C U R I N G H E A R T D I S E A S E

The 40 Biggest
Heart-Disease Lies

Forty things you have been told about your heart
that are not true — and what the evidence actually shows

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A note before you begin: this collection is education, not medical advice. It cannot know your history, your medications, or your risk, and nothing in it should be used to start, stop, or change any treatment, dose, diet, supplement, or exercise program — those decisions belong with a clinician who knows you. If you are having symptoms, seek medical care rather than reading further. Where the science is unsettled, this book says so.

How to Read This Collection

Each numbered entry stands alone. Read them in order or open the one you came for — nothing later depends on anything earlier. The headlines are deliberately provocative; the bodies are not. Every entry carries its own reference list, so you can check any claim without hunting through the back of the book.

The entries are grouped into five parts — lipids and testing, plaque and arteries, diet, exercise and aging, and risk and genetics — each mirroring a research section on CuringHeartDisease.com, where the deep-dive article behind every entry can be found.

What is in each entry

- **THE CLAIM —** the misconception stated plainly and fairly, in the form you would actually encounter it
- **THE HOOK —** the idea in one line
- **What the evidence actually shows —** three paragraphs of substance — mechanism, then trial or cohort evidence, then the practical consequence
- **The grain of truth —** what the myth gets right — because most of them get something right, and pretending otherwise would be its own kind of misinformation
- **BOTTOM LINE —** what to take away
- **Read more / See also —** where to go deeper on CuringHeartDisease.com
- **References —** the primary sources, numbered from 1 within each entry

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PART 1

Lipids, Medications and Testing

Cholesterol science, ApoB, statins, PCSK9 inhibitors, and what to actually measure.

Research hub: Lipids, Medications and Testing

01 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: Your Body Needs Cholesterol — So Lowering It Must Be Dangerous

THE CLAIM Every cell needs cholesterol to survive, therefore lowering LDL or ApoB with medication starves the body of something essential.

THE HOOK *Your cells need cholesterol. Your arteries do not need the delivery traffic.*

What the evidence actually shows

The argument collapses on a single distinction: the cholesterol inside your cells and the cholesterol travelling through your bloodstream are not the same pool, and they are not regulated the same way. Essentially every nucleated cell can manufacture its own cholesterol through the mevalonate pathway, and each one controls its internal supply through a tightly governed feedback system — sensing how much sterol it holds, adjusting synthesis up or down, and adding or retiring LDL receptors as needed. That machinery does not report to your blood test.

What circulates in plasma is something different: cargo in transit, packaged inside ApoB-containing lipoproteins. Those particles are the ones that cross the endothelium, become trapped in the arterial intima, and start the process that ends in plaque. Lowering their concentration reduces how much material is available to be deposited. It does not switch off a single cell's ability to make what it needs.

The clinical evidence makes the same point from the other direction. People with lifelong genetically low LDL — PCSK9 loss-of-function carriers, for instance — build healthy nervous systems, healthy hormones, and healthy cell membranes, while enjoying markedly less coronary disease. If low circulating LDL deprived cells of an essential substrate, these populations would show it. They do not.

THE GRAIN OF TRUTH The premise is correct — cholesterol genuinely is essential. The error is treating a transport-and-delivery number as if it were a supply-and-demand number.

BOTTOM LINE Cellular cholesterol is made on site and regulated locally. Blood ApoB is delivery traffic — and reducing traffic is not the same as cutting off supply.

Read the full article on CuringHeartDisease.com

- Your Cells Make Their Own Cholesterol - So Why Is Your Blood Full Of It?
- **See also:** The Million-Dollar Molecule: Why Your Body Pays a Fortune for Cholesterol

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02 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: LDL-C Is All You Need — ApoB Adds Nothing

THE CLAIM A standard LDL-C result always tells you how many artery-entering particles are circulating, so measuring ApoB is redundant.

THE HOOK *LDL-C weighs the cargo. ApoB counts the trucks.*

What the evidence actually shows

LDL-C and ApoB answer different questions. LDL-C reports how much cholesterol mass is being carried inside LDL particles. ApoB counts the particles themselves — one ApoB molecule per atherogenic lipoprotein, whether that particle is an LDL, a VLDL remnant, an IDL, or an Lp(a). Because it is one-per-particle, ApoB is a direct headcount of everything capable of lodging in an artery wall.

Most of the time the two numbers move together, and either one works. The problem is the minority of patients in whom they diverge — a state called discordance. Someone carrying many small, cholesterol-poor particles can post a reassuring LDL-C while running a high particle count. That pattern clusters in exactly the people who are already at elevated risk: insulin resistance, metabolic syndrome, high triglycerides, type 2 diabetes. When LDL-C and ApoB disagree, outcomes track ApoB.

This is why ApoB has moved from specialist interest to guideline-recognized risk marker. It is not that LDL-C is wrong; it is that LDL-C has a blind spot, the blind spot is not randomly distributed, and a single inexpensive test closes it.

THE GRAIN OF TRUTH For a large share of patients, LDL-C and ApoB give the same answer, and LDL-C remains a perfectly serviceable primary target. The case for ApoB is about who gets missed, not about LDL-C being useless.

BOTTOM LINE If you have high triglycerides, insulin resistance, or an LDL-C that looks better than the rest of your risk picture, ApoB is worth raising with your clinician — it is the situation where the two numbers most often disagree.

Read the full article on CuringHeartDisease.com

- ApoB: Longer, Higher, Worse
- **See also:** Why is ApoB important?

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03 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: A High LDL Is Harmless When You Are Metabolically Healthy

THE CLAIM Low triglycerides, normal glucose, and a lean body cancel out the arterial risk of a very high LDL.

THE HOOK *Metabolic health lowers your risk. It does not issue a permit for unlimited ApoB.*

What the evidence actually shows

Metabolic health and atherogenic particle burden are two separate risk axes, and being excellent on one does not neutralize the other. Insulin sensitivity, low visceral fat, and a favorable triglyceride profile genuinely reduce cardiovascular risk — they improve endothelial function, lower inflammatory tone, and reduce the production of small dense particles. None of that alters the physical chemistry by which an ApoB particle binds arterial proteoglycans and is retained.

The most-discussed test case is the lean mass hyper-responder phenotype: metabolically pristine individuals whose LDL-C rises sharply on carbohydrate restriction, often above 190 mg/dL, while triglycerides stay very low and HDL-C stays high. This is genuinely contested territory and deserves precision. The cross-sectional KETO trial imaged 80 such individuals after a mean of 4.7 years and found coronary plaque burden that was not significantly greater than in matched controls carrying 149 mg/dL less LDL-C — a null result the low-carbohydrate community reads as evidence of protection. What it did not show is that they had less plaque, which is what genuine protection would predict, and a single snapshot cannot describe a trajectory.

Familial hypercholesterolemia makes the same argument in a different population. Patients with FH are frequently lean, active, and metabolically unremarkable in every respect except one, and they develop premature coronary disease anyway. Lifetime particle exposure is doing the work.

THE GRAIN OF TRUTH Metabolic health matters enormously and is worth defending. It shifts risk downward, and a metabolically healthy person with a given ApoB is very probably better off than an insulin-resistant one with the same number. It just does not shift risk to zero, and it cannot be traded against an ApoB number.

BOTTOM LINE Treat metabolic health as a risk factor you have already fixed, not as a credit you can spend on the one you have not.

Read the full article on CuringHeartDisease.com

- Does metabolic health protect you from high cholesterol?

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04 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: Low Triglycerides Make a Very High LDL Safe on Keto

THE CLAIM Excellent triglycerides and a high HDL prove that a ketogenic-diet-induced LDL rise cannot promote plaque.

THE HOOK *A great triglyceride-to-HDL ratio tells you about insulin sensitivity. It tells you nothing about particle count.*

What the evidence actually shows

The triglyceride-to-HDL ratio is a rough marker associated with insulin resistance at population level — useful, but not a direct or universally reliable measure, and its performance varies by sex, ethnicity and metabolic state. It was never a measure of atherogenic particle burden, and it does not become one because the number is impressive.

Carbohydrate restriction reliably raises LDL-C in a subset of people, sometimes by 100 mg/dL or more, and the rise is largely a rise in particle number rather than a benign change in particle size. The cross-sectional KETO trial imaged exactly this population — LDL-C at or above 190 mg/dL, HDL-C at or above 60, triglycerides at or below 80 — and reported plaque burden not significantly greater than in matched controls carrying 149 mg/dL less LDL-C. Read carefully, that is a null finding in a snapshot rather than a demonstration of safety: the comparison group was matched on conventional risk factors, not selected for low ApoB, and the design cannot show how plaque in either group was changing over time.

A study designed to examine longitudinal plaque progression in this phenotype no longer stands. KETO-CTA followed this phenotype longitudinally and reported that baseline plaque, not ApoB, predicted progression — a result promoted widely as evidence against ApoB causality. JACC: Advances issued an Expression of Concern in January 2026, and in May 2026 the paper was retracted at the request of both the authors and the editors, after methodological concerns the editors judged too great to fix by correction. A retraction is not evidence for the opposite conclusion. But it removes the strongest published claim on that side of the argument, and anyone still citing it should stop.

What remains is a mechanism and a set of case reports. The lipid energy model offers a coherent account of why lean, glycogen-depleted people export more VLDL and therefore run higher LDL particle counts, and Norwitz's single-subject crossover — in which adding Oreo cookies to an LMHR's ketogenic diet lowered LDL-C more than high-intensity statin therapy did — is still published, still indexed, and a genuinely striking demonstration that the phenotype is diet-driven and rapidly reversible. Neither a mechanism nor an n-of-1 is an outcome study.

THE GRAIN OF TRUTH The honest position is that hard-endpoint evidence for this specific phenotype is thin in both directions — nobody has run an outcome trial in lean mass hyper-responders. A low triglyceride-to-HDL ratio is a real and favorable finding, and someone with this pattern is probably at lower risk than someone with the same LDL-C and insulin resistance. Anyone claiming certainty here, in either direction, is ahead of the data.

BOTTOM LINE If a diet doubles your ApoB, the diet's other benefits do not settle the question — and the study most often cited to settle it has been retracted. Measure the particle count, and decide with a clinician rather than from a podcast.

Read the full article on CuringHeartDisease.com

- The Big Cholesterol Lie, Part 2
- **See also:** Keto vs. Whole-Food Plant-Based: What's the hard evidence for performance, insulin sensitivity, heart disease reversal, and longevity?

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05 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: HDL Is the 'Good Cholesterol,' So Higher Is Always Better

THE CLAIM Raising HDL-C removes plaque and lowers risk regardless of what happens to ApoB.

THE HOOK *HDL-C is a thermometer, not a thermostat. Raising the reading does not lower the fever.*

What the evidence actually shows

Observational data showing that people with high HDL-C have fewer events is real and has been replicated for decades. The question is whether HDL-C is causing the lower risk or merely reporting on a healthier underlying state — and on that question the evidence indicates strongly that HDL-C concentration is a risk marker rather than a treatment target.

