

Review

The Carnivore Diet and Cardiovascular Disease Risk: A Narrative Review

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Abstract

The carnivore diet consists of meat, fish, eggs, and animal fats, and excludes all plant-based foods. Controlled prospective studies have not evaluated the implications of the carnivore diet for cardiovascular safety, and the existing evidence base remains limited in quality and scope. This narrative review evaluated the scientific literature on the carnivore diet to assess nutrient adequacy, metabolic effects, and alignment with established dietary guidelines for cardiovascular disease (CVD) prevention. We searched PubMed and Google Scholar through April 2026. Eligible studies included modeling research, surveys, case reports, and reviews of carnivore diets; findings from ketogenic and low-carbohydrate diets were drawn upon extensively, given the absence of carnivore-specific trial data. Carnivore diets provide high intakes of saturated fat, cholesterol, sodium, heme iron, and protein and lack fiber and essential nutrients such as folate, vitamin C, magnesium, and potassium. Reported short-term benefits (e.g., improved glycemic control, weight loss, and elevated HDL-cholesterol) are derived primarily from self-reported survey data and case reports, and are therefore highly



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susceptible to selection, recall, and reporting biases, alongside the absence of objective clinical verification. Significant elevations in total and LDL-cholesterol were also documented. The carnivore diet is inconsistent with public health guidelines recommending plant-based dietary patterns for CVD risk reduction, and current evidence does not support its long-term safety. Based on currently available evidence, the carnivore diet cannot be considered a heart-healthy eating pattern; however, well-designed prospective trials are needed to definitively characterize its long-term cardiovascular effects.

Keywords

Carnivore diet; low-carbohydrate diets; ketogenic diets; cardiovascular diseases; atherosclerosis; saturated fatty acids; low-density lipoprotein cholesterol; heart-healthy diet patterns

1. Introduction

The carnivore diet is a very low-carbohydrate diet that consists exclusively of animal-sourced foods, such as meat, fish, eggs, and animal fats, and aims to eliminate all plant-based foods [1-3]. Social media personalities and authors have promoted this dietary pattern, claiming health benefits and driving increasing interest in recent years [1, 3-5]. Health is the primary reason people report transitioning to a carnivore diet [5], and adherents report resolving a range of ailments, from obesity and diabetes to autoimmune conditions [1, 3, 4]. The official carnivore diet community website features over 1,000 anecdotes from individuals reporting relief from chronic diseases on this meat-exclusive diet [6].

The carnivore diet does not exist in isolation. It represents the terminal point of a decades-long debate over carbohydrate restriction, moving from Atkins-style low-carbohydrate regimens through therapeutic ketogenic diets to dietary patterns that exclude entire food groups by principle rather than by clinical indication. That debate remains unresolved on its central question: whether the favorable short-term metabolic changes produced by carbohydrate restriction are offset over time by the atherogenic lipid profile that accompanies the high saturated fat intakes such diets typically entail [7-9]. The carnivore diet intensifies both sides of this trade-off simultaneously: carbohydrate intake can approach zero. In contrast, saturated fat, cholesterol, and heme iron intakes typically reach levels rarely observed in any studied population. It does so, moreover, while removing the plant foods that ketogenic and low-carbohydrate patterns typically retain. It also belongs to a second, distinct category: exclusionary diets, in which entire food groups are eliminated. Exclusionary patterns warrant separate scrutiny because their risks arise not only from excess intake but also from what is structurally unobtainable. This applies to any pattern that removes a food group: where the excluded group is the principal dietary source of a nutrient, adequacy depends on deliberate substitution or supplementation. Two features distinguish the carnivore diet in this respect. Dietary fiber has no animal-source substitute, so no adjustment within the diet's own rules can supply it; the same is true of plant-derived phytochemicals, which likewise have no animal-source equivalent, although they are not classified as essential nutrients; and supplementation is

uncommon among adherents rather than routine, with 37% reporting no use of any vitamin supplement and 80% reporting never taking a multivitamin [3].

Existing reviews of ketogenic and low-carbohydrate diets have examined carbohydrate restriction as a metabolic intervention, typically in populations with obesity, type 2 diabetes (T2D), or epilepsy, and typically in patterns that retain non-starchy vegetables, nuts, and seeds [2, 10, 11]. Consequently, those reviews cannot address the two features that distinguish the carnivore diet: the complete elimination of dietary fiber and plant-derived micronutrients and phytochemicals, and protein intakes that commonly exceed three times the Recommended Dietary Allowance [1, 5]. Existing carnivore-specific publications have been scoping or narrative reviews of the diet's general health claims [11, 12], analyses confined to nutrient adequacy [1], or assessments restricted to athletic performance [10]; none has evaluated the diet specifically against cardiovascular risk. The present review is distinguished by its cardiovascular focus, its explicit separation of carnivore-specific evidence from the indirect ketogenic and low-carbohydrate literature used to contextualize it, and by its treatment of plant-food exclusion, rather than carbohydrate restriction alone, as a cardiovascular exposure.

Despite growing popularity, direct scientific evidence on the relationship between the carnivore diet and surrogate or clinical outcomes remains limited. Clinical trials examining health outcomes on a carnivore diet are lacking, and published research is confined to survey data, case reports, modeling studies, and scoping and narrative reviews [1-3, 5, 10-21]. Without empirical safety data, concerns have been raised that carnivore diets may pose significant health risks, particularly for cardiovascular diseases (CVD) [5, 15]. Abstinence from plant foods necessarily precludes the intake of dietary fiber, cardioprotective micronutrients and phytochemicals [1, 3]. The diet is also high in saturated fat and cholesterol, which are established contributors to elevated low-density lipoprotein (LDL) cholesterol levels and an increased atherosclerotic risk [7, 15].

Current public health recommendations for mitigating cardiovascular risk include increasing consumption of fruits, vegetables, and other whole plant foods and limiting saturated fat intake [22-24]. The divergence between these recommendations and the dietary composition of carnivore diets suggests a need to review the evidence to clarify whether this diet is a safe and heart-healthy eating pattern or whether the data indicate it may increase CVD risk. This question has public health implications that go beyond individual followers of the carnivore diet. Content promoting this diet is widely shared on social media platforms, podcasts, and commercial books, mainly by individuals who lack clinical or nutritional qualifications and operate outside any regulatory oversight or peer-review framework [4, 6, 25-27]. A content analysis of 1,169 Instagram posts from 19 carnivore diet content creators, whose median following was 114,000 and of whom approximately one in six held a medical or nursing qualification, found that every health-related claim was framed positively, that negative portrayals of science, politics, and the pharmaceutical industry recurred throughout, and that the most common position taken on dietary supplements was that they are unnecessary on this diet [27]. The diet is adopted preferentially by individuals seeking to resolve existing chronic disease [3, 5], and that population reports a measurable burden of cardiovascular risk at the point of adoption: in the largest survey, respondents reported having experienced overweight or obesity (46%), lipid abnormalities (21%), diabetes or insulin resistance (20%), hypertension (18%), and cardiovascular conditions (6%), and among those reporting pre-diet laboratory values the median LDL-cholesterol was 126 mg/dL [3]. The potential for population-level harm is therefore concentrated precisely where the margin for error is smallest. A clear, evidence-graded appraisal of

what is and is not known about the cardiovascular consequences of this diet is therefore a matter of public health relevance. This narrative review examined the carnivore diet, reported short-term health effects, and implications for CVD risk factors, clinical outcomes, and total mortality.

2. Materials and Methods

A narrative review was conducted to synthesize the existing scientific literature evaluating the effects of the carnivore diet on cardiovascular health, including both surrogate biomarkers and clinical endpoints. A literature search was conducted using PubMed and Google Scholar databases from their inception through April 2026, using combinations of the terms “carnivore diet,” “zero-carb diet,” “all-meat diet,” “animal-based diet,” “atherosclerosis,” “heart disease,” and “cardiovascular disease,” supplemented by hand-searching of the reference lists of relevant articles. The Boolean strings used in the original search were not recorded verbatim at the time. To provide a reproducible specification of the search rather than a reconstruction of it, the PubMed component was re-executed in full on 11 September 2026 using the structured strings set out below, with the number of records returned on that date reported for each. We report these strings as executed on that date, and readers re-running them will obtain the stated yields. We used two strings: the first to identify carnivore-specific literature, and the second to identify ketogenic and low-carbohydrate literature used as indirect evidence.

PubMed, primary search (carnivore-specific); 68 records returned on 11 September 2026:

(“carnivore diet”[tiab] OR “carnivorous diet”[tiab] OR “zero-carb diet”[tiab] OR “zero carbohydrate”[tiab] OR “all-meat diet”[tiab] OR “meat-only diet”[tiab] OR “exclusive meat diet”[tiab] OR “animal-based diet”[tiab]) AND (“Cardiovascular Diseases”[MeSH] OR “Atherosclerosis”[MeSH] OR “Cholesterol”[MeSH] OR “Lipids”[MeSH] OR “Blood Pressure”[MeSH] OR “Nutritional Status”[MeSH] OR cardiovascular[tiab] OR “heart disease”[tiab] OR atherosclerosis*[tiab] OR cholesterol[tiab] OR lipid*[tiab] OR “blood pressure”[tiab] OR nutrient*[tiab])

PubMed, secondary search (indirect ketogenic and low-carbohydrate evidence); 435 records returned on 11 September 2026:

(“Diet, Carbohydrate-Restricted”[MeSH] OR “Diet, Ketogenic”[MeSH] OR “low-carbohydrate”[tiab] OR ketogenic[tiab]) AND (“Cardiovascular Diseases”[MeSH] OR “Cholesterol, LDL”[MeSH] OR “Mortality”[MeSH] OR “Neoplasms”[MeSH]) AND (randomized controlled trial[pt] OR meta-analysis[pt] OR systematic review[pt] OR “Cohort Studies”[MeSH])

Google Scholar was searched using the following queries, with the first 200 records of each screened in relevance order:

“carnivore diet” AND (cardiovascular OR “heart disease” OR cholesterol OR atherosclerosis OR lipid OR mortality) “all-meat diet” OR “zero-carb diet” AND (cardiovascular OR cholesterol)

Filters applied to both databases were human studies and the English language. Google Scholar record yields are not reported because the platform does not return stable or reproducible record counts.

Inclusion criteria were articles that examined diets composed predominantly or exclusively of animal products, as well as articles reporting on nutrient intake or adequacy, cardiovascular risk

markers (e.g., blood lipids), or clinical CVD-related health outcomes. Given the substantial absence of carnivore diet-specific trial data, findings from ketogenic and low-carbohydrate diets were drawn upon extensively throughout this review. These diets share the carnivore diet's defining feature of severe carbohydrate restriction and its associated metabolic effects [3, 10, 11], though they differ importantly from carnivore diets in several respects. Ketogenic diets typically allow non-starchy vegetables, nuts, and seeds, so they differ in fiber intake, phytonutrient content, and protein levels [3, 10]. Very high protein intakes characteristic of carnivore diets may have distinct metabolic and cardiovascular effects not fully captured by research on ketogenic diets; therefore, findings from these related diets should be interpreted with appropriate caution when applied to carnivore-specific contexts. Acceptable article types included cross-sectional surveys, cohort studies, randomized controlled trials (RCTs), case reports, case series, meta-analyses, mechanistic studies, and scoping and narrative review articles. We excluded publications not written in English, those that did not address human diets, and those that did not report any CVD-related measures.

Carnivore-specific trial data are effectively absent. Ketogenic and low-carbohydrate literature was therefore used as indirect evidence and was selected and weighted according to explicit rules. Selection was restricted to the highest available tier of design for each question: randomized controlled trials, meta-analyses of randomized controlled trials, and prospective cohort studies were eligible, whereas uncontrolled ketogenic diet case series were not, on the grounds that they add no evidentiary value beyond the carnivore-specific case reports already included. We weighted studies based on the degree of compositional overlap between the studied diet and the carnivore diet for the specific exposure. Indirect evidence was accorded the greatest weight where the mechanism depends on carbohydrate restriction per se, which the two patterns share: for example, ketosis, glycemic response, and triglyceride lowering. It was accorded intermediate weight where the mechanism depends on total fat and saturated fat load, which overlap substantially but not completely. It was accorded the least weight, and is identified as such in the text, where the mechanism depends on features on which the patterns diverge: fiber intake, phytochemical provision, food processing level, and absolute protein load. Under this scheme, no conclusion in this review rests on indirect evidence alone where carnivore-specific data exist, and every claim supported only by indirect evidence is explicitly identified as an extrapolation at the point it is made.

We extracted data on nutrient composition, reported health benefits or adverse effects, changes in cardiovascular risk factors (e.g., serum lipids, blood pressure, glycemic markers, and inflammatory markers), and any available clinical cardiovascular outcomes. The review was organized into thematic sections on nutrient adequacy, metabolic and cardiovascular effects, reported benefits and harms, and alignment with established dietary guidelines. Formal risk-of-bias assessment was not appropriate for the primarily descriptive and observational literature available. We therefore evaluated the findings in the context of the study design limitations. We interpreted survey data, case reports, and case series as hypothesis-generating and susceptible to self-selection and reporting biases. Epidemiologic data and expert consensus statements were included to strengthen the contextual analysis and to assess the alignment or divergence of the carnivore diet with current evidence-based dietary recommendations for CVD prevention. Given the narrative nature of this review, a formal quality assessment tool was not applied. Instead, findings were interpreted in light of the study design hierarchy, with evidence from randomized controlled trials and prospective cohort studies weighted more heavily than cross-sectional surveys, case series, and case reports, the latter of which were treated as hypothesis-generating rather than confirmatory.

