VITALS VAULT

A VITALS VAULT FIELD GUIDE

Read the heart numbers beyond LDL in 20 minutes

For anyone with a basic cholesterol panel and a worry: know what each number adds and what to ask.

8

CHAPTERS

20 minutes

Every claim is tied to a named study. References are on the last page.

Educational only. Not medical advice.

THIS GUIDE IS FOR YOU IF

- You have a standard cholesterol panel and wonder what it leaves out.
- You have heart disease in your family, or a worry you cannot name.
- You have heard of ApoB or Lp(a) and want to know what they add.
- You want better questions for your next appointment.

HOW TO USE IT

Read one chapter at a time. Each opens with the one thing to remember. Keep your latest panel beside you and tick the boxes as you go. Bring the question lists and the one-page summary to your next appointment. Every range is cited to a study or guideline, never a personal target.

WHAT IS INSIDE

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CHAPTER 1

How plaque builds, slowly

THE ONE THING TO REMEMBER

Plaque is cholesterol-carrying particles stuck in the artery wall, collected over decades, long before any symptom.

Fat does not dissolve in blood, so cholesterol rides inside lipoproteins [fat-and-protein shipping packages]. Those with a protein called apoB [the protein on plaque-causing particles] can slip under the artery lining and get stuck. Immune cells swallow them and swell into foam cells, the first lesion. Over decades a cap forms and calcium shows up late. Think of silt settling on a riverbank: no single flood does it.

WHAT A STUDY FOUND

In autopsy series, no advanced lesions appeared before age 11, but by age 40 about 70% of people had intermediate to advanced lesions (nearly 700 autopsies per age group). Limit: older cohorts. Today's figures are likely lower, with less smoking and more cholesterol treatment.

Stary, Atlas of Atherosclerosis (book), 2003

50%

of men, and about a third of women, who will have a major cardiac event have the first one before age 65.

COMMON BELIEF

"Plaque is only an older person's problem."

WHAT THE EVIDENCE SAYS

Autopsies of accident victims aged 26 and 27 already showed oxidized apoB particles inside immune cells and a thickened artery lining. Fatty streaks have been seen in the aortas of children aged 4 to 8. These are small observational series, but they match the slow timeline.

Standard risk calculators look ten years ahead and are driven mostly by age and sex. Under about 55 to 60, almost no one crosses a treatment line, yet about half of heart attacks and strokes happen before 60. Some researchers argue for a 30-year view built on particle count. US guidelines still use the 10-year score. A score describes a group: 7.5% means 92.5 in 100 similar people have no event.

CHAPTER 2

What a standard panel reports, and what it estimates

THE ONE THING TO REMEMBER

A standard panel gives four numbers, and the LDL on it is usually a calculation, not a direct measurement.

A panel lists total cholesterol, LDL-C [cholesterol carried in LDL particles], HDL-C [cholesterol carried in HDL particles] and triglycerides [fat carried mostly in VLDL]. Think of a weather app's feels-like temperature: it is worked out from other readings. Usually close, but less reliable at the extremes. The panel is a snapshot of blood only. It shows nothing about the vessel wall.

On the panel	What it is	How it is obtained
Total cholesterol	All cholesterol in all carriers	Measured directly
HDL-C	Cholesterol in HDL carriers	Measured directly
Triglycerides	Fat in the blood	Measured; a recent meal can raise it
LDL-C	Cholesterol in LDL carriers	Usually calculated from the rest

The common estimate, the Friedewald formula, is total cholesterol minus HDL-C minus triglycerides divided by 5. The 5 assumes VLDL carries about five times more triglyceride than cholesterol. It counts other particle types as LDL, and it includes the cholesterol in Lp(a) [an inherited LDL-like particle]. A newer estimate (Martin-Hopkins) was proposed to replace it.

WHAT A STUDY FOUND

Friedewald and colleagues published the calculation behind most reported LDL-C in 1972. Their own advice was not to use it above triglycerides of 400 mg/dL. Limit: this is a history note. The 150 to 200 mg/dL range where errors can start is one lipid specialist's rule of thumb.

