

Anti-Inflammatory Effects of a Vegan Diet Versus the American Heart Association–Recommended Diet in Coronary Artery Disease Trial

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Background—Dietary interventions may play a role in secondary cardiovascular prevention. hsCRP (High-sensitivity C-reactive protein) is a marker of risk for major adverse cardiovascular outcomes in coronary artery disease.

Methods and Results—The open-label, blinded end-point, EVADE CAD (Effects of a Vegan Versus the American Heart Association–Recommended Diet in Coronary Artery Disease) trial randomized participants (n=100) with coronary artery disease to 8 weeks of a vegan or American Heart Association–recommended diet with provision of groceries, tools to measure dietary intake, and dietary counseling. The primary end point was high-sensitivity C-reactive protein. A linear regression model compared end points after 8 weeks of a vegan versus American Heart Association diet and adjusted for baseline concentration of the end point. Significance levels for the primary and secondary end points were set at 0.05 and 0.0015, respectively. A vegan diet resulted in a significant 32% lower high-sensitivity C-reactive protein (β , 0.68, 95% confidence interval [0.49–0.94]; $P=0.02$) when compared with the American Heart Association diet. Results were consistent after adjustment for age, race, baseline waist circumference, diabetes mellitus, and prior myocardial infarction (adjusted β , 0.67 [0.47–0.94], $P=0.02$). The degree of reduction in body mass index and waist circumference did not significantly differ between the 2 diet groups (adjusted β , 0.99 [0.97–1.00], $P=0.10$; and adjusted β , 1.00 [0.98–1.01], $P=0.66$, respectively). There were also no significant differences in markers of glycemic control between the 2 diet groups. There was a nonsignificant 13% reduction in low-density lipoprotein cholesterol with the vegan diet when compared with the American Heart Association diet (adjusted β , 0.87 [0.78–0.97], $P=0.01$). There were no significant differences in other lipid parameters.

Conclusions—In patients with coronary artery disease on guideline-directed medical therapy, a vegan diet may be considered to lower high-sensitivity C-reactive protein as a risk marker of adverse outcomes.

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Inflammation plays a central role in the progression of atherosclerosis and is associated with adverse cardiovascular events.^{1–6} Targeted anti-inflammatory therapy and

reductions in hsCRP (high sensitivity C-reactive protein) have been shown to reduce major adverse cardiovascular events in patients with established coronary artery disease (CAD).^{7–9} Approximately one third of patients with stable CAD have an hsCRP above the upper limit of normal, and there is a complex interplay between inflammation and glucometabolic disease that may further exacerbate cardiovascular risk.^{10–18} Furthermore, up to 45% of patients will continue to have elevated hsCRP and thus residual risk despite anti-inflammatory therapy.⁷ Therefore, investigation of additional strategies to address the residual risk associated with inflammation in these patients are warranted.

The American Heart Association's (AHA) strategy to reduce the health burden associated with CAD has a focus on a healthy diet and lifestyle.¹⁹ Although multiple dietary factors influence CAD, few studies examine a particular diet compared with the

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Clinical Perspective

What Is New?

- In a patient with coronary artery disease on guideline-directed medical therapy, a plant-based vegan diet may be an adjunctive treatment to lower high-sensitivity C-reactive protein.
- A vegan diet does not appear to provide greater benefit when compared with the American Heart Association–recommended diet in terms of weight loss, glycemic control, or dyslipidemia.

What Are the Clinical Implications?

- A vegan diet may be used to lower inflammation as measured by high-sensitivity C-reactive protein, a key player in the development of major adverse cardiovascular events.

standard recommended heart-healthy diet in patients with CAD.^{20–22} A plant-based vegan diet has been shown to significantly reduce adverse markers of poor cardiovascular health over time, but limited data include a heart-healthy diet as the comparator arm in patients receiving guideline-directed medical therapy.^{23–26} Lack of the same support resources in the intervention and control groups have confounded prior direct comparisons of the diets alone. Furthermore, several prior studies of a vegan diet provided full meals, limiting the generalizability of the results to patients in their home environment who do not have access to prepared meals.^{23,24} In sum, data on the effects of dietary strategies on systemic inflammation incremental to medical therapy are limited.

The aim of this study was to determine the effects of a vegan versus AHA-recommended diet on hsCRP, as well as other markers of inflammation, glucometabolic markers, and lipid profiles in patients with established CAD on guideline-directed medical therapy.

Methods

The data, analytic methods, and study materials will not be made available to other researchers for purposes of reproducing the results or replicating the procedure, as additional analyses may be forthcoming.

Study Cohort

The EVADE CAD (Effects of a Vegan Versus the American Heart Association-Recommended Diet in Coronary Artery Disease) trial design and rationale have been described in detail.²⁷ Briefly, between March 11, 2014, and February 2, 2017, 100 participants from New York University Langone Medical Center with a history of angiographically defined CAD ($\geq 50\%$ lesion in

an artery with ≥ 2 -mm caliber) underwent 1:1 randomization to either a vegan diet or the AHA-recommended diet. Major exclusion criteria included (1) history of an eating disorder; (2) already on a vegetarian or vegan diet; (3) use of steroids or nonsteroidal anti-inflammatory medications other than aspirin; (4) history of a myocardial infarction or coronary artery bypass graft surgery within the preceding 3 months; (5) presence of infection within the preceding 3 months; and (6) have a planned staged coronary revascularization or other surgical procedure during study period. Potential participants were also excluded if they had a score of >4 on any of the amotivational items or if the relative autonomy index (defined as average of answers for the 6 autonomous items—average of answers for the 6 controlled items) was ≤ 0 on a treatment self-regulation questionnaire²⁸ (Figure 1).

The New York University School of Medicine Institutional Review Board approved the study, and all participants provided written informed consent. The trial is registered at clinicaltrials.gov (NCT02135939). The Purjes Foundation (Salt Lake City, UT) was the primary sponsor of the trial and did not contribute to the study design or data analysis.

