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Caffeinated Coffee Consumption or Abstinence to Reduce Atrial Fibrillation

The DECAF Randomized Clinical Trial

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IMPORTANCE Conventional wisdom holds that caffeinated coffee is proarrhythmic. Coffee is the most commonly consumed caffeinated beverage in the US, and a randomized trial assessing caffeinated coffee consumption in patients with atrial fibrillation (AF) has not previously been performed.

OBJECTIVE To determine the effect of caffeinated coffee consumption compared with abstinence from coffee and caffeine on recurrent AF.

DESIGN, SETTING, AND PARTICIPANTS This was a prospective, open-label, randomized clinical trial enrolling 200 current or previous (within past 5 years) coffee-drinking adults with persistent AF, or atrial flutter with a history of AF, planned for electrical cardioversion from 5 hospitals in the US, Canada, and Australia between November 2021 and December 2024. The date of final follow-up was June 5, 2025.

INTERVENTION Patients were randomized in a 1:1 ratio to regular caffeinated coffee consumption vs coffee and caffeine abstinence for 6 months. Patients in the coffee consumption group were encouraged to drink at least 1 cup of caffeinated coffee daily. Patients in the abstinence group were encouraged to completely abstain from both caffeinated and decaffeinated coffee and other caffeine-containing products.

MAIN OUTCOMES AND MEASURES The primary end point was clinically detected recurrence of AF or atrial flutter over 6 months.

RESULTS Two hundred patients (mean [SD] age, 69 [11] years; 71% male) were randomized to caffeinated coffee consumption ($n = 100$) or coffee abstinence ($n = 100$). Baseline coffee intake was 7 cups (IQR, 7-18) per week in both groups. During follow-up, coffee intake in the consumption and abstinence groups was 7 (IQR, 6-11) and 0 (IQR, 0-2) cups per week, respectively, resulting in a between-group difference of 7 cups (95% CI, 7-7) per week. In the primary analysis, AF or atrial flutter recurrence was less in the coffee consumption (47%) than the coffee abstinence (64%) group, resulting in a 39% lower hazard of recurrence (hazard ratio, 0.61 [95% CI, 0.42-0.89]; $P = .01$). A comparable benefit of coffee consumption was observed with AF recurrence only. There was no significant difference in adverse events.

CONCLUSIONS AND RELEVANCE In this clinical trial of coffee drinkers after successful cardioversion, allocation to consumption of caffeinated coffee averaging 1 cup a day was associated with less recurrence of AF or atrial flutter compared with abstinence from coffee and caffeinated products.

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- + [Visual Abstract](#)
- + [Research Summary](#)
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Atrial fibrillation (AF) is the most common heart rhythm disorder. The prevalence of AF is rising, is estimated to affect up to 1 in 3 people during life, and is currently diagnosed in more than 10.5 million in the US alone.^{1,2} There is thus increasing interest in modifiable risk factors to reduce the burden of AF.³ Coffee is one of the most ubiquitously consumed substances and contains multiple active ingredients, the most recognized of which is caffeine. Indeed, coffee is the most commonly consumed caffeinated substance in the US.^{4,5} However, despite its widespread use, whether coffee has a beneficial, detrimental, or neutral effect on AF continues to be debated.

Caffeinated coffee has traditionally been considered proarrhythmic.⁶ It is commonly nominated by patients to be a frequent trigger for AF episodes, and physicians continue to advise that coffee reduction may minimize the effects of AF.^{7,8} In contrast, the recent Coffee and Real-time Atrial and Ventricular Ectopy (CRAVE) randomized trial found that caffeinated coffee consumption did not result in more premature atrial contractions that are known to trigger AF episodes.⁹⁻¹¹ Similarly, observational studies have generally reported no heightened or even a lower risk of AF among those who consumed coffee.¹²⁻¹⁶ However, observational studies are prone to confounding, and whether these findings are biased by systematic differences between coffee and noncoffee drinkers is unclear. An accurate understanding of any effect of caffeinated coffee on AF would be of great interest to patients and physicians alike. Thus, the current randomized clinical trial compared caffeinated coffee consumption vs abstinence from coffee and caffeine in patients with AF.

Methods

Study Design

The DECAF (Does Eliminating Coffee Avoid Fibrillation?) trial was an investigator-initiated, prospective, open-label, international, multicenter, randomized clinical trial. Patients were recruited from 5 tertiary hospitals in the US, Australia, and Canada. The trial was prospectively registered with ClinicalTrials.gov ([NCT05121519](#)) and approved by the University of California San Francisco, University of Adelaide, and University of Toronto institutional review boards. The trial protocol is in [Supplement 1](#). The trial is reported in accordance with the Consolidated Standards of Reporting Trials ([CONSORT](#)) guidelines.

Study Population

Patients meeting inclusion criteria were invited to participate and provided written informed consent ([Figure 1](#)). The inclusion criteria were age 21 years or older, sustained AF (or atrial flutter with a history of AF), planned direct current electrical cardioversion, coffee consumption of 1 cup per day or greater sometime in the past 5 years, willingness and ability to adhere to coffee abstinence or continuation, and life expectancy of at least 6 months. Patients who were not currently drinking coffee but otherwise met inclusion criteria were eligible. Key exclusion criteria were an established or adverse reaction to coffee, stated inability to adhere to coffee abstinence or continuation, AF ablation or cardiothoracic surgery

Key Points

Question Does consumption of caffeinated coffee have a beneficial, detrimental, or neutral effect on the risk of recurrent atrial fibrillation (AF) episodes?

Findings In this multicenter randomized clinical trial including 200 patients with persistent AF undergoing cardioversion, the risk of recurrent AF was significantly lower in the group allocated to coffee consumption (47%) compared with the abstinence group (64%).

Meaning Consumption of coffee and other caffeinated products may be reasonably considered in patients with AF.

within 3 months, and pregnancy or desire to conceive within 6 months. Baseline characteristics were determined by medical record review, and race, ethnicity, and lifestyle habits were determined by participant self-report.

