A HEAVENLY HEALTH HACKS COMPANION

Chapter 9 · "My Body, My Lab"

M46 MINISTRIES

CHOLESTEROL, *Plainly*

What it does, the myth of good and bad, what really causes heart disease, what makes a doctor reach for a statin — and what to ask before you say yes.

GUIDE 02

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M46 MINISTRIES · NOVEMBER 2026 · M46MINISTRIES.COM

GUIDE 02

CHOLESTEROL, *Plainly*

A plain-language guide. Short sentences, the numbers that matter, both sides named where doctors disagree. Every number is given in US units first, with the units Canadian labs use in [brackets] — so you can read your own report wherever it came from. Part 1 is the plain version; Part 2 goes deeper. Two pages are made to print: the questions (Section 9) and the tracker (Section 10).

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⚠️ Important — Read This First

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My Story — Why This Booklet Exists 📍

Teresa and I have tested for years. In 2026 we did extensive testing — blood, DNA and heart scans — partly for the writing of *Heavenly Health Hacks*, using ourselves as human laboratories. In February my total cholesterol came back at 303 mg/dL [7.8 mmol/L]. A doctor sees that number and reaches for the prescription pad. But a third of it was HDL, the return fleet, and the rest of my panel told a very different story.

What	Mine (February–April 2026)	What it means
Total cholesterol	303 mg/dL [7.8 mmol/L]	Looks alarming — but a third of it was HDL
Triglycerides ÷ HDL	0.46 [0.20] in February	Elite. My metabolism makes big, buoyant particles
Inflammation (hsCRP)	below detection	Very little fire in the artery walls
Lp(a)	under 10 nmol/L	No inherited sticky particle
ApoB — the particle count	**144 mg/dL [1.44 g/L]**	Too many particles, however healthy they were
CT angiogram	54 mm³ of plaque, 87% soft	Early plaque. The count had been high for years

A healthy engine carrying too many trucks, for too long. Low inflammation bought me time. It didn't buy immunity. The ratio alone was my blind spot; the count alone would have missed how healthy the engine was. You need both.

What I did. I changed the food — fewer saturated-fat extremes, more olive oil, more carbohydrate — took berberine and bergamot for four months (since stopped), and kept measuring. By November ApoB was 109 [1.09], a 24% drop in nine months, still above the 80 I'm aiming for. With my physicians, I chose no medication, regular ApoB tests and a repeat scan as the referee. That's my decision, on my data. It isn't a prescription for you.

Teresa's scan shows the other half. Same lifestyle, ApoB 112 [1.12] — and zero plaque. Hers hasn't been high for as long, and before menopause estrogen helps clear the particles. Plaque comes from how high the number is *and* how long it stays there.

Fix the root, not just the reading. Find out *why* your number is high before anyone treats it. Most of the roots — insulin resistance, a fatty liver, low thyroid, too much of one kind of fat — are fixable. Then measure, retest, and use medicine when the numbers and the scan say so. *(If you already take a statin, keep taking it — never stop on your own.)*

Start with the three steps in the box below.

💡 Start here — if you read nothing else

1. Get three numbers your doctor probably hasn't run: **ApoB**, **triglycerides ÷ HDL**, and **fasting insulin**.
2. If ApoB is over 80 mg/dL [0.80 g/L], find the *root* (Section 4) before anyone reaches for a prescription pad.
3. Make the three food moves — cut refined carbs and fructose, swap some saturated fat for olive oil and fish, add soluble fiber — then retest in 8–12 weeks, and let a scan referee if it stays high.

Bottom line: Count the trucks. Read the engine. Then decide — with a date on it.

Part 1 — Plainly

How to Use This Guide 💡

More people are told to start a statin than are told why their cholesterol is high. This guide is the "why." Chapter 9 of *Heavenly Health Hacks* tells my story; this is the manual behind it — what cholesterol does, how to read your own numbers, what makes a doctor reach for a prescription, and what to ask before you say yes.

Part 1 — Plainly is the short version: what cholesterol is, what really causes heart disease, how to read your numbers, and what moves them. **Part 2 — Going Deeper** covers how the liver sets the number, how the "normal" line moved, how doctors decide on a statin, and what statins buy and cost.

Every section opens with one line in bold — **Why it matters**. Read those lines alone and you have the booklet in a page.

How to read the evidence. "Solid" means large trials or consistent studies. "Moderate" means smaller or older studies pointing the same way. "Thin" means early, mixed or mostly marketing. Where the mainstream view and this book differ, I say so.

My numbers. My own results from 2026 appear through the booklet, marked 📍 where they stand alone. They are examples, not targets.

Units. US units first, Canadian [SI] units in brackets. To convert: cholesterol mg/dL ÷ 38.67 = mmol/L; triglycerides ÷ 88.57; ApoB mg/dL ÷ 100 = g/L.

📍 Cross-reference: *Heavenly Health Hacks* — Chapter 9 (My Body, My Lab) · Chapter 5 (Cellular Health — The Foundation of Life) · Chapter 22 (Supplementation) · The Essential 16, Biomarkers & Testing · *Bloodwork, Plainly* · *Men's Health, Plainly* · *Women's Health, Plainly*

1 What Cholesterol Is — and Why "Good" and "Bad" Are the Wrong Labels 🧱

Why it matters: Cholesterol is a building material your body makes on purpose. The number that matters is how many particles carry it — not "good" or "bad."

Cholesterol is a building material. Your body cannot live without it.

- Every cell wall in your body is made with it.
- Your hormones — testosterone, estrogen, cortisol — are built from it.
- Bile, which lets you digest fat and absorb vitamins A, D, E and K, is made from it.
- Vitamin D starts as cholesterol in your skin.
- Your brain holds a quarter of all the cholesterol in your body — most of it in the insulation on your nerve wiring — and makes every bit of it itself.
- The particles that carry it in your blood also soak up the toxins shed by bacteria; in animals, more LDL means better survival of a bad infection.

Three things show how much your body wants it. Building one molecule takes more than 30 chemical steps and real energy — and nearly every cell in your body can do it. The first food a human ever gets, breast milk, is rich in cholesterol; most formula has none. And your body makes about three-quarters of its own supply — not mainly in the liver, as people say, but in the cells that use it. What the liver controls is how much ends up in your

blood. Food adds the rest, and for about three people in four the cholesterol in food barely moves the number. In the fourth, LDL and HDL rise together and the particles get bigger, not more numerous.

Trucks, not cargo. Cholesterol is waxy — a sterol, not a fat you can burn, with no calories in it — and blood is water, so it can't travel on its own. It rides inside tiny particles. **LDL** and **HDL** are those particles — the trucks — not the cholesterol itself. Here is the one idea that makes everything else make sense:

- **LDL-C** is how much cholesterol the LDL trucks are carrying. A cargo number.
- **ApoB** is how many trucks there are. Every LDL truck carries exactly one ApoB tag, so ApoB counts trucks.
- **HDL** is the return fleet, collecting cholesterol from tissues and carrying it back to the liver.

Trucks, not cargo, are what get stuck in artery walls. So the truck count — ApoB — is the number to know. And **total cholesterol is not LDL**: it's all the cholesterol in all the trucks added together, the return fleet included. A high total can be high for a good reason — a third of mine was HDL. When a doctor says "your cholesterol is 300," the first question is: *which trucks?*

The myth. There is no good cholesterol and no bad cholesterol. There is one molecule, identical whether it's sitting in your brain, your adrenal gland, or an artery wall. The labels came from population studies in the 1970s and 80s that found heart disease tracked with LDL and ran opposite to HDL. Useful at the time. Wrong as biology — and the shorthand hides more than it shows, because LDL isn't one thing:

Kind of truck	What it is	Why it matters
Large, buoyant LDL (Pattern A)	Big, fluffy, cholesterol-rich particles	Less likely to squeeze into the wall and less likely to oxidize once there. The pattern of a healthy, insulin-sensitive metabolism. *Far less harmful — not harmless.* Enough of them for long enough still builds plaque; my own scan is the proof.
Small, dense LDL (Pattern B)	Small, compact particles, low in cholesterol, often carrying leftover triglyceride	Slip through more easily, stick to the wall more readily, oxidize faster, and stay in the blood longer because the liver's docks don't recognize them well. The pattern of insulin resistance. The dangerous one.
VLDL and remnants	The big fat-delivery trucks the liver sends out, and what's left after they unload	Rise with sugar, refined carbs, fructose and alcohol. Remnants are as plaque-forming as LDL, and they're what high triglycerides are telling you about.
Lp(a)	An LDL truck with an extra sticky protein bolted on	Inherited, about 90% genetic. Clot-prone and plaque-prone. Immune to diet. Test once in your life; if it's high, be stricter on everything else.