Friedewald, Levy, Fredrickson, 1972

COMMON BELIEF

"A calculated LDL cannot be trusted."

WHAT THE EVIDENCE SAYS

It is an estimate, and it is usually fine. It becomes less accurate when triglycerides are high (typically above 400 mg/dL) and when LDL is very low (below about 70 mg/dL). A non-fasting sample can skew it. A direct LDL measures it in the sample. Ask which one you have rather than calling yours wrong.

ASK YOUR CLINICIAN

- **Was my LDL calculated or measured directly?**
- **Was my sample fasting, and does that change my triglycerides?**
- **Are my triglycerides or LDL in a range where the estimate is less reliable?**

BEFORE YOU READ YOUR PANEL

- [ ] Date of the draw, and whether I was fasting
- [ ] Whether LDL says calculated or direct
- [ ] Units on the report (mg/dL or mmol/L)
- [ ] I was not acutely ill at the draw (lipids shift)
- [ ] If I menstruate, the cycle day (LDL can shift 10 to 12 mg/dL)

CHAPTER 3

Counting particles: apoB and non-HDL

THE ONE THING TO REMEMBER

Plaque is delivered by particles, so counting particles (apoB) can tell a different story than weighing the cholesterol inside them.

Each plaque-causing particle carries exactly one apoB. So LDL-C weighs the cargo while apoB counts the delivery trucks. Two people can both have LDL-C of 125 yet different truck counts, because smaller particles carry less each. About 90 to 95% of apoB particles are LDL. Advanced panels add apoB, Lp(a), particle counts and sizes, and hs-CRP [a sensitive inflammation marker].

Measure	What it tells you	Catch
LDL-C	Cholesterol inside LDL particles	Usually calculated; a stand-in for particle count
Non-HDL-C	Total minus HDL-C; all plaque-type cholesterol	High with normal apoB can hint at leftover remnants
ApoB	Count of plaque-causing particles	Separate test; guidelines do not require it
LDL-P (NMR)	LDL particle count by NMR	Also counts Lp(a) particles; some labs only

WHAT A STUDY FOUND

In people already on cholesterol-lowering treatment, apoB and non-HDL-C reflected leftover (residual) risk better than LDL-C. In other comparisons of people whose numbers disagree, outcomes tended to follow apoB. Limit: observational analyses, and sample size is not reported here.

Johannesen et al., J Am Coll Cardiol, 2021

COMMON BELIEF

"ApoB has replaced LDL-C everywhere."

WHAT THE EVIDENCE SAYS

Experts disagree on routine use. A 2019 European guideline favors apoB. The 2018 US cholesterol guideline mentions apoB but is built around LDL-C. A 2024 US lipid-society consensus statement backs apoB, yet most clinicians still do not order it.

90-95% **of apoB particles are LDL, so apoB works as a rough LDL particle count. In one rare disorder it reflects remnants instead.**

ASK YOUR CLINICIAN

- Can apoB be added to my next blood draw?
- Do my LDL-C and apoB tell the same story?
- What does my non-HDL-C show, and is it free on my report?

CHAPTER 4

Lp(a): the inherited number worth knowing once

THE ONE THING TO REMEMBER

Lp(a) is a mostly inherited particle that a standard panel never shows, and it is steady for life.

Lp(a) [pronounced L-P-little-a] is an LDL-like particle with an extra protein attached. Your level is set mostly by genes, reaches its adult level by about age 2 and stays steady for life. Think of a car's throttle: Lp(a) is the fixed baseline setting you were born with, while other numbers move with the gas pedal. Levels differ by ancestry, and about 15 to 20% of people have a high one.