Randomization

Because some patients have unsuccessful cardioversion or early recurrence of AF, randomization occurred only after sustained successful cardioversion. Patients were randomized 1:1 with stratification by study site to either caffeinated coffee consumption or abstinence from coffee and caffeine. If allocated to caffeinated coffee consumption, patients were encouraged to drink at least 1 cup of caffeinated coffee (or at least 1 espresso shot) and other caffeine-containing products every day as per their usual lifestyle. It was recommended that patients in the coffee consumption group not intentionally increase or decrease consumption of coffee or other caffeine-containing products. If allocated to the abstinence group, patients were encouraged to completely abstain from coffee, including decaffeinated coffee, and other caffeine-containing products.

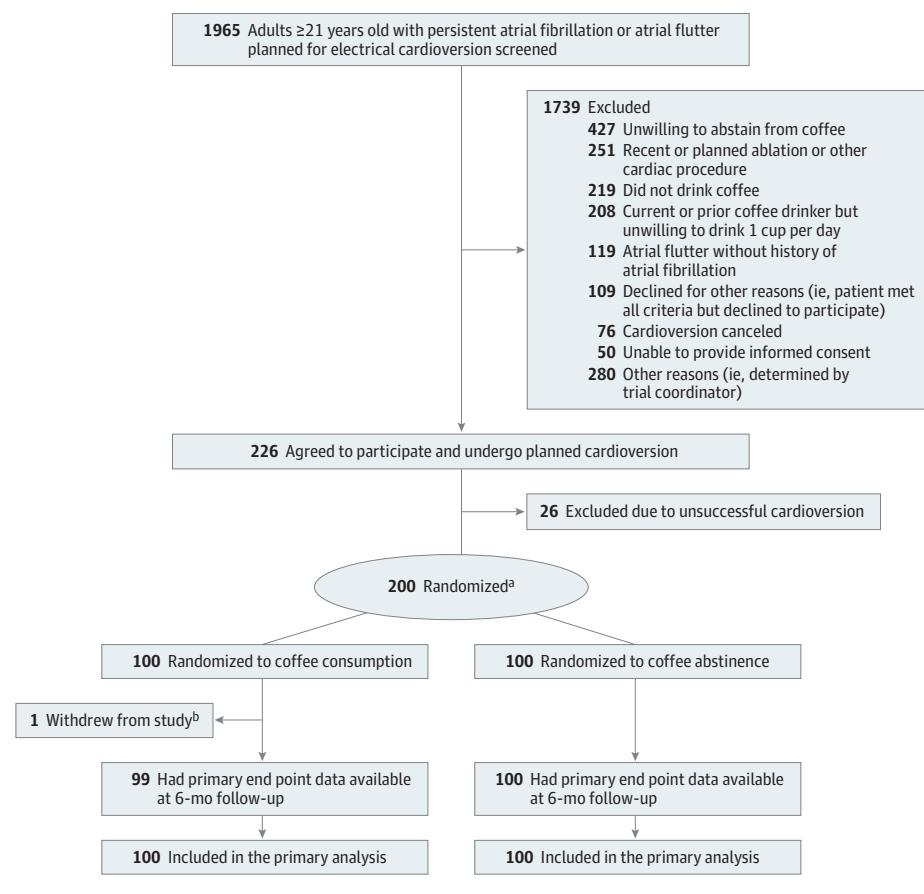
Follow-Up

Patients had study follow-up via telephone, video conference, or in-person at months 1, 3, and 6 where the randomized allocation was reinforced and the following ascertained: coffee and other caffeinated product consumption, medical history, medication inventory, recurrence of AF or atrial flutter, any consumer electrocardiogram data, and adverse events. The type and frequency of clinical follow-up and investigations were at the discretion of patients' regular physicians. All clinical health care documentation from regular physicians and electronic medical records, including all electrocardiograms (ECGs), wearable ECG monitors, and/or implantable cardiac device data, was reviewed to ascertain the primary end point of AF or atrial flutter recurrence and secondary end points. Every attempt was made to collect data for both primary and secondary end points until study censorship or the end of the 6-month follow-up period for all randomized participants.

Primary and Secondary End Points

The primary end point in an intention-to-treat analysis was clinically detected recurrence of AF or atrial flutter lasting 30 seconds or longer assessed in a time-to-event analysis. All episodes of AF or atrial flutter required confirmation by physician

Figure 1. Screening, Randomization, and Follow-Up in the DECAF Study



DECAF indicates Does Eliminating Coffee Avoid Fibrillation.

^aRandomization was stratified by study site.

^bThe 1 patient who withdrew (randomized to the coffee consumption group) did so on day 3 following study enrollment.

interpretation of an ECG, wearable ECG monitor, or implantable cardiac device electrograms. Prespecified secondary end points were the recurrence of AF and atrial flutter separately and adverse events before censorship (including myocardial infarction, stroke, heart failure exacerbation, emergency department visit, hospitalization, and death).

Statistical Analysis

For sample-size calculations, we assumed a 50% incidence of AF recurrence within 6 months following cardioversion.^{17,18} A clinically relevant effect size was assumed to approximate the effectiveness of commonly prescribed antiarrhythmic drugs for recurrent AF after cardioversion.¹⁸⁻²¹ To provide 80% power to detect a minimum 41% reduced relative hazard of AF, we enrolled 200 patients (100 per group) assuming a 1:1 randomization scheme, potential 10% loss to follow-up, and .05 2-tailed α level (eTable 1 in *Supplement 2*).

Baseline characteristics were summarized by randomization assignment. Continuous variables were described with means and SDs or medians and IQRs appropriate to distribution. Categorical variables were summarized with counts and percentages.

