



Testing for Lp(a) once in a person's lifetime is endorsed by the ESC/EAS, national guidelines, and the 2022 EAS consensus statement on Lp(a)<sup>1,13,15</sup>



Elevated Lp(a) is an important contributor to an individual's cardiovascular risk

Knowledge of elevated Lp(a) can...

- Contribute to clinical decision-making<sup>1,9</sup>
- Identify patients at high risk and improve cardiovascular risk management<sup>1</sup>
- Underline the need to intensify lipid lowering and control of all modifiable risk factors<sup>1,16,17</sup>
- Motivate patients to have close family members screened for Lp(a) for early risk detection<sup>1,9</sup>

Keypoints of the EAS Lp(a) consensus statement:<sup>1</sup>

- Lp(a) should be assessed in every adult at least once in their lifetime
- For Lp(a) values above 50 mg/dL, one must assume that the cardiovascular risk is increased by Lp(a). The higher the Lp(a) concentration, the higher the risk
- This additional risk is independent of other risk factors such as LDL-C

## 6 reasons to measure Lp(a)

- 1** Elevated Lp(a) is an independent, inherited, and causal risk factor for cardiovascular disease and aortic valve stenosis<sup>2,3,13</sup>
- 2** The 2019 ESC/EAS Guidelines and 2022 EAS consensus statement on Lp(a) recommend that Lp(a) measurement should be considered at least once in a lifetime of every adult<sup>1,13</sup>
- 3** Identification of elevated Lp(a) may reclassify a patient in a higher cardiovascular risk category<sup>1,14</sup>
- 4** By cascade screening, additional family members with elevated Lp(a) can be identified earlier<sup>1,9</sup>
- 5** Elevated Lp(a) is a reason for more aggressive treatment of other modifiable cardiovascular risk factors, including LDL-C and blood pressure, to reduce overall cardiovascular risk<sup>1,16,17</sup>
- 6** Lp(a) can be measured with a simple blood test<sup>9,13</sup>

**References:** 1. Kronenberg F, et al. *Eur Heart J*. 2022;43(39):3925–3946; 2. Tsimikas S, et al. *J Am Coll Cardiol*. 2018;71(2):177–192; 3. Kronenberg F, Utermann G. *J Intern Med*. 2013;273(1):6–30; 4. Schmidt K, et al. *J Lipid Res*. 2016;57:1339–1359; 5. Nordestgaard BG, Langsted A. *J Lipid Res*. 2016;57:1953–1975; 6. Kronenberg F, et al. *Curr Opin Lipidol*. 2022; 33(6):342–352; 7. Patel AP, et al. *Arterioscler Thromb Vasc Biol*. 2021;41:465–474; 8. Toth PP. *J Am Coll Cardiol*. 2020;75(21):2694–2697; 9. Reyes-Soffer G, et al. *Arterioscler Thromb Vasc Biol*. 2022;42(1):e48–e60; 10. Nordestgaard BG, et al. *Eur Heart J*. 2010;31:2844–2853; 11. Tsimikas S. *J Am Coll Cardiol*. 2017;69(6):692–711; 12. Willeit P, et al. *Lancet*. 2018;392(10155):1311–1320; 13. Mach F, et al. *Eur Heart J*. 2020;41(1):111–188; 14. Nurmohamed NS, et al. *Eur J Prev Cardiol*. 2022;29:769–776; 15. Pearson GJ, et al. *Can J Cardiol*. 2021;37(8):1129–1150; 16. Cegla J, et al. *Atherosclerosis*. 2019;291:62–70; 17. Perrot N, et al. *Atherosclerosis*. 2017;256:47–52.

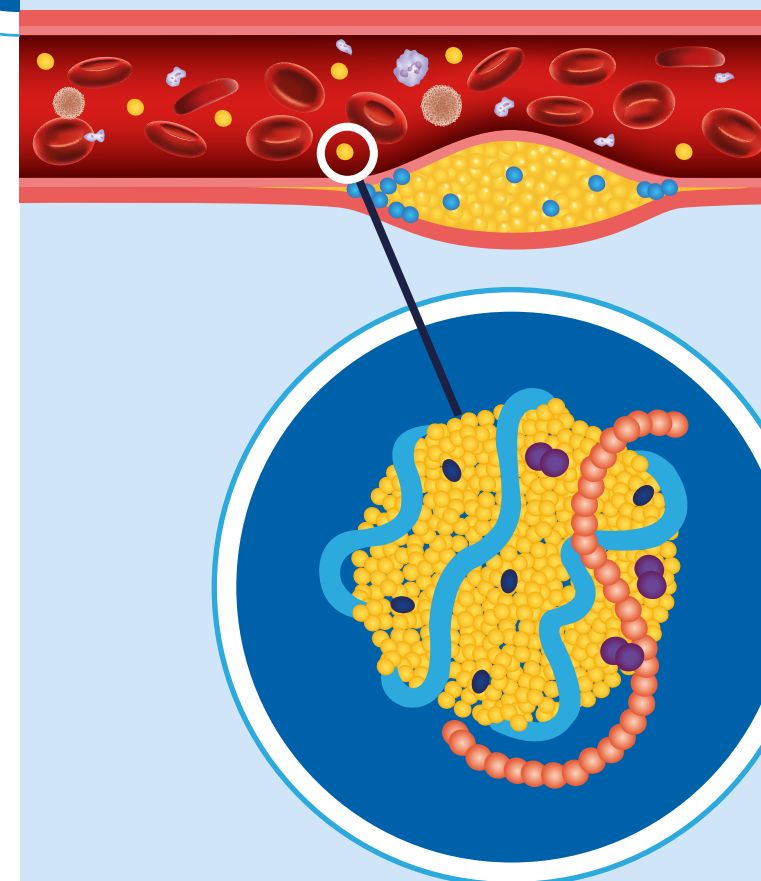
**Abbreviations:** apo(a), apolipoprotein(a); apoB, apolipoprotein B; EAS, European Atherosclerosis Society; ESC, European Society of Cardiology; LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a); OxPL, oxidized phospholipids.

