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REVIEW ARTICLE

Meat Consumption and Cerebrovascular Disease: An Updated Review of Stroke Risk and Mortality

AUTHORS & AFFILIATIONS

Jędrzej Czmyr (Corresponding Author) ORCID: 0000-0002-3898-5322 E-mail: jedrekcz@gmail.com University Clinical Center of the Medical University of Warsaw, Warsaw, Poland

Zuzanna Marczak ORCID: 0009-0002-9539-9255 E-mail: zuziam9@hotmail.com National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland

Maksymilian Sito ORCID: 0009-0009-5436-6706 E-mail: maksymilian.sito@gmail.com University Clinical Center of the Medical University of Warsaw, Warsaw, Poland

Filip Jurkowski ORCID: 0009-0003-0561-6162 E-mail: filipjurkowskiii@gmail.com District Medical Centre in Grójec, Grójec, Poland

Łukasz Wójcik ORCID: 0009-0003-1516-3478 E-mail: lukaszjaw571@gmail.com Nicolaus Copernicus Hospital, Copernicus PL Sp. z o.o., Gdańsk, Poland

Natalia Gawron ORCID: 0009-0009-4285-1755 E-mail: gawronnatalia@wp.pl Independent Public Healthcare Complex in Płońsk, Płońsk, Poland

Hubert Tomasz Bojanowski ORCID: 0009-0000-6899-6914 E-mail: hubert.bojan@gmail.com Medical University of Warsaw, Warsaw, Poland

Aleksandra Dobracka ORCID: 0009-0004-1009-3631 E-mail: lek.a.dobracka@gmail.com Masovian Bródnowski Hospital, Warsaw, Poland

Kinga Zachar ORCID: 0009-0007-8966-5867 E-mail: kinga.zachar@gmail.com Czerniakowski Hospital, Warsaw, Poland

Monika Jarowicz ORCID: 0009-0005-4752-2572 E-mail: monjarowicz@gmail.com University Clinical Centre of the Medical University of Warsaw, Warsaw, Poland

ABSTRACT

Introduction and aim of the study. Strokes are a leading cause of global mortality and disability. While diet is a key modifiable risk factor, the impact of specific proteins requires clarification. This review evaluates evidence on how unprocessed red meat, processed meat, white meat, and fish affect stroke risk to inform dietary guidelines.

Materials and methods. A literature search of PubMed, Embase, and the Cochrane Library focused on large-scale meta-analyses, prospective cohort studies, and established pathophysiology linking specific meat types with stroke outcomes.

State of knowledge. Evidence shows a distinct stroke risk gradient by meat type. Processed meat is a potent risk factor; a 50 g/day intake raises stroke risk by 11–17%, driven by sodium and nitrites inducing hypertension. Unprocessed red meat shows a modest association; a 100 g/day increase is linked to an 11–13% higher ischemic stroke risk, likely mediated by heme iron and the gut metabolite TMAO. Conversely, white meat has a neutral or slightly protective effect. Fish offers robust protection; its consumption reduces stroke risk by 14%, attributed to marine n-3 polyunsaturated fatty acids.

Conclusions. Protein choices significantly influence stroke risk. Limiting processed and unprocessed red meats, and substituting them with fish or white meat, is an effective stroke prevention strategy. Further research should clarify the impact of preparation methods and specific stroke subtypes.

KEYWORDS stroke; stroke risk; stroke mortality; meat consumption; red meat consumption; unprocessed red meat consumption; fish consumption; white meat consumption

1. Introduction and Aim of the Study

Cerebrovascular accidents, or strokes, represent a leading cause of mortality and long-term disability worldwide, imposing a massive global health and economic burden [1, 2]. With a growing and aging population, identifying and effectively managing modifiable risk factors for stroke is a critical global public health priority [2]. Among these modifiable risk factors, dietary habits play a fundamental and self-manageable role in both primary and secondary stroke prevention [1, 2].

Over recent decades, global meat consumption has steadily increased, driven significantly by economic development and nutritional transitions across the globe [3, 4]. While meat serves as a valuable dietary source of protein, essential amino acids, iron, and vitamins, excessive intake has raised substantial health concerns [3, 5]. High consumption of red meat and processed meat has been frequently implicated in the pathogenesis of major

chronic non-communicable diseases, including type 2 diabetes, cardiovascular disease, and various malignancies [3, 5].