The predominance of survey and case report data in the carnivore-specific literature introduces biases that consistently operate in one direction and must be carried forward into the interpretation of every finding in this review. Five are material. First, selection bias: the two available datasets were recruited from within the carnivore community itself, one through pro-carnivore social media groups [3] and one at a carnivore diet convention [5], so participation required sustained adherence and continued engagement with that community, conditions that systematically exclude individuals who abandoned the diet due to adverse effects, poor tolerance, or lack of benefit. The resulting samples are drawn from the favorable tail of the response distribution, and the reported prevalence of adverse effects, as low as under 1% in one survey [3], should be read as a lower bound of unknown magnitude rather than as an estimate. Second, reporting and recall bias: outcomes were retrospectively self-assessed by participants who were aware of the hypothesis under test and frequently invested in it, and biochemical values, where reported at all, were self-transcribed and not verified by an investigator or physician [3, 5, 28]. Third, publication bias specific to case reports: individual cases reach publication because they are instructive, which favors the striking result in either direction and precludes any inference about frequency; the published carnivore cases accordingly include both marked clinical improvement [21] and life-threatening harm [13, 29], and the ratio between them in the literature provides no information about the ratio in practice. Fourth, healthy user and co-intervention bias, and multicollinearity: adopting a carnivore diet removes ultra-processed foods, alcohol, added sugars, and refined grains, and frequently coincides with weight loss and increased attention to health behaviors more generally [3, 5], so improvements cannot be attributed to animal-food consumption rather than to these concurrent changes. Separating the effect of any single dietary exposure from the correlated dietary and lifestyle changes that accompany it is a general problem in nutrition research and has been examined in detail elsewhere [30]. Fifth, regression to the mean: because the diet is preferentially adopted by individuals seeking to resolve existing health problems [3, 5], initiation may coincide with periods in which symptoms or biomarkers are unusually poor, and in uncontrolled before-and-after observations, subsequent movement toward a level typical for that individual may be misattributed to the dietary intervention, particularly for outcomes that fluctuate over time. Consequently, we treat the survey and case report data assembled here as hypothesis-generating and are reported with their direction of bias made explicit; they are not used to support any causal claim, and they cannot exclude harms that the recruitment methods were structurally incapable of detecting.

To provide transparency about the composition of the evidence base, the carnivore-specific sources included in this review are summarized by study design in Table 1, together with the study designs for which no carnivore-specific evidence exists.

Table 1 Carnivore-specific sources included in this review, by study design. Counts refer to sources reporting on carnivore, all-meat, or exclusively animal-based diets as the exposure; the ketogenic and low-carbohydrate sources used as indirect evidence are not included.

Study design	n	References
Dietary modeling and nutrient analysis	2	[1, 31]
Cross-sectional surveys	2	[3, 5]
Case reports and case series	7	[13, 14, 17-19, 21, 29]
Scoping, narrative, and critical reviews	7	[10-12, 15, 16, 20, 32]
Commentaries on the carnivore diet literature	1	[28]
Popular and lay sources (books, websites)	8	[4, 6, 25, 26, 33-36]
Randomized controlled trials	0	None identified
Controlled feeding studies	0	None identified
Prospective cohort studies	0	None identified
Studies reporting clinical cardiovascular events	0	None identified
Studies reporting total mortality or cancer outcomes	0	None identified

2.1 Ethics Statement

Not applicable. This narrative review did not collect original data from human subjects, animals, cell lines, or plant material, and therefore did not require ethics committee review or approval.

3. Nutrient Composition of the Carnivore Diet

3.1 Elimination of Plant Foods and Nutrient Implications

Eliminating plant foods from the diet has important implications for nutrient adequacy. Plant-based foods are primary sources of nutrients, including dietary fiber, vitamin C, vitamin K1, folate, magnesium, potassium, numerous phytochemicals, and concentrated antioxidants, which are obtained in significantly smaller quantities in animal foods [37-39]. Conversely, animal foods are rich in vitamins A and B12, iron, zinc, and protein [40]. Table 2 summarizes the key nutrient characteristics of the carnivore diet relative to recommended intakes.

Table 2 Nutrient analysis of carnivore diet modeling.

Nutrient	Nutrient Reference Value Achievement
Fiber	×
Vitamin A	✓
Thiamin	×
Riboflavin	✓
Niacin	✓
Vitamin B6	✓
Folate	↔
Vitamin B12	✓
Vitamin C	×
Calcium	×
Iron	↔
Iodine	↔
Magnesium	×
Phosphorus	✓
Potassium	×
Selenium	✓
Sodium	×
Zinc	✓

Adapted from Goedeke et al., 2025. ✓ = intake meets or exceeds the nutrient reference value; × = intake falls below the nutrient reference value; ↔ = intake approximates the nutrient reference value.

3.2 Micronutrient Strengths and Excess Animal Protein

Dietary analyses of three-day food logs or 24-hour recalls from individuals following carnivore diets have not been published in the scientific literature. However, Goedeke et al. (2025) [1] examined four theoretical carnivore diet meal plans for adult men and women. The researchers compared their nutritional content with national nutrient reference values and found that carnivore diets can meet or exceed recommended intakes for vitamins A, B2, B3, B6, and B12, as well as phosphorus, selenium, and zinc. Modeled protein intake was also high, exceeding three times the adult Recommended Dietary Allowance of 0.8 g/kg/day [1]. This protein intake aligns with self-reported meat intake in a survey of 24 individuals following a carnivore diet, in which the mean was 626 g/day [5]. It is essential to meet daily protein needs, but high intakes of animal protein may also increase cardiovascular risk. Results from a meta-analysis of prospective cohort studies have demonstrated that higher intakes of animal protein are associated with increased risk of CVD mortality [41]. Carnivore diets have also been endorsed for providing retinol, vitamin K2, creatine, carnitine, and choline [5, 25, 26]. However, current evidence does not support the claim that animal-derived sources of these nutrients are necessary or associated with improvements in CVD risk or other health outcomes [42, 43].

The design of these meal plans matters for interpreting their results. Goedeke et al. (2025) built four single-day plans for hypothetical average Australian adults, two female and two male, with the

pairs differing according to whether dairy or organ meats were included as the source of nutrients otherwise difficult to obtain. They assessed the resulting nutrient profiles against Australian and New Zealand Nutrient Reference Values [1]. Phelan et al. (2023) similarly constructed menus for eight popular diets, including the carnivore diet, under the explicit instruction that each menu maximize adherence to the Dietary Guidelines for Americans within the constraints of that diet's own rules [31]. Trained investigators designed both sets of plans, using food composition databases, to achieve the best attainable nutrient profile the diet permits. They are best-case constructions rather than descriptions of practice. Even under those conditions, the carnivore menu constructed by Phelan et al. (2023) received the lowest total Healthy Eating Index score of the eight dietary patterns assessed. It provided the lowest intakes of fiber, vitamin D, calcium, and potassium, the four nutrients identified in the Dietary Guidelines for Americans as underconsumed by the US population [31]. Two features of the available survey data indicate that real-world adherence departs from them in an unfavorable direction. Most individuals following a carnivore diet report consuming organ meat less than once per week [3], whereas the modeled plans that met iron and several micronutrient reference values depended on organ meat inclusion [1]; and supplementation, the remaining route to adequacy, is neither routine among adherents nor encouraged by those promoting the diet: 80% and 47% of survey respondents reported never taking a multivitamin or other vitamin supplement, respectively [3]. The most common position taken on dietary supplements by prominent carnivore diet content creators is that they are unnecessary [27]. Self-reported mean meat intake of 626 g/day [5] also exceeds the quantities on which several modeled plans were based, implying correspondingly higher intakes of saturated fat, cholesterol, and heme iron than the models indicate. Accordingly, the modeled deficiencies described below should be understood as conservative.

3.3 Nutrient Deficiencies and Cardiovascular Consequences

Goedeke et al. (2025) identified several potential micronutrient deficiencies in carnivore diets, particularly those typically obtained from plant-based foods. The carnivore plans provided inadequate levels of vitamins B1 and C, as well as calcium, magnesium, and potassium. Some carnivore plans also had low folate, iodine, and iron, and all carnivore diets were virtually bereft of fiber [1]. These nutritional shortcomings are associated with increased CVD risk. For example, lower thiamin intake has been associated with increased risks of CVD and CVD mortality, especially in individuals with dyslipidemia [44]. Adequate thiamin status may mediate these relationships by influencing cardiac function, hemodynamic characteristics, and systemic vascular resistance [44]. Low dietary magnesium has been associated with increased risk of ischemic heart disease and stroke [45, 46]. Potential mechanisms include magnesium's effects on endothelium-dependent vasodilation and vascular tone, as well as on lipid and glucose metabolism, inflammation, and platelet function and aggregation [45]. Similarly, low dietary vitamin C has been associated with a significantly higher risk of CVD mortality, potentially because vitamin C contributes to nitric oxide production and release, which in turn influences arterial dilation, blood pressure, and endothelial function [47]. Goedeke et al. (2025) [1] speculated that vitamin C requirements may be lower in a carnivore because the body might recycle more vitamin C or use less in the absence of chronic high blood sugar. However, this argument remains hypothetical. The current evidence does not confirm that ketogenic or carnivorous metabolism can "spare" micronutrients to the extent needed to

prevent deficiencies. These findings collectively suggest that the micronutrient and fiber inadequacies common to carnivorous diets may increase the long-term risk of CVD.

The complete absence of dietary fiber warrants separate consideration, both because it is the single nutrient gap that no carnivore diet can close by any adjustment within the diet's own rules, and because the evidence linking fiber intake to cardiovascular outcomes is stronger than that for most individual nutrients. A series of systematic reviews and meta-analyses encompassing 185 prospective studies and 58 clinical trials found that the highest consumers of dietary fiber experienced a 15-30% lower rate of all-cause mortality, cardiovascular mortality, coronary heart disease incidence, and stroke incidence and mortality compared with the lowest consumers, with dose-response curves indicating progressively greater benefit across the observed intake range and the greatest risk reduction at intakes of 25-29 g/day; the certainty of fiber evidence was graded moderate, and the dose-response relationship was considered strong enough to suggest that these associations could be causal [48]. Randomized trials in the same synthesis demonstrated significantly lower body weight, systolic blood pressure, and total cholesterol with higher fiber intakes [48]. Carnivore diets provide none of this intake [1, 31], placing adherents not at the low end of the observed distribution but entirely outside it, at intakes for which no cohort data exist.

Several mechanisms connect this deficit to cardiovascular risk. Viscous soluble fiber binds bile acids in the intestinal lumen and increases their fecal excretion, obliging the liver to synthesize replacement bile acids from cholesterol, upregulating LDL receptor expression, and thereby lowering circulating LDL-cholesterol [49]. This pathway is wholly unavailable on a plant-free diet, and acts in direct opposition to the LDL-raising effect of the saturated fat load described in Section 3.4. Fermentable fiber is also the principal substrate for colonic production of short-chain fatty acids, particularly butyrate and propionate, which maintain colonocyte integrity and epithelial barrier function, attenuate systemic inflammatory signaling, and influence hepatic lipid metabolism [49]. Its removal may reduce short-chain fatty acid-producing bacterial populations, with potential consequences for inflammatory and metabolic regulation; near-complete exclusion of fiber has been linked specifically to loss of gut microbial diversity in carnivore and animal-based diets [10]. Fiber also contributes to glycemic regulation, satiety, and weight management [49]. The cardiovascular significance of the fiber deficit is therefore not reducible to the loss of a single protective mechanism: it removes a mechanism that lowers LDL-cholesterol, one that restrains systemic inflammation, and one that supports weight regulation, while the diet's macronutrient profile places additional load on all three.

An objective dietary quality metric independently confirmed the poor overall nutritional quality of the carnivore diet. Phelan et al. (2023) [31] evaluated eight popular diets using the Healthy Eating Index (HEI)-2015, a 100-point scoring tool based on alignment with Dietary Guidelines for Americans, that correlates with reductions in CVD mortality [50]. Results showed that the carnivore diet received the lowest total HEI score (26.7) of all diets analyzed [31]. This score compares unfavorably not only with several other diets, including the ketogenic diet (77.1) and the plant-based/vegan diet (84.1), but also with the average American HEI score of 59. Consistent with its total exclusion of plant foods, the carnivore diet scored 0.0 on the HEI adequacy subscales for total vegetables, greens, and beans; total fruit; whole fruit; and whole grains. Calcium and potassium intake were also identified as inadequate, and the overall nutritional adequacy of the carnivore diet was characterized as "potentially problematic." Notably, the menus used in this analysis were designed

to maximize adherence to Dietary Guidelines within each diet's rules, suggesting that real-world carnivore diets are likely of even lower dietary quality than the modeled score indicates [31].

3.4 Intakes of Sodium, Saturated Fat, Cholesterol, and Heme Iron

Nutrient analyses of the carnivore diet found other nutrients present in excessive quantities [1, 31]. Goedeke et al. (2025) [1] found that modeled intakes of sodium, saturated fat, and cholesterol significantly exceeded recommended limits. Sample meal plans provided 7,200-8,789 mg/day of sodium, 3-4 times the recommended maximum. This excess partly reflects the inclusion of salty, cured products and the liberal use of salt in meat preparation [1]. This finding is also reflected in objective dietary quality scoring: the carnivore diet received a score of 0.0 out of 10 for the sodium moderation component of the HEI-2015, indicating maximally excessive sodium intake (≥ 2.0 g/1000 kcal) [31]. Excessive sodium intake has been associated with increased CVD risk due to suppression of the renin-angiotensin-aldosterone system and expansion of plasma volume, which elevate cardiac output and blood pressure by increasing cardiac contractility and peripheral resistance [51].

Modeling research has also found that saturated fat levels were excessive in the carnivore diet [1, 31]. Nutrient analyses revealed that saturated fat accounts for 26-32% of total calories [1], levels that significantly exceed recommendations in the United States Dietary Guidelines for Americans 2025-2030, which limit saturated fat to <10% of calories [22]. On the HEI-2015, the carnivore diet also scored 0.0 out of 10 for the saturated fat moderation component, the lowest of all diets evaluated, and in stark contrast to most other popular diets, such as the plant-based (10.0) pattern, which scored substantially higher on this measure [31]. The evidence suggests that high saturated-fat intake increases CVD risk by raising LDL-cholesterol, an established risk factor for atherosclerosis, independent of other risk factors such as body mass index (BMI) and diabetes; this mechanism underlies the lipid findings discussed throughout Section 4 [52-54].

Goedeke et al. (2025) [1] did not calculate cholesterol provisions, but typical carnivore plans include cholesterol-rich foods such as eggs, liver, and shrimp, suggesting these diets are high in cholesterol. Dietary cholesterol has less effect on circulating concentrations than saturated fat in most individuals, but it contributes additively to the LDL elevation described above [55].

