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STATE OF THE ART REVIEW
Arrhythmias

Diet and risk of atrial fibrillation: a systematic review

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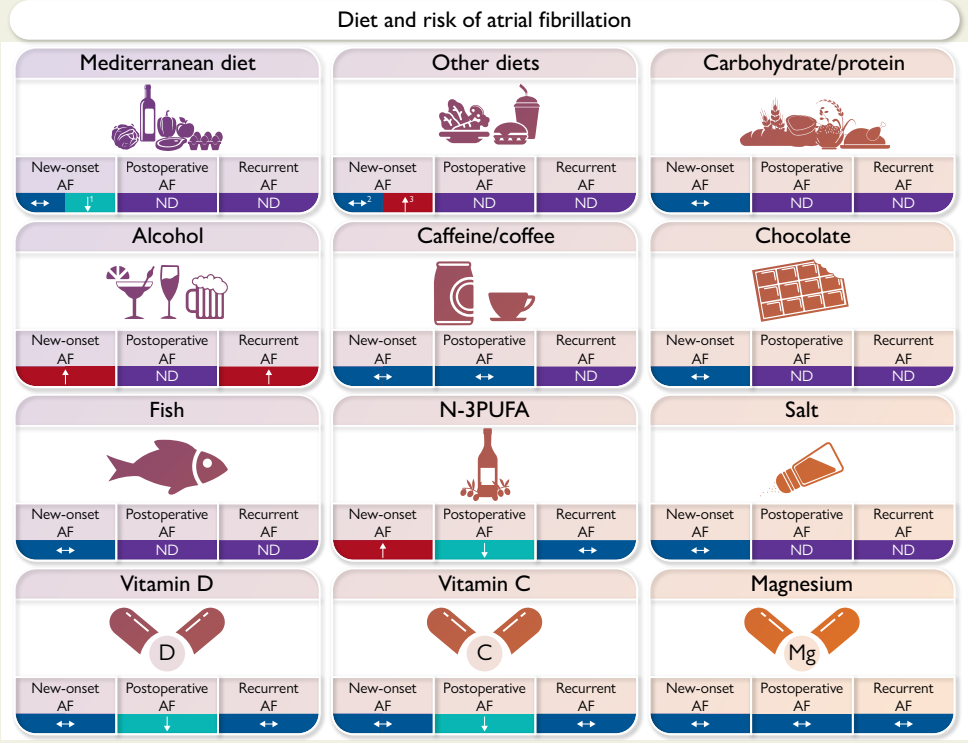
Graphical Abstract



Association between the most studied dietary patterns/components and atrial fibrillation. [1]enriched with extra virgin olive oil; [2]plant-based and Dietary Approaches to Stop Hypertension (DASH) diets; [3]ultra-processed food diet. AF, atrial fibrillation; MED-DIET, Mediterranean diet; ND, no data; PUFA, polyunsaturated fatty acids; ↔, neutral impact; ↑, increased risk; ↓, decreased risk.

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Abstract

Atrial fibrillation (AF) is the most prevalent sustained cardiac arrhythmia. Comprehensive modification of established AF risk factors combined with dietary interventions and breaking deleterious habits has been shown to reduce AF burden and recurrence. Numerous AF risk factors, such as diabetes, obesity or hypertension can be partially related to dietary and lifestyle choices. Therefore, dietary interventions may have potential as a therapeutic approach in AF. Based on available data, current guidelines recommend alcohol abstinence or reduction to decrease AF symptoms, burden, and progression, and do not indicate the need for caffeine abstention to prevent AF episodes (unless it is a trigger for AF symptoms). Uncertainty persists regarding harms or benefits of other dietary factors including chocolate, fish, salt, polyunsaturated and monounsaturated fatty acids, vitamins, and micronutrients. This article provides a systematic review of the association between AF and both dietary patterns and components. Additionally, it discusses potentially related mechanisms and introduces different strategies to assess patients' nutrition patterns, including mobile health solutions and diet indices. Finally, it highlights the gaps in knowledge requiring future investigation.

Keywords Atrial fibrillation • Arrhythmia • Diet • Nutrition • Lifestyle

Introduction

Despite continuous improvement in pharmacologic and catheter-based therapy, atrial fibrillation (AF) remains one of the greatest challenges in cardiology. Control of cardiovascular risk factors and concomitant diseases is a key component of current AF management.[1–3] Modifiable AF risk factors, such as diabetes, obesity, or hypertension, are partially related to diet and lifestyle choices, therefore dietary interventions may have potential as a therapeutic approach for AF as assessed in detail in a recent review.[4] However, no systematic review has been performed to address comprehensively the relationship between dietary components and AF while taking into account the type of AF and adverse outcomes in AF.

The dietary advice provided for patients with AF in international guidelines is constrained by the lack of consistent and sufficient evidence. While there is a suggestion to avoid alcohol to alleviate AF burden, and no conclusive evidence linking limited caffeine intake to the prevention of AF episodes,[3] uncertainty surrounds the potential risks or advantages of other dietary factors, such as chocolate, fish, salt, polyunsaturated, and monounsaturated fatty acids, as well as vitamins and micronutrients.

Herein, we provide a systematic review of available studies on the association between AF and dietary factors (*Graphical Abstract*). We discuss potentially involved arrhythmogenic and anti-arrhythmogenic mechanisms of dietary components and supplements and introduce different strategies to assess patients' nutrition patterns. We highlight the limitations of available studies, gaps in knowledge, and areas requiring future investigation.

Methods

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines.[5] The electronic databases (PubMed, Web of Science, and EMBASE) were systematically searched for articles published between 1 January 2000 and 23 June 2024. Two reviewers systematically conducted the search, with a third reviewer consulted in case of uncertainty. Main search strategy is provided in the Supplementary data online, *Appendix*. Non-English, non-original articles (except meta-analyses/pooled analyses), conference abstracts, and original articles that did not directly address the association between diet and AF were excluded (see Supplementary data online, *Figure S1*). The summary of available meta-analyses/pooled analyses is presented in *Table 1*.[6–64] The main results of the randomized controlled trials (RCTs) and prospective cohort studies are presented in Supplementary data online, *Table S1*, whereas results of the retrospective cohort, cross-sectional, case-control, and Mendelian randomization studies are presented in Supplementary data online, *Table S2*. Experimental studies on potentially involved proarrhythmic mechanisms of dietary components are presented in Supplementary data online, *Table S3*. Analysis of how inclusion of individual studies changed the results of subsequent emerging meta-analyse is presented in Supplementary data online, *Tables S4–S13*.

In this review article, we focused primarily on data from RCTs for a specific dietary pattern and component, and in the absence of these data (*Table 2*), we presented evidence from prospective cohort studies.

Dietary patterns

RCTs: There are no RCTs investigating the effects of dietary patterns on new-onset, post-operative or recurrent AF.