Mendelian randomization, which uses lifelong genetic variation as a natural randomized trial, found that variants raising HDL-C do not lower myocardial infarction risk, while variants lowering LDL-C do. The pharmacological record agrees: niacin added to statins failed to reduce events in AIM-HIGH and again in HPS2-THRIVE despite raising HDL-C, and the CETP inhibitor torcetrapib raised HDL-C by roughly 70% in ILLUMINATE while increasing mortality. Genetics plus two distinct drug mechanisms across three trials, and no benefit from raising the number in any of them.

There is also a J-shaped tail that surprises people. In two large Danish prospective cohorts totalling over 116,000 people, Madsen and colleagues found a U-shaped curve: all-cause mortality was lowest around 73 mg/dL in men and 93 mg/dL in women, and rose significantly above roughly 97 mg/dL in men and 135 mg/dL in women. That is an association rather than a demonstrated harm of HDL-C itself, but whatever the measurement is tracking, it is plainly not a quantity to maximize.

THE GRAIN OF TRUTH HDL particle function — cholesterol efflux capacity — may well matter biologically. But that is a different measurement from the HDL-C on a standard panel, and it is not yet a clinical target.

BOTTOM LINE Use HDL-C as a risk marker, not as a treatment target, and never as an offset against a high ApoB.

Read the full article on CuringHeartDisease.com

- Why Your 'Normal' Cholesterol is Actually a Warning Sign
- **See also:** Stop Trusting LDL! The Truth About ApoB, Insulin Resistance, & Diabetes

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06 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: Lipoprotein(a) Does Not Matter Because You Cannot Treat It

THE CLAIM Since no drug currently lowers Lp(a) with proven outcome benefit, testing for it is pointless.

THE HOOK *You cannot change your Lp(a) yet. You can change everything you do about it.*

What the evidence actually shows

Lp(a) is an LDL-like particle with an apolipoprotein(a) tail attached, it is roughly 80–90% genetically determined, it barely responds to diet or exercise, and it is causal — Mendelian randomization links genetically elevated Lp(a) to myocardial infarction and to calcific aortic stenosis. Somewhere around one in five people carry a clinically meaningful elevation, and most of them have never been tested.

The inference that an untreatable risk factor is not worth measuring does not survive contact with how prevention actually works. A high Lp(a) shifts overall risk upward, which in turn can change how aggressively everything modifiable is worth treating — the lipid goal — which may or may not be expressed as ApoB, depending on the guideline in use — blood pressure control, and how soon imaging is considered. Where those thresholds land is an individual clinical judgement, not a fixed rule. It also reframes the family — Lp(a) is inherited, so one result carries information for siblings and children.

The treatment picture is also changing quickly. RNA-based agents including pelacarsen, olpasiran and lepodisiran have produced Lp(a) reductions of 80% and above in phase 2, and the outcome trials are running now. Lp(a)HORIZON — 8,323 patients on pelacarsen, the first cardiovascular outcomes trial for any Lp(a)-lowering therapy — was still running as this was written, with OCEAN(a)-Outcomes testing olpasiran in roughly 7,300 patients behind it. By the time you read this, one or both may have reported; the answer is worth looking up, because it will determine whether Lp(a) becomes a treatable risk factor rather than only a measurable one. Either way, knowing your Lp(a) informs your risk assessment today and gives your relatives a reason to be tested.

THE GRAIN OF TRUTH It is fair to say that no therapy has yet proven that lowering Lp(a) lowers events. That is a statement about the drug pipeline, not about whether the risk factor is real.

BOTTOM LINE Lp(a) is measured once and does not meaningfully change — which makes it worth asking your clinician whether you have ever had it checked. The result should inform what you do about everything that is modifiable.

Read the full article on CuringHeartDisease.com

- Integrative Dynamics of Apolipoprotein B, Lipoprotein(a), and C-Reactive Protein in Atherosclerotic Progression

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Medical disclaimer: This article is for education only. It is not medical advice, it has not been reviewed by a treating clinician, and it cannot account for your individual circumstances. Nothing here should be used to start, stop, or change any medication, dose, diet, supplement, or exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

07 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: Statins Treat a Lab Number, Not Heart Disease

THE CLAIM Cholesterol medicines improve blood-test results without changing plaque or preventing events.

THE HOOK *The goal was never a prettier lab report. It is fewer heart attacks and strokes.*

What the evidence actually shows

If statins only moved a number, the trials would show a moved number and nothing else. Instead, the Cholesterol Treatment Trialists' meta-analysis of 26 randomized trials and roughly 170,000 participants found that each 1 mmol/L (about 39 mg/dL) reduction in LDL-C produced approximately a 22% proportional reduction in major vascular events over the trial follow-up — a proportional reduction per unit of LDL-C lowered, not a reduction compounding annually — and the relationship held across baseline risk, age, sex, and starting LDL.

Imaging trials confirm that the biology underneath the number is changing too. ASTEROID showed regression of coronary atheroma volume with very high-intensity statin therapy. GLAGOV showed further regression when a PCSK9 inhibitor was added on top of a statin, with the greatest change in those reaching the lowest LDL-C. Serial imaging also shows plaques becoming more fibrous and more calcified under treatment — the morphology of stabilization.

The consistency across drug classes is what makes the case hardest to explain any other way. Statins (CTT), ezetimibe (IMPROVE-IT), PCSK9 inhibitors (FOURIER), and bempedoic acid (CLEAR Outcomes) lower ApoB by entirely different mechanisms, and each reduced events roughly in proportion to how much it lowered it. That is what a causal exposure looks like, not a laboratory artifact.

THE GRAIN OF TRUTH Relative risk reduction and absolute benefit are different things. A 22% relative reduction is large for a high-risk patient and modest for a low-risk one — which is exactly why treatment should be matched to risk.

BOTTOM LINE Lipid-lowering is judged on events and on plaque, and it changes both. The lab number is how progress is tracked, not what is being treated.

Read the full article on CuringHeartDisease.com

- Should I take my cholesterol medicines? You bet.

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THE BIG LIE: Everyone Who Takes a Statin Gets Muscle Damage

THE CLAIM Muscle pain is a near-universal and unavoidable consequence of statin therapy.

THE HOOK *Muscle symptoms are common. Statin-caused muscle symptoms are much less common — and the difference is testable.*

What the evidence actually shows

Two facts are both true and are constantly confused with each other. Muscle aches are genuinely frequent in the population taking statins — middle-aged and older adults, many of them physically active. And in blinded comparison, the great majority of those aches occur at the same rate on placebo.

The SAMSON trial made this unusually concrete. Patients who had previously stopped statins because of side effects took, in randomized order, months of atorvastatin, months of placebo, and months of no tablet at all, recording symptoms daily. Symptom intensity on statin and on placebo was nearly identical, and both were substantially higher than on no tablet at all. Roughly 90% of the excess symptom burden produced by taking a statin tablet was also produced by taking a placebo tablet — meaning symptoms alone could not reliably tell participants which they were on. Half of them successfully restarted a statin afterwards.

The terms matter here and are routinely blurred. Myalgia means muscle ache without enzyme elevation and is common. Myopathy means symptoms with a raised creatine kinase. Rhabdomyolysis, the severe form with renal risk, is rarer still — the AHA statin safety statement cites hospitalized rhabdomyolysis at roughly 0.44 cases per 10,000 patient-years on statin monotherapy — about 0.004% in any given year of treatment, a rate rather than a lifetime probability — with the figure rising substantially when a statin is combined with gemfibrozil. Separating these matters, because which one a patient actually has determines whether they lose access to an effective therapy.

THE GRAIN OF TRUTH None of this means the symptoms are imagined. They are real, they are felt, and dismissing them is both unkind and clinically counterproductive. The question is what is causing them.

BOTTOM LINE Muscle symptoms deserve investigation rather than assumption. Clinicians have a structured way of telling true intolerance from coincidence, and most people who go through it end up able to take a lipid-lowering therapy of some kind. Do not simply stop on your own.

Read the full article on CuringHeartDisease.com

- Cholesterol for Athletes: How to Lower LDL Without Losing Your Edge (A Practical Guide to Statins)
- **See also:** Should I take my cholesterol medicines? You bet.

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

09 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: If One Statin Causes Symptoms, All Cholesterol Treatment Is Off the Table

THE CLAIM A bad experience with one medicine means no lipid-lowering option will be tolerable.

THE HOOK *One intolerable drug does not empty an entire toolbox.*

What the evidence actually shows

Statins are not interchangeable. They differ in how fat-soluble they are, in how long they last in the body, and in which liver enzymes break them down — which is why someone who cannot tolerate one drug in the class frequently tolerates a different one. Clinicians also have options around dose and frequency for the longer-acting agents. Which switch makes sense is a prescribing decision, and not one to improvise.

Beyond statins entirely, the modern toolbox includes ezetimibe, which blocks intestinal cholesterol absorption and reduced events on top of a statin in IMPROVE-IT; bempedoic acid, a prodrug activated in the liver but not in skeletal muscle, developed specifically for the statin-intolerant; PCSK9 monoclonal antibodies, which cut LDL-C by roughly 60% on a fortnightly or monthly injection and reduced events in FOURIER; and inclisiran, a small interfering RNA given twice yearly after loading, which produced comparable LDL-C reductions in the ORION phase 3 program — though its cardiovascular outcome trial had not reported at the time of writing, so it is an LDL-lowering agent with outcome evidence still pending.

Combination therapy also means no single agent has to carry the whole burden. A low-dose statin plus ezetimibe can reach the same target as a high-dose statin alone, often with better tolerability.

THE GRAIN OF TRUTH Some patients genuinely cannot take any statin, and their experience should be believed the first time. The point is that statin intolerance is a reason to change the plan, not to abandon it.

BOTTOM LINE Statin intolerance is a routing problem, not a dead end — at least four other drug mechanisms exist. If one medicine did not suit you, that is a conversation to reopen with your clinician rather than a door that closed.

Read the full article on CuringHeartDisease.com

- The Cholesterol Breakthrough: From Daily Pills to Twice-a-Year Shots

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10 LIPIDS, MEDICATIONS AND TESTING

THE BIG LIE: The Lowest Safe LDL Is the Population Average

THE CLAIM Once LDL reaches a 'normal' value, further reduction offers no benefit and may become unsafe.

THE HOOK *'Normal' describes the people around you. It does not describe the biology of a healthy artery.*

What the evidence actually shows

A population reference range tells you what is common, not what is optimal — and in a population where atherosclerosis is the leading cause of death, common is not a reassuring standard. Human newborns have an LDL-C around 30 mg/dL. Populations with lifelong low ApoB have very little coronary disease. There is no physiological argument that the adult Western average represents a floor.

