

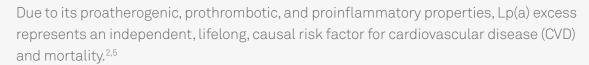
Lipoprotein (a) (Lp(a))



**Lipoprotein (a)** identifies proatherogenic lipoprotein and is a marker for cardiovascular risk stratification

## **Test description**

Lipoprotein (a) (Lp(a)) is an LDL-like particle with an apolipoprotein(a) bound to the apolipoprotein B subunit. Lp(a) levels are primarily genetically predetermined, and is elevated in 20%-30% of the population. <sup>2-4</sup>





## Clinical significance

- → Atherosclerotic cardiovascular disease (ASCVD) risk is positively correlated with Lp(a) levels<sup>6</sup>
- → Elevated Lp(a) is associated with a 5x risk for coronary artery stenosis<sup>7</sup>
- → Lp(a) excess is associated with a more than 3x increase in myocardial infarction and a 1.5x increase in cardiovascular mortality<sup>8</sup>
- → Individuals with elevated Lp(a) had 1.6x greater risk for ischemic stroke<sup>9</sup>

# Individuals suitable for testing

- → Individuals with 1 or more risk factors for the development of CVD
- → Individuals at borderline risk (5%-<7.5% 10-year ASCVD risk) or intermediate risk (7.5 %-<20% 10-year ASCVD risk)<sup>10</sup>
- → Personal or family history of premature ASCVD, familial hypercholesterolemia, or Lp(a) excess<sup>11</sup>
- → Individuals with a history of aortic stenosis, ischemic stroke, acute coronary syndrome, or progressive ASCVD despite optimal medical therapy<sup>5,11</sup>



# Lp(a) relative risk (nmol/L)

< 75 Optimal

75-125 Moderate

> 125 High

### **Treatment considerations\***

### A global risk reduction strategy should be considered when Lp(a) is elevated.

- → Optimize control of modifiable cardiovascular risk factors, such as lipids, blood pressure, and insulin resistance, based on the current guideline recommendations
- → Although diet and lifestyle do not lower Lp(a) levels, they can help to mitigate the cardiovascular risks attributable to it12
- → Lipid apheresis is used to treat familial hypercholesterolemia and can be considered for those with Lp(a) excess13
- → While there are no current FDA-approved pharmaceuticals for lowering Lp(a), PCSK9 inhibitors and niacin have been shown to reduce Lp(a) levels<sup>14,15</sup>
- An inverse relationship has been shown for Lp(a) levels and estrogen<sup>16</sup>

\*The treatment considerations are provided for educational purposes only and are not intended as medical advice. A healthcare provider's test selection, interpretation, diagnosis, and patient management decisions should be based on their education, clinical expertise, and assessment of the patient. Specific treatment plans should be provided and reviewed by the treating provider.

Test Name	Quest Test Code	CHL Test Code	CPT Codeª	Preferred Specimen(s)
Lipoprotein (a)	91729	91729	83695	Serum

\*The CPT codes provided are based on AMA guidelines and are for informational purposes only. CPT coding is the sole responsibility of the billing party. Please direct any questions regarding coding to the payer being billed.



Assess Lp(a) to identify risk for cardiovascular disease in your patients. For more information, contact your Cardiometabolic account executive.

#### References

References

1. Reyes-Soffer G, Ginsberg HN, Berglund L, et al. Lipoprotein(a): a genetically determined, causal, and prevalent risk factor for atherosclerotic cardiovascular disease: a scientific statement from the American Heart Association. ATVB. 2022;42(1). doi:10.1161/ATV.00000000000001472. Tsimikas S, Fazio S, Ferdinand KC, et al. NHLBI Working Group recommendations to reduce lipoprotein(a)-mediated risk of cardiovascular disease and aortic stenosis. JACC. 2018;71(2):177-192. doi:10.11016/j.jacc.2017.11.0143. Schmidt K, Noureen A, Kronenberg F, Utermann G. Structure, function, and genetics of lipoprotein (a). J Lipid Res. 2016;57(8):1339-1359. doi:10.1194/jlr.R0073144. Mack S, Coassin S, Rueedi R, et al. A genome-wide association meta-analysis on lipoprotein(a) concentrations adjusted for apolipoprotein(a) isoforms. J Lipid Res. 2017;58(9):1834-1844. doi:10.1194/jlr.M0762325. Wilson DP, Jacobson TA, Jones PH, et al. Use of Lipoprotein(a) in clinical practice: A biomarker whose time has come. A scientific statement from the National Lipid Association. J Clin Lipidol. 2019;13(3):374-392. doi:10.1016/j.jacl.2019.04.010.6. Finneran P, Pampana A, Khetarpal SA, et al. Lipoprotein(a) and coronary artery disease risk without a family history of heart disease. JAHA. 2021;10(5):e017470. doi:10.1161/JAHA.12.0107147077. Kamstrup PR, Tybjærg-Hansen A, Steffensen R, Nordestgaard BG. Genetic evidence that lipoprotein(a) associates with atherosclerotic stenosis rather than venous thrombosis. ATVB. 2012;32(7):1732-1741. doi:10.1161/ATVBAHA.112.248765.8. Kamstrup PR, Tybjærg-Hansen A, Steffensen R, Nordestgaard BG. Genetically elevated lipoprotein(a) and increased risk of myocardial infarction. JAMA. 2009;30(12):2331-2339. doi:10.1016/j.jacc.2019.208.019. Langsted A, Nordestgaard BG. Genetically elevated lipoprotein(a) and increased risk of myocardial infarction. JAMA. 2009;30(12):2331-2339. doi:10.1016/j.jacc.2019.03.524 10. Armstrup PR. Elevated Inpoprotein(a) and risk of inschemic struck. JACC. 2019;74(1):54-66. doi:10.1

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