Prospective cohort studies: In one prospective cohort study involving 24,713 participants, adherence to the EAT-Lancet diet, characterized by high consumption of healthy plant foods and moderate intake of fish while limiting meat, dairy, legumes, unsaturated fats, and sugars, was associated with a reduced risk of new-onset AF [hazard ratio (HR) 0.85, 95% confidence interval (CI) 0.73–0.98], particularly among those with a higher genetic predisposition to AF (HR 0.92, 95% CI 0.87–0.98).[65] Lower egg consumption was found to be a significant factor driving this association, although dietary data were collected only at baseline without considering changes over time. Nevertheless, three other prospective cohort studies, including the REasons for Geographic And Racial Differences in Stroke (REGARDS) study with 8,977 participants,[66] the UK Biobank study with 121,300 participants[67] and Women's Health Initiative with 123,330 women,[68] did not find associations between adherence to healthier dietary patterns such as Mediterranean diet (Med-diet), Dietary Approaches to Stop Hypertension (DASH), or plant-based diets and new-onset AF risk. In fact, only higher consumption of ultra-processed foods (5th vs. 0–2nd quintile) was linked to increased new-onset AF risk (HR 1.13, 95% CI 1.02–1.24).[67] Additionally, findings regarding specific nutrients were inconsistent across studies, with low carbohydrate intake associated with increased new-onset AF risk (HR 0.82, 95% CI 0.72–0.94 per 9.4% increment of energy from carbohydrates) in some analyses[69] and no relationship in others.[67]

Mediterranean diet. Med-diet is extensively studied regarding its impact on AF. It emphasizes high consumption of olive oil, unrefined cereals, fruits, and vegetables, moderate intake of fish, dairy, and red wine (≤1 drink/day), and low consumption of non-fish meat. Initial findings suggest potential benefits of the Med-diet in preventing AF development and

improving outcomes in AF patients. In the secondary analysis of the Prevención con Dieta Mediterránea (PREDIMED) trial, involving 6,705 participants, those on a Med-diet enriched with extra virgin olive oil had a 38% lower risk of new-onset AF compared with controls on Med-diet not enriched with nuts or extra virgin olive oil (HR 0.62, 95% CI 0.45–0.85).[70] Ongoing research, such as the PREvención Con DIeta Mediterránea de Arritmias Recurrentes (PREDIMAR) trial, aims to explore the effects of the Med-diet on recurrent AF after catheter ablation.[71]

There are no prospective cohort studies investigating the effects of dietary patterns on post-operative or recurrent AF.

Conclusions. No definitive evidence from prospective cohort studies currently backs any particular diet for lowering the risk of new-onset AF, including the Med-diet, which may only exhibit potential benefits when enhanced with extra virgin olive oil. Moreover, diets high in ultra-processed foods could potentially elevate the risk of new-onset AF.

Dietary components and supplements

Alcohol

RCTs: Several RCTs studied the effect of alcohol intake on recurrent AF in patients with paroxysmal/persistent AF.[72] In an RCT involving 140 regular drinkers (>10 drinks/week) with persistent AF undergoing electrical cardioversion, reducing alcohol consumption by nearly eightfold over 6 months led to decreased AF burden and longer freedom from recurrent AF (HR 0.55, 95% CI 0.36–0.84) compared with controls who continued their usual level of consumption.[73] Another RCT with 150 overweight individuals found that reducing alcohol intake to ≤30 g/week, as part of a comprehensive lifestyle modification program, resulted in fewer AF (2.5 vs. no change, $P = .01$) and cumulative AF duration (692-min decline vs. 419-min increase, $P = .002$).[74] The Individualized Studies of Triggers of Paroxysmal Atrial Fibrillation (I-STOP-AFib) RCT of 446 participants highlighted alcohol consumption as a significant trigger for near-term AF episodes among analyzed factors (e.g. caffeine, cold food, and drink, large meals, diet) [odds ratio (OR) 2.15, 95% CI 1.27–3.61].[75]

There are no RCTs investigating the effects of alcohol on new-onset or post-operative AF.

Prospective cohort studies: The largest study examining alcohol use and its association with new-onset AF among over 14 million participants found that those with alcohol dependence had over twice the risk of new-onset AF (HR 2.14, 95% CI 2.08–2.19).[76] Interestingly, this risk was particularly pronounced in individuals without other recognized AF risk factors. Other large studies, utilizing Korea's national health insurance system, revealed that each gram of alcohol consumed per week was associated with a 2% increase in new-onset AF risk (HR 1.02, 95% CI 1.01–1.03), with drinking frequency rather than the amount consumed linked to higher risk.[77] Surprisingly, certain patterns emerged, such as an inverse association between the amount of alcohol consumed per session and new-onset AF risk (HR 0.98, 95% CI 0.97–0.98 per gram). Additionally, analyses from the UK Biobank study of 403,281 individuals indicated a higher risk of new-onset AF with beer/cider consumption at any dose, while consumption of red wine, white wine, and spirits up to 10, 8, and 3 drinks/week, respectively, was not associated with increased risk.[78] Former drinkers were also found to have elevated new-onset AF risks compared with current drinkers. The ARIC study found that former drinkers had a 13% higher risk of new-onset AF for each decade of past alcohol consumption and a 4% higher risk for every additional drink per day. Conversely, each decade of abstinence was associated with a 20% lower risk of new-onset AF.[79] Meta-analyses of 13 prospective cohort studies, not including abovementioned studies,[78–82] revealed a linear relationship between alcohol consumption and new-onset AF risk in men, while in women, a J-shaped curve was observed, indicating a higher risk at alcohol consumption levels exceeding 1.4 drinks/day.[7] Another meta-analysis of 13 prospective cohort studies, differing by two studies compared with the previous meta-analysis, found that both low (<1 drink/day) and moderate (1–2 drinks/day) alcohol consumption were associated with an increased risk of new-onset AF in men (HR 1.14, 95% CI 1.01–1.28 and HR 1.09, 95% CI 1.07–1.11, respectively), but not in women.[9] Specifically, moderate beer consumption (~2 drinks/day) was associated with an elevated risk of new-onset AF (HR 1.11, 95% CI 1.02–1.21).

A prospective cohort study in 1,720 patients undergoing AF ablation demonstrated, that alcohol reduction of ≥1% (vs. <1%) from ~ 140 g/week during 12-month period was associated with lower rates of AF/atrial tachycardia recurrence (HR 0.63, 95% CI 0.52–0.77).[83] Finally, a recent meta-analysis of nine prospective cohort studies showed that moderate to high alcohol consumption was linked to a greater risk of recurrent AF after catheter ablation compared with minimal or no alcohol consumption (OR 1.45, 95% CI 1.06–1.99).[6]

There are no prospective cohort studies investigating the effects of alcohol on post-operative AF.

Conclusions. Based on RCTs (recurrent AF) and prospective cohort (new-onset AF) studies, alcohol consumption has been shown to have a dose-dependent relationship with AF. Most studies indicate that even low levels of intake (≥1 standard drink per week) may be associated with AF risk, especially in men and beer drinkers.

