



THE PREVELITY LIBRARY

VOL. 02 · LIPIDS & YOUR ARTERIES

Lipids for Longevity.

Heart disease is largely preventable — and most of the prevention is invisible. What your cholesterol numbers mean, what they miss, and what actually lowers your risk.

FREE PREVIEW EDITION

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PRE-VELITY

INNOVATIVE PREVENTION, PERSONALIZED FOR LONGEVITY.

FOREWORD

Heart disease is preventable. Most of the prevention is invisible.

Your feed is full of stories about good fats and bad fats, heart-healthy recipes and heart-attack survivors. Many of the people telling them mean well. What we've ended up with is a loop — the same half-right stories, shared again and again.

Meanwhile, heart and blood-vessel disease remains the leading cause of death, and much of it is preventable. The problem isn't a lack of tools. It's that the most important parts of the story — how many plaque-forming particles you carry, whether you've inherited a risk factor no diet can change, whether plaque is already building in your arteries — don't show up on the basic panel most people get once a year.

This guide explains what's actually going on: what lipids do, why they matter, how to measure them properly, and the full range of options — lifestyle, supplements, and medications — that your own physician may discuss with you. We've tried to be as clear about uncertainty as about evidence.

WHAT THIS IS — AND ISN'T**Education, so you can decide well with your doctor.**

This guide explains how physicians think about cholesterol and arterial health. It can't account for your history, and it isn't a treatment plan. PreVevity is strictly preventive: we screen, monitor, and refer. Any treatment is decided with your own physician.

DR. PATRICK MULLEN, MD
FOUNDING PHYSICIAN • PREVELITY

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SECTION 01

01.

The Silent Timeline.

Cholesterol isn't a villain. It's how it's carried, how it meets your blood vessels, and how long that goes on — that's where prevention lives.

INSIDE THIS SECTION

Cholesterol isn't the villain · A 40–50 year head start · You can't feel it

BACKGROUND

Essential building blocks — carried the wrong way, for too long.

When most people hear "cholesterol," they think of a bad lab number or a food they're not supposed to eat. The truth is more interesting. Lipids — cholesterol, triglycerides, and the particles that carry them — are essential. Without cholesterol you couldn't make hormones, cell membranes, or vitamin D. Without triglycerides you'd have no reliable energy reserve.

The problem isn't that you have cholesterol. It's how many particles carry it through your blood, how those particles interact with your artery walls, and how long that exposure lasts. High-risk lipids don't cause chest pain or breathlessness. They work silently, for decades, until a plaque ruptures and a heart attack or stroke arrives seemingly "out of nowhere."

A COMPOSITE CASE • DETAILS CHANGED

John, 52. "A little high, nothing to worry about."

He eats reasonably well, exercises a few times a week, and doesn't smoke. His LDL is 115, and he's told it's a little high but fine. Two years later, a routine stress test is normal and he feels reassured. A year after that, he has a heart attack on a morning run. Imaging afterward shows extensive plaque that had been building for years.

The lesson: the standard panel and the stress test weren't wrong. They just weren't built to see early disease.

THE TIMELINE

A 40–50 year head start.

Arteries feed every organ — heart, brain, kidneys, muscles, skin. That's why the same process behind heart attacks and strokes also contributes to vascular dementia, kidney disease, erectile dysfunction, and poor circulation in the legs.

And it starts early. Autopsy studies of young adults in their late teens and twenties have found fatty streaks inside the arteries — not dangerous yet, but the seed of what can grow into plaque over the following decades.

40–50

YEAR WINDOW

By your 30s and 40s, fatty streaks may have become small plaques. By your 50s and 60s, those plaques can narrow arteries or rupture. The window to change the course is long — if you use it.

The CARDIA study, following adults from ages 18–30, found that cumulative exposure to elevated LDL and related particles in early adulthood predicted coronary plaque and events decades later.

SYMPTOMS — OR THE LACK OF THEM

You can't feel cholesterol.

Most people with dangerous lipid profiles feel perfectly well until disease is advanced. When early signs do appear, they're easy to miss.

MEN**Erectile dysfunction**

The arteries involved are smaller than the coronaries, so narrowing can show up there first. It deserves a vascular conversation, not just a prescription.

WOMEN**Quieter symptoms**

Fatigue, breathlessness, indigestion, or shoulder discomfort instead of classic chest pain — and more often dismissed.

BRAIN**Vascular roots of dementia**

Poor vascular health raises the risk of both vascular dementia and Alzheimer's disease.

ELSEWHERE**Silent clues**

Leg cramps when walking, slow wound healing — circulation red flags worth mentioning to your doctor.

Risk isn't a switch. A "borderline" number over decades can do as much harm as a high number over a few years.