Goedeke et al. (2025) [1] modeled that iron intake was below the Recommended Dietary Intake (RDI) in the female meal plan that did not include liver. The female meal plan that included liver, as well as both male meal plans, exceeded their respective RDIs for iron [1]. The proportion of heme iron in animal-sourced foods is typically high and ranges from 33% of total iron in cooked liver to 70% of total iron in cooked beef [56]. High heme iron intake is associated with an increased risk of CVD, independent of potential confounding factors such as saturated fat and cholesterol intake [57, 58]. This association may be mediated by heme iron, which promotes oxidative stress and low-grade inflammation by catalyzing the formation of reactive oxygen species, thereby impairing endothelial function and atherosclerotic plaque development [58]. The abnormally high intake of heme iron that may characterize meat-only diets, even when modeled total iron intake appears modest, may therefore increase the risk of CVD.

3.5 Imbalance of Nutrient Provision and Implications for Cardiovascular Disease Risk

Nutrient analysis of carnivore diet meal plans indicates that these diets provide large amounts of protein and certain micronutrients, but are unbalanced, containing excessive quantities of

atherogenic nutrients and lacking in many components considered essential for CVD prevention [1, 31]. Organ meats have been recommended for nutritional adequacy in carnivore diets [16], but most individuals following a carnivore diet report consuming organ meat less than once per week [3]. Nutrient deficiencies are therefore likely without careful planning and supplementation. However, 80% and 47% of individuals following the carnivore diet reported never taking a multivitamin or other vitamins, respectively [3], and prominent carnivore diet content creators most commonly argue that dietary supplements are unnecessary [27]. Phytonutrients, such as polyphenols, have not been classified as essential nutrients, but evidence indicates that polyphenol intake is associated with reduced CVD risk [59]. The nutrient profile of the carnivore diet may therefore increase long-term CVD risk.

A distinction should be explicitly drawn between theoretical nutrient adequacy and observed intake, as the two are frequently conflated in discussions of this diet. All nutrient data presented in this section derive from modeling: no published study has collected three-day food records or 24-hour dietary recalls from individuals actually following a carnivore diet [1]. The modeling literature, therefore, establishes what the diet can deliver under optimal construction, not what adherents consume. Claims that the carnivore diet is nutritionally adequate rest on the former and are, within their assumptions, defensible for a subset of nutrients; they do not establish the latter. The only intake information available from adherents themselves is self-reported food group frequency and estimated meat quantity from two surveys [3, 5], which indicate infrequent organ meat consumption, minimal supplementation, and high total meat intake. This pattern diverges from the modeled plans at precisely the points on which their adequacy depends. Bridging this gap requires objective dietary assessment in free-living adherents, ideally with biochemical confirmation of nutrient status, and until such data exist, the nutrient adequacy of the carnivore diet as practiced remains unmeasured.

4. Metabolic and Cardiovascular Effects

Before considering individual risk factors, it is important to state the evidentiary status of this section clearly. There have been no randomized controlled trials or prospective cohort studies examining the carnivore diet in relation to any cardiovascular outcomes, whether surrogate or clinical. Consequently, every finding related to the carnivore diet mentioned below is derived from cross-sectional self-reports, uncontrolled case series, or individual case reports. These study designs cannot establish causality, estimate effect size, determine the frequency of any outcomes, or detect harms that may occur outside the observation period or in self-selected samples.

The case reports discussed in this section illustrate potential mechanisms and outline the range of observed outcomes; they are intended to generate hypotheses rather than prove typical effects. When a quantitative estimate is provided, it is sourced either from a single unreplicated source or from indirect research related to ketogenic and low-carbohydrate diets, and this is clearly identified. The lack of prospective or randomized carnivore-specific data is the main limitation of the existing evidence, which constrains all conclusions drawn in this section.

4.1 Glycemic Improvements

The macronutrient profile of carnivore diets also directly affects CVD risk factors such as insulin resistance. The carnivore diet is projected to contain less than 2% of calories from carbohydrates

[1], and low-carbohydrate diets have been shown to decrease blood glucose and insulin levels in overweight or obese patients with T2D [60]. Self-reports from many individuals with diabetes who followed the carnivore diet for six months or more revealed diabetes-related improvements, including a median reduction in A1c of 0.4% (while HbA1c was unchanged in the overall study sample; among those with diabetes, 100% discontinued other injectable medications, 92% of those with T2D discontinued insulin, and 84% discontinued oral diabetes medications) [3]. However, the study did not discuss whether participants discontinued medication use at the physician's direction or independently, without medical supervision. Consistent with these results, additional survey data from a broader cohort (including non-diabetic individuals) following a carnivore diet for a mean duration of 17 months revealed a mean reduction in A1c of -0.1%, which did not reach statistical significance ($p = 0.324$) [5]. Interventional trials are necessary to validate these findings, but preliminary data suggest carnivore diets may improve glycemic control in individuals with diabetes, with no clear benefit in non-diabetic populations.

A case report by Verma (2026) [21] provides further anecdotal evidence of glycemic improvement in a patient with T2D and illustrates the complexity of interpreting outcomes in medically complicated patients. A 58-year-old male with a 20-year history of T2D, hypertension, and Stage 4 Chronic Kidney Disease adopted a carnivore diet consisting of eggs, poultry, mutton, and fish cooked in animal fats or coconut oil. After approximately four months, his A1c decreased from 6.9% to 5.4%, and fasting insulin decreased from 4.21 to 2.48 mIU/L; his average blood pressure also improved to 125/87 mmHg, allowing discontinuation of all oral hypoglycemic and antihypertensive medications. Notably, despite the high animal protein intake, the patient's estimated glomerular filtration rate improved from 28.34 to 40 mL/min/1.73 m², suggesting that at least in this case, renal function was not adversely affected in the short term. Remission was maintained in the context of concurrent metastatic renal cell carcinoma, though the diet did not halt cancer progression, and the patient subsequently died from cancer-related complications [21]. The report did not document body weight before or after the dietary change, and the patient also adopted intermittent fasting, including 24-hour fasts, so the contribution of energy restriction and any accompanying weight loss to these improvements cannot be determined [21]. As a single case report, these findings cannot be generalized, and the long-term safety profile of such a restrictive dietary approach remains unknown.

4.2 Short-Term Weight Loss

Obesity is an established CVD risk factor, and dietary modifications have been recommended to facilitate weight reduction in individuals with obesity [61]. Consistent with research demonstrating weight loss from low-carbohydrate diets [62], many individuals following a carnivore diet also report significant short-term weight loss [3]. In a survey by Lennerz et al. (2021), the median BMI of respondents decreased from 27.2 to 24.3 kg/m² after adopting the carnivore diet, corresponding to a categorization from overweight to normal weight [3]. Evidence suggests that the observed weight loss is primarily attributable to reduced total energy intake, rather than to any unique metabolic advantage of low-carbohydrate intake. A meta-analysis by Naude et al. (2014) [63] found no significant difference in weight loss between low-carbohydrate diets, including high-fat and high-protein variants, and isocaloric balanced diets over periods of 12 months or longer. These findings suggest that caloric restriction, rather than macronutrient composition, is the principal driver of

weight reduction [63]. Consequently, weight loss on the carnivore diet may result from reduced overall energy intake.

4.3 Blood Pressure Reductions

Hypertension is another modifiable risk factor for CVD [64]. Weight loss alone can improve important biometrics, such as blood pressure [65]. Low-carbohydrate diets have therefore been shown to produce modest reductions in systolic (-1.41 mmHg) and diastolic (-1.71 mmHg) blood pressure [66], in line with reductions observed with other methods of energy restriction, increased physical activity, or both [67]. Lennerz et al. (2021) [3] found that 61% of individuals reported resolution of hypertension after adopting a carnivore diet. Mechanisms for these changes include the effects of short-term weight loss on decreasing plasma volume and improving vascular tone [66].

4.4 Elevated Total Cholesterol

The potential benefits should be weighed against significant negative changes in other cardiovascular risk markers. For example, total blood cholesterol is significantly associated with increased risk of death from CVD [68]. A quantitative analysis of metabolic ward studies demonstrated that saturated fat and dietary cholesterol significantly increase total blood cholesterol [69]. Very low-carbohydrate diets are typically high in saturated fat and dietary cholesterol, and have been shown to increase total cholesterol by 56.85 mg/dL [70].

Survey data indicate that total cholesterol changes are more pronounced after transitioning to a carnivore diet. Klement & Matzat (2025) [5] found that median total cholesterol increased from 224 mg/dL to 305 mg/dL on the carnivore diet ($p < 0.0001$). Case reports corroborate that adherence to a carnivore diet is associated with significantly elevated total cholesterol. Marmagkiolis et al. (2025) [13] described a man in his 40s who presented with yellowish nodules on his soles, elbows, and palms. He had been following a carnivore diet for approximately eight months when these xanthomas appeared. His baseline cholesterol levels were 210-300 mg/dL. Following the carnivore diet, his total cholesterol exceeded 1000 mg/dL [13].

Researchers also reported hypercholesterolemia in the early 20th century. Tolstoi (1929) [19] examined blood chemistry in two men who followed a carnivore diet for one year. In one subject (KA), baseline total cholesterol values were not reported, but they ranged from 200 to 800 mg/dL following adherence to the carnivore diet. In the second subject (VS), baseline total cholesterol was 263 mg/dL, and levels fluctuated between 212 and 300 mg/dL while adhering to the carnivore diet [19]. More recently, Norwitz and Soto-Mota (2024) [14] reported total cholesterol levels in two of 10 patients in a case series, with values of 479 mg/dL (Case 2, AN) and 392 mg/dL (Case 4, IA) while following a carnivore diet. Although these references are limited to survey data and case reports, they illustrate the potential for atherogenic lipid profiles to occur following a carnivore diet.

4.5 Elevated Low-Density Lipoprotein Cholesterol

LDL-cholesterol is significantly associated with the risk of CVD mortality [68], and interventions that lower LDL-cholesterol concentrations, whether pharmacological or dietary, consistently reduce CVD risk in a dose-response fashion [71]. As detailed in Section 3.4, saturated fat and dietary cholesterol intakes of this magnitude raise LDL-cholesterol [52, 55]. Researchers have also examined

low-carbohydrate diets, which are typically high in fat, in this context. Soto-Mota et al. (2024) conducted a meta-analysis of randomized controlled trials. They found that low-carbohydrate diets increased LDL-cholesterol by 41 mg/dL in adults with normal body weight, while no such increase was observed in overweight adults [8]. However, this meta-analysis has been subject to criticism for inaccuracies in data extraction, concerns about the choice of covariates, and the absence of comparisons with LDL-cholesterol responses to balanced-carbohydrate diets [9], and the results should therefore be interpreted with caution pending more rigorously designed trials.

Several mechanisms may underlie this BMI-dependent pattern. A recent review by DiMattia and Petersen (2026) synthesized evidence suggesting that individuals with normal BMI have lower hepatic cholesterol synthesis and greater LDL receptor responsiveness to dietary factors, such that a high-fat, low-carbohydrate diet more readily suppresses LDL receptor activity and reduces LDL clearance [72]. A complementary explanation is offered by the lipid energy model proposed by Norwitz and Soto-Mota (2024), which posits that carbohydrate restriction in individuals with low adiposity increases reliance on fat oxidation, prompting enhanced hepatic secretion of very-low-density lipoprotein, which is subsequently converted to LDL through peripheral lipolysis, resulting in elevated LDL-cholesterol and, paradoxically, low triglyceride levels [14]. DiMattia and Petersen (2026) further note that these proposed mechanisms remain incompletely understood and that the relative contributions of saturated fat intake, carbohydrate restriction, and body composition to LDL elevation in this population have not been formally disentangled [72].

Elevated LDL-cholesterol has also been reported in research detailing biochemical changes in adherents of the carnivore diet [3, 5, 14]. Lennerz et al. (2021) [3] found that individuals who followed the carnivore diet for at least six months reported elevated LDL-cholesterol levels, with a median value of 172 mg/dL. This value is approximately 70% higher than the upper end of the healthy range (<100 mg/dL). Klement & Matzat (2025) [5] reported median changes in LDL-cholesterol after transitioning to the carnivore diet, finding an increase from 157 mg/dL to 256 mg/dL ($p = 0.00024$). Norwitz and Soto-Mota (2024) [14] also presented elevated LDL-cholesterol in a case series. LDL-cholesterol levels were reported for three of 10 subjects following the carnivore diet, and values were consistently elevated: 399 mg/dL (Case 2, AN), 268 mg/dL (Case 4, IA), and 521 mg/dL (Case 9, BL). The authors hypothesized that certain individuals are lean mass hyper-responders (LMHR), a term referring to individuals who, when consuming a carbohydrate-restricted diet such as a ketogenic or carnivore diet [14], exhibit a triad of high LDL-cholesterol, high high-density lipoprotein (HDL)-cholesterol, and low triglycerides.

CVD risk in LMHR has recently been questioned, and Soto-Mota et al. (2025) [73] conducted the Keto-CTA study, a prospective cohort study to assess atherosclerotic plaque progression in 100 individuals with the LMHR phenotype who were following a ketogenic diet. Their median LDL-cholesterol concentration was 237 mg/dL, and their absolute increase in non-calcified plaque volume (NCPV) over one year was 18.9 mm³, which is approximately 3.8 times greater than the annualized NCPV progression in healthy individuals [73-76]. On the contrary, the Dietary Approaches to Stop Hypertension diet has been shown to result in regression of NCPV [77]. However, the journal retracted the Keto-CTA study approximately 11 months after publication due to methodological concerns [73].

Given the lack of robust data on plaque progression or cardiovascular outcomes in this population, the extensive lipid literature must be used to inform any assessment of cardiovascular risk in this cohort. The causal relationship between elevated LDL-cholesterol and atherosclerotic

cardiovascular disease is among the most strongly supported associations in medicine, validated by decades of epidemiological, genetic, and interventional research [71, 78, 79]. Evidence indicating that diet-induced increases in LDL-cholesterol are either benign or protective remains limited and contested [71, 78, 79]. Individuals experiencing substantial elevations in LDL-cholesterol in response to a ketogenic or carnivorous diet may face a significantly increased risk of CVD compared to those without such elevations, a finding of concern from both public health and clinical perspectives, given the magnitude of LDL increases consistently observed in this research [53, 54, 72]. DiMattia and Petersen (2026) further emphasize that the absence of long-term cardiovascular outcome data specific to this phenotype should not be interpreted as evidence of safety, given the extensive evidence base supporting LDL-cholesterol as a causal driver of atherosclerosis [72].