Recent large-scale meta-analyses of prospective cohort studies have increasingly demonstrated a dose-response relationship wherein high intake of red meat, and particularly processed meat, is positively associated with an elevated risk of total and ischemic stroke [2, 5, 6]. These adverse cerebrovascular effects are hypothesized to be driven by high concentrations of saturated fatty acids, cholesterol, and heme iron in red meat, alongside the high sodium and nitrite preservatives typical of processed meats [6]. Furthermore, emerging evidence suggests that the gut microbiome's metabolism of meat-derived nutrients into pro-atherosclerotic compounds, such as trimethylamine N-oxide (TMAO), may serve as a crucial mechanistic pathway for vascular damage [4].

Conversely, the consumption of white meat, such as poultry, appears to exhibit a neutral or even inverse (protective) relationship with stroke risk, highlighting the critical importance of differentiating meat types in nutritional research [1, 7]. Therefore, the objective of this literature review is to comprehensively evaluate the current epidemiological and mechanistic evidence regarding the impact of unprocessed red meat, processed meat, and white meat consumption on stroke incidence and mortality, thereby informing evidence-based clinical practice and dietary guidelines.

2. Material and Methods

A comprehensive literature review was conducted using electronic databases such as PubMed, Web of Science, Embase, and Google Scholar. The search strategy incorporated a combination of keywords, including: “stroke”, “stroke risk”, “stroke mortality”, “meat consumption”, “red meat consumption”, “unprocessed red meat consumption”, “fish consumption”, and “white meat consumption”.

3. State of Knowledge

3.1. Unprocessed Red Meat

The Influence of Unprocessed Red Meat Consumption on Stroke Incidence and Mortality

Epidemiological evidence evaluating the relationship between unprocessed (fresh) red meat consumption—such as beef, veal, pork, and lamb—and the incidence of stroke presents a complex and occasionally conflicting picture. Several comprehensive dose-response meta-analyses of prospective cohort studies have demonstrated a modest but statistically significant positive association between unprocessed red meat consumption and stroke risk. For instance, meta-analyses have reported that an increase of one serving per day (or approximately 100 g/day) of fresh red meat is associated with an 11% to 13% increased risk of total stroke [5, 6]. When stratified by stroke subtype, high consumption of unprocessed red meat is consistently associated with an elevated risk of ischemic stroke (relative risks ranging from 1.13 to 1.15), but no statistically significant associations have been observed for hemorrhagic stroke [2, 5, 6].

Conversely, findings from individual large-scale prospective studies, such as the cohorts of Swedish Men and the Swedish Mammography Cohort, did not find a significant association between the consumption of fresh, unprocessed red meat and the incidence of total stroke or any stroke subtype, despite finding strong associations for processed meat [8, 9]. Furthermore, evidence syntheses by the NutriRECS consortium concluded that reducing unprocessed red meat intake by 3 servings per week yields only a very small decrease in stroke risk (Relative Risk 0.94), grading the certainty of this evidence as low due to the observational nature of the data and potential residual confounding [10]. In terms of stroke-specific mortality, multiple meta-analyses have concluded that unprocessed red meat consumption is not significantly associated with an increased risk of stroke-related death [1].

Pathophysiology and Underlying Mechanisms

While unprocessed red meat lacks the high sodium and nitrate/nitrite preservatives that strongly drive hypertension and endothelial dysfunction in processed meats [1, 6, 9], fresh red meat exerts pathophysiological effects on the cerebrovascular system through its intrinsic nutritional profile. Several plausible biological mechanisms have been proposed to explain its role in atherothrombosis and ischemic stroke:

• Heme Iron and Oxidative Stress: Unprocessed red meat is a primary dietary source of highly bioavailable heme iron [8, 9]. As a redox-active transitional metal, free iron can catalyze the formation of highly reactive hydroxyl free radicals via the Fenton reaction [6]. Elevated systemic iron stores impose significant oxidative stress, leading to the peroxidation of lipids, protein modification, and DNA damage [6, 11]. In the vasculature, iron-mediated oxidative injury initiates atherogenesis by promoting endothelial dysfunction, vascular inflammation, and monocyte recruitment, thereby increasing the risk of cerebrovascular ischemia [9]. • Gut Microbiota and Trimethylamine N-oxide (TMAO): Unprocessed red meat is particularly rich in the nutrient L-carnitine. Upon ingestion, commensal intestinal microbiota metabolize L-carnitine into trimethylamine (TMA), which is subsequently absorbed and oxidized in the liver by flavin monooxygenases into trimethylamine N-oxide (TMAO) [4, 12]. Elevated circulating levels of TMAO are strongly atherogenic; they enhance platelet hyperreactivity, promote foam cell formation, decrease reverse cholesterol transport, and increase the overall risk of thrombosis and atherosclerosis, establishing a direct mechanistic link to ischemic stroke [4, 12]. • Saturated Fatty Acids and Cholesterol: Unprocessed red meat contains varying amounts of saturated fatty acids (SFAs) and dietary cholesterol. High intake of SFAs can elevate plasma total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides. This dyslipidemia accelerates the development of

