

Review

Interpreting Lower HDL Cholesterol in Plant-Based Diets: Mechanisms and Clinical Implications



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ABSTRACT

Plant-based dietary patterns are associated with improved cardiometabolic health, including lower concentrations of serum low-density lipoprotein cholesterol, blood pressure, adiposity, inflammation, and lower risk of cardiovascular disease. However, some studies report modestly reduced high-density lipoprotein (HDL) cholesterol concentrations in populations following plant-based diets (e.g., vegan or vegetarian), whereas other studies show null associations. This has created uncertainty about whether these changes in HDL cholesterol reflect clinically meaningful harm or a neutral physiologic response. This review examines the significance of lower HDL cholesterol in the context of plant-based diets and broader cardiometabolic risk. We describe plausible mechanistic pathways, including lower saturated fat and cholesterol intake, reduced apolipoprotein A-I secretion and HDL production, and formation of smaller HDL particles that are cleared more rapidly. We suggest that modest HDL cholesterol reductions observed with healthy and appropriately planned plant-based diets should be interpreted within the broader context of net cardiometabolic benefit rather than as an isolated reason for clinical concern. However, substantial heterogeneity across studies—in both results and methodology, including variation in the definitions of plant-based diets—limits causal directionality and interpretation of reported diet–disease associations. Future analyses should improve diet classification and comparator selection, while controlling for weight change and macronutrient composition to isolate any potential effects of plant-based diets on HDL-related outcomes.

Keywords: plant-based diet, vegetarian, vegan, high-density lipoprotein cholesterol, cardiometabolic risk

Statement of Significance

Modest reductions in HDL cholesterol are sometimes reported as a disadvantage or risk of plant-based dietary patterns. This review integrates lipid epidemiology, HDL function, dietary intervention evidence, and clinical guidance to suggest that small HDL cholesterol reductions should be interpreted within the broader atherogenic lipid and cardiometabolic risk profile rather than treated as a standalone adverse outcome.

Cardiometabolic Burden of Disease and Plant-Based Diets

Plant-based dietary patterns (e.g., vegetarian and vegan) are associated with improvements in cardiometabolic risk factors

such as LDL cholesterol, blood pressure, body mass index (BMI), and C-reactive protein, as well as lower overall risk of cardiovascular disease (CVD) incidence and mortality [1,2]. Furthermore, consumption of plant-based diets is associated with decreased risk of developing other noncommunicable disease

Abbreviations: ACC, American College of Cardiology; AHA, American Heart Association; apoA-I, apolipoprotein A-I; apoB, apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; CAD, coronary artery disease; CEC, cholesterol efflux capacity; CI, confidence interval; CVD, cardiovascular disease; MetS, metabolic syndrome; PDI, plant-based diet index; RCT, randomized controlled trial; RDN, registered dietitian nutritionist.

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<https://doi.org/10.1016/j.advnut.2026.100708>

Received 27 May 2026; Received in revised form 9 July 2026; Accepted 15 July 2026; Available online 17 July 2026

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outcomes such as type 2 diabetes, certain types of cancer, stroke, and all-cause mortality [1,3–5]. Despite these known associations, the global burden of cardiometabolic noncommunicable diseases remains substantial and continues to rise. In 2022, ~43% of adults worldwide were overweight, and 16% were living with obesity, whereas diabetes prevalence doubled to 14%, up from 7% in 1990, affecting ~600 million adults [6,7]. In the United States, where cardiometabolic burden is particularly high and well-documented through nationally representative surveillance data, ~72% of adults have overweight or obesity, with an estimated 58% having diabetes or prediabetes [8,9]. These conditions causally elevate CVD risk through mechanisms such as insulin resistance, atherogenic dyslipidemia, hypertension, chronic inflammation, and endothelial dysfunction; Mendelian randomization studies further demonstrate that genetic predisposition increases risk of CVD-related outcomes [10,11]. Moreover, a 2022 analysis found that only 6.8% of United States adults are metabolically healthy, defined as meeting all of the following criteria: fasting blood glucose of <100 mg/dL; systolic/diastolic blood pressure <120/<80 mmHg, respectively; triglycerides <150 mg/dL; HDL cholesterol $\geq$ 40 mg/dL (men) or $\geq$ 50 mg/dL (women); and waist circumference <102 cm (men) or <88 cm (women) [12].

The adoption of plant-based dietary patterns is increasing in high-income countries, driven by health concerns, environmental awareness, and product innovation [13,14]. This trend aligns with growing calls for dietary shifts toward plant-based diets to promote both human health and environmental sustainability. The Engage, Act, Transform (EAT)-Lancet Commission recommends a “planetary health diet” that is predominantly plant-based, with substantial increases in vegetables, fruits, legumes, nuts, and whole grains, alongside significant reductions in red meat and other animal-sourced foods for high-income countries, highlighting the possibility of such diet to prevent millions of premature deaths annually, simultaneously supporting global food security [15]. Relatedly, many countries have incorporated similar plant-based recommendations into their national dietary guidelines. For example, Mexico’s 2025–2030 dietary guidelines call for local, seasonal, and plant-based foods, celebrating traditional “Dieta de la Milpa” (maize, beans, squash, and chili) as a central model: emphasizing meat reduction while recommending that fruits and vegetables compose 50% of the plate [16]. In addition, the Nordic countries (Denmark, Finland, Iceland, Norway, and Sweden) follow joint dietary guideline recommendations that advocate for a predominantly plant-based diet rich in vegetables, fruits, berries, legumes, whole grains, nuts, and fish [17].

Notwithstanding the broad, yet predominantly cardiometabolic, health benefits gained from plant-based diets, some studies have identified an association with modestly lower concentrations of HDL cholesterol, alongside more pronounced decreases in atherogenic lipids [18]. Although this lower HDL cholesterol concentration may reflect lower saturated fat and cholesterol intake, and does not appear to negate the net cardiovascular protection conferred by plant-based dietary patterns, it raises important questions about HDL cholesterol’s functional role and whether such changes have clinical relevance. In the following sections, we examine the physiological functions of HDL cholesterol and its associations with cardiovascular health, followed by

potential mechanisms underlying HDL cholesterol reductions seen in populations following varying types of plant-based diets, to inform evidence-based guidance for researchers and clinicians managing patients interested in or adopting these patterns.

