Lipoprotein(a) Management Simplified: The National Lipid Association Lipid Simplified Series

What is Lipoprotein(a)?

Lipoprotein(a) [Lp(a)] is a unique lipoprotein. High Lp(a) levels are gentically determined and is recognized as an independent and causal risk factor for atherosclerotic cardiovascular disease (ASCVD).

Measure Lipoprotein(a)

Universal Lp(a) Screening	Additional Lipid Screening Intervals
Lp(a) levels should be measured at least once in every adult	Women after menopause
	Women after pregnancy
	Individuals who develop chronic kidney disease
	Individuals who develop hypothyroidism
	Individuals taking certain medications (statins slightly raise Lp(a) and PCSK9 inhibitors lower Lp(a))

Measure Lipoprotein(a)

Risk Stratification based on Lp(a) Level

Lp(a) Level	Impact on ASCVD RIsk
<75 nmol/L (<30 mg/dL)	Low
≥75 to <125 nmol/L (≥30 to <50 mg/dL)	Intermediate
≥ 125 nmol/L* (> 50 mg/dL)	High

Unit of Measurement

The NLA recommends using the nmol/L unit of measurement

*The nmol/L value of >105 nmol/L (>50 mg/dL) in the 2025 statement from ESC/EAS reflects the method for unit conversion employed.

Current Approaches to Lipid Lowering & Their Effect on Lp(a)

Approach	Expected Effect on Lp(a) Levels	Notes
Lipoprotein apheresis	60-75% reduction (acute); 25-40% (time-averaged)[23]	Approved by the FDA for HeFH patients with Lp(a) ≥60 mg/dL (≥130 nmol/L) and either documented coronary artery disease or documented peripheral artery disease[11]
Statins	11% increase on average, with some individuals exhibiting a more than 20% increase[13]	Mechanism of statin effect of Lp(a) is unknown; increase in Lp(a) does not outweigh benefit of statins
PCSK9 Inhibitors	20-30% reduction (mAb) 18-26% reduction (inclisiran) (siRNA); lerodalcibep (PCSK9-blocking adnectin); enlicitide (PCSK9-blocking peptide)[2]	Mechanism of effect of PCSK9 inhibition on Lp(a) lowering is controversial[24]; some evidence that patients with high Lp(a) derive additional benefit from PCSK9 inhibition
Lifestyle Modification	No consistent effect[25]	Includes diet, exercise, weight loss; some evidence that a diet high in saturated fat modestly lowers Lp(a)[25]
Aspirin	No effect[14]	Some evidence from clinical studies for a cardiovascular benefit for high-Lp(a) in elderly patients in primary prevention; can be considered using a patient-centered approach in individuals with elevated Lp(a)
Ezetimibe	No effect[14]	
Bempedoic acid	No effect[14]	
Niacin	20-30% reduction[26]	Use not recommended due to no overall CV benefit (including in patients with high Lp(a)) and adverse effects of treatment

Frequently Asked Questions

How is Lp(a) distinct from Low-Density Lipoprotein Cholesterol?

Lp(a) is a unique lipoprotein class. It is similar to low-density lipoprotein (LDL) but additionally contains the unique apolipoprotein(a) (apo(a)) protein. Apo(a) likely accounts for the recognized causal role and independent contributions to ASCVD of elevated Lp(a). Lp(a) preferentially carries highly proinflammatory oxidized phospholipids, and elevated Lp(a) increases ASCVD risk even when other risk factors including LDL-C are well-controlled.

Lp(a) levels are mostly genetically determined and hence resistant to lipid-lowering therapy as well as dietary interventions. Because of this, Lp(a) levels tend to be stable over a person's lifetime such that one Lp(a) measurement is usually sufficient to ascertain risk.

How are Plasma Lp(a) Levels Determined?

The metabolism of Lp(a) is distinct from that of other apoB-containing lipoproteins. Lp(a) levels are primarily determined by the LPA gene that encodes apo(a). Smaller gene (and hence apo(a) protein) sizes generally result in higher Lp(a) levels. The rate of Lp(a) biosynthesis is more important to determining Lp(a) levels than clearance from plasma by the liver.

What Plasma Level of Lp(a) is Correlated with Increased Risk for ASCVD? Does this Vary in Different Populations?

Most guidelines define plasma Lp(a) concentrations ≥125 nmol/L (>50 mg/dL) as conferring increased risk for ASCVD. While different populations may show differences in median Lp(a) levels, the levels conferring increased risk are the same.

What Approaches are Currently Available for Reducing ASCVD Risk in Individuals with Elevated Lp(a)?

Guidelines and scientific publications universally recommend reducing overall ASCVD risk in high-Lp(a), primary prevention patients through optimal control of modifiable ASCVD risk factors. The presence of elevated Lp(a) should be an indication for more intensified LDL-C lowering and management of other risk factors.

How Can I Find a Lipid Specialist?

The 2026 ACC/AHA Multisociety Guideline on the Management of Dyslipidemia indicate that a Lipid Specialist may be required to manage patients with Lp(a) levels ≥200 nmol/L (≥75 mg/dL). A Lipid Specialist is a clinician with advanced expertise in lipid management, certified by the American Board of Clinical Lipidology. Find a Lipid Specialist at learnyourlipids.com/find-a-clinician/.

Scan QR Code to Find a Lipid Specialist