atherosclerotic plaques in the large cerebral and carotid arteries, directly contributing to disruptions in cerebral blood flow and the pathogenesis of cerebral infarction [6, 8, 9].

3.2. Processed Meat Consumption

The Influence of Processed Red Meat Consumption on Stroke Incidence

Epidemiological evidence strongly and consistently links the consumption of processed red meat—such as bacon, sausages, hot dogs, ham, and salami—to a significantly elevated risk of cerebrovascular events. Unlike unprocessed red meat, which shows modest or occasionally neutral associations with stroke, processed meat emerges as a potent and independent risk factor for stroke incidence and cardiovascular mortality.

Extensive dose-response meta-analyses have demonstrated that the consumption of processed meat is associated with a markedly higher risk of total and ischemic stroke. For instance, an increase of just 50 g/day of processed meat (approximately equivalent to one hot dog or two slices of bacon) is associated with an 11% to 17% increased risk of total stroke [2, 13]. Cohort studies, including the Cohort of Swedish Men and the Swedish Mammography Cohort, have corroborated these findings, reporting substantial increases in the incidence of cerebral infarction (ischemic stroke) among individuals in the highest quintiles of processed meat consumption [8]. Similar to unprocessed red meat, processed meat consumption shows no statistically significant association with the incidence of hemorrhagic stroke [5].

Pathophysiology and Underlying Mechanisms: The Effect of Processing

The deleterious cerebrovascular effects of processed red meat extend beyond its inherent saturated fat and cholesterol content. The specific methods used to preserve, cure, and flavor these meats introduce novel, highly atherogenic and vasoactive compounds. The processing itself is the primary driver of the excess stroke risk observed in epidemiological studies, operating through the following key mechanisms:

• Extreme Sodium Load and Hypertension: The most profound effect of meat processing is the massive addition of sodium. Processed red meats contain approximately 400% more sodium than fresh, unprocessed red meats. This extreme sodium load is a primary driver of elevated systemic blood pressure, which is the single most important modifiable risk factor for stroke [8, 14]. High dietary sodium significantly increases peripheral vascular resistance, impairs arterial compliance, and promotes vascular stiffness, directly precipitating the hemodynamic stress that leads to cerebrovascular events [6, 8]. • Nitrate and Nitrite Preservatives: To prevent bacterial growth and improve color and flavor, processed meats are treated with chemical preservatives, resulting in approximately 50% higher nitrate levels compared to unprocessed meat [3, 15, 16]. In the gastrointestinal tract and systemic circulation, these nitrites and nitrates are converted into highly reactive nitrogen species, including peroxynitrite and N-nitroso compounds (nitrosamines) [3, 17]. These processing byproducts induce severe endothelial dysfunction and promote atherosclerosis by increasing oxidative stress and vascular inflammation [17, 18]. • Metabolic Dysregulation from Additives: The chemical additives introduced during processing also exert systemic metabolic effects that compound stroke risk. Nitrosamines and related compounds have been shown to be toxic to pancreatic beta-cells, impairing glucose tolerance and reducing insulin secretion [3, 6, 16]. By promoting insulin resistance and endothelial dysfunction simultaneously, these processing chemicals accelerate the atherothrombotic processes that culminate in ischemic stroke [6, 17].

3.3. White Meat Consumption

The Influence of White Meat Consumption on Stroke Incidence

In contrast to the deleterious cerebrovascular effects associated with red and processed meats, epidemiological evidence evaluating the consumption of white meat—primarily

poultry such as chicken and turkey—generally demonstrates a neutral or slightly protective association with the risk of stroke.