The primary end point of clinically detected recurrence of AF or atrial flutter lasting 30 seconds or longer was analyzed as a time-to-event outcome. Kaplan-Meier plots were

used to descriptively compare the survival distributions of the primary end point by randomization group. Cox proportional hazards regression models were used to formally analyze the primary end point with prespecified adjustment for study site (used in the randomization process as a stratification factor). In prespecified sensitivity analyses, we additionally adjusted for other baseline characteristics that were either prognostic factors or imbalanced between the groups. The proportional hazards assumption was assessed with no strong evidence of violation found (eMethods and eFigure 1 in *Supplement 2*). Point estimates for hazard ratios (HRs), Wald-based 95% confidence intervals (CIs), and P values were reported. The primary intention-to-treat analysis included all participants regardless of adherence with randomized allocation. To explore the consistency of the treatment effect with respect to the primary end point, subgroup analyses were undertaken. The trial was not powered to compare the treatment effects across subgroups. CIs and interaction-related P values were not adjusted for multiplicity. For secondary end points, AF and atrial flutter recurrence separately were assessed with Cox hazard regression models, and adverse events compared with χ^2 tests. Statistical analyses were conducted using SAS software, version 9.4 (SAS Institute). A 2-tailed P value of .05 was considered statistically significant. The last protocol and statistical analysis

Table 1. Baseline Patient Characteristics

Variable	Coffee consumption (n = 100)	Coffee abstinence (n = 100)
Demographics		
Age, mean (SD), y	68.2 (12.3)	70.4 (10.1)
Sex, No. (%)		
Male	76 (76)	65 (65)
Female	24 (24)	35 (35)
Height, mean (SD), cm	175.7 (9.7)	174.0 (10.1)
Weight, mean (SD), kg	94.1 (28.5)	92.5 (25.4)
Body mass index, mean (SD) ^a	30.3 (7.9)	30.4 (7.5)
Race, No. (%)	n = 99	n = 96
American Indian/Alaska Native	1 (1)	0
Asian	9 (9)	7 (7)
Black or African American	3 (3)	1 (1)
More than 1 race	4 (4)	0
Native Hawaiian or Other Pacific Islander	2 (2)	0
Other ^b	4 (4)	4 (4)
White	76 (77)	84 (88)
Ethnicity, No. (%)	n = 65	n = 60
Hispanic or Latino	8 (12)	5 (8)
Not Hispanic or Latino	57 (88)	55 (92)
Education, No. (%)		
>Undergraduate degree	46 (46)	42 (42)
Some college	18 (18)	12 (12)
≤High school	29 (29)	41 (41)
Other ^b	7 (7)	5 (5)
Medical history, No. (%) ^c		
Hypertension	65 (65)	63 (63)
Obstructive sleep apnea	29 (29)	26 (26)
Heart failure	23 (23)	21 (21)
Cardiomyopathy	17 (17)	21 (21)
Diabetes	14 (14)	14 (14)
Stroke or transient ischemic attack	10 (10)	9 (9)
Coronary artery disease	9 (9)	16 (16)
Permanent pacemaker	9 (9)	9 (9)
Implantable cardioverter defibrillator	6 (6)	7 (7)
Myocardial infarction	3 (3)	4 (4)
Peripheral vascular disease	2 (2)	3 (3)
AF history		
History of paroxysmal AF, No. (%) ^d	25 (25)	35 (35)
Coexisting atrial flutter, No. (%)	3 (3)	8 (8)
Time since first diagnosis of AF, median (IQR), y	2.4 (0.4-6.9)	2.6 (0.4-6.1)
Duration of current AF episode, median (IQR), d ^e	60 (22-143)	60 (22-150)
CHA ₂ DS ₂ -VASC score, mean (SD) ^f	2.5 (1.7)	2.6 (1.5)
AF symptom score, No. (%) ^g		
No physical limitation	52 (52)	57 (57)
Slight physical limitation	33 (33)	25 (25)
Marked physical limitation	14 (14)	17 (17)
Severe symptoms at rest	1 (1)	1 (1)
Previous AF ablation	20 (20)	12 (12)

(continued)

Table 1. Baseline Patient Characteristics (continued)

Variable	Coffee consumption (n = 100)	Coffee abstinence (n = 100)
Medication history, No. (%)		
Non-vitamin K antagonist oral anticoagulant	96 (96)	91 (91)
β-Blocker	71 (71)	70 (70)
Antiarrhythmic therapy ^h	53 (53)	51 (51)
Class IC	13 (13)	10 (10)
Class III	40 (40)	41 (41)
Digoxin	9 (9)	4 (4)
Non-dihydropyridine calcium channel blockers	5 (5)	8 (8)
Lifestyle history, No. (%)		
Coffee and other caffeinated products, No. (%) ⁱ		
Drip coffee consumption	49 (49)	45 (45)
Espresso drink consumption	37 (37)	36 (36)
Tea consumption	37 (37)	28 (28)
Chocolate	28 (28)	28 (28)
Decaffeinated coffee consumption	7 (7)	11 (11)
Baseline coffee consumption, median (IQR), cups/week ^j	7 (7-18)	7 (7-18)
Patient-reported coffee never triggering AF, No. (%)	60 (60)	65 (65)
Patient-reported no coffee abstinence symptoms, No. (%)	61 (61)	64 (64)
Prior physician advice to decrease coffee, No. (%)	13 (13)	19 (19)
Alcohol consumption, No. of drinks/wk		
None	46 (46)	47 (47)
<1	8 (8)	9 (9)
1-3	21 (21)	19 (19)
4-7	12 (12)	12 (12)
8-14	7 (7)	6 (6)
15-21	1 (1)	5 (5)
>21	5 (5)	2 (2)
Investigations		
Left ventricular ejection fraction, mean (SD), %	54.0 (10.9)	53.6 (14.2)
Left atrial volume index, mean (SD), mL/m ²	46.2 (15.1)	44.5 (14.9)

Abbreviations: AF, atrial fibrillation; CHA₂DS₂-VASC, congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, stroke or transient ischemic attack or thromboembolism, vascular disease, age 65-74 years, and female sex.

^a Calculated as weight in kilograms divided by height in meters squared

^b The other category includes the option of unknown or not reported.

^c Medical history was determined by a combination of medical record review and patient self-report.

^d These patients had a pattern of paroxysmal AF prior to the current episode of persistent AF that led to them being planned for electrical cardioversion.

^e The duration of the current AF episode was determined by the treating physician to the best of their knowledge based on symptoms and/or documented AF.

