

LIFEVerity™

The Biomarker Reference

Eight measurements. One consistent standard.

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A field companion to The Biomarker Map

HOW TO USE THIS REFERENCE

Context before certainty.

Each entry separates conventional context from evidence context, names a common misread, and points to the chapter in The Biomarker Map where fuller reasoning and integration appear.

Numeric policy

This reference states diagnostic thresholds, staging boundaries, and guideline-defined risk categories when they clarify what a measurement means. It does not reproduce treatment goals or convert population guidance into a personal target; those depend on overall risk, clinical context, and shared decision-making.

Educational boundary

General education about biological measurements and health concepts. Nothing here interprets individual results, recommends testing or treatment, replaces care from a qualified clinician, or creates a physician-patient relationship.

THE EIGHT ENTRIES

Entry 01 Apolipoprotein B (ApoB)

Entry 02 Lipoprotein(a)

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Editorial source check: 11 October 2026. The online entries remain the current source if this compiled edition and the website ever differ.

ENTRY 01 · PARTICLE BURDEN

Apolipoprotein B (ApoB)

A marker of atherogenic particle number, rather than the cholesterol those particles carry.

CONVENTIONAL CONTEXT	Reported in mg/dL. Laboratories may display their own intervals.
EVIDENCE CONTEXT	No universal ApoB cutoff defines health. Its usefulness depends on baseline risk and whether it adds information beyond LDL-C and non-HDL-C.
IN BIOMARKER MAP	Chapter 12 - Apolipoprotein B

ApoB answers a different question from LDL cholesterol. LDL-C estimates cholesterol mass inside LDL particles; ApoB more closely reflects the number of circulating atherogenic particles.

Why the distinction matters

Each atherogenic lipoprotein particle carries one molecule of apolipoprotein B. When particles carry different amounts of cholesterol, two people can have similar LDL-C values but different particle burdens. That discordance is why ApoB can add information when the conventional cholesterol panel and the broader metabolic picture do not align.

The 2026 ACC/AHA dyslipidemia guideline describes selective ApoB measurement as useful for assessing residual cardiovascular risk in people with cardiovascular-kidney-metabolic syndrome, type 2 diabetes, elevated triglycerides, or known cardiovascular disease after LDL-C and non-HDL-C goals have been reached. It is an additional risk marker - not a standalone diagnosis.

Why this entry does not give one ApoB target

Current ACC/AHA guidance uses ApoB to refine risk and guide decisions about treatment intensification; it does not define one universal ApoB diagnostic cutoff. Published ApoB treatment goals vary by risk tier. Because those goals depend on a person's complete cardiovascular context, this general reference does not reproduce them as if they were population categories.

THE COMMON MISREAD

A reassuring LDL value is not always the whole answer.

LDL-C can be accurate while leaving particle number incompletely described. The useful question is whether ApoB changes the interpretation of the complete risk picture, not whether one marker "wins."

SOURCES

- 2026 ACC/AHA Guideline on the Management of Dyslipidemia - ACC summary - Selective use of ApoB for residual-risk assessment.
- 2026 ACC/AHA Dyslipidemia Guideline - JACC - Full multisociety clinical practice guideline.

EDUCATIONAL BOUNDARY	This entry does not interpret an individual ApoB or LDL-C result, establish a personal target, or recommend testing or treatment.

ENTRY 02 · INHERITED RISK

Lipoprotein(a)

An inherited particle that can materially change cardiovascular-risk interpretation.

CONVENTIONAL CONTEXT	Reported in nmol/L or mg/dL. The units are not exactly interconvertible.
EVIDENCE CONTEXT	Risk rises across a continuum. Published categories support risk stratification but do not function as personal treatment targets.
IN BIOMARKER MAP	Chapter 13 - Lipoprotein(a)

Lp(a) resembles an LDL particle with an additional apolipoprotein(a) component. Its concentration is largely genetically determined and usually changes little with lifestyle.

