



**FUNCTIONAL
MEDICINE**

Continuing Education

Beyond Traditional Lipids: Case-Driven Cardiometabolic Risk Assessment with Advanced Biomarkers

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Disclosures

- Dr. Hartzler
 - Paid speaker for NovoNordisk Diabetes and Abbott Diabetes
 - Member of the Scientific Advisory Board for Designs for Health

Objectives

- Describe the clinical significance of advanced cardiometabolic biomarkers including PLAC® activity, oxidized LDL, small dense LDL, Lp(a), apolipoproteins (A1/B), and inflammatory markers and how they complement traditional lipid testing.
- Interpret real patient case data from the Cardiovascular Risk Profile and differentiate between patterns suggesting elevated active atherogenesis, plaque instability, or systemic inflammation.
- Apply evidence-based interventions tailored to specific biomarker abnormalities, including lifestyle protocols (nutrition, physical activity, weight optimization), targeted supplementation, and when appropriate, medication considerations.
- Construct individualized care plans integrating biomarker findings with traditional risk scores (e.g., Framingham/ACC/AHA algorithms) and patient phenotypes to mitigate cardiometabolic risk more precisely.
- Evaluate outcomes and follow-up strategies by selecting appropriate re-assessment intervals and actionable clinical goals driven by patient results and best practice evidence.

Top Five CVD Risk Factors

Hypertension

Diabetes

Dyslipidemia

Obesity

Smoking

Standard Panels

Standard Lipid Panel

Core Measurements

Total Cholesterol

LDL-C (Calculated)

HDL-C

Triglycerides

What Standard Panels Miss

Particle Number & Quality

ApoB / LDL-P

Total atherogenic particle count

sdLDL & Size

Small, dense, pro-atherogenic

Oxidized LDL

Modified, inflammatory LDL

Lipoprotein(a)

Genetic thrombotic risk

Inflammation & Metabolism

PLAC & hs-CRP

Vascular inflammation/
instability

Insulin & LP-IR

Early metabolic resistance

Why Go Beyond Traditional Lipids?

The "CHD Gap"

~50%

of CHD/MI Patients Have "Normal" Lipids

Coronary events occur frequently despite standard panels showing values within reference ranges.

<100 mg/dL

Admission LDL-C in CAD Hospitalization

Analysis of 136,905 hospitalizations (2000-2006) showed nearly half had optimal LDL levels.

Limitations of LDL-C

Discordance Matters

- LDL-C underestimates risk when discordant with apoB, LDL-P, or non-HDL-C. Low LDL-C does not always mean low particle burden.

Predictive Ratios

- Ratios like TC/HDL-C and TG/HDL-C often predict mortality better than LDL-C alone, especially in metabolic syndrome.

The Goal of Advanced Testing

To identify risk factors standard panels miss:

- ✓ Particle Burden
- ✓ Particle Quality
- ✓ Oxidative Injury
- ✓ Inflammation

LDL-C Discordance



Why Discordance Matters

- Occurs when LDL-C levels are low or normal, but atherogenic particle number (ApoB or LDL-P) is elevated.
- Standard LDL-C underestimates cardiovascular risk when discordant. Particle number drives risk more than cholesterol content.



EPIC-Norfolk Study Evidence

- 21,448 participants without diabetes or CHD between 45 and 79 years of age were followed for 11 years
- A total of 2,086 participants developed CHD during follow-up
- Independently of their LDL-C levels, participants with high non-HDL-C levels, high TG levels, or with an elevated TC/HDL-C ratio were at increased CHD risk

Non-HDL Cholesterol & Key Ratios

The Calculation

$$\text{Non-HDL-C} = \text{Total-C} - \text{HDL-C}$$

What It Captures

Represents the cholesterol content of ALL atherogenic, ApoB-containing particles:



More comprehensive than LDL-C alone

Clinical Pearl

Non-HDL-C outperforms LDL-C for risk prediction in many studies (e.g., Framingham, lipid-lowering trials) and requires no fasting.