Comprehensive meta-analyses of prospective cohort studies highlight this divergent risk profile. For example, a large-scale meta-analysis demonstrated that the highest category of white meat consumption was associated with a statistically significant 13% reduction in the incidence of total stroke compared to the lowest category of intake [1]. Similarly, another recent and extensive evidence synthesis found a robust inverse association between white meat consumption and all-cause mortality, alongside a neutral association for both cardiovascular mortality and non-fatal cardiovascular events, including ischemic and hemorrhagic stroke [7].

It is crucial to note that the preparation methods of poultry can significantly confound these associations. While fresh poultry is generally neutral or protective—with one cohort even observing that saturated fatty acids derived specifically from poultry were associated with a significantly lower stroke incidence [19]—other studies have reported conflicting data. For instance, a recent large cohort study identified a small but significant positive association between poultry intake and incident cardiovascular disease; however, researchers noted this finding was likely heavily driven by the high consumption of deep-fried poultry (e.g., fried chicken), introducing atherogenic compounds generated during high-temperature frying [20, 21].

Pathophysiology and Underlying Mechanisms

The differing cerebrovascular impact of white meat compared to red meat is largely attributed to its distinct macronutrient and micronutrient composition, which avoids the primary drivers of atherosclerosis [7]:

• Favorable Fatty Acid Profile and Cholesterol Lowering: Poultry is characterized by a lower total fat content and a substantially higher proportion of polyunsaturated fatty acids

(PUFAs) compared to mammalian meat [7, 22]. When PUFAs act as a primary dietary source of fatty acids, they effectively lower plasma total cholesterol and low-density lipoprotein (LDL) cholesterol concentrations [1, 23]. By mitigating hyperlipidemia, white meat consumption avoids driving the atherogenesis and plaque formation in large cerebral arteries that typically precipitates ischemic stroke [1]. • Reduced Heme Iron Content and Oxidative Stress: The defining characteristic of white meat is its markedly lower concentration of myoglobin and highly bioavailable heme iron [1, 7, 24]. Because it lacks this high iron load, white meat consumption does not provoke the systemic iron-catalyzed oxidative stress and lipid peroxidation associated with red meat [1, 7]. Consequently, substituting white meat for red meat helps preserve endothelial function, limits vascular inflammation, and prevents the oxidative damage that actively promotes cerebrovascular ischemia [1, 7].

3.4. Fish Consumption

The Influence of Fish Consumption on Stroke Incidence

In stark contrast to the cardiovascular risks associated with red and processed meats, epidemiological evidence consistently demonstrates that fish consumption is inversely associated with the risk of stroke, offering a highly protective cerebrovascular profile.

Large-scale, dose-response meta-analyses of prospective cohort studies have firmly established this inverse relationship, particularly concerning ischemic stroke [25]. For example, comprehensive data syntheses have revealed that each additional 100 g/day of fish consumed is associated with a 14% reduction in the risk of stroke [13]. Furthermore, compared to individuals who never or rarely consume fish, those who eat just one serving of fish per week exhibit a statistically significant 14% lower stroke risk, while consuming 2 to 4 servings per week reduces the risk by 9%, and 5 or more servings per week reduces it by 13% [2]. Studies exploring dietary substitutions within the UK Biobank cohort also demonstrated

that "fish eaters" (who exclude mammalian meat but eat fish) had a 21% lower incident risk of stroke compared to regular meat-eaters [26].

The protective effects of fish become especially clear when analyzed through the lens of specific ischemic stroke subtypes. Studies using statistical substitution models have shown that replacing unprocessed or processed red meat with fish is associated with a significantly lower incidence of large artery atherosclerosis [27]. Similarly, substituting unprocessed red meat specifically with fatty fish correlates with a significantly reduced risk of small-vessel occlusion [27].

However, researchers have noted an important nuance regarding cardioembolic stroke. While fish protects against atherothrombotic events, some cohort data suggest that a high intake of marine n-3 polyunsaturated fatty acids (PUFAs), or substituting poultry with lean fish, may paradoxically be associated with a higher rate of cardioembolism [27, 28]. It is hypothesized that this could be mediated by a potential positive association between very high fish intake and the development of atrial fibrillation, the primary driver of cardioembolic strokes, though these findings require cautious interpretation due to smaller sample sizes for this specific subtype [27].