From a clinical management perspective, regular lipid monitoring is recommended in individuals following ketogenic or low-carbohydrate diets. DiMattia and Petersen (2026) [72] note that moderate carbohydrate reintroduction (50-100 g/d) may substantially reverse diet-induced LDL-cholesterol elevations, with pharmacologic therapy providing additional lowering where needed. Furthermore, coronary artery calcium scoring may be useful in asymptomatic patients with marked LDL-cholesterol elevation to help guide individualized risk discussions, a practical framework that clinicians managing patients on carnivore diets may find applicable [72].

4.6 Effects on High-Density Lipoprotein Cholesterol and Triglycerides

Additional CVD risk factors include low HDL-cholesterol and high triglyceride levels [80]. Improvements in one or both of these biomarkers have been reported in individuals following a carnivore diet [3, 5]. Low-carbohydrate diets have been associated with increases in plasma HDL-cholesterol by 3.9 mg/dL and decreases in triglyceride levels by 13.4 mg/dL [66]. Similarly, Lennerz et al. (2021) [3] observed that participants following the carnivore diet reported high HDL cholesterol (median 68 mg/dL) and low triglyceride levels. Klement & Matzat (2025) [5] found that HDL-cholesterol levels were within the normal range before and after transitioning to a carnivore diet. These concentrations increased from 64 mg/dL to 78 mg/dL following this dietary change ($p = 0.0404$). However, triglyceride changes were not statistically significant [5]. Additional anecdotal data were presented by Norwitz and Soto-Mota (2024) [14] in a case series of 10 patients with severe inflammatory bowel disease (IBD) who were treated with a carnivore diet. HDL-cholesterol values were reported for three patients: 70 mg/dL (Case 2, AN), 91 mg/dL (Case 4, IA), and 125 mg/dL (Case 9, BL) while following a carnivore diet. Triglyceride values were also presented in three patients, with values of 83 mg/dL (Case 2, AN), 51 mg/dL (Case 4, IA), and 49 mg/dL (Case 9, BL) while following a carnivore diet. Each of these values, both for HDL-cholesterol and triglycerides, is within the normal range. However, comparison with values prior to the dietary change was not included, which limits interpretation of the carnivore diet's effect on this variable [14].

Furthermore, although HDL-cholesterol is a useful biomarker for predicting CVD risk, the inverse association between HDL-cholesterol and CVD risk does not appear to be causal [81, 82], limiting its relevance as a therapeutic endpoint. This contrasts with the well-established causal role of LDL-cholesterol in atherosclerosis [54, 78] and suggests that diet-induced increases in HDL-cholesterol cannot be interpreted as independently cardioprotective [82]. Nonetheless, increased HDL-cholesterol is commonly associated with low-carbohydrate diets and is attributed to a higher intake of unsaturated fats, which have been shown to raise HDL levels [83]. The concomitant reduction in

triglycerides is thought to result from a lower intake of refined carbohydrates, which reduces carbohydrate-induced hypertriglyceridemia by limiting hepatic *de novo* lipogenesis and insulin secretion [83]. However, triglyceride reductions of similar magnitude are also observed with other weight-loss methods, suggesting this effect may be largely attributable to energy restriction rather than to carbohydrate restriction specifically [84].

However, research on carnivore and ketogenic diets reporting triglyceride improvements has primarily involved individuals without significant underlying metabolic dysfunction. The generally observed pattern of low triglycerides on carbohydrate-restricted diets may not persist in individuals with pre-existing impairment of triglyceride clearance. A recent case report by Squire and Ghosh (2026) [29] illustrates this distinction. A 61-year-old man with a BMI of 43.7 kg/m², T2D, hypothyroidism, and dyslipidemia transitioned from a ketogenic diet to a carnivore diet over two years. He presented to the emergency department with acute pancreatitis and was found to have triglyceride levels exceeding 4,425 mg/dL, a life-threatening elevation. The authors attributed this to multifactorial chylomicronemia precipitated by the carnivore diet's high saturated fat load in the context of concurrent insulin resistance and hypothyroidism. Obesity, hyperglycemia, and insulin resistance elevate triglycerides by increasing hepatic very-low-density lipoprotein synthesis. At the same time, hypothyroidism contributes as an acquired cause of impaired chylomicron clearance, compounded by the high saturated fat load of the carnivore diet [29]. This case illustrates that the lipid effects of carnivore diets are highly context-dependent: while triglyceride reductions are commonly observed in metabolically healthy individuals, those with pre-existing impairment of triglyceride metabolism may face a substantially elevated risk of severe, acute hypertriglyceridemia and its complications [29].

4.7 Effects on Inflammatory Markers

Inflammatory markers are recognized as independent contributors to cardiovascular risk, with C-reactive protein (CRP) serving as a widely utilized biomarker of systemic inflammation that correlates with increased cardiovascular risk [85]. In a survey conducted by Lennerz et al. (2021) [3], 73 participants provided matched CRP measurements before and after adopting a carnivore diet. The median CRP level decreased from 1.0 mg/dL (interquartile range [IQR]: 0.3-3.3) to 0.7 mg/dL [reported in mg/dL per the original publication; equivalent to 10.0 and 7.0 mg/L, respectively, in standard clinical units, indicating pre-diet values consistent with low-grade systemic inflammation] (IQR: 0.3-1.5) ($p < 0.01$) [2]. Similarly, γ -glutamyltransferase, an indicator of liver stress, oxidative burden, and inflammation that is also associated with cardiovascular risk [86], declined from a median of 18 to 15 U/L ($p < 0.01$) [3]. A proposed mechanism that may modulate these anti-inflammatory effects involves β -hydroxybutyrate directly inhibiting the NLRP3 inflammasome, a key mediator of pro-inflammatory cytokine signaling, and the elimination of pro-inflammatory simple sugars further reduces systemic inflammation [2].

These reductions in inflammatory and hepatic stress markers are potentially favorable. However, they must be interpreted with caution: the observed decline in CRP may partly reflect weight loss or removal of ultra-processed foods [87, 88] rather than any specific benefit of animal-based eating, and the magnitude of change (from approximately 1.0 to 0.7 mg/dL in conventional units) represents a modest shift with uncertain clinical significance. Furthermore, the self-reported nature of these data, the absence of a control group, and the selection bias inherent to social media surveys

limit any causal interpretation. Other carnivore diet studies have not reported changes in inflammatory markers, and longer-term controlled trials are needed to determine whether reductions in inflammatory markers are sustained and translate into reduced cardiovascular event rates.

4.8 Overall Assessment of Cardiometabolic Tradeoffs

The reported effects of the carnivore diet do not point in a single direction, and their inconsistency is the central finding of this section. On one side, adherents report, and limited self-reported laboratory data appear to corroborate, weight loss, improved glycemic control, reduced triglycerides, increased HDL-cholesterol, and reduced C-reactive protein [3, 5, 21]. On the other hand, the same datasets document elevated total and LDL-cholesterol levels, the latter of which is the exposure for which a causal relationship to atherosclerotic cardiovascular disease is established beyond reasonable doubt and whose lowering reduces events in a dose-dependent fashion [54, 68, 71, 78]. These observations conflict directly, and four factors shape how we should interpret that conflict.

First, the favorable changes are largely attributable to co-interventions rather than to animal-food consumption: weight loss, removal of ultra-processed foods, added sugars, and refined grains, and reduced total energy intake each independently produce improvements in glycemia, triglycerides, blood pressure, and C-reactive protein [63, 66, 87, 88], and all occur on transition to a carnivore diet. A dietary pattern that retained plant foods while achieving the same energy deficit would be expected to produce the same benefits without the LDL elevation.

Second, the two sets of findings are not equivalent in evidence. The favorable changes are reported for biomarkers whose relationship to cardiovascular events is, in several cases, associational but unlikely to be causal: Mendelian randomization and intervention data indicate that raising HDL-cholesterol does not by itself reduce cardiovascular risk [81, 82], and triglyceride reductions of comparable magnitude follow from energy restriction by any means [84]. By contrast, the unfavorable change is in LDL-cholesterol, the only biomarker for which causality has been established.

Third, the two effects operate over different timescales. Glycemic and weight changes are observed within weeks to months and are reported by adherents on that horizon [3, 21]; atherosclerosis accrues over years to decades. The cumulative exposure to LDL-cholesterol concentrations of the magnitude reported, a median of 172 mg/dL in one survey [3] and individual values of 268-521 mg/dL in a case series [14], would not be expected to manifest clinically within the median 14-month adherence period these datasets capture. The absence of reported events in a 14-month window is therefore uninformative about risk and should not be read as reassurance.

Fourth, when objective intermediate measures of atherosclerosis have been reported, they do not align with subjective reports. Coronary artery calcium scores increased from a median of 55 to 81 among the 15 survey participants who reported paired values [3]. This signal is weak because the sample size is far too small for inference, and calcium score progression can, in some contexts, reflect stabilization of previously non-calcified plaque rather than disease progression [89, 90]. It is nonetheless the only objective vascular measurement in the carnivore literature, and it points away from the direction implied by the subjective reports. The resolution of this inconsistency is not that one set of findings is false; rather, short-term symptomatic and metabolic improvement and long-

term atherosclerotic risk are distinct outcomes that can move in opposite directions simultaneously, and the available evidence measures the first with far greater sensitivity than the second. Long-term cardiovascular safety cannot be inferred from short-term metabolic improvement, and in the presence of sustained, substantial LDL-cholesterol elevation, it cannot be assumed without controlled, prospective data.

5. Reported Outcomes and Clinical Findings

5.1 Limited Empirical Evidence and Methodological Challenges

Research on health outcomes for people following the carnivore diet is limited. No interventional trials have tested the carnivore diet for effects on intermediate cardiovascular outcomes such as plaque progression or arterial function, or cardiovascular events such as heart attacks or strokes. Self-reported health changes from surveys and case reports that focus on specific conditions are weaker forms of evidence that may be used to hypothesize regarding potential long-term health outcomes. However, these reports have inherent limitations. Their findings must be considered in conjunction with numerous caveats, including the absence of control groups and selection, reporting, and recall biases.

5.2 Reported Outcomes from Online Surveys

A recent study by Klement & Matzat (2025) [5] explored the subjective experiences of 24 individuals from Germany who had followed a self-conceived carnivore diet for at least one month. The survey used a self-designed questionnaire and qualitative interviews with four participants, to gather deeper insights. Most respondents reported subjective improvements in their chronic diseases, overall health, hunger, sleep, mental clarity, focus, memory, strength, endurance, and energy. However, the authors noted that these data were self-reported and not verified by an external examiner such as a physician, which limits internal validity, including selection bias (individuals who self-select carnivore diets may differ systematically from the general population) and healthy user bias [5].

Lennerz et al. (2021) [3] published the largest dataset on self-reported health outcomes and adherence to the carnivore diet. They collected retrospective data from 2,029 adults who self-reported following a carnivore diet for at least 6 months. Respondents reported subjective improvements across various health domains. In addition to weight loss and diabetes-related improvements, a high percentage of participants reported improvements in chronic conditions, including obesity or being overweight (93%) and diabetes or insulin resistance (98%), since adopting and adhering to a carnivore diet. Participants self-rated overall well-being as improved by 66-91% and energy by approximately 80%. Adverse symptoms were reported at low prevalence (ranging from <1% to 5.5%) and mainly consisted of transient gastrointestinal issues, such as constipation (3.1%) and diarrhea (5.5%), as well as occasional muscle cramps (4.0%) in a small percentage of participants. Classic deficiency diseases such as scurvy or beriberi were not reported, although it is unclear whether these diseases were specifically surveyed. High levels of overall satisfaction were reported, and the median time following a carnivore diet was 14 months, suggesting that many found the diet sustainable over the intermediate term [3]. These findings suggest that the carnivore diet is subjectively well tolerated for at least 6 months in motivated individuals and may be

associated with various improvements, likely related to weight loss and the removal of ultra-processed foods from the diet.

The study by Lennerz et al. (2021) [3] had significant limitations, primarily because it relied on self-reported data from a social media survey. Such survey data are prone to selection bias because individuals who experienced adverse health outcomes or did not benefit from the carnivore diet may have been less inclined to participate. Self-reported improvements in health status and various conditions are also difficult to verify without objective measurements or a control group for comparison. These perceived benefits may therefore reflect the placebo effect or concurrent lifestyle changes. The survey included self-reported laboratory data from a subset of participants, which confirmed markedly elevated LDL-cholesterol levels. Median coronary artery calcium (CAC) scores also increased from 55 to 81 in the 15 participants who reported both pre- and post-diet measures. This observation should be interpreted with considerable caution, for the reasons set out in Section 4.8. Nonetheless, this signal warrants attention and prospective investigation in larger, controlled studies. However, the survey did not systematically assess key CVD indicators, including blood pressure, inflammatory markers, or comprehensive measures of vascular health. Additionally, reporting and recall bias, along with the influence of online echo chambers, further complicates interpretation, given that participants were mainly recruited through pro-carnivore social media pages [3, 28].

5.3 Early Case Studies

Early-twentieth-century case reports also reported health outcomes associated with adherence to the carnivore diet [18, 19]. Two healthy, physically active adult male volunteers, both experienced Arctic researchers participating in a closely monitored inpatient and outpatient study, adhered to a carnivore diet for one year and reported no apparent loss of physical or mental vigor, maintaining their usual activities without increased fatigue. Initial weight loss was observed in both subjects during the first week, after which body weight stabilized for the remainder of the year. Blood pressure was unchanged in one participant, and systolic blood pressure decreased in the other participant. No clinical evidence of vitamin deficiency was noted.

McClellan and Du Bois (1930) similarly reported no clinical evidence of vitamin deficiency or renal impairment in two subjects monitored in a metabolic ward over 31-110 days as part of their year-long experiment [17]. Tolstoi (1929) [18] conducted glucose tolerance tests immediately after the meat diet was discontinued, which demonstrated a marked reduction in carbohydrate tolerance in both subjects, with one exhibiting glycosuria. However, both subjects showed normal responses to the glucose tolerance tests after two to four weeks on a mixed diet [18].

These glucose tolerance findings, glycosuria and prolonged hyperglycemia in response to carbohydrate reintroduction after a year of carnivore eating, suggest meaningful impairment of insulin sensitivity. Importantly, these experiments were conducted under tightly controlled research conditions in two men, and their findings may not generalize to broader or more vulnerable populations [17-19].