Two people can have the same LDL-C and completely different risk, because one carries it in a few big trucks and the other in a swarm of small ones. That is why a "normal" cholesterol number can hide a high truck count — Section 3 shows how to catch it.

Why HDL is called "good." People with high HDL have less heart disease, and that's real. But HDL isn't good because of the number; it's good because of the *work*. Three things the label hides:

- **You can't buy it.** Drugs that raised HDL — niacin, the CETP inhibitors — raised the number beautifully and prevented little or nothing. The one CETP drug that helped a little did it by lowering ApoB, not by raising HDL.

- **It can go bad.** In chronic inflammation and diabetes, HDL particles become oxidized and dysfunctional — they stop collecting and can even start depositing. Same number on the lab sheet, opposite job.
- **More isn't always better.** In the largest study — 116,000 Danes — men's death rate was lowest around 73 mg/dL [1.9 mmol/L] and began to climb above about 97 [2.5]; women's was lowest around 93 [2.4]. That's why the book's range has a top as well as a bottom (50–90 mg/dL [1.29–2.33 mmol/L]). My 91 [2.35] — 90 [2.33] in November — sits in the flat zone. My triglycerides were 42 mg/dL [0.47 mmol/L], and 30 [0.34] by November. Together they read as a healthy metabolism, which is what the book's "excellent" means. Pushing it higher would buy nothing.

HDL is a *marker*, not a lever. A high HDL with low triglycerides says the metabolism is sound and the trucks are big. You get it by fixing insulin resistance, not by chasing the number.

Where the two camps stand. Mainstream lipidology says the count beats the size: once you know how many trucks there are, big or small adds little. The functional camp (Krauss, Hyman, Attia on this point) says the pattern is the earliest, cheapest window into insulin resistance. Both are right about what they measure — count the trucks, and read the pattern, never one without the other.

What drives Pattern B, and what reverses it. Small dense LDL is made from triglyceride-rich VLDL, so it rises with whatever fills the liver with fat — sugar, refined starch, fructose, alcohol, insulin resistance, belly fat, inactivity — and falls when they fall. Cutting refined carbohydrate shifts particles from small to large within weeks. Saturated fat, by contrast, tends to make particles *larger* while raising their number — my profile exactly.

How to know yours. The two-number shortcut: high LDL with *low* triglycerides (ratio under 1 [0.44]) is almost always Pattern A; high LDL with *high* triglycerides (ratio over 2 [0.87]) is almost always Pattern B. If you want it measured, an NMR LipoProfile reports particle number, small dense count, and average size (Pattern A above 20.5 nm). Then count the trucks with ApoB either way.

💡 **Carry two numbers in your head.** *Triglycerides ÷ HDL* says whether your body is making dangerous trucks. *ApoB* says how many there are. I had an elite ratio and a high count in the same body — which is why the ratio alone was my blind spot, and why the count alone would have missed how healthy the engine was. Never one without the other.

🔑 **Remember:** Count the trucks with ApoB. Read the engine with triglycerides ÷ HDL. Never one without the other.

2 What Actually Causes Heart Disease 🔥

Why it matters: The particles are the fuel and your metabolism is the fire. You need to measure both.

People say "it's not LDL." Half of that is right. The other half can hurt you.

The half that's right. LDL-C — the cargo number on a normal blood test — is a weak predictor by itself. In a study of 136,905 Americans admitted with a heart attack, three-quarters had "normal" LDL-C. Most people who have heart attacks have normal cholesterol, and most people with high cholesterol never have one. Treating that one number with a pill while ignoring blood sugar, blood pressure, smoking, sleep and inflammation is the mistake.

The half that's wrong. The trucks are not innocent. ApoB particles are the *fuel*. No fuel, no fire. This is as settled as anything in medicine. People born with genes that keep ApoB low all their lives almost never get coronary disease. People born with very high ApoB get heart attacks in their thirties, with no other risk factor. And every drug that lowers ApoB, by whatever route, lowers heart attacks in proportion. In the biggest study of heart attacks ever done — 52 countries, 30,000 people — the balance of ApoB trucks against the return fleet (ApoB ÷ ApoA-1) was the single largest cause.

So the honest sentence is: **LDL-C alone is a poor guide. ApoB particles are the fuel. Your metabolism decides how fast and how dangerously the fuel burns.**

🔍 **How it happens.** A truck slips into the artery wall and gets stuck. It oxidizes — goes rancid. Immune cells come to clean it up, eat it, and die there. Over years that becomes plaque. A heart attack is different and fast: the cap on a soft plaque tears, a clot forms on it, and the artery closes. So two things decide your future — how much plaque you build, and whether it's the soft, inflamed kind that tears or the hard, scarred kind that mostly doesn't.

The causes, roughly in order. Each one adds fuel, lights the fire, or makes clots more likely:

1. **Insulin resistance and high blood sugar** — damages the wall, makes small dense trucks, raises blood pressure. The root under most of the others.
2. **Too many ApoB trucks, for too many years** — the fuel. Time counts; "my LDL has been high for years but my inflammation is low" is reassurance with an expiry date.
3. **Smoking and dirty air** — the strongest fire-starter there is.
4. **High blood pressure** — pounding on the wall every heartbeat.
5. **Chronic inflammation** — belly fat, gum disease, poor sleep, infections, oxidized seed-oil fats. An hsCRP of 2 mg/L or more is a risk-enhancer in both the US and Canadian guidelines — the fire raises risk on its own, before any cholesterol number.
6. **Lp(a)** — the sticky truck. One in five people has a lot of it.
7. **Damaged fats** — oxidized LDL is what the immune cells eat; seed-oil fat is the easiest to oxidize. Mainstream medicine ranks this lower than this book does.
8. **Sleep apnea and short sleep; chronic stress, depression and loneliness** — psychosocial stress was the third-biggest factor in that 52-country study.
9. **Sitting still** — low fitness predicts early death better than almost anything.
10. **The clot itself** — sticky platelets, dehydration, the 6 a.m. stress surge.

Mainstream medicine treats number 2 with a pill and the rest with advice. This book treats the rest as the disease and expects number 2 to improve as they do. Often it does. Not always, and not always enough. The honest path does both: put the fire out, measure the fuel, and let a scan referee.

🔍 **An early warning most men are never told.** The arteries that supply an erection are smaller than the ones that feed the heart, and they often show damage first. In 9,457 men followed for up to seven years, new erectile dysfunction raised the risk of heart attack, stroke and other heart events by 25% — in the same range as smoking or a family history of heart attack. If it shows up, don't start with a pill. Ask for ApoB, fasting insulin, HbA1c and a blood-pressure check, and read them with this section in mind.

The people with the cleanest arteries. The **Tsimane**, a forager people in the Bolivian Amazon: LDL around 90 mg/dL [2.3 mmol/L], active all day, no smoking, no insulin resistance — and 85% of them had a calcium score of zero from age 40 up. Their inflammation is actually *high*, from constant infection, and their arteries are still clean. Hunter-gatherer groups generally run LDL of 50–75 mg/dL [1.3–1.9 mmol/L], roughly where a human liver settles without a modern diet on top of it. Mainstream reads that as: the common thread is low ApoB for life. This book reads it as: the common thread is also no insulin resistance, no seed oils, constant movement, no smoking — you can't pull one strand out and call it the cause. Either way, nobody in those villages lets ApoB climb to 144 mg/dL [1.44 g/L] over fifteen years.

🔑 **Remember:** No fuel, no fire — but fuel without fire still adds up over time.

3 Reading Your Numbers

Why it matters: Five tests read the particles, the engine and the fire together. A standard panel reads none of them well.