Cholesterol Dynamics and HDL Cholesterol’s Role in Health and Plant-Based Diets

Cholesterol is an essential lipophilic molecule that serves as a structural component of cell membranes, acting as a precursor for vitamin D, steroidal hormones (e.g., cortisol and adrenal androgens), sex hormones (e.g., estrogen, testosterone, and progesterone), and bile acids needed for digesting dietary fats and absorbing fat-soluble vitamins [19]. Atherosclerosis is driven in large part by the accumulation of atherogenic lipoproteins, particularly LDL particles, within the arterial wall. Elevated concentrations of circulating LDL particles permeate damaged endothelium, undergo oxidation, which triggers uptake by macrophages into foam cells, initiating plaque formation and vascular inflammation [19].

Diet modulates circulating LDL cholesterol through multiple pathways. Dietary cholesterol has a modest and nonlinear effect on serum LDL cholesterol; a meta-regression from controlled feeding trials demonstrates that increasing intake from ~0 mg/d to 400 mg/d (the equivalent found in ~2 large eggs) raises LDL cholesterol by ~8 mg/dL, whereas further increases from 400 mg/d to 800 mg/d would only contribute an additional ~2 mg/dL [20]. In contrast, saturated fatty acids (SFAs) exhibit a stronger and mostly linear association, with each 1% of energy replaced by polyunsaturated fatty acids (PUFAs) lowering LDL cholesterol by ~2.1 mg/dL, and smaller reductions when replaced by monounsaturated fatty acids (MUFAs) (~1.6 mg/dL) or carbohydrates (~1.3 mg/dL). Accordingly, plant-based diets typically improve overall lipid profiles by simultaneously minimizing both dietary cholesterol and saturated fat while supplying higher quantities of unsaturated fats and soluble fiber, binding bile acids, and promoting cholesterol excretion [21].

HDL particles are the smallest and densest, yet most heterogeneous in size, of the lipoprotein classes. These variations directly influence biological activities and may explain why concentration alone is an imperfect marker of risk prevention [22]. The primary atheroprotective mechanism of HDL is reverse cholesterol transport, the only pathway for net removal of excess cholesterol from peripheral tissues to the liver for excretion. Cholesterol efflux capacity (CEC) is a functional assay that quantifies the ability of HDL to promote the removal of cholesterol from macrophages; higher CEC values indicate more efficient reverse cholesterol transport [23]. Beyond reverse cholesterol transport, HDL exhibits antioxidant properties through the inhibition of LDL oxidation and reactive oxygen species formation; anti-inflammatory actions, including suppression of endothelial adhesion molecules and modulation of macrophage cytokine release; and promotion of nitric oxide production, enhancing vasodilation and inhibiting thrombosis [24]. These actions are highly dependent on HDL particle quality and composition: dysfunction (e.g., in inflammatory states) can convert HDL to a proinflammatory particle, emphasizing that

quantity (i.e., serum HDL cholesterol) does not always equate to function.

Epidemiologic studies have demonstrated an inverse association between serum HDL cholesterol concentrations and CVD risk, with a pooled analysis of 68 prospective cohort studies (N = 302,430) demonstrating that each 1 SD higher HDL cholesterol (~15 mg/dL) reduced coronary artery disease (CAD) risk by 20% to 25% [25]. However, current evidence indicates a nonlinear, U-shaped relationship [26,27], and causality between elevated HDL cholesterol alone and lower risk of CVDs has yet to be firmly established [28]. Mendelian randomization studies have shown that genetically elevated HDL cholesterol does not reduce CVD events, suggesting that HDL cholesterol is largely a marker rather than a causal mediator [29–31]. In contrast, CEC may be a stronger predictor of CVD risk than HDL cholesterol alone. Two recent meta-analyses have confirmed that higher CEC is associated with lower risk of CAD and adverse cardiovascular events, independent of HDL cholesterol concentration. In a 2021 meta-analysis of 20 studies, high concentrations of CEC (defined study-dependently as above the cohort median or in the top quartile/tertile) were associated with a 37% lower risk of adverse cardiovascular events compared with low concentrations. A 2022 meta-analysis of 18 studies found a linear dose–response relationship, whereby each 20% increase in CEC was associated with a 10% reduction in CAD risk [32,33].

These findings indicate that HDL cholesterol is an incomplete surrogate for HDL biology: although HDL particles perform several cardioprotective functions, circulating HDL cholesterol concentration does not fully capture these effects and may reflect broader metabolic processes rather than a direct mediator of CVD risk, and measurement of HDL cholesterol does not

accurately characterize HDL function [27,34]. Indeed, it has been hypothesized that low HDL cholesterol may serve as an indirect marker of CVD risk, given its confounded association with obesity, diabetes, and impaired glucose tolerance [35].

In addition, subspecies of HDL may have differing influences on cardiometabolic outcomes. For example, some subspecies of HDL, including those that contain apoC1 or apoE, are linked to a lower risk of CVD, whereas others, such as HDL-containing complement C3 or α -2-macroglobulin, are associated with increased risk of CVD [36], presenting an additional potential explanation for discrepancies in findings between HDL cholesterol and CVD, and further substantiating a need for more precise measurements.

These distinctions are essential for interpreting whether observed diet-related reductions reflect impaired reverse cholesterol transport or largely a neutral physiologic response with uncertain cardiometabolic relevance.

Associations between Plant-Based Diets and HDL Cholesterol

Plant-based diets have diverse classifications but typically refer to the consumption of fruits, vegetables, grains, legumes, nuts and seeds, herbs, spices, while excluding various forms of animal products (Table 1).