5.4 Summary and Future Directions

Table 3 provides an overview of key findings from available studies and reports on carnivore diets, which highlight this duality of effects. Case reports and survey data provide intriguing, albeit limited,

insights into the relationship between adherence to a carnivore diet and CVD risk. However, these research designs are not equipped to establish causality or investigate long-term safety issues. The potential benefits should be evaluated alongside considerable methodological limitations and evidence of harm. High-quality, prospective research is needed to determine whether the carnivore diet can support sustained health or whether its restrictive nature and associated risks outweigh its short-term benefits.

Table 3 Overview of findings from original research into the relationship between the carnivore diet and measures of health.

Study Design	Population	Methods	Reported Improvements	Reported Adverse Effects
Dietary Modeling and Nutrient Analysis				
Dietary Modeling and Nutrient Analysis [1]	Hypothetical average Australian adults (2 female, 2 male).	Developed 4 meal plans (dairy vs. offal) for a single day. Analyzed nutrient adequacy against Australian and New Zealand NRVs.	Met NRVs for riboflavin, niacin, phosphorus, zinc, Vitamin B6, Vitamin B12, selenium, Vitamin A; exceeded sodium. Anecdotal benefits for inflammation mentioned.	Inadequate fiber, thiamin, vitamin C, calcium, magnesium, potassium; Inconsistent in meeting iron, folate, iodine requirements. Potential long-term micronutrient deficiencies.
Dietary Modeling and HEI Analysis [31]	Hypothetical meal plans for 8 popular diets (including carnivore), designed to maximize DGA compliance within each diet's rules.	Scored each diet on the HEI-2015 (0-100). Assessed micronutrient adequacy against RDAs. Average energy intake calculated per diet.	Most RDAs were met for animal-derived nutrients (e.g., protein) and for the majority of nutrients in the seven diets. Near-zero added sugars and refined grains (consistent with diet rules).	Lowest HEI-2015 score of all diets analyzed (26.7/100; average American score: 59). Scored 0.0/5 for vegetables and fruit components and 0.0/10 for whole grains, saturated fat, and sodium components. Calcium and potassium inadequate. Fiber absent. Nutritional adequacy rated "potentially problematic." Lowest average energy intake (1,302 kcal/day).
Surveys				
Survey [3]	2029 adults self-identifying as following a carnivore diet for ≥6 months.	Social media survey on dietary intake, health status, symptoms.	High satisfaction (95% improved overall health). Improved well-being (66-91%), improvements in various medical conditions (48-98%). BMI decreased (27.2 → 24.3 kg/m ² , p < 0.01). CRP decreased (1.0 → 0.7 mg/dL, p < 0.01; n = 73 pairs). HDL-	Low prevalence of adverse symptoms (<5.5%), including GI, muscular, and dermatologic. Lipid abnormalities variably affected (27% new/worsening). Markedly elevated LDL-cholesterol (median 172 mg/dL; pre-diet: 126 mg/dL, p < 0.01) and TC (median 256 mg/dL; pre-diet: 209

			cholesterol increased (58 → 68 mg/dL, $p < 0.01$); TG decreased (83 → 68 mg/dL, $p < 0.01$). In the T2D subset: A1c reduced (-0.4%), 84% discontinued oral diabetes medications, 92% of T2DM participants discontinued insulin.	mg/dL, $p < 0.01$). GGT decreased (18 → 15 U/L, $p < 0.01$). Coronary artery calcium score increased (55 → 81) in subset reporting paired values.
Survey [5]	24 adults self-identifying as following carnivore diet for ≥ 1 month.	Survey including a self-designed questionnaire on health status, chronic disease, and lifestyle changes; qualitative interviews with four participants; collection of self-reported biochemical blood parameters.	Subjective improvements in the majority of respondents in chronic diseases, overall health, hunger regulation, sleep quality, mental clarity, focus, memory, strength, endurance, and energy.	Total and LDL-cholesterol significantly increased. Reductions in subjective ratings of strength and endurance in the minority of respondents.
Case Series and Reports				
Case Series [17]	Two healthy adult men followed a carnivore diet for 31-110 days.	Monitored clinical condition, urine, kidney function, and fat metabolism.	No clinical vitamin deficiency. Kidney function normal.	Transient intestinal issues in one subject.
Case series [14]	10 patients with histologically confirmed IBD (UC, Crohn's).	Retrospective analysis of patients adopting carnivore-ketogenic diet. Analyzed medical records, labs, 48-h food logs, IBDQ-32 scores (pre & on diet).	Remission of IBD in all 10 patients; symptoms returned upon diet deviation. Improved IBDQ-32 scores.	Not reported.
Case series [18]	Two healthy adult men followed a carnivore diet for one year.	Two glucose tolerance tests per subject: after the carnivore diet, and after 2-4 weeks of a general diet. Monitored blood sugar and urine.	Normal glucose tolerance after resuming mixed diet.	Marked blood sugar rise and prolonged hyperglycemia post-meat diet. One subject had glycosuria. Lowered carbohydrate tolerance.

Case Series [19]	Two healthy adult men.	Carnivore diet for one year. Monthly blood sample analysis for various chemical constituents.	Blood composition slightly affected, mostly within normal range. No changes suggesting renal damage.	Plasma lipemia, high cholesterol soon after starting. One subject had elevated non-protein nitrogen pre-diet.
Case Report [21]	58-year-old male with 20-year T2D history, hypertension, and Stage 4 CKD.	Carnivore diet (eggs, poultry, mutton, fish, animal fats/coconut oil). Clinical and laboratory monitoring over ~4 months.	T2D and hypertension remission (~4 months); all medications discontinued. A1c: 6.9% → 5.4%. Fasting insulin: 4.21 → 2.48 mIU/L. eGFR improved: 28.34 → 40 mL/min/1.73 m ² despite Stage 4 CKD.	Patient died from metastatic renal cell carcinoma complications; cancer did not respond to diet.
Case Report [29]	61-year-old man with T2D, hypothyroidism, obesity, and dyslipidemia.	Transitioned from ketogenic to carnivore diet over 2 years. Emergency presentation; clinical and laboratory monitoring during hospitalization and follow-up.	Weight loss of 40 kg over 2 years. Glycemic improvement (A1c 7.9% at presentation without diabetes medication).	Acute pancreatitis. Severe hypertriglyceridemia (>4,425 mg/dL). Hepatomegaly and severe hepatic steatosis. Multifactorial chylomicronemia attributed to carnivore diet in context of insulin resistance and hypothyroidism.

Abbreviations: A1c, hemoglobin A1c; BMI, body mass index; CKD, chronic kidney disease; CRP, C-reactive protein; DGA, Dietary Guidelines for Americans; eGFR, estimated glomerular filtration rate; GGT, γ -glutamyltransferase; GI, gastrointestinal; HEI, Healthy Eating Index; IBD, inflammatory bowel disease; IBDQ-32, Inflammatory Bowel Disease Questionnaire-32; LDL, low-density lipoprotein; NRVs, Nutrient Reference Values; RDA, Recommended Dietary Allowance; T2D, type 2 diabetes; TC, total cholesterol; TG, triglyceride; UC, ulcerative colitis.

The overall quality of currently available evidence has been formally characterized as very limited. A scoping review by Lietz et al. (2026) categorized most carnivore diet studies as levels III-IV on the National Health and Medical Research Council evidence hierarchy, citing small sample sizes, short study durations, absence of control groups, and heavy reliance on self-reported social media surveys as primary weaknesses [11]. This assessment is consistent with Waśkiewicz's (2026) observation that no RCTs directly examine carnivore diets in athletes, and that most conclusions about performance contexts are extrapolated from distinct but related ketogenic or low-carbohydrate models [10]. The majority of available trials are short (weeks), precluding assessment of long-term health, recovery, or cumulative training adaptations [10]. From a sports and performance perspective, Waśkiewicz (2026) [10] noted that while the carnivore diet markedly increases fatty acid oxidation capacity, this adaptation does not consistently translate into improved functional performance, as fat oxidation is more oxygen-costly and slower in ATP resynthesis than carbohydrate oxidation, with muscle glycogen remaining a primary limiting factor for high-intensity activity. Very high protein intakes (often >2.5 g/kg/day) support lean mass maintenance but increase reliance on gluconeogenesis, and eliminating plant foods reduces populations of short-chain fatty acid (SCFA)-producing bacteria, potentially compromising gut integrity and inflammatory regulation. Extremely restrictive diets also carry a risk of promoting disordered eating behaviors in competitive athletic populations [10].

6. Alignment with Dietary Guidelines and Broader Evidence

The carnivore diet diverges markedly from evidence-based dietary patterns endorsed by leading health authorities, including the American Heart Association, World Health Organization, and the United States Department of Agriculture. These institutions consistently recommend diets rich in vegetables, fruits, whole grains, legumes, and nuts, while limiting intake of saturated fat, sodium, and red and processed meats [22-24]. Clinical practice guidelines from 2010 to 2021, issued by major health organizations and medical societies, consistently align regarding cardioprotective dietary recommendations. These recommendations commonly include increasing plant food intake, such as legumes, nuts, and seeds [91]. In contrast, the carnivore diet eliminates all plant-based food groups. It relies heavily on red and processed meats, resulting in high intakes of saturated fat and sodium, both of which are established risk factors for CVD [51, 92]. Recent large-scale analyses found that greater plant-based food intake was associated with lower CVD and mortality risks, whereas increased intake of red and processed meats was associated with elevated cardiovascular risk [93, 94]. Additional meta-analytic data indicate that replacing animal-based foods, including red and processed meat, poultry, and eggs, with plant-based foods, such as nuts, legumes, and whole grains, was positively associated with cardiometabolic benefits and reduced mortality risk [95].

Ethnographic examples, such as the traditional diets of the Maasai and Inuit populations, have often been cited to support claims that carnivore diets can promote cardiovascular health [4, 25, 26, 33-36]. However, these references lack contextual validity in modern settings. For example, the Maasai diet was not exclusively carnivorous and was consumed amid high physical activity, chronic caloric restriction, and shorter life expectancy [32]. Moreover, paleopathological evidence suggests that atherosclerosis was still present in pre-modern populations, despite lower rates of diagnosis of lifestyle-related diseases [96]. The applicability of these ancestral patterns to contemporary, sedentary, high-calorie environments is therefore highly questionable. No randomized controlled

trials or longitudinal cohort studies support the exclusion of all plant foods as a sustainable or cardioprotective strategy.

A vast and growing body of literature supports the benefits of diets that promote the inclusion of whole plant foods for the prevention of CVD [97-100]. Potential harms from nutrient imbalances and hyperlipidemia, along with lack of long-term safety data, underscore the misalignment between the carnivore diet and current public health and clinical guidelines.

6.1 Contrast with Established Cardioprotective Dietary Patterns

The divergence between the carnivore diet and guideline-endorsed patterns is most instructive when set against the two dietary patterns for which cardiovascular benefit has been demonstrated in randomized trials rather than inferred from observational data. The contrast is informative on three levels: the strength of the evidence supporting each pattern, the direction of their compositional differences, and the mechanisms through which each is understood to act.

On evidence, the asymmetry is categorical. Researchers have tested the Mediterranean pattern against hard cardiovascular endpoints in two landmark randomized trials. In the Lyon Diet Heart Study, patients randomized after a first myocardial infarction to a Mediterranean-type diet experienced 14 cardiac deaths and non-fatal myocardial infarctions versus 44 in the prudent Western-diet control group over a mean of 46 months ($P = 0.0001$), with adjusted risk ratios across composite endpoints ranging from 0.28 to 0.53 [101]. In the PREDIMED trial (Prevencion con Dieta Mediterranea), 7,447 participants at high cardiovascular risk but without prevalent disease were randomized to a Mediterranean diet supplemented with extra-virgin olive oil, the same diet supplemented with mixed nuts, or a reduced-fat control diet; after a median 4.8 years, hazard ratios for major cardiovascular events were 0.69 (95% CI 0.53-0.91) and 0.72 (95% CI 0.54-0.95) respectively [102]. The Dietary Approaches to Stop Hypertension (DASH) pattern was likewise established in a controlled feeding trial: among 459 adults, the combination diet rich in fruits, vegetables, and low-fat dairy and reduced in saturated and total fat lowered systolic and diastolic blood pressure by 5.5 and 3.0 mmHg more than the control diet, and by 11.4 and 5.5 mmHg more among the 133 participants with hypertension [103]; dose-response meta-analysis of 17 prospective cohort analyses across 13 publications subsequently found each 5-point increment in DASH adherence associated with hazard ratios of 0.95 for all-cause, 0.96 for cardiovascular, and 0.97 for cancer mortality [104]. In contrast, the carnivore diet has not been studied in any randomized trial, prospective cohort study, or controlled feeding study of any duration or for any cardiovascular endpoint.

In terms of composition, the differences are not just variations in degree; they represent differences in direction. Both Mediterranean and DASH patterns are built on the food groups the carnivore diet eliminates, namely vegetables, fruits, legumes, whole grains, nuts, and seeds, and both explicitly limit the foods on which the carnivore diet is built, namely red meat, saturated fat, and, in the case of DASH, sodium [22-24, 91]. On every axis along which these patterns are defined, the carnivore diet lies at the opposite pole: fiber intake of approximately zero against the 25-29 g/day at which cardiovascular risk reduction is maximal [48]; saturated fat at 26-32% of energy [1] against the under 10% these patterns target; modeled sodium intakes of 7,200-8,789 mg/day [1] against the 1,500-2,300 mg/day recommended by current guidelines [22, 23]; and no provision of

the polyphenols, unsaturated fats from plant sources, or phytochemicals through which both patterns are understood to act [37-39, 59].

