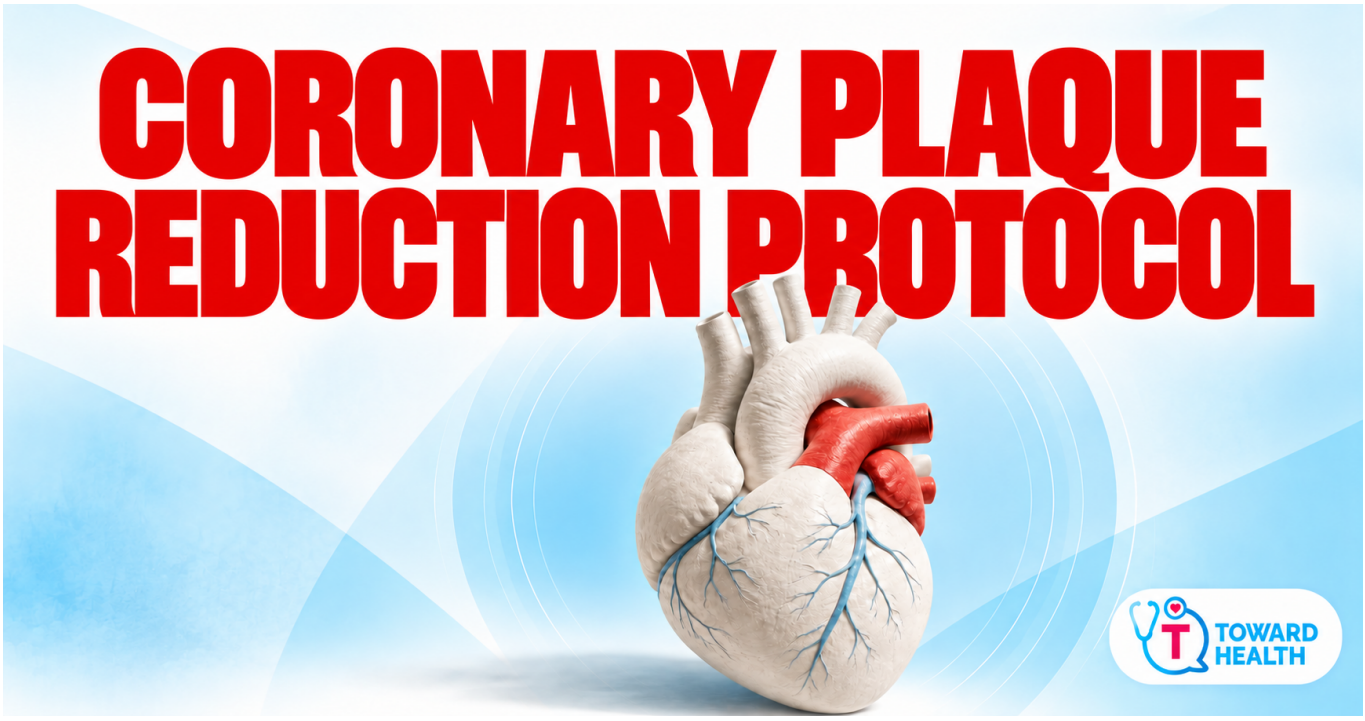
TOWARD HEALTH

CORONARY PLAQUE REDUCTION PROTOCOL

A practical guide to metabolic health, treatment options and coronary plaque.



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toward.health/plaque-reduction

Coronary plaque can shrink, grow more slowly or become more stable. Start by identifying and treating metabolic risk, including insulin resistance, obesity or excess abdominal fat, metabolic syndrome and diabetes status. Blood pressure, smoking and existing plaque also shape the plan.

Plaque deserves a plan

Plaque contains different mixtures of fat, inflammatory tissue, fibrous tissue and calcium. Reducing its volume, making its composition more stable and preventing a heart attack are related goals, but they are different measurements. You can improve cardiovascular health without making every speck of calcium disappear.