Mechanisms. In animal models, both acute and chronic alcohol exposure increases vulnerability to AF by altering conduction velocities, refractory periods,[84] and promoting atrial fibrosis.[85] Acute alcohol exposure affects cardiac ion channels, calcium-handling proteins, and mitochondrial function, leading to sarcoplasmic calcium leaks[86] and increased reactive oxygen species,[85] contributing to AF development. In humans, binge drinking activates the sympathetic nervous system followed by a rebound parasympathetic response,[87] reducing atrial refractory periods.[88] Long-term alcohol consumption leads to left atrial remodeling,[89] enlargement,[90] and mechanical dysfunction,[87] promoting AF episodes and progression.

Caffeine/coffee

RCTs: A small RCT with 110 participants found that caffeine intake 1.2 g/day before cardiac surgery had no effect on post-operative AF risk.[91]

There are no RCTs investigating the effects of caffeine/coffee on new-onset or recurrent AF.

Prospective cohort studies: Previous prospective cohort studies on caffeine's effects (found in coffee, tea, cola, chocolate snack, energy drink, or given as a supplement) on new-onset AF consistently show no association,[92–96] despite caffeine being the most commonly self-reported trigger for AF-related adverse events.[75] However, the relationship between coffee intake and AF risk is complex. In the largest prospective cohort study involving 449,563 participants, a U-shaped relationship was observed between coffee consumption and new-onset AF risk, with the lowest risk seen in those consuming 4–5 cups/day (HR 0.88, 95% CI 0.83–0.94).[97] Sub-analyses found similar relationships for

Table 1 Meta-analyses/pooled analyses regarding diet/dietary supplements and atrial fibrillation risk

Author	Design (# of studies)	Patients/AF cases	Exposure/intervention	Outcome	Association
Alcohol					
Grindal, 2023[6]	CP (9)	5,436/1,713	Alcohol intake (≥1 vs. <1 SD/week)	Recurrent AF	OR 1.45, 95% CI 1.06–1.99
Jiang, 2022[7]	CP (13)	10,151,366/214,365	Alcohol intake (per 1 SD/day), all	New-onset AF	RR 1.06, 95% CI: 1.03–1.08
			Alcohol intake (per 1 SD/day), men		Linear relationship
			Alcohol intake (per 1 SD/day), women		J-shaped relationship (peak at 1.4 drinks)
Giannopoulos, 2022[8]	CP (15) CC (1)	13,044,007/305,433	Alcohol intake (per 1 g/week)	New-onset AF	J-shaped association (peak at 14 SD)
Yang, 2022[9]	CP (13)	10,266,315/222,293	Alcohol intake (1–2 SD/day vs. no), all	New-onset AF	HR 1.14, 95% CI 1.07–1.21
			Alcohol intake (1–2 SD/day vs. no), men		HR 1.09, 95% CI 1.07–1.11
			Alcohol intake (1–2 SD/day vs. no), women		None
			Alcohol intake (<1 SD/day vs. no)		None
			Alcohol intake (<1 SD/day vs. no), men		HR 1.14, 95% CI 1.01–1.28
			Alcohol intake (<1 SD/day vs. no), women		None
Zhang, 2022[10]	CP (13)	645,626/23,079	Alcohol intake (>24 vs. <2 g/day)	New-onset AF	HR 1.30, 95% CI 1.20–1.41
			Alcohol intake (12–24 vs. <2 g/day)		HR 1.12, 95% CI 1.06–1.18
			Alcohol intake (<12 vs. <2 g/day)		None
Gallagher, 2017[11]	CP (9)	249,496/13,996	Alcohol intake (>2 SD/day vs. <1 SD/week)	New-onset AF	HR 1.34, 95% CI 1.20–1.49
			Alcohol intake (1–2 SD/day vs. <1 SD/week)		HR 1.11, 95% CI 1.05–1.18
			Alcohol intake (6–7 vs. <1 SD/week)		None
Larsson, 2014[12]	CP (7)	206,073/12,554	Alcohol intake (per 12 g/day)	New-onset AF	RR 1.08, 95% CI 1.06–1.10
Kodama, 2011[13]	CP (9) CC (5)	130,820/7,558	Alcohol intake (per 10 g/day)	New-onset AF	RR 1.08, 95% CI 1.05–1.10
Samokhvalov, 2010[14]	CP (4) CC (2)	63,124/4,767	Alcohol intake (per 12 g/day)	New-onset AF	RR 1.08, 95% CI 1.04–1.12
Caffeine/coffee					
Cao, 2022[15]	CP (10)	723,825/30,169	Daily coffee intake (per 1 cup/day)	New-onset AF	RR 0.98, 95% CI 0.97–1.00
Krittanawong, 2021[16]	CP (8) CR (1) CC (2)	361,143/17,704	Daily coffee intake (≥5 vs. 1–2 cups/day)	New-onset AF	None
Abdelfattah, 2018[17]		176,675/8,897		New-onset AF	OR 1.16; 95% CI 1.07–1.26

Continued

Table 1 *Continued*

Author	Design (# of studies)	Patients/AF cases	Exposure/intervention	Outcome	Association
	CP (6) CC (1)		Caffeine/coffee intake (<4 cups or <436 mg/day vs. ≥4 cups or ≥436 mg/day)		
Larsson, 2015[18]	CP (5)	248,910/10,406	Coffee intake (per 2 cups/day)	New-onset AF	None
Cheng, 2014[19]	CP (6)	228,465/4,261	Caffeine intake (per 300 mg/day)	New-onset AF	None
Caldeira, 2013[20]	CP (6) CC (1)	115,993/4,225	Habitual caffeine exposure (yes vs. no)	New-onset AF	None
Chocolate					
Larsson, 2017[21]	CP (5)	180,454/16,356	Chocolate intake (per 2 servings/week)	New-onset AF	None
Fish					
Li, 2017[22]	CP (6)	206,811/12,913	Fish intake (per 1 serving/week)	New-onset AF	None
Khawaja, 2012[23]	CP (6) CC (1)	56,931/1,672	Fish or n-3 PUFA intake (high vs. low quartile/day)	New-onset AF	None
Nuts					
Becerra-Tomas, 2019[24]	CP (2)	53,965/10,867	Nuts intake (high vs. low categories)	New-onset AF	RR 0.85, 95% CI 0.71–0.99
n-3 PUFA					
Garg, 2023[25]	CP (11)	41,335/6,173	Blood linoleic acid (per interquintile range)	New-onset AF	None
			Blood arachidonic acid (per interquintile range)		None
Qian, 2023[26]	CP (17)	54,799/77,720	Blood EPA (per interquintile range)	New-onset AF	None
			Blood docosapentaenoic acid (per interquintile range)		HR 0.89, 95% CI 0.83–0.95
			Blood DHA (per interquintile range)		HR 0.90, 95% CI 0.85–0.96
Gencer, 2021[27]	RCT (7)	81,210/2,905	n-3PUFA supplementation (vs. control)	New-onset AF	HR 1.25, 95% CI 1.07–1.46
Kow, 2021[28]	RCT (6)	75,120/2,053	n-3PUFA supplementation (vs. control)	New-onset + recurrent AF	RR 1.31, 95% CI 1.13–1.51
Jia, 2021[29]	RCT (8)	83,112/3,050	n-3PUFA supplementation (vs. control)	New-onset AF	RR 1.24, 95% CI 1.11–1.38
Lombardi, 2021[30]	RCT (5)	50,277/1,153	n-3PUFA supplementation (vs. control)	New-onset AF	RR 1.37, 95% CI 1.22–1.54
Lombardi, 2020[31]	RCT (5)	125,763/2,622	n-3PUFA supplementation (>1 g/day vs. control)	New-onset AF	IRR 1.35, 95% CI 1.10–1.66
			n-3PUFA supplementation (≤1 g/day vs. control)		None
Wang, 2018[32]	RCT (14)	3,570/1,209	n-3PUFA supplementation (vs. control)	Post-operative AF	RR 0.84, 95% CI 0.73–0.98
Jiang, 2017[33]	RCT (4)	1,268/625	n-3PUFA supplementation (vs. control)	Recurrent AF	None
Guo, 2014[34]	RCT (11)	3,137/956	n-3PUFA supplementation and/or VC, VE (vs. control)	Post-operative AF	OR 0.62, 95% CI 0.44–0.86