Understanding the mechanism matters because the components understood to confer the benefits of Mediterranean and DASH dietary patterns are specifically those that a plant-free diet cannot provide. While the Mediterranean pattern substitutes unsaturated for saturated fat and DASH substitutes potassium- and fiber-rich plant foods for sodium-dense processed foods, the carnivore diet reverses both substitutions. Meta-analytic data indicate that replacing animal-based foods, including red and processed meat, poultry, and eggs, with plant-based foods such as nuts, legumes, and whole grains is associated with cardiometabolic benefit and reduced mortality [95]; the carnivore diet represents the maximal expression of the opposite exchange. The comparison, therefore, does not merely show that the carnivore diet is unsupported, but that Mediterranean and DASH patterns are supported. It shows that the carnivore diet is constructed by inverting the specific compositional features attributed to the demonstrated benefits of those patterns. The same contrast holds in the same direction for predominantly plant-based patterns, although the evidence there is observational rather than trial-based: greater plant-based food intake is associated with lower cardiovascular disease and mortality risk, and replacing animal-based foods with plant-based foods is associated with cardiometabolic benefit [93, 95, 97-99]. No trial has compared a plant-rich pattern directly with a carnivore diet, and the inference that such patterns are more supportive of cardiovascular health does not depend on one: it rests on the trial evidence for those patterns themselves, and on the fact that the carnivore diet is defined by the inversion of the compositional and mechanistic features to which their benefit is attributed.

7. Total Mortality and Cancer Outcomes

7.1 Rationale for Considering Total Mortality

Evaluating a dietary pattern solely against cardiovascular endpoints is incomplete. The objective of dietary prevention is to extend survival and healthy life, and any dietary change alters exposure to multiple disease processes simultaneously; a pattern that reduces cardiovascular mortality while increasing mortality from cancer or other causes would not extend life and could shorten it. Total mortality integrates these competing effects and, for that reason, is the endpoint against which dietary patterns are ultimately judged [23, 24]. This consideration is directly relevant to the carnivore diet, which primarily claims benefits such as diabetes remission, weight loss, and resolution of inflammatory conditions. These benefits are specific to certain diseases. However, several key characteristics of the diet, especially the high consumption of red and processed meats and the complete lack of fiber and plant foods, may increase cancer risk and affect overall mortality, independent of cardiovascular effects. The case report by Verma (2026) discussed in Section 4.1 illustrates the distinction concretely: the patient achieved sustained remission of T2D and hypertension and improvement in renal function, and died from complications of metastatic renal cell carcinoma that the diet did not arrest [21]. Disease-specific improvement and survival are not interchangeable outcomes.

7.2 Available Evidence

No study has examined total mortality, cancer incidence, or cancer mortality in individuals following a carnivore diet. The available surveys and case series are neither designed nor powered for mortality endpoints. The longest documented adherence in the published literature, one year in the 1920s metabolic ward studies [17-19] and a median of 14 months in the largest survey [3], is short relative to the latency of the outcomes in question. The following information is therefore based entirely on indirect evidence about the exposures that are maximized or eliminated by the carnivore diet and is subject to the interpretive constraints outlined in Section 7.3.

Cohort studies of low-carbohydrate diet scores have consistently found that the relationship with mortality depends on the food sources replacing carbohydrates. In pooled analysis of the Nurses' Health Study and the Health Professionals Follow-up Study (85,168 women and 44,548 men; 21,233 deaths), an animal-based low-carbohydrate score was associated with higher all-cause mortality (hazard ratio comparing extreme deciles 1.23, 95% CI 1.11-1.37), cardiovascular mortality (HR 1.14, 95% CI 1.01-1.29), and cancer mortality (HR 1.28, 95% CI 1.02-1.60), whereas a vegetable-based low-carbohydrate score was associated with lower all-cause (HR 0.80, 95% CI 0.75-0.85) and cardiovascular mortality (HR 0.77, 95% CI 0.68-0.87) [105]. The Atherosclerosis Risk in Communities cohort and an accompanying meta-analysis of 432,179 participants found a U-shaped relationship between carbohydrate intake and mortality, with lowest risk at 50-55% of energy from carbohydrate and elevated risk below 40% (pooled HR 1.20, 95% CI 1.09-1.32); crucially, mortality increased when carbohydrate was exchanged for animal-derived fat or protein (HR 1.18, 95% CI 1.08-1.29) and decreased when the substitution was plant-based (HR 0.82, 95% CI 0.78-0.87) [106]. In National Health and Nutrition Examination Survey participants followed for a mean 6.4 years, those in the lowest-carbohydrate quartile had the highest risk of overall (32%), cardiovascular (50%), cerebrovascular (51%), and cancer (36%) mortality; pooled analysis of nine prospective cohorts comprising 462,934 participants in the same report found higher all-cause (RR 1.22, 95% CI 1.06-1.39), cardiovascular (RR 1.13, 95% CI 1.02-1.24), and cancer mortality (RR 1.08, 95% CI 1.01-1.14) with greater low-carbohydrate adherence [107], and an earlier meta-analysis of four cohorts (272,216 participants) similarly found elevated all-cause mortality with high low-carbohydrate scores (RR 1.31, 95% CI 1.07-1.59) [108]. The same source-dependence has been observed when carbohydrate-restricted patterns are classified by quality rather than by animal or plant origin: unhealthy low-carbohydrate scores were associated with higher total mortality (HR 1.07, 95% CI 1.02-1.11 per 20-percentile increment) and healthy scores with lower total mortality (HR 0.91, 95% CI 0.87-0.95) in 37,233 US adults [109], and among 10,101 adults with incident T2D, vegetable- and healthy-emphasis low-carbohydrate scores were associated with significantly lower total, cardiovascular, and cancer mortality while animal- and unhealthy-emphasis scores showed no such association [110]. The carnivore diet is, by construction, the extreme case of the animal-source pattern in every one of these analyses.

The International Agency for Research on Cancer classifies processed meat as carcinogenic to humans (Group 1) and red meat as probably carcinogenic to humans (Group 2A), based on evidence for colorectal cancer [111]. A systematic review and meta-analysis of 148 prospective publications found red meat consumption associated with higher incidence of colorectal cancer (RR 1.10, 95% CI 1.03-1.17) and of colon, rectal, breast, endometrial, lung, and hepatocellular cancer, and processed meat associated with an 18% greater colorectal cancer risk and with higher incidence of colon, rectal,

breast, and lung cancer [112]. For total mortality, the NIH-AARP Diet and Health Study followed 536,969 adults for 16 years. They found red meat intake in the highest versus lowest fifth associated with all-cause mortality (HR 1.26, 95% CI 1.23-1.29) and with death from nine separate causes; heme iron and processed meat nitrate/nitrite were each independently associated with increased all-cause and cause-specific mortality, with mediation analysis attributing 37.0-72.0% of the excess mortality associated with processed red meat to nitrate and 20.9-24.1% to heme iron [113]. Pooled individual-level analysis of six US cohorts (29,682 participants) found each additional two servings per week of processed meat (HR 1.03, 95% CI 1.02-1.05) or unprocessed red meat (HR 1.03, 95% CI 1.01-1.05) associated with all-cause mortality, with no such association for poultry or fish [114]. These findings are directly related to the discussion of heme iron in Section 3.4. The same exposure associated with cardiovascular risk due to oxidative stress and inflammation [57, 58] is also independently linked to total mortality [113].

The converse exposures are equally relevant. Higher dietary fiber intake is associated with 15-30% lower all-cause mortality, cardiovascular mortality, and colorectal cancer incidence, with dose-response relationships strong enough to suggest causality [48]. However, the carnivore diet provides no fiber. As a result, the protective benefits associated with guideline-concordant dietary patterns are not only diminished on this diet but are completely unattainable.

7.3 Interpretation and Limitations of This Evidence

Three constraints apply to the preceding synthesis and should be weighed by readers. First, none of this evidence is carnivore-specific; it concerns exposures associated with the carnivore diet, not the diet itself. Second, the cohorts described above sampled dietary patterns far removed from a carnivore diet: participants in the lowest-carbohydrate categories typically consumed 30-40% of their energy as carbohydrate [105, 106], whereas the carnivore diet provides under 2% [1]. The relationships observed are therefore being extrapolated well beyond the range in which they were measured, and the shape of the dose-response curve in that range is unknown; this extrapolation may overstate or understate risk, but it provides no basis for assuming that risk diminishes at more extreme intakes. Third, these are observational data subject to residual confounding, dietary measurement error, and the possibility that individuals with high animal-food intakes differ systematically in unmeasured ways. Despite these caveats, the cohorts summarized in Section 7.2 show a consistent effect direction across geographic locations and analytical methods. Additionally, the coherence between epidemiological associations and established mechanisms, such as LDL elevation, oxidative stress from heme iron, endogenous formation of *N*-nitroso compounds, and fiber deficiency, is notable. Furthermore, the dose-response analyses show a biological gradient.

The conclusion drawn from the available evidence is precise and definitive: there is currently no evidence that the carnivore diet enhances longevity or reduces overall mortality rates, and no studies have been conducted to effectively establish such evidence. The existing indirect evidence regarding restriction of healthy carbohydrate sources, high intakes of red and processed meats, heme iron, and the lack of fiber and plant foods uniformly suggests increased overall and cancer mortality. Therefore, prospective evaluations of the carnivore diet should also focus on total mortality and cancer incidence as key endpoints, rather than limiting the assessment to cardiovascular measures. A dietary pattern that improves select short-term cardiometabolic

markers but increases non-cardiovascular mortality would contradict the objective of dietary prevention.

8. Priorities for Future Research

The evidentiary gaps identified throughout this review are specific enough to address with targeted study designs. Four priorities follow, ordered by feasibility. The most immediately tractable question is what a carnivore diet does to cardiovascular risk factors under conditions in which intake is known rather than self-reported. A randomized controlled feeding study of 8-12 weeks, comparing an isocaloric carnivore diet against a guideline-concordant comparator and against a ketogenic diet that retains non-starchy vegetables, nuts, and seeds, would separate the effects of carbohydrate restriction from those of plant-food exclusion, a distinction the present literature cannot make and on which the interpretation of every extrapolation in this review depends. A three-arm design with weight held constant would further separate the diet's metabolic effects from those of the energy deficit that accompanies it in practice [63]. Prespecified endpoints should include apolipoprotein B and LDL particle number alongside LDL-cholesterol, given the protein-rich, fiber-free composition; lipoprotein(a); ambulatory blood pressure, given the modeled sodium loads [1]; and objective measures of nutritional status for the nutrients identified as inadequate in modeling studies, particularly thiamin, vitamin C, magnesium, and potassium [1, 31]. Such a study would also generate the first objective dietary intake data for this pattern, addressing the theoretical-versus-actual gap described in Section 3.5.

Feeding studies cannot address duration or clinical outcomes. A prospective cohort study that recruits individuals currently following the carnivore diet, with regular objective measurements of lipid panels, coronary artery calcium scores, coronary computed tomography angiography, blood pressure, and nutrient biomarkers at enrollment and at designated intervals, would offer the first unbiased description of the long-term effects of this diet over several years instead of just months. Three design features are essential to avoid reproducing the limitations of the existing surveys: recruitment must extend beyond the advocacy channels through which the existing samples were assembled, since those channels are the origin of the selection bias described in Section 2; the cohort must retain and follow individuals who discontinue the diet, whose outcomes are the principal blind spot of the current literature; and outcome ascertainment must be conducted by investigators rather than by self-report. Total mortality and cancer incidence should be prespecified alongside cardiovascular endpoints, for the reasons given in Section 7.1. Nesting such a cohort within an existing prospective study infrastructure would improve comparability and reduce cost.

Three mechanistic questions are unresolved but consequential. The first concerns the extreme LDL response to carbohydrate restriction in some individuals: the lean mass hyper-responder phenotype and the lipid energy model have been proposed to explain it [14, 72], but the relative contributions of saturated fat intake, carbohydrate restriction, and body composition have not been experimentally disentangled [72], and doing so requires controlled manipulation of each variable in isolation. Whether the elevated LDL-cholesterol observed in this phenotype carries the same atherogenic risk as comparable elevations of other etiology is the single most consequential open question for the individuals most likely to adopt this diet. The second concerns heme iron: the proposed pathway from heme iron to oxidative stress, endothelial dysfunction, and plaque development [57, 58] has not been characterized at the intake levels typical of a meat-exclusive diet,

and its independent association with total mortality [113] makes this a priority beyond cardiovascular risk alone. The third concerns the gut microbiome: complete elimination of fermentable substrate depletes short-chain fatty acid-producing populations [10, 49], and controlled feeding of an exclusively animal-based diet alters gut microbial community structure within days, increasing bile-tolerant organisms and reducing those that ferment plant polysaccharides [115]; the consequences for intestinal barrier integrity and systemic inflammatory tone under sustained high animal-protein intake warrant direct characterization in adherents.

Two further lines of work follow from the public health framing set out in the Introduction. The prevalence, demographic distribution, and duration of carnivore diet adherence are unknown, and adding items that capture exclusionary dietary patterns to established population surveys would help determine the scale of the exposure. Separately, the reported discontinuation of prescribed medication by substantial proportions of adherents with diabetes, without documented physician involvement [3], is a patient safety signal that merits direct investigation, as is the question of how clinicians can most effectively communicate lipid-related risk to patients who have encountered extensive online material encouraging them to disregard it.

Until studies of this kind are conducted, the cardiovascular and mortality consequences of the carnivore diet will remain a matter of extrapolation. The purpose of the present review is to establish what that extrapolation can and cannot support, and to specify the questions that carnivore-specific research would need to answer.

9. Limitations

This review has several limitations to consider when interpreting its findings. First, as a narrative rather than a systematic review, the synthesis reflects the authors' judgment in selecting, weighting, and interpreting the available literature. Narrative reviews are inherently susceptible to confirmation bias, and readers should note that the conclusions presented here represent one reasonable interpretation of a limited and heterogeneous evidence base. Second, most carnivore diet literature consists of self-reported survey data, case reports, and modeling studies. These study designs cannot establish causality, are prone to selection and reporting biases, and cannot address long-term safety. Third, this review draws extensively on research on ketogenic and low-carbohydrate diets as a proxy for the effects of the carnivore diet. While these diets share severe carbohydrate restriction as a defining feature, they differ from carnivore diets in important ways, including fiber content, phytonutrient availability, food processing level, and typical protein intake; extrapolation from this broader literature should therefore be made with caution. Fourth, surrogate markers such as LDL-cholesterol and coronary artery calcium scores, while well-validated as cardiovascular risk indicators, are not equivalent to clinical event data. No studies have directly examined carnivore diet adherence in relation to hard cardiovascular outcomes such as myocardial infarction or stroke. Fifth, no carnivore-specific data exist for total mortality or cancer outcomes. The evidence in Section 7 concerns the individual exposures the carnivore diet maximizes or eliminates rather than the pattern itself, and derives from cohorts whose lowest-carbohydrate participants nonetheless consumed substantially more carbohydrate than a carnivore diet provides; its application here is therefore an extrapolation beyond the measured range.