Study Design

This trial utilized a prospective, randomized, open-label, blinded end point study design.²⁹ A 1-to-1 randomization sequence was computer generated by the study biostatistician. Only the biostatistician and a member of the cardiovascular clinical research center, who was not a member of the study team, had access to this sequence and provided the dietary assignment via e-mail after the participant provided written informed consent. Participants were randomized at least 7 days after invasive coronary angiography so that medication regimens would be as stable as possible during the study period. The active study duration was 8 weeks, with an interim visit at 4 weeks and a final visit at 8 weeks. Between visits, participants in both treatment groups had access to the study's registered dietitian by telephone and e-mail, and a 24-hour dietary recall was performed twice a week on random days. In addition, participants were instructed to fill out a 4-day food record during the 1 week before each of the 3 visits. Groceries that supported the dietary recommendations of the intervention assignment were provided on a weekly basis, along with a cookbook (Simply Vegan, Baltimore: Vegetarian Resource Group, 2012; or AHA Low-Fat, Low-Cholesterol Cookbook, New York: Clarkson Potter, 2008), handouts from the *Nutrition Care Manual* of the Academy of Nutrition and Dietetics (www.eatright.org), a 2-week sample menu, food scale, measuring spoons, and measuring cups. As previously described, the dietary intervention was kept as similar as possible, with only substitution of animal-based protein for plant-based protein on handouts, grocery menus, 2-week sample menus, and

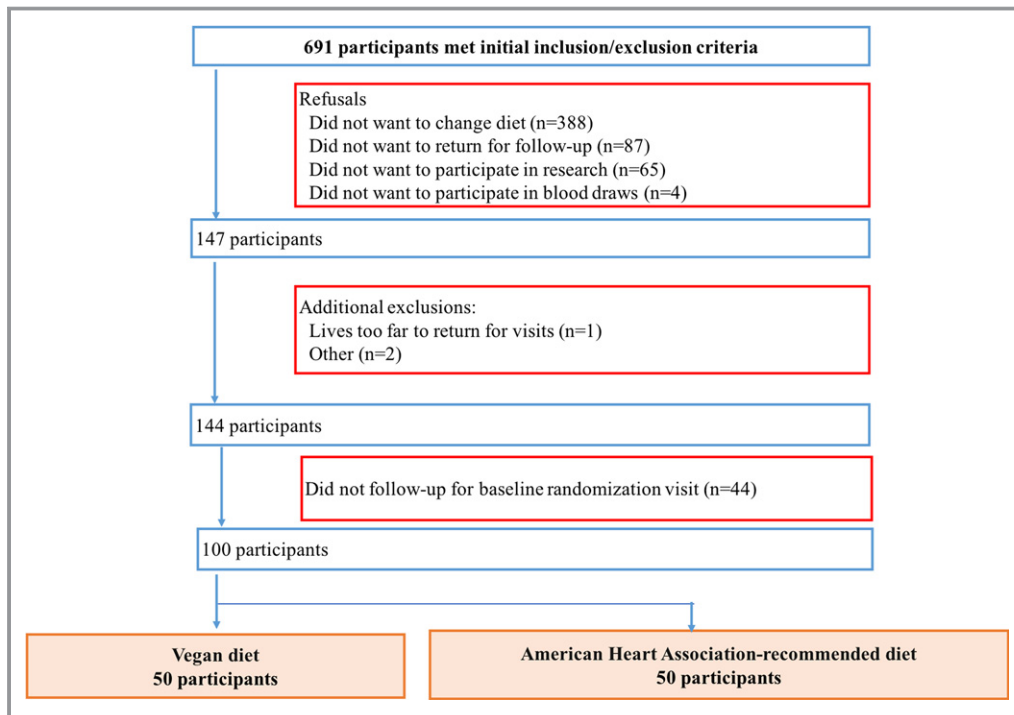


Figure 1. Study cohort.

recipes.²⁷ Physical activity was assessed at each of the 3 study visits using the International Physical Activity Questionnaire-Short Form questionnaire.³⁰

Measures of Dietary Adherence

Adherence was assessed each week using the information collected during the two 24-hour dietary recalls as previously described.²⁷ Briefly, the following adherence method was developed with the aim to use a practical and quantifiable way of capturing animal protein in a diet using similar criteria for both groups. Furthermore, the data to evaluate adherence were obtained in the same manner in both groups. A script was developed by the study registered dietitian to allow for consistent interactions with the study participants and reduce bias throughout the study. Participants in the vegan diet group received 1 point for abstinence from each of the following: (1) meat/poultry/eggs, (2) dairy, and (3) seafood. Participants in the vegan diet group could earn up to 3 points on each of the two 24-hour dietary recalls, and a score of 5 to 6 was defined as adherent to the vegan diet for that week. Participants in the AHA-recommended diet group received 1 point for consumption of each of the following: (1) ≤ 5 oz of animal protein/day, (2) only low-fat/fat-free dairy if dairy was consumed, (3) fish ≥ 2 times/week. A score of 4 to 5 defined adherence to the AHA-recommended diet for that week. Of note, if a participant in the AHA group did not report fish intake on both dietary recalls, participants were asked if they had been eating fish at least 2 times per week

at the end of the second 24-hour dietary recall. Participants were determined to be adherent at the 4-week interim and 8-week final follow-up visits if they were adherent for at least 2 of the 3 weeks evaluated between visits. To better examine whether the dietary intakes of the participants were in the spirit of the guidelines, MyPlate servings of fruits, vegetables, and whole grains were also determined in each diet group using Food Processor 11.0.137 (ESHA Research, Salem, OR).

End Points

The primary end point was hsCRP concentration. Secondary end points included the following inflammatory markers: white blood cell count and subtypes (neutrophil/lymphocyte ratio and monocyte subtypes), white blood cell cellular adhesion molecules (neutrophil-surface expression of L-selectin to CD11b ratio, soluble L-selectin, soluble E-selectin, intracellular adhesion molecule, vascular cellular adhesion molecule), other markers of neutrophil activity (neutrophil gelatinase-associated lipocalin, myeloperoxidase), and urine F2-isoprostane/creatinine ratio.

Other secondary end points included anthropometric data (body mass index, waist circumference), glycemic markers (fasting blood glucose concentration, hemoglobin A_{1c} level, blood insulin concentration), lipid profiles (total cholesterol concentration, non-high-density lipoprotein [HDL] cholesterol concentration, low-density lipoprotein [LDL] cholesterol concentration [calculated], LDL size, LDL particle number, small

LDL particle number, oxidized LDL concentration, very low-density lipoprotein size, large very low-density lipoprotein particle number, triglycerides concentration, HDL cholesterol concentration, HDL size, HDL particle number, large HDL particle number), and quality of life as measured by the EuroQol 5 dimensions questionnaire.

Endothelium activity was measured, as an exploratory end point, in a subset of participants using the EndoPat Device (Itamar Medical Ltd, Caesarea, Israel). Major adverse cardiovascular and cerebrovascular event was defined as the composite of all-cause mortality, myocardial infarction, stroke or transient ischemic attack, and repeat coronary revascularization. Participants were directly asked about interim clinical events during study visits. Source documents were collected, and the reported events were adjudicated by the study investigators blinded to treatment allocation.

The Cleveland HeartLab, Inc (Cleveland, OH) measured certain markers of inflammation (hsCRP, myeloperoxidase, hemogram with differential), the comprehensive lipid profiles, and glucometabolic parameters at no cost. Itamar Medical Ltd (Caesarea, Israel) provided the EndoPat machine to measure endothelial function. These sponsors also did not contribute to the study design or data analysis. All other secondary end points were measured at New York University School of Medicine.