Why it behaves differently

Because Lp(a) is relatively stable and independently associated with atherosclerotic cardiovascular disease, current guidance treats it primarily as a risk-classification marker. The 2024 National Lipid Association update supports measuring it at least once in every adult for risk stratification; the 2026 ACC/AHA guideline also recommends at least one adult measurement.

The NLA statement classifies values below 75 nmol/L as lower risk, 75 to <125 nmol/L as intermediate, and 125 nmol/L or higher as high risk. For results reported in mg/dL, the corresponding published categories are below 30, 30 to <50, and 50 or higher. These are risk categories, not diagnoses. Mass and molar units should not be converted with a fixed factor because particle size varies.

THE COMMON MISREAD

Treating Lp(a) as a lifestyle score.

Its value is not that it grades effort. It can reveal inherited risk that is not apparent from habits or a standard lipid panel and can change how the rest of the risk picture is interpreted.

SOURCES

- 2024 National Lipid Association focused update on Lp(a) - Adult testing recommendation, categories, units, and interpretation.
- 2026 ACC/AHA Dyslipidemia Guideline - ACC summary - At-least-once adult measurement and role in risk refinement.

EDUCATIONAL BOUNDARY	This entry does not interpret an individual Lp(a) result or recommend testing, medication, supplements, or other treatment.

ENTRY 03 · COMPENSATION

Fasting insulin

How much insulin is present after fasting to help keep glucose regulated.

CONVENTIONAL CONTEXT	Intervals vary substantially by assay and laboratory.
EVIDENCE CONTEXT	There is no universal optimum. Routine insulin testing is not recommended for most people with diabetes or at risk; interpretation requires the clinical context.
IN BIOMARKER MAP	Chapters 17-18 - Insulin and Compensation

Fasting insulin can contribute to a metabolic pattern, but it is not a standardized diagnostic line and it does not have a universally accepted "optimal" value.

Why the number resists a simple target

Insulin assays are not sufficiently harmonized for one laboratory's value to be treated as interchangeable with another's. Direct measurement of insulin resistance also requires research methods such as the hyperinsulinemic-euglycemic clamp. For routine clinical interpretation, expert laboratory guidance emphasizes the consequences and surrounding pattern - glucose tolerance, blood pressure, body composition, triglycerides, HDL cholesterol, and other findings - rather than insulin concentration alone.

ADA/AACC guidance does not recommend routine insulin or proinsulin testing for most people with diabetes or at risk for diabetes or cardiovascular disease. Repeated measurements should not be treated as a validated stand-alone measure of metabolic health.

THE COMMON MISREAD

A printed laboratory interval is not a universal metabolic verdict.

An in-range result does not establish metabolic health, while a single higher result does not diagnose insulin resistance. Method, fasting conditions, medications, physiology, and the accompanying pattern matter.

SOURCES

• Guidelines and Recommendations for Laboratory Analysis in the Diagnosis and Management of Diabetes Mellitus - American Diabetes Association and laboratory-medicine guidance on insulin assays and insulin-resistance measurement.

EDUCATIONAL BOUNDARY	This entry does not define an optimal fasting-insulin value, diagnose insulin resistance, or recommend testing or treatment.

ENTRY 04 · LONGER-TERM GLYCEMIA

Hemoglobin A1c

An estimate of average glycemia over the life of circulating red blood cells.

CONVENTIONAL CONTEXT	Below 5.7%; 5.7-6.4%; and 6.5% or higher on appropriately confirmed testing.
EVIDENCE CONTEXT	Diagnostic categories are standardized. Treatment goals are individualized, and discordance with glucose may signal interference.
IN BIOMARKER MAP	Chapter 19 - Hemoglobin A1c

HbA1c is standardized for screening and diagnosis, but the number remains an estimate shaped by both glucose exposure and red-cell biology.

Categories and context

The American Diabetes Association's 2026 Standards classify HbA1c below 5.7% as below the prediabetes range, 5.7-6.4% as prediabetes, and 6.5% or higher as meeting a diagnostic criterion for diabetes when appropriately confirmed. These are diagnostic categories, not universal treatment goals.