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Novartis Sverige AB, Box 1218, 164 28 Kista.  
Telefon 08-732 32 00, [www.novartis.se](http://www.novartis.se)

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## Testing for Lipoprotein(a): Why, How, and in Whom?

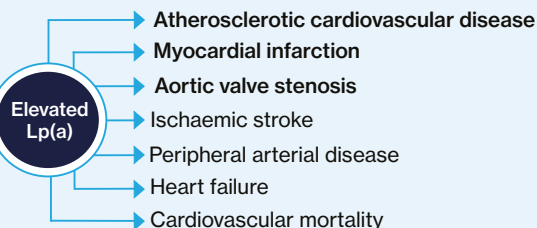
Elevated lipoprotein(a) is an inherited, independent, and causal risk factor for atherosclerotic cardiovascular disease





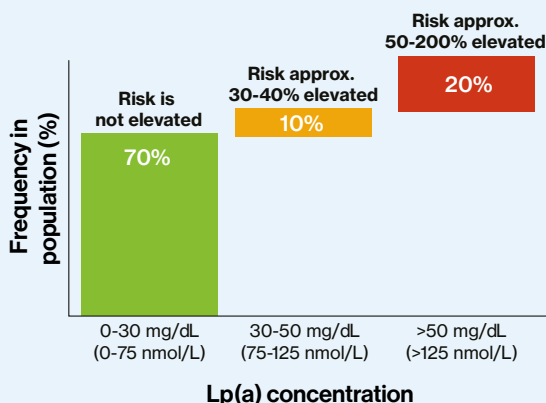
Elevated Lp(a) is an independent, inherited, and causal risk factor for atherosclerotic cardiovascular disease and aortic valve stenosis<sup>1-5</sup>

Genetics



Measurement of Lp(a) can help to identify hidden cardiovascular risk

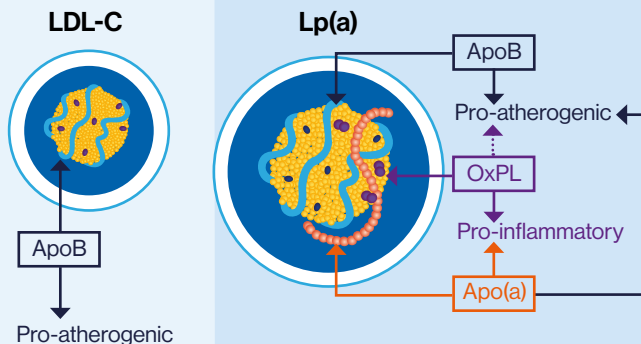
Risk for myocardial infarction as a function of Lp(a)<sup>6</sup>



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Risk of cardiovascular events increases continuously with increasing Lp(a) concentration<sup>6,7</sup>

## Structure of Lp(a)



- Lp(a) is an apoB-containing particle in which apo(a) is covalently bound to the apoB<sup>3,8</sup>
- In addition to the pro-atherogenic properties of apoB, Lp(a) possesses pro-inflammatory properties due to apo(a) and oxidized phospholipids<sup>2,9</sup>

Elevated Lp(a) levels are genetically determined and remain fairly constant throughout a person's life



Approximately 20% of the population have plasma Lp(a) levels that are considered elevated (defined as >50 mg/dL or >125 nmol/L)<sup>10</sup>



Lp(a) levels are >90% genetically determined by the *LPA* gene, which controls the production of Lp(a)<sup>1,3,4,11</sup>



Circulating Lp(a) levels are only minimally affected by age, sex, diet, and physical activity<sup>1,3</sup>

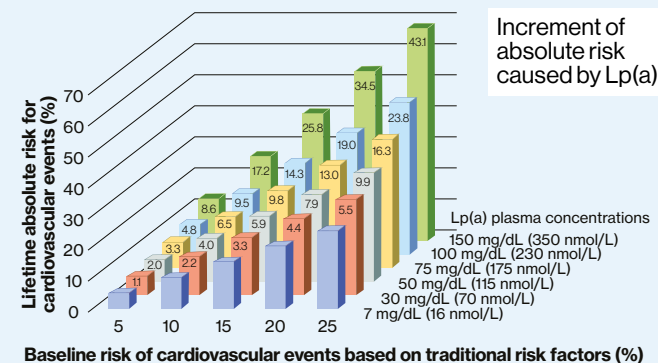
Easy access to Lp(a) testing is needed

The EAS consensus statement recommends:

- Lp(a) should be measured at least once in adults to identify those with high cardiovascular risk<sup>1,11,12</sup>
- Elevated Lp(a) can contribute to cardiovascular risk even in patients treated with lipid-lowering therapies<sup>1,11,13</sup>
- Lp(a) testing is also recommended in youth with a history of ischaemic stroke or a family history of premature ASCVD or high Lp(a)<sup>1</sup>
- Cascade (family) testing for high Lp(a) is recommended in the settings of familial hypercholesterolaemia or family or personal history of (very) high Lp(a)<sup>1</sup>
- Several laboratory assays are widely available internationally for measuring Lp(a)<sup>13</sup>
- Important note: Lp(a) concentrations are reported in 2 different units – either molar in nmol/L or mass units in mg/dL<sup>13</sup>



Elevated Lp(a) adds to the overall ASCVD risk and triggers a risk reclassification<sup>1,14</sup>



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