Pathophysiology and Underlying Mechanisms

The cerebrovascular benefits of fish are primarily attributed to its unique lipid profile, particularly its high concentration of marine n-3 polyunsaturated fatty acids (PUFAs), mainly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) [13, 27, 28]. These long-chain fatty acids fundamentally alter vascular biology and hemodynamics through several key mechanisms:

• Anti-atherosclerotic and Endothelial Function: Marine n-3 PUFAs exert potent anti-inflammatory effects and directly improve endothelial function [2, 13]. High tissue concentrations of EPA are strongly linked to the preservation of microvascular function and

the inhibition of atherosclerotic plaque progression in large cerebral arteries [28, 29]. • Lipid Modulation and Hemodynamics: Consuming fish in place of saturated fat-rich mammalian meat favorably modulates the lipid profile by lowering plasma triglycerides [2, 27]. Furthermore, n-3 PUFAs are known to reduce systemic blood pressure, a primary modifiable risk factor for stroke [30]. • Anti-thrombotic Properties: Long-chain n-3 PUFAs actively inhibit platelet aggregation and adhesion, reducing overall thrombosis risk and protecting against the ischemic blockages that cause cerebral infarctions [2, 31]. • Additional Nutrients: Beyond omega-3s, fish is a prime source of other cardioprotective nutrients, including vitamin D, iodine, selenium, and specific amino acids like taurine, which has been independently suggested to have a protective cerebrovascular effect [26, 27].

Table 1. Summary of the Association Between Meat Type, Stroke Risk, and Underlying Pathophysiology

Dietary Source	Impact on Stroke Risk	Key Pathophysiological Mechanisms
Unprocessed Red Meat	Modest increase in risk: A 100 g/day increase is associated with an 11% to 13% higher risk of total stroke. Consistently linked to ischemic stroke but shows no significant association with hemorrhagic stroke.	**Heme Iron:** Catalyzes hydroxyl free radicals, leading to oxidative stress and lipid peroxidation. **TMAO:** Gut microbiota metabolize L-carnitine into TMAO, promoting foam cell formation and platelet hyperreactivity. **Lipid Profile:** High saturated fats elevate LDL cholesterol, accelerating atherosclerosis.
Processed Meat	Potent increase in risk: Consumption of just 50 g/day is associated with an 11% to 17% increased risk of total stroke. Considered an independent and strong risk factor.	**Sodium Overload:** Contains ~400% more sodium than fresh meat, directly driving hypertension and vascular stiffness. **Nitrates/Nitrites:** Converted into nitrosamines that induce severe endothelial dysfunction and vascular inflammation. **Metabolic Stress:** Additives can impair insulin secretion and promote systemic insulin resistance.
White Meat (Poultry)	Neutral to slightly protective: High consumption levels are associated with a 13% reduction in total stroke incidence compared to low intake.	**Favorable Lipids:** Higher proportion of PUFAs helps lower plasma LDL cholesterol. **Low Heme Iron:** Reduced concentrations of myoglobin prevent the iron-catalyzed oxidative damage seen with red meat.

Dietary Source	Impact on Stroke Risk	Key Pathophysiological Mechanisms
Fish	Robust protection: Each 100 g/day increase is associated with a 14% reduction in stroke risk. Replacing red meat with fish significantly lowers the incidence of large artery atherosclerosis.	**n-3 PUFAs (EPA/DHA):** Exert potent anti-inflammatory effects and improve endothelial function. **Anti-thrombotic:** Long-chain fatty acids inhibit platelet aggregation, protecting against ischemic blockages. **Hemodynamics:** Helps reduce systemic blood pressure and lower plasma triglycerides.

4. Conclusions

Current epidemiological and mechanistic evidence underscores a clear gradient of cerebrovascular risk associated with different types of meat consumption. Processed red meat emerges as the most significant dietary threat, demonstrating a strong and consistent dose-response relationship with increased stroke incidence, primarily driven by excessive sodium loads and pro-oxidative preservatives. Unprocessed red meat confers a more modest, yet significant, risk specifically for ischemic stroke, likely mediated by heme iron-induced oxidative stress and the gut microbiota-derived metabolite TMAO.

Conversely, the consumption of white meat appears largely neutral or mildly protective, while fish consumption provides robust cerebrovascular benefits, significantly reducing the risk of ischemic events through the anti-inflammatory and anti-thrombotic actions of marine n-3 polyunsaturated fatty acids.