Continued

Table 1 *Continued*

Author	Design (# of studies)	Patients/AF cases	Exposure/intervention	Outcome	Association
	RCT (8)		n-3PUFA supplementation (vs. control)		None
	RCT (3)		n-3PUFA supplementation + VC, VE (vs. control)		OR 0.32, 95% CI 0.17–0.60
Zhang, 2014[35]	RCT (8)	2,687/848	n-3PUFA supplementation (vs. control)	Post-operative AF	None
Costanzo, 2013[36]	RCT (8)	2,687/848	n-3PUFA supplementation (vs. control)	Post-operative AF	OR 0.84, 95% CI 0.71–0.99
Mariani, 2013[37]	RCT (8)	4,677/1,753	n-3PUFA supplementation (vs. control)	Post-operative AF	None
	RCT (8)			Recurrent AF	None
Benedetto, 2013[38]	RCT (3)	431/181	n-3PUFA supplementation (vs. control)	Post-operative AF	None
He, 2013[39]	RCT (6)	2,184/979	n-3PUFA supplementation (vs. control)	Post-operative AF	OR 0.66, 95% CI 0.49–0.88
	RCT (5)			Recurrent AF	None
Xin, 2013[40]	RCT (8)	2,687/405	n-3PUFA supplementation (vs. control)	Post-operative AF	None
Cheng, 2013[41]	RCT (8)	1,990/894	n-3PUFA supplementation (vs. control)	Recurrent AF	None
Khawaja, 2012[23]	RCT (11) CP (7)	56,931/1,672	n-3PUFA supplementation (vs. control)	Post-operative + recurrent AF	None
Cao, 2012[42]	RCT (6)	759/455	n-3PUFA supplementation (vs. control)	Recurrent AF	None
	RCT (3)		– precardioversion (≥4 weeks)		OR 0.39, 95% CI 0.25–0.61
Armaganijan, 2011[43]	RCT (4)	538/205	n-3PUFA supplementation (vs. control)	Post-operative AF	None
Liu, 2010[44]	RCT (10)	1,955/903	n-3PUFA supplementation (vs. control)	Post-operative AF	None
				Recurrent AF	None
Salt					
Bhagavathula, 2020[45]	CP (3) CR (1) MR (1)	1,421,826/133,645	Salt intake (per 1 g/day)	New-onset AF	None
Vitamin D					
Ding, 2023[46]	CP (6)	28,694/2,917	Blood VD (per 10 ng/mL)	New-onset AF	HR 0.95, 95% CI 0.93–0.97
Hameed, 2023[47]	RCT (3)	448/91	VD supplementation, preoperatively (yes vs. no)	Post-operative AF	RR 0.60; 95% CI 0.40–0.90
Rahimi, 2021[48]	CR (1) CC (4)	669/219	Blood VD, preoperatively (per nmol/L)	Post-operative AF	mean difference −2.85, 95% CI −5.51;−0.20
Öztürk, 2020[49]	CR (1) CC (5)	769/269	Blood VD, preoperatively (ND)	Post–operative AF	mean difference −0.46, 95% CI −0.79;−0.12
Liu, 2019[50]	CC (4)	74,885/6,519	Blood VD, pre-/post-operatively (per 10 ng/mL)	Post-operative AF	RR 0.44, 95% CI 0.24–0.82
	CP (5) CR (1) CC (4)		Blood VD (per 10 ng/mL)	New-onset AF	RR 0.88, 95% CI 0.78–0.98

Continued

Table 1 *Continued*

Author	Design (# of studies)	Patients/AF cases	Exposure/intervention	Outcome	Association
Huang, 2017[51]	CR (1) CC (5)	1,252/528	Blood VD (ND)	Post-operative + new-onset AF	None
Zhang, 2016[52]	CP (5) CC (4)	27,307/3,571	Blood VD (per ng/mL)	New-onset AF	OR 0.92, 95% CI 0.87–0.97
Vitamin C					
Shi, 2018[53]	RCT (13)	1,956/615	VC supplementation alone + other therapy, PO	Post-operative AF	RR 0.68, 95% CI 0.54–0.87
	RCT (9)		– VC supplementation alone		RR 0.75, 95% CI 0.63–0.90
	RCT (4)		= VC supplementation + other therapy		RR 0.32, 95% CI 0.20–0.53
Hemila, 2017[54]	RCT (15)	2,050/629	VC supplementation, PO/IV	Post-operative + recurrent AF	RR 0.73, 95% CI 0.64–0.83
	RCT (6)		VC supplementation, IV, outside US	Post-operative AF	RR 0.64, 95% CI 0.53–0.78
	RCT (4)		VC supplementation, PO, outside US		RR 0.27, 95% CI 0.15–0.48
Hu, 2017[55]	RCT (8)	1,060/370	VC supplementation, PO/IV	Post-operative AF	OR 0.47, 95% CI 0.36–0.62
Baker, 2016[56]	RCT (11)	1,330/430	VC supplementation, PO/IV	Post-operative AF	OR 0.44, 95% CI 0.32–0.61
Polymeropoulos, 2016[57]	RCT (9)	1,037/372	VC supplementation, PO	Post-operative AF	OR 0.48, 95% CI 0.34–0.67
Magnesium					
Curran, 2023[58]	RCT (4)	4,654/696	$MgSO_4$ supplementation, IV	New-onset + post-operative AF	None
Chaudhary, 2019[59]	RCT (20)	2,430/630	$MgSO_4$ supplementation, IV	Post-operative AF	None
Duan, 2015[60]	RCT (7)	997/187	$MgSO_4$ supplementation, IV	Post-operative AF	RR 0.75; 95% CI 0.58–0.97
Cook, 2013[61]	RCT (21)	3,950/940	$MgSO_4$ supplementation, IV	Post-operative AF	None
Gu, 2012[62]	RCT (7)	1,028/192	$MgSO_4$ supplementation, IV	Post-operative AF	RR 0.64, 95% CI 0.50–0.83
Henyan, 2005[63]	RCT (7)	1,234/287	$MgSO_4$ supplementation, IV	Post-operative AF	OR 0.66, 95% CI 0.51–0.87
Shiga, 2004[64]	RCT (12)	1,649/388	$MgSO_4$ supplementation, IV	Post-operative AF	RR 0.71, 95% CI 0.55–0.93