The trial evidence points the same way. In FOURIER, a prespecified analysis found that patients reaching LDL-C values below 20 mg/dL — and even below 10 — continued to show lower event rates, with a monotonic relationship and no signal of harm across the safety endpoints examined, including neurocognitive outcomes, new-onset diabetes, hemorrhagic stroke, and muscle events. IMPROVE-IT found the same directional benefit at more modest levels.

The genetic evidence closes the loop. PCSK9 loss-of-function carriers spend an entire lifetime at very low LDL-C, with substantially reduced coronary risk and no recognized deficiency syndrome. Long duration at low levels appears to be safe as well as beneficial.

THE GRAIN OF TRUTH 'How low is safe' and 'how low is worth it' are different questions. Follow-up in the very-low-LDL trials is measured in years rather than decades, and the absolute benefit of pushing lower is small in genuinely low-risk people.

BOTTOM LINE A lab's reference range describes the population, not your target. Targets are set against individual risk, and in people with established disease they are generally well below 'normal' — worth understanding before reading a result as reassurance.

Read the full article on CuringHeartDisease.com

- Cholesterol: How Low is Too Low?

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PART 2

Plaque, Arteries and Disease

How plaque forms, why it becomes dangerous, how it stabilizes, and what imaging can and cannot see.

Research hub: Plaque, Arteries and Disease

THE BIG LIE: Heart Disease Starts in Old Age

THE CLAIM Atherosclerosis appears late in life, so cholesterol exposure in childhood and young adulthood barely matters.

THE HOOK *The heart attack is the last event in a story that started decades earlier.*

What the evidence actually shows

Autopsy studies of people who died young of unrelated causes settled this question before most current risk calculators were written. The Pathobiological Determinants of Atherosclerosis in Youth study examined the arteries of 15- to 34-year-olds killed in accidents, homicides, and suicides, and found early atherosclerotic change to be common in this age range and raised fibrous plaques already present in a substantial minority — with extent tracking non-HDL cholesterol, smoking, blood pressure, and obesity. The Bogalusa Heart Study found the same association in children and young adults.

What this means practically is that atherosclerosis is a slow accumulation, not a late-life event. The relevant exposure is not today's number but the cumulative burden of atherogenic lipoproteins over time. Domanski and colleagues measured this as cumulative LDL-C exposure; 'ApoB-years' is a useful analogy borrowed from pack-years, not a validated clinical metric. Two people with identical LDL-C at fifty can have very different arteries if one of them spent three decades at 160 mg/dL and the other did not.

This is precisely where a short-term risk calculator can give false reassurance if it is used to answer a lifetime question. A ten-year risk estimate for a healthy 35-year-old will be low almost regardless of their lipids, because ten-year risk is dominated by age. Lifetime risk in the same person can be very high. The calculator is not wrong; it is answering a question about the next decade when the disease is operating on a forty-year timescale.

THE GRAIN OF TRUTH Events do cluster in later life, and age is genuinely the strongest single predictor of a near-term event. The mistake is inferring that the disease only exists when it becomes visible.

BOTTOM LINE The most valuable prevention window opens long before anyone feels unwell — and closes quietly while ten-year risk scores still look reassuring.

Read the full article on CuringHeartDisease.com

- A disease you are born with
- **See also:** From the Womb to Reversal: Undoing a Lifetime of Arterial Damage

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

12 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: You Can Wait Until Your Cholesterol Is 'Really Bad'

THE CLAIM There is little benefit to lowering atherogenic particles early, because treatment works just as well after decades of exposure.

THE HOOK *Arteries keep a ledger. Later treatment credits the account; it does not erase the entries.*

What the evidence actually shows

The Domanski analysis followed young adults for decades and found that cumulative LDL-C exposure predicted later cardiovascular events independently of the LDL-C measured at the end of follow-up. Two people arriving at midlife with the same number did not carry the same risk if they had taken different routes to get there. Time at exposure was doing work that the current value could not see.

Human genetics reinforces the point with unusual force. Variants that lower LDL-C modestly but from birth are associated with far greater proportional risk reduction per unit of LDL-C than the same reduction achieved by starting a drug in a patient's sixties. Ference and colleagues quantified this across LDL and blood pressure variants. The caveat matters: Mendelian estimates describe lifelong exposure beginning at conception and are not direct predictions of what starting a drug at 30 or 50 would deliver — duration, adaptation and population selection all differ. What they establish is that exposure duration is doing real work.

None of this argues against late treatment, which remains clearly beneficial — the statin trials were largely conducted in older adults. It argues that lowering atherogenic lipoproteins earlier prevents more cumulative exposure than the same reduction achieved decades later, and that 'wait and see' has a cost that does not appear on any single lab report. What early action should mean — diet, treating a secondary cause, medication, or simply closer monitoring — depends on the degree of elevation, family history, other risk factors, and the patient's own preferences.

THE GRAIN OF TRUTH Starting later still helps substantially, and no one should conclude that a late diagnosis is a lost cause. The point is about maximizing benefit, not about writing anyone off.

BOTTOM LINE Prevention is a dose-and-duration problem. Lower sooner generally beats lower later, because the arterial wall integrates over time.

Read the full article on CuringHeartDisease.com

- Can high cholesterol in your 20s and 30s cause a heart attack later even if you lower it?

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

13 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: Plaque Is Permanent Concrete

THE CLAIM Once atherosclerotic plaque forms it can only get worse; stabilization or regression is impossible.

THE HOOK *Plaque is living tissue with a metabolism, not poured concrete.*

What the evidence actually shows

A plaque is not a deposit sitting inside a pipe. It is a lesion within the artery wall containing lipid, macrophages and foam cells, smooth muscle cells, extracellular matrix, neovessels, and calcium — all of it turning over. Because plaque is biologically active tissue rather than an inert deposit, its lipid content, inflammation, fibrous tissue and calcification can all change when the arterial environment changes.

The proof of principle is old. Armstrong, Warner and Connor showed in 1970 that atheroma in rhesus monkeys regressed when a high-cholesterol diet was reversed. Modern serial imaging has confirmed the same phenomenon in humans: ASTEROID demonstrated regression of percent atheroma volume under very high-intensity statin therapy, and GLAGOV showed additional regression when evolocumab was added, with the effect scaling as achieved LDL-C fell.

But the more important change may be compositional rather than volumetric. Imaging studies under intensive lipid lowering report less lipid-rich plaque and increased calcification or fibrous tissue — changes consistent with greater stability. Volume regression is usually modest, a few percent. The interpretation that stabilization accounts for much of the early clinical benefit is widely held and mechanistically plausible, but it is an inference from imaging and event data rather than something any single trial has quantified directly.

THE GRAIN OF TRUTH Regression is real but generally small and slow, and it is not the same as clearance. Anyone promising that plaque can be dissolved away is overselling what the imaging trials show.

BOTTOM LINE Expect stabilization rather than disappearance — and understand that a plaque does not have to vanish for the risk it carries to fall substantially.

Read the full article on CuringHeartDisease.com

- Can heart disease be reversed?
- **See also:** Reversal of Coronary Atherosclerosis: Mechanistic Insights from Human and Primate Studies

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14 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: A Stent Cures Atherosclerosis

THE CLAIM Opening one narrowed segment removes the underlying disease from the coronary arteries.

THE HOOK *A stent fixes a place. Prevention treats a process.*

What the evidence actually shows

Atherosclerosis is diffuse. By the time one lesion is severe enough to stent, the same biology is operating throughout the coronary tree, in segments that look unremarkable on angiography because the artery has remodelled outward to preserve the lumen. Treating the visible narrowing leaves the process untouched everywhere else.

The trial evidence on stable disease is consistent and, for many patients, counterintuitive. COURAGE found that adding PCI to optimal medical therapy in stable coronary disease did not reduce death or myocardial infarction. ISCHEMIA, in patients with moderate-to-severe demonstrable ischemia, reached the same conclusion for the primary endpoint. Revascularization reliably relieves angina and improves quality of life, and in acute coronary syndromes meeting the criteria for urgent intervention it is guideline-indicated and reduces mortality. What these trials show is narrower than 'stents don't help': adding routine PCI to guideline-directed medical therapy did not reduce death or infarction in these broad stable populations. Prognostic benefit still applies in selected settings — left main disease, certain complex anatomy, reduced left-ventricular function — and PCI and CABG are not interchangeable. Current chronic coronary disease guidelines treat revascularization as an individualized decision.

There is also a mismatch between where stents go and where events come from. PROSPECT followed patients after acute coronary syndrome with intravascular imaging and found that many subsequent events were associated with lesions that had not appeared severely obstructive at baseline. Attributing events to treated versus untreated sites is methodologically complex and not all of these events were infarctions or deaths — but the pattern illustrates why treating the most visible narrowing does not eliminate risk elsewhere.

THE GRAIN OF TRUTH In acute coronary syndromes where urgent revascularization is indicated, timely PCI reduces mortality — the benefit depends on presentation and timing rather than applying to every infarction equally. Stents also relieve genuine, disabling angina. The claim being corrected here is specifically about curing the disease, not about whether the procedure has value.

BOTTOM LINE A stent treats a specific lesion. Long-term protection still requires managing the diffuse disease — lipids, blood pressure, tobacco, glucose, physical activity, appropriate antithrombotic therapy, and adherence.

Read the full article on CuringHeartDisease.com

- Treating the Biology, Not the Pipes: The Radical New Approach to Heart Disease
- **See also:** The Ins and Outs of Cardiac Stents

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Medical disclaimer: This article is for education only. It is not medical advice, it has not been reviewed by a treating clinician, and it cannot account for your individual circumstances. Nothing here should be used to start, stop, or change any medication, dose, diet, supplement, or exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

15 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: A Heart Attack Is Just a Slowly Closing Pipe

THE CLAIM Heart attacks happen only when a plaque gradually grows until the artery reaches complete blockage.

THE HOOK *Most heart attacks are a sudden clot on a damaged surface, not a slow squeeze to zero.*

What the evidence actually shows

The dominant mechanism is acute thrombosis on a disrupted plaque. A lipid-rich lesion with a thin fibrous cap ruptures, exposing highly thrombogenic core material to flowing blood; platelets aggregate and a clot forms within minutes. A second mechanism, plaque erosion, involves loss of the endothelial layer over a more fibrous lesion without frank rupture. Intravascular OCT series such as Jia's put it at a substantial minority of acute coronary syndromes, disproportionately in younger patients, women and smokers — though reported proportions vary considerably with the population studied and whether the diagnosis is made by pathology or by imaging.

Crucially, the culprit lesion frequently was not the tightest narrowing beforehand. Pathological series from sudden coronary death and prospective imaging studies both show that many culprit sites were only mildly to moderately stenotic before the event. The features associated with rupture are compositional — large necrotic core, thin cap, positive remodelling, spotty calcification, macrophage infiltration. Luminal stenosis is an incomplete predictor of which lesion will cause a future event, and composition is not a perfect one either.

This explains a pattern that otherwise looks paradoxical: total plaque burden predicts events better than maximum stenosis. A wide field of moderate, lipid-rich disease presents many possible culprits. A single severe stenosis may be clinically important, but the worst percentage on the report does not capture how many other plaques exist or what they are made of.