Conditions that change red-cell turnover or the relationship between HbA1c and glycemia can distort the result. The ADA advises evaluating possible interference when A1c and glucose are substantially discordant and using plasma-glucose criteria in selected conditions, including some hemoglobin variants, pregnancy, G6PD deficiency, HIV, and disorders affecting red-cell turnover.

THE COMMON MISREAD

Treating one value as a direction.

The same HbA1c can represent a stable pattern, improvement from a higher level, or progression from a lower one. Trend and biological context change what the snapshot can support.

SOURCES

• ADA Standards of Care in Diabetes - 2026: Diagnosis and Classification - Diagnostic categories, assay requirements, confirmation, and conditions affecting interpretation.

EDUCATIONAL BOUNDARY	This entry does not diagnose prediabetes or diabetes, establish an individual glycemic goal, or recommend treatment.

ENTRY 05 · REGULATORY PATTERN

Thyroid tests: TSH, free T4, and T3

A set of related thyroid signals rather than a single verdict.

CONVENTIONAL CONTEXT	TSH intervals are laboratory- and population-dependent; free-hormone values are method-dependent.
EVIDENCE CONTEXT	TSH and free T4 form the central pattern. T3 and antibodies are used selectively according to the question.
IN BIOMARKER MAP	Chapter 25 - Thyroid

TSH is often the first thyroid test considered, but its meaning depends on the integrity of the pituitary-thyroid axis and its relationship to free thyroid hormone measurements.

Why the panel matters

The American Thyroid Association describes TSH as generally more sensitive than free T4 because of their log-linear relationship. Free T4 more directly reflects circulating unbound thyroxine and is commonly interpreted alongside TSH. T3 testing has selected uses, particularly in hyperthyroidism, and may remain normal even in hypothyroidism. Free T3 assays can be unreliable and are not routinely helpful.

Results depend on method-specific reference intervals. The ATA's 2023 review emphasizes that different instrument systems can produce different absolute values and that interference from biotin, medications, antibodies, illness, and other factors should be considered when results and the clinical picture do not agree.

THE COMMON MISREAD

Treating TSH as self-explanatory.

A TSH value is part of a regulatory system. Free T4, the clinical question, medications, illness, timing, assay method, and - when appropriate - antibodies or other tests determine how much meaning it can bear.

SOURCES

- 2023 American Thyroid Association commissioned review of TSH, T4, and T3 - Assay variation, reference intervals, interference, and relationships among thyroid tests.
- American Thyroid Association: Thyroid Function Tests - Patient-facing explanation of TSH, free T4, and T3.

EDUCATIONAL BOUNDARY	This entry does not interpret an individual thyroid panel, diagnose thyroid disease, or recommend medication or supplements.

ENTRY 06 · FILTRATION ESTIMATE

eGFR with cystatin C

An estimate of kidney filtration whose slope can be as important as its current value.

CONVENTIONAL CONTEXT	G1 is 90 mL/min/1.73 m² or higher. G1 and G2 alone do not establish chronic kidney disease without other markers of kidney damage. A value below 60 sustained for at least three months contributes to the CKD definition.
EVIDENCE CONTEXT	Trend, albuminuria, age, muscle mass, illness, and confirmatory cystatin C can change the meaning of a single estimate.
IN BIOMARKER MAP	Chapter 31 - Kidney Function and Longevity

eGFR is an estimate, not a direct measurement. Creatinine is the usual starting point, while cystatin C can refine the estimate when creatinine may be misleading.

One function, several lenses

KDIGO recommends creatinine-based eGFR as the most widely used initial assessment. Creatinine is affected by factors other than filtration, particularly muscle mass and body habitus. For adults at risk for chronic kidney disease, KDIGO recommends using the combined creatinine-cystatin C estimate to determine the GFR category when cystatin C is available.

Kidney evaluation is two-dimensional: filtration category and evidence of damage such as albuminuria. A value below 60 mL/min/1.73 m² that persists for at least three months contributes to the definition of chronic kidney disease, but duration, albuminuria, trajectory, age, medications, acute illness, and non-filtration influences all affect interpretation.