Begin by reviewing insulin resistance, abdominal fat or obesity, metabolic-syndrome features and diabetes status. Existing plaque, symptoms, smoking, blood pressure, family history and lipids also help determine how aggressively to act. The aim is fewer heart attacks and strokes, better function and a plan you can sustain. [1]

Start with metabolic health

Insulin resistance, abdominal obesity, metabolic syndrome and diabetes are central to coronary risk. Serial imaging studies link metabolic syndrome and diabetes to greater progression of coronary calcium or plaque. Another imaging study found more subclinical atherosclerosis among insulin-resistant people with normal HbA1c. Metabolic health therefore belongs at the front of a plaque-reduction plan. [30] [31] [32]

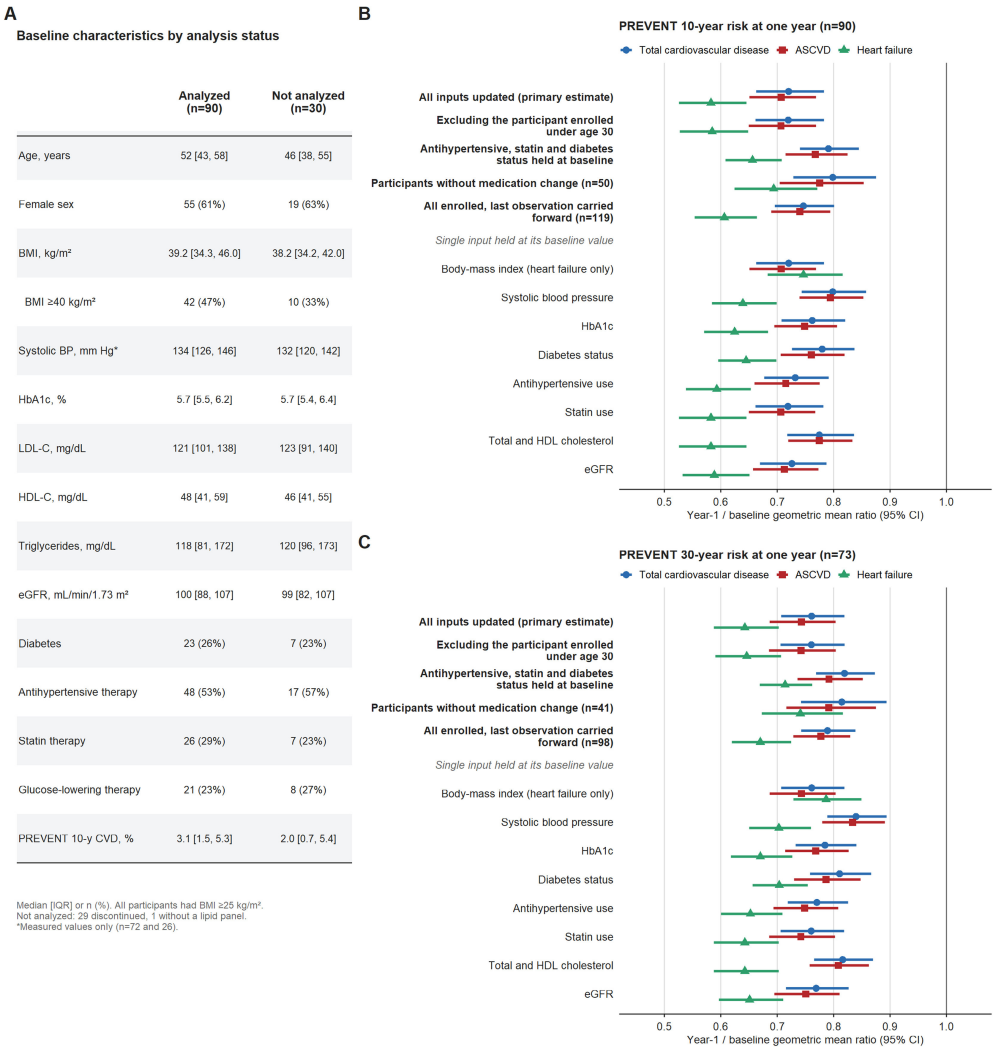
Establish diabetes status and follow glucose and HbA1c, but also review waist and weight trends, blood pressure, triglycerides and HDL. Work with your care team on sustainable nutrition, activity, weight reduction when appropriate and diabetes treatment. Reassess as risk factors change and adjust medicines safely. Smoking cessation and other indicated coronary treatments should proceed alongside this work. [1]

Our newest research: improving metabolic health

Our 2026 American Journal of Preventive Cardiology paper examined a year of telehealth metabolic care centered on a therapeutic ketogenic diet. This retrospective cohort included 120 adults; 90 had paired PREVENT estimates. Estimated 10-year risks fell by 29% for atherosclerotic cardiovascular disease, 28% for total cardiovascular disease and 42% for heart failure. [2]