THE GRAIN OF TRUTH Severe stenosis is not irrelevant — it causes angina, it correlates with total burden, and very tight lesions do occlude. But many infarctions arise from acute thrombosis at lesions that were not previously the most severe narrowing.

BOTTOM LINE Stability and total plaque burden predict events better than the worst percentage on the report.

Read the full article on CuringHeartDisease.com

- A New Way of Looking Inside Your Heart Without Surgery - The Best Way to Predict a Heart Attack

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

THE BIG LIE: Inflammation Makes Cholesterol Irrelevant

THE CLAIM Because plaque is an inflammatory lesion, ApoB-containing particles are innocent bystanders.

THE HOOK *Inflammation is the fire. ApoB is the fuel that was delivered first.*

What the evidence actually shows

Sequence matters, with one caveat: retention of ApoB particles in susceptible arterial regions is the central initiating event, but endothelial biology, disturbed flow and immune signaling interact with it from the earliest stages rather than waiting their turn. ApoB particles cross the endothelium, bind to intimal proteoglycans, and are held in place. Retained and modified lipoproteins then trigger the immune response — monocyte recruitment, macrophage foam cell formation, cytokine signaling. The response-to-retention model, formalized by Williams and Tabas, has held up for three decades because it explains why the disease occurs where it occurs and why it does not occur without sufficient particle exposure.

The anti-inflammatory trials confirm the partnership rather than displacing lipids. CANTOS showed that canakinumab reduced events without changing LDL-C, and LoDoCo2 showed the same for low-dose colchicine in chronic coronary disease — both real, both important, both delivering benefit on top of statin therapy rather than instead of it. Ridker's pooled analysis of three statin trials found that residual inflammatory risk and residual cholesterol risk both predicted outcomes, and that they identify different patients.

A further line of evidence is a negative one: in populations with lifelong very low ApoB, atherosclerotic disease is markedly reduced. Inflammation can accelerate and destabilize atherosclerosis and shapes how dangerous a plaque becomes, but classic lipid-rich plaque remains strongly dependent on exposure to ApoB-containing particles. Other arterial diseases exist — the inflammatory vasculopathies — but they are not this disease.

THE GRAIN OF TRUTH Residual inflammatory risk is genuinely underappreciated, and in some statin-treated patients hsCRP predicts events better than LDL-C does. That is an argument for measuring inflammation where it would change management — not for adding hsCRP to every panel.

BOTTOM LINE Lower the fuel first: ApoB reduction matched to risk. Where established disease and persistent inflammatory risk coexist, anti-inflammatory therapy is a discussion to have with a clinician — colchicine requires patient selection, and canakinumab is not used for routine prevention.

Read the full article on CuringHeartDisease.com

- Which Came First - the Lipid or the Inflammation? Apolipoprotein B and the Chicken-and-Egg Problem of Atherogenesis
- **See also:** Inflammation, Lipoproteins, and the End of the LDL-Centric Era

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17 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: A Calcium Score of Zero Means Zero Atherosclerosis

THE CLAIM A CAC score of zero proves there is no coronary plaque, and therefore makes a very high LDL harmless.

THE HOOK *Zero calcium is not zero plaque. Calcium is the scar, not the wound.*

What the evidence actually shows

Coronary artery calcium is one of the strongest negative risk markers available in asymptomatic middle-aged and older adults, and a score of zero legitimately moves risk downward. But calcification is a late feature of atherosclerosis. Early lesions — soft, lipid-rich, non-calcified, and disproportionately the rupture-prone ones — do not register on a calcium scan at all.

Noncalcified plaque is not one thing — it ranges from relatively fibrous lesions to lipid-rich, higher-risk phenotypes — and none of it is counted by a standard calcium score. This blind spot is also not evenly distributed. It is largest in exactly the people most likely to be reassured by a zero: those under about forty or forty-five, in whom disease has not had time to calcify, and those with familial hypercholesterolemia or other severe hypercholesterolemia. In the multinational FH cohort study, a substantial proportion of patients with a CAC of zero nonetheless had non-calcified plaque on CT angiography.

A zero also has an expiry date. MESA data on the 'warranty period' show that the duration of low risk conferred by a zero score depends heavily on age and overall risk factor burden — a matter of years, not a lifetime, and shorter in higher-risk individuals.

THE GRAIN OF TRUTH In selected borderline- or intermediate-risk adults, a CAC of zero is genuinely powerful and is one of the findings that can support a decision to treat less intensively or to wait. It does not carry the same weight against severe hypercholesterolemia, diabetes, smoking, or strong premature family history. The test is excellent; the over-reading of it is the problem.

BOTTOM LINE A zero lowers estimated near-term risk in the right patient. It does not erase the significance of severe or prolonged ApoB exposure, and it is not a clearance certificate.

Read the full article on CuringHeartDisease.com

- Power of Zero: No Calcium – No Risk?
- **See also:** The Heart Test Your Doctor Isn't Ordering

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

THE BIG LIE: A Normal Stress Test Means Clean Arteries

THE CLAIM Performing well on a stress test rules out substantial coronary plaque.

THE HOOK *A stress test asks whether blood gets through. It does not ask what is in the wall.*

What the evidence actually shows

Stress testing is a physiology test. It asks whether blood flow becomes inadequate under demand — and it answers that question reasonably well. What it does not do is image the artery wall. Crucially, anatomy and physiology do not map onto each other by any single threshold: in the FAME study, a substantial share of angiographically severe-looking lesions were not physiologically significant, and plenty of moderate ones were. Diffuse disease and microvascular dysfunction can produce ischemia with no single tight epicardial lesion at all. A normal stress result does not exclude non-obstructive plaque, because such plaque may simply not reduce perfusion enough to register.

Compensatory mechanisms widen the gap further. Arteries remodel outward as plaque accumulates, preserving the lumen while the wall thickens, so substantial disease can develop with a normal-caliber channel. Collateral circulation can maintain perfusion around a significant stenosis. High fitness may also allow someone to remain functional despite substantial disease. The extent to which athletic adaptation reduces the sensitivity of stress testing has not been established by the imaging cohorts discussed elsewhere in this collection, and should be treated as a plausible hypothesis rather than a demonstrated effect.

The practical consequence is a specific and recurrent clinical trap: a reassuring stress test in a fit patient with a strong family history and an untested ApoB. The test answered its question correctly. It was simply not the question that needed asking.

THE GRAIN OF TRUTH Stress testing is the right test when the question is inducible ischemia, exercise capacity, symptoms under load, or the functional significance of a known lesion — and it detects things anatomy cannot, including arrhythmia, blood-pressure response and functional limitation. Contemporary guidelines increasingly favor coronary CT angiography when the question is instead whether plaque or obstructive anatomy is present. Different tests, different questions.

BOTTOM LINE Match the test to the question. Ischemia and exercise symptoms need functional testing; plaque and stenosis need anatomical imaging; and a lipid panel measures atherogenic exposure, not plaque.

Read the full article on CuringHeartDisease.com

- How Being Fit Can Hide a Heart Attack
- **See also:** How Your Workout Blocks Pain: The Deadly Secret of the Athlete's Heart

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19 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: Calcified Plaque Is Always More Dangerous Than Soft Plaque

THE CLAIM More calcium automatically means a plaque is more likely to rupture.

THE HOOK *Dense calcium may mark a steadier plaque. Total calcium still marks accumulated disease.*

What the evidence actually shows

Two things are true at once, and holding both is the whole skill here. At the level of an individual lesion, densely calcified plaque is generally more stable than lipid-rich plaque with a thin fibrous cap — the classifications derived from sudden coronary death pathology are unambiguous that thin-cap fibroatheroma, not dense calcification, is the rupture-prone phenotype. Spotty microcalcification within a soft lesion is a different and more concerning finding than a uniformly calcified one.

At the level of a whole person, however, a higher total calcium score reliably predicts a higher absolute event rate. This is not a contradiction: calcium is a marker of how much disease has accumulated overall, and someone with a great deal of calcified plaque has had a great deal of atherosclerosis. Density signals stability; quantity signals burden.

Treatment illustrates the distinction neatly. Statins can increase plaque calcium density while reducing lipid content and event rates, so an Agatston score may rise on effective therapy. Serial calcium scoring is genuinely hard to interpret — the score combines area and density, and is affected by measurement variability, baseline burden, aging and real progression. A rising score under treatment cannot be read as simple failure, and equally cannot be read as proof of success.

THE GRAIN OF TRUTH This nuance is easy to over-rotate into 'calcium is good.' It is not. A CAC of 400 is a serious finding whatever the density, and the composition argument applies within a plaque, not across patients.

BOTTOM LINE At lesion level, dense macrocalcification is often steadier than thin-cap lipid-rich plaque. At patient level, a larger total burden still means more disease and higher risk. Read composition and quantity as two separate signals.

Read the full article on CuringHeartDisease.com

- Coronary Calcium and Athletic Risk
- **See also:** Peak Fitness, Hidden Calcium: The Athlete's Paradox of Plaque Buildup

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THE BIG LIE: Feeling Fine Means Your Arteries Are Fine

THE CLAIM Coronary disease produces reliable warning symptoms long before it becomes dangerous.

THE HOOK *Coronary disease can stay completely silent until the heart attack.*

What the evidence actually shows

Coronary atherosclerosis commonly stays silent until the disease is advanced or a plaque becomes acutely unstable. Classic exertional angina reflects impaired coronary flow reserve, which can arise from obstructive stenosis, diffuse disease, vasospasm or microvascular dysfunction — and none of those is an early finding. A plaque can grow for decades, remodel outward, and announce nothing. Coronary disease can and does remain undetected until myocardial infarction or sudden cardiac arrest.

High functional capacity does not exclude coronary plaque. Substantial subclinical disease has been documented in asymptomatic masters athletes with otherwise favorable risk profiles, which is the finding that matters here. Whether fitness additionally masks symptoms — through collateral recruitment, high flow reserve, or a trained tolerance for exertional discomfort — is a common clinical intuition that the observational evidence does not establish. Treat it as a reason not to be reassured by fitness, not as a demonstrated mechanism.

The practical implication is that symptoms are not a risk assessment. Assessment starts with standard lipids, consideration of ApoB and Lp(a), blood pressure, glycemic status, smoking, family history and other clinical factors. Imaging belongs in the picture only where it is likely to meaningfully refine risk or change management — it is not a routine step for every asymptomatic adult.

THE GRAIN OF TRUTH Symptoms, when they do appear, matter enormously and should never be dismissed. The failure mode here is the inverse inference: treating the absence of symptoms as evidence of absence of disease.

BOTTOM LINE Silence is the natural state of this disease, not a signal of health. Screen on risk, not on how you feel.