^f The CHA₂DS₂-VASC score is a measure of the risk of stroke among persons with AF; scores range from 0 to 9, with higher scores indicating a greater risk.

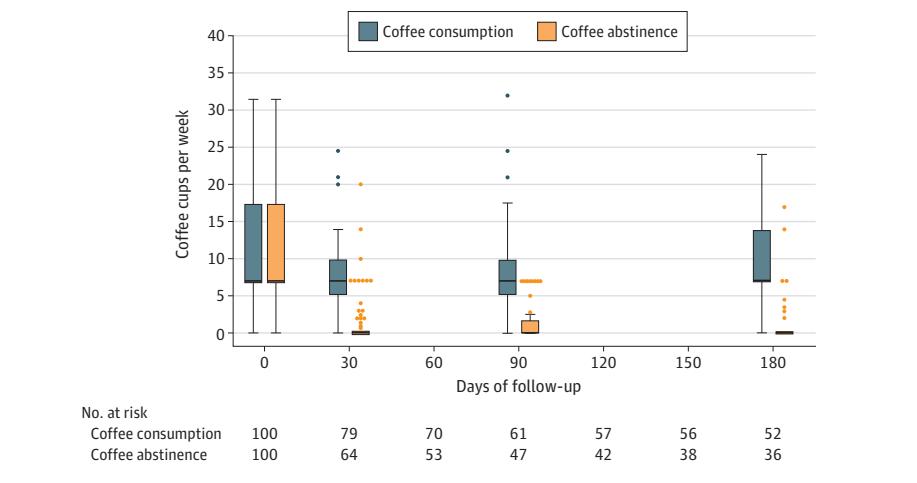
^g An indication of AF symptom severity was self-reported from these categorical options.

^h Class IC agents included flecainide and propafenone, and class III agents included sotalol, dofetilide, amiodarone, and dronedarone.

ⁱ See eTables 4 and 5 in Supplement 2 for more details on consumption of coffee and other caffeinated products.

^j Coffee intake was self-reported in standard cups (8 oz or 240 mL).

Figure 2. Changes in Coffee Intake by Randomization Group



Box-and-whisker plots showing changes in coffee intake among patients in the caffeinated coffee consumption group and the coffee abstinence group. Coffee intake was self-reported in standard cups (approximately 8 oz or 240 mL). The bottom edge, middle black line, and top edge of the boxes represent the 25th percentile, median, and 75th percentile values, respectively. The whiskers are plotted using the Tukey method, extending from the 25th percentile minus 1.5 times the IQR to the 75th percentile plus 1.5 times the IQR. Some of the values for the boxes and whiskers are similar or identical in several plots. Values outside the whiskers are plotted as individual points.

plan were finalized before the database was locked and data analysis commenced.

Results

Patient Characteristics

Of 1965 potential patients who were screened for participation, 1739 did not meet eligibility criteria and were excluded (Figure 1). One-quarter of patients screened were not willing to abstain from coffee, caffeine, or both (427 patients, 25%). Due to either personal preference or physician advice, another one-quarter of screened patients were either current or prior coffee drinkers but unwilling to consume at least 1 cup per day (208 patients, 12%) or did not drink coffee (219 patients, 13%). After a further 26 patients were excluded due to unsuccessful cardioversion, a total of 200 patients were randomly assigned to consumption of caffeinated coffee (100 patients) or abstinence from coffee and caffeine (100 patients). The majority were randomized on the day of cardioversion (eTable 2 in *Supplement 2*). Except for 1 patient withdrawal, all patients had primary and secondary end point data available at 6 months' follow-up.

Baseline characteristics were largely balanced between the groups, although there were numerical imbalances in sex, coronary artery disease, previous AF ablation, coexisting atrial flutter, and history of paroxysmal AF (Table 1). Approximately half of patients (52%) were taking antiarrhythmic medication (Table 1; eTable 3 in *Supplement 2*).

Coffee Intake and Other Caffeinated Products

Baseline coffee intake was similar between the groups, with the most frequent amount of coffee intake being 7 cups per week, or 1 cup per day (40%; Figure 2; eTable 4 in *Supplement 2*). Coffee intake did not significantly change in the coffee consumption group over the trial period, with baseline and follow-up intake of 7 (IQR, 7-18) and 7 (IQR, 6-11) cups per week, respectively (percentage change, 0% [IQR, -33% to 14%]; Figure 2; eFigure 2 in *Supplement 2*). Patients in the coffee ab-

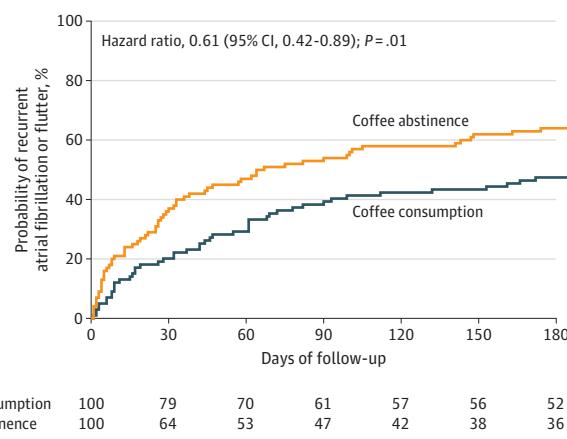
stinence group reduced their coffee intake from 7 (IQR, 7-18) to 0 (IQR, 0-2) cups per week (percentage change, -100% [IQR, -100% to -77%]). As a result, there was a between-group difference in coffee intake of 7 (95% CI, 7-7), 7 (95% CI, 6-7), and 7 (95% CI, 7-7) cups per week at months 1, 3, and 6, respectively, and a between-group difference of 7 cups (95% CI, 7-7) per week over the entire trial period. Intake of other caffeinated products, such as tea, chocolate, energy drinks, soda, and decaffeinated coffee, was numerically higher in the coffee consumption group during the trial period. However, most were not significantly different, except for the addition of sugar to coffee, which was greater in the consumption group (eTable 5 in *Supplement 2*).