Median weight fell 12.2%, systolic pressure fell 10.7 mmHg, HbA1c fell 0.48 percentage points and triglycerides fell 34 mg/dL. Across the cohort, 125 medications were discontinued. The study measured metabolic improvement and estimated cardiovascular risk, rather than changes in coronary plaque or observed heart attacks. [2]

At Toward Health, nutrition changes come with coaching, clinician follow-up, remote monitoring and medication adjustment. Individualized carbohydrate reduction can help people work on glucose, weight and appetite together. Follow-up matters as blood sugar and pressure improve, especially for people using medications that can cause low glucose or low blood pressure. Read about our newest research



A

Baseline characteristics by analysis status

	Analyzed (n=90)	Not analyzed (n=30)
Age, years	52 [43, 58]	46 [38, 55]
Female sex	55 (61%)	19 (63%)
BMI, kg/m²	39.2 [34.3, 46.0]	38.2 [34.2, 42.0]
BMI ≥40 kg/m²	42 (47%)	10 (33%)
Systolic BP, mm Hg*	134 [126, 146]	132 [120, 142]
HbA1c, %	5.7 [5.5, 6.2]	5.7 [5.4, 6.4]
LDL-C, mg/dL	121 [101, 138]	123 [91, 140]
HDL-C, mg/dL	48 [41, 59]	46 [41, 55]
Triglycerides, mg/dL	118 [81, 172]	120 [96, 173]
eGFR, mL/min/1.73 m²	100 [88, 107]	99 [82, 107]
Diabetes	23 (26%)	7 (23%)
Antihypertensive therapy	48 (53%)	17 (57%)
Statin therapy	26 (29%)	7 (23%)
Glucose-lowering therapy	21 (23%)	8 (27%)
PREVENT 10-y CVD, %	3.1 [1.5, 5.3]	2.0 [0.7, 5.4]

Median [IQR] or n (%). All participants had BMI ≥25 kg/m².
Not analyzed: 29 discontinued, 1 without a lipid panel.
*Measured values only (n=72 and 26).

B

PREVENT 10-year risk at one year (n=90)

Total cardiovascular disease · ASCVD · Heart failure

All inputs updated (primary estimate)
Excluding the participant enrolled under age 30
Antihypertensive, statin and diabetes status held at baseline
Participants without medication change (n=50)
All enrolled, last observation carried forward (n=119)
Single input held at its baseline value
Body-mass index (heart failure only)
Systolic blood pressure
HbA1c
Diabetes status
Antihypertensive use
Statin use
Total and HDL cholesterol
eGFR

0.5 0.6 0.7 0.8 0.9 1.0
Year-1 / baseline geometric mean ratio (95% CI)

C

PREVENT 30-year risk at one year (n=73)

Total cardiovascular disease · ASCVD · Heart failure

All inputs updated (primary estimate)
Excluding the participant enrolled under age 30
Antihypertensive, statin and diabetes status held at baseline
Participants without medication change (n=41)
All enrolled, last observation carried forward (n=98)
Single input held at its baseline value
Body-mass index (heart failure only)
Systolic blood pressure
HbA1c
Diabetes status
Antihypertensive use
Statin use
Total and HDL cholesterol
eGFR

0.5 0.6 0.7 0.8 0.9 1.0
Year-1 / baseline geometric mean ratio (95% CI)

Published results from our one-year metabolic-care study. The figure shows participant characteristics and changes in PREVENT-estimated cardiovascular risk. Calkins and colleagues, American Journal of Preventive Cardiology, 2026, Figure 1. © The authors, CC BY-NC-ND 4.0. View full-size figure

Build the foundation every day

Start with food you can sustain, adequate nutrition and less reliance on highly processed foods. Track blood sugar and blood pressure rather than guessing from weight alone.

