



Nutritional Approaches to Reducing the Risk of Stroke

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Abstract

Stroke remains a leading cause of mortality and long-term disability worldwide, with diet representing a major modifiable risk factor. Although substantial evidence links nutrition to stroke prevention, effective translation into clinical practice requires integration of dietary patterns, specific nutrients, biological mechanisms, and emerging personalized approaches. This review aimed to synthesize current evidence on nutritional strategies for stroke prevention, encompassing macronutrients, micronutrients, dietary patterns, nutrigenomics, chrononutrition, and gut microbiome-related mechanisms, and to translate these findings into practical clinical implications. A systematic narrative review was conducted in accordance with PRISMA 2020 principles. PubMed/MEDLINE, Scopus, Web of Science, and ScienceDirect were searched for peer-reviewed English-language studies published between January 2000 and April 2024. Eligible studies included randomized controlled trials, observational studies, systematic reviews, and meta-analyses evaluating dietary exposures and stroke-related outcomes. Due to heterogeneity in study design and outcomes, 48 eligible studies were synthesized narratively. Evidence consistently supports the protective role of comprehensive dietary patterns—particularly the Mediterranean, DASH, and MIND diets—in reducing stroke risk. These patterns exert benefits through convergent mechanisms, including blood pressure reduction, lipid profile improvement, attenuation of inflammation and oxidative stress, and preservation of endothelial function. Macronutrient quality, especially fat type and dietary fiber intake, and key micronutrients such as potassium, magnesium, folate, and sodium further modulate stroke risk. Emerging evidence

indicates that genetic variation, meal timing, and gut microbiome composition may influence individual responses to dietary interventions. In conclusion, nutrition plays a central role in both primary and secondary stroke prevention. Integrating evidence-based dietary patterns with attention to nutrient quality, chrononutrition, and individual risk profiles may enhance clinical effectiveness. Future randomized trials are needed to strengthen causal inference and support implementation of personalized nutritional strategies in clinical and public health practice.

Keywords: Stroke prevention, Dietary patterns, Macronutrients and micronutrients, Nutrigenomics, Chrononutrition

Introduction

Globally, stroke remains one of the leading causes of death and long-term disability, affecting an estimated 13.7 million people annually and contributing to over 100 million cases of stroke-related morbidity as of 2023⁽¹⁾. The pathogenesis of stroke is complex, involving both non-modifiable factors such as age, sex, and genetics, and modifiable factors including diet, hypertension, diabetes, and dyslipidemia. Among these, dietary habits represent a particularly potent modifiable determinant—capable of influencing nearly 80% of stroke cases through effects on inflammation, oxidative stress, lipid metabolism, and endothelial function^(2,3).

Over recent decades, nutritional research has progressed from focusing on single nutrient effects to recognizing the broader importance of dietary patterns in reducing stroke risk. Comprehensive diets such as the Mediterranean, DASH, and plant-based diets have demonstrated consistent benefits in reducing cardiovascular and cerebrovascular morbidity, largely through mechanisms involving improved blood pressure control, enhanced lipid profiles, and reduction of oxidative stress⁴. These dietary patterns are naturally rich in antioxidants, polyphenols, and unsaturated fats, which collectively contribute to anti-inflammatory and neuroprotective effects that help preserve vascular integrity.

In parallel, advances in nutrigenomics have deepened understanding of how genetic polymorphisms influence individual responses to diet, paving the way for personalized nutrition strategies that optimize stroke prevention outcomes⁽⁵⁾. Meanwhile, chrononutrition, an emerging field in nutritional science, explores how meal timing and circadian rhythm alignment modulate metabolic and vascular health, offering novel insights into temporal optimization of nutrient intake to lower stroke risk⁽⁶⁾.

Furthermore, growing evidence highlights the gut-brain axis as a critical intermediary between diet and cerebrovascular function. Diet-driven modulation of the gut microbiome

influences systemic inflammation, lipid metabolism, and vascular health—key processes linked to stroke pathophysiology. Recent innovations in artificial intelligence (AI) and machine learning have begun to transform nutritional epidemiology by enabling predictive modeling of diet–gene–microbiome interactions, facilitating more individualized dietary recommendations for stroke prevention^(7,8).

Accordingly, this review synthesizes the latest evidence on nutritional approaches to reducing stroke risk, emphasizing the transition from isolated nutrient analysis to an integrated understanding of dietary patterns, gene–diet interactions, and chrononutrition. The overarching aim is to delineate how these interconnected domains contribute to stroke prevention and to identify emerging opportunities for precision nutrition and public health implementation.

Methods

Study Design and Reporting Framework

This review was conducted as a systematic narrative review and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. The review aimed to synthesize evidence on nutritional strategies for stroke prevention, including dietary patterns, specific nutrients, nutrigenomics, chrononutrition, and gut microbiome–related mechanisms.

Search Strategy

A comprehensive literature search was performed across four electronic databases: PubMed/MEDLINE, Scopus, Web of Science, and ScienceDirect. The search covered publications from January 2000 to April 2024. Search terms were developed using combinations of Medical Subject Headings (MeSH) and free-text keywords related to stroke and nutrition, including stroke, ischemic stroke, cerebrovascular disease, diet, nutrition, dietary patterns, Mediterranean diet, DASH diet, MIND diet, nutrigenomics, chrononutrition, and meal timing. Boolean operators (“AND”, “OR”) were applied to refine the search. In addition, reference lists of relevant reviews and guidelines were manually screened to identify additional eligible studies.

Eligibility Criteria

Studies were eligible for inclusion if they met the following criteria: (1) randomized controlled trials, cohort studies, case–control studies, cross-sectional studies, systematic reviews, or meta-analyses; (2) conducted in human participants aged ≥ 18 years; (3) evaluated dietary patterns, specific nutrients (macro-, micro-, or phytonutrients), nutrigenomic factors, or

chrononutrition-related exposures; (4) reported stroke incidence, recurrence, mortality, or established stroke risk factors (e.g., blood pressure, lipid profiles, inflammation, endothelial function); and (5) published as peer-reviewed articles in English.

Studies were excluded if they were animal or in vitro studies, editorials, commentaries, conference abstracts, non-English publications, did not assess nutritional exposures or stroke-related outcomes, or lacked sufficient methodological detail to support data synthesis.

Study Selection

A total of 1,278 records were identified, including 1,248 records from database searches and 30 records from reference screening. After removal of duplicates, 582 unique records remained and were screened by title and abstract. During this stage, 409 records were excluded due to irrelevance. The remaining 173 articles underwent full-text assessment. Following full-text review, 125 articles were excluded for predefined reasons, including lack of nutrition relevance, absence of stroke-related outcomes, animal or in vitro design, non-English language, editorial format, or methodological limitations. Ultimately, 48 studies met all inclusion criteria and were included in the qualitative synthesis. The study selection process is illustrated in the PRISMA flow diagram (Figure 1).