Follow-Up and Primary End Point

In addition to 1-, 3-, and 6-month study follow-ups, patients had a mean (SD) of 5.8 (6.5) clinical and 1.9 (2.0) cardiology health care encounters, including 2.1 (2.3) ECGs, during the study period (eTable 6 in *Supplement 2*). A majority (53%) had a continuous recording device during follow-up, such as a consumer device, wearable ECG monitor, and/or implanted cardiac device (eTable 7 in *Supplement 2*).

At 6 months' follow-up, recurrence of AF or atrial flutter was documented in 111 patients (56%), representing 47 patients (47%) in the caffeinated coffee consumption group and 64 patients (64%) in the abstinence group (eTables 8, 9, and 10 in *Supplement 2*). In the primary intention-to-treat analysis adjusted for study site, the time to recurrence was longer in the coffee consumption group than in the abstinence group (Figure 3), resulting in a 39% lower hazard of AF or atrial flutter recurrence with coffee consumption (HR, 0.61 [95% CI, 0.42-0.89]; $P = .01$). A similar benefit for caffeinated coffee consumption was also observed in prespecified sensitivity analyses adjusting for baseline characteristics that were either prognostic factors and/or numerically imbalanced between the groups (eTable 11 in *Supplement 2*). This benefit appeared to be consistent across most analyses of subgroups except for ablation history ($P = .04$), though this was not adjusted for multiplicity and should be interpreted cautiously (eTable 12 in *Supplement 2*).

Figure 3. Time to Recurrence of Atrial Fibrillation or Flutter



Kaplan-Meier survival curve showing the probability of recurrent atrial fibrillation or flutter in the coffee consumption and abstinence groups. All patients in both groups were followed up until recurrence or 6 months' follow-up. Hazard ratio and *P* value are from a Cox proportional hazard regression model including prespecified adjustment for study site used in the randomization process.

Table 2. Adverse Events^a

Variable	Coffee consumption (n = 100)	Coffee abstinence (n = 100)
Emergency department visits	13	16
Any cardiovascular	7	8
AF or atrial flutter related	5	5
Heart failure	1	2
Myocardial infarction	2	0
Syncope	0	2
Stroke or transient ischemic attack	0	0
Hospitalizations	23	21
Any cardiovascular	17	17
AF or atrial flutter related	10	15
Heart failure	1	2
Myocardial infarction	2	0
Syncope	0	1
Stroke or transient ischemic attack	0	0
Death	0	0

Abbreviation: AF, atrial fibrillation.

^a Numbers represent participants with at least 1 such event. Categories are not exclusive.

Both groups had a similar proportion of recurrent AF or atrial flutter detected by a consumer device, pacemaker or defibrillator with atrial lead, or implantable loop recorder. However, more patients in the abstinence group (78%) had their AF detected by a 12-lead ECG compared with the coffee consumption group (57%), and more in the coffee consumption group (21%) had their AF detected by a wearable ECG monitor compared with the abstinence group (0%) (eTable 9 in *Supplement 2*).

Secondary Analyses

There was a similar benefit of caffeinated coffee consumption on the secondary end point of AF recurrence only (HR, 0.62 [95% CI, 0.43-0.91]; *P* = .01; eFigure 3 and eTable 13 in *Supple-*

ment 2). A lower hazard of atrial flutter recurrence was also seen with caffeinated coffee consumption, although atrial flutter recurrence occurred in only 6% of patients and the between-group difference did not reach statistical significance (HR, 0.37 [95% CI, 0.10-1.41]; *P* = .14; eFigure 4 and eTable 13 in *Supplement 2*). There were numerically more AF or atrial flutter-related hospitalizations in the abstinence compared with the consumption group (15 vs 10, respectively), with other adverse events similar between the groups (Table 2).

Discussion

In this randomized clinical trial of patients with persistent AF undergoing cardioversion, consumption of caffeinated coffee compared with abstinence from coffee and caffeine was associated with a significantly lower recurrence of AF or atrial flutter. The current results contrast with the traditional assumption that coffee promotes atrial arrhythmogenesis, but fit with some observational data on the subject.¹²⁻¹⁶ These findings could be considered in patients with AF and a personal preference for consumption of coffee and other caffeinated products.

Conventional wisdom has held that caffeinated coffee is proarrhythmic.^{6,8,22} In contrast, most¹²⁻¹⁶ (although not all²³) observational studies have suggested a neutral or beneficial association of coffee with AF. However, nonrandomized studies are prone to residual confounding that cannot be definitely excluded. The present trial, where measured and unmeasured confounders should have been balanced via randomized allocation, suggests that caffeinated coffee consumption may reduce recurrence of persistent AF compared with abstinence from coffee and other caffeinated products. Moreover, a continued separation of survival curves over time implies that this difference may be more attributable to a benefit of coffee consumption rather than harm from abrupt coffee cessation and withdrawal.

Several mechanisms may be responsible for these findings. Coffee contains numerous biological compounds, the most recognized being caffeine. Some data suggest that caffeinated

but not decaffeinated coffee is associated with reduced AF.¹³ Caffeine concentrations associated with typical coffee consumption result in blockade of A₁ and A_{2a} adenosine receptors.²⁴ Adenosine facilitates AF induction, an effect thought to be due to sympathoexcitatory effects, shortening of atrial refractoriness, and ectopic triggers.^{25,26} Caffeine may thus have adenosine-mediated antiarrhythmic properties, as has been suggested in a canine study on AF inducibility.²⁷

Coffee also appears to have anti-inflammatory properties.^{28,29} As systemic inflammation is an AF risk factor, coffee might reduce AF risk by decreasing inflammation.³⁰⁻³² AF can be vagally mediated, and perhaps the catecholaminergic effects of caffeine are therefore protective.^{33,34} Caffeine is also a diuretic, potentially reducing blood pressure and AF risk.^{35,36}

Coffee consumption may also influence other relevant behaviors. Coffee drinkers may consume fewer unhealthy drinks.³⁷ However, soda intake was greater in the coffee consumption group. Similarly, adding sugar to coffee was expectedly greater in the consumption group. These differences would, if anything, be expected to result in greater obesity, diabetes, and AF.^{38,39} Conversely, random assignment to coffee consumption in the CRAVE trial resulted in approximately 1000 more steps each day.⁹ Physical activity reduced AF recurrence in other trials^{40,41} and, in addition to other modifiable factors such as alcohol^{42,43} and obesity,⁴⁴ is now recommended as part of AF management.³ It seems plausible that greater physical activity may have been in part responsible for the benefit of coffee consumption seen.