Build aerobic fitness and strength, stop smoking, protect sleep and address persistent stress. Poor sleep, fatigue or snoring deserve attention because an untreated problem can make daily changes harder. A plan that fits your work, family and physical capacity is more useful than a demanding routine you cannot maintain. [3]

Exercise can affect plaque directly. In CENIT, six months of supervised interval training after a coronary procedure reduced percent atheroma volume (the share of measured vessel volume occupied by plaque) by about 1.2 percentage points; the control group received usual preventive care. Participants continued medical treatment. For people with established heart disease, cardiac rehabilitation or a supervised program provides a practical way to build exercise intensity. [4] [1]

Know what your tests are measuring

A CAC scan measures calcified coronary plaque. CCTA can show calcified and noncalcified plaque, narrowing and plaque composition. Stress testing evaluates whether the heart develops inadequate blood supply during exertion or medication-induced stress. These tests answer different questions; a normal stress test does not mean the arteries contain no plaque.

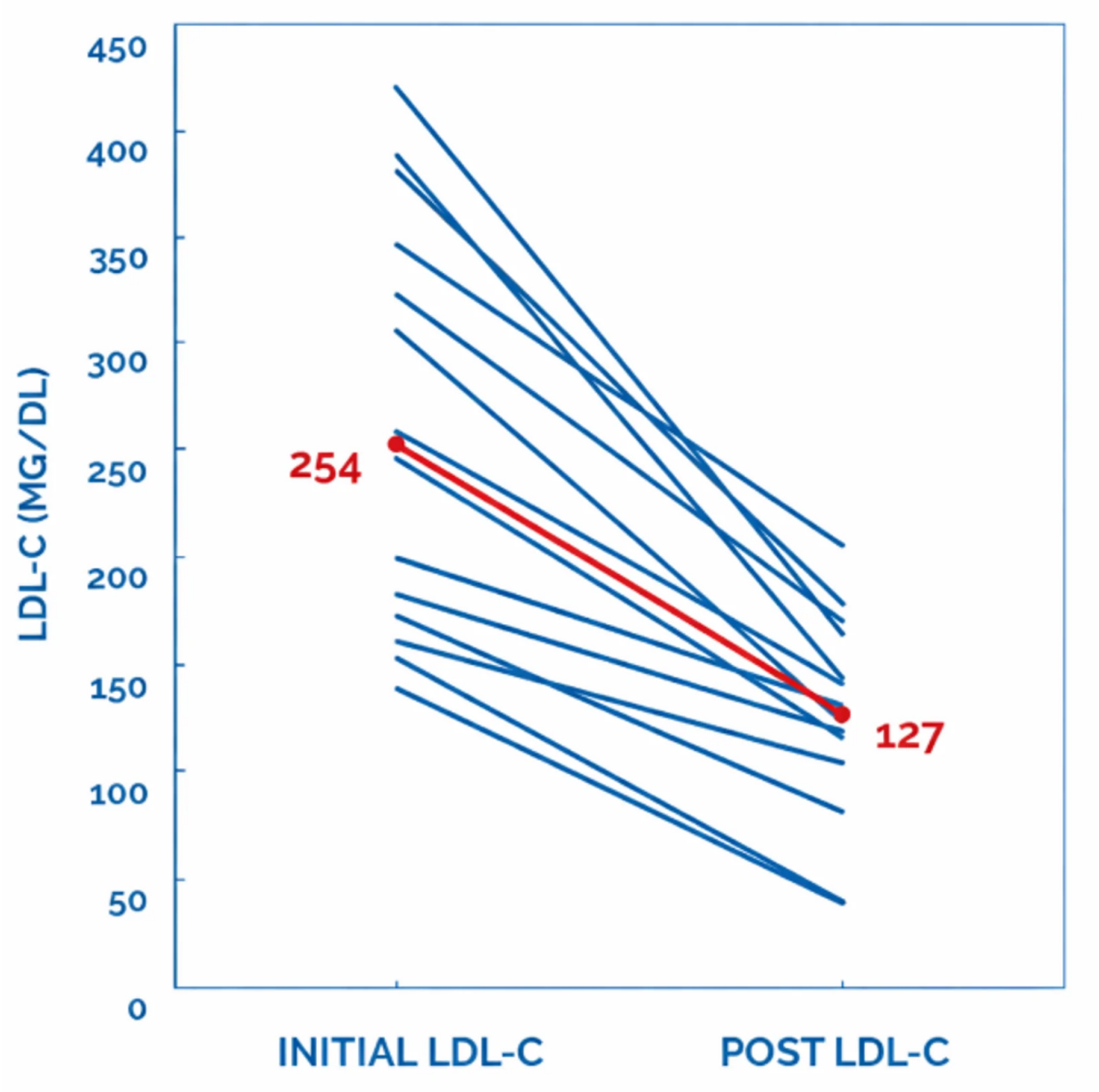
Age, symptoms and family history give a scan its context. A zero calcium score often indicates lower near-term risk, but it does not exclude soft plaque or explain new symptoms. Whether to repeat imaging depends on what the result would change in your care. Compare CAC, CCTA, HeartFlow, Cleerly and stress tests