Item	What it means	Catch
Unit: nmol/L	Counts particles; the better measure	50 mg/dL is roughly 125 nmol/L
Unit: mg/dL	Weighs the whole particle	Depends on the extra protein's size
High flag	About 50 mg/dL or 125 nmol/L and up	Some groups use 30 mg/dL; labs differ
Guidelines	Canada, Europe: test once in everyone	US narrower in 2022; a 2024 society statement backs one test

WHAT A STUDY FOUND

Clarke and colleagues compared about 3,000 people with coronary disease and about 3,000 without (PROCARDIS). Gene variants that raise Lp(a) were more common in those with disease. Genes are fixed at birth, so this work helped show Lp(a) is a cause, not a bystander. Limit: no drug trial has yet shown fewer heart attacks from lowering it.

Clarke et al., N Engl J Med, 2009

COMMON BELIEF

"High Lp(a) means a heart attack is coming."

WHAT THE EVIDENCE SAYS

Not necessarily. Some people with very high levels have no plaque, while others with moderate levels need bypass surgery in their 50s. Researchers are still studying why. It adds risk; it does not decide it. Aortic stenosis [stiffening, narrowing of the aortic valve] is the condition most tied to high Lp(a).

What moves it? Cholesterol medicines of the statin class do not lower it, and in most studies raised it about 10%. Two other drug classes (niacin and PCSK9 antibodies) lower it roughly 20 to 30%, but neither has shown fewer heart attacks from that drop. A gene-silencing drug cut it about 80% in a phase 2 trial; outcome trials were pending in the 2022 source.

ASK YOUR CLINICIAN

- Has my Lp(a) ever been measured, and in which unit?
- Does my family history change whether I should be tested?
- If it is high, how should that change how my other numbers are read?

CHAPTER 5

Triglycerides, HDL and blood sugar

THE ONE THING TO REMEMBER

Triglycerides, HDL-C and blood sugar are linked clues to how your body handles fuel. They are clues, not verdicts.

When insulin runs high, a protein called apoC-III slows how fast blood fats are cleared. Triglycerides and VLDL particles [triglyceride-rich particles from the liver] rise, the liver makes more particles, apoB goes up and HDL-C falls. Think of a loading dock: when trucks unload slowly, they queue up and more are dispatched.

Marker	What it hints at	Limit
Triglycerides	Fat in blood; often rises with insulin resistance	Guideline high is 150; some say apoB holds most of its signal
HDL-C	Cholesterol in HDL particles	High does not cancel risk; raising it with drugs failed
TG to HDL-C ratio	Rough insulin gauge; under 1.0 suggests good	One clinician's rule; poor in African ancestry
AIP index	Log of TG to HDL-C; tracks small LDL	Top group had 28% more events; observational
A1C	Average blood sugar over 100 to 120 days	Misses spikes; some conditions skew it
TyG index	Triglycerides and glucose in one score	Screening estimate; studies disagree on its edge

WHAT A STUDY FOUND

Voight and colleagues used gene variants that raise or lower HDL-C, fixed at birth. They did not change heart attack risk the way a protective HDL would. A drug class that raised HDL-C a lot cut events only about 9% in one trial, in line with its 9% drop in LDL and apoB (an expert's recollection). Limit: HDL-C is still a useful clue.

Voight et al., Lancet, 2012

WHAT A STUDY FOUND

In 10,232 adults aged 45 to 79 without diagnosed diabetes (EPIC-Norfolk), event and death rates were lowest at A1C below 5%. Each 1-point rise went with 1.24 to 1.28 times the all-cause death risk, after adjusting for smoking, cholesterol, blood pressure and weight. Limit: observational, and blood conditions can skew A1C.

Khaw et al., EPIC-Norfolk, 2004

COMMON BELIEF

"High HDL, the good cholesterol, means I am protected."

WHAT THE EVIDENCE SAYS

High HDL-C does not cancel risk. In a rare disorder, very high HDL-C goes with advanced plaque. In people of African ancestry it is not reliably protective. Check apoB whatever HDL-C says. Low HDL-C is still a useful clue to insulin resistance.