10. Conclusion

Anecdotal reports and limited survey data suggest that the carnivore diet may improve glycemic control and select cardiovascular risk markers in the short term, potentially driven in part by concurrent weight loss, while other markers, most notably LDL-cholesterol, may worsen. The current evidence does not support the long-term safety or efficacy of carnivore diets in preventing cardiovascular disease. Concerns remain regarding its high levels of saturated fat, sodium, cholesterol, and heme iron, as well as its deficiencies in fiber and essential micronutrients, all of which are consistently associated with increased cardiovascular risk. Excluding all plant-based foods is inconsistent with public health and clinical practice guidelines, which recommend dietary patterns that include whole plant foods to reduce the burden of cardiovascular disease. Appeals to ancestral or ethnographic examples as justification for adopting a carnivore diet lack translational relevance, failing to account for differences in lifestyle, environment, caloric availability, and longevity that limit their applicability to modern populations.

Without long-term safety evidence, the carnivore diet cannot currently be recommended as a heart-healthy eating pattern and remains at odds with the prevailing scientific consensus. Moreover, indirect evidence on the exposures it maximizes and eliminates consistently points to increased total and cancer mortality, so its consequences cannot be presumed neutral simply because they are unmeasured. The urgency of resolving these questions is a function of the gap between adoption and evidence: a dietary pattern disseminated at scale through social media and commercial publishing, adopted preferentially by individuals with existing chronic disease, and associated in the available data with LDL-cholesterol concentrations ranging from a median approximately 70% above the recommended upper limit to individual values exceeding it fivefold, has never been evaluated in a single controlled prospective study. If these concerns translate into increased cardiovascular events with long-term adherence, the consequences would fall disproportionately on a population already carrying a high burden of cardiovascular risk. These effects could quietly accumulate over the years as atherosclerosis progresses without any noticeable symptoms. Conversely, if the diet is shown to be safe or beneficial in specific subgroups, definitive evidence will be essential to support that conclusion. Well-designed prospective trials with predefined cardiovascular endpoints are therefore needed without further delay; in the interim, clinicians should be aware that patients adopting this pattern may present with marked hypercholesterolemia and may have discontinued prescribed therapy based on information encountered online.

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Author Contributions

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David Goldman consults for Metabite, Inc. and has consulted with Soy Nutrition Institute Global. Thomas Waterfall consults for Game Changers Initiative, and consults for and owns stock in FYTA, Inc. Matthew Nagra previously had partnerships via affiliate links with FYTA and Complement® and has consulted with Soy Nutrition Institute Global.

Data Availability Statement

No new data were created or analyzed in this study. Data sharing is not applicable to this article.

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References

1. Goedeke S, Murphy T, Rush A, Zinn C. Assessing the nutrient composition of a carnivore diet: A case study model. *Nutrients*. 2025; 17: 140.
2. Dyńka D, Rodzeń Ł, Rodzeń M, Pacholak-Klimas A, Ede G, Sethi S, et al. Ketogenic diets for body weight loss: A comparison with other diets. *Nutrients*. 2025; 17: 965.
3. Lennerz BS, Mey JT, Henn OH, Ludwig DS. Behavioral characteristics and self-reported health status among 2029 adults consuming a “carnivore diet”. *Curr Dev Nutr*. 2021; 5: nzab133.
4. Saladino P. *The carnivore code: Unlocking the secrets to optimal health by returning to our ancestral diet*. Boston, MA: Houghton Mifflin Harcourt; 2020.
5. Klement RJ, Matzat JS. Subjective experiences and blood parameter changes in individuals from Germany following a self-conceived “carnivore diet”: An explorative study. *Cureus*. 2025; 17: e82521.
6. Carnivore.Diet. Carnivore diet success stories [Internet]. San Francisco, CA: Carnivore.Diet; 2026. Available from: <https://carnivore.diet/carnivore-diet-success-stories/>.
7. Landry MJ, Crimarco A, Gardner CD. Benefits of low carbohydrate diets: A settled question or still controversial? *Curr Obes Rep*. 2021; 10: 409-422.
8. Soto-Mota A, Flores-Jurado Y, Norwitz NG, Feldman D, Pereira MA, Danaei G, et al. Increased low-density lipoprotein cholesterol on a low-carbohydrate diet in adults with normal but not high body weight: A meta-analysis. *Am J Clin Nutr*. 2024; 119: 740-747.
9. Gonzalez JT, Lolli L, Atkinson G. Does BMI moderate the LDL cholesterol response to low-carbohydrate diets? *Am J Clin Nutr*. 2024; 120: 274-275.

10. Waśkiewicz Z. Carnivore and animal-based diets in sport: A critical evaluation of current evidence and future perspectives for precision nutrition. *Nutrients*. 2026; 18: 998.
11. Lietz A, Dapprich J, Fischer T. Carnivore diet: A scoping review of the current evidence, potential benefits and risks. *Nutrients*. 2026; 18: 348.
12. Wilson HE, Moe SM. You are what you eat-should it be all meat?: Impact of the carnivore diet on the risk of kidney stone development. *Am J Clin Nutr*. 2025; 121: 197-202.
13. Marmagkiolis K, Caballero J, Iliescu C. Yellowish nodules on a man consuming a carnivore diet. *JAMA Cardiol*. 2025; 10: 297.
14. Norwitz NG, Soto-Mota A. Case report: Carnivore-ketogenic diet for the treatment of inflammatory bowel disease: A case series of 10 patients. *Front Nutr*. 2024; 11: 1467475.
15. Palmer RD. The protein paradox, carnivore diet & hypertrophy versus longevity. Short term nutrition and hypertrophy versus longevity. *Nutr Health*. 2025; 31: 419-428.
16. O’Hearn A. Can a carnivore diet provide all essential nutrients? *Curr Opin Endocrinol Diabetes Obes*. 2020; 27: 312-316.
17. McClellan WS, Du Bois EF. Clinical calorimetry: XLV. Prolonged meat diets with a study of kidney function and ketosis. *J Biol Chem*. 1930; 87: 651-668.
18. Tolstoi E. The effect of an exclusive meat diet lasting one year on the carbohydrate tolerance of two normal men. *J Biol Chem*. 1929; 83: 747-752.
19. Tolstoi E. The effect of an exclusive meat diet on the chemical constituents of the blood. *J Biol Chem*. 1929; 83: 753-758.
20. Rana MS, Khan SS. Balancing autoimmune nutrition: Evaluating the carnivore diet’s risks and the protective role of plant-based foods in personalized care. *Aust Herb Insight*. 2025; 8: 1-22. doi: 10.25163/ahi.8110187.
21. Verma A. Reversing 20 years of diabetes using the carnivore diet in India: A case report. *Clin Nutr Res*. 2026; 15: 72-78.
22. U.S. Department of Agriculture, U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2025-2030 [Internet]. Washington, D.C.: USDA/HHS; 2026. Available from: <https://cdn.realfood.gov/DGA.pdf>.
23. Lichtenstein AH, Appel LJ, Vadiveloo M, Hu FB, Kris-Etherton PM, Rebholz CM, et al. 2021 dietary guidance to improve cardiovascular health: A scientific statement from the American Heart Association. *Circulation*. 2021; 144: e472-e487.
24. World Health Organization. Healthy diet [Internet]. Geneva, Switzerland: WHO; 2020. Available from: <https://www.who.int/news-room/fact-sheets/detail/healthy-diet>.
25. Karen JB. The carnivore diet code for beginners: Meat-based recipes for weight loss, healing, and optimal health: A 28-day meal plan handbook. Redwood City, CA: PublishDrive; 2023.
26. Baker S. Carnivore diet. Las Vegas, NV: Victory Belt Publishing; 2019.
27. Lietz A, Dapprich J, Fischer T. Preferences and recommendations from content creators on carnivore diets: A social media analysis. *J Health Popul Nutr*. 2026; 45: 137.
28. Kirwan R, Mallett GS, Ellis L, Flanagan A. Limitations of self-reported health status and metabolic markers among adults consuming a “carnivore diet”. *Curr Dev Nutr*. 2022; 6: nzac037.
29. Squire P, Ghosh M. Acute pancreatitis caused by severe hypertriglyceridemia in a 61-year-old man on a carnivore diet. *CMAJ*. 2026; 198: E622-E625.

30. Nagra M, Goldman DM, Belury MA, Messina M. Concerns about the health effects of industrially produced seed oils are without scientific foundation: A scoping narrative review of the clinical and observational evidence. *Crit Rev Food Sci Nutr.* 2026; 66: 5386-5445.
31. Phelan JM, Joyce JM, Bode K, Rosenkranz SK. Opportunities for maximizing the dietary quality of fad diets. *Nutrients.* 2023; 15: 4526.
32. Goldman DM, Waterfall TJ, Nagra M. Traditional Maasai dietary practices and their inapplicability to modern carnivore diets: A narrative review. *Cureus.* 2025; 17: e78448.
33. Teicholz N. *The big fat surprise: Why butter, meat and cheese belong in a healthy diet.* New York, NY: Simon and Schuster; 2014.
34. Cho J. *Carnivore cure: Meat-based nutrition and the ultimate elimination diet to attain optimal health.* Austin, TX: Nutrition with Judy; 2020.
35. Greene J. *Carnivore diet: A guide to eat meat, get lean, and stay healthy an alternative for paleo and keto diet.* Sugar Hill, CA: Novelty Publishing LLC.; 2020.
36. Bennett TW. *The carnivore diet for optimal health: Easy ways to lose weight and burn fat.* 2020.
37. Carlsen MH, Halvorsen BL, Holte K, Bøhn SK, Dragland S, Sampson L, et al. The total antioxidant content of more than 3100 foods, beverages, spices, herbs and supplements used worldwide. *Nutr J.* 2010; 9: 3.
38. Hever J, Cronise RJ. Plant-based nutrition for healthcare professionals: Implementing diet as a primary modality in the prevention and treatment of chronic disease. *J Geriatr Cardiol.* 2017; 14: 355-368.
39. Liu RH. Health-promoting components of fruits and vegetables in the diet. *Adv Nutr.* 2013; 4: 384S-92S.
40. Sheffield S, Fiorotto ML, Davis TA. Nutritional importance of animal-sourced foods in a healthy diet. *Front Nutr.* 2024; 11: 1424912.
41. Chen Z, Glisic M, Song M, Aliahmad HA, Zhang X, Moumdjian AC, et al. Dietary protein intake and all-cause and cause-specific mortality: Results from the Rotterdam Study and a meta-analysis of prospective cohort studies. *Eur J Epidemiol.* 2020; 35: 411-429.
42. Goldman DM, Warbeck CB, Barbaro R, Khambatta C, Nagra M. Assessing the roles of retinol, vitamin K2, carnitine, and creatine in plant-based diets: A narrative review of nutritional adequacy and health implications. *Nutrients.* 2025; 17: 525.
43. Goldman D, Nagra M. Choline adequacy and health outcomes in vegetarian and vegan diets. *J Acad Nutr Diet.* 2025; 2. doi: 10.20935/AcadNutr8085.
44. Wen H, Niu X, Zhao R, Wang Q, Sun N, Ma L, et al. Association of vitamin B1 with cardiovascular diseases, all-cause and cardiovascular mortality in US adults. *Front Nutr.* 2023; 10: 1175961.
45. Del Gobbo LC, Imamura F, Wu JH, de Oliveira Otto MC, Chiuve SE, Mozaffarian D. Circulating and dietary magnesium and risk of cardiovascular disease: A systematic review and meta-analysis of prospective studies. *Am J Clin Nutr.* 2013; 98: 160-173.
46. Larsson SC, Orsini N, Wolk A. Dietary magnesium intake and risk of stroke: A meta-analysis of prospective studies. *Am J Clin Nutr.* 2012; 95: 362-366.
47. Jayedi A, Rashidy-Pour A, Parohan M, Zargar MS, Shab-Bidar S. Dietary and circulating vitamin C, vitamin E, β -carotene and risk of total cardiovascular mortality: A systematic review and dose-response meta-analysis of prospective observational studies. *Public Health Nutr.* 2019; 22: 1872-1887.

48. Reynolds A, Mann J, Cummings J, Winter N, Mete E, Te Morenga L. Carbohydrate quality and human health: A series of systematic reviews and meta-analyses. *Lancet*. 2019; 393: 434-445.
49. Ramezani F, Pourghazi F, Eslami M, Gholami M, Khonsari NM, Ejtahed HS, et al. Dietary fiber intake and all-cause and cause-specific mortality: An updated systematic review and meta-analysis of prospective cohort studies. *Clin Nutr*. 2024; 43: 65-83.
50. Hao X, Li D. The Healthy Eating Index-2015 and all-cause/cause-specific mortality: A systematic review and dose-response meta-analysis. *Adv Nutr*. 2024; 15: 100166.
51. Wang YJ, Yeh TL, Shih MC, Tu YK, Chien KL. Dietary sodium intake and risk of cardiovascular disease: A systematic review and dose-response meta-analysis. *Nutrients*. 2020; 12: 2934.
52. Sacks FM, Lichtenstein AH, Wu JH, Appel LJ, Creager MA, Kris-Etherton PM, et al. Dietary fats and cardiovascular disease: A presidential advisory from the American Heart Association. *Circulation*. 2017; 136: e1-e23.
53. Zhang Y, Pletcher MJ, Vittinghoff E, Clemons AM, Jacobs Jr DR, Allen NB, et al. Association between cumulative low-density lipoprotein cholesterol exposure during young adulthood and middle age and risk of cardiovascular events. *JAMA Cardiol*. 2021; 6: 1406-1413.
54. Boren J, Chapman MJ, Krauss RM, Packard CJ, Bentzon JF, Binder CJ, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: Pathophysiological, genetic, and therapeutic insights: A consensus statement from the European Atherosclerosis Society Consensus Panel. *Eur Heart J*. 2020; 41: 2313-2330.
55. Carson JA, Lichtenstein AH, Anderson CA, Appel LJ, Kris-Etherton PM, Meyer KA, et al. Dietary cholesterol and cardiovascular risk: A science advisory from the American Heart Association. *Circulation*. 2020; 141: e39-e53.
56. Archundia-Herrera MC, Nunes F, Barrios ID, Park CY, Bell RC, O'Brien KO. Development of a database for the estimation of heme iron and nonheme iron content of animal-based foods. *Curr Dev Nutr*. 2024; 8: 102130.
57. Fang X, An P, Wang H, Wang X, Shen X, Li X, et al. Dietary intake of heme iron and risk of cardiovascular disease: A dose-response meta-analysis of prospective cohort studies. *Nutr Metab Cardiovasc Dis*. 2015; 25: 24-35.
58. Yang W, Li B, Dong X, Zhang XQ, Zeng Y, Zhou JL, et al. Is heme iron intake associated with risk of coronary heart disease? A meta-analysis of prospective studies. *Eur J Nutr*. 2014; 53: 395-400.
59. Kiyimba T, Yiga P, Bamuwanye M, Ogwok P, Van der Schueren B, Matthys C. Efficacy of dietary polyphenols from whole foods and purified food polyphenol extracts in optimizing cardiometabolic health: A meta-analysis of randomized controlled trials. *Adv Nutr*. 2023; 14: 270-282.
60. Tian W, Cao S, Guan Y, Zhang Z, Liu Q, Ju J, et al. The effects of low-carbohydrate diet on glucose and lipid metabolism in overweight or obese patients with T2DM: A meta-analysis of randomized controlled trials. *Front Nutr*. 2025; 11: 1516086.
61. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: A scientific statement from the American Heart Association. *Circulation*. 2021; 143: e984-e1010.
62. Leung LY, Tam HL, Ho JK. Effects of ketogenic and low-carbohydrate diets on the body composition of adults with overweight or obesity: A systematic review and meta-analysis of randomised controlled trials. *Clin Nutr*. 2025; 46: 10-18.