CUMULATIVE EXPOSURE

The flip side is encouraging: prevention works at any stage. Lowering exposure and inflammation can slow plaque growth and help stabilize it — but only if we look before the body forces us to.

SECTION 02

02.

Lipids 101.

Five numbers, two processes, one outcome. Once you can name the players, most of the confusion about cholesterol falls away.

INSIDE THIS SECTION

The five numbers · Inflammation's double hit · How a plaque forms · Inflammatory conditions and the heart

THE CARRIERS

Fats don't dissolve in blood. They ride in particles.

Lipids aren't water-soluble, so your body packages them in lipoproteins — tiny particles of fat and protein that ferry cholesterol and triglycerides through the bloodstream. Which particles you carry, and how many, matters far more than the "total cholesterol" line on a basic panel.

FIGURE 01 • THE FIVE NUMBERS

A DELIVERY SYSTEM, A RECYCLING CREW, AND A WILD CARD.

NUMBER	ROLE	WHAT TO KNOW
LDL	Delivery truck	Delivers cholesterol to cells. When particles are plentiful, they slip into the artery wall, where they can be oxidized and trigger inflammation.
HDL	Recycling crew	Carries excess cholesterol back to the liver. More is generally better — but very high HDL (roughly above 100 in men, 120 in women) hasn't proven protective and may reflect dysfunctional particles.
Triglycerides • VLDL	Energy carrier	High fasting triglycerides often travel with insulin resistance and more small, dense LDL particles — a flag to look at metabolic health (see Vol. 03).
ApoB	Particle count	Every plaque-forming particle carries exactly one ApoB, so it counts them directly. More particles, more chances for plaque.
Lp(a)	Wild card	Inherited, elevated in roughly one in five people, sticky and clot-promoting, and barely moved by lifestyle. Often the culprit when fit, healthy people have early heart disease.

ApoB and Lp(a) belong on every modern lipid panel. Lp(a) is largely fixed by your genes, so it usually needs measuring only once.

THE DOUBLE HIT

Cholesterol seeds the problem. Inflammation fans the flames.

Lipid particles start the process by entering the artery wall. What turns a fatty streak into dangerous plaque is the body's inflammatory response. The worst outcomes happen when both are present — which is why lowering LDL without addressing inflammation, or vice versa, leaves risk on the table.

THE SMOKE DETECTOR

hs-CRP

High-sensitivity CRP picks up low-grade inflammation. Even with normal LDL, a raised hs-CRP is associated with more heart attacks and strokes.

DIRECT INJURY

Smoking

Damages the artery lining, makes arteries stiffer and platelets stickier, and accelerates plaque. There's no safe level, including secondhand smoke.

FIGURE 02 • HOW A PLAQUE FORMS

FROM SMOOTH LINING TO RUPTURE — A DECADES-LONG PROCESS.

01

Dysfunction

Blood pressure, glucose, smoking, or inflammation damage the lining.



02

Entry

ApoB particles slip in, oxidize, and draw immune cells.



03

Buildup

A fibrous cap forms; the plaque grows, often silently.



04

Event

The cap ruptures, a clot forms: heart attack or stroke.

The heart attack that came "out of nowhere" was often decades in the making. The same process plays out in the kidneys, legs, and brain under different names.

INFLAMMATORY CONDITIONS

When the immune system raises the stakes.

In many chronic inflammatory diseases, the leading cause of death isn't the condition itself — it's cardiovascular disease. Persistent immune activation keeps the artery lining inflamed, so the same cholesterol exposure does more damage. Standard risk calculators weren't built for this, and they tend to underestimate it.

RHEUMATOID ARTHRITIS

Higher cardiovascular mortality

Substantially higher than the general population, driven by chronic joint and vascular inflammation.

LUPUS

Early heart attacks

Young women with lupus face markedly higher heart attack rates than their peers.

HIV

Even when controlled

Higher vascular inflammation and earlier atherosclerosis, even with the virus well suppressed.

Psoriasis, inflammatory bowel disease, and other autoimmune conditions follow the same pattern to varying degrees. If you live with any of these, your lipid numbers deserve closer attention than a calculator alone suggests — and it's worth making sure your specialist and your primary care physician are talking to each other about your heart.

When inflammation is chronic, the same cholesterol does more damage. Measure accordingly.

THE DOUBLE-HIT PRINCIPLE

END OF THE FREE PREVIEW

The rest of this guide.

You know the players now. The complete edition covers how to measure them properly — ApoB, Lp(a), and when imaging your arteries changes the picture — then everything that lowers risk: food, training, sleep, stress, supplements with real evidence, and the medications your doctor may discuss.

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