The five tests. If you run nothing else, run these. Four come from one tube of blood.

#	Test	What it tells you	"Normal" on the lab sheet	Optimal
1	**ApoB**	How many trucks	under 105 mg/dL [1.05 g/L]	**under 80 mg/dL [0.80 g/L]**
2	**Triglycerides ÷ HDL**	What kind of trucks; how your metabolism is running	not reported	**under 1.0** (mg/dL) or **under 0.44** (mmol/L); under 0.5 [0.22] is elite
3	**Fasting insulin**	Whether your engine is straining — years before sugar rises	up to 25 µIU/mL [174 pmol/L]	**2–5 µIU/mL [14–35 pmol/L]**
4	**hsCRP**	How much fire	under 3 mg/L	**under 0.36 mg/L** — "below detection" is the goal
5	**HbA1c**	Your three-month average blood sugar	under 5.7%	**under 5.4%**
★	**Lp(a)** — once in your life	The sticky truck you inherited	under 100 nmol/L	under 75 nmol/L; if high, be stricter on everything else

Read them together: ApoB says how many trucks. The ratio says what kind. Insulin and HbA1c say whether the engine that makes the bad kind is running. hsCRP says whether the wall is inflamed enough to trap them. The dangerous profile is the common one: a so-so ApoB with a ratio over 2 [0.87], insulin over 10 µIU/mL [69 pmol/L] and CRP over 2 mg/L — which a standard blood test calls "normal."

⏱️ **Practical.** Fast twelve hours — water only; black coffee can nudge glucose and insulin, so skip it on test days. Don't test hsCRP within two weeks of a cold or an injury. Insulin is the one most doctors won't order unless you ask — ask. Retest every two to three months while you're changing something, then once or twice a year — and compare readings from the same season. Cholesterol runs higher in winter: about 4 mg/dL [0.1 mmol/L] in men and 5 [0.13] in women, and 11–18 [0.3–0.5] in people already over 240 [6.2], mostly because blood is more concentrated in the cold. In one year-long study, 22% more people were flagged "high" in winter than in summer. One winter reading should never decide a prescription. **The next five:** vitamin D (40–80 ng/mL [100–200 nmol/L]), ferritin (men 75–150, women 50–150 ng/mL [µg/L]), GGT (under 20 — early fatty liver), a full thyroid set (TSH, free T4, free T3, reverse T3, TPO antibodies), and the omega-3 index (over 8%).

The standard lipid panel, line by line. A standard panel measures three things — total cholesterol, HDL and triglycerides — and *calculates* LDL-C from them: **Total = LDL-C + HDL-C + VLDL cholesterol**, where VLDL is estimated as triglycerides ÷ 5 (÷ 2.2 in mmol/L). The lab's "normal" is where most of the people it tests land — and the lab tests a lot of sick people. The optimal column is where the healthy sit.

Line on the sheet	Lab "normal"	Optimal	What to look for
Total cholesterol	under 200 mg/dL [5.2 mmol/L]	no target on its own	*Which trucks made it high?* High total with high HDL and low triglycerides is a different animal from high total with low HDL and high triglycerides.
LDL-C	under 135 mg/dL [3.5 mmol/L]	context-dependent — read it with triglycerides, then confirm with ApoB	A calculated estimate of cargo; unreliable when triglycerides are high. High LDL-C with *low* triglycerides: big trucks — ask for ApoB before anyone reaches for a pad. High LDL-C with *high* triglycerides: small trucks — fix the metabolism first.
HDL-C	over 40 mg/dL [1.0 mmol/L] (men), 50 [1.3] (women)	50–90 mg/dL [1.29–2.33 mmol/L]	A marker, not a lever. Low HDL beside high triglycerides is the insulin-resistance signature.
Triglycerides	under 150 mg/dL [1.7 mmol/L]	under 80 mg/dL [0.90 mmol/L]	The most diet-sensitive line on the sheet — sugar, refined starch, fructose, alcohol. The only line that needs fasting.
Non-HDL (total − HDL)	Canada's treatment line is 162 mg/dL [4.2 mmol/L]	under 130 mg/dL [3.4 mmol/L]	All the truck cargo in one number — the best stand-in when ApoB isn't on the sheet.
Triglycerides ÷ HDL	not reported — do the division yourself	under 1.0 (mg/dL) / 0.44 (mmol/L)	Insulin sensitivity and truck size in one number. See below.
Total ÷ HDL	over 6 is flagged	under 3.5 (same number in mg/dL or mmol/L)	Overall balance.
ApoB (ask for it)	under 105 mg/dL [1.05 g/L]	under 80 mg/dL [0.80 g/L]	The truck count. The line the statin conversation should be about.
ApoB ÷ ApoA-1 (if both are run)	—	under 0.6 (same number in either unit)	Bad trucks against good — the strongest single predictor in the 52-country study.

The ratio — the one number your doctor won't calculate

Divide your triglycerides by your HDL. That's it. Both are on every standard panel, and the lab never does the division for you, because mainstream risk tools don't use it. This book treats it as the first number to look at, for three reasons:

- **It reads insulin resistance** before glucose moves. Triglycerides rise and HDL falls as insulin climbs; the ratio catches it a decade before HbA1c does.
- **It reads truck size.** A high ratio means the liver is pumping out triglyceride-rich VLDL, which is where small, dense LDL comes from. In a Harvard case-control study of 680 people, those in the top quarter of the ratio

had sixteen times the heart-attack risk of the bottom quarter — a small study, and larger cohorts since have found a smaller effect, but the direction has held.

- **It moves fast.** Cut refined carbohydrate and fructose and the ratio falls within weeks — the quickest feedback you'll get from a blood test.

Ratio (mg/dL)	Ratio (mmol/L)	What it says
under 0.5	under 0.22	Elite — insulin-sensitive, big trucks
under 1.0	under 0.44	Optimal
1.0–2.0	0.44–0.87	Watch — insulin is starting to run high
over 2.0	over 0.87	Act — this is a metabolic signal, not a number to watch and wait

Two cautions. The target changes with the unit — under 1.0 in mg/dL is under 0.44 in mmol/L, and a Canadian reader who uses the US target will think they're in trouble when they're not. And the ratio cannot see the count: a perfect ratio with a high ApoB is exactly my case. Mainstream medicine's reason for not using it is fair — ApoB and non-HDL predict events more directly — but nothing on the standard sheet reads your metabolism as cheaply or as early.

Two more things to know. Labs wobble: HDL moves 10–15% between draws, LDL about 10% — one surprising number means retest in a month, not panic. Units: cholesterol mg/dL ÷ 38.67 = mmol/L; triglycerides ÷ 88.57; ApoB mg/dL ÷ 100 = g/L.

The advanced tests add what the standard panel can't see: ApoB (truck count), Lp(a) (once), an NMR LipoProfile (truck size and number), and imaging — a **calcium score** (CAC), which shows hardened plaque only, or a **CT angiogram with plaque analysis** (CCTA), which shows the soft plaque too and is the fuller picture.

Where ApoB fits — reading it against LDL-C and total cholesterol

Total cholesterol can't see trucks at all. LDL-C sees the cargo in one kind of truck, by estimate. ApoB counts every truck that can get stuck — LDL, VLDL, remnants and Lp(a) — because each carries exactly one ApoB tag, and it's measured directly. That's why it beats both.

What to expect. In a typical person, ApoB runs about three-quarters to four-fifths of the LDL-C number (both in mg/dL). The medians from 12,688 American adults not on a statin:

If LDL-C is	ApoB is usually about
70 mg/dL [1.8 mmol/L]	60 mg/dL [0.60 g/L]
100 mg/dL [2.6 mmol/L]	80 mg/dL [0.80 g/L]
190 mg/dL [4.9 mmol/L]	140 mg/dL [1.40 g/L]

There's a real spread around those medians — at an LDL-C of 100 [2.6], half of people had an ApoB between 75 and 86 mg/dL [0.75–0.86 g/L], and 95% fell between 66 and 99 [0.66–0.99]. That spread is the whole reason to measure it.