Read the full article on CuringHeartDisease.com

- You Feel Fine - That's the Problem
- **See also:** Fit, Fast, and Almost Dead: How a World-Class Cyclist Discovered Silent Heart Disease

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21 PLAQUE, ARTERIES AND DISEASE

THE BIG LIE: If the Arteries Look Clean, It Was Not a Real Heart Attack

THE CLAIM A myocardial infarction with non-obstructive coronary arteries on angiography must be a false alarm or a lab error.

THE HOOK *MINOCA is a real heart attack with a real cause — just not the one the angiogram was looking for.*

What the evidence actually shows

Myocardial infarction with non-obstructive coronary arteries accounts for somewhere between 5% and 10% of all infarctions, and it is a working diagnosis rather than a final mechanism — the label applies once infarction criteria are met and obstructive disease is excluded, at which point the search begins rather than ends. Patients are younger on average and more often women, and their prognosis is not benign — mortality at twelve months is meaningfully elevated compared with people who did not have an event.

The underlying mechanisms are identifiable when looked for. Plaque erosion or rupture of a non-obstructive lesion with subsequent spontaneous thrombolysis leaves an angiographically unimpressive artery behind. Coronary vasospasm, spontaneous coronary artery dissection, coronary microvascular dysfunction, and coronary embolism all produce genuine myocardial injury without a fixed stenosis. Intracoronary imaging and cardiac MRI frequently reveal what angiography missed.

MINOCA is best understood as a working diagnosis rather than a final one: the patient has met infarction criteria and has non-obstructive arteries, and the job that follows is to identify which mechanism was responsible — typically with cardiac MRI, intracoronary imaging, and provocation testing where indicated. Treatment then follows the mechanism. Where plaque disruption is found or strongly suspected, secondary prevention generally follows an atherothrombotic path. That is not automatically the right answer for spontaneous coronary artery dissection, where a conservative approach is often preferred, or for vasospasm, which is managed differently again. The point for a patient is that the mechanism should be pursued and named — applying one template to all of them is its own error.

THE GRAIN OF TRUTH Myocarditis, takotsubo syndrome, and pulmonary embolism are not types of MINOCA — they are alternative diagnoses that the workup is designed to identify and rule out before MINOCA stands. That distinction is the reason the workup exists, not a reason to skip it.

BOTTOM LINE An open artery does not undo the infarct. MINOCA needs a mechanism identified and then treatment matched to it — not a discharge slip.

Read the full article on CuringHeartDisease.com

- The Invisible Infarction: A Deep Research Verification of Mechanisms, Diagnosis, and Management in MINOCA

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PART 3

Diet and Nutrition

What to eat, what to avoid, and the evidence behind every claim — including the great diet debates.

Research hub: Diet and Nutrition

22 DIET AND NUTRITION

THE BIG LIE: Mediterranean and Whole-Food Plant-Based Diets Are Interchangeable

THE CLAIM Both patterns affect ApoB, plaque, and clinical outcomes in essentially the same way.

THE HOOK *Two good diets can sit on different rungs of the same ladder.*

What the evidence actually shows

These patterns have different evidence bases and different mechanisms, and conflating them costs precision. A Mediterranean pattern carries the strongest randomized outcome evidence in primary prevention — PREDIMED reported reduced major cardiovascular events with supplementation by extra-virgin olive oil or nuts — and it is unusually sustainable, which matters more than any nutrient argument if a diet has to last thirty years.

A very-low-fat whole-food plant-based pattern is aimed at a different target. By minimizing saturated fat and dietary cholesterol and maximizing viscous fiber, it is designed to push LDL-C and ApoB further than a Mediterranean pattern typically does — though no head-to-head randomized trial has compared the two patterns on ApoB with matched adherence, so the ranking is inferred from their separate effects on the known dietary levers rather than demonstrated directly. The Lifestyle Heart Trial and its extension paired this with exercise and stress management and reported angiographic regression — a smaller and less generalizable trial than PREDIMED, but aimed at plaque rather than at population event rates.

The useful framing is a ladder rather than a rivalry. If the goal is broad risk reduction with high adherence, a Mediterranean pattern is an excellent rung. If the goal is the largest achievable dietary ApoB reduction — someone with established disease pursuing regression — the very-low-fat plant-based rung goes higher, at the cost of being harder to sustain.

THE GRAIN OF TRUTH The ladder is an editorial framework for thinking about dietary intensity, not an established comparative hierarchy. Both patterns beat a typical Western diet decisively, and the difference between them is far smaller than the difference between either one and what most people actually eat.

BOTTOM LINE Match the pattern to what you need it to do — broad outcome evidence, or the largest achievable lipid reduction — and then to what you can sustain. The ranking between them is a working framework, not an established hierarchy.

Read the full article on CuringHeartDisease.com

- What is the difference between Mediterranean and whole-food plant-based diets for heart disease?

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

23 DIET AND NUTRITION

THE BIG LIE: Only One Named Diet Can Reverse Heart Disease

THE CLAIM A single branded dietary program has uniquely proven that it reverses coronary disease.

THE HOOK *No diet owns the biology of regression. The mechanism is available to anyone who reaches it.*

What the evidence actually shows

Regression appears to require atherogenic particle exposure falling far enough, for long enough, alongside control of the other causal drivers — blood pressure, glycemia, weight, smoking and physical activity all contribute rather than ApoB acting alone. That is a physiological condition, not a brand. A large sustained reduction in atherogenic lipoprotein exposure is one biologically plausible contributor, and the imaging trials demonstrating regression most convincingly used drugs rather than food — which at least suggests the mechanism is exposure reduction rather than anything proprietary. Whether different dietary patterns achieving similar ApoB changes produce comparable plaque change has not been directly tested.

Branded programs also bundle. The Lifestyle Heart Trial combined a very-low-fat plant-based diet with aerobic exercise, stress management training, smoking cessation, and group support. The result was real, but the design cannot attribute it to the diet alone, and the participant numbers were small. Reading it as proof that one named diet uniquely reverses disease overstates what a bundled intervention in a few dozen people can establish.

This matters practically because branding creates false binaries. Patients who cannot sustain one named program frequently conclude that regression is closed to them. It is biologically plausible that another sustainable approach producing comparable improvements in ApoB and the other major risk factors would deliver similar benefit — though no comparative imaging trial has tested that directly.

THE GRAIN OF TRUTH The very-low-fat whole-food plant-based approach does have the strongest direct angiographic-regression pedigree among dietary patterns, and that deserves acknowledgement rather than flattening — which is why the linked article carries the headline it does. Read 'the only diet proven' as a claim about which pattern has been directly imaged, not as a claim that no other route to the same ApoB would fail.

BOTTOM LINE Ask what a diet does to ApoB, blood pressure, and glucose — and whether it can be sustained. The label is not the mechanism.

Read the full article on CuringHeartDisease.com

- The Only Diet Proven to Shrink Arterial Plaque (It's Not Mediterranean!)
- **See also:** Can Diet Really Reverse Heart Disease?

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24 DIET AND NUTRITION

THE BIG LIE: Seed Oils Are Poison

THE CLAIM Common plant oils inherently inflame arteries and are worse for the heart than saturated animal fats.

THE HOOK *The question is never 'is this food good?' It is 'good compared with what?'*

What the evidence actually shows

Nutrition works by substitution, because calories displace calories. The relevant comparison for a seed oil is what it replaces. Controlled feeding studies are consistent: replacing saturated fat with polyunsaturated fat lowers LDL-C and ApoB. Mensink's meta-analysis of sixty controlled trials quantified the effect on lipids, and the network meta-analysis by Schwingshackl and colleagues ranked oils and solid fats on the same basis. Replacing saturated fat with refined carbohydrate does not deliver the same benefit — which is where much of the confusion originates.

The linoleic acid inflammation argument has not held up where it has been measured. A systematic review of randomized controlled trials by Johnson and Fritsche found that increasing dietary linoleic acid did not raise circulating inflammatory markers in healthy adults. A pooled individual-level analysis of 30 cohorts by Marklund and colleagues found that higher biomarker-measured linoleic acid was associated with lower, not higher, cardiovascular event rates. These are selected circulating markers in largely healthy adults, and observational associations — they do not close off every proposed oxidation or postprandial mechanism, but they are inconsistent with the claim that linoleic acid broadly raises risk.

Two separate issues also get merged here. Repeatedly reheated commercial frying oil is chemically not the same product as fresh oil, and concerns about degradation compounds in that setting are a live research question rather than a settled one. Either way it is a question about a cooking process, not about using fresh unsaturated plant oil in place of butter or lard at home.

THE GRAIN OF TRUTH 'Seed oil' is a marketing category, not a uniform exposure — soybean, canola, sunflower and corn oils differ in fatty-acid profile and use. And many foods containing them are ultra-processed and energy-dense, arriving alongside refined starch, sodium and low fiber, which makes it hard to assign risk to the oil itself.

BOTTOM LINE Judge the swap, not the ingredient. Unsaturated plant oil replacing saturated fat lowers ApoB; that is the finding.

Read the full article on CuringHeartDisease.com

- Are Seed Oils Healthy, True or False?

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25 DIET AND NUTRITION

THE BIG LIE: Butter Is Heart-Healthy Because It Is Natural

THE CLAIM A food's traditional or natural status determines its effect on ApoB and cardiovascular risk.

THE HOOK *'Natural' is a marketing category. Your LDL receptor has never heard of it.*

What the evidence actually shows

The liver responds to fatty acid composition, not provenance. Butter is high in saturated fatty acids — roughly half the product by weight, and a considerably larger share of its total fatty acids — dominated by palmitic and myristic acids. Myristic acid in particular raises LDL-C, largely by reducing hepatic clearance, though the size of that response varies with dietary context and between individuals. Fewer receptors means slower clearance of ApoB particles from plasma, which means a higher circulating particle count. That mechanism is indifferent to whether the fat came from a pasture or a factory.

The AHA presidential advisory on dietary fats pooled the core randomized trials and estimated that replacing saturated fat with polyunsaturated fat reduced cardiovascular events by roughly a quarter. That figure is a synthesis of a small set of older trials rather than a single head-to-head result, and it should be read as an estimate of dietary effect rather than as a like-for-like comparison with drug therapy. The comparator matters: the benefit appears when saturated fat is replaced by unsaturated fat, and largely disappears when it is replaced by refined starch and sugar.

Cheese and fermented dairy complicate the picture slightly and honestly: observational data associate them with less cardiovascular risk than their saturated fat content alone would predict, a proposed food-matrix effect that remains under investigation and is not established. Whatever it turns out to be, it is a claim about fermented dairy products, not a rehabilitation of butterfat as a category.

THE GRAIN OF TRUTH Butter in small quantities within an otherwise excellent diet is not the deciding factor in anyone's cardiovascular fate. The claim being corrected is that natural status confers cardiovascular benefit — not that any butter is catastrophic.

BOTTOM LINE Fatty acid composition and the overall pattern determine the outcome. How old or how traditional the food is does not enter the calculation.