Statistical Analyses

As previously described,²⁷ sample size was calculated using preliminary data from our cardiac catheterization laboratory (mean hsCRP concentration, 2.07 ± 0.57 mg/L), and based on an estimated decrease in mean hsCRP concentration by 20% with the vegan diet as compared with the AHA diet, significance level of 0.05, and power of 0.80. Using a 2-sided 2-sample t test, the number of participants needed in each group was estimated to be 30. After adjusting for a 40% floor effect (including possible participant drops), the sample size in each group was increased to 50.

Summary data are presented as median [interquartile range] for continuous variables and proportion (frequency) for categorical variables. Continuous data were compared between the 2 dietary groups with the Mann–Whitney test, and categorical data were compared between the 2 dietary groups with Fisher’s exact test or chi-squared test. Changes in dietary and physical activity data within each dietary group over time were compared using a related-samples Wilcoxon signed-rank test.

The primary analyses compared the 2 dietary strategies for the change in end points from baseline to 8 weeks. End points after 8 weeks of dietary intervention were assessed in the vegan diet group with AHA-recommended diet group as the reference using a linear regression model. Model 1 was adjusted for the baseline concentration of the end point. Model 2 was

additionally adjusted for covariates that may affect systemic inflammation—age, race, baseline waist circumference, diabetes mellitus, and prior myocardial infarction. Given their skewed distribution, the end points were log-transformed for these analyses and then transformed back to their original scale for presentation. Back-transformed beta estimates of treatment (vegan versus AHA-recommended diet) along with 95% confidence intervals were reported to assess the magnitude of the effect size. Statistical significance was tested using a 2-sided alpha level of 0.05 for the primary end point and 0.0015 for the secondary end points after Bonferroni adjustment for multiple comparisons. All analyses were performed as intention-to-treat. Statistical analyses were performed using the IBM SPSS Statistics software, version 23 (IBM Corporation, Armonk, NY) and SAS 9.4 (SAS Institute Inc, Cary, NC).

Results

Baseline Characteristics

Baseline demographic and clinical characteristics are shown in Table 1. Participants in the vegan diet group were older than those in the AHA diet group (63.0 [57.0–68.0] versus 59.5 [53.0–67.0], $P=0.06$). The majority of participants were white males and had a history of coronary revascularization. More than three quarters of the participants had dyslipidemia, and more than half had hypertension. Although more than half of the participants had a history of tobacco use, only a minority of participants actively smoked. Diabetes mellitus and prior myocardial infarction were present in $\approx 30\%$ of participants, but other major comorbidities were present in less than one fifth of the participants.

Baseline medication use and ancillary cardiac data are also shown in Table 1. Almost all participants in both diet groups were on aspirin and statin therapy, while a majority were also on a P2Y₁₂-inhibitor. More than half of the patients were on high-dose statin therapy (atorvastatin 40–80 mg or rosuvastatin 20–40 mg). The majority of participants had normal left ventricular function and significant CAD on invasive coronary angiography.

Follow-Up Characteristics

Two participants withdrew from the trial, both of whom were from the vegan diet group. One withdrew after 1 week and the other after 2 weeks. One participant in the AHA diet group refused a blood draw at the 8-week final visit.

Clinical covariates

Of the 4 users of tobacco within the 6 months before randomization in the vegan diet group, 2 did not use tobacco

Table 1. Baseline Demographic and Clinical Characteristics of Participants With Angiographically Defined Coronary Artery Disease Randomized to the Vegan Versus American Heart Association–Recommended Diet

	Vegan Diet (n=50)	American Heart Association–Recommended Diet (n=50)	P Value
Age, y	63.0 [57.0–68.0]	59.5 [53.0–67.0]	0.06
Male sex	86% (43)	84% (42)	0.99
Race			0.17
White	92% (46)	80% (40)	
Black	2% (1)	12% (6)	
Asian	6% (3)	6% (3)	
Other	0	2% (1)	
Hispanic ethnicity	8% (4)	10% (5)	0.99
Tobacco use*	58% (29)	50% (25)	0.55
Current tobacco use	8% (4)	2% (1)	0.36
Hypertension	64% (32)	62% (31)	0.99
Dyslipidemia	78% (39)	78% (39)	0.99
Diabetes mellitus	24% (12)	36% (18)	0.28
Prior myocardial infarction	24% (12)	38% (19)	0.19
Prior coronary revascularization	90% (45)	92% (46)	0.99
Congestive heart failure requiring diuretic treatment	0	4% (2)	0.50
Stroke or transient ischemic attack	0	2% (1)	0.99
Carotid artery disease [†]	14% (7)	18% (9)	0.60
Lower extremity artery disease [‡]	2% (1)	6% (3)	0.62
Chronic renal dysfunction	0	4% (2)	0.50
Dialysis	0	2% (1)	0.99
Aspirin	92% (46)	96% (48)	0.68
Any P2Y12 inhibitor	86% (43)	84% (42)	0.99
Statin	94% (47)	96% (48)	0.99
High-dose statin [§]	58% (29)	56% (28)	0.99
β-Blocker	66% (33)	62% (31)	0.84
Calcium channel blocker	20% (10)	16% (8)	0.80
Angiotensin-converting enzyme inhibitor or angiotensin receptor blocker	56% (28)	52% (26)	0.84
Long-acting nitrate	4% (2)	4% (2)	0.99
Ranolazine	2% (1)	6% (3)	0.62
Left ventricular ejection fraction			0.31
Normal or borderline	94% (45)	87% (39)	
Mildly/moderately reduced	6% (3)	13% (6)	
Severely reduced	0	0	
Number of vessels diseased on invasive coronary angiography			0.49
1	30% (15)	38% (19)	
2	38% (19)	40% (20)	
3	32% (16)	22% (11)	
Number of vessels diseased on invasive coronary angiography [¶]			0.69
0	6% (3)	2% (1)	
1	46% (23)	52% (26)	

Continued

Table 1. Continued

	Vegan Diet (n=50)	American Heart Association–Recommended Diet (n=50)	P Value
2	30% (15)	32% (16)	
3	18% (9)	14% (7)	

Continuous data are presented as median [interquartile range] and compared using the Mann–Whitney test. Categorical data are presented as proportion (frequency) and compared using Fisher's exact test or χ^2 test.

*Tobacco use was defined as ever smoked >100 cigarettes or 5 cigars or pipes in lifetime, and current tobacco use was defined as use within past 6 months.

†Carotid disease was defined as $\geq 50\%$ stenosis or qualitatively moderate or severe stenosis reported on carotid imaging or prior carotid artery revascularization.

‡Lower extremity peripheral artery disease was defined as $\geq 50\%$ stenosis or qualitatively moderate or severe stenosis reported on lower extremity arterial imaging or prior lower extremity artery revascularization.