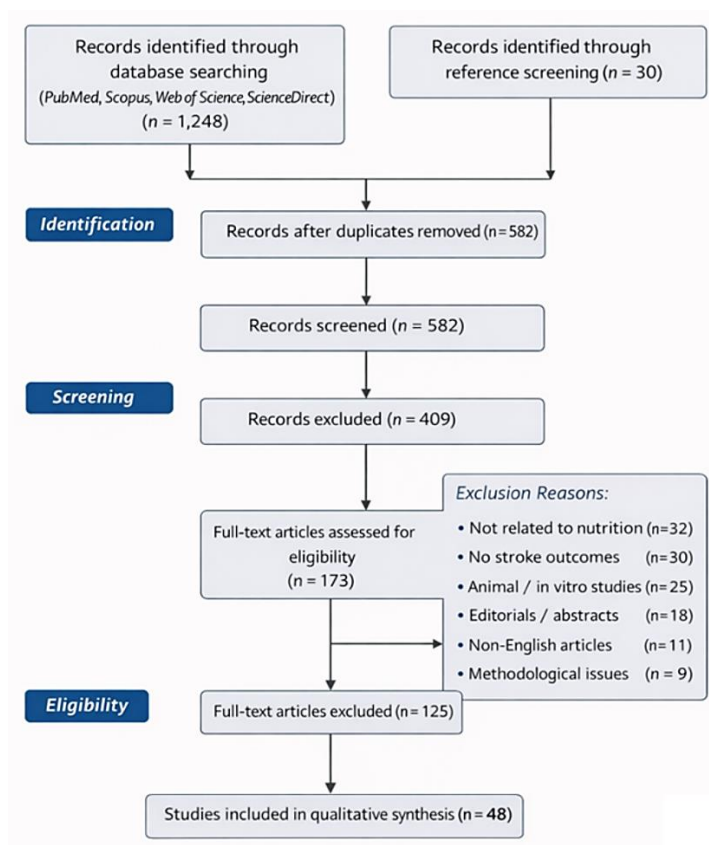


Figure 1 PRISMA 2020 flow diagram of study selection

Data Extraction

Data extraction was conducted independently by two reviewers using a standardized extraction form. Extracted information included author and year of publication, study design and geographic setting, population characteristics and sample size, dietary exposure or intervention, stroke-related outcomes or intermediate biomarkers, key findings and effect estimates, and reported confounding variables with corresponding adjustment methods. Any discrepancies were resolved through consensus discussion.

Quality Assessment and Risk of Bias

Methodological quality was assessed according to study design. Observational studies were evaluated using the Newcastle–Ottawa Scale (NOS), while randomized controlled trials were assessed using the Cochrane Risk of Bias Tool version 2.0 (RoB 2.0). Quality assessment was performed independently by two reviewers, with disagreements resolved by consensus. Studies were categorized as having low, moderate, or high risk of bias, and quality considerations were incorporated into the interpretation of findings.

Data Synthesis

Due to heterogeneity in study designs, dietary exposures, and outcome measures, a quantitative meta-analysis was not performed. Instead, findings were synthesized narratively and organized into thematic domains, including macronutrients, micronutrients, dietary patterns, nutrigenomics, chrononutrition, and gut microbiome-related mechanisms. Where available, dose-response relationships, effect sizes, and biological mechanisms—such as inflammation, oxidative stress, lipid metabolism, endothelial function, and neurovascular protection—were summarized.

Ethics and Limitations

All data were from published sources; no ethical approval was required. Limitations included variation in dietary assessment tools, outcome definitions, and regional diet patterns.

Stroke Overview: Pathogenesis and Clinical Features

Stroke, medically termed cerebrovascular accident (CVA), is a life-threatening event resulting from an interruption or marked reduction of cerebral blood flow, leading to deprivation of oxygen and nutrients and subsequent neuronal death. Strokes are classified into two main types: ischemic and hemorrhagic. Ischemic stroke, accounting for approximately 85% of cases, results from arterial obstruction by a thrombus or embolus, whereas hemorrhagic stroke arises from the rupture of weakened cerebral vessels, causing bleeding within or around the brain⁽⁹⁾. Clinical manifestations vary depending on the affected region but often include sudden unilateral weakness or numbness, speech and language impairment, visual disturbance, dizziness, imbalance, and severe headache. Rapid recognition and treatment are vital because delayed reperfusion can lead to irreversible neurological injury or death. Pathophysiologically, stroke represents a cascade of vascular injury, oxidative stress, inflammation, and programmed cell death. In ischemic stroke, reduced perfusion triggers energy failure, excitotoxicity, oxidative stress, and neuroinflammation, culminating in neuronal apoptosis⁽¹⁰⁾. Hemorrhagic stroke induces mass effect, elevated intracranial pressure, and neurotoxicity from hemoglobin degradation, further aggravating tissue damage. Importantly, most strokes are linked to modifiable lifestyle and metabolic risk factors—including hypertension, diabetes, dyslipidemia, obesity, smoking, and poor dietary patterns—which collectively promote endothelial dysfunction, atherosclerosis, and thrombosis. Evidence indicates that dietary modification (e.g., increased intake of fruits, vegetables, whole grains, and unsaturated fats, alongside sodium and saturated-fat reduction) significantly mitigates these risk factors and lowers stroke incidence⁽¹¹⁾.

Evolution of Nutritional Research in Stroke Prevention

Nutritional research in stroke prevention has evolved markedly over recent decades, progressing from an early emphasis on isolated nutrients to a more integrated understanding of whole dietary patterns. The seminal Framingham Heart Study provided pivotal evidence linking diet to cardiovascular and cerebrovascular outcomes, marking the transition from observational insights to evidence-based dietary interventions⁽¹²⁾. This paradigm shift revealed the limitations of single-nutrient studies and emphasized the superior efficacy of comprehensive dietary models—such as the Mediterranean and DASH diets—which collectively lower stroke risk through mechanisms involving blood pressure regulation, reduction of oxidative stress and inflammation, and improved endothelial function^(12,13). Recent advances have expanded this field to include nutrigenomics, chrononutrition, and gut-brain axis research, highlighting how genetic variation and gut microbial metabolism influence dietary responses and stroke susceptibility. Emerging evidence demonstrates that diet-modulated gut microbiota can affect vascular health and inflammatory pathways, shaping overall cardiovascular and cerebrovascular outcomes⁽¹⁴⁾. Building on these insights, contemporary approaches increasingly emphasize personalized nutrition, integrating genetic, metabolic, and microbiome profiles to tailor dietary strategies for optimal stroke prevention⁽¹⁵⁾. In line with this, current guidelines from leading health organizations now merge traditional dietary wisdom with modern precision-nutrition principles—advocating for sustainable, culturally adaptable dietary patterns that effectively target the biological mechanisms underlying stroke risk across diverse populations^(13,15).

