INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

OPEN ACCESS

Review Paper

Diet and Lifestyle Modifications in the Prevention and Management of Stroke: A Comprehensive Narrative Review

Shivam Pawar*, Kelgaonkar Prajakta, Potdar Sukrut, Awale Prathamesh, Sawandkar Shivshankar

Channabasweshwar Pharmacy College, Latur.

ARTICLE INFO

Published: 25 Aug 2026
Keywords:
stroke; primary prevention; secondary prevention; diet; Mediterranean diet; DASH diet;
physical activity; smoking cessation; obesity; sleep; lifestyle modification
DOI:
10.5281/zenodo.22095987

ABSTRACT

Background: Stroke remains one of the leading causes of death and long-term disability worldwide, with the Global Burden of Disease (GBD) 2021 study estimating 11.9 million incident strokes, 93.8 million prevalent strokes, and 7.3 million stroke-related deaths in a single year. An estimated 90% of the population-attributable risk of stroke is explained by a limited number of modifiable behavioural and metabolic factors, most of which are directly linked to diet and lifestyle . Objective: This narrative review synthesises current evidence on the role of dietary patterns and lifestyle behaviours in the primary and secondary prevention of stroke, and translates this evidence into the framework of contemporary clinical guidelines. Methods: A structured search of PubMed, Embase, Cochrane Library, and major cardiovascular/neurology journals was conducted, prioritising landmark randomised controlled trials, large prospective cohort studies, dose–response meta-analyses, and the 2024 American Heart Association/American Stroke Association (AHA/ASA) Guideline for the Primary Prevention of Stroke. Forty primary sources were elected for synthesis. Results: Adherence to a Mediterranean dietary pattern reduces composite cardiovascular events,including stroke, by up to 30% in randomised trials , while higher adherence to the Dietary Approaches to Stop Hypertension (DASH) diet is associated with an approximately 12% lower risk of stroke in meta-analysis . Each additional 200 g/day of fruit and vegetable intake lowers stroke risk by roughly 16%, whereas each 5 kg/m² increment in body-mass index (BMI) raises stroke risk by approximately 10% . Regular moderate-to-vigorous physical activity reduces stroke incidence by 20–25% , smoking cessation substantially reduces excess stroke risk within a few years, and both very short and very long sleep duration are associated with a U-shaped increase in stroke risk . Excess sodium intake and low potassium intake independently increase stroke risk, while the sodium-to-potassium ratio is an emerging composite marker of dietary risk

***Corresponding Author:** Shivam Pawar
Address: *Channabasweshwar Pharmacy College, Latur.*
Email ✉: shivampawar.me@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

INTRODUCTION

Stroke is defined as a sudden focal neurological deficit caused by an ischaemic or haemorrhagic disturbance of cerebral blood flow. It remains the second-leading cause of death and the third-leading cause of combined death and disability worldwide, ranking just behind ischaemic heart disease [1,2]. According to the Global Burden of Disease (GBD) 2021 study, stroke accounted for 7.3 million deaths globally in 2021 — 10.7% of all deaths — with the absolute burden continuing to rise despite falling age- standardised incidence rates in many high-income regions, largely as a consequence of population ageing and growth [1,2,3]. What distinguishes stroke from many other major causes of global mortality is the striking concentration of risk within a small number of modifiable factors. The landmark INTERSTROKE case-control study, conducted across 32 countries in Asia, the Americas, Europe, Africa, the Middle East, and Australia, demonstrated that ten potentially modifiable risk factors together account for approximately 90% of the population-attributable risk (PAR) of stroke, a proportion that was remarkably consistent across regions, ethnic groups, sexes, and age bands [4,5,6]. Hypertension carried the single largest attributable risk, but physical inactivity, an unhealthy diet (characterised by low fruit, vegetable, and fish intake and high sodium intake), abdominal obesity, smoking, cardiac causes, alcohol intake, psychosocial stress, and diabetes together compounded this risk substantially [6]. This concentration of risk within behavioural and dietary domains has profound clinical and public health implications: stroke is, to a very large extent, a preventable disease. Diet and lifestyle modification are not adjuncts to pharmacological risk-factor management but are foundational, evidence-based interventions in their own right, formally recognised as Class 1 recommendations in the 2024 AHA/ASA Guideline for the Primary Prevention of Stroke, which explicitly incorporates the American Heart Association's "Life's Essential 8" construct — diet, physical activity, nicotine exposure, sleep, body weight, blood lipids, blood glucose, and blood pressure — as the organising framework for both cardiovascular and brain health [7]. This review synthesises the current evidence base on dietary patterns (Mediterranean and DASH diets, sodium and potassium intake, fruit, vegetable and fibre intake, red and processed meat, fish and omega- 3 fatty acids, and sugar-sweetened/ultra-processed foods) and lifestyle behaviours (physical activity, smoking, alcohol use, body weight, and sleep) in relation to both primary and secondary stroke prevention, and situates this evidence within current clinical guidelines and practical implementation challenges.