Plant-based diets are associated with no [1,40] to small but statistically significant decreases in HDL cholesterol [1,4,41]. Here, we summarize the impact of plant-based diets on HDL cholesterol: 1) compared with omnivorous diets, 2) as an intervention (compared with no control or intervention), and 3) in

TABLE 1
Common variations of plant-based dietary patterns and plant-based diet indices

Diet type or index	Included/positively scored	Excluded/reverse scored	Definition/characteristics
<i>Dietary patterns</i>			
Vegetarian	Dairy, eggs	All meat, fish	A predominantly plant-based diet excluding meat and fish but allowing dairy and eggs (lactoovovegetarian). Subvariations include lactovegetarian (dairy but no eggs) and ovovegetarian (eggs but no dairy).
Pescatarian	Fish/seafood, dairy, eggs	Red meat, poultry	A largely vegetarian diet that also includes seafood.
Flexitarian	Occasional meat, fish, dairy, eggs	None strictly	A broad, “part-time” vegetarian diet that primarily follows a plant-based approach but allows animal products on occasion.
Vegan	None	All animal products (meat, dairy, eggs, honey, gelatin)	A strict plant-based diet that excludes all animal products and byproducts such as honey and gelatin.
Whole-foods, plant-based	None	All animal products	A vegan diet that focuses on health rather. Emphasizes minimally processed whole foods (fruits, vegetables, whole grains, legumes, nuts, seeds) and is typically low in fat.
<i>Diet indices</i>			
Plant-based diet index	All plant food groups	Animal food groups	A graded index (18–90) based on quintile scoring (1–5 per food group). Positive scores for higher intake of plant foods; reverse scores for higher intake of animal foods.
Healthful plant-based diet index	Healthy plant foods (whole grains, fruits, vegetables, nuts, legumes, vegetable oils, tea/coffee)	Less healthy plant foods and all animal foods	A graded index (18–90) based on quintile scoring (1–5 per food group). Positive scores for higher intake of healthy plant foods; reverse scores for higher intake of less healthy plant foods and animal foods.
Unhealthful plant-based diet index	Less healthy plant foods (fruit juices, sugar-sweetened beverages, refined grains, potatoes, sweets/desserts)	Healthy plant foods and all animal foods	A graded index (18–90) based on quintile scoring (1–5 per food group). Positive scores for higher intake of less healthy plant foods; reverse scores for higher intake of healthy plant foods and animal foods.

Dietary pattern definitions adapted from Clem and Barthel [37] and Ostfeld [38]; plant-based diet indices adapted from Satija et al. [39].

intervention settings compared with other therapeutic diets. We focus primarily on vegetarian and vegan diets, which our literature search identified as the most frequently defined plant-based dietary patterns in studies examining associations with HDL cholesterol (Supplemental Table 1). To inform our review, all umbrella, systematic review, and meta-analyses examining plant-based (e.g., vegan and vegetarian) diets and cardiometabolic health or lipid profiles that included HDL cholesterol were retrieved through literature searches. Individual studies cited within each review that included HDL cholesterol as an outcome were further reviewed in detail to describe the frequency of examined plant-based diets (Supplemental Table 1).

Comparison of plant-based diets to omnivore diets

Observational analyses result in null to small but statistically significant decreases in HDL cholesterol. For example, Yokoyama et al. [18] examined the effects of vegetarian diets (including vegan, lacto, lactoovo, pesco, and/or semivegetarian) in adults aged ≥ 20 y for ≥ 1 y compared with omnivorous diets, pooling data across 30 observational studies ($N = 10,143$). Compared with omnivores, vegetarian diets were associated with decreased HDL cholesterol [-3.6 mg/dL (-0.093 mmol/L)] [42]. Furthermore, in a meta-analysis by Dinu et al. [43], across 86 cross-sectional and 10 prospective cohort studies with clinically healthy adult subjects (≥ 18 y, $N = 6194$), vegetarian diets were associated with lower HDL cholesterol [-2.72 mg/dL (-0.0703 mmol/L)] compared with omnivorous diets. However, in the same review, vegan diets ($N = 1175$) yielded no difference in HDL cholesterol compared with omnivorous diets [-1.54 mg/dL (-0.04 mmol/L); 95% confidence interval (CI): -2.96 , -0.12 , $P = 0.61$] [43]. It should be noted that the reported 95% CI does not cross null (which would imply $P < 0.05$), but the authors explicitly state $P = 0.61$ and describe the result in the text as “nonsignificant lower HDL cholesterol.” This internal consistency is almost certainly a typographical error; nonetheless, we report their stated nonsignificance. Zhang et al. [44] further found no significant difference in HDL cholesterol [standard mean difference = 0.8 mg/dL (-0.02 mmol/L); 95% CI: -7.3 , $+8.5$ mg/dL (-0.19 , 0.22 mmol/L)] between omnivorous diets and vegetarian diets in a meta-analysis of 12 cross-sectional and cohort studies with freely living adults (≥ 18 y, $N = 4177$).

Intervention studies have also yielded mixed findings. In a meta-analysis of randomized controlled trials (RCTs), Wang et al. [45] found that vegetarian diets (including vegan, ovovegetarian, and lactoovovegetarian), lasting 3–74 wk, reduced HDL cholesterol by -3.9 mg/dL [-0.10 mmol/L; 95% CI: -5.4 , -2.3 mg/dL (-0.14 , -0.06 mmol/L)] compared with omnivore diets among both healthy individuals and those with chronic illnesses ($N = 832$, including diet-related conditions, overweight, and obesity). Furthermore, Yokoyama et al. [18] assessed data from 19 clinical trials ($N = 1484$) in which participants followed vegetarian diets for ≥ 4 wk, which were associated with a modest reduction in HDL cholesterol of -2.4 mg/dL (-0.09 mmol/L).

However, additional intervention evidence suggests no effect of vegetarian and vegan diets on HDL cholesterol. For example, in an umbrella review of meta-analyses of RCTs on popular diets, Dinu et al. [46] reported no difference in HDL cholesterol [mean difference (MD) = -1.2 mg/dL (-0.03 mmol/L); 95% CI: -3.1 , $+0.8$ mg/dL (-0.08 , 0.02 mmol/L)] between vegetarian ($N =$

329) and nonvegetarian ($N = 337$) diets. The authors noted substantial variability and a lack of specificity in how control and vegetarian diets were defined across studies: some meta-analyses grouped lactoovovegetarian and vegan diets together (or labeled them collectively as “plant-based”), whereas others evaluated them separately, and there was also noted heterogeneity in the definition of the nonvegetarian control diets [46]. In a second umbrella review of experimental studies, Chew et al. [40] found a small, nonsignificant reduction in HDL cholesterol [MD = -1.5 mg/dL (-0.04 mmol/L); 95% CI: -3.1 , 0.0 mg/dL (-0.08 , 0.00 mmol/L)] when comparing plant-based diets (including vegetarian, vegan, lactoovovegetarian, pescovegetarian, and semi-vegetarian) to non-plant-based diets.