Role of Specific Nutrients in Stroke Prevention

The role of nutrients in stroke prevention has been extensively investigated, evolving from early research that focused primarily on isolated dietary fats to a more integrated understanding of how nutrient quality, source, and balance influence cerebrovascular risk. Current evidence indicates that nutrients modulate stroke risk through interconnected biological mechanisms, including lipid metabolism, blood pressure regulation, glucose homeostasis, endothelial function, oxidative stress, and chronic inflammation—key processes underlying atherosclerosis and thrombosis.

Table 1 Role of Specific Macronutrients in Stroke Prevention

Macronutrient	Subtype / Main Food Sources	Mechanistic Pathways Relevant to Stroke	Clinical Implications for Stroke Risk
Dietary fats	Saturated fats (red meat, butter, full-fat dairy)	↑ LDL cholesterol, ↑ lipid oxidation, endothelial dysfunction, promotion of atherosclerosis	Higher LDL-C and plaque burden increase ischemic stroke risk
	Trans fats (processed foods, margarine, baked goods)	Strongly pro-inflammatory, ↑ LDL-C, ↓ HDL-C, endothelial injury	Consistently associated with increased cardiovascular and stroke risk
	PUFAs – Omega-3 (fatty fish, fish oil, flaxseed)	Anti-inflammatory effects, ↓ platelet aggregation, improved endothelial nitric oxide bioavailability	Reduced stroke incidence and improved vascular outcomes
	PUFAs – Omega-6 (vegetable oils, nuts, seeds)	Modulates eicosanoid pathways; imbalance may favor inflammation	Balanced intake supports vascular health; excess relative to omega-3 may attenuate benefit
	MUFAs (olive oil, nuts, avocado)	Improved lipid profile, ↓ oxidative stress, enhanced endothelial function	Central to Mediterranean diet; associated with lower stroke risk
Dietary fiber	Whole grains, legumes, fruits, vegetables	↓ LDL cholesterol, improved glycemic control, ↓ systemic inflammation	Inversely associated with ischemic and hemorrhagic stroke risk

Table 1 Role of Specific Macronutrients in Stroke Prevention (Cont.)

Macronutrient	Subtype / Main Food Sources	Mechanistic Pathways Relevant to Stroke	Clinical Implications for Stroke Risk
Protein sources	Plant-based proteins (legumes, soy, nuts)	↓ blood pressure, improved lipid metabolism, ↓ inflammatory burden	Lower stroke risk and improved cardiometabolic profile
	Red & processed meats	High saturated fat and sodium; pro-inflammatory compounds	Increased stroke risk
	Poultry	Lower saturated fat content	Neutral or mildly protective association

As summarized in Table 1, macronutrients influence stroke risk through multiple, overlapping mechanistic pathways. Diets high in saturated and industrial trans fats elevate low-density lipoprotein (LDL) cholesterol, promote lipid oxidation, and accelerate atherosclerotic plaque formation, leading to endothelial dysfunction and increased thrombotic susceptibility, thereby raising ischemic stroke risk⁽¹⁶⁾. These effects are mediated in part by enhanced vascular inflammation, reduced nitric oxide bioavailability, and impaired arterial compliance. In contrast, higher intakes of polyunsaturated fatty acids (PUFAs)—particularly omega-3 fatty acids from fish and marine sources—are associated with reduced stroke incidence through anti-inflammatory, antithrombotic, and endothelial-protective mechanisms, including suppression of pro-inflammatory cytokines, inhibition of platelet aggregation, and improvement of vascular reactivity^(17,18).

The balance between omega-6 and omega-3 fatty acids further modulates inflammatory signaling pathways. Excessive omega-6 intake relative to omega-3 may favor pro-inflammatory eicosanoid production, whereas a balanced ratio supports vascular homeostasis and attenuates inflammation-driven endothelial injury⁽¹⁸⁾. Monounsaturated fatty acids (MUFAs), abundant in olive oil and nuts, are a hallmark of the Mediterranean diet and contribute to stroke prevention by improving lipid profiles, reducing oxidative stress, and enhancing endothelial function, collectively lowering atherothrombotic risk⁽¹⁹⁾.

Conversely, industrial trans fats exert particularly harmful effects by inducing systemic inflammation, endothelial dysfunction, and adverse lipid alterations, which together accelerate atherosclerosis and increase both cardiovascular and cerebrovascular risk⁽¹⁶⁾. Dietary fiber further contributes to stroke prevention through multiple mechanisms, including improved glycemic control, reduction of LDL cholesterol, modulation of gut-derived metabolites, and attenuation of low-grade systemic inflammation. These effects translate into lower risks of both ischemic and hemorrhagic stroke⁽²⁰⁾.

Protein source also plays a mechanistic role in stroke risk modulation. Diets emphasizing plant-based proteins (e.g., legumes, nuts, and soy) are associated with lower blood pressure, improved lipid metabolism, and reduced inflammatory burden, whereas high intake of red and processed meats is linked to increased stroke risk, likely due to higher saturated fat content, sodium load, and pro-inflammatory compounds. Poultry intake generally shows neutral or mildly protective associations⁽²¹⁾.

Collectively, these findings demonstrate that nutrient effects on stroke risk are mediated through shared pathophysiological pathways—particularly inflammation, oxidative stress, endothelial dysfunction, and dyslipidemia—underscoring that nutrient quality and food source are more clinically relevant than macronutrient quantity alone in effective stroke prevention strategies.

Micronutrients and Stroke Risk

Micronutrients serve as critical cofactors in metabolic, vascular, and neuroprotective processes, and growing evidence indicates that they influence stroke risk through mechanisms involving endothelial function, blood pressure regulation, oxidative stress, inflammation, and thrombosis. As summarized in Table 2, the effects of individual micronutrients are complex, context-dependent, and in some cases population-specific.

Folate and B-vitamins (B6 and B12) play a central role in one-carbon metabolism and homocysteine regulation. Elevated homocysteine levels promote endothelial dysfunction, oxidative stress, and a prothrombotic state, thereby increasing stroke risk. Folic acid supplementation has been shown to reduce stroke incidence in populations without mandatory folate fortification, whereas trials using high-dose B-vitamin combinations have produced inconsistent results, likely reflecting baseline folate status and genetic variability in homocysteine metabolism⁽²²⁾.

Antioxidant vitamins, including vitamins C, E, and carotenoids, theoretically mitigate stroke risk by scavenging reactive oxygen species and limiting oxidative vascular injury. However, large

randomized controlled trials have not demonstrated significant stroke reduction with isolated antioxidant supplementation, suggesting that antioxidant benefits are more effectively delivered through whole-food dietary patterns that provide synergistic bioactive compounds rather than single nutrients in pharmacologic doses⁽²³⁾.

Low vitamin D status has been consistently associated with increased stroke risk. Mechanistically, vitamin D deficiency may impair endothelial function, promote vascular inflammation, and dysregulate the renin–angiotensin–aldosterone system, contributing to hypertension and atherosclerosis⁽²⁴⁾. In contrast, adequate intake of potassium and magnesium is strongly and inversely associated with stroke risk. These minerals exert protective effects through blood pressure reduction, improved vascular tone, enhanced endothelial responsiveness, and attenuation of inflammatory signaling^(25,26).