AF, atrial fibrillation; CC, case-control; CI, confidence interval; CP, cohort prospective; CR, cohort retrospective; CS, cross-sectional; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; HR, hazard ratio; IV, intravenous; ND, no data; OR, odds ratio; PO, per os; PUFA, polyunsaturated fatty acid; RCT, randomized controlled trial; RR, risk ratio; SD, standard drink; US, United States; VC, vitamin C; VD, vitamin D; VE, vitamin E.

Table 2 Available evidence on specific dietary component and type of atrial fibrillation

Diet aspect	New-onset AF		Post-operative AF		Recurrent AF	
	Randomized controlled trial	Prospective cohort study	Randomized controlled trial	Prospective cohort study	Randomized controlled trial	Prospective cohort study
Dietary patterns	No	Yes	No	No	No	No
Med-diet	No	Yes	No	No	No	No
Alcohol	No	Yes	No	No	Yes	Yes
Caffeine/coffee	No	Yes	Yes	No	No	No
Chocolate	No	Yes	No	No	No	No
Fish	No	Yes	No	No	No	No
MUFA/SFA	No	Yes	No	No	No	No
PUFA	Yes	Yes	Yes	Yes	Yes	Yes
Salt	No	Yes	No	No	No	No
Vitamin D	Yes	Yes	Yes	Yes	No	Yes
Vitamin C	No	Yes	Yes	No	Yes	No
Magnesium	No	Yes	Yes	Yes	Yes	Yes

The table was created using data from Supplementary data online, *Table S1*.
AF, atrial fibrillation; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid.

both ground (HR 0.77, 95% CI 0.68–0.87) and instant coffee (HR 0.85, 95% CI 0.79–0.91), but no association with decaffeinated coffee. In line, relying on data from the UK Biobank, genetic variants related to caffeine metabolism did not influence the relationship between coffee intake and new-onset AF risk.[98] Conversely, an analysis of the Multi-Ethnic Study of Atherosclerosis (MESA) prospective cohort reported an overall trend of increasing coffee exposure correlating with increased new-onset AF risk in those consuming 2–3 cups/day (HR 1.57, 95% CI 1.14–2.20) and ≥6 cups/day (HR 2.15, 95% CI 1.29–3.61).[99] However, a meta-analysis of 10 prospective cohort studies, not including the aforementioned studies,[97–99] suggested a 2% reduced risk of AF with each additional cup of coffee consumed daily [risk ratio (RR) 0.98, 95% CI 0.97–1.00],[15] but this effect was not observed for caffeinated coffee alone. Another meta-analysis, including 8 prospective, 1 retrospective cohort, and 2 case-control studies, indicated that mixed consumption of caffeine or coffee does not increase new-onset AF risk,[16] and when studies with moderate bias were excluded, higher caffeine/coffee consumption (≥5 vs. 1–2 cups/day) was associated with a decreased risk of new-onset AF by 10% (RR 0.90, 95% CI 0.82–0.95).

There are no prospective cohort studies investigating the effects of caffeine/coffee on post-operative or recurrent AF.

Conclusions. Available prospective cohort studies showed no overall association between caffeine/coffee intake and new-onset AF risk. However, pooled results from high-quality studies with adjustments for possible confounders showed a reduction in new-onset AF risk with habitual caffeine intake. A single RCT showed no overall association between caffeine consumption and post-operative AF risk.

Mechanisms. Caffeine affects the heart by antagonizing various adenosine receptors. Research on human atrial myocytes from patients with AF suggests that adenosine-mediated pathways could increase spontaneous calcium release from the sarcoplasmic reticulum, potentially initiating AF.[100] However, long-term use may lead to habituation. For instance, in the Coffee and Real-time Atrial and Ventricular Ectopy (CRAVE) trial, consuming caffeinated coffee for 14 days did not significantly increase daily premature atrial contractions,[101] a potent predictor of AF.[102] Additionally, habitual caffeine intake may have cardioprotective effects by mitigating the effects of endogenous adenosine. In dogs, escalating doses of caffeine reduced AF propensity through autonomic mechanisms.[103] Moreover, caffeine's pro-catecholamine effects may counteract vagal AF,[104] while the anti-inflammatory properties of common caffeinated beverages like tea and coffee could reduce AF risk.[105]

Chocolate

RCTs: There are no RCTs investigating the effects of chocolate on new-onset, post-operative, or recurrent AF.

Prospective cohort studies: In the largest prospective cohort study involving 55,502 participants, higher chocolate intake (between 6 servings/week and 1 serving/month vs. <1 serving/month) was associated with a 10%–20% lower rate of new-onset AF, with no effect observed for daily chocolate consumption compared with <1 serving/month.[106] Noteworthy, in a sub-analysis stratified by sex, the risk of new-onset AF was lower among women than men at each level of chocolate intake; in women, the significant association was only seen for 1 serving/week (HR 0.79, 95% CI 0.66–0.95). Conversely, results from two other large prospective cohort studies, the Women's Health Study of 33,638 women[94], and Physicians' Health Study of 18,819 men,[107] did not find any association between chocolate intake and new-onset AF. Accordingly, a meta-analysis of five studies (including the three aforementioned studies[94,106,107] showed no overall association between chocolate consumption and new-onset AF risk).[21]

There are no prospective cohort studies investigating the effects of chocolate on post-operative or recurrent AF.

Conclusions. Current prospective cohort studies do not indicate an association between chocolate consumption and an increased risk of new-onset AF.

Mechanisms. Chocolate contains flavanols, which have antioxidant, anti-inflammatory, and antiplatelet properties, as well as positive effects on angiotensin-converting enzyme activity and glucose transport.[108] These mechanisms have been demonstrated to prevent the development of atrial arrhythmogenic substrate.[105] However, chocolate also contains methylxanthines like caffeine and theobromine, which could potentially have a neutral or pro-arrhythmogenic effect on AF.[100,103] It's worth noting that chocolate is often consumed in high-calorie processed forms rich in sugar and fat, and modern manufacturing processes may lead to significant losses (>80%) of the beneficial flavanols found in cocoa beans.[109]

Fish

RCTs: There are no RCTs investigating the effects of fish on new-onset, post-operative, or recurrent AF.