§Atorvastatin 40 to 80 mg or rosuvastatin 20 to 40 mg.

||Defined as $\geq 50\%$ diameter stenosis by visual estimate, physiologically significant lesion as assessed by pressure gradient, or prior revascularization.

*Defined as $\geq 70\%$ diameter stenosis by visual estimate, physiologically significant by pressure gradient, or prior revascularization.

during the 8-week study period, 1 did not use tobacco between the 4-week interim and 8-week final visit, and 1 continued to use tobacco throughout the 8 weeks of the active study. The 1 user of tobacco within the 6 months before randomization in the AHA diet group did not use tobacco during the 8-week study period.

A low proportion of participants underwent a cardiac rehabilitation program during the 8-week study period in both the vegan (n=4) and AHA (n=5) diet groups.

Nutrient and physical activity data

Dietary intake data over the active study period are shown in Table 2. At baseline, there were no significant differences in reported dietary intake of key nutrients between the 2 diet groups. Over time, reported intakes of energy, protein, fat, and saturated fat significantly decreased in both groups. However, at the end of the study period, reported energy intake was higher ($P=0.01$), while protein intake was lower ($P<0.001$), in the vegan versus AHA diet group. Reported carbohydrate intake increased over the active study period in the vegan diet group ($P=0.02$) and, at the end of the study period, was higher in the vegan versus AHA diet group ($P<0.001$). Although the reported dietary fiber intake increased over the study period in both diet groups, at the end of the study period, dietary fiber intake was higher in the vegan versus AHA diet group ($P<0.001$). While intake of grains and vegetables increased in the vegan diet group over the study period ($P=0.01$ and $P<0.001$, respectively), at the end of the study period, only the intake of grains was significantly higher in the vegan versus AHA diet groups ($P<0.001$). Finally, micronutrient data show a lower intake of vitamin B₁₂, zinc, and omega-3 fatty acids over the study period in the vegan group ($P<0.001$), and at the end of the study period, these micronutrients were significantly lower in the vegan versus AHA diet groups ($P<0.001$).

Physical activity data over the active study period are shown in Table 3. Overall, measures of physical activity did not differ between diet groups at baseline or at the end of the study period.

Dietary adherence

Even with inclusion of the 2 trial withdrawals from the vegan diet group, there was a higher rate of dietary adherence as determined by 24-hour dietary recall data among participants in the vegan versus AHA diet groups (4-week interim visit: 96% versus 84%, $P=0.09$; 8-week final visit: 94% versus 70%, $P=0.003$).

End Points

Markers of inflammation

HsCRP concentrations over time with the vegan and AHA diet groups are shown in Table 4. The median change in hsCRP concentration over the study period was significantly lower in the vegan versus AHA diet groups (Figure 2). After adjustment for baseline concentrations, the vegan diet resulted in a significant 32% lower concentration of hsCRP when compared with the AHA diet (β estimate, 0.68 [95% confidence interval, 0.49–0.94], $P=0.02$) (Table 5). After additional adjustment for age, race, baseline waist circumference, presence of diabetes mellitus, and prior myocardial infarction, there remained a significant 33% lower concentration of hsCRP with the vegan versus AHA diet (adjusted β estimate, 0.67 [0.47–0.94], $P=0.02$). Measures of white blood cell activity did not differ over time between groups (Table 5).

Anthropometric data, glycemic markers, and lipid profiles

Although weight loss, as measured by body mass index and waist circumference, was observed in both diet groups (Table 4), the degree of these reductions did not significantly differ between diet groups (Table 5). Furthermore, there were no significant differences in fasting glucose, hemoglobin A_{1c}, or insulin concentrations between diet groups (Table 5).

After adjustment for baseline concentration, the vegan diet resulted in a nonsignificant 12% reduction in LDL cholesterol compared with the AHA diet (β estimate, 0.88 [0.80–0.96], $P=0.008$) (Table 5). After additional adjustment for age, race,

Table 2. Nutrient Data in Participants With Angiographically Defined Coronary Artery Disease Randomized to the Vegan Versus American Heart Association–Recommended Diet for 8 Weeks

	Vegan Diet (n=50)			American Heart Association–Recommended Diet (n=50)			P Value Between Groups	
	Baseline	8 Weeks	P Value	Baseline	8 Weeks	P Value	Baseline	8 Weeks
Energy, kcal	1871 [1470–2447]	1715 [1482–2052]	0.07	1761 [1452–2295]	1531 [1197–1817]	<0.001	0.66	0.01
Protein, g	96 [72–110]	50 [40–70]	<0.001	86 [64–116]	80 [66–92]	0.04	0.39	<0.001
Energy from protein, %	20.2 [15.4–24.2]	12.9 [10.5–14.1]	<0.001	18.7 [16.9–20.3]	21.4 [18.2–23.7]	0.001	0.30	<0.001
Carbohydrates, g	216 [164–283]	242 [210–289]	0.02	205 [158–258]	170 [140–222]	0.01	0.52	<0.001
Energy from carbohydrates, %	45.4 [37.6–52.0]	57.5 [52.5–63.3]	<0.001	43.2 [38.8–50.1]	47.9 [39.8–54.0]	0.10	0.64	<0.001
Fiber, g	21 [15–27]	36 [30–43]	<0.001	22 [15–28]	25 [20–30]	0.003	0.56	<0.001
Fat, g	69 [46–100]	56 [46–72]	0.006	68 [50–94]	48 [37–65]	<0.001	0.79	0.05
Energy from fat, %	35.3 [27.1–41.0]	29.9 [26.5–36.4]	0.03	37.1 [31.2–40.8]	30.2 [25.8–37.1]	0.002	0.28	0.88
Saturated fat, g	20 [12–36]	9 [6–12]	<0.001	18 [12–28]	10 [8–15]	<0.001	0.38	0.03
Energy from saturated fat, %	11.0 [7.2–13.5]	4.5 [3.6–5.3]	<0.001	9.2 [7.0–11.8]	6.6 [4.7–8.1]	<0.001	0.22	<0.001
Trans fatty acids, g	0.52 [0.10–0.93]	0.06 [0.00–0.74]	0.15	0.38 [0.11–0.67]	0.14 [0.05–0.43]	0.005	0.41	0.40
Cholesterol, mg	271 [169–403]	0 [0–2]	<0.001	227 [135–368]	142 [110–240]	0.003	0.13	<0.001
Sodium, mg	2347 [1540–3130]	1890 [1547–2269]	0.03	2226 [1370–2872]	1497 [1139–2090]	<0.001	0.22	0.006
Vitamin B ₁₂ , µg	4.6 [2.4–8.6]	1.2 [0.1–2.6]	<0.001	3.2 [1.7–4.6]	3.5 [2.3–5.4]	0.54	0.009	<0.001
Calcium, mg	750 [489–1010]	577 [438–807]	0.01	610 [411–770]	695 [477–847]	0.14	0.032	0.25
Iron, mg	14.3 [9.7–19.4]	14 [11–20]	0.56	13.7 [10.0–20.8]	12.4 [8.7–16.4]	0.12	0.864	0.06
Zinc, mg	8 [5–14]	5.1 [3.6–6.2]	<0.001	7.7 [5.1–10.3]	7.4 [5.6–9.3]	0.73	0.241	<0.001
Omega-3 fatty acids, mg	1.3 [0.8–2.0]	0.5 [0.3–1.1]	<0.001	1.4 [0.7–2.0]	1.3 [0.8–1.7]	0.10	0.761	<0.001
MyPlate—grain*	4.9 [3.4–6.5]	6.0 [4.8–7.6]	0.01	5.2 [3.7–6.0]	4.7 [3.1–6.0]	0.20	0.71	<0.001
MyPlate—vegetable†	1.6 [1.1–2.4]	2.5 [1.7–4.3]	<0.001*	2.1 [1.3–3.0]	2.4 [1.5–3.3]	0.32	0.10	0.29
MyPlate—fruit†	1.3 [0.5–2.4]	1.7 [0.8–2.8]	0.07	1.3 [0.2–2.1]	1.5 [0.8–2.0]	0.13	0.67	0.22