Plant-based diets compared with no intervention or control

Plant-based diets are generally associated with small but statistically significant decreases in HDL cholesterol when compared with minimal intervention. For example, in a network meta-analysis of 48 RCTs among participants with type 2 diabetes, with interventions lasting ≥ 12 wk, vegetarian and vegan diets significantly lowered HDL cholesterol [MD = -2.7 mg/dL (-0.07 mmol/L); 95% CI: -5.4 , -0.4 mg/dL (-0.14 , -0.01 mmol/L)] compared with no or minimal intervention ($N = 5360$) [47]. Meta-analyses comparing vegan diets with usual care or minimal intervention have similarly reported small reductions in HDL cholesterol in a general healthy population [MD = -3.1 mg/dL (-0.08 mmol/L); 95% CI: -4.3 , -1.5 mg/dL (-0.11 , -0.04 mmol/L)] [41] and for primary CVD prevention [MD = -3.1 mg/dL (-0.08 mmol/L); 95% CI: -4.3 , -1.5 mg/dL (-0.11 , -0.04 mmol/L), $N = 449$ participants] [48].

Plant-based diets compared with other therapeutic diets

Although plant-based diets are generally associated with small decreases in HDL cholesterol when compared with usual care or minimal intervention, they often yield no difference in comparison with other therapeutic diet interventions. For example, in a meta-analysis of 9 RCTs ($N = 664$) among individuals with type 2 diabetes, of which 8 reported HDL cholesterol data ($N = 632$), Vigiouliouk et al. [49] found no significant difference in HDL cholesterol (MD = -1.2 mg/dL (-0.03 mmol/L); 95% CI: -3.1 , $+0.8$ mg/dL (-0.08 , 0.02 mmol/L)] when comparing vegetarian dietary patterns (primarily vegan and low-fat vegan diets, with some lactovegetarian and plant-based protein variants) diets to various control diets (including conventional diabetes diets, animal-based protein diets, usual care, and portion-controlled diets). Similarly, among participants with overweight or obesity, meta-analysis results from Xu et al. [50] report no significant change in HDL cholesterol [MD = -5.8 mg/dL (-0.15 mmol/L); 95% CI: -11.2 , 0.0 mg/dL (-0.29 , 0.00 mmol/L)] when comparing various vegetarian diets (lactoovovegetarian, low-fat vegan, low-calorie vegetarian, and whole-food plant-based) compared with control diets (including Mediterranean, usual care, calorie- and fat-restricted, and meat-based) across 6 studies ($N = 724$).

Experimental studies examining vegan diets yield similar conclusions: in a meta-analysis of 5 studies among people with type 2 diabetes ($N = 342$), Kashyap et al. [51] found no

difference in HDL cholesterol [MD = +0.4 mg/dL (0.01 mmol/L); 95% CI: −7.7 mg/dL, +8.5 mg/dL (−0.20, 0.22 mmol/L)] between vegan diets and conventional diabetes diets (based on American or Korean Diabetes Association guidelines). In addition, for primary prevention of CVD among the general population and those at increased risk of CVD ($N = 256$), vegan diets yielded no difference in HDL cholesterol compared with various other dietary interventions [MD = −0.4 mg/dL (−0.01 mmol/L); 95% CI: −3.1 mg/dL, +1.9 mg/dL (−0.08, 0.05 mmol/L)] [48]. Finally, a meta-analysis of 9 studies ($N = 698$) reported that among adults with overweight, type 2 diabetes, or prediabetes, vegan diets yielded no difference in HDL cholesterol [MD = −2.3 mg/dL (−0.06 mmol/L); 95% CI: −4.6 mg/dL, +0.4 mg/dL (−0.12, 0.01 mmol/L)] between vegan diets and control diets (including American/Korean Diabetes Association guidelines with energy deficit, Mediterranean, and high-carbohydrate lactoovovegetarian diets) [52].

Exploring the reported discrepancies in direction of findings

Original research, systematic reviews, meta-analyses, and umbrella reviews have yielded heterogeneous findings regarding the association between plant-based diets and HDL cholesterol. We propose several potential reasons for these discrepancies.

First, definitions of what constitutes “plant-based” vary widely in the literature, including discrepancies on definitions of subtypes such as “vegetarian.” Some review group vegan through semivegetarian patterns together as an exposure [18], whereas others examine a single subtype in isolation (e.g., vegan diets only) [41,48,51,52]. However, even within consistent diets and definitions, diet composition can differ. For example, Termannsen et al. [52] included low-fat vegan diets ad libitum [53–58], a low-carbohydrate vegan diet with 40% energy restriction [59], and other vegan diets ad libitum—all vegan diets with markedly different caloric, macronutrient, and micronutrient composition [60]. Notably, although some studies include lactoovovegetarian and semivegetarian diets as “vegetarian” diets, other reviews place similar diets in the control group. Control groups also vary widely, ranging from Mediterranean diets [61,62], American and Korean Diabetes Association guidelines [58,60], meat-based diets [63], low-fat diets [56], calorie- and fat-restricted diets [64], usual care [54,55], and high-carbohydrate lactoovovegetarian diets [59]. Some studies do not define the control diet used [46]. In an attempt to quantify the extent of this heterogeneity, Supplemental Table 1 disaggregates the 236 primary studies cited in this review (including those within all cited meta-analyses, umbrella reviews, and narrative reviews) and extracts the plant-based diets