Prospective cohort studies: Some prospective cohort studies suggest a beneficial association between total fish intake and new-onset AF,[110] while others show no association[96,111–113] or even a U-shaped relationship with the lowest AF risk associated with a fish intake of 40 g/day.[114] One study found a 21% reduction in new-onset AF (RR 0.79, 95% CI 0.65–0.95) among individuals consuming lean fish ≥3 times/week compared with none.[111] Conversely, individuals consuming dark fish >4/week (vs. <1/week) had a 6.53-fold higher risk of new-onset AF (HR 6.53, 95% CI 2.65–16.06).[96] A meta-analysis of six studies (without aforementioned one[114]), found no association between higher fish consumption and new-onset AF risk, even in additional subgroup and dose-response analyses.[22]

There are no prospective cohort studies investigating the effects of fish on post-operative or recurrent AF.

Conclusions. The inconsistency of data from available prospective cohort studies make it challenging to determine whether consuming fish influences new-onset AF risk. RCTs are not available.

Mechanisms. The properties of fish consumption are seen in the action of polyunsaturated and monounsaturated fatty acids discussed in *Monounsaturated fatty acids and saturated fatty acids* and *Salt*, respectively.

Polyunsaturated fatty acids

Polyunsaturated fatty acids (PUFAs), encompassing both n-3 and n-6 fatty acids, are often studied for their health benefits, primarily focusing on docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) whose main source is fish. On the other hand, the most abundant plant-based PUFA is α-linolenic acid.[115]

RCTs: The Vitamin D and Omega-3 (VITAL) Rhythm study, involving 25,871 participants, found no significant change in AF risk with n-3PUFA supplementation (840 mg/day; EPA:DHA 1.2:1) over median 5.3 years.[116] Conversely, in the Long-Term Outcomes Study to Assess Statin Residual Risk with Epanova in High Cardiovascular Risk Patients with Hypertriglyceridemia (STRENGTH) trial, which included 13,078 participants, treatment with four-fold higher dose of n-3PUFA with a larger EPA content (EPA:DHA 2.8:1) and for shorter time of median 38 months, compared with the VITAL trial, was associated with increased risk of new-onset AF, and therefore terminated early.[117] In line, in the recent Randomized Trial for Evaluation in Secondary Prevention Efficacy of Combination Therapy–Statin and Eicosapentaenoic Acid (RESPECT-EPA) trial, EPA supplementation (1.8 g/day) for median 5 years resulted in a significant increase in new-onset AF (3.1% vs. 1.6%, $P = .017$) compared with the control group.[118] According to the available meta-analyses, one of seven RCTs (including two aforementioned RCTs)[116,117] suggested an increased AF risk with n-3PUFA supplementation (HR 1.25, 95% CI 1.07–1.46), particularly at high (>1 g/day) doses.[27]

In the Omega-3 Fatty Acids for Prevention of Post-operative Atrial Fibrillation (OPERA) RCT involving 1,516 participants undergoing cardiac surgery, preoperative (8–10 g over 2–5 days) and post-operative (2 g/day over 10 days) n-3PUFA treatment did not significantly affect the incidence of post-operative AF.[119] However, a meta-analysis of 14 RCTs, including the OPERA trial, indicated a 16% reduction (RR 0.84, 95% CI 0.73–0.98) in post-operative AF incidence with n-3PUFA supplementation.[32] Notably, this effect was observed when the EPA:DHA ratio was <1 and when placebo consisted of usual care, but not when placebo was non-fish oils. Previous four meta-analyses examining the same eight RCTs showed conflicting results, with some indicating no effect on post-operative AF[34,35,40] and others suggesting a reduced risk[36] with n-3PUFA supplementation. The difference between these meta-analyses could be explained by differently assigned weights for articles as well as differently calculated risk metrics (OR[36] vs. RR[35]). Additionally, one meta-analysis found that combining n-3PUFA supplementation with vitamins C and E resulted in a 68% reduction in post-operative AF incidence compared with control (OR 0.32; 95% CI 0.17–0.60).[34]

A large RCT involving 663 participants with paroxysmal or persistent AF found that n-3PUFA supplementation did not reduce the risk of recurrent AF compared with placebo.[120] Similarly, a meta-analysis of eight RCTs, including the aforementioned study,[120] found no benefit of n-3PUFA treatment in preventing recurrent AF after cardioversion.[41] However, in a sensitivity analysis, continuous administration of n-3PUFA at least 4 weeks before cardioversion was associated with a decreased recurrent AF rate (OR 0.39, 95% CI 0.25–0.61), while initiating treatment <4 weeks before or after cardioversion did not show this benefit.

Prospective cohort studies: Prospective studies assessing α-linolenic acid intake using dietary[121] or plasma[122] records for the new-onset AF reported no association. A meta-analysis of two prospective cohort studies analyzed the association between nuts, which are natural sources of α-linolenic acid. Compared with the lowest category of nut consumption, the highest one was associated with a reduced risk of AF (RR 0.85, 95% CI 0.73–0.99)[24]. Pooled analyses of 17 prospective cohort studies indicated a protective effect of certain n-3PUFAs (HR 0.90, 95% CI 0.85–0.96 for DHA and HR 0.89, 95% CI 0.82–0.95 for docosapentaenoic acid) against new-onset AF, but only when transported by circulating phospholipids and as such incorporated into all cell membranes, not as free fatty acid fraction.[26]

The prospective cohort studies investigating the effects of PUFA on new-onset, post-operative or recurrent AF are presented in supplemental material.

Conclusions. RCTs have shown that long-term, high-dose n-3PUFA supplementation increases the risk of new-onset AF, and that short-term n-3PUFA supplementation decreases the risk of post-operative AF. Of note, in comparison to studies with new-onset AF as the endpoint, the n-3PUFA therapy exposure in studies with post-operative AF as the endpoint was shorter, was combined with additional active molecules (like vitamins) and included a usual care control group, which may have contributed to the contrasting results. There is limited RCT data to definitively establish whether n-3PUFA can prevent recurrent AF.

Mechanisms. Experimental studies suggest that n-3PUFAs, particularly DHA, may reduce vulnerability to AF[123,124] by modulating the autonomic nervous system, reducing inflammation, oxidative stress, fibrosis,[123] and altering connexin expression levels.[125] While some

research indicates that n-3PUFAs may not directly affect atrial electrical remodeling induced by atrial tachycardia,[126] others suggest they may inhibit specific ion currents (I_{to}, I_{Kur}, I_{Na}) in human atrial myocytes,[124] potentially influencing AF development.

Monounsaturated fatty acids and saturated fatty acids

RCTs: There are no RCTs investigating the effects of fatty acids on new-onset, post-operative, or recurrent AF.