Clinical Application

Better Predictor of CVD

- Superior to LDL-C, especially in patients with high triglycerides, diabetes, or metabolic syndrome where remnant particles are elevated.

Key Ratios

TC/HDL-C Ratio
> 4.0
Increased Risk

TG/HDL-C Ratio
> 3.0
Insulin Resistance

Therapeutic Goals

Moderate Risk < 130 mg/dL

High Risk < 100 mg/dL

Very High Risk < 85 mg/dL

Advanced Cardiovascular Risk Profile

Category	Key Biomarkers	Clinical Significance
Lipoprotein Particles	LDL-P, HDL-P, small LDL-P, LDL size/pattern	Measures particle burden and size quality. Small, dense LDL (sdLDL) is more atherogenic.
Apolipoproteins & Genetic	apoB, apoA1, Lp(a)	ApoB captures total atherogenic load. Lp(a) is an independent, genetic risk factor.
Inflammation & Oxidation	oxLDL, PLAC Activity, hs-CRP, homocysteine	Identifies active plaque instability, vascular inflammation, and oxidative modification.
Metabolic & Glycemic	glucose, fasting insulin, LP-IR score, 1,5-AG	Detects insulin resistance and glycemic variability early, often years before diabetes.
Adipokines	leptin, adiponectin	Signaling molecules linking adipose tissue dysfunction to cardiometabolic risk.

CardioMetabolic Profile; serum

LIPIDS/RATIOS	RESULT / UNIT		REFERENCE INTERVAL	CARDIOVASCULAR RISK				
				LOW RISK	MODERATE RISK	HIGH RISK		
Total Cholesterol	173	mg/dL	< 200					
Triglycerides	269	mg/dL	< 150					
HDL Cholesterol	41	mg/dL	> 60					
LDL Cholesterol (calculated)	90.0	mg/dL	< 100					
VLDL Cholesterol (calculated)	43.0	mg/dL	< 30.0					
Non-HDL Cholesterol (calculated)	134	mg/dL	< 130					
OxLDL Cholesterol	44	U/L	< 60					
sdLDL Cholesterol (calculated)	41	mg/dL	< 35					
Lp(a)	< 4	mg/dL	< 30					
Total Cholesterol : HDL-C	4.3		< 4.0					
LDL-C : HDL-C	2.2		< 2.0					
OxLDL-C : LDL-C	0.49		< 0.45					
sdLDL-C : LDL-C	0.45		< 0.34					
Apo B : Apo A-1	0.80		< 0.80					
RISK FACTORS/INFLAMMATORY MARKERS								
PLAC (LP-PLA ₂ Activity)	168	U/L	< 151					
Homocysteine	5.1	µmol/L	< 11.0					
hsCRP	22	mg/L	< 1.0					
APOLIPOPROTEINS				PERCENTILE				
				2.5 th	16 th	50 th	84 th	97.5 th
Apolipoprotein A-1	126	mg/dL	115 - 220					
Apolipoprotein B	104	mg/dL	50 - 130					
METABOLIC RISK MARKERS								
Insulin	43.8	µIU/mL	2.8 - 18.0					
Glucose	146	mg/dL	70.0 - 100					
1,5-Anhydroglucitol (1,5 AG)	17	ug/mL	6.8 - 29					
*Leptin	49	ng/mL	4.0 - 39					
*Adiponectin	1.0	µg/mL	4.0 - 20					
Leptin : Adiponectin ratio	48.3		0.20 - 3.40					
Cystatin C	1.1	mg/L	0.5 - 1.5					
Creatinine	0.8	mg/dL	0.6 - 1.3					
eGFR (calculated)	81	mL/min	> 60					

Oxidized LDL (oxLDL)

What It Reflects

Oxidized LDL represents the oxidative modification of LDL particles. Unlike native LDL, oxLDL is not recognized by normal LDL receptors but is taken up by macrophage scavenger receptors, leading directly to foam cell formation and plaque progression.