2. GLOBAL BURDEN AND EPIDEMIOLOGY OF STROKE

The GBD 2021 study estimated 11.9 million incident strokes and 93.8 million stroke survivors worldwide in a single year, with stroke constituting 10.7% of all deaths globally — the third most common GBD- classified cause of death after ischaemic heart disease and COVID-19 [1,2]. Ischaemic stroke accounts for the majority of cases, though intracerebral and subarachnoid haemorrhage contribute disproportionately to mortality and disability-adjusted life-years (DALYs) [1]. Notably, the reduction in age-standardised stroke incidence observed between 1990 and 2015 has stagnated since 2015, with rising incidence, death, prevalence, and DALY rates observed in Southeast Asia, East Asia, Oceania, lower Socio-Demographic Index (SDI) countries, and adults younger than 70 years [1]. A parallel GBD analysis focused on adults aged 15–39 years found that ischaemic stroke, while numerically less common than ischaemic heart

disease in this age group, is disproportionately driven by dietary risk factors, underscoring that stroke prevention can no longer be considered solely a disease of later life [3]. Risk-factor attribution analyses within GBD 2021 identify high systolic blood pressure, high body-mass index, high fasting plasma glucose, tobacco use, high ambient temperature, low physical activity, dietary risks (including diets high in sodium and sugar-sweetened beverages and low in fruits, vegetables, and whole grains), and high alcohol use as the dominant contributors to the modifiable burden of stroke, mirroring and extending the findings of INTERSTROKE [1,2,3]. This convergence between global observational epidemiology and case-control data provides a coherent, actionable rationale for diet- and lifestyle-centred stroke prevention programmes at both the individual and population level.

3. THE MODIFIABLE RISK FACTOR FRAMEWORK: EVIDENCE FROM INTERSTROKE

INTERSTROKE remains the single most influential epidemiological study establishing the modifiable risk factor paradigm for stroke. In its first phase (6,000 participants, 22 countries), ten factors — hypertension, current smoking, waist-to-hip ratio, diet, physical activity, diabetes mellitus, alcohol intake, psychosocial stress and depression, cardiac causes, and the ratio of apolipoproteins B to A1 — were collectively associated with over 90% of the PAR for both ischaemic stroke and intracerebral haemorrhage [5]. The expanded second phase, encompassing more than 13,000 stroke cases and 13,000 matched controls across 32 countries, confirmed this ten-factor model while revealing important regional variation in the relative magnitude (though not the direction) of individual risk factors, with diet showing particularly notable regional heterogeneity [6]. Within the diet-related component of INTERSTROKE, a directly modifiable diet risk score derived from consumption of fruits, vegetables, and meat/fish was independently associated with stroke risk across all regions studied, and a related INTERSTROKE analysis of urinary sodium and potassium excretion confirmed that higher sodium and lower potassium excretion were independently and jointly associated with increased odds of both ischaemic and haemorrhagic stroke [6,19]. Hypertension showed a stronger association with intracerebral haemorrhage, whereas smoking, diabetes, dyslipidaemia, and cardiac causes were more strongly linked to ischaemic stroke, indicating that diet and lifestyle modification operate through partially distinct pathophysiological mechanisms depending on stroke subtype [6]. This risk-factor architecture directly informs the structure of the remainder of this review, which considers dietary and lifestyle domains in turn before integrating them within current secondary prevention and guideline frameworks.

4. DIETARY MODIFICATIONS AND STROKE RISK

4.1 The Mediterranean Diet

The Mediterranean dietary pattern — characterised by high intake of vegetables, fruits, legumes, whole grains, nuts, and extra-virgin olive oil, moderate fish and poultry intake, and low intake of red/processed meat and refined sugars — has the strongest randomised-trial evidence base of any dietary pattern studied in relation to stroke. The (PREDIMED) trial randomised 7,447 individuals at high cardiovascular risk to a Mediterranean diet supplemented with extra-virgin olive oil, a Mediterranean diet supplemented with mixed nuts, or a control low-fat diet [8]. After a median follow-up of 4.8 years, both Mediterranean diet arms achieved a 28–30% relative risk reduction in the composite primary

endpoint of myocardial infarction, stroke, or cardiovascular death; notably, when the three components of this composite were examined individually, stroke was the only endpoint that reached statistical significance on its own (hazard ratio 0.61, 95% CI 0.44–0.86), suggesting a particularly strong protective signal for cerebrovascular events specifically [8]. Mechanistic sub-studies of PREDIMED have linked these benefits to improvements in endothelial function, blood pressure, glycaemic control, lipid profile, reduced platelet aggregation and thrombogenicity, decreased low-grade systemic inflammation, and reduced initiation of antithrombotic medications during follow-up, indirectly reflecting a lower incident burden of atherothrombotic disease [9,10]. The PREDIMED-Reus sub-study additionally demonstrated a reduced incidence of type 2 diabetes with the Mediterranean diet, a metabolic pathway of particular relevance to ischaemic stroke risk [9]. Although subsequent re-analyses identified minor randomisation irregularities at some centres, leading to a formal retraction and republication of the trial, the overall magnitude and direction of the cardiovascular and stroke benefit were preserved in sensitivity analyses excluding the affected sites, and the totality of evidence — spanning observational cohorts and mechanistic sub-studies — continues to support a genuine protective effect [8,11]. On the strength of this evidence, the 2024 AHA/ASA primary prevention guideline issued its highest- strength (Class 1) recommendation in favour of a Mediterranean dietary pattern, supplemented with extra-virgin olive oil or nuts, for individuals at intermediate-to-high cardiovascular risk, citing the PREDIMED-derived relative and absolute stroke risk reduction as pivotal evidence [7].