Data are presented as median [interquartile range] and compared within groups using related-samples Wilcoxon signed-rank test and between groups using the Mann–Whitney test. Four-day food record data were used to generate these data. Baseline food record data were missing from 1 participant in the AHA-recommended diet group. Final food record data at 8 weeks were missing from 2 participants in the vegan diet group.

*Ounce equivalent.

†Cup equivalent.

baseline waist circumference, presence of diabetes mellitus, and prior myocardial infarction, there remained a nonsignificant 13% reduction in LDL cholesterol with the vegan versus AHA diet (adjusted β estimate, 0.87 [0.78–0.97], $P=0.01$). Other lipid parameters did not significantly differ over time between diet groups (Table 5).

Quality of life

Quality of life as measured by the EuroQol 5 dimensions index increased over time in both groups (Table 4). However, these increases were not significantly different between the vegan and AHA diet groups (EuroQol 5 dimensions index adjusted β estimate, 1.00 [0.96–1.05], $P=0.13$; current health state score adjusted β estimate, 1.01 [0.94–1.08], $P=0.22$) (Table 5).

Endothelial function

Endothelial function, as assessed by the EndoPat device, was available in 80% ($n=40$) of participants in the vegan diet group and 74% ($n=37$) of participants in the AHA group at both baseline and 8-week final visits. There was no significant change in endothelial function over time (vegan diet: baseline 1.96 [1.62–2.70] to 8-week final 1.88 [1.61–2.61], $P=0.86$; AHA diet: baseline 2.12 [1.85–2.48] to 8-week final 1.84 [1.68–2.13], $P=0.12$). When abnormal endothelial function was defined as an EndoPat score <1.67 , no significant change in status of endothelial function (abnormal versus normal) was noted in 75% of vegan and 84% of AHA diet participants ($P=0.41$); a change was noted to go from normal to abnormal in 10% of vegan and 14% of AHA diet participants ($P=0.73$);

Table 3. Physical Activity Data in Participants With Angiographically Defined Coronary Artery Disease Randomized to the Vegan Versus American Heart Association Recommended Diet for 8 Weeks

	Vegan Diet (n=50)			American Heart Association-Recommended Diet (n=50)			P Value Between Groups	
	Baseline	8 Weeks	P Value	Baseline	8 Weeks	P Value	Baseline	8 Weeks
Vigorous metabolic equivalents (METs)	0 [0–1920]	360 [0–2040]	0.27	0 [0–960]	120 [0–1440]	0.03	0.27	0.54
Moderate METs	480 [0–1680]	720 [180–1560]	0.67	110 [0–1440]	480 [0–1120]	0.32	0.33	0.24
Walking METs	1386 [495–2426]	1386 [693–2772]	0.87	718 [248–2079]	743 [594–2376]	0.08	0.07	0.21
Total METs	3612 [1386–7971]	3947 [2106–5493]	0.43	2120 [668–4512]	2541 [1040–4434]	0.03	0.05	0.10
Moderate+vigorous activity (min)	240 [0–660]	360 [105–683]	0.81	143 [0–450]	210 [0–520]	0.09	0.24	0.18

Data are presented as median [interquartile range] and compared within groups using related-samples Wilcoxon signed-rank test and between groups using Mann–Whitney test and. Physical activity data are shown as time over 7 days. Baseline physical activity data were missing from 2 participants in the vegan diet group and 2 participants in the AHA-recommended diet group. Final physical activity data at 8 weeks were missing from 4 participants in the vegan diet group.

and a change was noted to go from abnormal to normal in 15% of vegan and 3% of AHA diet participants ($P=0.11$).

Clinical events

No participants had a myocardial infarction, underwent a repeat coronary revascularization, or died during the 8-week study period. No participants had a cerebrovascular event in the vegan diet group; 2 participants in the AHA diet group had a probable transient ischemic attack as determined by a clinical neurologist consultant.

Discussion

This randomized, open-label, blinded end-point trial demonstrated a significantly greater reduction in hsCRP with a vegan versus AHA-recommended diet in patients with established CAD on guideline-directed medical therapy. The degree of weight loss, as measured by both body mass index and waist circumference, did not significantly differ between the 2 diet groups. Markers of glycemic control and lipid profiles, overall, also did not significantly differ in the vegan diet group when compared with the AHA-recommended diet group.

Strengths of the current study include the use of the AHA-recommended diet as the comparator arm and the use of similar dietary counseling strategies and pharmacologic interventions in both arms with a focus on the only difference being consumption of plant-based versus animal-based protein.²⁷ Earlier studies evaluated the effects of a vegan diet versus a control group that consisted of no intervention.^{23–26} One study randomized 46 patients with established or likely CAD to either a 1-month program of vegan diet with prepared meals and stress management or no intervention.²³ This study demonstrated an increase in exercise duration and a decrease in plasma cholesterol concentrations with the vegan diet. The 48 patients with angiographically documented CAD randomized to

a low-fat vegetarian diet in the Lifestyle Heart Trial demonstrated regression of atherosclerosis burden over 1 year, while the no-intervention group demonstrated progression of disease over the study period.²⁵ Of note, none of these patients were on lipid-lowering medications during the study period. A more recent single-arm prospective cohort study evaluated the effects of the MultiSite Cardiac Lifestyle Intervention Program, which consisted of a low-fat, whole-foods, plant-based diet; exercise; stress management; and group support meetings, in 56 patients with established or likely CAD and 75 patients with at least 3 cardiac risk factors or diabetes mellitus.²⁶ This intervention demonstrated significant decreases in multiple indices, including C-reactive protein concentrations, body mass index, waist/hip ratio, insulin concentrations, and lipid profiles over the 3 months of the study.