Pathology

1. Modification

ROS modifies LDL apoB & lipids

2. Inflammation

Triggers endothelial dysfunction

3. Foam Cells

Unregulated uptake by macrophages

Clinical Associations

- Present across all stages of atherosclerosis
- Elevated in chronic inflammatory conditions (RA, Hashimoto's)
- Strong predictor of acute coronary syndromes
- High Level = Active Oxidative Stress

Small Dense LDL (sdLDL) & LDL-P

Why Particle Size & Number Matter

Standard LDL-C measures the concentration of cholesterol within particles, but LDL Particle Number (LDL-P) measures the actual burden of atherogenic particles. Small dense LDL (sdLDL) particles are more dangerous because they penetrate the arterial wall more easily and are more susceptible to oxidation.

Atherogenicity

1. Penetration

Smaller size = easier entry into subendothelial space

2. Oxidation

Reduced antioxidant content = highly oxidizable

3. Clearance

Lower affinity for LDL receptor = longer residence time

Pattern A vs. B

Pattern A (Lower Risk)

- Large, buoyant LDL particles predominating.

Pattern B (Higher Risk)

- Small, dense LDL particles predominating.

- Low LDL-P represents lower risk
- High numbers of small, dense LDL particles- highest risk (Pattern B “bad”)
- High numbers of large, buoyant LDL-P represent a higher risk (Pattern A “not as bad”)

Highest Risk: <u>HIGH</u> number of <u>SMALL</u> LDL particles	Low Risk: <u>LOW</u> number of <u>SMALL</u> LDL particles
High Risk: <u>HIGH</u> numbers of <u>LARGE</u> LDL particles	Lowest Risk: <u>LOW</u> number of <u>LARGE</u> LDL particles

Lp(a)

ASCVD Risk Enhancer

Structure & Pathogenicity

- An LDL-like particle with apolipoprotein(a) covalently bound to apoB100. It is pro-atherogenic (LDL-like) and pro-thrombotic/antifibrinolytic

Independent Risk Factor

- Genetically determined levels remain stable throughout life. Risk increases in a linear, concentration-dependent manner.

Clinical Threshold

> 30 mg/dL (or > 75 nmol/L)- Intermediate Risk
> 50 mg/dL (or > 125 nmol/L)- High Risk

Universal screening increasingly advocated (measure once in adulthood)

Therapeutic Challenge

Statins do not lower Lp(a) and may paradoxically increase levels by 10-20%.

Apolipoprotein B (ApoB)

Mechanism of Action

ApoB is the primary structural protein found on all potentially atherogenic lipoprotein particles.

Key Principle: 1:1 Ratio

There is exactly one molecule of ApoB per atherogenic particle (VLDL, IDL, LDL, Lp(a)). Therefore, measuring ApoB provides a direct count of total atherogenic particle number.

Therapeutic Goals

Optimal/Low-Risk: < 90 mg/dL

Very High-Risk: < 80 mg/dL

Extremely High-Risk: < 70 mg/dL

Superior Risk Prediction

- Outperforms LDL-C as a predictor of cardiovascular events, especially in metabolic syndrome.
- Direct measure of particle burden vs. cholesterol content (LDL-C).
- Unaffected by fasting status, making it convenient for patients.

Clinical Pearl: Insulin Resistance

ApoB is frequently elevated in patients with insulin resistance, diabetes, and hypertriglyceridemia, even when LDL-C appears normal (discordance).

Apolipoprotein A-1 (ApoA-1)

Protective Function

Major HDL Protein

- Structural backbone of High-Density Lipoprotein particles.

Reverse Cholesterol Transport

- Facilitates the removal of excess cholesterol from peripheral tissues back to the liver for excretion.