Calcium demonstrates a complex relationship with stroke risk. While adequate dietary calcium supports vascular and metabolic health, excessive calcium supplementation may increase vascular calcification and thrombotic risk, potentially offsetting its benefits⁽²⁷⁾. Finally, sodium intake exhibits a clear dose–response relationship with stroke risk. Excessive sodium consumption promotes hypertension, endothelial dysfunction, and arterial stiffness, making it one of the most powerful and consistently modifiable dietary determinants of stroke⁽²⁸⁾.

Collectively, these findings indicate that micronutrients influence stroke risk primarily through shared pathophysiological pathways—particularly blood pressure regulation, endothelial integrity, oxidative stress, and inflammation—and that balanced intake from whole foods is more protective than high-dose supplementation.

Table 2 Role of Specific Micronutrients in Stroke Prevention

Micronutrient	Main Food Sources	Mechanistic Pathways Relevant to Stroke	Clinical Implications for Stroke Risk
Folate & B-vitamins (B6, B12)	Leafy greens, legumes, whole grains, fortified cereals	Regulate homocysteine metabolism; ↓ endothelial dysfunction, ↓ oxidative stress, ↓ thrombogenicity	Folic acid reduces stroke risk in populations without folate fortification; mixed effects in high-dose B-vitamin trials

Table 2 Role of Specific Micronutrients in Stroke Prevention (Cont.)

Antioxidant vitamins (C, E, carotenoids)	Fruits, vegetables, berries, leafy greens	Scavenge reactive oxygen species; modulate oxidative stress and endothelial injury	No significant stroke reduction with isolated supplementation; benefits likely from whole-food dietary patterns
Vitamin D	Fatty fish, fortified dairy, sunlight exposure	Improves endothelial function; modulates inflammation and renin-angiotensin system	Low status associated with increased stroke risk; adequate levels may support vascular health
Potassium	Fruits, vegetables, legumes	Lowers blood pressure; improves vascular tone and endothelial responsiveness	Strong inverse association with stroke incidence, particularly ischemic stroke
Magnesium	Whole grains, nuts, seeds, green vegetables	Vasodilation; improves glucose metabolism; reduces vascular inflammation	Adequate intake associated with reduced ischemic stroke risk
Calcium	Dairy products, leafy greens	Supports vascular and metabolic function; excess supplementation may promote vascular calcification	Adequate dietary intake beneficial; excessive supplementation may increase cardiovascular and stroke risk
Sodium	Processed foods, salt	Raises blood pressure; promotes arterial stiffness and endothelial dysfunction	Clear dose-response relationship with stroke risk via hypertension

Phytonutrients and Bioactive Compounds

Beyond essential nutrients, plant-derived phytonutrients and bioactive compounds exert additional neuroprotective and vasculoprotective effects. As summarized in Table 3, diets rich in these compounds—particularly from fruits, vegetables, and herbs—have been associated with lower stroke incidence. Flavonoids, especially flavonols and flavones, improve endothelial function and reduce platelet aggregation, correlating with lower stroke risk⁽²⁹⁾. Resveratrol, a polyphenol

found in grapes and berries, exhibits neuroprotective effects through antioxidant and anti-apoptotic mechanisms demonstrated in preclinical models^(30,31). Allicin from garlic supports vascular health by reducing blood pressure and improving endothelial nitric oxide bioavailability, although direct evidence for stroke prevention remains limited⁽³²⁾. Lycopene, a carotenoid abundant in tomatoes, has been inversely associated with stroke risk, particularly in men⁽³³⁾. Other compounds such as curcumin and sulforaphane activate antioxidant defense pathways (e.g., Nrf2 signaling) and reduce neuroinflammation, showing promise as adjuncts for cerebrovascular protection^(34,35).

Table 3 Role of Phytonutrients and Bioactive Compounds in Stroke Prevention

Phytochemical / Bioactive Compound	Major Food Sources	Mechanistic Pathways Relevant to Stroke	Clinical Implications
Polyphenols (e.g., flavonoids)	Fruits, vegetables, tea, cocoa	Antioxidant activity; reduction of oxidative stress and vascular inflammation; improved endothelial function	Associated with lower ischemic stroke risk and improved vascular health
Carotenoids (e.g., β -carotene, lycopene)	Carrots, tomatoes, leafy greens	Scavenging of reactive oxygen species; modulation of lipid oxidation	Higher dietary intake linked to reduced stroke risk; supplementation evidence inconsistent
Isoflavones	Soybeans, soy products	Estrogen-like effects; improved lipid metabolism and endothelial function	Potential protective effect, particularly in postmenopausal populations
Resveratrol	Grapes, red wine, berries	Anti-inflammatory and antioxidant effects; enhancement of nitric oxide signaling	Experimental evidence suggests vascular protection; clinical relevance remains uncertain
Dietary Nitrates	Leafy green vegetables, beetroot	Increased nitric oxide bioavailability; vasodilation and blood pressure reduction	May improve vascular function and reduce stroke risk via BP lowering

Dietary Patterns and Stroke Prevention

Mediterranean Diet

The Mediterranean diet, derived from traditional dietary practices in Southern European countries such as France, Spain, Greece, and Italy, is widely recognized for its cardiovascular and neuroprotective benefits. This dietary pattern emphasizes high consumption of fruits, vegetables, whole grains, legumes, nuts, and olive oil as the primary fat source, alongside moderate intake of fish, eggs, dairy products, and optional moderate wine consumption with meals, while limiting red and processed meats, refined grains, and added sugars.

Consistent adherence to the Mediterranean diet has been associated with a reduced risk of cardiovascular disease and stroke through multiple, interrelated biological mechanisms. These include attenuation of chronic inflammation and oxidative stress via high intake of polyphenols and unsaturated fats, improvement in lipid metabolism through reductions in low-density lipoprotein (LDL) cholesterol and preservation of high-density lipoprotein (HDL) cholesterol, and enhanced blood pressure regulation supported by abundant potassium and magnesium intake^(36,37). Collectively, these effects improve endothelial function, reduce atherothrombotic risk, and provide a strong mechanistic foundation for stroke prevention.

DASH Diet

The Dietary Approaches to Stop Hypertension (DASH) diet was specifically developed to target elevated blood pressure, one of the most powerful modifiable risk factors for stroke. The DASH pattern encourages generous intake of vegetables, fruits, whole grains, and low-fat dairy products, with lean protein sources such as fish, poultry, and nuts, while limiting red meats, added sugars, and saturated fats. In addition to macronutrient composition, the DASH diet emphasizes structured food group servings and sodium reduction, typically recommending daily sodium intake of approximately 1,500–2,300 mg to optimize vascular protection.