4.2 The Dash Diet

The Dietary Approaches to Stop Hypertension (DASH) diet emphasises vegetables, fruits, legumes and nuts, whole grains, and low-fat dairy while restricting red and processed meat, sodium, and sugar- sweetened beverages. Because hypertension is the single largest modifiable risk factor for stroke, and DASH was originally designed as a blood-pressure-lowering dietary intervention, its relevance to stroke prevention is direct and mechanistically coherent [12]. In a prospective analysis of 74,404 Swedish men and women followed via the national patient and cause-of-death registers, higher adherence to a modified DASH score was associated with a significantly lower incidence of total stroke, with the association being particularly strong for haemorrhagic stroke [12]. A pooled dose–response meta-analysis of 12 prospective cohort studies comprising over 548,000 participants confirmed that higher DASH adherence was associated with a 12% reduction in stroke risk (relative risk 0.88, 95% CI 0.83–0.93), with the protective association appearing somewhat stronger in Asian than in Western populations, possibly reflecting differences in baseline sodium intake and dietary sodium-to-potassium ratios [13]. A case-control study conducted in Lebanon similarly found that high DASH-diet adherence was independently protective against ischaemic stroke and was further associated with a lower level of post-stroke disability among those who did have a cerebrovascular event, an observation with direct relevance to functional recovery in addition to primary prevention [14]. A large prospective cohort from the China Multi-Ethnic Cohort study further demonstrated an interaction between DASH adherence and other dietary exposures (such as spicy food consumption) on incident stroke risk, illustrating that dietary patterns act cumulatively rather than in isolation [15].

4.3 Sodium And Potassium Intake

Sodium and potassium intake are two of the most extensively studied single-nutrient determinants of

stroke risk, acting predominantly — though not exclusively — through their effects on blood pressure. A meta-analysis of prospective studies estimated that a reduction in dietary sodium intake of approximately 5 g/day (85 mmol/day) is associated with a 23% reduction in stroke risk [16]. Complementing this, a meta- analysis of 16 prospective cohort studies found that higher dietary potassium intake was linearly associated with lower stroke risk, independent of blood pressure adjustment in several of the pooled cohorts, suggesting mechanisms beyond blood pressure lowering alone, potentially including direct vasculo-protective, antioxidant, and anti-thrombotic effects of potassium [16,17]. A subsequent nonlinear dose–response meta-analysis encompassing over 260,000 participants and more than 10,000 stroke events demonstrated that stroke risk increased linearly with increasing sodium intake and, independently, with an increasing dietary sodium-to-potassium ratio, without evidence of a protective J- or U-shaped curve at low sodium intakes for stroke outcomes specifically [18]. This finding is reinforced by an INTERSTROKE case-control analysis of directly measured urinary sodium and potassium excretion, which confirmed that the combination of high sodium and low potassium intake conferred the greatest odds of both ischaemic and haemorrhagic stroke, and that population-level strategies emphasising potassium-rich foods (such as fruits, vegetables, legumes and low-sodium salt substitutes) alongside sodium restriction may be more effective than sodium restriction in isolation [19]. This evidence base underpins the 2024 AHA/ASA guideline's new formal discussion of potassium-enriched salt substitution as a population and individual-level stroke prevention strategy [7].

4.4 Fruits, Vegetables, And Dietary Fibre

Fruit and vegetable consumption shows one of the most consistent inverse dose–response relationships with stroke risk among all dietary exposures studied. An early meta-analysis of cohort studies found that each additional daily portion of fruit was associated with an 11% reduction in stroke risk, and each additional portion of combined fruit and vegetables with a 5% reduction [20]. A larger meta-analysis of prospective cohort studies confirmed a clear linear dose–response relationship, with high (versus low) combined fruit and vegetable intake associated with substantially reduced stroke risk, and fruit intake alone showing a somewhat stronger protective association than vegetable intake alone [20,21]. A comprehensive dose–response meta-analysis of 95 studies quantified this relationship precisely: each 200 g/day increment in combined fruit and vegetable intake was associated with a 16% reduction in stroke risk (summary relative risk 0.84, 95% CI 0.76–0.92), a considerably larger effect size than that observed for coronary heart disease or total cancer in the same analysis [22]. Dietary fibre, a major constituent of fruits, vegetables, legumes, and whole grains, appears to confer additional and partially independent benefit. In a large Swedish cohort, dietary fibre intake was inversely associated with stroke incidence in a dose-dependent manner, with cereal fibre showing a particularly robust association [23]. Proposed mechanisms include improved lipid profiles, reduced postprandial glycaemic and insulinemic excursions, favourable modulation of the gut microbiome and its metabolites (notably short-chain fatty acids), and improved endothelial function, in addition to fibre-rich foods' typically high potassium and antioxidant content [21,23].

4.5 Red And Processed Meat

In contrast to plant-based foods, higher intake of red and, in particular, processed meat is consistently associated with increased stroke risk.

A meta-analysis of prospective cohort studies found that higher red meat intake and higher processed meat intake were each associated with significant increases in stroke risk, with the association for processed meat being notably stronger, likely reflecting the contribution of sodium, nitrites, and advanced glycation end-products used in meat processing and preservation [24]. A subsequent dose–response meta-analysis confirmed that a significant elevation in total stroke risk emerged once total red meat intake exceeded approximately 50 g/day and once processed red meat intake exceeded even minimal amounts, while no significant association was observed between meat intake and haemorrhagic stroke specifically [25]. An overview of systematic reviews and meta-analyses evaluating thirteen distinct food groups in relation to stroke outcome corroborated these findings, identifying red and/or processed meat as the only food group consistently and adversely associated with stroke risk across the reviewed evidence, while nuts, fruits, vegetables, dairy foods, fish, tea, and moderate coffee/chocolate consumption showed protective associations [26].