An additional strength of the current study is that the changes in end points noted were in participants with established CAD already on guideline-directed medical therapy. Although only 35% of participants in the current study had hsCRP concentrations ≥ 2.0 mg/L at baseline, >90% of participants had prior coronary revascularization, >60% of participants had a prior myocardial infarction, and <5% of participants had nonobstructive CAD on invasive angiography. Despite significant reductions in major adverse cardiovascular outcomes with lipid-lowering statin therapy in patients with CAD,^{31,32} there remains a residual risk of adverse outcomes in this patient population. In the Further Cardiovascular Outcomes Research with PCSK9 Inhibition in Subjects with Elevated Risk study, there remained a 9.8% rate of major adverse cardiovascular events in the PCSK9 (proprotein convertase subtilisin/kexin type 9) inhibitor therapy group where the median LDL cholesterol was decreased to 30 mg/dL.³³ Anti-inflammatory strategies, on the other hand, provide incremental benefit on clinical outcomes. The recently published CANTOS (Canakinumab Antiinflammatory Thrombosis Outcome Study) demonstrated

Table 4. Markers of Cardiovascular Risk in Participants With Angiographically Defined Coronary Artery Disease Randomized to the Vegan Versus American Heart Association–Recommended Diet for 8 Weeks

	Vegan Diet (n=50)			American Heart Association–Recommended Diet (n=50)		
	Baseline	4 Weeks	8 Weeks	Baseline	4 Weeks	8 Weeks
Primary endpoint						
High-sensitivity C-reactive protein, mg/L	1.25 [0.9–2.3]	1.00 [0.6–2.3]	1.10 [0.7–2.1]	1.15 [0.7–2.8]	1.10 [0.6–3.6]	1.10 [0.7–2.9]
Secondary end points						
White blood cell subtypes						
White blood cells, K/μL	6.5 [5.1–7.9]	6.2 [5.1–7.3]	5.9 [4.8–7.4]	5.7 [5.0–7.0]	5.3 [4.6–6.6]	5.3 [4.5–6.4]
Neutrophil/lymphocyte ratio	2.5 [1.9–3.5]	2.6 [2.0–3.4]	2.6 [1.8–3.3]	2.1 [1.4–2.8]	1.8 [1.4–2.6]	2.0 [1.6–2.6]
Monocytes, classical subtype, %	73 [65–81]	75 [68–79]	71 [65–80]	71 [65–75]	70 [63–73]	68 [49–72]
Monocytes, intermediate subtype, %	3.6 [2.1–5.4]	4.4 [2.4–6.6]	4.2 [2.1–5.1]	3.9 [2.4–6.1]	4.6 [3.1–7.5]	4.8 [3.0–6.1]
Monocytes, nonclassical subtype, %	9 [7–16]	10 [7–16]	11 [5–16]	14 [9–17]	14 [11–23]	15 [12–20]
White blood cell cellular adhesion molecules						
Neutrophil surface expression ratio of L-selectin to CD11b, MFI	1.3 [0.7–2.4]	0.7 [0.4–1.9]	1.2 [0.9–2.7]	1.1 [0.8–2.1]	0.9 [0.6–1.6]	1.4 [0.8–1.9]
Soluble L-selectin, ng/mL	12.7 [8.4–15.3]	13.4 [9.3–15.6]	13.7 [10.1–15.7]	14.6 [10.2–17.7]	13.3 [9.8–18.3]	13.6 [9.4–18.2]
Soluble E-selectin, ng/mL	37.8 [28.0–71.9]	37.6 [29.3–68.5]	37.2 [30.9–47.6]	50.6 [32.5–89.8]	46.7 [32.4–70.6]	42.4 [30.4–68.4]
Soluble intercellular adhesion molecule-1, ng/mL	225.1 [158.5–366.4]	224.9 [154.2–308.5]	229.5 [153.9–343.2]	235.5 [159.6–357.7]	230.3 [167.7–307.3]	251.5 [159.1–372.9]
Soluble vascular cell adhesion molecule-1, ng/mL	304.7 [250.1–358.2]	325.2 [249.6–428.3]	316.9 [257.1–400.2]	343.1 [247.5–420.6]	325.1 [247.1–440.4]	346.2 [244.8–422.7]
Other markers of neutrophil activity						
Neutrophil gelatinase-associated lipocalin, ng/mL	59.8 [51.3–78.0]	58.9 [49.6–82.6]	60.9 [49.9–75.4]	53.8 [40.1–79.0]	56.7 [42.6–79.8]	57.0 [42.5–78.3]
Myeloperoxidase, pmol/L	277 [232–330]	267 [219–322]	267 [213–351]	274 [220–368]	262 [202–357]	276 [187–343]
Other inflammatory marker						
Urine F2-isoprostane/creatinine ratio	0.36 [0.25–0.52]	0.41 [0.06–0.52]	0.35 [0.01–0.54]	0.21 [0.01–0.40]	0.21 [0.01–0.36]	0.20 [0.01–0.35]
Anthropometric data						
Body mass index, kg/m ²	30.5 [26.7–34.2]	29.4 [25.7–32.9]	29.0 [25.5–32.4]	30.9 [25.8–33.4]	29.8 [25.4–32.6]	29.5 [25.2–32.4]
Waist circumference, cm	107 [96–120]	103 [95–118]	102 [94–117]	107 [98–117]	106 [98–113]	105 [97–113]
Glycemic markers						
Fasting glucose, mg/dL	101 [92–115]	97 [93–108]	97 [91–109]	103 [94–121]	102 [94–123]	100 [95–125]
Hemoglobin A _{1c} , %	5.8 [5.6–6.3]	5.7 [5.5–6.2]	5.7 [5.5–6.0]	6.0 [5.7–6.8]	5.9 [5.6–6.5]	5.9 [5.6–6.5]
Insulin, μU/mL	10.9 [8.1–17.4]	10.3 [7.0–16.4]	10.5 [6.8–14.6]	12.0 [5.6–19.7]	10.0 [5.5–16.3]	10.2 [5.6–13.8]
Lipid profile						
Total cholesterol, mg/dL	136 [120–164]	121 [111–150]	127 [116–154]	146 [123–169]	138 [119–163]	142 [124–160]
Non-HDL cholesterol, mg/dL	95 [74–114]	81 [66–103]	89 [71–106]	97 [79–114]	95 [78–116]	91 [82–113]