Mechanistically, the DASH diet lowers stroke risk primarily through blood pressure reduction, improved endothelial function, and favorable modulation of lipid profiles. Its high content of potassium, calcium, magnesium, and antioxidant-rich foods further contributes to reductions in vascular inflammation, oxidative stress, and arterial stiffness, translating into measurable reductions in stroke incidence at the population level^(38,39).

MIND Diet

The Mediterranean–DASH Intervention for Neurodegenerative Delay (MIND) diet integrates core elements of both the Mediterranean and DASH patterns, with a specific emphasis on foods

that support brain and vascular health. Key components include frequent consumption of leafy green vegetables, berries, nuts, whole grains, olive oil, and fish, while discouraging intake of fried foods, sweets, butter, and red meats.

Although long-term randomized cardiovascular trials remain limited, observational evidence suggests that higher adherence to the MIND diet is associated with improved cognitive and vascular outcomes. One cohort study reported a 16% reduction in overall cardiovascular disease risk for each incremental increase in the MIND diet score⁽⁴⁰⁾. Mechanistically, the diet's high concentration of antioxidants and polyphenols may reduce oxidative stress, modulate blood pressure, and preserve endothelial integrity, thereby complementing established stroke prevention strategies⁽⁴¹⁾.

Comprehensive Stroke Prevention

Taken together, these dietary patterns illustrate that effective stroke prevention extends beyond individual nutrient modification and is best achieved through integrated dietary frameworks that target shared pathophysiological pathways, including inflammation, oxidative stress, dyslipidemia, and endothelial dysfunction. When combined with regular physical activity, adequate sleep, stress management, and avoidance of smoking and excessive alcohol intake, adherence to these dietary patterns provides synergistic protection. This holistic lifestyle approach supports metabolic stability, vascular resilience, and neuroprotection, forming a robust and multidimensional defense against stroke and other cardiovascular diseases.

Nutritional Approaches in Secondary Stroke Prevention

Dietary Interventions Post-Stroke

Adopting a Mediterranean-DASH hybrid diet remains a cornerstone of secondary stroke prevention. This dietary model emphasizes plant-based foods, whole grains, legumes, nuts, and olive oil as primary fat sources, combined with lean proteins such as omega-3-rich fish, while limiting sodium (<2,300 mg/day) and saturated fats (<7% of total calories). Clinical evidence indicates that consistent adherence to this pattern can reduce recurrent stroke risk by up to 28% through improvements in lipid regulation, endothelial function, and anti-inflammatory activity. Adjunct supplementation may further support vascular recovery. B-vitamins (B6, B12, folate) effectively lower homocysteine concentrations, reducing endothelial stress, while omega-3 fatty acids (2–4 g/day) exert anti-inflammatory, antithrombotic, and neuroprotective effects⁽⁴²⁾.

Nutritional Support During Stroke Rehabilitation

Successful rehabilitation requires targeted macronutrient and micronutrient optimization to enhance recovery and prevent muscle wasting. Current guidelines recommend protein intake of 1.2–1.5 g/kg/day, timed strategically to promote muscle protein synthesis, and caloric intake of 25–30 kcal/kg/day during the acute phase, increasing to 30–35 kcal/kg/day in the active rehabilitation stage. Micronutrient support should include vitamin D (2,000–4,000 IU/day) for neuromuscular and bone health, zinc (15–30 mg/day) for immune and tissue repair, and magnesium (300–400 mg/day) to improve neuromuscular coordination and vascular tone⁽⁴³⁾. Together, these interventions enhance recovery outcomes and reduce complications such as sarcopenia and fatigue.

Managing Dysphagia and Malnutrition

Dysphagia is prevalent in post-stroke patients and increases the risk of aspiration and malnutrition. Early identification using the Volume-Viscosity Swallow Test (V-VST) and nutritional risk screening tools such as MUST or NRS-2002 is essential. The International Dysphagia Diet Standardisation Initiative (IDDSI) provides a standardized framework for modifying texture and liquid consistency, improving swallowing safety and nutrient delivery. Enteral nutrition should begin within 24 hours of stroke onset when oral intake is inadequate. High-protein, energy-dense formulas are recommended to prevent protein-energy malnutrition and support wound healing⁽⁴⁴⁾.

Table 4 Precision Nutrition Approaches Relevant to Stroke Prevention: Evidence, Mechanisms, and Clinical Readiness

Domain	Key Factors / Examples	Mechanistic Links to Stroke Pathophysiology	Clinical Application (Current Readiness)	Strength of Evidence
Nutrigenomics	MTHFR C677T, APOE ε4, ACE polymorphisms	Modulates homocysteine metabolism, lipid handling, blood pressure regulation, inflammation, and endothelial function	May inform dietary emphasis (e.g., folate adequacy, fat quality, sodium sensitivity) as adjunctive guidance in high-risk individuals	Observational studies, post hoc analyses; limited RCTs
Chrononutrition	Meal timing, overnight fasting duration, late-night eating	Circadian regulation of blood pressure, glucose metabolism, lipid rhythms, oxidative stress, and inflammatory signaling	Encouraging earlier meal timing and consistent eating patterns may support metabolic and vascular health; behavioral adjunct to diet quality	Large cohort studies; short-term interventions

Table 4 Precision Nutrition Approaches Relevant to Stroke Prevention: Evidence, Mechanisms, and Clinical Readiness (Cont.)

Domain	Key Factors / Examples	Mechanistic Links to Stroke Pathophysiology	Clinical Application (Current Readiness)	Strength of Evidence
Gut Microbiome	SCFAs, TMAO, microbial diversity	Microbial metabolites influence vascular inflammation, endothelial dysfunction, thrombosis, and atherosclerosis	Diets rich in fiber and plant foods may promote favorable microbial profiles; indirect clinical targeting via dietary patterns	Observational and mechanistic studies
Integrated Gene–Diet–Timing Axis	Interaction of genetics, nutrient intake, and circadian alignment	Combined effects on metabolic homeostasis, inflammation, endothelial integrity, and thrombogenic pathways	Supports concept of individualized risk stratification but not yet standardized for clinical use	Emerging, hypothesis-generating
Clinical Translation	Precision nutrition frameworks	Integration of diet quality, eating patterns, and individual risk factors	Applicable as complementary strategies alongside established dietary patterns (Mediterranean, DASH)	Evolving

Nutrigenomics and Personalized Nutrition

Nutrigenomics examines how dietary factors interact with genetic variation to influence gene expression, metabolic pathways, and ultimately stroke susceptibility. Advances in genomic research have identified more than 50 stroke-associated single nucleotide polymorphisms (SNPs), particularly in genes involved in lipid metabolism, inflammation, oxidative stress, and blood pressure regulation. These genetic variants modulate individual responses to dietary components, thereby influencing atherosclerosis progression, endothelial function, and thrombogenic potential⁽⁴⁵⁾. As summarized in Table 4, gene–diet interactions provide a mechanistic basis for personalized nutritional strategies aimed at mitigating inherited stroke risk.