4.6 Fish And Omega-3 Fatty Acids

Fish consumption, and the long-chain omega-3 polyunsaturated fatty acids (eicosapentaenoic acid and docosahexaenoic acid) it provides, has been extensively studied in relation to stroke. A meta-analysis of prospective cohort studies found that fish consumption was inversely associated with stroke risk, with both fatty and lean fish showing protective associations, indicating that the benefit is not attributable solely to omega-3 fatty acid content but likely also reflects the broader nutrient profile of fish (including selenium, vitamin D, and taurine) and the displacement of red/processed meat from the diet [27,28]. A systematic review and meta-analysis focused specifically on long-chain omega-3 fatty acids and cerebrovascular disease found moderate inverse associations for dietary omega-3 intake, though associations for circulating omega-3 biomarkers and for supplementation in randomised trials were more modest and less consistent, suggesting that the benefit of fish consumption may be only partially mediated by omega-3 fatty acids per se and may also reflect broader dietary pattern effects [29].

4.7 Sugar-Sweetened Beverages And Ultra-Processed Foods

The evidence base linking sugar-sweetened beverages (SSBs) and ultra-processed foods directly to stroke risk is less mature than that for the food groups above but is rapidly accumulating and directionally consistent with harm. Large US cohort analyses (the Nurses' Health Study, Nurses' Health Study II, and Health Professionals Follow-up Study) found that higher intake of ultra-processed foods was associated with increased cardiovascular disease and coronary heart disease risk, with a smaller signal for stroke specifically, while a complementary meta-analytic “Burden of Proof” modelling approach found that processed meat and sugar-sweetened beverages were each independently associated with increased cardiometabolic disease risk, including ischaemic heart disease [30,31]. The GBD 2021 risk-factor attribution analysis specifically identified diets high in sugar-sweetened beverages as a substantial and growing contributor to global ischaemic stroke DALYs between 1990 and 2021, reflecting rising consumption in low- and middle-income countries undergoing nutrition transition [3]. Mechanistically, SSBs and ultra-processed foods are thought to increase stroke risk through their contribution to obesity, insulin resistance, dyslipidaemia, hypertension, and systemic inflammation, several of which are independently established stroke risk factors in their own right [3,30,31,44].

5. LIFESTYLE MODIFICATIONS AND STROKE RISK

5.1 Physical Activity And Exercise

Physical inactivity is among the ten modifiable risk factors identified by INTERSTROKE and is independently associated with stroke risk across virtually all major epidemiological analyses [6]. A foundational meta- analysis of 23 cohort and case-control studies found that highly active individuals had a 25% lower risk of stroke incidence or mortality compared with low-active individuals in cohort studies (relative risk 0.75, 95% CI 0.69–0.82), with an even larger protective association observed in case-control studies [32]. A subsequent reanalysis using more robust statistical modelling of leisure-time physical activity data confirmed statistically significant risk reductions of 21–28% for moderate-to-high levels of leisure-time activity in both men and women, with stability of the pooled estimate demonstrated across cumulative meta-analyses dating back to 2005 [33]. More recent and larger dose–response meta-analyses have refined understanding of the shape of this relationship. A pooled analysis of over 2.6 million participants from 14 international prospective cohorts found a nonlinear inverse association between total physical activity and stroke risk, with each 10 metabolic-equivalent-hour-per-week (MET-h/wk) increment reducing risk by approximately 1% up to a plateau around 130 MET-h/wk (corresponding to a maximum 13% risk reduction), while moderate-to- vigorous physical activity showed an L-shaped association with the greatest single increment of benefit (19% risk reduction) achieved at approximately 19 MET-h/wk — a level roughly equivalent to the commonly recommended 150 minutes per week of moderate-intensity activity [34]. A separate dose–response meta-analysis of leisure-time versus occupational physical activity found that leisure-time activity, but not occupational activity, was significantly associated with reduced stroke risk, with a 22% risk reduction plateauing at approximately 25 MET-h/wk, a distinction with important implications for how physical activity counselling is tailored to different occupational groups [35]. Collectively, this evidence supports a pragmatic public health message: meaningful stroke-risk reduction begins with modest amounts of regular activity, with continued but diminishing incremental benefit at higher volumes.

5.2 Smoking Cessation

Cigarette smoking is one of the most potent and rapidly reversible risk factors for stroke. In the landmark Nurses' Health Study, current smokers had more than double the age-adjusted risk of total stroke compared with never-smokers (relative risk 2.58, 95% CI 2.08–3.19), while former smokers' risk had declined substantially towards that of never-smokers (relative risk 1.34, 95% CI 1.04–1.73), demonstrating that much — though not all — of the excess risk attenuates after cessation [36]. A large nationwide South Korean cohort study of nearly 3.8 million adults found that smoking cessation was associated with significantly reduced risk of both myocardial infarction and ischaemic stroke compared with continued smoking, and importantly, this cardiovascular benefit was preserved even among quitters who experienced substantial post-cessation weight gain, addressing a common patient concern that often undermines cessation attempts [37]. A separate Korean cohort study examining graded reduction versus complete cessation of smoking found that complete cessation, but not partial reduction in cigarette consumption, was associated with significantly lower risk of ischaemic stroke, subarachnoid haemorrhage, and myocardial infarction, indicating that “cutting down” without full cessation confers limited cardiovascular protection and that complete abstinence should remain the clinical target [38]. These findings

collectively support smoking cessation as one of the highest yield, most rapidly effective lifestyle interventions available for stroke risk reduction, with benefit accruing within a small number of years of quitting.