Read the full article on CuringHeartDisease.com

- The Big Cholesterol Lie: Uncovering the Diet Industry's Organized Denial

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26 DIET AND NUTRITION

THE BIG LIE: Coconut Oil Is a Superfood

THE CLAIM Coconut oil's medium-chain triglycerides make it metabolically special and protective for the heart.

THE HOOK *Coconut oil raises LDL like the saturated fat it mostly is.*

What the evidence actually shows

The medium-chain triglyceride argument does not describe the product on the shelf. Definitions vary, and lauric acid at 12 carbons sits on the boundary — chemically it is sometimes classed as medium-chain, but physiologically it is absorbed and transported much more like a long-chain fat than like the caprylic and capric acids used in purified MCT products. Coconut oil is not the MCT of the supplement aisle. Coconut oil is approximately 80–90% saturated fat, a higher proportion than butter or lard.

Trial data match the composition. A meta-analysis of sixteen clinical trials published in Circulation found that coconut oil significantly raised LDL-C compared with non-tropical vegetable oils — by around 10 mg/dL — while producing no benefit on body weight, waist circumference, or measures of glycemia. HDL-C also rose, which is what generated much of the favorable marketing, and which the HDL evidence tells us not to bank on.

The 'traditional populations thrive on coconut' argument compares whole coconut consumed within a high-fiber, physically active, largely unprocessed dietary pattern against extracted oil added to a Western diet. These are not the same exposure.

THE GRAIN OF TRUTH Coconut oil is fine as a culinary ingredient used sparingly for flavor or texture. The correction is to the health claim, not to the existence of the food.

BOTTOM LINE Treat coconut oil as a saturated fat with a good publicist — not as a therapeutic agent.

Read the full article on CuringHeartDisease.com

- The Myth of Coconut Oil as a Health Food: How Industry Influences What You Eat!

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27 DIET AND NUTRITION

THE BIG LIE: Olive Oil Is a Medicine That Reverses Plaque

THE CLAIM Adding olive oil to any diet dissolves coronary plaque because of its polyphenols.

THE HOOK *Olive oil is an excellent replacement. It is not a solvent.*

What the evidence actually shows

The strongest evidence for olive oil comes from PREDIMED, where a Mediterranean pattern supplemented with extra-virgin olive oil reduced major cardiovascular events relative to a low-fat advice control. That is a trial of a dietary pattern with olive oil in it, in a Mediterranean population, against a comparator diet. It is not a trial of adding olive oil to whatever someone was already eating, and it did not measure plaque.

Mechanistically, olive oil's benefit is substitution plus matrix. Replacing saturated fat with monounsaturated fat modestly lowers LDL-C and ApoB, and extra-virgin oil carries polyphenols with plausible antioxidant and endothelial effects. Both are plausible. Neither has been shown to make olive oil independently reverse established coronary plaque — regression in the imaging literature required large sustained reductions in atherogenic particles, generally pharmacological. PREDIMED also cannot separate how much of its benefit came from the oil, the wider pattern, or the dietary support participants received.

Addition without substitution is the specific failure mode. Olive oil is about 120 calories per tablespoon. Poured generously onto an already energy-dense diet, it adds calories without displacing the saturated fat that was the problem — which can blunt the expected benefit and promote weight gain.

THE GRAIN OF TRUTH Extra-virgin olive oil is a genuinely good choice with respectable outcome evidence behind it, and it is a much better default cooking fat than butter. It just is not doing what the strongest claims say it does.

BOTTOM LINE Use olive oil to replace worse fats, not to supplement them — and expect improved risk factors, not dissolved plaque.

Read the full article on CuringHeartDisease.com

- Is Olive Oil Actually Healthy?

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28 DIET AND NUTRITION

THE BIG LIE: Dietary Cholesterol Is the Only Food Issue That Matters

THE CLAIM Avoiding cholesterol-containing foods is sufficient, regardless of saturated fat, fiber, calories, or overall pattern.

THE HOOK *Your blood cholesterol reflects how your body makes, absorbs and clears it — not just what the food label says.*

What the evidence actually shows

Endogenous synthesis outweighs dietary intake. The body manufactures substantially more cholesterol each day than a typical diet delivers, and most people partially down-regulate absorption and synthesis in response to what they eat. The AHA science advisory on dietary cholesterol reviewed this evidence and concluded that the effect of dietary cholesterol on serum LDL-C is smaller and more variable between individuals than was assumed for decades — the reasoning behind dropping a specific numerical daily limit in favor of an overall dietary pattern.

For most people, saturated fat is a larger and more consistent dietary determinant of LDL-C than dietary cholesterol is, acting largely on hepatic LDL-receptor expression — though individual responses vary, and fiber, weight change, energy balance, trans fat and plant sterols all contribute. Reduce receptor availability and ApoB particles linger in circulation regardless of how little cholesterol was eaten. This is why the composition of a food matters more than its cholesterol column: many high-cholesterol foods are also high in saturated fat, and it is generally the latter that is driving the lipid response.

The rest of the pattern carries real weight too. Viscous fiber interrupts enterohepatic bile acid recirculation and lowers LDL-C — Brown and colleagues quantified the effect across controlled trials — while plant sterols compete for micellar absorption, and energy balance and weight change alter lipoprotein metabolism substantially. The AHA advisory also documents genuine inter-individual variation, with hyper-responders and hypo-responders to dietary cholesterol both described. Whether people with diabetes respond differently has been raised but not settled — the evidence is inconsistent and heavily confounded by what those foods are eaten alongside. That variation is an argument for measuring your own response rather than assuming it.

THE GRAIN OF TRUTH Dietary cholesterol is not irrelevant, particularly at high intakes and particularly in hyper-responders and people with diabetes. It is one input among several, not the only one.

BOTTOM LINE Manage the whole pattern — saturated fat, fiber, energy balance, and substitution — hold it steady long enough to mean something, then review the lipid response with a clinician.

Read the full article on CuringHeartDisease.com

- Cholesterol: What Is It Good For?

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29 DIET AND NUTRITION

THE BIG LIE: Saturated Fat Is Red Meat's Only Cardiovascular Problem

THE CLAIM If you choose lean cuts and keep saturated fat low, red meat carries no remaining cardiovascular risk.

THE HOOK *The red meat question runs past saturated fat — into pathways nobody has yet pinned down.*

What the evidence actually shows

A recurring observation in cohort studies is that red and processed meat intake associates with cardiovascular risk to a degree that saturated fat content alone does not obviously account for. Residual confounding is a real alternative explanation for that gap and cannot be dismissed. But it has also driven a search for additional mechanisms, and several candidates now have experimental support.

The best characterized is the trimethylamine N-oxide pathway. Gut microbes metabolize dietary L-carnitine and phosphatidylcholine — both abundant in red meat — into trimethylamine, which the liver oxidizes to TMAO. Wang and colleagues linked the pathway to atherosclerosis in animal models; Koeth showed carnitine driving it; and Tang found that elevated fasting TMAO predicted major adverse cardiovascular events in patients undergoing coronary evaluation. Long-term vegans and vegetarians produced markedly less TMAO after a carnitine challenge in Koeth's work, consistent with differences in gut microbial metabolism.

But TMAO has a serious problem as a causal story, and it deserves stating plainly. Circulating TMAO tracks renal function closely, so it rises in people whose kidneys are already impaired — a group at high cardiovascular risk for entirely separate reasons. It also rises after eating fish, a food that associates with lower cardiovascular risk rather than higher. Whether TMAO is a mediator, a marker of impaired renal and metabolic health, or some of both remains unresolved. The other candidates are weaker still: Neu5Gc, a non-human sialic acid absorbed from red meat against which humans generate antibodies, is hypothesis-generating work rather than an established human cardiovascular mechanism, and heme iron is similar. The sodium and nitrite load in processed meat rests on firmer ground than either.

THE GRAIN OF TRUTH None of these pathways is established as causal in humans, and this section is a set of open questions rather than findings. The best-established concern still differs by product and comparator: saturated fat raises LDL-C, while processed meats carry substantial sodium and preservative exposure on top of that. Residual confounding also remains a live explanation for the cohort signal. The linked article's 'cover up' headline refers to the commercial framing around red meat marketing, not to a claim that TMAO causality has been proven and suppressed.

BOTTOM LINE 'Lean' addresses the best-established mechanism. Whether the proposed gut-microbial and immune pathways add meaningfully on top of it is genuinely unresolved — an argument for moderation and for watching the literature, not for alarm.

Read the full article on CuringHeartDisease.com

- Is red meat killing you through your gut? The great TMAO cover up
- **See also:** Is Your Immune System at War With Your Steak?

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30 DIET AND NUTRITION

THE BIG LIE: A Daily Glass of Wine Protects Your Heart

THE CLAIM Moderate drinking is cardioprotective, so a glass a day is a heart-healthy habit.

THE HOOK *The J-curve was mostly built out of who was in the comparison group.*

What the evidence actually shows

The protective association is real in the observational data, and the leading explanation for it is bias rather than biology. The reference category — non-drinkers — was contaminated by former drinkers who had quit because of illness, a bias known as sick-quitter. Moderate drinkers in Western cohorts also tended to be wealthier, better educated, more physically active, and better connected to healthcare. Confounding by socioeconomic status was doing much of the work attributed to ethanol.

Mendelian randomization reduces those confounders by using genetic variants that constrain alcohol tolerance — not a literal randomized drinking trial, and dependent on its own assumptions about pleiotropy and instrument strength, but far less vulnerable to sick-quitter bias. Millwood and colleagues, working in a Chinese cohort of half a million people where variants in ALDH2 and ADH1B strongly constrain intake, found that genetically predicted alcohol consumption raised blood pressure and stroke risk in a linear, dose-dependent way — with no protective threshold. Biddinger's analysis in UK Biobank found similarly that apparent benefit at low intake disappeared after adjustment for lifestyle confounders, with risk rising exponentially at higher intakes.

Alcohol also carries cardiovascular harms that the J-curve discussion tends to omit: it raises blood pressure dose-dependently, it is a well-recognized trigger for atrial fibrillation, and sustained heavy intake causes cardiomyopathy. The 2018 Global Burden of Disease analysis concluded that the intake minimizing total health loss across all causes is zero — a conclusion that has been debated since, and one that concerns overall burden rather than cardiovascular endpoints specifically.

THE GRAIN OF TRUTH For many adults the absolute cardiovascular effect of one drink a day is modest, and this is a personal decision with more than cardiovascular inputs. Total health risk varies substantially by age, sex, medical history and drinking pattern, and for some people no alcohol is the right answer. The correction here is to the idea that drinking is a health intervention.

BOTTOM LINE No dose of alcohol has been established to improve cardiovascular outcomes, so it should not be started or recommended for heart protection. People who do drink should weigh amount, pattern and their own medical risks honestly.

Read the full article on CuringHeartDisease.com

- The Glass Half-Empty: Why Science is Toasting Goodbye to “Heart-Healthy” Drinking

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.

31 DIET AND NUTRITION

THE BIG LIE: Fish Oil Capsules Are Proven Heart Protection

THE CLAIM Over-the-counter omega-3 supplements reliably prevent heart attacks.