Superior Predictor

- Higher levels correlate with lower CVD incidence more strongly than HDL-C alone.

Optimal Target Levels

Men

> 120 mg/dL

Women

> 140 mg/dL

Clinical Significance of Low Levels

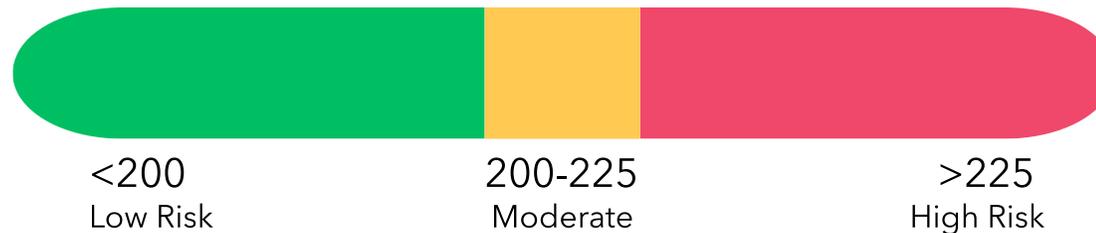
Low ApoA-1 is frequently associated with:

- Metabolic Syndrome
- Insulin Resistance
- Type 2 Diabetes
- Increased CVD Risk

PLAC (LP-PLA2 Activity)

- Lipoprotein-associated phospholipase A2 (Lp-PLA2) is a vascular-specific inflammatory enzyme produced by macrophages and foam cells within atherosclerotic plaque.
 - reflects **active inflammation within the vessel wall** and the likelihood of plaque rupture

PLAC Activity Ranges



Inflammation & Vascular Integrity

Biomarker & role	Reference ranges	Clinical significance	Key interventions
<p>hs-CRP (High-sensitivity C-reactive Protein)</p> <p>Liver-derived acute phase reactant; primary marker of systemic inflammation</p>	<p>Optimal: <1.0 mg/L</p>	<p>Strong univariate predictor of CV events. Reflects inflammatory burden from visceral fat, infection, or autoimmune activity.</p>	<ul style="list-style-type: none"> • Anti-inflammatory diet • Glycemic control • Weight loss/Visceral fat reduction • Smoking cessation • Treat gum disease/infections • Stress management
<p>Homocysteine</p> <p>Amino acid intermediate in methionine metabolism; marker of methylation status and endothelial health</p>	<p>Optimal: 5.0-7.2 umol/L</p>	<p>Elevations impair endothelial nitric oxide, promote oxidative stress, and increase thrombotic risk. Often signals B-vitamin insufficiency (folate, B12, B6), impaired methylation, insulin resistance, or renal dysfunction.</p>	<ul style="list-style-type: none"> • Anti-inflammatory lifestyle • correct B-vitamin insufficiency (folate, B12, B6) • Manage IR and smoking

Glycemia and Insulin: Early Detection of Risk

Parameter	Reference ranges	Clinical significance & context
Fasting Blood Glucose (FBG)	Optimal: 72–85 mg/dL	Early Warning Sign: Risk for CVD and T2D rises significantly even in the "high-normal" range (91–99 mg/dL). Standard reference ranges (<100 mg/dL) often miss early metabolic dysfunction.
Fasting Insulin	Optimal: 1–5 mIU/L	Primary Driver: Hyperinsulinemia is a major driver of sdLDL formation, triglyceride elevation, and systemic inflammation. Elevated insulin indicates resistance often years before glucose rises.

Evidence-Based Interventions

 Low-glycemic / Mediterranean dietary pattern

 Combined resistance + aerobic training

 Weight optimization (visceral fat reduction)

 Sleep hygiene & stress management (cortisol control)

Adipokines

Leptin

Primary Role
"Satiety hormone" regulating energy balance and immune response. Resistance leads to hyperleptinemia.