Continued

Table 4. Continued

	Vegan Diet (n=50)			American Heart Association-Recommended Diet (n=50)		
	Baseline	4 Weeks	8 Weeks	Baseline	4 Weeks	8 Weeks
	LDL cholesterol (calculated), mg/dL	73 [54–90]	58 [47–77]	63 [50–85]	75 [60–94]	71 [55–91]
LDL size, nm	20.6 [20.3–21.1]	20.5 [20.2–20.9]	20.5 [20.2–20.9]	20.6 [20.3–20.8]	20.5 [20.3–21.0]	20.6 [20.2–21.0]
LDL particle number, nmol/L	924 [704–1118]	759 [593–1014]	840 [656–1045]	924 [680–1109]	861 [675–1144]	842 [764–1110]
Small LDL particle number, nmol/L	444 [276–571]	421 [318–532]	469 [385–580]	400 [264–572]	398 [295–592]	396 [321–579]
Oxidized LDL, U/L	34 [25–41]	30 [25–35]	31 [26–38]	36 [28–40]	33 [27–42]	33 [28–41]
VLDL size, nm	47 [45–52]	47 [45–51]	48 [46–51]	47 [44–50]	46 [44–50]	47 [44–49]
Large VLDL-particle number, nmol/L	2.6 [1.5–4.3]	2.6 [1.7–4.1]	2.5 [1.9–3.7]	2.3 [1.5–3.5]	2.3 [1.4–3.3]	2.2 [1.4–3.5]
Triglycerides, mg/dL	112 [78–126]	107 [78–126]	102 [81–132]	105 [68–142]	100 [73–138]	85 [70–134]
HDL cholesterol, mg/dL	45 [38–53]	44 [35–50]	43 [40–51]	45 [35–55]	39 [35–54]	43 [36–56]
HDL size, nm	9.0 [8.7–9.4]	8.9 [8.5–9.3]	8.9 [8.6–9.3]	9.0 [8.5–9.5]	8.8 [8.7–9.4]	9.0 [8.5–9.6]
HDL particle number, μ mol/L	30 [28–33]	30 [27–33]	31 [28–34]	31 [27–37]	29 [26–33]	29 [25–34]
Large HDL particle number, μ mol/L	4.4 [2.1–6.7]	3.9 [2.6–5.6]	4.1 [2.4–5.7]	3.4 [2.0–6.6]	3.4 [2.1–6.2]	3.8 [1.7–7.2]
Quality-of-life survey						
EuroQol 5 dimensions index	0.84 [0.80–1.00]	1.00 [0.83–1.00]	1.00 [0.83–1.00]	0.84 [0.81–1.00]	1.00 [0.83–1.00]	1.00 [0.83–1.00]
Current health state score	75 [65–82]	81 [75–90]	85 [80–90]	70 [60–80]	75 [70–85]	80 [75–85]

End points are presented as median [interquartile range]. HDL indicates high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein.

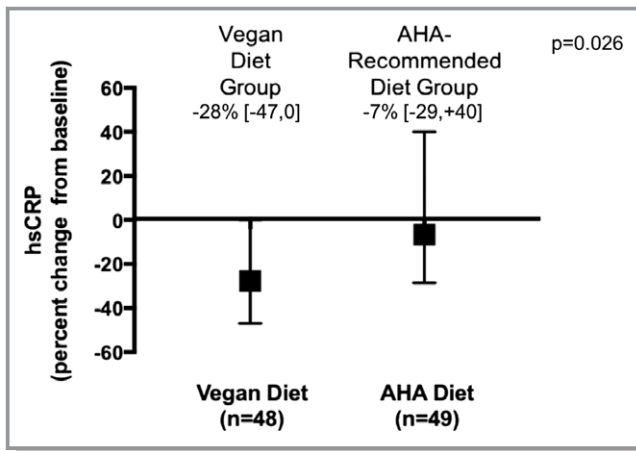


Figure 2. Percent change in hsCRP (high-sensitivity C-reactive protein) over the study period in participants with angiographically defined coronary artery disease randomized to the vegan vs American Heart Association (AHA)-Recommended diet. Data shown as median [interquartile range] and compared between diet groups using the Mann–Whitney test.

significant reductions in major adverse cardiovascular events with a monoclonal antibody to interleukin-1 β in patients with prior myocardial infarction and elevated baseline hsCRP concentrations.⁸ This reduction in outcomes was noted on a background of optimal medical therapy independent of lipid profiles. Notably, there was a heterogeneity of treatment effect among CANTOS participants, who achieved an hsCRP <2 mg/dL compared with \geq 2 mg/dL. Although the current study was not powered for clinical outcomes, it is possible that decrease in hsCRP with the vegan diet may provide added secondary cardiovascular prevention benefit when compared with the AHA-recommended diet.

The observed reductions in hsCRP and LDL cholesterol concentrations with the vegan diet may be independent or interdependent. The decrease in LDL cholesterol in the vegan group did not meet statistical significance, and there was no significant difference in oxidized LDL concentrations over time in either group or between groups. If the anti-inflammatory effect of the vegan diet is independent of its lipid-lowering effects, the underlying mechanism remains unclear. Subtypes of inflammatory cells nor the cellular adhesion molecules that play a significant role in adhesion of inflammatory cells to injured or inflamed endothelium did not differ over time between the 2 diet interventions. However, a significantly higher amount of dietary fiber was observed in the vegan arm of the current study, and studies have shown that a diet high in fiber and low in fat is associated with less inflammation and lower incidence of major cardiovascular outcomes.^{34–36} While the anti-inflammatory effect of fiber is well established, the underlying mechanism remains unclear. Data suggest increased fiber intake may restore gut microbiota, which may, in turn, improve the inflammatory profile.³⁴

Other dietary components such as carbohydrate intake may provide important context for the findings in the current study. A recent interim analysis from the international PURE (Prospective Urban Rural Epidemiology) study, a prospective observational cohort study, which did not include the United States, evaluated 135 335 participants and demonstrated a greater likelihood of all-cause mortality in participants who ate the most carbohydrates (average 77% of energy intake) versus those who ate the least (average 46% of energy intake).³⁷ In the current study, although there was a significantly higher carbohydrate intake in the vegan group, the median proportion of carbohydrate intake was 57% of the energy intake, much lower than the group at risk in the PURE analysis. Nonetheless, patients on a vegan diet should be encouraged to monitor their carbohydrate intake. In the PURE study, participants who ate the most total fat (average 35% of energy intake) compared with those who ate the least (average 11% of energy intake) had a lower likelihood of all-cause mortality. These results were consistent across the different types of fats. In the current study, there were no significant differences in the reported intake of total fat as a proportion of total energy intake between the 2 diet groups (30%). However, the intake of saturated fat was significantly lower in the vegan group, which may explain the numerical improvement in LDL-cholesterol concentration in that group.