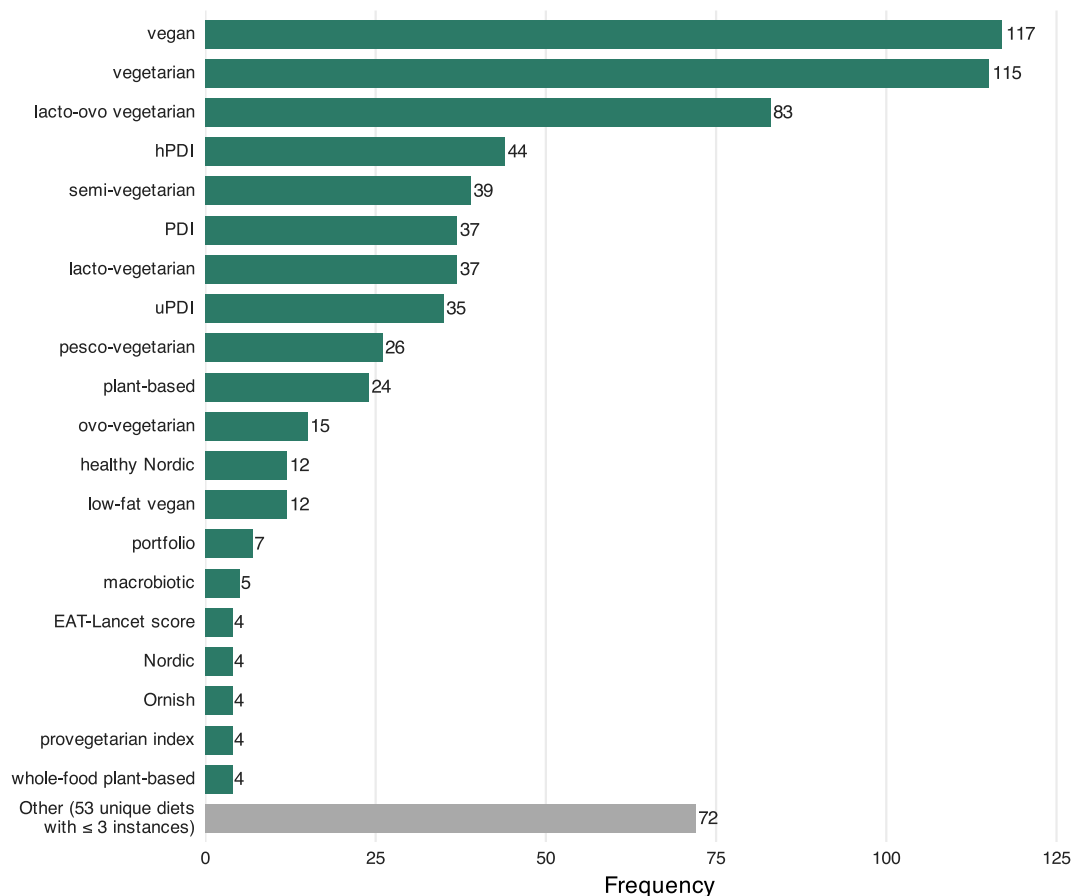


FIGURE 1. Frequency of plant-based diet definitions across the cited evidence base. Bars show extracted diet definition instances, not necessarily mutually exclusive studies (i.e., 1 study could contribute >1 dietary exposure). “Other” includes 72 instances across 53 unique diets with ≤3 instances each. A full catalog of the studies included in this plot can be found in Supplemental Table 1. This table represents only studies cited in this narrative review, including those within cited meta-analyses, umbrella reviews, and narrative reviews. Engage Act Transform (EAT)-Lancet; hPDI, healthful plant-based diet index; PDI, plant-based diet index; uPDI, unhealthful plant-based diet index.

used and their respective definitions. [Figure 1](#) demonstrates substantial definitional dispersion across the evidence base. Although vegan and vegetarian were most frequent, the literature also included various plant-based diet indices: semi-vegetarian, lactovegetarian, ovovegetarian, lactoovovegetarian, and pescovegetarian patterns, as well as plant-based indices including the plant-based diet index (PDI), unhealthful PDI, and healthful PDI; low-fat and other macronutrient specifications; and numerous low-frequency or study-specific classifications. This long tail of dietary labels underscores that pooled estimates for “plant-based,” “vegetarian,” or “vegan” diets often combine meaningfully different exposures, biasing associations or limiting comparability across studies and complicating interpretation of findings.

Plant-based diet subtypes are frequently aggregated for the purpose of evidence synthesis, yet they may markedly differ in composition, with individual foods impacting HDL cholesterol concentrations in varying directions and magnitudes. For example, lactoovovegetarian diets often include eggs, which are associated with higher HDL cholesterol [65,66], although not consistently [67]. Pescatarian diets may include fatty fish, which have been shown to increase HDL cholesterol, likely because of their eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) contents [68,69]. Fatty fish is not typically included in other plant-based eating patterns, potentially explaining inconsistencies in plant-based diet-related HDL cholesterol trends among studies with aggregated plant-based diet exposures. In a similar vein, self-report dietary data are subject to measurement bias, both generally [70] and specific to vegetarian identification. For example, Vinnari et al. [71] found that 80% of self-identified vegetarians did not meet the vegetarian definition based on reported consumption behaviors. Similarly, Gilsing et al. [72] found that 50% of self-reported vegetarians also reported meat or fish intake in food frequency questionnaires. Such misclassification further complicates efforts to define consistent exposures when examining associations between plant-based dietary patterns and HDL cholesterol.

Review papers including only vegan diets generally associate them with decreases in HDL cholesterol compared with no intervention [41,47,48], potentially providing a clearer examination of associations with strictly plant-based dietary patterns, given their more uniform exclusion of all animal-source foods. Still, vegan diets vary in composition, as noted above, and may include or exclude foods that impact HDL cholesterol concentrations: for example, soy foods [73] and alcohol [68,69] associate with increased HDL cholesterol, refined sugar with decreased HDL cholesterol [68,69]. Individual consumption of foods is typically not specified in explorations of dietary pattern consumption and HDL cholesterol, pointing to challenges in the assessment of the impacts of eating patterns that may be compositionally diverse among those who follow them.