Non-statin options for LDL and plaque

Ezetimibe

Ezetimibe reduces intestinal cholesterol absorption. In PRECISE-IVUS, adding it to atorvastatin produced greater plaque regression than atorvastatin alone over 9-12 months: median percent atheroma volume changes were -1.4 versus -0.3 percentage points. IMPROVE-IT also found fewer composite cardiovascular events after acute coronary syndromes: 32.7% versus 34.7% at seven years with ezetimibe plus simvastatin versus simvastatin alone. [5] [6]

Our Lipids in Health and Disease case series explored a notable response during carbohydrate restriction. Among 14 patients selected for a large response to ezetimibe, the median paired LDL reduction was 53.2%; 12 used ezetimibe alone and two also used statins. This helps identify a useful treatment option for selected patients with diet-associated LDL elevations. The result describes these responders, rather than the average response everyone should expect. Plaque and cardiovascular events were not measured. [7]



LDL cholesterol before and after ezetimibe in the selected case series; each blue line represents a participant. Gonzalez and colleagues, Lipids in Health and Disease, 2026, Figure 2. © The authors, CC BY-NC-ND 4.0. View full-size figure

PCSK9 inhibitors

These injections can produce substantial additional LDL lowering. GLAGOV tested evolocumab added to statins: percent atheroma volume fell 0.95 percentage points versus a 0.05-point increase with placebo at 78 weeks. FOURIER measured clinical outcomes: its composite endpoint occurred in 9.8% versus 11.3% over a median 2.2 years. The endpoint combined cardiovascular death, heart attack, stroke, unstable-angina hospitalization and coronary revascularization. Injection-site reactions are a possible tradeoff. [8] [9]

Triglycerides and inflammation also matter

Prescription EPA

EVAPORATE studied prescription icosapent ethyl in people with coronary disease and elevated triglycerides already taking statins. Low-attenuation plaque volume fell 17% over 18 months, compared with a 109% increase in the placebo group. Low-attenuation plaque is a softer, lipid-rich component measured on CCTA. [10]

In REDUCE-IT, the composite cardiovascular endpoint occurred in 17.2% versus 22.0% over a median 4.9 years. Hospitalization for atrial fibrillation or flutter was more frequent, 3.1% versus 2.1%. Serious bleeding occurred in 2.7% versus 2.1%; that difference did not reach statistical significance. These results apply to prescription EPA in the studied patients, not ordinary fish-oil supplements. [11]

Colchicine

Low-dose colchicine targets inflammation. LoDoCo2 found fewer cardiovascular events in people with chronic coronary disease. In the 12-month EKSTROM imaging trial, colchicine slowed overall plaque progression, while the primary low-attenuation plaque measurement did not differ significantly. Kidney function, digestive tolerance and medication interactions help determine whether it fits an individual treatment plan. [12] [13] [1]

Blood pressure control and aspirin

For someone with high blood pressure, sustained control may be the single most important modifiable step to help prevent further coronary calcium and plaque progression. Higher blood pressure is linked to faster progression, and treating hypertension lowers cardiovascular risk. Nutrition, activity and medication can all help achieve lasting control. [1] [33] [34]

Aspirin does not shrink plaque, but it helps prevent clots and has mild anti-inflammatory activity. A positive coronary calcium score can prompt a discussion of low-dose aspirin as a real option, especially when Lp(a) is elevated. The decision should weigh bleeding risk alongside calcium burden and overall cardiovascular risk. [35] [36] [37]

Vitamin K2, vitamin D and nattokinase

Vitamin K2 and D3

Recent trials suggest a role in slowing calcification when substantial calcium is already present. AVADEC was neutral overall, with a signal in the higher-CAC subgroup. In DANCODE, people starting at CAC 400 or above had a smaller two-year calcium-score increase with K2 plus D3: 196 versus 248 units. Total plaque volume increased similarly in both groups. Slower calcification is a different result from removing plaque or preventing a heart attack. [14] [15]