THE COMMON MISREAD

Reading one estimate as a measured verdict.

A stable eGFR of 65 over many years and a rapid fall to 65 are not the same pattern. The equation used, the marker used, and the slope all matter.

SOURCES

- KDIGO 2024 Clinical Practice Guideline for Chronic Kidney Disease - GFR evaluation, creatinine and cystatin C equations, albuminuria, and chronicity.

EDUCATIONAL BOUNDARY	This entry does not diagnose kidney disease, interpret an individual eGFR, or recommend testing, referral, or treatment.

ENTRY 07 · IRON AND INFLAMMATION

Ferritin

One measurement that can reflect iron stores and inflammation at the same time.

CONVENTIONAL CONTEXT	Intervals vary by sex, age, assay, population, and inflammatory state.
EVIDENCE CONTEXT	Ferritin belongs with transferrin saturation and inflammatory context. It often raises a question before it answers one.
IN BIOMARKER MAP	Chapters 9 & 14 - Standard Panels; Inflammation

Ferritin is useful because it reflects stored iron. It is difficult because it is also an acute-phase reactant that may rise with inflammation.

Why one value can answer two questions

A low ferritin concentration can support iron depletion before hemoglobin or mean corpuscular volume changes. A higher value can reflect greater iron stores, inflammation, liver disease, infection, metabolic conditions, or other processes. WHO guidance therefore treats ferritin as a valuable iron-status marker while emphasizing adjustment and interpretation in the presence of inflammation.

Transferrin saturation supplies a different view of iron availability and is often needed to distinguish an iron-storage question from an inflammatory one. Laboratory intervals also vary by age, sex, assay, and population.

THE COMMON MISREAD

Treating ferritin as a standalone iron verdict.

A raised value is not synonymous with iron overload, and a value near the lower end of a printed interval does not by itself exclude depleted stores. The surrounding iron studies and inflammatory context decide which question the number is answering.

SOURCES

- WHO guideline on the use of ferritin concentrations to assess iron status - Ferritin as an iron-status indicator and the effect of inflammation on interpretation.

EDUCATIONAL BOUNDARY	This entry does not interpret an individual ferritin result, diagnose iron deficiency or iron overload, or recommend supplements or treatment.

ENTRY 08 · MODEL OUTPUT

Biological age

An estimate of aging-related patterns - not a direct measurement of how old the body "really" is.

CONVENTIONAL CONTEXT	No universal reference range. Results depend on the clock, sample, model, and laboratory.
EVIDENCE CONTEXT	Different clocks answer different questions. Model uncertainty and added value beyond established measures remain central limitations.
IN BIOMARKER MAP	Chapter 26 - Biological Age

Biological-age clocks compress multiple measurements into a single estimate. The result depends on what the model was trained to predict and the population in which it was developed.

Different clocks, different questions

Some clocks are trained primarily to reproduce chronological age. Others are designed around mortality, health outcomes, molecular patterns, or pace of aging. They may use DNA methylation, proteins, metabolites, clinical measurements, functional measures, imaging, or combinations of data. Two clocks can therefore produce different answers from the same person without either being a simple laboratory error.

An npj Aging perspective published online in December 2025 emphasizes that a useful clock must be accurate, consistent, generalizable, and explicit about prediction uncertainty. It also asks whether the clock adds meaningful information beyond established risk measures. Those requirements are not yet met uniformly across available tests.

THE COMMON MISREAD

Treating an estimate as the thing itself.

A younger or older score can create false reassurance or unnecessary alarm if the model, uncertainty, sample, and intended purpose are ignored. A change in one clock does not automatically establish a change in health or longevity.

SOURCES

- Do we actually need aging clocks? - npj Aging (2026; online 2025) - Perspective on validation, uncertainty, clinical meaning, and the purposes aging clocks can and cannot yet serve.

EDUCATIONAL BOUNDARY	This entry does not validate a commercial clock, interpret an individual biological-age result, or recommend testing or interventions.