5.3 Alcohol Consumption

The relationship between alcohol consumption and stroke risk is more complex than that of the other lifestyle factors reviewed here, following a J-shaped or dose-dependent pattern that differs by stroke subtype and by sex. A meta-analysis of 19 cohort and 16 case-control studies found that light-to-moderate alcohol consumption (fewer than 12 g/day, roughly under one standard drink) was associated with a modestly reduced relative risk of total stroke compared with abstention, whereas heavy consumption (more than 60 g/day) was associated with substantially increased risk; the association for ischaemic stroke specifically followed a clear J-shaped curve, while for haemorrhagic stroke the relationship was more linear and unfavourable even at moderate intake levels [39]. A systematic review and meta-analysis that separately examined stroke morbidity and mortality by subtype and sex found a monotonic, near-linear increase in haemorrhagic stroke risk with increasing alcohol consumption in men, while women showed a J-shaped curve for ischaemic stroke with the lowest risk observed at consumption below one drink per day and a protective effect persisting up to approximately three drinks per day before risk increased [40]. A large Korean population-based cohort study examining change in alcohol consumption over time found that sustained mild-to-moderate drinking was associated with reduced stroke risk relative to sustained abstention, while sustained heavy drinking or an increase in consumption over time was associated with elevated risk, reinforcing that both absolute level and consistency of consumption matter [41]. A comprehensive dose–response meta- analysis further quantified these patterns, finding that the point of maximal protection differed substantially by sex, with a considerably lower threshold in women than in men [42]. Given the heterogeneity of this evidence, the absence of a demonstrated causal protective effect in Mendelian randomisation analyses (which found a log-linear increase in stroke risk with genetically predicted alcohol intake above modest thresholds, without evidence of benefit at low-moderate intake), and the well- established non-cardiovascular harms of alcohol, current guidelines do not recommend initiating alcohol consumption for cardiovascular or stroke-protective purposes, and instead emphasise moderation or abstention [7,39,42].

5.4 Body Weight And Obesity

Excess adiposity is independently associated with increased stroke risk, both directly and through its metabolic sequelae (hypertension, insulin resistance, dyslipidaemia, and systemic inflammation). A large dose–response meta-analysis of 44 prospective cohort studies encompassing over 4.4 million participants and more than 100,000 incident strokes found that each 5-unit increment in BMI was associated with a 10% increase in stroke risk (summary relative risk 1.10, 95% CI 1.06–1.13), with a clear J-shaped dose– response curve showing no excess risk below a BMI of approximately 24 kg/m² but a progressively steepening risk gradient above 25 kg/m² [44]. Notably, this and related analyses suggest that a substantial portion of the excess stroke risk associated with overweight and obesity is mediated through intermediate metabolic risk factors — elevated blood pressure, dyslipidaemia, and hyperglycaemia — which has led to increasing clinical interest in the concept of “metabolically healthy obesity” as a potentially lower-risk phenotype [44,45]. However, a meta-analysis of prospective cohort studies specifically examining

metabolic obesity phenotypes found that even individuals with "metabolically healthy" obesity carried an elevated risk of stroke compared with metabolically healthy normal-weight individuals, indicating that the absence of overt metabolic abnormality does not fully neutralise the cerebrovascular risk conferred by excess adiposity itself, and reinforcing that weight management remains an appropriate target even in individuals without overt metabolic derangement [43,45]. These findings support the 2024 AHA/ASA guideline's continued emphasis on healthy body weight as one of the eight components of Life's Essential 8 relevant to stroke prevention, alongside newer guidance on the emerging role of glucagon-like peptide- 1 receptor agonist medications in high-risk individuals with obesity and diabetes [7].

5.5 Sleep Duration And Quality

Sleep has emerged over the past two decades as an independently important, modifiable determinant of cerebrovascular risk, and is now formally incorporated into the AHA's Life's Essential 8 construct [7]. A prospective study and meta-analysis by Leng and colleagues, combining new cohort data with a systematic review of the existing literature, found that both short sleep duration (typically defined as ≤5–6 hours per night) and long sleep duration (typically ≥8–9 hours per night) were associated with increased stroke risk relative to approximately 7 hours of sleep, describing a U-shaped or J-shaped dose–response relationship that has since been replicated across multiple independent meta-analyses [46]. A separate meta-analysis of observational studies similarly found that both short and long sleep durations were associated with significantly increased stroke risk, with long sleep duration showing the stronger and more consistent association across studies, and each additional hour of sleep beyond 7 hours associated with an approximately 13% relative increase in total stroke risk in dose–response modelling [47]. A comprehensive meta-analysis incorporating 43 cohort studies confirmed these U-shaped patterns for both stroke incidence and stroke mortality, with short sleep duration associated with a hazard ratio of approximately 1.29 for incident stroke and long sleep duration associated with a hazard ratio of approximately 1.46, effect sizes broadly comparable in magnitude to those seen for several traditional vascular risk factors [46,47]. Proposed mechanisms linking suboptimal sleep duration to stroke risk include heightened systemic inflammation (elevated C-reactive protein, interleukin-6, and fibrinogen), impaired glucose metabolism and insulin resistance, sympathetic nervous system activation and blood pressure dysregulation, and, in the case of long sleep duration, a possible marker of underlying subclinical illness, depression, or sleep-disordered breathing (notably obstructive sleep apnoea) rather than a purely causal pathway [46]. These findings support incorporating sleep health — both duration and, where applicable, screening and treatment of obstructive sleep apnoea — into comprehensive stroke prevention counselling.