ASK YOUR CLINICIAN

- **Do my triglycerides and HDL-C hint at insulin resistance?**
- **Was my glucose or A1C measured, and can I see it?**
- **Should I repeat these fasting?**

CHAPTER 6

Inflammation and hs-CRP

THE ONE THING TO REMEMBER

hs-CRP is a general inflammation signal, not a heart test on its own, and a cold can throw it off.

hs-CRP [a sensitive inflammation marker] works like a smoke alarm: it says something is burning, not where. It rises with infections, fatty liver and metabolic problems. A cold can push it to 6 or 7 when normal is around 1. For heart purposes, two readings at least two weeks apart are advised, because it varies from person to person.

Level	Category (AACE/ACE)	Note
Under 1 mg/L	Normal	Standard lab range is 0 to 1.0
1 to 3 mg/L	Intermediate cardiovascular risk	Some experts flag 2+; US cutoffs are 1 and 3
Above 3 mg/L	High risk category	Check for recent illness first
About 6 to 7	Seen with a cold, for example	Repeat when well

WHAT A STUDY FOUND

A meta-analysis pooled 14 studies and 83,995 people. Higher hs-CRP went with higher all-cause and cardiovascular death, especially at 1 mg/L or above. Limit: association only. hs-CRP is non-specific, so it cannot say where the inflammation comes from.

Li et al., meta-analysis, 2017

WHAT A STUDY FOUND

In 10,181 health-checkup adults, mean hs-CRP was 0.5 mg/L with no fatty liver, 0.9 with mild and 1.6 with moderate to severe fatty liver. Limit: cross-sectional, so it shows a link, not a cause. It shows how many different things can move one hs-CRP number.

Yu et al., 2018

COMMON BELIEF

"A normal hs-CRP means my arteries are clear."

WHAT THE EVIDENCE SAYS

No. One lipid specialist described a person with a coronary calcium score of 10 and an hs-CRP under 1, so it can miss early plaque. Inflammation markers are linked to risk but not proven to cause it. Experts differ on how much weight they deserve.

ASK YOUR CLINICIAN

- **Was my hs-CRP drawn when I was well?**
- **Should I repeat it in a few weeks?**
- **What else could be raising it, such as liver fat or an inflammatory condition?**

CHAPTER 7

Blood pressure, measured properly

THE ONE THING TO REMEMBER

One office reading is not your blood pressure. A few weeks of correct home readings give a truer picture.

Blood pressure injures the vessel lining mechanically, which helps plaque particles get in. In a pooled analysis of about 1 million adults, each 20 mmHg [the unit for blood pressure] higher systolic (10 diastolic) went with about double the risk of death from stroke or heart disease. Readings swing with posture, talking and stress, so method matters. Think of a scale on a tilted floor.

Common error	Approximate effect
No 5-minute rest first	10 to 20 mmHg higher
Talking during the reading	10 to 15 mmHg higher
Full bladder	10 to 15 mmHg higher
Back unsupported, feet dangling	5 to 10 mmHg higher
Crossed legs	5 to 8 mmHg higher
Automatic cuff estimating the numbers	Can read 10 to 15 mmHg high

HOME READING ROUTINE

- [ ] No caffeine, exercise or smoking for 30 minutes before
- [ ] Empty bladder, sit quietly 5 minutes, back supported
- [ ] Feet flat, arm at heart level, cuff on bare skin
- [ ] Cuff fits your arm; a wrong size shifts the number
- [ ] Take two readings a minute apart and average them
- [ ] Log readings over several weeks and bring the log

WHAT A STUDY FOUND

SPRINT enrolled about 10,000 adults aged 50 or older with systolic pressure of 130 or more and high risk, without diabetes. Aiming under 120 versus under 140 (reached 121.4 vs 136.2) cut cardiovascular events 25% (1.6 points absolute at 3 years) and deaths 27%. It stopped early, at about 3.3 years. Limit: high-risk people without diabetes, not a rule for everyone.

SPRINT Research Group, N Engl J Med, 2015

120/80

Current US categories: normal is under 120/80; elevated is 120 to 129 with diastolic under 80; stage 1 is 130 to 139 or 80 to 89; stage 2 is 140/90 or above.