Three things an ApoB result tells you about your LDL-C:

1. **ApoB about where LDL-C predicts.** The two agree; your LDL-C is telling the truth about the truck count.
2. **ApoB higher than LDL-C predicts.** More trucks than the cargo suggests — small, dense trucks, each carrying little. The insulin-resistance pattern; common with high triglycerides, belly fat and diabetes. Your risk is *higher* than the LDL-C says. This is the case a standard panel misses: a "normal" LDL-C sitting on top of a high ApoB.

3. **ApoB lower than LDL-C predicts.** Fewer, bigger, cholesterol-rich trucks. Your risk is *lower* than the LDL-C implies — but the count still counts.

When the two disagree, follow ApoB. In the CARDIA study, young adults whose ApoB ran higher than their LDL-C suggested had more coronary calcium twenty-five years later. In the UK Biobank — 390,000 people — once ApoB was known, LDL-C and triglycerides added nothing to predicting a heart attack. Mainstream and functional medicine agree on this one.

A size read you can do yourself. Divide LDL-C by ApoB. In US units (both mg/dL), around 1.3–1.4 is typical [from a Canadian report — LDL-C in mmol/L ÷ ApoB in g/L — about 3.4–3.6]. Lower — toward 1.2 [3.1] and under — means the cargo is spread thin across many small trucks (Pattern B). Higher — 1.5 [3.9] and up — means big, cholesterol-rich trucks (Pattern A). A rough guide, not a diagnosis; the NMR measures it.

What it means for the decision. If your doctor is deciding on a statin from LDL-C alone, ask for ApoB first. In Canada it's already on the guideline as an equal trigger and an equal target. The book's target is under 80 mg/dL [0.80 g/L]; Dr. Attia argues for under 60 mg/dL [0.60 g/L] for anyone serious about prevention.

My own panel, read properly

Total 303 mg/dL [7.8 mmol/L], LDL-C 202 [5.22], HDL 91 [2.35], triglycerides 42 [0.47] — so 303 = 202 + 91 + about 10 in VLDL (in Canadian units, 7.8 = 5.2 + 2.35 + 0.2). A third of my "high cholesterol" was the return fleet.

- **First look:** a doctor sees 303 and 202 and reaches for the prescription pad.
- **Second look:** triglycerides 42 [0.47] and HDL 91 [2.35], ratio 0.46 [0.20] — elite insulin sensitivity, big fluffy trucks. The NMR confirmed it: Pattern A at 22.0 nm, small dense essentially zero.
- **Third look:** ApoB 144 mg/dL [1.44 g/L] — right where an LDL-C of 202 [5.22] predicts (near 150 [1.50]), so the LDL-C was telling the truth: too many trucks, however fluffy. The LDL-P on the same NMR, 1,537 nmol/L against a target under 1,000, said the same thing in a different unit. The size read agreed: 202 ÷ 144 = 1.40 [5.22 ÷ 1.44 = 3.63] — mid-range.
- **Fourth look:** hsCRP undetectable, Lp(a) under 10 nmol/L — low fire, no sticky truck.
- **Fifth look:** the CT angiogram — 54 mm^3 of plaque, 87% soft, zero of the highest-risk kind, 1.1% of the artery wall.

All five at once: a healthy engine carrying too many trucks for too long, with low inflammation keeping the damage early. Fuel without fire bought me time; it did not buy immunity. Ten weeks later LDL-C read 213 [5.50] while ApoB fell to 128 [1.28] — the two moved in opposite directions between draws, a reminder that LDL-C is a calculation from a different lab on a different day and ApoB is the measured one. By November 2026, with more carbohydrate on the plate and the saturated fat moderated rather than piled on, LDL-C read 172 [4.45], triglycerides 30 [0.34], HDL 90 [2.33], the ratio 0.33 [0.15] — and ApoB 109 mg/dL [1.09 g/L], down from 144 in February and 128 in the spring. The size read moved the right way too: 172 ÷ 109 = 1.58 [4.45 ÷ 1.09 = 4.08] — bigger trucks, and fewer of them than an LDL-C of 172 would predict. A 24% drop in trucks in nine months, still above the 80 I'm aiming for. I changed several things at once — fewer saturated-fat extremes, more carbohydrate, and, for four of those nine months, berberine and bergamot (since stopped) — so I can't credit any one of them; the direction is what I can report. The plan: fewer trucks, keep the fire out, rescan.

4 Where a High Number Comes From — and What Moves It 🌱

Why it matters: A high number is a symptom. Find the root, and you often fix the number with it.

A high number is a symptom. The question is *why this body is making or keeping more trucks than it clears.* In rough order of how often each is the answer:

1. **Insulin resistance and a fatty liver.** The most common root. Signs: triglycerides over 150 mg/dL [1.7 mmol/L], ratio over 2 [0.87], HbA1c over 5.6%, a waist more than half your height, liver enzymes creeping up. Fix this and the panel often fixes itself.
2. **More saturated fat than your liver handles.** Modest effect in most people, large in some. The extreme case is the lean, fit, low-carb eater whose ApoB climbs on a high-fat diet. That was me.
3. **Genes.** Inherited high cholesterol affects about 1 in 250 — LDL over 190 mg/dL [4.9 mmol/L] from childhood, family history of early heart disease. If that's you — LDL-C over 190 since your twenties, or a parent or sibling with a heart attack before 55 (men) or 65 (women) — ask for a genetic test. This is the one group where early medication is least controversial, and where waiting costs the most: the engine can be perfect and the trucks still too many. Lp(a) is separate.
4. **Low thyroid.** Thyroid hormone opens the docks. Hashimoto's is the usual cause; ask for TPO antibodies, free T3 and reverse T3, not just TSH.
5. **Menopause.** LDL commonly rises 10–20% through it.
6. **Fast weight loss or long fasting.** Temporary. Retest after your weight settles. The same flood of cholesterol into bile is why crash diets and long fasts are a classic cause of gallstones. Three gallstones in four are mostly cholesterol — and they go with high triglycerides, low HDL and a fatty liver, not with a high blood cholesterol. Same root as item 1, different organ.
7. **Kidney or liver disease.**
8. **Medications** — some blood-pressure pills, steroids, some antidepressants and antipsychotics, some contraceptives.
9. **Chronic inflammation or infection** — gum disease included.
10. **Iron overload** — ferritin over 300–400 ng/mL [µg/L] oxidizes the trucks.
11. **Alcohol** — raises triglycerides, especially beer.
12. **Short sleep and chronic stress.** Not only a slower road to the same place — stress raises the number directly. In 199 healthy adults, the third whose cholesterol jumped most during a stressful task in the lab were three times as likely to have LDL over the treatment line three years later, whatever their starting number. The liver sends out more trucks to move fuel for a fight that never comes.

What moves the number — ranked by effect on ApoB, and by how well each is proven. One rule first: for almost every drug and diet change, ApoB falls about four-fifths as much as LDL-C does in percentage terms, because particles shrink before they disappear. The drugs are included so the food and lifestyle levers can be read against them honestly.

Proven to prevent heart attacks — the drugs

Lever	Effect on ApoB	How well proven
PCSK9 injection	down 45–55%	Outcome trials (FOURIER, ODYSSEY)
High-intensity statin	down 35–45%	Dozens of outcome trials
Moderate statin	down 25–35%	Same
Ezetimibe — alone, or added to a statin	down 11–16%	IMPROVE-IT
Bempedoic acid	down about 12% (LDL-C 18%; 38% with ezetimibe)	CLEAR Outcomes 2023 — 13% fewer events in people who can't take a statin

Lever	Effect on ApoB	How well proven
Red yeast rice	down 15–20% — it *is* a low-dose statin (monacolin K)	One Chinese outcome trial; potency and contamination of products are uncontrolled