6. SECONDARY PREVENTION AND LIFESTYLE MODIFICATION AFTER STROKE

The importance of diet and lifestyle modification does not diminish after a first stroke event; if anything, it intensifies, because stroke survivors face a substantially elevated risk of recurrence. Recurrent stroke risk has been estimated at 6- to 15-fold higher than the risk of first stroke in the age- and sex-matched general population, with approximately 12–13% of survivors experiencing a recurrent event within the first year and a continued annual recurrence rate of 4–6% thereafter; recurrent stroke is itself an independent predictor of mortality, disability, and institutionalisation [48,49]. Because up to 90% of

strokes — first-ever and recurrent alike — are attributable to the same core set of modifiable risk factors, secondary prevention guidelines uniformly recommend that lifestyle counselling on diet, physical activity, smoking cessation, alcohol moderation, and stress reduction be initiated during the acute hospital admission and continued systematically through outpatient and primary care follow-up [48]. Randomised and quasi-experimental evaluations of structured secondary-prevention lifestyle interventions have generally found that such programmes are effective at improving lifestyle behaviours (smoking status, dietary quality, physical activity levels) and intermediate physiological outcomes (blood pressure, lipid profile, body composition), although the evidence base for a direct effect on hard outcomes such as stroke recurrence itself remains less mature, reflecting the long follow-up periods and large sample sizes required to detect such effects, as well as substantial heterogeneity in intervention design, intensity, and delivery setting across trials [51]. A systematic review synthesising trials of counselling and educational interventions found consistent improvement in modifiable physiological risk markers, particularly systolic blood pressure, though effects on health behaviours themselves were more variable and appeared to depend heavily on intervention intensity, use of structured behaviour-change techniques, and continuity of support after hospital discharge [52]. Qualitative research among stroke survivors and family members has identified important barriers to sustained lifestyle change, including limited provision of individualised lifestyle information at the point of discharge, cognitive and communication impairments that limit engagement with standard educational materials, post-stroke depression, motor impairment limiting physical activity participation, and a lack of structured longer-term follow-up once patients transition from specialist stroke services back to primary care [50]. These findings have motivated the development of theory-informed behaviour-change interventions — for example, programmes explicitly grounded in the Health Action Process Approach or the Theory of Planned Behaviour — designed to equip stroke survivors not merely with knowledge of risk factors but with the specific self-regulatory skills (goal-setting, action planning, relapse-management strategies) required to sustain lifestyle change over the long term, an approach increasingly reflected in pilot and definitive randomised secondary-prevention trials [48,53].

7. CONTEMPORARY CLINICAL GUIDELINES: THE 2024 AHA/ASA FRAMEWORK

The 2024 AHA/ASA Guideline for the Primary Prevention of Stroke, replacing the prior 2014 iteration, represents the most authoritative and current synthesis of diet- and lifestyle-related stroke prevention evidence, and explicitly organises its recommendations around the AHA's Life's Essential 8 construct: diet, physical activity, nicotine exposure, sleep health, body weight, blood lipids, blood glucose, and blood pressure [7]. Among its notable updates relative to the 2014 guideline, the 2024 document elevates the Mediterranean dietary pattern to a Class 1 (strong) recommendation for individuals at intermediate-to- high cardiovascular risk, explicitly citing the PREDIMED trial's stroke-specific findings as pivotal supporting evidence, and introduces new, more detailed guidance on potassium-enriched salt substitution as a practical, population-scalable strategy for improving the dietary sodium-to-potassium ratio [7]. The guideline further recommends periodic (every 1–5 years) formal cardiovascular risk estimation in adults aged 40–79 years to guide individualised lifestyle and pharmacological decision-making, periodic screening for modifiable stroke risk factors

(including sleep disorders and adverse social determinants of health) in all adults 18 years and older, and blood-pressure treatment targets aligned with lifestyle modification as first-line therapy for stage 1 hypertension in lower-risk individuals [7]. Importantly, the guideline situates diet and lifestyle counselling not as a stand-alone intervention but as an integrated component of a broader risk-based prevention strategy that also encompasses statin therapy, antihypertensive and antithrombotic management, newer pharmacological agents such as GLP-1 receptor agonists in appropriate high-risk individuals with diabetes and obesity, and structured attention to social determinants of health that may otherwise undermine adherence to lifestyle recommendations [7].