There are several limitations to the current study. First, the study cohort reflects 14% of the patients who met initial inclusion/exclusion criteria, so generalizability may be limited. Second, the study is not powered to assess for differences in major adverse cardiovascular and cerebrovascular event. However, lowering hsCRP has been shown to be associated with lower outcomes in patients with prior myocardial infarction, and study participants will be followed for 5 years during which clinical event data are captured.⁷ Third, the vegan diet was only compared with 1 other diet—the AHA-recommended diet. The study did not include additional comparator arms, such as the Mediterranean Diet. However, both groups were counseled to incorporate protein from plant-based sources, including nuts and nut butters, and encouraged to use unsaturated oils, including olive oil.²⁰ Furthermore, olive oil was used as an ingredient for food preparation in recipes provided to both groups. Fourth, participants may have underreported their intake on the food records. Fifth, in the context of the dietary adherence protocol used, an AHA group participant who consumed no animal protein or no dairy would be considered adherent to the AHA-recommended diet. However, such a participant would favor the null hypothesis. Finally, the exact mechanism of potential underlying benefit remains incompletely elucidated given the lack of change in the white blood cell–related markers. Nonetheless, this is the first randomized trial to evaluate the effects of a vegan diet compared with one of the more

Table 5. Between Group Comparison of Markers of Cardiovascular Risk in Participants With Angiographically Defined Coronary Artery Disease Randomized to the Vegan Versus AHA-Recommended Diet for 8 Weeks

	Model 1			Model 2		
	β Estimate	95% Confidence Interval	P Value	β Estimate	95% Confidence Interval	P Value
AHA-recommended diet	Reference	Reference
Vegan diet						
Primary end point						
High-sensitivity C-reactive protein, mg/L	0.68	0.49–0.94	0.02	0.67	0.47–0.94	0.02
Secondary end points						
White blood cell subtypes						
White blood cells, K/ μ L	1.04	0.92–1.16	0.55	1.06	0.93–1.20	0.37
Neutrophil/lymphocyte ratio	1.15	0.94–1.41	0.16	1.20	0.96–1.49	0.10
Monocytes, classical subtype, %	1.25	1.07–1.45	0.005	1.09	1.04–1.45	0.02
Monocytes, intermediate subtype, %	0.65	0.44–0.95	0.03	0.69	0.46–1.05	0.08
Monocytes, nonclassical subtype, %	0.54	0.36–0.81	0.003	0.56	0.37–0.85	0.008
White blood cell cellular adhesion molecules						
Neutrophil surface expression ratio of L-selectin to CD11b, MFI	1.75	0.44–6.95	0.42	3.05	0.77–12.00	0.11
Soluble L-selectin, ng/mL	1.05	0.93–1.18	0.46	1.04	0.92–1.18	0.55
Soluble E-selectin, ng/mL	0.79	0.58–1.07	0.12	0.84	0.60–1.17	0.30
Soluble intercellular adhesion molecule-1, ng/mL	1.03	0.71–1.50	0.86	1.11	0.75–1.64	0.62
Soluble vascular cell adhesion molecule-1, ng/mL	1.07	0.97–1.19	0.18	1.09	0.97–1.22	0.13
Other markers of neutrophil activity						
Neutrophil gelatinase-associated lipocalin, ng/mL	0.99	0.88–1.12	0.87	1.03	0.91–1.18	0.61
Myeloperoxidase, pmol/L	1.04	0.92–1.18	0.53	1.03	0.90–1.17	0.68
Other inflammatory marker						
Urine F2-isoprostane/creatinine ratio	1.17	0.64–2.15	0.61	1.30	0.68–2.48	0.43
Anthropometric data						
Body mass index, kg/m ²	0.99	0.98–1.00	0.07	0.99	0.97–1.00	0.10
Waist circumference, cm	1.00	0.98–1.01	0.73	1.00	0.98–1.01	0.66
Glycemic markers						
Fasting glucose, mg/dL	0.92	0.85–1.00	0.06	0.93	0.86–1.01	0.10
Hemoglobin A _{1c} , %	0.99	0.97–1.01	0.36	1.00	0.98–1.02	0.91
Insulin, μ U/mL	1.04	0.86–1.26	0.70	1.04	0.85–1.26	0.72
Lipid profile						
Total cholesterol, mg/dL	0.95	0.89–1.01	0.08	0.94	0.88–1.01	0.09
Non-HDL cholesterol, mg/dL	0.92	0.84–1.00	0.05	0.92	0.84–1.01	0.08
LDL cholesterol (calculated), mg/dL	0.88	0.80–0.96	0.008	0.87	0.78–0.97	0.01
LDL size, nm	1.00	0.99–1.00	0.40	1.00	0.99–1.00	0.32
LDL particle number, nmol/L	0.91	0.82–1.02	0.10	0.91	0.81–1.03	0.13
Small LDL particle number, nmol/L	1.17	0.96–1.42	0.12	1.20	0.97–1.47	0.09
Oxidized LDL, U/L	0.92	0.82–1.03	0.13	0.93	0.83–1.05	0.24
VLDL size, nm	1.03	0.99–1.07	0.21	1.02	0.98–1.07	0.29
Large VLDL particle number, nmol/L	1.15	0.92–1.43	0.22	1.15	0.91–1.46	0.23

Continued

Table 5. Continued

	Model 1			Model 2		
	β Estimate	95% Confidence Interval	P Value	β Estimate	95% Confidence Interval	P Value
Triglycerides, mg/dL	1.06	0.94–1.21	0.35	1.06	0.93–1.22	0.38
HDL-cholesterol, mg/dL	1.02	0.97–1.08	0.42	1.02	0.96–1.08	0.47
HDL size, nm	0.99	0.96–1.01	0.23	0.99	0.96–1.01	0.32
HDL particle number, $\mu\text{mol/L}$	1.07	1.02–1.13	0.01	1.07	1.02–1.13	0.01
Large HDL particle number, $\mu\text{mol/L}$	1.00	0.85–1.18	0.98	0.98	0.83–1.16	0.82
Quality-of-life survey						
EuroQol 5 dimensions index	1.00	0.96–1.04	0.96	1.00	0.96–1.05	0.13
Current health state score	1.00	0.94–1.07	0.95	1.01	0.94–1.08	0.22

End points after 8 weeks of dietary intervention were assessed in the vegan diet group with AHA-recommended diet group as the reference using a linear regression model. Model 1: adjusted for the baseline concentration of the end point. Model 2: adjusted for the baseline concentration of the end point, age, race, baseline waist circumference, diabetes mellitus, and prior myocardial infarction. Significance for primary end point is set at an alpha level of 0.05 and for the secondary end points at an alpha level of 0.0015 after Bonferroni correction. AHA indicates American Heart Association; HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein.

commonly referenced dietary recommendations in the United States on multiple parameters of inflammation, glucometabolic, and lipid profiles in patients with established CAD on guideline-directed medical therapy.

In conclusion, in patients with CAD and an elevated hsCRP despite guideline-directed medical therapy, a vegan diet may be considered to further lower this marker of adverse outcomes. The vegan diet does not appear to provide significant added benefit when compared with the AHA-recommended diet in terms of weight loss, glycemic control, or lipid profile improvement.

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Disclosures

None.

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