Proven on the number — food and lifestyle

Lever	Effect on ApoB	How well proven
Swap some saturated fat for olive oil, avocado, nuts, fish — keep some; it's a needed fat, just not the dominant one	down 5–12% (LDL-C 10–15% for a big swap)	Controlled feeding studies — the strongest diet evidence there is. My 11% in ten weeks, alongside other changes, fits.
Soluble fiber — psyllium 10 g a day, oats, beans	about 5% (LDL-C about 7%)	Solid meta-analysis
Plant sterols, 2 g a day	down 6–8%	Solid on the number; no outcome trials
Lose belly fat	down 5–10% with substantial loss, more if insulin-resistant; triglycerides much more	Consistent
Exercise — high volume (about 20 miles [32 km] a week of jogging, or the equivalent)	LDL-C doesn't move, but *particle number and small dense LDL fall and size rises*; small effect at low volume	One good trial (STRRIDE)
Fix insulin resistance — refined carbs, fructose, sleep, movement	down 5–10%; triglycerides 20–50%; small dense LDL falls	Consistent — but in lean people a very-low-carb, very-high-fat diet can *raise* ApoB
Fix low thyroid	down 10–30% — only if you're hypothyroid	Consistent

Supplements — proven on the number only, with weaker trials

Lever	Effect on ApoB	How well proven
Berberine, 500–1,500 mg a day with meals (the book cycles it 8 weeks on, 2 off as a precaution for the gut; dihydroberberine is easier on the stomach)	LDL-C down about 17 mg/dL [0.45 mmol/L], roughly 12–15%; ApoB measured in only two small trials	Moderate certainty on LDL-C, with high variation between trials and signs of publication bias; no outcome trials; interacts with some drugs
Citrus bergamot	LDL-C down 8–40% across twelve small trials, several unblinded; no ApoB data	Low certainty; no outcome trials
Niacin	down 15–20% alone, and Lp(a) 20–30%	Added to a statin, the extra ApoB drop was small, it prevented nothing and it caused harm (AIM-HIGH, HPS2-THRIVE). A caution, not a lever.

Little or no effect on ApoB

Lever	What it does instead
Omega-3 (EPA/DHA, 2–3 g)	Triglycerides down 20–30%; ApoB about unchanged. Take it for triglycerides and the omega-3 index, not for ApoB.
Cutting eggs and dietary cholesterol	Minimal for most people

Together, the food and lifestyle levers can move ApoB 10–25% in a motivated person — enough for some, not for all. For a lean, insulin-sensitive person with a high number, the ones that actually move it are the saturated-fat swap, fiber, sterols and high-volume exercise.

Part 2 — Going Deeper

The reference: read the section you need. Each one opens with why it matters.

5 Your Liver Sets the Number 🏭

Why it matters: How many "docks" your liver keeps open decides your ApoB more than anything you ate today.

Think of your liver as a port. It loads trucks and it unloads them.

Loading. The liver makes cholesterol and packs it, with fat, into VLDL trucks. As the body unloads the fat, those trucks shrink into LDL. So most LDL in your blood started as a truck leaving your liver. A fatty liver sends out more trucks, carrying extra fat — that's high triglycerides. **High triglycerides are a liver report: a sugar problem, not a fat problem.**

Unloading. The liver pulls LDL trucks back out of your blood through **receptors** — docking stations on the liver's surface. A truck docks, gets pulled in, and the dock goes back out for another. How many docks are open decides your ApoB more than anything you ate today.

What opens and closes the docks. The liver cell checks its own cholesterol level. Running low, it opens more docks; full, it closes some. Every drug and every food rule works through that switch:

Opens more docks (lower ApoB)	Closes docks (higher ApoB)
Healthy thyroid	Low thyroid
Good insulin sensitivity	Insulin resistance, fatty liver
Estrogen	Menopause
Olive oil, avocado, nuts in place of some saturated fat	Lots of saturated fat filling the liver cell
Soluble fiber (oats, psyllium, beans) — bile is the body's only real exit for cholesterol; fiber catches it so the liver must make more, from your blood	—
Statins — they block the factory, so the hungry cell opens more docks	—
Ezetimibe — blocks absorption in the gut, same effect	—
Berberine — protects docks from PCSK9, a protein that destroys them	PCSK9 — some people make a lot of it from birth
—	Fast weight loss or long fasts (temporary — stored fat flooding the liver)

Saturated fat doesn't clog anything. It fills the liver cell so it closes a few docks. That's why swapping some butter and beef fat for olive oil, alongside other changes, helped drop my ApoB 16 mg/dL [0.16 g/L] in ten weeks. One more thing worth knowing: your brain doesn't use blood cholesterol at all. It makes its own, behind the blood-brain barrier.

🔍 **Does a statin make the liver produce less cholesterol?** Yes — but that is not mainly how it lowers your number. A statin concentrates in the liver and blocks the first big enzyme on the cholesterol assembly line (HMG-CoA reductase). The liver makes less — and immediately fights back: it builds several times more of that enzyme, makes more PCSK9, and the gut absorbs more cholesterol from food. Whole-body production falls only about 20–40%, and the total amount of cholesterol in your body hardly changes. What changes is the

blood: the liver cell, sensing a shortage, opens many more docks and pulls LDL trucks out of circulation faster. Blood LDL falls 30–55% — mostly from faster clearance, not less production. A statin doesn't drain the pool; it moves cholesterol out of the pipes and into the liver, where the bile exit can get at it. The side effects come from the part that *is* about production. **Where a statin works:** mostly in the liver, but some of the drug reaches other tissues — more with the fat-soluble statins (atorvastatin, simvastatin, lovastatin) than the water-soluble ones (rosuvastatin, pravastatin). The same assembly line makes CoQ10, so CoQ10 falls too — in the liver and in muscle, which is where the aches can begin. Smaller amounts likely reach the testes as well, which is the probable reason testosterone dips a little (Section 8).

6 The Line Moved 📉

Why it matters: The "normal" line has moved again and again in sixty years. Biology didn't change; the line did.

300 wasn't alarming. In 1963 the *New England Journal of Medicine* printed the normal range for total cholesterol as 150 to 280 mg/dL [3.9–7.2 mmol/L]. A man who walked into his doctor's office in 1970 with a total of 300 [7.8] was told nothing. My own 303 [7.8] would have been unremarkable to my grandfather's doctor and is an emergency to mine. Human biology did not change in sixty years. The line did.

Year	The rule of the day
1963	*NEJM* normal range: total cholesterol 150–280 mg/dL [3.9–7.2 mmol/L]
1984	US consensus panel: a total over 240 [6.2] (ages 40 and up) is flagged for treatment — diet first, drugs if diet fails
1987	Lovastatin — the first statin — approved
1988	First national guideline: total under 200 [5.2] "desirable," LDL under 130 [3.4]
2001	LDL under 100 [2.6] called "optimal"
2004	LDL under 70 [1.8] for the highest-risk patients
2013	US guideline switches to a risk calculator: a statin for most people over a 7.5% ten-year risk
2019	European guideline: LDL under 55 [1.4] for the highest-risk

Each step moved millions of people from "normal" to "treat." Some of that reflects better evidence about particles and risk. Some of it reflects who funds guideline panels. Both things are true. What matters is that you know the line is a line, drawn by people — and that the thing to measure is not where you sit against it but what your particles, your engine and your arteries are actually doing.

What the population's cholesterol did. The average American adult's total cholesterol fell from about 222 mg/dL [5.7 mmol/L] in 1960 to 206 [5.3] by the early 1990s — before statins, from less saturated and trans fat in the food supply and less smoking — and to under 190 [4.9] by 2018, while the share of adults over 45 on a statin went from 2% to roughly a quarter or more. The drop after 1990 happened mostly in people over 45, exactly where statin use climbed; the government's own analysts attribute it mainly to the medication, since diet surveys showed little change in those years.

What heart disease did. Deaths from coronary heart disease, adjusted for age, fell about 70% from their 1968 peak to the 2010s. The careful accounting (the IMPACT model, *New England Journal*, 2007) splits the 1980–2000 drop roughly in half: about 47% from treatments — clot-busters, stents, bypass, blood-pressure drugs, statins — and about 44% from risk factors falling: smoking, blood pressure, and cholesterol. Lower population

cholesterol, from diet and drugs together, got credit for about a quarter of the whole decline. Over the same decades obesity tripled and diabetes quadrupled, and in the IMPACT accounting those two *added back* about 18% of the deaths everything else had prevented. Since about 2011 the fall in heart-disease deaths has stalled, and in adults under 55, deaths in hospital after a first severe heart attack rose between 2011 and 2022.