THE HOOK *Eating fish and swallowing fish oil are not the same intervention, and the trials show it.*

What the evidence actually shows

The large, well-powered supplement trials were negative. VITAL randomized nearly 26,000 adults to 1 g daily of marine omega-3 and found no significant reduction in the primary composite cardiovascular endpoint. ASCEND, in over 15,000 people with diabetes, found no significant benefit on serious vascular events. These large trials did not show a significant reduction in their primary cardiovascular endpoints at the doses and formulations tested.

The apparent exception requires care. REDUCE-IT tested 4 g daily of icosapent ethyl — a purified, high-dose, prescription EPA product, not a supplement — in patients with elevated triglycerides on statin therapy, and reported a substantial reduction in events. But STRENGTH, testing a high-dose EPA/DHA combination in a similar population, was stopped for futility. Why the two trials diverged remains unresolved. Proposed explanations include the EPA-only formulation, the achieved EPA concentrations, differing biological effects of DHA, differences in population and adherence, and the possibility that REDUCE-IT's mineral-oil comparator adversely affected its control group.

Meanwhile eating fish is associated with better cardiovascular outcomes in many cohorts — though part of that association may reflect what the fish replaces and the broader dietary pattern it sits in. Whole foods carry a matrix, a substitution effect, and a set of dietary habits that an isolated capsule does not reproduce.

THE GRAIN OF TRUTH High-dose prescription icosapent ethyl has a legitimate evidence base and guideline standing in selected higher-risk patients — typically those with raised triglycerides already on statin therapy, though the tested and approved populations are more nuanced than any one profile. It is a prescribing decision, not a supplement-aisle purchase.

BOTTOM LINE Over-the-counter fish-oil capsules should not be assumed to prevent heart attacks. Eating fish is a reasonable part of a dietary pattern; high-dose prescription EPA is a separate, targeted therapy for selected higher-risk patients and an individual clinical decision.

Read the full article on CuringHeartDisease.com

- Heart Disease and Fish Oil

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PART 4

Exercise, Athletes and Aging

Performance science, cardiac adaptation in athletes, and the limits of fitness as protection.

Research hub: Exercise, Athletes and Aging

THE BIG LIE: Elite Fitness Makes You Immune to Heart Disease

THE CLAIM A high VO_2 max, competitive results, and a lean physique prevent coronary atherosclerosis.

THE HOOK *Fitness is among the most powerful risk reducers available. It is not immunity.*

What the evidence actually shows

Cardiorespiratory fitness is one of the most powerful predictors of survival known, and nothing here should be read as diminishing it. But risk reduction and immunity are different claims, and several imaging studies of predominantly male masters endurance athletes point firmly to the first.

Merghani and colleagues imaged masters endurance athletes selected for a low atherosclerotic risk profile — no smoking, favorable lipids, no diabetes — and found a higher prevalence of coronary plaque in the male athletes than in matched sedentary controls, including more calcified plaque and more athletes with a CAC above 300. Aengevaeren's study of lifelong exercise volume found the most active athletes had a greater coronary plaque burden, though with a more calcified and plausibly more stable composition.

These are cross-sectional studies in largely male, self-selected cohorts, and they establish that plaque is present rather than explaining why. The proposed mechanism — that training does not alter the physical chemistry of ApoB retention, so an athlete with elevated lifetime ApoB, elevated Lp(a), or inherited risk runs the same accumulation process as anyone else — comes from the wider lipid literature, not from these cohorts, which were deliberately selected for low conventional risk. What the athlete studies show is that high fitness does not exclude coronary plaque and cannot substitute for risk assessment.

THE GRAIN OF TRUTH Fit people still do far better on average than unfit people. These studies examined plaque presence and composition rather than long-term outcomes, so they should not be read as evidence that endurance exercise raises clinical event risk. The finding is that plaque exists despite the fitness.

BOTTOM LINE Fitness substantially improves health and survival, but it does not rule out coronary plaque. Athletes need the same lipid panel, family history and risk assessment as everyone else.

Read the full article on CuringHeartDisease.com

- Fit, Fast, and Almost Dead: How a World-Class Cyclist Discovered Silent Heart Disease

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33 EXERCISE, ATHLETES AND AGING

THE BIG LIE: You Can Outrun High ApoB

THE CLAIM Enough training volume neutralizes years of exposure to atherogenic lipoproteins.

THE HOOK *Training improves nearly every cardiovascular risk domain — except, reliably, the particle burden.*

What the evidence actually shows

Exercise improves endothelial function, insulin sensitivity, blood pressure, autonomic balance, inflammatory tone, body composition, and mortality across essentially every population studied. What it does not reliably do is produce a large reduction in atherogenic particle number. Reviews of training trials, including Mann and colleagues' synthesis across aerobic, resistance, and combined modalities, find that the most consistent lipid effects are on triglycerides and HDL-C, while changes in LDL-C are smaller and inconsistent and depend heavily on accompanying weight and dietary change. Improvements in triglycerides and HDL-C reflect better metabolic health, and triglyceride-rich remnant particles are themselves atherogenic — but neither number is a substitute for correcting an elevated ApoB count.

The arithmetic follows. Someone whose ApoB sits well above target is facing a gap that training alone is unlikely to close, while the exposure integral keeps accumulating in the meantime. This is the specific situation in which highly disciplined athletes lose years — doing everything right in a domain that cannot solve the problem they actually have.

The constructive version of this is not a trade-off but a stack: keep the training, and address the particle burden separately by the means that actually move it. The athlete imaging cohorts were not designed to test whether athletes with low ApoB fare better than athletes with high ApoB, so that combination is an inference from the wider lipid literature rather than a finding from those studies — but it is where the mechanism points.

THE GRAIN OF TRUTH Exercise reduces cardiovascular events through many pathways, and would be expected to lower absolute risk even when ApoB stays high — an inference from the wider exercise literature rather than something the studies here tested directly. Training can also lower ApoB indirectly, through weight loss and improved insulin sensitivity. It is additive protection, not substitutable protection.

BOTTOM LINE Keep training, then measure the result. Where atherogenic particle burden stays above the risk-based lipid goal you and your clinician have set — which may be expressed as ApoB, depending on the guideline in use — that gap is closed by dietary, metabolic and, where appropriate, pharmacological means.

Read the full article on CuringHeartDisease.com

- Do endurance sports help or hurt arteries?

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THE BIG LIE: Exercise Erases Plaque

THE CLAIM Training physically scrubs cholesterol deposits out of artery walls.

THE HOOK *Exercise does not scrub the artery clean — and the calcified plaque seen in some athletes does not prove training put it there.*

What the evidence actually shows

There is no mechanism by which mechanical flow removes lipid from within the arterial intima. Plaque sits in the vessel wall, not in the lumen, and shear stress acting on the endothelial surface does not extract material from beneath it. Regression requires reducing the influx of atherogenic particles and enabling macrophage efflux — a metabolic process, not a hydraulic one.

What the athlete imaging data show is a difference in composition at one point in time — not a shift. Aengevaeren's cohort found that the most active athletes had more plaque overall, with a higher proportion calcified and a lower proportion mixed than less active participants; Merghani found a similar predominance of calcified lesions. Because neither study imaged the same plaques before and after a training exposure, they cannot establish that exercise converted soft plaque into calcified plaque. What they establish is that highly trained people can carry a lot of plaque, much of it calcified.

Densely calcified plaque is generally regarded as the more stable phenotype, which is reassuring as far as it goes. But these cohorts were not powered for clinical events, so any explanation built on their event rates is speculation resting on a premise the studies did not establish. And a plaque is still a plaque: whatever its composition, it reflects an exposure that has not been addressed.

THE GRAIN OF TRUTH The practical message is emphatically not to train less — the survival evidence for fitness is overwhelming. These are observational, cross-sectional cohorts showing an association between training volume and plaque composition. They do not show causation in either direction, and whether that composition translates into fewer events in athletes has not been tested.

BOTTOM LINE Exercise delivers major cardiovascular benefits, but it has not been shown to erase established plaque or to stabilize it. Reducing plaque burden turns on lowering atherogenic particle exposure, alongside blood pressure, glucose and tobacco.

Read the full article on CuringHeartDisease.com

- Exercise Doesn't Erase Arterial Plaque - It Turns It to Stone!

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35 EXERCISE, ATHLETES AND AGING

THE BIG LIE: Cardio Is the Only Exercise That Helps Your Heart

THE CLAIM Resistance training builds muscle but contributes nothing to cardiovascular health.

THE HOOK *The heart benefits from lifting too — and a surprisingly modest weekly dose may capture most of it.*

What the evidence actually shows

Resistance training has its own cardiovascular evidence base, separate from aerobic exercise — though it is observational. In a large cohort followed for over a decade, Liu and colleagues found resistance exercise associated with lower cardiovascular event and mortality rates, with the association persisting after adjustment for aerobic activity. Stamatakis's pooled analysis across eleven population cohorts reported a similar association for strength-promoting exercise. These are cohorts, not randomized trials, so they establish association rather than proof of independent causal benefit.

The mechanisms are partly distinct from aerobic training, and here the evidence is randomized rather than observational. A meta-analysis of 46 randomized trials in over 2,000 adults with type 2 diabetes found resistance training lowered HbA1c and fasting glucose against non-exercise control, and Cornelissen and Smart's pooled analysis of exercise trials found dynamic resistance training reduced resting blood pressure. Resistance work also improves body composition in ways that reduce visceral adiposity, and preserves muscle mass and strength, which become increasingly important for physical function, glucose metabolism and fall prevention with age.

The dose-response also looks different from aerobic exercise. The association appears at relatively low volumes — on the order of one to two sessions weekly in several analyzes — and appears to flatten at higher volumes. Some analyzes suggest it may reverse at very high volumes, but the participant numbers at that end are small and confounding is difficult to exclude, so that tail should be held loosely.

THE GRAIN OF TRUTH Aerobic fitness has an especially strong and extensively documented association with cardiovascular survival; the cohorts here were not designed to rank the two against each other. Resistance training provides physiological and functional benefits that complement aerobic exercise, which is why guidelines recommend both.

BOTTOM LINE Treat two sessions of resistance work weekly as cardiovascular training, not just cosmetic — with attention to technique and sensible progression, and medical review first where that is warranted.

Read the full article on CuringHeartDisease.com

- Can pumping iron help your pumping heart?
- **See also:** Stop Wasting Away as You Age…the Emerging Science of Antiaging!

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THE BIG LIE: A Young Athlete's Cardiac Arrest Is Random Bad Luck

THE CLAIM Sudden cardiac death in young athletes is unpredictable and unpreventable, so screening and planning are pointless.