It is worth emphasizing that the caffeine in this study was naturally occurring and coffee consumption was within normal ranges. It would be inappropriate to extrapolate any perceived benefits to high-dose caffeine, and particularly to synthetic products such as energy drinks that might contain other substances. However, given the apparent protective effects, strategies to encourage or even initiate caffeinated coffee as a proactive strategy to prevent AF may be worthwhile investigating.

Limitations

This trial had several limitations. First, it was pragmatic in design, and the primary end point was clinically detected AF or atrial flutter. While clinical follow-up was per usual with no loss to follow-up, and a majority of participants used continuous recording devices, there was no protocol-mandated AF detection method or schedule. However, follow-up and detection methods were similar between groups, except for wearable ECG monitors used more frequently in the coffee consumption group, which would have, if anything, likely biased results in the opposite direction to the current findings. Second, the sample size was modest, similar to other trials on lifestyle factors.⁴² Although the results were statistically sig-

nificant and appeared robust to sensitivity analyses, a chance finding remains possible.

Third, there were some numeric imbalances in baseline characteristics; while sensitivity analyses adjusting for these yielded similar results, the possibility that they influenced the current findings cannot be excluded. Fourth, the study was not blinded. While AF recurrence was detected and confirmed by treating physicians, and not by study coordinators, blinding would have minimized risk of bias. Similarly, participants were not blinded to the exposure, although this did allow for the full and real-life experience of consuming caffeinated coffee, and the study was not intended to identify specific mechanistic constituents.

Fifth, only a minority of screened patients were willing to participate. While many did not want to abstain from coffee for the length of this trial, many also believed that coffee worsened AF. While it is possible this was an effect of the long-held conventional wisdom, it remains possible that some select individuals are truly triggered by coffee. If so, this subpopulation may have been underrepresented. Of note, in the I-STOP-AFib trial, those who believed caffeine to be an acute trigger for AF did not demonstrate that effect in randomized N-of-1 trials, suggesting the belief may more likely stem from received (rather than evidence-based) wisdom.⁴⁵ Sixth, paroxysmal AF may not respond in the same manner as persistent AF to coffee and requires further study. Because patients with persistent AF were enrolled, the study did not seek to determine AF burden.

Seventh, adherence in the abstinence group was also suboptimal, with only 69% not consuming any coffee. However, this does raise the possibility that these results might underestimate the true magnitude of benefit from coffee. Self-reported coffee consumption data may be subject to misclassification in the form of recall bias. However, any nondifferential bias or differential bias more likely from those in the abstinence group underreporting actual coffee consumption would bias the current results to the null. Eighth, while no significant differences in adverse events were observed, this trial was not specifically powered to detect a difference in these less common events, although these results are consistent with neutral or beneficial observational associations of coffee with these outcomes.⁴⁶

Conclusions

Among previously coffee-drinking patients with AF referred for cardioversion, random allocation to typical consumption of caffeinated coffee averaging 1 cup a day was associated with less recurrence of AF and atrial flutter compared with abstinence from coffee and caffeinated products.

ARTICLE INFORMATION

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analyses and for the fidelity of the trial to the protocol.

Concept and design: Wong, Cheung, Olglin, Marcus. **Acquisition, analysis, or interpretation of data:** Wong, Cheung, Montenegro, Oo, Peña, Tang, Tu, Wall, Dewland, Moss, Gerstenfeld, Tseng, Hsia,