How to read the three lines together. Population cholesterol fell, partly from food and partly from pills. Heart attacks fell with it — but smoking, blood pressure and emergency care did at least as much, and the fuel-only strategy ran straight into a diabetes epidemic it couldn't touch. That is the whole argument of this book in three lines: lowering the trucks works, and a nation on statins with a fatty liver is still a nation having heart attacks. Fix the engine and the fire, measure the fuel, and use the pill when the numbers and the scan say so — not instead of the rest.

7 What Makes a Doctor Reach for a Statin 🩺

Why it matters: Your doctor is following a flowchart. Knowing it lets you take part in the decision.

Your doctor is following a flowchart. Knowing it lets you be a participant, not a patient. In Canada (the 2021 guideline — the thresholds are printed on your LifeLabs report):

A statin is recommended regardless of calculated risk if you have: - a past heart attack, stent, bypass or other diagnosed artery disease, or an aortic aneurysm (plaque found on a screening scan isn't on Canada's list, but it weighs heavily — see below); - diabetes and you're over 40; - chronic kidney disease, if you're 50 or older; - **LDL-C of about 193 mg/dL [5.0 mmol/L] or higher** — the US line is 190 mg/dL [4.9 mmol/L] — or inherited high cholesterol.

Otherwise, they calculate your 10-year risk (Framingham): - **20% or more:** statin. - **10–19%:** statin if LDL-C is 135 mg/dL [3.5 mmol/L] or higher, or ApoB is 105 mg/dL [1.05 g/L] or higher, or non-HDL is 162 mg/dL [4.2 mmol/L] or higher. - **Under 10%:** statin if LDL-C is about 193 mg/dL [5.0 mmol/L] or higher (the inherited line again), or ApoB is 145 mg/dL [1.45 g/L] or higher. Between 5% and 9%, the 10–19% lines also apply: LDL-C 135 mg/dL [3.5 mmol/L] or ApoB 105 mg/dL [1.05 g/L].

Things that tip a borderline case toward a statin: Lp(a) of 100 nmol/L or higher; hsCRP of 2 mg/L or higher; a parent or sibling with early heart disease (men under 55, women under 65); any calcium on a scan; South Asian or Indigenous ancestry; metabolic syndrome. In the US the shape is the same with slightly different cut-points, and a calcium score of zero lets a middle-risk person hold off. Notice what is *not* on the flowchart: the triglyceride ÷ HDL ratio, fasting insulin, and particle size. The system decides on cargo and a calculator; it never asks how your engine is running.

Where I land. LDL-C 202 [5.22] in February, 213 [5.50] in April, 172 [4.45] in November — the first two above both the US 190 mg/dL [4.9 mmol/L] line and Canada's 193 mg/dL [5.0 mmol/L] line. ApoB 144, then 128, then 109 mg/dL [1.09 g/L]. Plaque on the scan. The LDL-C alone meets the line in both countries, and the plaque made the case stronger, which is why a preventive clinic recommended a statin plus ezetimibe. The standard risk calculator (Framingham) scores me on age, cholesterol and blood pressure. On my February numbers — 55, total 303 [7.8], HDL 91 [2.35], blood pressure 120/80, non-smoker — it gives about a 9% chance of a heart attack or stroke in ten years: low risk. My father had his first bypass at 51, and Canada's version doubles the score for that, to about 18%: middle risk. So the guideline and the clinic agree. Neither one sees my elite insulin sensitivity or my low inflammation. I chose no medication, with a verified omega-3, regular ApoB tests, and a repeat scan as the referee — a decision made with my physicians, on my data, with my history. It is not a prescription for you.

8 Statins — What They Buy, What They Cost ⚠️

Why it matters: Statins help most people who already have disease and least healthy people with a high number. Know your number needed to treat.

What they do is in Section 5: they slow the cholesterol factory, mostly in the liver, and the hungry liver opens more docks. LDL falls 30–55%. The same factory line makes CoQ10, which is where most side effects begin.

What they buy. For every 39 mg/dL [1 mmol/L] drop in LDL-C, heart attacks and strokes fall about 20–25% over five years. What that means for *you* depends entirely on your starting risk — which is what "number needed to treat" (NNT) captures: how many people take the drug, for how long, for one of them to be spared an event. A "25% lower risk" means something completely different at 2% risk and at 20%.

Who you are	Heart attacks prevented per 100 people treated for 5 years	NNT over 5 years
Already had a heart attack or stent	about 2–3, plus about 1 death	about 39 to prevent one heart attack; about 83 to prevent one death
Plaque on a scan, diabetes, or 20% ten-year risk	2–3	about 40–50 — where most cardiologists feel the case is clear
Men with LDL around 190 mg/dL [4.9 mmol/L] and no heart disease (the WOSCOPS trial)	about 2	about 42 — high numbers alone, in middle-aged men, do carry risk
Middle risk, no plaque (the pooled primary-prevention trials)	about 1	about 104 for a heart attack; about 154 for a stroke; death not clearly reduced
Low risk, high number only, no other factors	less than 1	150–400+ — hundreds take the drug for one person's benefit

The drug earns its reputation in the top two rows. A healthy person with a high number sits in the bottom row.

Two more honest numbers. Averaged across everyone who takes a statin for about five years, the *average* postponement of death works out to a few days — about 3 days for people without heart disease and 4 for those who already have it, in the one analysis that did the arithmetic. True, and misleading in both directions: it averages the many who gain nothing with the few who gain years. The other side of the ledger — **number needed to harm** — is roughly 1 in 250 for a new case of diabetes over four years, something like 1 in 10 for muscle complaints as people report them but far fewer in trials where nobody knows who's on the drug, and about 1 in 100,000 for serious muscle breakdown.

Do your own. Take your ten-year risk from the calculator your doctor uses, halve it for five years, and divide by about 0.3 (the share of events a typical statin dose removes). A 5% five-year risk: $1 \div (0.05 \times 0.3) \approx 67$. A 2% five-year risk: $1 \div (0.02 \times 0.3) \approx 167$. That is the number to put beside the side effects before you decide.

What they cost — real rates, not folklore.

- **Muscle aches:** 5–10% of users report them. Across nineteen placebo-controlled trials and 124,000 people, 27.1% on a statin reported muscle pain or weakness against 26.6% on placebo — in the first year the drug caused about one in fifteen of the reports, and after the first year none. And in trials where people took statin and placebo in alternating months without knowing which, 90% of the aches showed up on placebo too. Real for some; much rarer than the reputation. Muscle damage you can measure (myopathy): about 1 in 1,000 to 1 in 10,000; the dangerous form, rhabdomyolysis, about 1 in 100,000.

- **CoQ10 loss:** real and consistent — blood CoQ10 falls 30–50%. Take 100–200 mg of ubiquinol with a statin. It restores the blood level the drug lowers; whether it prevents the aches is unsettled — the trials are small and split, and since most reported aches show up on placebo too, there may be less to fix than the reputation says. Cheap, safe, reasonable.
- **New diabetes:** real and modest — about 1 extra case per 250 people over four years, mostly in people already close to it.
- **Memory complaints:** on the label; not borne out in trials. Switching to a "water-soluble" statin (rosuvastatin, pravastatin) may help — the evidence is case reports, not trials.
- **Testosterone:** falls a little on average — about 4% — with some men falling much more. The full picture, statins and the wider war on cholesterol, is below.
- **Calcium score goes up:** statins harden soft plaque. Mainstream calls that stabilizing; this book also raises the possibility that it runs through the vitamin K2 pathway — a hypothesis, not a demonstrated mechanism. Either way, a calcium score can't be used to track a statin — a CT angiogram can.
- **Too low?** Below an LDL of 70 mg/dL [1.8 mmol/L], two large studies — 96,000 Chinese adults and 28,000 American women — saw bleeding strokes rise, 1.7 to 2.7 times at the lowest levels; at 70 or above there was no extra risk. The numbers are small (0.8% of the women over 19 years, against 0.4%), the drug trials that drove LDL very low haven't shown it clearly, and the clot-strokes prevented outnumber the bleeds. But a target has a floor as well as a ceiling. The book's ApoB target of 80 sits above it.
- **Liver enzymes:** rise in about 1%, usually mild; serious liver injury is rare enough that routine liver tests were dropped in 2012.
- **Lawsuits:** as far as I can find, no lawsuit over a statin now on the market has succeeded on side effects. The one that did — cerivastatin (Baycol) — followed deaths from rhabdomyolysis; Bayer withdrew the drug in 2001 and settled. The U.S. Lipitor diabetes cases were dismissed for lack of proof and the dismissal was upheld on appeal in 2018. Online claims to the contrary are selling something.