Other types of heterogeneity may also contribute to mixed associations. Duration of exposure differs: observational reviews often require 1 to 2 y on the diet for inclusion [18,44], whereas interventions range from 2 wk to over 2 y [40,50]. Medication reporting also varies, with some original research studies and review papers documenting lipid-lowering, antihypertensive, or diabetes medication use and dosing changes [18,45,47,49,52,55,74,75]. Others did not mention or address medication use [1,4,41,43,44,46,50]. Given that some of these pharmacological

therapies can impact HDL cholesterol [76,77], variations in assessing and accounting for medication use may further explain heterogeneity, although 1 review that stratified by lipid-lowering medication use found no difference in HDL cholesterol [45]. Finally, study populations varied substantially, ranging from healthy adults [2,41,43,45], general populations [44,51], to individuals with diabetes [47], overweight and obesity [50], and mixed populations that included those with chronic diseases [45,52], further complicating comparisons across reviews.

Potential Mechanisms for HDL Reduction with Plant-Based Diets

The most prominent explanation for plant-based diet-mediated HDL cholesterol reduction is macronutrient substitution. Controlled feeding meta-analyses show that replacing carbohydrates with fat raises HDL cholesterol, whereas replacing fat with carbohydrate lowers HDL cholesterol and tends to raise triglycerides; in Mensink et al.'s [78] meta-analysis of 60 controlled trials, replacing 1% of energy from carbohydrate with saturated fat, monounsaturated fat, and polyunsaturated fat increased HDL cholesterol by ~0.47, 0.34, and 0.28 mg/dL, respectively. Mechanistic tracer data support this interpretation: in an isocaloric trial, a low-fat, low-saturated-fat, low-cholesterol diet reduced HDL cholesterol by 15%, driven primarily by an ~8% reduction in hepatic secretion of apolipoprotein A-I (apoA-I), the principal structural protein of HDL comprising ~70% of its protein mass [79,80]. This reduction in apoA-I secretion occurred without a change in the catabolic rate, indicating that lower serum HDL cholesterol reflects decreased HDL particle production rather than increased clearance. No subsequent studies have definitively established the molecular basis for this reduced apoA-I secretion; Vélez-Carrasco et al. [80] speculated that lower dietary fat and cholesterol may reduce the systemic requirement for HDL-mediated cholesterol transport, thereby downregulating apoA-I secretion, although they could not disentangle independent contributions of dietary components.

A parallel mechanism occurs as higher-carbohydrate, lower-fat diets promote cholesteryl ester transfer protein-mediated exchange, in which cholesteryl esters are transferred from HDL to triglyceride-rich lipoproteins in exchange for triglycerides [81]. The resulting triglyceride-enriched HDL particles are preferred substrates for hepatic lipase. This generates smaller, denser HDL3 particles and promotes apoA-I dissociation from the HDL particle, accelerating its clearance from circulation [82,83]. HDL3-associated apoA-I may also exhibit antioxidant properties through methionine residues in its structure, which reduce reactive lipid hydroperoxides to inactive (i.e., less harmful) hydroxides, thereby interrupting damaging lipid peroxidation chain reactions [84]. Consequently, the smaller, triglyceride-rich particles generated under these conditions may have altered functional capacity; although some evidence suggests that HDL3 subtypes can retain cholesterol efflux activity, many studies indicate that triglyceride enrichment and the associated remodeling often impair antiatherogenic properties (including reduced antioxidant and anti-inflammatory functions) compared with larger, buoyant HDL2 particles, which are generally more effective in mediating reverse cholesterol

transport and protecting LDL from oxidative modification [84]. These apoA-I-related mechanisms reinforce that lower serum HDL cholesterol with plant-based diets reflects changes in both HDL particle quantity and quality.

HDL responses observed in dietary interventions are also often confounded by concurrent shorter-term weight loss. In the Eco-Atkins trial ($N = 47$), where 2 markedly different macronutrient variations of vegetarian diets (low carbohydrate, high fat compared with high carbohydrate, low fat), produced equivalent decreases in HDL cholesterol despite comparable weight loss under energy restriction across 4 wk, suggesting that energy deficit may obscure macronutrient-specific effects [85]. Further showing that weight loss may mask this association, the Complete Health Improvement Program cohort followed 5046 participants over 30 d while encouraging an ad libitum low-fat, plant-based diet, reporting a 8.7% decrease in HDL cholesterol alongside a 3.2% reduction in BMI [86]. They also reported that although a total of 323 patients resolved baseline-classified metabolic syndrome, 112 participants acquired metabolic syndrome resulting from reduced HDL cholesterol [86]. Short-term reductions in HDL cholesterol warrant cautious interpretation in the context of long-term steady states: in a meta-analysis of 73 RCTs with ≥ 6 to 12 mo follow-up, sustained weight loss was associated with increases in HDL cholesterol of ~ 0.46 mg/dL per kg lost ($N = 32,496$), corresponding to ~ 2 to 5 mg/dL increases with typical weight reductions (~ 10 – 20 kg) [87]. These findings suggest that HDL cholesterol responses to dietary interventions may be nonlinear and temporally dynamic, with short-term changes during ongoing weight loss differing from longer-term effects.

Other mechanisms likely play secondary roles or are less documented than weight and macronutrient pathways. For example, in the Prevention of Cardiovascular Disease With Med or Veg Diets study, 30 participants were enrolled in a randomized cross-over trial comparing the effects of a Mediterranean compared with a vegetarian dietary pattern; despite similar serum HDL cholesterol concentrations, the vegetarian dietary pattern reduced CEC by $\sim 9\%$ relative to the Mediterranean dietary pattern, indicating potential differences in HDL functionality independent of serum concentration [88]. By contrast, other characteristic components of plant-based diets, including higher soluble fiber and phytosterol intake, appear to have minimal direct effects on HDL cholesterol; although both substantially lower LDL cholesterol, meta-analyses show little to no change in HDL cholesterol when these components are studied in isolation [89,90]. Notably, HDL function can improve independent of serum HDL cholesterol concentration: in a randomized cross-over trial, polyphenol-rich olive oil increased CEC and improved HDL particle composition (larger, triglyceride-poor particles) without changing HDL cholesterol concentrations, further demonstrating that qualitative improvements in HDL may not be fully represented by HDL cholesterol alone [91].

Collectively, these findings support a conceptual framework (Figure 2): plant-based diet composition may reduce atherogenic lipid burden while producing context-dependent HDL cholesterol changes, such that modest HDL cholesterol reductions should be interpreted within the broader cardiometabolic risk profile, rather than as evidence of impaired cardiometabolic health.