ASK YOUR CLINICIAN

- Is my home cuff validated, and is the size right for my arm?
- What reading range counts as normal for me?
- If readings stay high, should a cause beyond lifestyle be checked?

CHAPTER 8

Putting it together: your numbers and your family

THE ONE THING TO REMEMBER

No single number settles risk. Read them together, and tell your clinician your family history.

Number	What it adds	Its limit
LDL-C	Cholesterol in LDL; the usual start	Often estimated; can understate particle count
ApoB	Counts plaque-causing particles	Experts differ on routine use
Non-HDL-C	All plaque-type cholesterol, free on a panel	Cholesterol, not a particle count
Lp(a)	Inherited particle; test once	Level alone does not say who gets disease
Triglycerides, HDL-C, A1C	Clues to insulin resistance	Clues, not causes; apoB covers much
hs-CRP	General inflammation	Non-specific; a cold skews it
Blood pressure	Direct strain on vessel lining	Needs correct repeated readings

Family history is not on your lab report, yet it changes how you read it. Familial hypercholesterolemia (FH) [inherited very high LDL cholesterol] is usually flagged by LDL-C above 190 mg/dL, and risk at any age is much higher than with LDL-C below 130. One preventive cardiologist put FH at about 1 in 250 people. Early heart disease in relatives, or a high Lp(a), is worth telling a clinician.

WHAT A STUDY FOUND

Ference and colleagues compared lower LDL-C and risk across three study types. Each 1 mmol/L (about 40 mg/dL) lower went with about 22% lower risk over a median 5 years in randomized trials, about 32% over 12 years in cohorts, and more again in genetic studies with a median 52 years of exposure. Limit: designs differ. It shows time matters, not what to do.

Ference et al., Eur Heart J, 2017

YOUR FAMILY HEART HISTORY

Relative and how they are related _____

Heart attack, stent, stroke or valve problem _____

Age when it happened _____

Any known LDL-C, apoB or Lp(a) result _____

Date of my own last panel _____

ASK YOUR CLINICIAN

- Given my family history, which tests beyond a basic panel make sense?
- Do my LDL-C and apoB agree, and has my Lp(a) been checked?
- How should I measure blood pressure at home, and how often?
- When should I repeat these tests?
- What result would change the plan, and what would not?

BRING TO YOUR APPOINTMENT

- [ ] My latest panel with date, units and fasting status
- [ ] My filled-in family history page
- [ ] A few weeks of home blood pressure readings
- [ ] A list of my medicines and supplements

THE WHOLE GUIDE ON ONE PAGE

If you remember nothing else

1. Plaque builds over decades as apoB particles stick in artery walls; early lesions appear in young people.
2. A basic panel reports cholesterol amounts, and the LDL on it is usually calculated, not measured.
3. ApoB counts plaque-causing particles; if it disagrees with LDL-C, risk tends to follow it. Experts differ on use.
4. Lp(a) is mostly inherited and steady for life; several guidelines support testing it once.
5. Triglycerides, HDL-C and A1C are clues to insulin resistance; a high HDL-C does not cancel risk.
6. hs-CRP is a non-specific inflammation signal; repeat it when you are well.
7. Blood pressure needs correct, repeated readings; one office number can mislead.
8. Family history plus your own numbers, read together and repeated over time, tell the story.

WHAT THIS GUIDE CANNOT DO

This guide cannot tell you your risk, diagnose anything, or say what to do about a result. A number describes a group, not you. Many findings here are associations, some are expert opinion, and experts disagree on how widely to use apoB and particle tests. It does not suggest taking or avoiding any medicine. Blood tests also cannot show how much plaque is in your arteries.

A FREE NEXT STEP

Our short, free page on the blood markers that give a fuller picture of heart health covers the numbers in this guide in one place. Your own results, repeated over time, are the piece a guide cannot give you.

vitalsvault.com/lp/heart-cardiometabolic

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This guide is educational. It is not medical advice and not a diagnosis, and it does not replace a clinician who knows your history. Study findings describe groups of people and may not apply to you.