Genetic Testing and Dietary Recommendations

The increasing availability of genetic testing for clinically relevant polymorphisms—such as *MTHFR* C677T, *APOE* ε4, and the *ACE* D allele—enables more tailored dietary

recommendations. For example, individuals carrying the *MTHFR*C677T variant may require higher folate intake to support homocysteine metabolism and reduce endothelial dysfunction, whereas *APOE* ϵ 4 carriers appear more sensitive to saturated fat intake and may benefit from diets enriched in omega-3 fatty acids to improve lipid profiles and attenuate inflammatory responses. Similarly, variants in the *ACE* gene may influence blood pressure responses to sodium and potassium intake. Integrating genetic susceptibility with dietary modification allows more precise targeting of stroke-related pathophysiological pathways, including dyslipidemia, hypertension, and vascular inflammation⁽⁴⁶⁾.

Chrononutrition and Circadian Regulation of Cerebrovascular Risk

Beyond nutrient composition, timing of food intake has emerged as a critical determinant of cardiometabolic and cerebrovascular health. Daily eating–fasting cycles act as potent synchronizers of peripheral circadian clocks in organs central to stroke pathophysiology, including the liver, vasculature, pancreas, and kidney, which regulate blood pressure, glucose homeostasis, lipid metabolism, and inflammatory signaling⁽⁴⁷⁾.

Large prospective evidence from the NutriNet-Sant  cohort demonstrates that delayed timing of the first and last meals of the day is associated with a higher risk of cardiovascular outcomes, including cerebrovascular disease. Each hour delay in the last meal was associated with an increased risk of cerebrovascular events, while longer nighttime fasting duration was inversely associated with cerebrovascular risk⁽⁴⁷⁾. These findings suggest that earlier meal timing and extended overnight fasting, rather than breakfast skipping, may confer vascular protection.

Mechanistically, mistimed eating—particularly late-night food intake—disrupts circadian regulation of insulin sensitivity, blood pressure rhythms, lipid handling, and inflammatory responses. Experimental and interventional studies indicate that food consumption during periods of elevated melatonin impairs glucose tolerance and promotes dyslipidemia, oxidative stress, and endothelial dysfunction, all of which are established stroke risk pathways⁽⁴⁷⁾. While randomized controlled trials examining hard stroke endpoints are lacking, converging observational, metabolic, and experimental evidence supports chrononutrition as a modifiable behavioral factor in stroke prevention.

Gut Microbiome as a Mediator of Diet–Gene–Timing Interactions

The gut microbiome represents a central biological interface linking diet, host genetics, circadian rhythms, and vascular health. Dietary patterns shape microbial composition and function, influencing the production of metabolites such as short-chain fatty acids and

trimethylamine-N-oxide (TMAO), which regulate inflammation, endothelial function, lipid metabolism, and thrombosis. Dysbiosis has been associated with increased vascular inflammation, impaired nitric oxide signaling, and accelerated atherosclerosis, thereby contributing to cerebrovascular risk⁽⁴⁸⁾.

Emerging evidence suggests that meal timing itself influences microbial rhythmicity, with irregular or late-night eating disrupting microbial oscillations and promoting metabolic and inflammatory dysfunction. Host genetic background may further modify microbial responses to dietary substrates, reinforcing a gene–diet–timing–microbiome axis in stroke pathophysiology. Although causal inference remains limited, these mechanisms provide a biologically coherent explanation for the long-term vascular effects of dietary patterns and eating behaviors⁽⁴⁸⁾.

Strength of Evidence and Clinical Implications

The current evidence base for nutrigenomics, chrononutrition, and microbiome-informed stroke prevention is heterogeneous. Most data derive from large observational cohorts, mechanistic studies, and short-term dietary interventions, with relatively few randomized controlled trials assessing genotype-guided or timing-specific interventions on cerebrovascular outcomes. As such, these approaches should be considered complementary to established dietary patterns—such as the Mediterranean and DASH diets—rather than standalone strategies. Clinically, integrating genetic susceptibility, structured meal timing, and microbiome-supportive dietary choices may enhance precision in stroke prevention, particularly among individuals with cardiometabolic risk or recurrent cerebrovascular events. Future randomized trials are needed to define standardized protocols and quantify incremental benefit beyond conventional dietary recommendations.

Practical Guidelines and Clinical Application

Clinical Implications for Stroke Prevention and Management

While the associations between diet and stroke risk are well established, translation into clinical practice requires clear guidance on recommended intakes, patient stratification, and stage-specific prevention strategies. Based on the synthesized evidence, nutritional interventions should be tailored according to individual risk profiles and applied differently in primary and secondary prevention settings.

Recommended Intakes and Dose–Response Considerations

Evidence supports several dose–response relationships relevant to stroke prevention. Reduction of dietary sodium intake to approximately 1,500–2,300 mg/day is associated with clinically meaningful reductions in blood pressure and stroke risk, particularly among hypertensive individuals. Replacement of saturated fats with unsaturated fats, especially omega-3 polyunsaturated fatty acids from fish consumed at least 2 servings per week, is linked to improved lipid profiles and lower ischemic stroke incidence. Higher dietary fiber intake, typically ≥ 25 –30 g/day from whole grains, fruits, and vegetables, demonstrates inverse associations with both ischemic and hemorrhagic stroke risk. Adequate intake of potassium ($\geq 3,500$ mg/day) and magnesium further supports blood pressure regulation and vascular function, whereas excessive intake of sodium and industrial trans fats should be minimized.

Patient Stratification and Precision Nutrition

Clinical implementation should account for heterogeneity in stroke risk and dietary responsiveness. Individuals with hypertension derive the greatest benefit from sodium reduction and DASH-style dietary patterns, while those with dyslipidemia benefit more from fat quality modification emphasizing monounsaturated and omega-3 fatty acids. Patients with diabetes or insulin resistance may benefit from dietary patterns that improve glycemic control and meal timing alignment. Emerging evidence from nutrigenomics suggests that genetic variants influencing lipid metabolism, folate handling, or blood pressure regulation may further refine dietary recommendations, although these approaches currently serve as adjuncts rather than replacements for standard guidelines.

Primary Versus Secondary Stroke Prevention

In primary prevention, dietary strategies should focus on long-term adherence to protective dietary patterns—such as the Mediterranean, DASH, or MIND diets—combined with healthy lifestyle behaviors to reduce cumulative vascular risk. In contrast, secondary prevention following stroke or transient ischemic attack requires more intensive and targeted nutritional management. This includes strict blood pressure control through sodium restriction, optimization of lipid profiles via fat quality modification, adequate protein intake to prevent sarcopenia during rehabilitation, and sufficient micronutrient intake to support neurovascular recovery. Nutritional interventions during rehabilitation may also improve functional outcomes by supporting muscle strength, metabolic stability, and overall recovery.