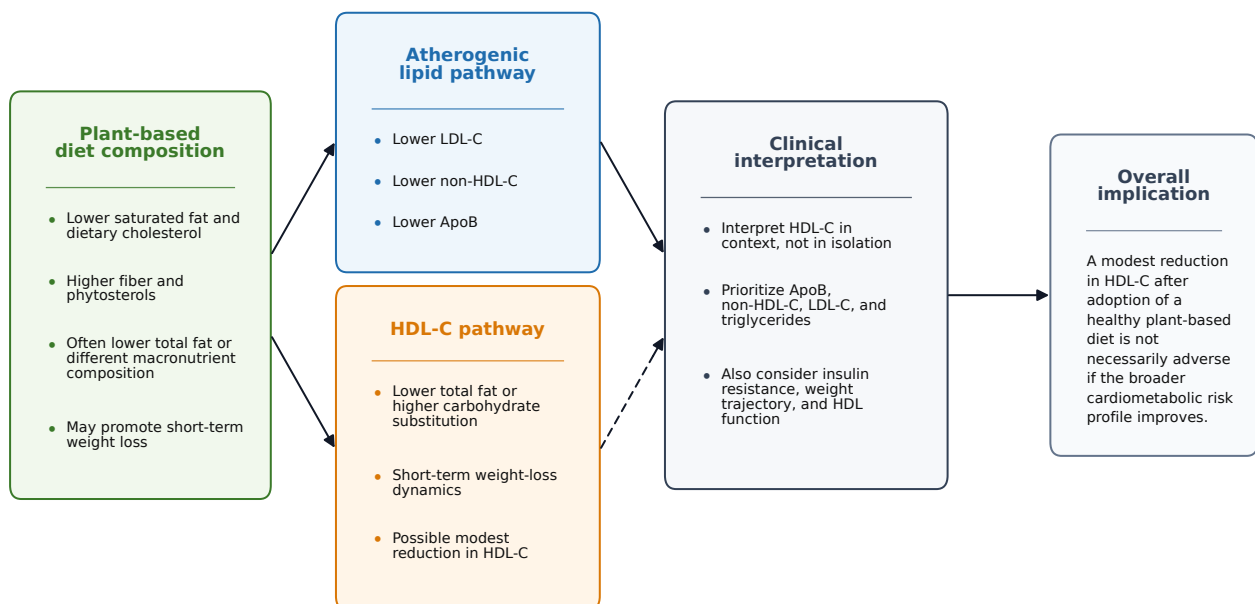


FIGURE 2. Conceptual pathways linking plant-based diets, HDL cholesterol, and cardiometabolic risk. This figure summarizes plausible pathways through which the adoption of a healthy plant-based dietary pattern may influence lipid markers and overall cardiometabolic risk. Plant-based diets may improve atherogenic lipid markers through lower saturated fat and dietary cholesterol intake, higher fiber and phytosterol intake, and changes in body weight. Modest reductions in HDL cholesterol may occur in some settings, particularly with lower total fat intake, higher-carbohydrate substitution, or short-term weight loss, but should not be interpreted in isolation. Clinical interpretation should prioritize apoB, non-HDL cholesterol, LDL cholesterol, triglycerides, insulin resistance, weight trajectory, and, where available, markers of HDL function. The dashed arrow indicates that lower HDL cholesterol may require contextual interpretation rather than implying an adverse effect. apoB, apolipoprotein B.

Clinical Practice Implications

Appropriately planned vegan and vegetarian diets are nutritionally adequate across the life span [92] and are endorsed by major professional organizations for cardiometabolic risk reduction. For example, the Academy of Nutrition and Dietetics endorses all vegetarian diets (including vegan diets) as having a “likely benefit” regarding CVD and CAD incidence in general healthy adult populations [93]. In addition, the 2026 American College of Cardiology (ACC)/American Heart Association (AHA) Guidelines on the Management of Dyslipidemia endorse predominantly plant-based dietary patterns (including vegetarian and vegan) as having the strongest evidence for lowering LDL cholesterol and reducing atherosclerotic CVD (ASCVD) risk, with additional benefits for apolipoprotein B (apoB), blood pressure, glycemic control, and body weight management [94].

Vegetarian and vegan diets have been associated with reduced CVD incidence [relative risk (RR): 0.85 (0.79, 0.92)] and CVD mortality [hazard ratio: 0.92 (0.85, 0.99)] among healthy adults when compared with nonvegetarian diets [2]. In addition to cardiovascular benefits, plant-based diets are associated with reductions in risk of certain cancers and type 2 diabetes, and, among those with type 2 diabetes, reductions in hemoglobin A1c, decreased need for diabetic medications, and improved insulin sensitivity [4,75].

Because of the lack of causality established between HDL cholesterol and risk of CVD, and the lack of evidence to show that therapeutic interventions targeting HDL cholesterol are efficacious in producing outcome-related CVD benefits, evidence to support the use of HDL cholesterol as a therapeutic target is limited [95]. In fact, some practice guidelines eschew specific clinical recommendations related to HDL cholesterol [96,97]. The modest HDL cholesterol reductions (~2–4 mg/dL, when present) observed with plant-based diets do not appear to attenuate their net benefits, particularly in the context of the clinically significant improvements in LDL cholesterol, inflammation, and insulin sensitivity. This reduction may be largely explained by physiologic macronutrient substitution (i.e., lower saturated fat and cholesterol, higher-carbohydrate intake) and transient effects of caloric deficit or weight loss rather than intrinsic impairment of HDL particle function. Therefore, we suggest that modest decreases in HDL cholesterol should not justify discouragement of otherwise health-supporting, healthy plant-based dietary patterns.

There is insufficient evidence to support dietary interventions targeting increased HDL cholesterol [96]. We suggest providers follow approaches outlined by the Academy of Nutrition and Dietetics and the ACC/AHA that emphasize a health-promoting approach to plant-based diets that maximize their potential health benefits and minimize their potential risks, when appropriate and in line with patient goals, values, preferences, and cultural or religious needs. Furthermore, we do emphasize targeted lowering of LDL cholesterol because it remains a clinically relevant biomarker with causally established links to ASCVD risk, based on genetic studies, Mendelian randomization studies, epidemiological prospective cohort studies, and RCTs of LDL cholesterol-reducing therapies [95, 98]. Examples of how to support health-promoting plant-based diets in clinical practice, including recommendations for those

at risk of CVD, are outlined below, although they should not replace clinical judgment.