Cardiometabolic Risk
High levels independently associated with atherosclerosis, MI, and stroke.

Highest Risk Combination

Elevated Leptin + Elevated hs-CRP = Synergistic CVD Risk

Adiponectin

Primary Role
Insulin-sensitizing hormone with vascular protective effects. Enhances fatty acid oxidation.

Cardiometabolic Risk
Levels are inversely related to visceral fat and insulin resistance. Low levels predict metabolic syndrome.

Clinical Pearl

Evaluate alongside Fasting Insulin, TG/HDL-C ratio, and LP-IR to phenotype insulin resistance severity.

1,5-Anhydroglucitol (1,5 AG)

Mechanism: Structural analog of glucose. Competes for reabsorption when blood glucose spikes >180 mg/dL.

Clinical Utility: Captures postprandial hyperglycemic excursions often missed by A1c (average) or FBG (fasting).

Low Levels: Indicate recent significant glucose spikes (within 1-2 weeks).

Comprehensive Biomarker Reference Table

BIOMARKER	WHAT IT MEASURES	OPTIMAL RANGE	ELEVATED RISK	CLINICAL PATTERN
🔗 LIPOPROTEIN PARTICLES				
LDL-P	Total LDL particle number	< 1000 nmol/L	> 1000 nmol/L	<i>Discordance: Low LDL-C but high LDL-P = High Risk</i>
ApoB	Total atherogenic particles	< 80 mg/dL	> 90-100 mg/dL	<i>Strongest predictor; tracks with LDL-P</i>
sdLDL	Small, dense LDL concentration	< 20 mg/dL	> 20 mg/dL	<i>Pattern B; associated with MetS & high TG</i>
Lp(a)	Genetic, pro-thrombotic LDL	< 30 mg/dL	> 30-50 mg/dL	<i>Independent genetic risk; family history</i>
🔥 INFLAMMATION & OXIDATION				
oxLDL	Oxidatively modified ApoB	< 60 U/L	> 60 U/L	<i>Active atherogenesis & oxidative stress</i>
PLAC	Lp-PLA2 enzyme activity	< 200 nmol/min/mL	> 225 nmol/min/mL	<i>Vascular inflammation; unstable plaque</i>
hs-CRP	Systemic inflammation	< 1.0 mg/L	> 2.0-3.0 mg/L	<i>General inflammation; CVD risk multiplier</i>
Homocysteine	Methylation/Vascular stress	5.0 - 7.2 µmol/L	> 10-15 µmol/L	<i>Endothelial dysfunction; B-vitamin need</i>
📊 METABOLIC & GLYCEMIC				
Fasting Insulin	Insulin resistance status	2 - 5 mIU/L	> 10 mIU/L	<i>Early IR detection before glucose rises</i>
LP-IR Score	Lipoprotein IR composite	< 45 (0-100 scale)	> 50	<i>Multifactorial insulin resistance metric</i>
1,5-AG	Postprandial glucose control	> 14 µg/mL	< 10 µg/mL	<i>Detects glucose spikes missed by A1c</i>
FBG	Fasting glucose	72 - 85 mg/dL	> 90-100 mg/dL	<i>"High-normal" indicates rising risk</i>
🏠 ADIPOKINES				
Leptin	Adipose signaling/Energy	Variable by BMI/Gender	High relative to BMI	<i>Leptin resistance; inflammatory obesity</i>
Adiponectin	Insulin sensitizing hormone	High is better	Low levels	<i>Low levels correlate with IR & MetS</i>

Putting It Altogether



Robert

46-Year-Old Male

Occupation

IT project manager

Chief Concern

“I want to avoid what happened to my father.”

BMI

29.1kg/m²

Blood pressure

132/84 mmHg

Medical History

Past Medical History

Prediabetes noted 1 year ago (HbA1c previously 5.7%)

Family History

Father: MI at age 54

Paternal grandfather: died of stroke at age 67

Mother: hypertension

Current Medications

None reported

Lifestyle

Dietary Pattern

High refined carbohydrates, low fiber intake. Frequent dining out.