In VitaK-CAC, a two-year randomized trial in people with symptomatic coronary disease, vitamin K2 alone (MK-7, 360 micrograms daily) slowed the rise in coronary calcium versus placebo by an adjusted 22 Agatston units (P = .02). This finding concerns calcium progression, not demonstrated plaque regression or fewer heart attacks. [38]

Nattokinase

A retrospective study of 1,062 people using nattokinase at 10,800 fibrinolytic units (FU) daily for 12 months reported smaller carotid plaque measurements in 66.5% of those assessed. That is the proportion of people who improved, not the percentage of plaque removed. A separate randomized trial using 2,000 FU daily found no benefit on carotid atherosclerosis measures over three years. The higher-dose study reported plaque regression, but these studies did not demonstrate reduced cardiovascular events or mortality. [16] [17]

Where statins fit and what to watch

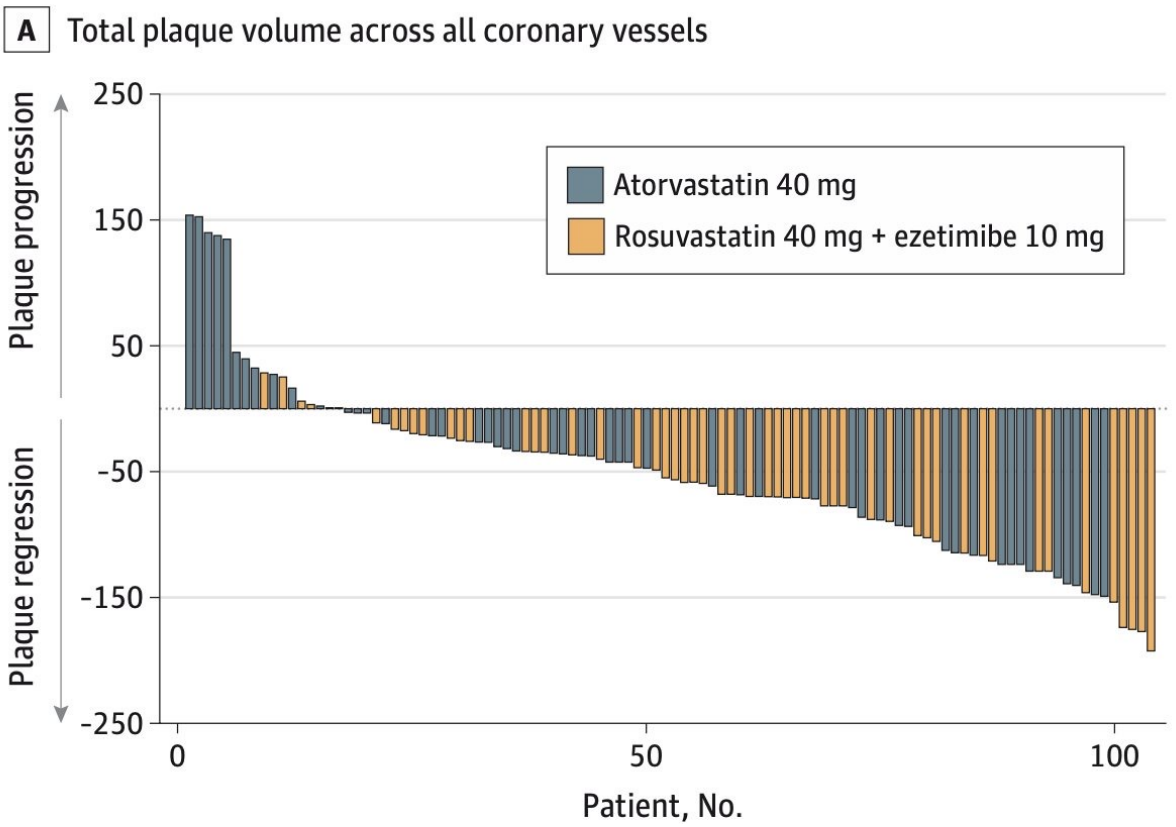
Statins reduce heart attacks and strokes in appropriate patients, with larger absolute benefits when baseline risk is higher. They can also reduce coronary plaque: in SATURN, percent atheroma volume fell 0.99 percentage points with atorvastatin and 1.22 with rosuvastatin over 104 weeks; the difference between regimens was not statistically significant. They are one part of the treatment conversation, alongside metabolic care, preferences and alternatives. [18] [19]