From a clinical and public health perspective, stroke prevention strategies should prioritize the limitation of processed and unprocessed red meats. Dietary guidelines should emphasize the substitution of these high-risk proteins with fish, poultry, or plant-based alternatives to mitigate the global burden of stroke. Further research is warranted to better understand the nuances of stroke subtypes—particularly the potential association between high fish intake and cardioembolism—and to account for the confounding effects of various cooking and preparation methods.

Disclosure

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REFERENCES

1.Kim K, Hyeon J, Lee SA, Kwon SO, Lee H, Keum N, et al. Role of total, red, processed, and white meat consumption in stroke incidence and mortality: a systematic review and meta-analysis of prospective cohort studies. J Am Heart Assoc. 2017;6(9):e005983. doi:10.1161/JAHA.117.005983

2.Deng C, Lu Q, Gong B, Li L, Chang L, Fu L, et al. Stroke and food groups: an overview of systematic reviews and meta-analyses. Public Health Nutr. 2018;21(4):766-776. doi:10.1017/S1368980017003093

3.Wolk A. Potential health hazards of eating red meat. J Intern Med. 2017;281(2):106-122. doi:10.1111/joim.12543

4.Chen F, Hu W, Chen S, Si A, Zhang Y, Ma J. Stroke mortality attributable to high red meat intake in China and South Korea: an age-period-cohort and joinpoint analysis. Front Nutr. 2022;9:921592. doi:10.3389/fnut.2022.921592

5.Yang C, Pan L, Sun C, Xi Y, Wang L, Li D. Red meat consumption and the risk of stroke: a dose-response meta-analysis of prospective cohort studies. J Stroke Cerebrovasc Dis. 2016;25(5):1177-1186. doi:10.1016/j.jstrokecerebrovasdis.2016.01.040

6.Kaluza J, Wolk A, Larsson SC. Red meat consumption and risk of stroke: a meta-analysis of prospective studies. Stroke. 2012;43(10):2556-2560. doi:10.1161/STROKEAHA.112.663286

7.Lupoli R, Vitale M, Calabrese I, Giosuè A, Riccardi G, Vaccaro O. White meat consumption, all-cause mortality, and cardiovascular events: a meta-analysis of prospective cohort studies. Nutrients. 2021;13(2):676. doi:10.3390/nu13020676

8.Larsson SC, Virtamo J, Wolk A. Red meat consumption and risk of stroke in Swedish men. Am J Clin Nutr. 2011;94(2):417-421. doi:10.3945/ajcn.111.015115

9.Larsson SC, Virtamo J, Wolk A. Red meat consumption and risk of stroke in Swedish women. Stroke. 2011;42(2):324-329. doi:10.1161/STROKEAHA.110.596510

10.Zeraatkar D, Han MA, Guyatt GH, Vernooij RWM, El Dib R, Cheung K, et al. Red and processed meat consumption and risk for all-cause mortality and cardiometabolic outcomes: a systematic review and meta-analysis of cohort studies. Ann Intern Med. 2019;171(10):703-710. doi:10.7326/M19-0655

11.Wang DD, Li Y, Nguyen XM, Ho YL, Hu FB, Willett WC, et al. Red meat intake and the risk of cardiovascular diseases: a prospective cohort study in the Million Veteran Program. J Nutr. 2024;154(3):886-895. doi:10.1016/j.tjnut.2023.12.051

12.Bechthold A, Boeing H, Schwedhelm C, Hoffmann G, Knüppel S, Iqbal K, et al. Food groups and risk of coronary heart disease, stroke and heart failure: a systematic review and dose-response meta-analysis of prospective studies. Crit Rev Food Sci Nutr. 2019;59(7):1071-1090. doi:10.1080/10408398.2017.1392288

13.Allen TS, Bhatia HS, Wood AC, Momin SR, Allison MA. State-of-the-art review: evidence on red meat consumption and hypertension outcomes. Am J Hypertens. 2022;35(8):679-687. doi:10.1093/ajh/hpac064

14.Grosso G, La Vignera S, Condorelli RA, Godos J, Marventano S, Tieri M, et al. Total, red and processed meat consumption and human health: an umbrella review of observational studies. Int J Food Sci Nutr. 2022;73(6):726-737. doi:10.1080/09637486.2022.2050996

15.Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. Circulation. 2010;121(21):2271-2283. doi:10.1161/CIRCULATIONAHA.109.924977

16.Borowiecka M, Góralczyk W, Zając M, Brzezińska A. Red meat and different dietary patterns and cardiovascular health - the review. J Educ Health Sport. 2021;11(9):282-288. doi:10.12775/JEHS.2021.11.09.034