Physical Activity

Sedentary job. Walks/plays basketball occasionally on weekends.

Alcohol Intake/Smoking Status

Moderate (5-7 drinks/week)/Non-Smoker

Sleep

6–6.5 hours/night; reports snoring; wakes tired

Baseline Labs

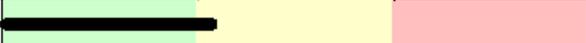
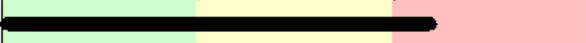
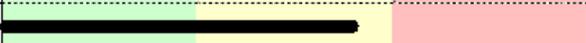
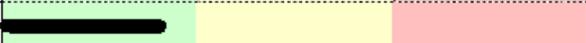
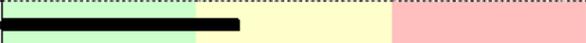
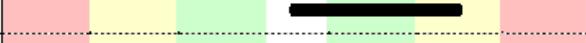
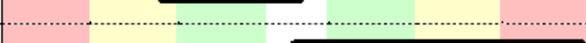
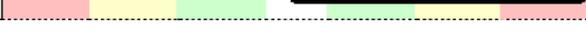
- HbA1c: 5.8%
- Glucose: 96 mg/dL
- ALT: 34 U/L
- AST: 27 U/L
- GGT: 42 U/L
- Creatinine: 0.9 mg/dL
- eGFR: >90
- TSH: 2.1
- Vitamin D: 27 ng/mL

LIPIDS/RATIOS	RESULT / UNIT		REFERENCE INTERVAL	CARDIOVASCULAR RISK		
				LOW RISK	MODERATE RISK	HIGH RISK
Total Cholesterol	198	mg/dL	< 200			NORMAL
Triglycerides	168	mg/dL	< 150			HIGH
HDL Cholesterol	47	mg/dL	> 60			LOW
LDL Cholesterol (calculated)	104	mg/dL	< 100			NEAR OPTIMAL
Non-HDL Cholesterol (calculated)	151	mg/dL	< 130			HIGH

Total Chol/HDL Ratio: 4.2 (rr < 3.5 optimal) **MODERATE RISK**

Triglycerides/HDL Ratio: 3.6 (rr < 2 optimal) **INCREASED RISK**

Advanced Cardiometabolic Biomarkers

OxLDL Cholesterol	62	U/L	< 60		MILDLY ELEVATED
sdLDL Cholesterol (calculated)	42	mg/dL	< 35		ELEVATED
Lp(a)	18	mg/dL	< 30		NO RISK
RISK FACTORS/INFLAMMATORY MARKERS					
PLAC (LP-PLA ₂ Activity)	205	U/L	< 151		MODERATE RISK
Homocysteine	8.2	μmol/L	< 11.0		HIGH NORMAL
hsCRP	1.4	mg/L	< 1.0		MILDLY ELEVATED
APOLIPOPROTEINS				PERCENTILE	
				2.5 th 16 th 50 th 84 th 97.5 th	
Apolipoprotein A-1	146	mg/dL	115 - 220		NORMAL
Apolipoprotein B	108	mg/dL	50 - 130		ELEVATED
METABOLIC RISK MARKERS					
Insulin	13	μIU/mL	2.8 - 18.0		NOT OPTIMAL
Glucose	96	mg/dL	70.0 - 100		NOT OPTIMAL
1,5-Anhydroglucitol (1,5 AG)	8.7	ug/mL	6.8 - 29		MODERATE RISK
*Leptin	28.5	ng/mL	4.0 - 39		
*Adiponectin	8.5	μg/mL	4.0 - 20		
Leptin : Adiponectin ratio	3.35		0.20 - 3.40		

Clinical Interpretation

Insulin resistance–driven atherogenic dyslipidemia

- Key signals:
 - Elevated LDL particle number
 - Small dense LDL
 - Elevated ApoB
 - High TG / HDL ratio
 - Elevated fasting insulin

Secondary Contributors

- Visceral adiposity
- Mild systemic inflammation
- Early oxidative modification of LDL

Intervention Plan: Lifestyle Core

Nutrition



Mediterranean / Low-Glycemic

- Emphasize whole plants, lean proteins, and healthy fats.