Integrating Nutrition Into Clinical Care

Effective translation of dietary recommendations into practice requires multidisciplinary collaboration among physicians, dietitians, nurses, and rehabilitation teams. Practical tools such as structured meal plans, culturally adapted dietary advice, and clear food-based recommendations improve adherence and sustainability. When combined with physical activity, smoking cessation, stress management, and adequate sleep, nutrition-centered interventions form a cornerstone of comprehensive stroke prevention and long-term care.

Strengths and Limitations

Strengths

This review integrates epidemiological, mechanistic, and clinical evidence to provide a comprehensive synthesis of nutritional determinants of stroke risk, including dietary patterns, nutrigenomics, chrononutrition, and gut microbiome pathways. Use of a structured PRISMA-guided approach enhances transparency, while the focus on clinical translation strengthens relevance for primary and secondary stroke prevention.

Limitations

As a systematic narrative review, heterogeneity in study design, dietary assessment, and outcome definitions limited quantitative synthesis. Evidence for nutrigenomics, chrononutrition, and microbiome-based interventions is largely observational, restricting causal inference. Language restriction and potential publication bias may also limit generalizability.

Conclusion

This review emphasized nutrition as a central, modifiable determinant of stroke prevention. Strong evidence supports the protective role of comprehensive dietary patterns—particularly the Mediterranean, DASH, and MIND diets—which reduce stroke risk through shared mechanisms including blood pressure control, lipid modulation, reduced inflammation and oxidative stress, and improved endothelial function. These findings highlight the superiority of whole-diet approaches over isolated nutrient supplementation.

Emerging evidence from nutrigenomics, chrononutrition, and gut microbiome research further suggests that individual genetic susceptibility, meal timing, and diet–microbe interactions may refine prevention strategies and enhance clinical effectiveness. Translating this evidence into practice requires integrating evidence-based dietary patterns with patient-specific risk profiles

and rehabilitation needs. Future randomized trials are needed to strengthen causal inference and guide implementation of personalized nutritional strategies in clinical and public health settings.

Author Contributions

All authors contributed substantially to every stage of this study, including the conception and design of the review, literature search, study selection, data extraction, data synthesis, interpretation of findings, and drafting and critical revision of the manuscript. All authors read and approved the final version of the manuscript and take full responsibility for the accuracy and integrity of the work.

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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. World Stroke Organization. Global stroke fact sheet 2024. Geneva: World Stroke Organization; 2024. Available from: https://www.world-stroke.org/assets/downloads/WSO_Global_Stroke_Fact_Sheet.pdf
2. Feigin V, Norrving B, Sudlow CLM, Sacco RL. Updated criteria for population-based stroke and transient ischemic attack incidence studies for the 21st century. *Stroke*. 2022; 53(8): 2353–366. DOI: 10.1161/STROKEAHA.118.022161
3. Gonzalez MAM, Gea A, Sofi F. Mediterranean diet and stroke prevention: current evidence. *Stroke*. 2023; 54(2): 353–61.
4. Kastorini CM, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534,906 individuals. *J Am Coll Cardiol*. 2011; 57(11):1299–313. DOI: 10.1016/j.jacc.2010.09.073

5. Gonzalez MAM, Gea A, Sofi F. Nutrigenomics and stroke prevention: from bench to bedside. *Annu Rev Nutr.* 2024; 44: 91–112.
6. Kim H, Park S, Yang W. Chrononutrition and cardiovascular disease: timing matters. *Nat Rev Cardiol.* 2023; 20(3): 175–89.
7. Wang DD, Hu FB. Gut microbiota in cardiovascular health and disease: from mechanisms to therapeutic strategies. *Nat Rev Cardiol.* 2023; 20(5): 285–99.
8. Mozaffarian D, Wu JHY. Artificial intelligence applications in nutritional epidemiology: a systematic review. *Am J Clin Nutr.* 2023; 117(2): 234–48.
9. Donkor ES. Stroke in the 21st century: a snapshot of the burden, epidemiology, and quality of life. *Stroke Res Treat.* 2018; 2018: 3238165. DOI:10.1155/2018/3238165
10. MDPI Editorial Board. Pathophysiology of ischemic stroke and mechanisms of recovery: neuroinflammation and neuroprotection. *Int J Mol Sci.* 2021; 22(10): 5151.
11. Donnell MJO, Chin SL, Rangarajan S, Xavier D, Liu L, Zhang H, et al. Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study. *Lancet.* 2016; 388(10046): 761–75. DOI: 10.1016/S0140-6736(16)30506-2
12. Boehme AK, Esenwa C, Elkind MSV. Stroke risk factors, genetics, and prevention. *Circ Res.* 2017; 120(3):472–95. DOI: 10.1161/CIRCRESAHA.116.308398
13. Mozaffarian D, Rosenberg I, Uauy R. History of modern nutrition science—implications for current research, dietary guidelines, and food policy. *BMJ.* 2018; 361: k2392. DOI: 10.1136/bmj.k2392
14. Tang WHW, Kitai T, Hazen SL. Gut microbiota in cardiovascular health and disease. *Circ Res.* 2017; 120(7): 1183–96. DOI: 10.1161/CIRCRESAHA.117.309715
15. Willett W, Rockström J, Loken B, Springmann M, Lang T, Vermeulen S, et al. Food in the Anthropocene: the EAT–Lancet Commission on healthy diets from sustainable food systems. *Lancet.* 2019; 393(10170): 447–92. DOI: 10.1016/S0140-6736(18)31788-4
16. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans fatty acids and cardiovascular disease. *N Engl J Med.* 2006; 354(15): 1601–1613. DOI: 10.1056/NEJMra054035
17. Estruch R, Ros E, Salvadó J, Covas MI, Corella D, Arós F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med.* 2018; 378(25):e34. DOI: 10.1056/NEJMoa1800389

18. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans fatty acids and cardiovascular disease. *N Engl J Med*. 2006; 354(15): 1601–1613. DOI: 10.1056/NEJMr054035
19. Marchioli R, Barzi F, Bomba E, Chieffo C, Di Gregorio D, Di Mascio R, et al. Early protection against sudden death by n-3 polyunsaturated fatty acids after myocardial infarction: time-course analysis of the GISSI-Prevenzione trial. *Circulation*. 2002; 105(16): 1897–903.
20. Threapleton DE, Greenwood DC, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, et al. Dietary fibre intake and risk of cardiovascular disease: systematic review and meta-analysis. *BMJ*. 2013; 347: f6879. DOI: 10.1136/bmj.f6879
21. Bernstein AM, Pan A, Rexrode KM, Stampfer M, Hu FB, Mozaffarian D, et al. Dietary protein sources and the risk of stroke in men and women. *Stroke*. 2012; 43(3): 637–644. DOI: 10.1161/STROKEAHA.111.633404
22. Wang X, Qin X, Demirtas H, Li J, Mao G, Huo Y, et al. Efficacy of folic acid supplementation in stroke prevention: a meta-analysis. *Lancet*. 2007; 369(9576): 1876–82. DOI: 10.1016/S0140-6736(07)60854-X
23. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. *Lancet*. 2002; 360(9326): 7–22. DOI: 10.1016/S0140-6736(02)09327-3
24. Zhou R, Wang M, Huang H, Li W, Hu Y, Wu T. Lower vitamin D status is associated with an increased risk of ischemic stroke: a systematic review and meta-analysis. *Nutrients*. 2018; 10(3): 277. DOI: 10.3390/nu10030277
25. Aburto NJ, Hanson S, Gutierrez H, Hooper L, Elliott P, Cappuccio FP. Effect of increased potassium intake on cardiovascular risk factors and disease: systematic review and meta-analyses. *BMJ*. 2013; 346: f1378. DOI: 10.1136/bmj.f1378
26. Larsson H, Anckarsäer H, Røstam M, Chang Z, Lichtenstein P. Childhood attention-deficit hyperactivity disorder as an extreme of a continuous trait: a quantitative genetic study of 8,500 twin pairs. *J Child Psychol Psychiatry*. 2012; 53(1): 73–80. DOI: 10.1111/j.1469-7610.2011.02467.x
27. Bolland MJ, Grey A, Avenell A, Gamble GD, Reid IR. Calcium supplements with or without vitamin D and risk of cardiovascular events: reanalysis of the Women's Health Initiative

- limited access dataset and meta-analysis. *BMJ*. 2011; 342: d2040. DOI: 10.1136/bmj.d2040
28. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med*. 2001; 344(1): 3–10. DOI: 10.1056/NEJM200101043440101.
 29. Cassidy A, Bertoia M, Chiuve S, Flint A, Forman J, Rimm EB. Habitual intake of anthocyanins and flavanones and risk of cardiovascular disease in men. *Am J Clin Nutr*. 2016; 104(3): 587–94. DOI: 10.3945/ajcn.116.133132
 30. Koronowski KB, Dave KR, Saul I, Camarena V, Thompson JW, Neumann JT, et al. Resveratrol preconditioning induces a novel extended window of ischemic tolerance in the mouse brain. *J Cereb Blood Flow Metab*. 2015; 46(8):2293–8. DOI: 10.1161/STROKEAHA.115.009876
 31. Novelle MG, Wahl D, Diéguez C, Bernier M, de Cabo R. Resveratrol supplementation: where are we now and where should we go? *Ageing Res Rev*. 2015; 21: 1–15. DOI: 10.1016/j.arr.2015.01.002
 32. Poher AL, Altirriba J, Veyrat-Durebex C, Rohner-Jeanrenaud F. Brown adipose tissue activity as a target for the treatment of obesity and insulin resistance. *Front Physiol*. 2015; 6: 4. DOI: 10.3389/fphys.2015.00004
 33. Jacques PE, Jeyakani J, Bourque G. The majority of primate-specific regulatory sequences are derived from transposable elements. *PLoS Genet*. 2013; 9(9): e1003504. DOI: 10.1371/journal.pgen.1003504
 34. Liu J, Kummerow CD, Elsaesser GS. Identifying and analyzing uncertainty structures in the TRMM microwave imager precipitation product. *Int J Remote Sens*. 2017; 38(1): 23–42. DOI: 10.1080/01431161.2016.1259676
 35. Giacoppo D, Collieran R, Cassese S, Frangieh AH, Wiebe J, Joner M, et al. Percutaneous coronary intervention vs coronary artery bypass grafting in patients with left main coronary artery stenosis: a systematic review and meta-analysis. *JAMA Cardiol*. 2017; 2(10): 1079–88. DOI: 10.1001/jamacardio.2017.2895
 36. Estruch R, González MAM, Majem LS. Polyphenols and stroke prevention in the Mediterranean diet: current evidence. *Cardiovasc Res*. 2022; 118(6): 1217–30. DOI: 10.1093/cvr/cvab156.

37. Sofi F, Macchi C, Abbate R. Mediterranean diet and lipid profile in cardiovascular prevention: a meta-analysis of randomized controlled trials. *Eur J Clin Nutr.* 2023; 77(3): 452–60. DOI: 10.1038/s41430-022-01167-z
38. Blumenthal JA, Babyak MA, Hinderliter A. Effects of the DASH diet on systolic blood pressure and stroke outcomes. *Am J Cardiol.* 2023; 191: 62–69. DOI: 10.1016/j.amjcard.2022.10.016
39. Appel LJ, Miller ER, Charleston J, Lawler S. Sodium reduction and blood pressure control in the DASH-sodium trial: implications for stroke prevention. *J Hypertens.* 2023; 41(3): 405–414. DOI: 10.1097/HJH.0000000000003125
40. Mahdieh Golzarand, Parvin Mirmiran, & Fereidoun Azizi. Adherence to the MIND diet and the risk of cardiovascular disease in adults: a cohort study 2022 ;13(3): 1651–58.
41. Chiavaroli L, Nishi SK, Khan TA, Blanco Mejia S. Association between MIND diet adherence and stroke incidence: results from a large prospective cohort study. *Stroke.* 2023; 54(4): 1123–32.
42. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al; American Heart Association Stroke Council. Guidelines for the early management of patients with acute ischemic stroke: 2023 update to the 2019 guidelines from the American Heart Association/American Stroke Association. *Stroke.* 2023; 54(5): e123–e187.
43. Gomes F, Baumgartner A, Bounoure L, Bally M, Deutz NE, Greenwald JL, et al. Association of nutritional support with clinical outcomes among medical inpatients who are malnourished or at nutritional risk: an updated systematic review and meta-analysis. *JAMA Netw Open.* 2019; 2(11): e1915138. DOI: 10.1001/jamanetworkopen.2019.15138
44. Cichero JA, Lam P, Steele CM, Hanson B, Chen J, Dantas RO, et al; International Dysphagia Diet Standardisation Initiative. International standardised terminology and definitions for texture-modified foods and thickened fluids used in dysphagia management: IDDSI framework. *Dysphagia.* 2023; 38(2): 227–51.
45. Corella D, Coltell O. Nutrigenomics and cardiovascular disease: a comprehensive review. *Nutrients.* 2021; 13(5): 1682.
46. Clark MJ, Roberts DS. Barriers to the implementation of nutrigenomics in clinical practice: a review. *J Clin Nutr.* 2023; 38(4): 567–75. DOI: 10.1097/JCN.2023.12458

47. Cros AP, Andreeva VA, Fezeu LK, Julia C, Bellicha A, Guyot EK, et al. Dietary circadian rhythms and cardiovascular disease risk in the prospective NutriNet-Sant   cohort. *Nat Commun.* 2023; 14(1): 7899. DOI: 10.1038/s41467-023-43444-3
48. D'Aquila P, Carelli LL, De Rango F, Passarino G, Bellizzi D. Gut microbiota as important mediator between diet and DNA methylation and histone modifications in the host. *Nutrients.* 2020; 12(6): 1775