17.Associations of the consumption of unprocessed red meat and processed meat with the incidence of cardiovascular disease and mortality, and the dose-response relationship: a systematic review and meta-analysis of cohort studies. Crit Rev Food Sci Nutr. 2023;63(27):8443-8456. doi:10.1080/10408398.2022.2058461

18.Vogtschmidt YD, Soedamah-Muthu SS, Imamura F, Givens DI, Lovegrove JA. Replacement of saturated fatty acids from meat by dairy sources in relation to incident cardiovascular disease: the European prospective investigation into cancer and nutrition (EPIC)-Norfolk study. Am J Clin Nutr. 2024;119(6):1495-1503. doi:10.1016/j.ajcnut.2024.04.007

19.Zhong VW, Van Horn L, Greenland P, Carnethon MR, Ning H, Wilkins JT, et al. Associations of processed meat, unprocessed red meat, poultry, or fish intake with incident cardiovascular disease and all-cause mortality. JAMA Intern Med. 2020;180(4):503-512. doi:10.1001/jamainternmed.2019.6969

20.Sun Y, Liu B, Snetselaar LG, Robinson JG, Wallace RB, Peterson LL, et al. Association of fried food consumption with all cause, cardiovascular, and cancer mortality: prospective cohort study. BMJ. 2019;366:l4897. doi:10.1136/bmj.k5420

21.Bergeron N, Chiu S, Williams PT, King M, Krauss RM. Effects of red meat, white meat, and nonmeat protein sources on atherogenic lipoprotein measures in the context of low compared with high saturated fat intake: a randomized controlled trial. Am J Clin Nutr. 2019;110(1):24-33. doi:10.1093/ajcn/nqz035

22.Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. PLoS Med. 2010;7(3):e1000252. doi:10.1371/journal.pmed.1000252

23.Klurfeld DM. Research gaps in evaluating the relationship of meat and health. Meat Sci. 2015;109:86-95. doi:10.1016/j.meatsci.2015.05.022

24.Quintana Pacheco DA, Sookthai D, Wittenbecher C, Graf ME, Schübel R, Johnson T, et al. Red meat consumption and risk of cardiovascular diseases-is increased iron load a possible link? Am J Clin Nutr. 2018;107(1):113-119. doi:10.1093/ajcn/nqx014

25.Petermann-Rocha F, Parra-Soto S, Gray S, Anderson J, Welsh P, Gill J, et al. Vegetarians, fish, poultry, and meat-eaters: who has higher risk of cardiovascular disease incidence and mortality? A prospective study from UK Biobank. Eur Heart J. 2021;42(12):1136-1143. doi:10.1093/eurheartj/ehaa939

26.Venø SK, Bork CS, Jakobsen MU, Lundbye-Christensen S, Bach FW, McLennan PL, et al. Substitution of fish for red meat or poultry and risk of ischemic stroke. Nutrients. 2018;10(11):1648. doi:10.3390/nu10111648

27.Spence JD. Nutrition and risk of stroke. Nutrients. 2019;11(3):647. doi:10.3390/nu11030647

28.Mason RP, Libby P, Bhatt DL. Emerging mechanisms of cardiovascular protection for the omega-3 fatty acid eicosapentaenoic acid. Arterioscler Thromb Vasc Biol. 2020;40(5):1135-1147. doi:10.1161/ATVBAHA.119.313286

29.Miller PE, Van Elswyk M, Alexander DD. Long-chain omega-3 fatty acids eicosapentaenoic acid and docosahexaenoic acid and blood pressure: a meta-analysis of randomized controlled trials. Am J Hypertens. 2014;27(7):885-896. doi:10.1093/ajh/hpu024

30.Bergeron N, Chiu S, Williams PT, King SM, Krauss RM. Effects of red meat, white meat, and nonmeat protein sources on atherogenic lipoprotein measures in the context of low compared with high saturated fat intake: a randomized controlled trial. Am J Clin Nutr. 2019;110(1):24-33. doi:10.1093/ajcn/nqz035

31.Johansson A, Acosta S, Mutie PM, Sonestedt E, Engström G, Drake I. Components of a healthy diet and different types of physical activity and risk of atherothrombotic ischemic stroke: A prospective cohort study. Front Cardiovasc Med. 2022;9:993112. Published 2022 Oct 14. doi:10.3389/fcvm.2022.993112