Prospective cohort studies: A prospective cohort study of 33,665 women found no significant link between dietary fat intake and the risk of developing new-onset AF.[127] However, when considering the type of AF, saturated and monounsaturated fatty acids showed associations with persistent AF (RR 1.47, 95% CI 1.04–2.09 and RR 0.67, 95% CI 0.46–0.98 per 5% increment of energy from saturated and monounsaturated fatty acids, respectively) but not paroxysmal AF.[127] Conversely, prospective cohort study involving 1,872 participants found that while most monounsaturated fatty acids did not correlate with new-onset AF risk, nervonic acid was an exception, showing a higher risk association (HR 1.18, 95% CI 1.08–1.29).[128]

There are no prospective cohort studies investigating the effects of fatty acids on post-operative or recurrent AF.

Conclusions. Single prospective cohort studies make it impossible to draw a clear conclusion about the relationship between saturated and monounsaturated fatty acids and new-onset AF.

Mechanisms. The role of fatty acids in AF pathophysiology is discussed in our previous review.[129] In brief, fatty acids could potentially trigger AF via atrial inflammation and oxidative stress enhancement.

Salt

RCTs: There are no RCTs investigating the effects of vitamin D on new-onset, post-operative or recurrent AF.

Prospective cohort studies: The largest study on salt intake based on urinary sodium excretion and new-onset AF included 473,080 participants from the UK Biobank.[130] It found a U-shaped association (first quintile: HR 1.20, 95% CI 1.08–1.32 and fifth quintile: HR 1.15, 95% CI 1.03–1.27) among men but a non-significant J-shaped association among women. However, a meta-analysis of three prospective (including the above study,[130] one retrospective cohort study and one Mendelian randomization study), found no association between new-onset AF and salt intake.[45]

There are no prospective cohort studies investigating the effects of salt on post-operative or recurrent AF

Conclusions. There is no definitive data from prospective cohort studies linking salt intake to a higher risk of new-onset AF.

Mechanisms. In animal studies, a high-salt diet leads to atrial fibrosis,[131–133] increased sympathetic nerve activity,[131] and activation of certain potassium currents.[133] It also affects AF duration and atrial refractory periods by altering calcium-handling and reducing gap junction expression.[132] Inhibition of a specific sodium-proton exchanger subtype reduces AF susceptibility, preserves atrial function, and lessens fibrosis and slow conduction areas.[134]

Vitamin D

RCTs: The VITAL Rhythm study, the largest RCT on vitamin D's impact on new-onset AF risk, found no significant difference in new-onset AF risk with vitamin D supplementation compared with placebo.[116] Similarly, in a Women's Health Initiative trial involving 16,801 postmenopausal women, supplementation with calcium and vitamin D showed no difference in new-onset AF risk compared with placebo.[135] Regarding post-operative AF, the impact of serum vitamin D is uncertain, with some data suggesting both protective and neutral effects. A recent meta-analysis of three RCTs showed that vitamin D supplementation before cardiac surgery significantly reduced post-operative AF incidence (RR 0.60; 95% CI 0.40–0.90).[47] Furthermore, subgroup analysis of aforementioned meta-analysis,[50] revealed that higher serum vitamin D levels were associated with decreased post-operative AF risk (RR 0.44, 95% CI 0.24–0.82 per 10 ng/mL increase).

There are no RCTs investigating the effects of vitamin D on recurrent AF.

Prospective cohort studies: In a prospective cohort study of 200 patients with persistent AF, lower serum vitamin D levels were linked to failure of electrical cardioversion to restore sinus rhythm (19 ± 4.7 vs. 29 ± 4.9 ng/mL in cardioversion success, $P < .01$).[136] Similarly, a subanalysis ($n = 900$ participants) of the VITAL Rhythm study showed a higher risk of persistent AF in participants randomized to receive vitamin D.[137]

The prospective cohort studies investigating the effects of vitamin D on new-onset or post-operative AF are presented in supplemental material (see Supplementary data online, *Table S1*).

Conclusions. While RCTs failed to show a protective effect of high vitamin D levels in patients with new-onset AF, RCTs showed, that n-3PUFA reduces post-operative AF. There is not enough data from RCTs and prospective cohort studies on the connection between recurrent AF and vitamin D to make any definitive conclusions.

Mechanisms. Several possible mechanisms link vitamin D deficiency with AF, including activation of the renin-angiotensin system, structural remodeling of the left atrium, atrial electromechanical delay, and shortened duration of atrial action potentials.[138]

Vitamin C

RCTs: Most research on the impact of vitamin C on AF risk has focused on post-operative populations and small (<100 participants) RCTs. A meta-analysis of 13 RCTs showed that vitamin C combined with other therapies (beta blockers or statin) had a stronger preventive effect (RR 0.32, 95% CI 0.20–0.53) than vitamin C alone (RR 0.75, 95% CI 0.63–0.90).[53] Oral vitamin C was more effective than intravenous administration in preventing post-operative AF based on studies conducted outside the United States. A meta-analysis of 15 RCTs found that oral vitamin C had a stronger preventive effect against post-operative AF (RR 0.27, 95% CI 0.15–0.48) compared with intravenous vitamin C (RR 0.64, 95% CI 0.53–0.78), with studies conducted outside the United States.[54]

In a 120-participant RCT, intravenous vitamin C before cardioversion and oral supplementation afterward showed no significant difference in recurrent AF risk compared with placebo.[139] However, a small RCT of 44 participants found that oral vitamin C reduced recurrent AF frequency compared with no supplementation (RR 0.13, 95% CI 0.02–0.92).[140] Another small RCT on intravenous vitamin C administration before catheter ablation ($n = 20$ participants) did not show a significant reduction in recurrent AF compared with placebo ($n = 10$ participants).[141]

There are no RCTs investigating the effects of vitamin C on new-onset AF.

Prospective cohort studies: Single data from European Prospective Investigation into Cancer (EPIC) Norfolk prospective cohort study, involving 8,760 men and 10,530 women, found an inverse relationship between plasma vitamin C levels and new-onset AF risk in women (HR 0.87, 95% CI 0.78–0.97 per 20 μmol/L increase) but not in men.[142]

There are no prospective cohort studies investigating the effects of vitamin C on post-operative or recurrent AF.

Conclusions. Findings from several small RCTs suggest that perioperative oral vitamin C supplementation, combined with other therapies, may lower the incidence of post-operative AF. However, insufficient data from prospective cohort studies and RCTs exist to definitively confirm whether vitamin C can reduce the risk of new-onset or recurrent AF.

Mechanisms. While the exact mechanism of how vitamin C supplementation benefits is not fully understood, it's thought that vitamin C may reduce oxidative stress and inflammation, potentially offering protection against AF,[143] particularly in contexts like cardiac surgery.