8. IMPLEMENTATION CHALLENGES AND FUTURE DIRECTIONS

Despite a robust and largely consistent evidence base, translating diet and lifestyle evidence into sustained real-world behaviour change remains the principal challenge in stroke prevention. Barriers operate at multiple levels: individual (competing health priorities, low health literacy, cognitive or motor impairment after stroke, psychological factors including post-stroke depression), interpersonal and social (limited family or caregiver support, socioeconomic constraints on access to healthy foods), health-system (fragmented handover between acute stroke services and primary care, limited reimbursement for structured lifestyle counselling, workforce capacity constraints), and structural/environmental (food environments dominated by ultra-processed and sodium-dense products, limited access to safe spaces for physical activity, and broader social determinants of health) [7,48,50]. Several priorities emerge for future research and practice. First, larger and longer-duration randomised trials of structured, theory-informed secondary-prevention lifestyle interventions are needed to establish a direct causal link between such programmes and hard outcomes such as stroke recurrence, rather than intermediate physiological markers alone [51,52]. Second, further dose–response and mechanistic work is warranted on newer dietary exposures — ultra-processed foods, sugar-sweetened beverages, and the gut microbiome — where the evidence base, while directionally consistent with harm, remains less mature than for established dietary patterns such as the Mediterranean and DASH diets [3,30,31]. Third, given the striking regional variation in the relative contribution of individual risk factors observed in INTERSTROKE and GBD analyses, prevention strategies will likely need to be regionally and culturally tailored rather than applied uniformly, particularly as the burden of stroke continues to shift towards lower- and middle-income countries and younger age groups [1,3,6]. Finally, integration of digital health tools, behaviour-change technique frameworks, and social-determinants-of-health screening into routine primary and stroke-service care represents a promising, though still incompletely evaluated, avenue for improving the durability of diet and lifestyle modification after both first-ever and recurrent stroke [7,48,53].

CONCLUSION

Stroke is, to an unusually large extent among major non-communicable diseases, a preventable condition, with approximately 90% of its population-attributable risk explained by a compact set of modifiable behavioural and metabolic factors [4,5,6]. Dietary pattern — particularly adherence to a Mediterranean or DASH-style diet, adequate potassium intake relative to sodium, generous fruit, vegetable, and fibre consumption, moderate fish intake, and limitation of red/processed meat and sugar-sweetened beverages — together with regular physical activity, smoking cessation, alcohol

moderation, healthy body weight, and adequate, regular sleep, constitute a coherent and extensively evidence-based framework for both primary and secondary stroke prevention [7–47]. The formal incorporation of this evidence into the 2024 AHA/ASA guideline and the Life's Essential 8 construct provides clinicians with a clear, actionable, and increasingly high-grade recommendation structure [7]. The principal challenge going forward is not the generation of further observational evidence but the design and scaled implementation of effective, equitable, and sustainable strategies for translating this evidence into durable individual and population- level behaviour change.

REFERENCES

1. GBD 2021 Stroke Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurology. 2024.
2. GBD 2021 Collaborators. Global, regional, and national burden of ischemic stroke, 1990–2021: an analysis of data from the Global Burden of Disease Study 2021. eClinicalMedicine. 2024.
3. Global, regional, and national trends in ischaemic stroke burden and risk factors among adults aged 20+ years (1990–2021): a systematic analysis of data from the Global Burden of Disease study 2021 with projections into 2050. Frontiers in Public Health. 2025.
4. O'Donnell M, Xavier D, Diener C, Sacco R, Lisheng L, Zhang H, et al.; INTERSTROKE investigators. Rationale and design of INTERSTROKE: a global case-control study of risk factors for stroke. Neuroepidemiology. 2010;35:36–44.
5. O'Donnell MJ, Xavier D, Liu L, Zhang H, Chin SL, Rao-Melacini P, et al.; INTERSTROKE investigators. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case- control study. Lancet. 2010;376:112–123.
6. O'Donnell MJ, Chin SL, Rangarajan S, Xavier D, Liu L, Zhang H, et al.; INTERSTROKE investigators. Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study. Lancet. 2016;388:761–775.
7. Bushnell C, Kernan WN, Sharrief AZ, Chaturvedi S, Cole JW, Cornwell WK, et al. 2024 Guideline for the Primary Prevention of Stroke: A Guideline From the American Heart Association/American Stroke Association. Stroke. 2024;55.
8. Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, et al.; PREDIMED Study Investigators. Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. New England Journal of Medicine. 2018;378:e34.
9. Salas-Salvadó J, Bulló M, Babio N, Martínez-González MÁ, Ibarrola-Jurado N, Basora J, et al. Reduction in the incidence of type 2 diabetes with the Mediterranean diet: results of the PREDIMED-Reus nutrition intervention randomized trial. Diabetes Care. 2011;34:14–19.
10. Castro-Barquero S, Ribó-Coll M, Lassale C, Tresserra-Rimbau A, Castañer O, Pintó X, et al. Mediterranean diet decreases the initiation of use of vitamin K epoxide reductase inhibitors and their associated cardiovascular risk: a randomized controlled trial. Nutrients. 2020;12:3895.
11. Guasch-Ferré M, Salas-Salvadó J, Ros E, Estruch R, Corella D, Fitó M, Martínez-González MA; PREDIMED Investigators.