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REFERENCES

1. Kornej J, Börschel CS, Benjamin EJ, Schnabel RB. Epidemiology of atrial fibrillation in the 21st century: novel methods and new insights. *Circ Res*. 2020;127(1):4-20. doi:[10.1161/CIRCRESAHA.120.316340](https://doi.org/10.1161/CIRCRESAHA.120.316340)
2. Noubiap JJ, Tang JJ, Teraoka JT, Dewland TA, Marcus GM. Minimum national prevalence of diagnosed atrial fibrillation inferred from California acute care facilities. *J Am Coll Cardiol*. 2024;84(16):1501-1508. doi:[10.1016/j.jacc.2024.07.014](https://doi.org/10.1016/j.jacc.2024.07.014)
3. Chung MK, Eckhardt LL, Chen LY, et al; American Heart Association Electrocardiography and Arrhythmias Committee and Exercise, Cardiac Rehabilitation, and Secondary Prevention Committee of the Council on Clinical Cardiology; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular and Stroke Nursing; and Council on Lifestyle and Cardiometabolic Health. Lifestyle and risk factor modification for reduction of atrial fibrillation: a scientific statement from the American Heart Association. *Circulation*. 2020;141(16):e750-e772. doi:[10.1161/CIR.00000000000000748](https://doi.org/10.1161/CIR.00000000000000748)
4. Loftfield E, Freedman ND, Dodd KW, et al. Coffee drinking is widespread in the United States, but usual intake varies by key demographic and lifestyle factors. *J Nutr*. 2016;146(9):1762-1768. doi:[10.3945/jn.116.233940](https://doi.org/10.3945/jn.116.233940)
5. Mitchell DC, Knight CA, Hockenberry J, Teplansky R, Hartman TJ. Beverage caffeine intakes in the US. *Food Chem Toxicol*. 2014;63:136-142. doi:[10.1016/j.fct.2013.10.042](https://doi.org/10.1016/j.fct.2013.10.042)
6. Hughes JR, Amori G, Hatsuhami DK. A survey of physician advice about caffeine. *J Subst Abuse*. 1988;1(1):67-70. doi:[10.1016/S0899-3289\(88\)80009-9](https://doi.org/10.1016/S0899-3289(88)80009-9)
7. Groh CA, Faulkner M, Getabechia S, et al. Patient-reported triggers of paroxysmal atrial fibrillation. *Heart Rhythm*. 2019;16(7):996-1002. doi:[10.1016/j.hrthm.2019.01.027](https://doi.org/10.1016/j.hrthm.2019.01.027)
8. Joglar JA, Chung MK, Armbruster AL, et al; Peer Review Committee Members. 2023 ACC/AHA/ACCP/HRS guideline for the diagnosis and management of atrial fibrillation: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*. 2024;149(1):e1-e156. doi:[10.1161/CIR.0000000000001193](https://doi.org/10.1161/CIR.0000000000001193)
9. Marcus GM, Rosenthal DG, Nah G, et al. Acute effects of coffee consumption on health among ambulatory adults. *N Engl J Med*. 2023;388(12):1092-1100. doi:[10.1056/NEJMoa2204737](https://doi.org/10.1056/NEJMoa2204737)
10. Dewland TA, Vittinghoff E, Mandyam MC, et al. Atrial ectopy as a predictor of incident atrial fibrillation: a cohort study. *Ann Intern Med*. 2013;159(11):721-728. doi:[10.7326/0003-4819-159-11-201312030-00004](https://doi.org/10.7326/0003-4819-159-11-201312030-00004)
11. Haïssaguerre M, Jaïs P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med*. 1998;339(10):659-666. doi:[10.1056/NEJM19980903391003](https://doi.org/10.1056/NEJM19980903391003)
12. Kim EJ, Hoffmann TJ, Nah G, Vittinghoff E, Delling F, Marcus GM. Coffee consumption and incident tachyarrhythmias: reported behavior, mendelian randomization, and their interactions. *JAMA Intern Med*. 2021;181(9):1185-1193. doi:[10.1010/jamainternmed.2021.3616](https://doi.org/10.1010/jamainternmed.2021.3616)
13. Chieng D, Canovas R, Segan L, et al. The impact of coffee subtypes on incident cardiovascular disease, arrhythmias, and mortality: long-term outcomes from the UK Biobank. *Eur J Prev Cardiol*. 2022;29(17):2240-2249. doi:[10.1093/europc/zwac189](https://doi.org/10.1093/europc/zwac189)
14. Larsson SC, Drca N, Jensen-Urstad M, Wolk A. Coffee consumption is not associated with increased risk of atrial fibrillation: results from two prospective cohorts and a meta-analysis. *BMC Med*. 2015;13:207. doi:[10.1186/s12916-015-0447-8](https://doi.org/10.1186/s12916-015-0447-8)
15. Krittawong C, Tunhasirivat A, Wang Z, et al. Is caffeine or coffee consumption a risk for new-onset atrial fibrillation? a systematic review and meta-analysis. *Eur J Prev Cardiol*. 2021;28(12):e13-e15. doi:[10.1177/2047487320908385](https://doi.org/10.1177/2047487320908385)
16. Cheng M, Hu Z, Lu X, Huang J, Gu D. Caffeine intake and atrial fibrillation incidence: dose response meta-analysis of prospective cohort studies. *Can J Cardiol*. 2014;30(4):448-454. doi:[10.1016/j.cjca.2013.12.026](https://doi.org/10.1016/j.cjca.2013.12.026)
17. Pluymakers NAHA, Dudink EAMP, Luermans JGLM, et al; RACE 7 ACWAS Investigators. Early or delayed cardioversion in recent-onset atrial fibrillation. *N Engl J Med*. 2019;380(16):1499-1508. doi:[10.1056/NEJMoa1900353](https://doi.org/10.1056/NEJMoa1900353)
18. Singh BN, Singh SN, Reda DJ, et al; Sotalol Amiodarone Atrial Fibrillation Efficacy Trial (SAFE-T) Investigators. Amiodarone versus sotalol for atrial fibrillation. *N Engl J Med*. 2005;352(18):1861-1872. doi:[10.1056/NEJMoa041705](https://doi.org/10.1056/NEJMoa041705)
19. Singh BN, Connolly SJ, Crijns HJ, et al; EURIDIS and ADONIS Investigators. Dronedarone for maintenance of sinus rhythm in atrial fibrillation or flutter. *N Engl J Med*. 2007;357(10):987-999. doi:[10.1056/NEJMoa054686](https://doi.org/10.1056/NEJMoa054686)
20. Fetsch T, Bauer P, Engberding R, et al; Prevention of Atrial Fibrillation after Cardioversion Investigators. Prevention of atrial fibrillation after cardioversion: results of the PAFAC trial. *Eur Heart J*. 2004;25(16):1385-1394. doi:[10.1016/j.ehj.2004.04.015](https://doi.org/10.1016/j.ehj.2004.04.015)
21. Kochiadakis GE, Igoumenidis NE, Hamilos ME, et al. Sotalol versus propafenone for long-term maintenance of normal sinus rhythm in patients with recurrent symptomatic atrial fibrillation. *Am J Cardiol*. 2004;94(12):1563-1566. doi:[10.1016/j.amjcard.2004.08.041](https://doi.org/10.1016/j.amjcard.2004.08.041)
22. Hansson A, Madsen-Härdig B, Olsson SB. Arrhythmia-provoking factors and symptoms at the onset of paroxysmal atrial fibrillation: a study based on interviews with 100 patients seeking hospital assistance. *BMC Cardiovasc Disord*. 2004;4:13. doi:[10.1186/1471-2261-4-13](https://doi.org/10.1186/1471-2261-4-13)
23. Sehrawat O, Mehra NS, Kowlgi NG, et al. Association between coffee consumption and incident atrial fibrillation (from the Multi-Ethnic Study of Atherosclerosis [MESA]). *Am J Cardiol*. 2023;186:5-10. doi:[10.1016/j.amjcard.2022.10.025](https://doi.org/10.1016/j.amjcard.2022.10.025)