63. Naude CE, Schoonees A, Senekal M, Young T, Garner P, Volmink J. Low carbohydrate versus isoenergetic balanced diets for reducing weight and cardiovascular risk: A systematic review and meta-analysis. *PLoS One*. 2014; 9: e100652.
64. Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L, et al. Global burden of hypertension and systolic blood pressure of at least 110 to 115 mm Hg, 1990-2015. *JAMA*. 2017; 317: 165-182.
65. Yang S, Zhou Z, Miao H, Zhang Y. Effect of weight loss on blood pressure changes in overweight patients: A systematic review and meta-analysis. *J Clin Hypertens*. 2023; 25: 404-415.
66. Dong T, Guo M, Zhang P, Sun G, Chen B. The effects of low-carbohydrate diets on cardiovascular risk factors: A meta-analysis. *PLoS One*. 2020; 15: e0225348.
67. Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM. Influence of weight reduction on blood pressure: A meta-analysis of randomized controlled trials. *Hypertension*. 2003; 42: 878-884.
68. Jung E, Kong SY, Ro YS, Ryu HH, Shin SD. Serum cholesterol levels and risk of cardiovascular death: A systematic review and a dose-response meta-analysis of prospective cohort studies. *Int J Environ Res Public Health*. 2022; 19: 8272.
69. Clarke R, Frost C, Collins R, Appleby P, Peto R. Dietary lipids and blood cholesterol: Quantitative meta-analysis of metabolic ward studies. *BMJ*. 1997; 314: 112.
70. Joo M, Moon S, Lee YS, Kim MG. Effects of very low-carbohydrate ketogenic diets on lipid profiles in normal-weight (body mass index <25 kg/m²) adults: A meta-analysis. *Nutr Rev*. 2023; 81: 1393-1401.
71. Silverman MG, Ference BA, Im K, Wiviott SD, Giugliano RP, Grundy SM, et al. Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: A systematic review and meta-analysis. *JAMA*. 2016; 316: 1289-1297.
72. DiMattia ZS, Petersen KS. Ketogenic diets and low-density lipoprotein cholesterol in adults with normal weight: An emerging clinical challenge. *J Am Heart Assoc*. 2026; 15: e048903.
73. Soto-Mota A, Norwitz NG, Manubolu VS, Kinnering A, Wood TR, Earls J, et al. Longitudinal data from the KETO-CTA study: Plaque predicts plaque, ApoB does not. *JACC Adv*. 2025; 4: 101686. Retraction in: *JACC Adv*. 2026; 5: 102824.
74. Kirwan R, Elliott P, Flanagan A, McGowan G, Nagra M, Mughal I, et al. Rapid plaque progression amongst lean mass hyper-responders following a ketogenic diet with elevated ApoB and LDL-cholesterol. 2025. doi: 10.31219/osf.io/78bph_v1.
75. Aldana J, Kinnering A, Krishnan S, Ichikawa K, Budoff M, Karlsberg R. Atherosclerotic plaque progresses over time in healthy individuals without MACE, risk factors, or interventions. *Circulation*. 2024; 150: A4139340.
76. Soto-Mota A, Norwitz NG, Manubolu VS, Kinnering A, Wood TR, Earls J, et al. Reply: The keto CTA study. *JACC Adv*. 2025; 4: 101862.
77. Henzel J, Kępka C, Kruk M, Makarewicz-Wujec M, Wardziak Ł, Trochimiuk P, et al. High-risk coronary plaque regression after intensive lifestyle intervention in nonobstructive coronary disease: A randomized study. *JACC Cardiovasc Imaging*. 2021; 14: 1192-1202.
78. Ference BA, Ginsberg HN, Graham I, Ray KK, Packard CJ, Bruckert E, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European Atherosclerosis Society Consensus Panel. *Eur Heart J*. 2017; 38: 2459-2472.

79. Yang R, Wu S, Zhao Z, Deng X, Deng Q, Wang D, et al. Causal association between lipoproteins and risk of coronary artery disease-a systematic review and meta-analysis of Mendelian randomization studies. *Clin Res Cardiol.* 2026; 115: 175-184.
80. Lee JS, Chang PY, Zhang Y, Kizer JR, Best LG, Howard BV. Triglyceride and HDL-C dyslipidemia and risks of coronary heart disease and ischemic stroke by glycemic dysregulation status: The Strong Heart Study. *Diabetes Care.* 2017; 40: 529-537.
81. Ganjali S, Watts GF, Banach M, Reiner Ž, Nachtigal P, Sahebkar A. The Yin and Yang of high-density lipoprotein and atherosclerotic cardiovascular disease: Focusing on functionality and cholesterol efflux to reframe the HDL hypothesis. *Curr Med Chem.* 2021; 28: 6066-6081.
82. Kjeldsen EW, Thomassen JQ, Frikke-Schmidt R. HDL cholesterol concentrations and risk of atherosclerotic cardiovascular disease-insights from randomized clinical trials and human genetics. *Biochim Biophys Acta Mol Cell Biol Lipids.* 2022; 1867: 159063.
83. Chawla S, Tessarolo Silva F, Amaral Medeiros S, Mekary RA, Radenkovic D. The effect of low-fat and low-carbohydrate diets on weight loss and lipid levels: A systematic review and meta-analysis. *Nutrients.* 2020; 12: 3774.
84. Hasan B, Nayfeh T, Alzuabi M, Wang Z, Kuchkuntla AR, Prokop LJ, et al. Weight loss and serum lipids in overweight and obese adults: A systematic review and meta-analysis. *J Clin Endocrinol Metab.* 2020; 105: 3695-3703.
85. Piccolo R, Laino A, Vitale AP, Franzone A, Giacoppo D, Bossone E, et al. Association between C-reactive protein and adverse events in secondary cardiovascular prevention: A systematic review and meta-analysis of prognostic factor studies. *Atherosclerosis.* 2026; 412: 120574.
86. Wang J, Zhang D, Huang R, Li X, Huang W. Gamma-glutamyltransferase and risk of cardiovascular mortality: A dose-response meta-analysis of prospective cohort studies. *PLoS One.* 2017; 12: e0172631.
87. Kemalasari I, Fitri NA, Sinto R, Tahapary DL, Harbuwono DS. Effect of calorie restriction diet on levels of C reactive protein (CRP) in obesity: A systematic review and meta-analysis of randomized controlled trials. *Diabetes Metab Syndr.* 2022; 16: 102388.
88. Sajan K, Anthireddy N, Matarazzo A, Furtado C, Hennekens CH, Ferris A. Ultra-processed foods and increased high sensitivity C-reactive protein. *Am J Med.* 2025; 138: 1742-1745.
89. Rodriguez-Granillo GA, Carrascosa P, Bruining N. Progression of coronary artery calcification at the crossroads: Sign of progression or stabilization of coronary atherosclerosis? *Cardiovasc Diagn Ther.* 2016; 6: 250-258.
90. McEvoy JW, Blaha MJ, DeFilippis AP, Budoff MJ, Nasir K, Blumenthal RS, et al. Coronary artery calcium progression: An important clinical measurement? A review of published reports. *J Am Coll Cardiol.* 2010; 56: 1613-1622.
91. Cara KC, Goldman DM, Kollman BK, Amato SS, Tull MD, Karlsen MC. Commonalities among dietary recommendations from 2010 to 2021 clinical practice guidelines: A meta-epidemiological study from the American College of Lifestyle Medicine. *Adv Nutr.* 2023; 14: 500-515.
92. Reynolds AN, Hodson L, De Souza R, Vlietstra L, Mann J. Saturated fat and trans-fat intakes and their replacement with other macronutrients: A systematic review and meta-analysis of prospective observational studies. Geneva, Switzerland: World Health Organization; 2023.

93. Capodici A, Mocciaro G, Gori D, Landry MJ, Masini A, Sanmarchi F, et al. Cardiovascular health and cancer risk associated with plant based diets: An umbrella review. *PLoS One*. 2024; 19: e0300711.
94. Zhang X, Liang S, Chen X, Yang J, Zhou Y, Du L, et al. Red/processed meat consumption and non-cancer-related outcomes in humans: Umbrella review. *Br J Nutr*. 2023; 130: 484-494.
95. Neuenschwander M, Stadelmaier J, Eble J, Grummich K, Szczerba E, Kiesswetter E, et al. Substitution of animal-based with plant-based foods on cardiometabolic health and all-cause mortality: A systematic review and meta-analysis of prospective studies. *BMC Med*. 2023; 21: 404.
96. Zimmerman MR. The paleopathology of the cardiovascular system. *Tex Heart Inst J*. 1993; 20: 252-257.
97. Wang Y, Liu B, Han H, Hu Y, Zhu L, Rimm EB, et al. Associations between plant-based dietary patterns and risks of type 2 diabetes, cardiovascular disease, cancer, and mortality-a systematic review and meta-analysis. *Nutr J*. 2023; 22: 46.
98. Gan ZH, Cheong HC, Tu YK, Kuo PH. Association between plant-based dietary patterns and risk of cardiovascular disease: A systematic review and meta-analysis of prospective cohort studies. *Nutrients*. 2021; 13: 3952.
99. Quek J, Lim G, Lim WH, Ng CH, So WZ, Toh J, et al. The association of plant-based diet with cardiovascular disease and mortality: A meta-analysis and systematic review of prospect cohort studies. *Front Cardiovasc Med*. 2021; 8: 756810.
100. Goldman DM, Nagra M, Warbeck C, Winings E, Landry MJ. Plant-based diets: Nutritional considerations and public health implications. In: *Handbook of public health nutrition: International, national, and regional perspectives*. Cham, Switzerland: Springer Cham; 2025. pp. 1-27.
101. De Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: Final report of the Lyon Diet Heart Study. *Circulation*. 1999; 99: 779-785.
102. Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. *N Engl J Med*. 2018; 378: e34.
103. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med*. 1997; 336: 1117-1124.
104. Soltani S, Arablou T, Jayedi A, Salehi-Abargouei A. Adherence to the Dietary Approaches to Stop Hypertension (DASH) diet in relation to all-cause and cause-specific mortality: A systematic review and dose-response meta-analysis of prospective cohort studies. *Nutr J*. 2020; 19: 37.
105. Fung TT, Van Dam RM, Hankinson SE, Stampfer M, Willett WC, Hu FB. Low-carbohydrate diets and all-cause and cause-specific mortality: Two cohort studies. *Ann Intern Med*. 2010; 153: 289-298.
106. Seidemann SB, Claggett B, Cheng S, Henglin M, Shah A, Steffen LM, et al. Dietary carbohydrate intake and mortality: A prospective cohort study and meta-analysis. *Lancet Public Health*. 2018; 3: e419-e428.
107. Mazidi M, Katsiki N, Mikhailidis DP, Sattar N, Banach M. Lower carbohydrate diets and all-cause and cause-specific mortality: A population-based cohort study and pooling of prospective studies. *Eur Heart J*. 2019; 40: 2870-2879.

108. Noto H, Goto A, Tsujimoto T, Noda M. Low-carbohydrate diets and all-cause mortality: A systematic review and meta-analysis of observational studies. *PLoS One*. 2013; 8: e55030.
109. Shan Z, Guo Y, Hu FB, Liu L, Qi Q. Association of low-carbohydrate and low-fat diets with mortality among US adults. *JAMA Intern Med*. 2020; 180: 513-523.
110. Hu Y, Liu G, Yu E, Wang B, Wittenbecher C, Manson JE, et al. Low-carbohydrate diet scores and mortality among adults with incident type 2 diabetes. *Diabetes Care*. 2023; 46: 874-884.
111. Bouvard V, Loomis D, Guyton KZ, Grosse Y, El Ghissassi F, Benbrahim-Tallaa L, et al. Carcinogenicity of consumption of red and processed meat. *Lancet Oncol*. 2015; 16: 1599-1600.
112. Farvid MS, Sidahmed E, Spence ND, Mante Angua K, Rosner BA, Barnett JB. Consumption of red meat and processed meat and cancer incidence: A systematic review and meta-analysis of prospective studies. *Eur J Epidemiol*. 2021; 36: 937-951.
113. Etemadi A, Sinha R, Ward MH, Graubard BI, Inoue-Choi M, Dawsey SM, et al. Mortality from different causes associated with meat, heme iron, nitrates, and nitrites in the NIH-AARP Diet and Health Study: Population based cohort study. *BMJ*. 2017; 357: j1957.
114. 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: 503-512.
115. David LA, Maurice CF, Carmody RN, Gootenberg DB, Button JE, Wolfe BE, et al. Diet rapidly and reproducibly alters the human gut microbiome. *Nature*. 2014; 505: 559-563.