Practical, evidence-based recommendations for clinicians and providers:

- *Emphasizing diet quality and nutritional adequacy.* Counsel patients to prioritize whole, minimally processed foods (vegetables, legumes, nuts, seeds, whole grains), with inclusion of polyphenol-rich foods (berries, nuts) and sources of soluble fiber (oats, beans).
- *Limit unhealthy plant-based foods.* Advise minimizing ultra-processed foods high in sodium, added sugars, refined grains, trans fats, and tropical oils (e.g., sweets and sugar-sweetened beverages), because diets rich in these foods are associated with increased risk of dyslipidemia, insulin resistance, obesity, type 2 diabetes, and CVD [5].
- *Monitor and address nutrient adequacy.* Plant-based diets can increase the risk of deficiencies in certain nutrients, including vitamin B-12, iron, iodine, choline, vitamin D, and (for vegan diets specifically) calcium; routinely evaluate and monitor these nutrients [93].
- *Optimize for dyslipidemia risk reduction.* For patients with or at risk of dyslipidemia, encourage the incorporation of additional fiber, nuts, and soy protein, along with the replacement of saturated fat with unsaturated fats to further lower LDL cholesterol [94].
- *Incorporate complementary lifestyle behaviors.* Recommend ≥150 min/wk of moderate-intensity aerobic exercise, which may increase HDL cholesterol and improve HDL functionality in addition to other cardiometabolic benefits [99]. The 2026 ACC/AHA Guidelines also endorse healthy sleep habits (7–9 h of quality sleep per night) as part of broader lifestyle support for cardiometabolic health [94].
- *Engage cross-disciplinary collaboration.* Integrate registered dietitian nutritionists (RDNs) and nutrition and dietetics technicians, registered as part of the care team, to provide nutrition counseling and education. RDNs improve management of dyslipidemia [100,101] and cardiometabolic outcomes among adults with overweight and obesity [102].

Opportunities to Advance Understanding of HDL Cholesterol and Plant-Based Diets

Existing evidence suggests that plant-based dietary patterns improve several markers of cardiometabolic risk (e.g., LDL cholesterol), yet the clinical interpretation of small reductions in HDL cholesterol remains unclear because few studies have concurrently assessed whether these changes occur alongside reduced atherogenic particle burden or altered HDL function. Future trials should therefore treat HDL cholesterol as an incomplete metric rather than a sufficient endpoint and incorporate broader lipid and HDL-related outcomes, when available, including apoB, LDL particle number, HDL particle number and size, apoA-I, CEC, and validated measures of HDL anti-inflammatory or antioxidant function.

Methodological heterogeneity also remains a major limitation. Studies vary in the definition of plant-based diets, what comparator diet is used, and whether confounding factors are controlled for, such as weight change, macronutrient

composition, and caloric deficit. Future studies should distinguish between plant-based diet subtypes (e.g., vegan and lacto-ovo-vegetarian), differentiate whole food from ultraprocessed, and furthermore consider the comparison of macronutrient and food composition associated with changes in HDL cholesterol. In addition, when tested, nutrient substitutions should be specified. Improved reporting and harmonization among interventions and outcomes would strengthen comparability across trials.

Furthermore, there is a lack of clarity regarding the types of HDL cholesterol particles generated from plant-based diet consumption; for example, it is not fully understood whether plant-based diets result in atherogenic, antiatherogenic HDL cholesterol particles, or both. Understanding HDL cholesterol composition associated with dietary changes may offer a better indication of the health impacts associated with such changes. To further elucidate the role of dietary change and HDL cholesterol, short-duration mechanistic feeding trials may be needed to determine whether reductions in HDL cholesterol reflect changes in apoA-I production, HDL remodeling, or reverse cholesterol transport. In contrast, longer free-living randomized trials could be employed to assess adherence, medication interactions, and whether HDL-related associations occur alongside overall cardiometabolic risk. Such studies would also be able to examine whether socioeconomic context and food access interact with dietary composition and intervention adherence, influencing HDL-related outcomes. The deliberate inclusion of diverse populations, particularly groups underrepresented in previous trials, would further bolster the evidence and aid in investigating diet–HDL effect modification by sex, metabolic status, and life stage.

In conclusion, plant-based diets can be nutritionally adequate and health promoting, with beneficial associations for cardiometabolic outcomes, particularly through reductions in LDL cholesterol and other markers of atherogenic risk. Seemingly paradoxical to their health benefits, some evidence associates modest decreases in HDL cholesterol when compared with omnivorous diets or no intervention. However, inconsistency observed across study designs, coupled with evidence suggesting null effects or no difference compared with other therapeutic diets, suggests caution when interpreting these findings in isolation. Presently, evidence does not support using HDL cholesterol as an independent therapeutic target, and HDL cholesterol concentration alone cannot determine whether observed changes represent physiologic harm or neutrality. Furthermore, the magnitude of HDL cholesterol reduction is typically minimal in comparison with concurrent reductions in LDL cholesterol, which has established causality for lowered ASCVD risk. As such, we advise cautious interpretation of reductions in HDL cholesterol, while considering the context of a patient's overall cardiometabolic risk profile. Working with patients interested in following plant-based diets to ensure nutrition adequacy, balanced composition, and whole food-forward approaches will maximize their health benefits while minimizing risks.

Author contributions

The authors' responsibilities were as follows – KB: conceptualized the review; all authors: performed the literature review and evidence synthesis, collaboratively wrote the manuscript,

critically reviewed and revised all materials, and approved the final version.

Declaration of Generative AI and AI-assisted technologies in the writing process

The authors declare that no generative AI or AI-assisted technologies were used in the writing of this manuscript.

Conflict of interest

The authors declare no conflicts of interest.

Funding

The authors reported no funding received for this study.

Data availability

As a narrative review paper, no new data were analyzed during the writing of this paper; thus, data sharing is not applicable.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.advnut.2026.100708>.

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