24. Conlay LA, Conant JA, deBros F, Wurtman R. Caffeine alters plasma adenosine levels. *Nature*. 1997;389(6647):136. doi:10.1038/38160

25. Ip JE, Cheung JW, Chung JH, et al. Adenosine-induced atrial fibrillation: insights into mechanism. *Circ Arrhythm Electrophysiol*. 2013;6(3):e34-e37. doi:10.1161/CIRCEP.113.000480

26. Kabell G, Buchanan LV, Gibson JK, Belardinelli L. Effects of adenosine on atrial refractoriness and arrhythmias. *Cardiovasc Res*. 1994;28(9):1385-1389. doi:10.1093/cvr/28.9.1385

27. Rashid A, Hines M, Scherlag BJ, Yamanashi WS, Lovallo W. The effects of caffeine on the inducibility of atrial fibrillation. *J Electrocardiol*. 2006;39(4):421-425. doi:10.1016/j.jelectrocard.2005.12.007

28. Jung S, Kim MH, Park JH, Jeong Y, Ko KS. Cellular antioxidant and anti-inflammatory effects of coffee extracts with different roasting levels. *J Med Food*. 2017;20(6):626-635. doi:10.1089/jmf.2017.3935

29. Tajik N, Tajik M, Mack I, Enck P. The potential effects of chlorogenic acid, the main phenolic components in coffee, on health: a comprehensive review of the literature. *Eur J Nutr*. 2017;56(7):2215-2244. doi:10.1007/s00394-017-1379-1

30. Marcus GM, Whooley MA, Glidden DV, Pawlikowska L, Zaroff JG, Olglin JE. Interleukin-6 and atrial fibrillation in patients with coronary artery disease: data from the Heart and Soul Study. *Am Heart J*. 2008;155(2):303-309. doi:10.1016/j.ahj.2007.09.006

31. Chung MK, Martin DO, Sprecher D, et al. C-reactive protein elevation in patients with atrial arrhythmias: inflammatory mechanisms and persistence of atrial fibrillation. *Circulation*. 2001;104(24):2886-2891. doi:10.1161/hc4901.101760

32. Aviles RJ, Martin DO, Apperson-Hansen C, et al. Inflammation as a risk factor for atrial fibrillation. *Circulation*. 2003;108(24):3006-3010. doi:10.1161/01.CIR.0000103131.70301.4F

33. Mandyam MC, Vedantham V, Scheinman MM, et al. Alcohol and vagal tone as triggers for paroxysmal atrial fibrillation. *Am J Cardiol*. 2012;110(3):364-368. doi:10.1016/j.amjcard.2012.03.033

34. Hou Y, Scherlag BJ, Lin J, et al. Ganglionated plexi modulate extrinsic cardiac autonomic nerve input: effects on sinus rate, atrioventricular conduction, refractoriness, and inducibility of atrial fibrillation. *J Am Coll Cardiol*. 2007;50(1):61-68. doi:10.1016/j.jacc.2007.02.066

35. Seal AD, Bardis CN, Gavrieli A, et al. Coffee with high but not low caffeine content augments fluid and electrolyte excretion at rest. *Front Nutr*. 2017;4:40. doi:10.3389/fnut.2017.00040

36. Rieg T, Steigle H, Schnermann J, Richter K, Osswald H, Vallon V. Requirement of intact adenosine A1 receptors for the diuretic and natriuretic action of the methylxanthines theophylline and caffeine. *J Pharmacol Exp Ther*. 2005;313(1):403-409. doi:10.1124/jpet.104.080432

37. Dewland TA, van Dam RM, Marcus GM. Coffee and cardiovascular disease. *Eur Heart J*. 2025;46(36):3546-3554. doi:10.1093/eurheartj/ehaf421

38. Tu SJ, Gallagher C, Elliott AD, et al. Associations of dietary patterns, ultra-processed food and nutrient intake with incident atrial fibrillation. *Heart*. 2023;109(22):1683-1689. doi:10.1136/heartjnl-2023-322412

39. Henn M, Glenn AJ, Willett WC, Martínez-González MA, Sun Q, Hu FB. Coffee consumption, additive use, and risk of type 2 diabetes-results from 3 large prospective United States cohort studies. *Am J Clin Nutr*. 2025;121(3):695-702. doi:10.1016/j.ajcnut.2025.01.017

40. Elliott AD, Verdicchio CV, Mahajan R, et al. An exercise and physical activity program in patients with atrial fibrillation: the ACTIVE-AF randomized controlled trial. *JACC Clin Electrophysiol*. 2023;9(4):455-465. doi:10.1016/j.jacep.2022.12.002

41. Malmo V, Nes BM, Amundsen BH, et al. Aerobic interval training reduces the burden of atrial fibrillation in the short term: a randomized trial. *Circulation*. 2016;133(5):466-473. doi:10.1161/CIRCULATIONAHA.115.018220

42. Voskoboinik A, Kalman JM, De Silva A, et al. Alcohol abstinence in drinkers with atrial fibrillation. *N Engl J Med*. 2020;382(1):20-28. doi:10.1056/NEJMoa1817591

43. Wong CX, Tu SJ, Marcus GM. Alcohol and arrhythmias. *JACC Clin Electrophysiol*. 2023;9(2):266-279. doi:10.1016/j.jacep.2022.10.023

44. Wong CX, Sun MT, Odutayo A, et al. Associations of epicardial, abdominal, and overall adiposity with atrial fibrillation. *Circ Arrhythm Electrophysiol*. 2016;9(12):e004378. doi:10.1161/CIRCEP.116.004378

45. Marcus GM, Modrow MF, Schmid CH, et al. Individualized studies of triggers of paroxysmal atrial fibrillation: the I-STOP-AFib randomized clinical trial. *JAMA Cardiol*. 2022;7(2):167-174. doi:10.1001/jamacardio.2021.5010

46. Freedman ND, Park Y, Abnet CC, Hollenbeck AR, Sinha R. Association of coffee drinking with total and cause-specific mortality. *N Engl J Med*. 2012;366(20):1891-1904. doi:10.1056/NEJMoa1112010