THE HOOK *Most of these events have a cause, and survival depends on something the venue decided months earlier.*

What the evidence actually shows

Sudden cardiac death in young athletes is rare but not random. The identified causes cluster into a recognizable set: hypertrophic cardiomyopathy, anomalous coronary artery origin, arrhythmogenic cardiomyopathy, ion channelopathies including long QT and catecholaminergic polymorphic ventricular tachycardia, myocarditis, and commotio cordis. Maron's analysis of 1,866 deaths in US athletes established the distribution, and Harmon's work quantified incidence in NCAA athletes — with higher reported incidence among male athletes and among Black male basketball players in particular — a disparity that should not be read as a simple biological effect of race, and that likely reflects sport distribution, screening access, structural factors and case ascertainment alongside any biological contribution.

In athletes over about 35, the picture shifts decisively toward atherosclerotic coronary disease — which is the same disease this collection is about, and which is modifiable. Warning signs also precede a meaningful fraction of events: exertional syncope, exertional chest pain, unexplained performance decline, and a family history of sudden death under 50 are all findings that warrant evaluation rather than reassurance.

Survival is where preparation shows up most starkly. Drezner's prospective registry of US high schools found that immediate bystander CPR and rapid on-site defibrillation were among the strongest modifiable determinants of survival, alongside factors nobody controls such as presenting rhythm, underlying cause and location. Survival rates in venues with a rehearsed emergency action plan and an accessible AED are dramatically higher than where the response has to be improvised.

THE GRAIN OF TRUTH Having an identifiable cause is not the same as being detectable in advance, and many events remain hard to predict through routine screening. Universal ECG screening in particular is genuinely contested on cost-effectiveness and false-positive grounds, and history, examination, ECG, echocardiography and genetic testing answer different questions. Emergency preparedness is not contested.

BOTTOM LINE Treat exertional syncope, exertional chest pain, and a family history of early sudden death as findings to investigate — and make sure the venue has an AED and a plan.

Read the full article on CuringHeartDisease.com

- How not to die young

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PART 5

Risk, Genetics and Special Populations

Who is most at risk, why genetics matters, and what changes for specific groups.

Research hub: Risk, Genetics and Special Populations

THE BIG LIE: Family History Means Prevention Is Futile

THE CLAIM Genetic risk fixes your destiny, so lifestyle, testing, and medication cannot meaningfully change outcomes.

THE HOOK *Genes set the starting line and the slope. They do not set the finish.*

What the evidence actually shows

The most directly relevant evidence is Khera's analysis across three prospective cohorts and a randomized trial population, using a polygenic risk score for coronary disease. Participants in the highest genetic risk quintile who adhered to a favorable lifestyle — not smoking, no obesity, regular physical activity, a healthy diet — had roughly half the coronary event rate of those at equally high genetic risk with an unfavorable lifestyle. Because lifestyle was observed rather than randomly assigned, that is a strong association rather than a proven causal halving. But high genetic risk clearly raised the baseline without eliminating the gradient across lifestyle.

Familial hypercholesterolemia demonstrates the same principle at the extreme end. Untreated heterozygous FH carries a very high lifetime risk of premature coronary disease. Identified early and treated adequately, that trajectory changes substantially — which is precisely why the European Atherosclerosis Society guidance frames FH as an underdiagnosis problem rather than an untreatable one. The tragedy of FH is missed detection, not absent therapy.

Family history is also a summary variable that hides actionable specifics. A strong history can resolve into a measurable, treatable finding — an elevated Lp(a), an FH-consistent lipid profile, early hypertension, diabetes, smoking patterns, or a shared environment — though it sometimes remains unexplained after all of those have been checked. Investigating it converts a vague inheritance into a target.

THE GRAIN OF TRUTH Genetic risk is real and substantial, and lifestyle does not cancel it. In Khera's analysis a favorable lifestyle was associated with lower risk at every genetic-risk level, but it did not erase the gap between the high- and low-risk groups — which is an argument for treating high genetic risk more intensively, not for giving up. These are group-level estimates and do not predict any individual's outcome.

BOTTOM LINE A strong family history supports earlier and more detailed risk assessment, and more intensive control of what is modifiable — not resignation.

Read the full article on CuringHeartDisease.com

- Double-Dosed: Is Your Family Tree a Bio-Hazard? Here's the Life-Saving Information
- **See also:** Children With 60-Year-Old Arteries: The Extreme Reality of HoFH

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THE BIG LIE: Good Genes Make You Bulletproof

THE CLAIM A family full of long-lived relatives who ignored their health means you can do the same.

THE HOOK *Everyone knows an Uncle Joe. Nobody builds a survival estimate from a sample of one.*

What the evidence actually shows

The Uncle Joe argument is survivorship bias with a first name. The relatives who smoked for fifty years and died at ninety are memorable; the ones who died at fifty-eight are, statistically and conversationally, less present in family lore. Reasoning from the survivors systematically overestimates protection.

Protective genotypes do exist and are genuinely powerful. Cohen and colleagues showed that PCSK9 loss-of-function carriers had substantially reduced coronary risk; the Exome Sequencing Project found APOC3 loss-of-function variants associated with lower triglycerides and less coronary disease; and Nioi and colleagues identified an ASGR1 variant linked to reduced risk. They work by different routes — LDL-receptor availability, triglyceride-rich lipoprotein metabolism, and a hepatic pathway still being characterized — rather than through one shared mechanism. PCSK9 and APOC3 variants clearly alter atherogenic lipoprotein metabolism; the ASGR1 pathway is less completely characterized. Some of these have inspired effective drugs — PCSK9 inhibition most obviously — though starting a therapy in midlife is not biologically identical to inheriting a variant that has been acting since birth.

Meanwhile, a favorable polygenic score does not license inaction. Lifetime risk data show that the burden of modifiable risk factors at midlife predicts events powerfully across genetic strata, and smoking, hypertension, and diabetes are not neutralized by good ancestry. Genetic advantage lowers the baseline; it does not remove the slope.

THE GRAIN OF TRUTH Genetics matters enormously, and a genuinely favorable inheritance is a real asset. The error is treating an anecdote about a relative as if it were a personal genotype.

BOTTOM LINE Do not infer personal protection from a long-lived relative. Assess your own lipids, Lp(a), blood pressure, glycemia, smoking exposure and overall clinical risk instead.

Read the full article on CuringHeartDisease.com

- The Genetic Lottery: Why You Can't Count on Being "Bulletproof"
- **See also:** Can Good Genes Beat Bad Lipids?

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39 RISK, GENETICS AND SPECIAL POPULATIONS

THE BIG LIE: Heart Disease Is a Man's Problem Until Well After Menopause

THE CLAIM Women are protected by estrogen and do not need to think about cardiovascular risk until old age.

THE HOOK *Cardiovascular disease is the leading cause of death in women — and the risk trajectory changes years before the last period.*

What the evidence actually shows

Cardiovascular disease kills more women than any other cause worldwide, and the Lancet Women and Cardiovascular Disease Commission documented that it remains under-recognized, under-studied, under-diagnosed, and under-treated in women across health systems. The perception of it as a male disease is itself a contributor to worse outcomes.

The menopause transition is a distinct period of cardiometabolic change rather than a threshold that is crossed at the final menstrual period. The AHA scientific statement on the topic describes adverse shifts in lipids — including rises in LDL-C and ApoB — along with changes in body composition, visceral fat distribution and vascular function that begin during perimenopause. Separating what menopause itself drives from what chronological aging drives is genuinely difficult, and both are at work. Prevention timed to 'after menopause' arrives after the change it was meant to pre-empt.

Women also carry sex-specific risk enhancers that standard calculators frequently omit: pre-eclampsia and gestational hypertension, gestational diabetes, preterm delivery, polycystic ovary syndrome, premature menopause before 40, and autoimmune conditions. One point deserves stating directly, because the question is asked constantly: menopausal hormone therapy should not be started for the purpose of preventing cardiovascular disease. The WHI analyzes showed that age and time since menopause substantially change the risk-benefit picture, and formulation and route matter too — it is a decision about menopausal symptoms, made individually with a clinician, not a cardiovascular prevention strategy. Certain conditions are also disproportionately female — MINOCA, spontaneous coronary artery dissection, and coronary microvascular disease among them. On symptoms, the older teaching that women typically present 'atypically' has been overstated: chest pain remains the most common presenting symptom in both sexes, with substantial overlap, and treating atypical presentation as the female norm risks the very delays it was meant to prevent.

THE GRAIN OF TRUTH Event rates genuinely are lower in premenopausal women than in age-matched men, and that difference is real. It is a delay in the curve, not exemption from it.

BOTTOM LINE Prevention should start well before menopause, and it should include reproductive history: hypertensive disorders of pregnancy, gestational diabetes and preterm delivery all carry later cardiovascular meaning. An obstetric history is a cardiovascular history. Perimenopause is an important reassessment point, not the starting line.

Read the full article on CuringHeartDisease.com

- Does estrogen protect women's arteries from plaque?
- **See also:** Menopause Endocrine Cardiovascular Risks

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THE BIG LIE: Once You Start Medication, Lifestyle No Longer Matters

THE CLAIM Drugs and lifestyle are competing philosophies; choosing one makes the other unnecessary.

THE HOOK *They are teammates, not rival religions — and they cover different ground.*

What the evidence actually shows

The two approaches do overlapping but non-identical work. Lipid-lowering drugs achieve reductions that are difficult for most people to reach through lifestyle alone: a high-intensity statin commonly lowers LDL-C by at least 50%, a PCSK9 inhibitor by roughly 60%, and more in combination. ApoB falls substantially too, though not always by an identical percentage. Diet and exercise reach less far on that single axis. But they act on blood pressure, insulin sensitivity, body composition, inflammatory tone, thrombotic risk, cardiorespiratory fitness, sleep, and mood — the majority of which no lipid-lowering drug addresses at all.

The trial evidence assumes both. The major lipid-lowering trials generally included background preventive care or lifestyle advice, though adherence and intensity varied. Their results therefore support adding medication to usual preventive management rather than substituting for it. And the residual risk that remains after excellent lipid control is substantial — that is exactly the space that blood pressure, glycemia, fitness, weight, and tobacco occupy.

There is also a practical interaction that gets overlooked. Sustained weight loss and improved insulin sensitivity improve triglycerides, blood pressure, glycemia and sometimes ApoB. Whether that should change a medication dose is a clinical judgement about achieved targets, not an automatic consequence. The two strategies make each other more effective.

THE GRAIN OF TRUTH There is a real and reasonable preference question about how much medication someone wants to take, and patients are entitled to weight it differently. The false part is the premise that starting one means abandoning the other.

BOTTOM LINE Use both where indicated. Medication produces large, reliable reductions in atherogenic lipoproteins; lifestyle improves several additional risk pathways — though neither covers everything, and Lp(a), established plaque and inherited risk persist regardless.

Read the full article on CuringHeartDisease.com

- How I Reversed My Heart Disease
- **See also:** Can Modern Medicine Save a Couch Potato? The 40-Year Triplet Experiment

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exercise program — those decisions belong with a qualified clinician who knows your history. If you are having symptoms, seek medical care rather than reading further.