⚠ Statin safety — what to know before you start, and while you're on one.

- **Get emergency care the same day** for severe, unexplained muscle pain or weakness with dark, cola-coloured urine, fever or feeling unwell. That is rhabdomyolysis — rare, but it can damage the kidneys. Stop the statin and go.
- **Weakness that doesn't fade weeks after stopping** needs a CK blood test and an anti-HMGCR antibody test. It can be a rare autoimmune muscle disease (about 2–5 in 100,000 users) that needs treatment of its own and means no statin again.
- **Interactions cause most serious muscle injury.** Tell every doctor and pharmacist you take a statin. The main ones: certain antibiotics (clarithromycin, erythromycin), some antifungals (itraconazole, ketoconazole), HIV and hepatitis C drugs, cyclosporine, gemfibrozil, colchicine, high-dose niacin — and large amounts of grapefruit juice with simvastatin, lovastatin or atorvastatin. Red yeast rice *is* a statin; never take it with one. Pravastatin and rosuvastatin have the fewest interactions.
- **Higher risk of muscle side effects:** age over 80, small body size, kidney disease, low thyroid, heavy alcohol, and Asian ancestry (rosuvastatin starts at a lower dose). A gene test (SLCO1B1) flags people who shouldn't take high-dose simvastatin.
- **Liver:** test liver enzymes before you start; report yellowing skin or eyes, dark urine, upper-belly pain or unusual tiredness.
- **Blood sugar:** check HbA1c before you start and three months in.
- **Memory or confusion:** report it. In the FDA's review the cases were uncommon and went away when the statin stopped.
- **Pregnancy and breastfeeding:** most women stop a statin as soon as they learn they're pregnant; the FDA lifted its absolute ban in 2021 so that the very highest-risk women can decide with their doctors. Don't

breastfeed on a statin.

- **Never stop on your own** if you've had a heart attack, stent or stroke — talk to your doctor first.

⚠ **The Statin Spiral.** Chapter 9 of the book draws it: lower cholesterol → lower testosterone → less drive → less muscle → more fat → more insulin resistance → another prescription. The *shape* is real — one drug's side effect treated with the next drug is a pattern every family has watched. Two links are solid: the diabetes risk, and CoQ10 → tired muscles → less exercise → more belly fat → worse insulin sensitivity. Two links are weaker than the page says, and I'm saying so: the testosterone drop from the drug is small on average, and statins have not been shown to thin bones or cause depression.

Cholesterol and testosterone — what the science actually shows

"Your hormones are made from cholesterol, so lowering cholesterol must lower testosterone." The first half is true. The second half is true in some ways and false in others, and the difference matters. There are three ways the war on cholesterol could touch testosterone, and the evidence is different for each:

Route	What happens to testosterone	How sure
A statin — blocks cholesterol-making mostly in the liver, but a little reaches other tissues, likely including the testes	Down about 0.66 nmol/L [about 19 ng/dL], roughly 4%, on average across five trials in 501 men. Some men drop much more, and a man near the bottom of the range can be pushed under it. Gene studies that mimic a statin show the same drop in men.	Solid — small on average, real
Low blood cholesterol by itself — PCSK9 injections, ezetimibe, or people born with low LDL	No change. In trials that drove LDL below 40 mg/dL [1 mmol/L] — some under 15 [0.4] — for a year, testosterone, cortisol and the pituitary hormones held. Gene studies that mimic PCSK9 drugs and ezetimibe show no testosterone effect. The testes and adrenals make most of their own cholesterol. Only people born with almost *no* LDL at all show strain: the adrenals respond less under prolonged stress, and women make less progesterone.	Solid
A low-fat diet — the "eat lean" advice that came with the cholesterol scare	Down 10–15% — about 60 ng/dL [2.1 nmol/L] — in six controlled diet studies of 206 men, and up to 26% on low-fat vegetarian diets. Free testosterone fell too. The drop was larger in men of European ancestry.	Moderate — small, older studies, but consistent

What that means. The biggest effect of the anti-cholesterol era on men's hormones probably came from the plate, not the pill: a low-fat diet lowers testosterone about three times as much as a statin does on average. Low cholesterol in the blood isn't the lever. The statin's effect most likely comes from the small amount of drug that reaches the testes and slows cholesterol-making there — which is why drugs that lower LDL another way don't touch testosterone. And none of this explains the big picture. Young American men's testosterone fell from about 605 to 450 ng/dL [21 to 15.6 nmol/L] between 1999 and 2016; men aged 15 to 39 barely take statins, and fat intake had already stopped falling by then. The drivers this book is about — belly fat, insulin resistance, sitting still, short sleep, endocrine-disrupting chemicals and a long list of medications — carry most of that weight.

So the spiral the evidence carries is: *statin → a small hormone dip in most men, a bigger one in some → tired, achy muscles in some people → less movement → more belly fat and insulin resistance → lower testosterone and higher diabetes risk → the next prescription.* The hormones are in it; they enter through the belly fat more than through the drug. That is enough of a case without the parts it won't carry.

✅ **If you're a man on a statin, or about to start one:** test total testosterone, SHBG and free or bioavailable testosterone before you start and about three months in — morning, fasting. Keep dietary fat in the meal — olive oil, eggs, fish, some butter and red meat — rather than going low-fat, and lift weights. If testosterone falls into the low range, that's a conversation about dose, a different drug or a different route — not a reason to stop on your own.

✅ **If you take one.** Ask for the lowest dose that reaches the target and whether a water-soluble statin suits you — in small studies, rosuvastatin every other day kept most of the daily dose's effect with fewer complaints. Take ubiquinol with it. Keep K2 and vitamin D in your routine (no K2 with warfarin unless your doctor manages it). Have thyroid and vitamin D checked *before* you start — both, when low, raise LDL and cause the muscle aches that get blamed on the drug. Lift weights; it's the direct antidote to the spiral. Retest in six to eight weeks. And never stop on your own — stopping suddenly after a heart attack or stent raises risk in the following weeks.

✅ **If you decline one.** Ezetimibe (LDL-C down about 20%, almost no muscle effect) and bempedoic acid (works in the liver only) exist, and a PCSK9 injection is the heavy tool. The levers in Section 4 are the other route. Whatever you choose, put a plan and a retest date under it. High ApoB plus plaque plus no plan is not an option — in either direction.

9 Questions to Ask Your Doctor 🗣️

Bring this list. A good doctor will welcome it.

About the tests

1. Can you order ApoB with my next panel? If it isn't covered, what would it cost privately?
2. Can you add fasting insulin and hsCRP? I'd like to see my insulin sensitivity and inflammation, not just my cholesterol.
3. Have I ever had Lp(a) tested? If not, can we do it once?
4. Can we check a full thyroid picture — TSH, free T3, reverse T3 and TPO antibodies — along with vitamin D and ferritin?

About my numbers

5. Is my LDL calculated or measured? Could high triglycerides be distorting it?
6. Which of my numbers is driving your concern — the LDL cargo, the particle count, or the inflammation?

About the cause

7. Before we treat the number, what do you think is *causing* it? Could we test for the cause first?
8. If I change my diet and retest in eight to twelve weeks, would you re-decide then?

About imaging

9. Would a calcium score change your recommendation? Is a CT angiogram with plaque analysis available to me, since a calcium score misses soft plaque?

About a statin, if one is recommended

10. Which trigger am I meeting — the LDL-C-over-193 [5.0] line, my calculated risk, or plaque? And what is my number needed to treat — how many people like me would take this for five years to prevent one heart attack? The number needed to harm?
11. If I take it: the lowest dose that reaches the target, a water-soluble statin, CoQ10 alongside — and when do we retest? Are ezetimibe or bempedoic acid options, alone or instead?
12. If I'd rather try lifestyle first: what number, and what date, would make you say it's time for medication? And who do I call if I get muscle pain?