Greater plaque reduction with combination therapy

Statins can reduce coronary plaque while increasing plaque calcification and calcium density. [39] Ezetimibe can enhance plaque regression when added to statin therapy. [5]

In FLOWPROMOTE, rosuvastatin 40 mg plus ezetimibe 10 mg reduced total plaque volume by 21.1% and noncalcified plaque by 28.2% over 18 months, compared with 13.6% and 20.0% with atorvastatin 40 mg. Calcified plaque increased similarly in both groups: the combination achieved greater plaque reduction with a similar change in calcified plaque. [40]

Figure 2. Waterfall Plots of Changes in Total Plaque Volume, Noncalcified Plaque Volume, and Calcified Plaque Volume Across All Coronary Vessels at 18 Months



Each bar represents one participant's change in total coronary plaque volume after 18 months. Bars below zero show regression; bars above zero show progression. Gray: atorvastatin. Gold: rosuvastatin plus ezetimibe. Mortensen et al., *JAMA Cardiology*, 2026. Figure 2A.

Blood sugar and insulin resistance

Statins can raise glucose and HbA1c, and reduced insulin sensitivity has been measured in some studies. More intensive treatment carries a greater risk of a new diabetes diagnosis, particularly near the diagnostic threshold. Large blinded trials found about 1.2 additional diabetes diagnoses per 1,000 people treated for a year with low- or moderate-intensity statins. Follow glucose trends and discuss the regimen if metabolic control worsens. [20] [21]

Muscle symptoms and CoQ10

Muscle pain or weakness deserves attention. Blinded trials found about 11 additional reports per 1,000 people during the first treatment year. Statins lower circulating CoQ10, and some high-dose studies also found lower muscle CoQ10. Tissue effects vary, and supplements have not consistently relieved symptoms. Investigating medication interactions, dose and other causes can help find a treatment you tolerate. [22] [23] [24] [25]

Vitamin K2

Laboratory and animal research suggests interference with production of MK-4, a form of K2; human studies have reported changes in vitamin K-related biomarkers. [26] [27]

Uncommon serious effects

Uncommon serious effects include severe muscle injury, autoimmune muscle disease and significant liver injury. Severe weakness, dark urine or jaundice warrant prompt medical evaluation. Symptoms can justify a different dose, regimen or treatment. They should not simply be dismissed. [28]

Follow the health changes, not just the calcium number

Track symptoms, exercise capacity, glucose, blood pressure, lipids and treatment tolerance. A rising calcium score during statin treatment does not, by itself, prove failure: PARADIGM linked statin use with more calcification alongside slower noncalcified plaque progression and fewer new high-risk features. Interpret follow-up imaging in that context. [29]

Build your plaque-reduction plan

Bring your scan report, medication list and recent labs into one conversation with your care team. Identify the metabolic changes you can make, decide which treatments offer worthwhile benefit, and agree on how you will measure progress. A useful plan treats the person and the disease together.

Explore heart health and screening

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Additional safety references

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Safety notes

Medication changes. Review treatment changes with your medical team. Do not independently start, stop or change prescription medicines or aspirin. [1]

Food changes and fasting. Agree on monitoring and medication adjustments before major carbohydrate reduction or fasting, especially if you take insulin or other medicines that can cause low blood sugar. Continue monitoring blood pressure as treatment and nutrition change. [42]

Exercise. If you have established coronary disease, discuss an appropriate exercise plan and whether supervised cardiac rehabilitation is suitable. [41]

Warfarin and vitamin K. Discuss vitamin K, including K2 supplements, with your medical team. Keep vitamin K intake from food and supplements consistent rather than making abrupt changes. [43]

Supplements. Bring your complete medication and supplement list to your clinician or pharmacist. Check for interactions and bleeding risk, particularly when taking anticoagulants or antiplatelet medicines. [44]

New symptoms. Seek urgent medical attention for new or worsening chest symptoms. Severe weakness, dark urine or jaundice also warrant prompt medical evaluation. [1] [28]

Medical disclaimer

This protocol provides general education and does not replace individualized medical care. Treatment choices should reflect your symptoms, medical history, test results, medications and preferences. Study results do not guarantee an individual outcome.