The PREDIMED trial, Mediterranean diet and health outcomes: how strong is the evidence? Nutrition, Metabolism and Cardiovascular Diseases. 2017;27:624–632.
12. Larsson SC, Wallin A, Wolk A. Dietary Approaches to Stop Hypertension diet and incidence of stroke: results from 2 prospective cohorts. Stroke. 2016;47:986–990.
13. Adherence to the dietary approaches to stop hypertension diet and risk of stroke: a systematic review and dose–response meta-analysis of prospective cohort studies. Medicine (Baltimore). 2018.
14. Adherence to Dietary Approaches to Stop Hypertension (DASH) diet as a protective factor for ischemic stroke and its influence on disability level: a case–control study in Lebanon. 2024.
15. China Multi-Ethnic Cohort Study Group. Eating spicy food, Dietary Approaches to Stop Hypertension (DASH) score, and their interaction on incident stroke in southwestern Chinese aged 30–79: a prospective cohort study. Nutrients. 2023;15:1222.
16. D'Elia L, Barba G, Cappuccio FP, Strazzullo P. Potassium intake, stroke, and cardiovascular disease: a meta- analysis of prospective studies. Journal of the American College of Cardiology. 2011;57:1210–1219.
17. Larsson SC, Orsini N, Wolk A. Dietary potassium intake and risk of stroke: a dose–response meta-analysis of prospective studies. Stroke. 2011;42:2746–2750.
18. Jayedi A, Zargar MS, Ghomashi F, Shab-Bidar S. Dietary sodium, sodium-to-potassium ratio, and risk of stroke: a systematic review and nonlinear dose–response meta-analysis. Clinical Nutrition. 2019;38:1092–1100.
19. O'Donnell MJ, Mente A, et al.; INTERSTROKE investigators. Urinary sodium and potassium, and risk of ischemic and hemorrhagic stroke (INTERSTROKE): a case–control study. 2021.
20. He FJ, Nowson CA, MacGregor GA. Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. Lancet. 2006;367:320–326.
21. Hu D, Huang J, Wang Y, Zhang D, Qu Y. Fruits and vegetables consumption and risk of stroke: a meta-analysis of prospective cohort studies. Stroke. 2014;45:1613–1619.
22. Aune D, Giovannucci E, Boffetta P, Fadnes LT, Keum N, Norat T, Greenwood DC, Riboli E, Vatten LJ, Tonstad S. Fruit and vegetable intake and the risk of cardiovascular disease, total cancer and all-cause mortality — a systematic review and dose–response meta-analysis of prospective studies. International Journal of Epidemiology. 2017;46:1029–1056.
23. Larsson SC, Wolk A. Dietary fiber intake is inversely associated with stroke incidence in healthy Swedish adults. Journal of Nutrition. 2014;144:1952–1955.
24. Kaluza J, Wolk A, Larsson SC. Red meat consumption and risk of stroke: a meta-analysis of prospective studies. Stroke. 2012;43:2556–2560.
25. Chen GC, Lu DB, Pang Z, Liu QF. Red meat consumption and the risk of stroke: a dose–response meta-analysis of prospective cohort studies. PMID: 26935118.
26. Foroughi M, et al. Stroke and food groups: an overview of systematic reviews and meta-analyses. PMID:
1. 29143697.
27. Xun P, Qin B, Song Y, Nakamura Y, Kurth T, Yaemsiri S, Djoussé L, He K. Fish consumption and risk of stroke and its subtypes: accumulative evidence from a meta-analysis of prospective cohort studies. European Journal of Clinical Nutrition. 2012;66:1199–1207.

28. Effects of fatty and lean fish intake on stroke risk: a meta-analysis of prospective cohort studies. Lipids in Health and Disease. 2018;17:264.
29. Association between fish consumption, long chain omega-3 fatty acids, and risk of cerebrovascular disease: systematic review and meta-analysis. Mayo Clinic Proceedings. 2017.
30. Health effects associated with consumption of processed meat, sugar-sweetened beverages and trans fatty acids: a Burden of Proof study. Nature Medicine. 2025.
31. Ultra-processed food intake and cardiovascular disease, coronary heart disease, and stroke risk in three US prospective cohorts. 2025.
32. Lee CD, Folsom AR, Blair SN. Physical activity and stroke risk: a meta-analysis. Stroke. 2003;34:2475–2481.
33. Kelley GA, Kelley KS. Leisure time physical activity reduces the risk for stroke in adults: a reanalysis of a meta- analysis using the inverse-heterogeneity model. Stroke Research and Treatment. 2019;2019:8264502.
34. Li Z, Zhang Z, Zhang Y, Zhang C, Li X, Tian C, Liang J, Ma T, Huang W, Lei J. Association of total and moderate-to-vigorous physical activity with stroke risk: a dose–response meta-analysis of 2,639,086 participants from 14 international prospective cohort studies. International Journal of Stroke. 2026.
35. Leisure-time and occupational physical activity and risk of cardiovascular disease incidence: a systematic review and dose–response meta-analysis of prospective cohort studies. 2024.
36. Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Rosner B, Speizer FE, Hennekens CH. Smoking cessation and decreased risk of stroke in women. JAMA. 1993;269:232–236.
37. Cho JH, Kwon HM, Park SE, Jung JH, Han KD, Park YG, et al. Protective effect of smoking cessation on subsequent myocardial infarction and ischemic stroke independent of weight gain: a nationwide cohort study. PLoS One. 2020;15:e0235276.
38. Song YM, Cho HJ. Risk of stroke and myocardial infarction after reduction or cessation of cigarette smoking: a cohort study in Korean men. Stroke. 2008;39:2432–2438.
39. Reynolds K, Lewis B, Nolen JD, Kinney GL, Sathya B, He J. Alcohol consumption and risk of stroke: a meta- analysis. JAMA. 2003;289:579–588.
40. Patra J, Taylor B, Irving H, Roerecke M, Baliunas D, Mohapatra S, Rehm J. Alcohol consumption and the risk of morbidity and mortality for different stroke types — a systematic review and meta-analysis. BMC Public Health. 2010;10:258.

HOW TO CITE: Shivam Pawar, Kelgaonkar Prajakta, Potdar Sukrut, Awale Prathamesh, Sawandkar Shivshankar, Diet and Lifestyle Modifications in the Prevention and Management of Stroke: A Comprehensive Narrative Review, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 8, 4183-4196, https://doi.org/10.5281/zenodo.22095987