If a doctor won't order ApoB, won't test for a cause, or treats "retest in twelve weeks" as unreasonable, that tells you something too. You are allowed to be a participant in this.

10 My Tracker 📋

Print this page. Fill it in after each test. Trends tell the truth.

Test	Optimal	Date: _____	Date: _____	Date: _____	Date: _____
ApoB	under 80 mg/dL [0.80 g/L]				
Triglycerides ÷ HDL	under 1.0 (mg/dL) [0.44 (mmol/L)]				
LDL-C	read with triglycerides; confirm with ApoB				
HDL	50–90 mg/dL [1.29–2.33 mmol/L]				
Triglycerides	under 80 mg/dL [0.90 mmol/L]				
Non-HDL	under 130 mg/dL [3.4 mmol/L]				
Fasting insulin	2–5 µIU/mL [14–35 pmol/L]				
hsCRP	under 0.36 mg/L				
HbA1c	under 5.4%				
Lp(a) (once)	under 75 nmol/L				
Calcium score or CT angiogram	zero, or no growth				
Notes (season, diet, weight, medication)					

Key Takeaways 💡

1. Cholesterol is a building material your body makes on purpose. LDL and HDL are the trucks that carry it, not "bad" and "good" cholesterol — and total cholesterol is all the trucks added together.
2. ApoB — the truck count — is the number that matters, read with the triglyceride ÷ HDL ratio, which says what kind of trucks you're making. Where LDL-C and ApoB disagree, follow ApoB.
3. Your liver sets the number through its docks. A statin doesn't drain cholesterol; it starves the liver so it opens more docks — and starves CoQ10 along the way.
4. LDL-C alone is a poor guide. ApoB particles are the fuel; insulin resistance, inflammation, smoking and blood pressure are the fire. Low fire slows the damage; it doesn't stop it.
5. "Normal" ranges are full of sick people, and the line has moved again and again in sixty years. Know the line is a line — and measure your particles, your engine and your arteries instead.
6. Statins help most in people who already have disease and least in healthy people with a high number. Ask for your number needed to treat before you decide anything.
7. Find the root before you treat the number — then put a plan and a retest date under whatever you choose. High ApoB plus plaque plus no plan is not an option.
8. Plaque comes from how high ApoB is *and* how long it stays there. Know your number early, and keep measuring.

Action Steps ✅

1. 🩸 Order the five: ApoB, a fasting lipid panel (for the ratio), fasting insulin, hsCRP, HbA1c. Add Lp(a) once, and a full thyroid set if LDL is high.
2. 🧮 Calculate your triglyceride ÷ HDL ratio in one unit, and your LDL-C ÷ ApoB. Write both down.
3. 🔍 If ApoB is above 80 mg/dL [0.80 g/L], work through the roots in Section 4 before anything else.
4. 🥑 Make the three food moves that move trucks: cut refined carbohydrate and fructose; swap some saturated fat for olive oil, avocado and fish; add soluble fiber daily.
5. ⏱️ Retest in 8–12 weeks. If ApoB is still high — or you're over 40 with other risk — ask for a calcium score, or a CT angiogram with plaque analysis if you can get one.
6. 🗣️ Take the Section 9 questions to your appointment.
7. 📋 Copy your results into the tracker in Section 10.

Reflection Questions 🤔

1. Which of your five numbers have you actually seen — and which have you been letting a single "cholesterol number" stand in for?
2. If your doctor recommended a statin tomorrow, which trigger would it be — the number, the calculator, or a scan — and would you know enough to ask?
3. What would it take for you to treat the fire — your sleep, your sugar, your stress, your movement — with the same seriousness the system gives the fuel?

Sources: Heavenly Health Hacks, Fourth Edition (Chapters 5, 9 and 22); normal-range history (JAMA Cardiology 2017 editorial, citing the 1963 NEJM table of normal values); NIH Consensus Development Conference, Lowering Blood Cholesterol to Prevent Heart Disease (JAMA 1985); Satish et al., JAHA 2026 (first heart attacks in adults 18–54, 2011–2022); Lamiquiz-Moneo et al. 2019 systematic review of citrus bergamot (12 studies); NHANES/NCHS cholesterol trend reports (Carroll et al., JAMA 2005 and 2012; NCHS Data Briefs 177 and 363); Ford et al., NEJM 2007 (IMPACT model of the US coronary mortality decline); Canadian Cardiovascular Society 2021 dyslipidemia guideline (Pearson et al.); ACC/AHA 2018 cholesterol

guideline; EAS consensus on LDL causality (Ference 2017); Cholesterol Treatment Trialists' meta-analyses; Sachdeva et al. 2009 (LDL in 136,905 heart-attack admissions); INTERHEART (Yusuf 2004); Kaplan et al. 2017 (Tsimane); Ridker 2023 (residual inflammation vs residual LDL); SAMSON and StatinWISE (nocebo trials); Banach et al., Mayo Clinic Proceedings 2015, Qu et al., JAHA 2018, and Taylor et al., Atherosclerosis 2015 (CoQ10 and statin muscle symptoms); Bayer's withdrawal of cerivastatin (Baycol), 2001; Sattar 2010 (statins and diabetes); WOSCOPS (Shepherd 1995); TheNNT.com primary- and secondary-prevention summaries; Kristensen et al., BMJ Open 2015 (postponement of death); Schooling et al., BMC Medicine 2013 (statins and testosterone, meta-analysis of randomized trials); Schooling et al., eLife 2020 (Mendelian randomization of statins, PCSK9 inhibitors and ezetimibe on testosterone); Illingworth et al., J Clin Endocrinol Metab 1982 (adrenal function in hypobetalipoproteinemia); FDA statin labelling changes, February 2012 (liver, memory, blood sugar, interactions) and FDA Drug Safety Communication, July 2021 (pregnancy and breastfeeding); current statin prescribing information (myopathy, rhabdomyolysis, immune-mediated necrotizing myopathy, drug interactions); Blom 2015 and the BERSON trial (PCSK9 inhibition and steroid hormones); Lokeshwar 2021 (testosterone decline in young US men, NHANES 1999–2016); Whittaker 2021 (low-fat diets and testosterone); Gaziano et al., Circulation 1997 (triglyceride-to-HDL ratio and myocardial infarction); Sayed et al., JAMA Cardiology 2024 (ApoB distribution across LDL-C levels, NHANES 2005–2016); Marston et al., JAMA Cardiology 2022 (ApoB and myocardial infarction, UK Biobank); Wilkins et al., JACC 2016 (ApoB–LDL-C discordance and coronary calcium, CARDIA); Silbernagel et al., Biomedicines 2022 (LDL apoB-to-LDL-C ratio and small dense LDL, LURIC); Madsen et al., European Heart Journal 2017 (extreme high HDL and mortality, Copenhagen); Thompson et al., JAMA 2005 (erectile dysfunction and subsequent cardiovascular disease); Bryan Elliott's own LifeLabs and Cleerly reports, April 2026, and his November 2026 lipid panel, as told in Chapter 9 of Heavenly Health Hacks; Steptoe & Brydon, Health Psychology 2005 (stress responses and lipids three years later); Ockene et al., Archives of Internal Medicine 2004 (SEASONS study); Fernandez, Food & Function 2010, and Blesso & Fernandez, Nutrients 2018 (dietary cholesterol responders); Rist et al., Neurology 2019 (Women's Health Study, lipids and hemorrhagic stroke); Ma et al., Neurology 2019 (Kailuan cohort, LDL and intracerebral hemorrhage); Vreugdenhil et al., JCI 2001, and Netea et al., JCI 1996 (lipoproteins and bacterial endotoxin); Delplanque et al., JPGN 2015 (lipids in breast milk and formula); Hui et al., Pediatrics 2019 (breastfeeding and adolescent lipids); Dietschy, Turley & Spady, J Lipid Res 1993, and Turley et al., J Lipid Res 1995 (the liver's share of cholesterol synthesis); Björkhem & Meaney, ATVB 2004, and Vance, Dis Model Mech 2012 (brain cholesterol); Pathology Outlines and the World Gastroenterology Organisation on gallstone composition.