Soluble Fiber Focus

- Goal: 30–45 g/day (legumes, oats, chia) to bind bile acids and lower LDL-P.

Omega-3 Rich Foods

- Fatty fish (salmon, sardines) 2-3x/week for TG lowering.

Specific Reductions

- Minimize refined carbs, added sugars, and alcohol

Sleep



Duration & Quality

- Aim for 7–9 hours nightly. Prioritize consistency in sleep/wake times.

Screening

- Assess for Obstructive Sleep Apnea (OSA) if snoring or fatigue present (common in metabolic syndrome).

Environment

- Cool, dark room; limit blue light exposure 60-90 min before bed.

Physical Activity



Aerobic Base

- 150–300 min/week moderate intensity (Zone 2) to improve mitochondrial function.

Resistance Training

- 2–3 days/week to improve insulin sensitivity and muscle glucose uptake.

"Exercise Snacking"

- Post-meal walks (10-15 min) to blunt postprandial glucose/insulin excursions.

Stress Resilience



Mindfulness Practice

- Daily 10-15 min meditation or breathwork to lower sympathetic tone.

HRV Biofeedback

- Consider heart rate variability training to improve vagal tone.

Nature Exposure

- Regular time outdoors ("forest bathing") associated with reduced cortisol.

Weight Optimization



Realistic Goal Setting

- Aim for 5–10% weight loss over 3–6 months to significantly improve metabolic markers.

Waist Circumference

- Track waist-to-height ratio (optimal < 0.5) as a better metric than BMI.

Meal Timing

- Consider time-restricted eating (e.g., 12-hour window) to support metabolic health.

Targeted Supplementation

Omega-3 Fatty Acids (EPA+DHA)

- 2–4 g/day for significant triglyceride lowering and shift from sdLDL to larger buoyant particles.

Curcumin (Bioavailable Form)

- 500–1000 mg/day for potent anti-inflammatory effects and reduction of lipid peroxidation.

Berberine HCL

- 500 mg 2–3x/day with meals; activates AMPK similarly to metformin, improving insulin sensitivity and lipid profile.

Magnesium (Glycinate/Malate)

- 300–400 mg/day; critical for insulin receptor tyrosine kinase activity.

Alpha-Lipoic Acid (ALA)

- 300–600 mg/day for antioxidant defense and improved glucose uptake.

Chromium / Vanadium

- Trace minerals to support insulin signaling pathways.

Coenzyme Q10 (Ubiquinol)

- 100–200 mg/day for antioxidant support and mitochondrial function

Key Takeaways

- **Traditional Panels Miss Critical Risk**
 - Standard LDL-C fails to predict ~50% of events. Relying solely on basic lipid panels leaves significant residual risk undetected, especially in metabolic syndrome.
- **Particle Number & Quality Matter**
 - **ApoB / LDL-P** (burden), **sdLDL** (quality), **oxLDL**(oxidative stress), and **PLAC** (instability) provide a superior, granular view of atherogenic risk.
- **Treat the Phenotype, Not Just the Number**
 - Identify the root cause: **Insulin Resistance** (high TG/HDL, LP-IR) vs. **Inflammation** (hs-CRP) vs. **Genetic**(Lp(a)). Tailor lifestyle and meds accordingly.
- **Track Progress with Actionable Targets**
 - Set clear intervals (8-12 weeks) and specific goals. Use biomarkers to validate the effectiveness of interventions.

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