Magnesium

RCTs: Individual RCTs suggest oral magnesium supplementation can be beneficial against post-operative AF.[144,145] A trial with 200 participants showed that those given oral magnesium before and after cardiac surgery had a lower risk of post-operative AF compared with a placebo (RR 0.45, 95% CI 0.23–0.91).[145] However, a meta-analysis of 20 RCTs found no association between intravenous magnesium supplementation and post-operative AF risk.[59]

In RCT of 170 patients with persistent AF, oral magnesium therapy, whether used alone or in combination with sotalol, did not affect the recurrence rate of AF following elective cardioversion.[146]

There are no RCTs investigating the effects of magnesium on new-onset AF.

Prospective cohort studies: In the Atherosclerosis Risk in Communities (ARIC) prospective cohort study involving 14,290 participants, low serum magnesium levels were linked to AF development (HR 1.34, 95% CI 1.16–1.54), but oral magnesium intake was not.[147] A single study suggests that oral magnesium supplementation may either reduce (with low doses) or increase (with high doses) new-onset AF risk.[148] A Danish nationwide register-based study of over 4.2 million individuals found a slight beneficial effect on new-onset AF associated with increased magnesium levels in drinking water up to 10 mg/L [incidence RR (IRR) 0.98, 95% CI 0.97–1.00], though the correlation was generally favorable (IRR 1.04, 95% CI 1.04–1.05 per 10 mg/L increase).[110]

The prospective cohort studies investigating the effects of magnesium on post-operative AF are presented in supplemental material.

Conclusions. There is not enough evidence from prospective cohort studies (new-onset) and RCTs (post-operative AF, recurrent AF) to definitively confirm whether magnesium supplementation can reduce the AF risk.

Mechanisms. Intravenous magnesium directly impacts myocardial potassium channels, affects calcium and sodium channels indirectly, prolongs the PR interval, and extends the refractory period of atrioventricular node conduction.[149]

Nutrition control

Various methods, like daily food logs, 24 h dietary recalls, food frequency questionnaires, and diet quality indexes, can assess diet-AF relationships. These approaches offer insights, but intermittent diet assessment simplifies the complex dietary intake over time.[150] The limitations include potential recall bias, also influenced by current diet and incomplete questionnaire responses. Additional indicators are needed to validate data accuracy. For instance, the PREDIMED trial used objective biomarkers (urinary hydroxytyrosol and plasma α-linolenic acid) to measure adherence to specific dietary components (extra-virgin olive oil and nut consumption, respectively).[70]

The diet has a substantial effect on the gut microbiome, which consists of all microorganisms and their genetic material in the gastrointestinal tract.[151] This, in turn, influences the metabolome, the total number of metabolites in an organism, through its role in fermenting food and host-derived substrates. More research on the metabolomic signatures related to diet and AF has been performed previously.[152,153] Metabolomic studies help identify specific biomarkers in the blood that correlate with dietary patterns and can provide insights into how diet influences the risk and progression of AF.

Many diet-tracking apps have emerged to help individuals understand dietary patterns and lose weight. A recent review[154] evaluated 7 top diet-tracking apps over a 2-week period, recording real-time food consumption for three consecutive days (2 week days and 1 weekend day) for each app. While app features varied, they all emphasized self-efficacy by assisting users in tracking their diet and progress toward goals. Future studies should assess if certain apps enhance users' self-motivation to meet dietary goals. Combining heart rhythm/rate control with diet-tracking could offer valuable insights into physiological responses to nutrients. Notably, these apps lack tracking potential upstream (e.g. hunger, hormone levels, personal preference) and downstream effects of diet (e.g. satiety, stress, and taste), which could be incorporated in future app designs.

Limitations and gaps in knowledge

There are many limitations, which are related to the design of most available studies focusing on the relation between diet and AF. Most studies have assessed the association between AF and diet by patient self-reported amount and type of food consumption, rather than by objective blood or urine samples. Quantifying dietary intake can be challenging due to varying definitions of food/alcohol serving sizes and long-term adherence to diets. Methodological differences, such as diverse scoring systems for evaluating dietary adherence, can also hinder result interpretation. Another shortcoming is that information on type of specific drink/food (e.g. milk vs. dark chocolate) is not often taken into account. Also, most studies are conducted within European ancestry, rather than other ethnicities. Additionally, the presence of AF is often established from a new AF diagnosis derived from patient records based on electrocardiogram documentation of often symptomatic AF episodes, rather than dedicated long-term heart rhythm monitoring. The possibility of reverse causality in the association between specific drink/food consumption and AF cannot be excluded. Under this assumption, those participants with higher rates of risk factors or illnesses try to avoid, for example, coffee consumption due to the previously noted belief of a deleterious effect of coffee on cardiovascular health. Studies also differ in terms of adjustment for confounding factors (sun exposure, physical activity, racial/ ethnic differences, and comorbidity including obesity) which might add another level of heterogeneity. The role of less validated supplements like green tea, iron, zinc, and copper, as well as different sensory agents, including flavour enhancers and sweeteners on AF remains unclear. A limitation with meta-analyses owes to heterogeneity of included studies.

RCTs and cohort studies are used in nutritional epidemiology, and both methods have their inherent advantages and limitations. RCTs balance confounding factors and minimize bias through randomization and blinding but are often expensive, complex, and may not represent the general population. Although cohort studies may be more generalizable with diverse populations and may provide insights into long-term dietary effects,

they may be prone to confounding, recall bias, and dropouts over time. This, for example, might partially explain the discordance between observational and RCT evidence for n-3PUFA supplementation in AF. Future studies should focus on objective rhythm monitoring and diet consumption monitoring. In future RCTs, health-neutral placebos rather than vegetable oil administration (such as olive oil with its cardio protective effects) as placebo should be considered. Interventions supporting the weight loss process like ketogenic diet, intermittent fasting or pharmaceutics use (for example glucagon-like peptide-1 receptor agonists) have taken off in popularity. Some of them have been shown to improve cardiovascular health via reducing blood pressure, glucose, and weight.[155] Further experimental and clinical studies are needed to assess whether such interventions are beneficial in the population of patients with AF.

Conclusions

This systematic review summarizes existing evidence regarding the association between AF and the wide variety of dietary patterns and components. In summary, alcohol raises AF risk, while caffeine/coffee, chocolate, fish consumption, and magnesium show no clear association with AF risk. Long-term, high-dose n-3PUFA supplementation, based on RCTs, increases new-onset AF risk. N-3PUFA, vitamin D, and vitamin C may lower post-operative AF risk. Data on the influence of dietary factors on AF progression (burden/recurrences) is sparse. Currently, no specific diet has definitive evidence for reducing new-onset AF risk, including the Med-diet, which may only show potential benefits when supplemented with extra virgin olive oil. Additionally, diets high in ultra-processed foods may increase new-onset AF risk. High-quality data from RCTs is rarely available, and the results of most meta-analyses of partially low-quality observational studies are often inconclusive. Further evidence is required to allow clear recommendations concerning diet in patients with AF in future guidelines.

Supplementary data

Supplementary data are available at *European Heart Journal* online.

Declarations

Disclosure of Interest

All authors declare no disclosure of interest for this contribution.

Data Availability

The data underlying this article are available in the article and in its supplementary material.

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