Indonesian Journal of Cardiology
Indonesian J Cardiol 2026:47
pISSN; 2830-3105 / eISSN: 2964-7304
doi: 10.30701/ijc.1846

Original Article

Prevalence and Lipid Profile Associations of Elevated Lipoprotein(a) Among Indonesian Adults: A Cross-Sectional Study

Jason Wirandy Haryanto[1], Kevin Jonatan Sandi[1], Surya Sinaga Immanuel[1], Angely Christie Rumayar[2], Leonardo Paskah Suciadi[3]

Abstract

Background: Lipoprotein(a) [Lp(a)] is a genetically determined lipoprotein and an independent risk factor for Atherosclerotic Cardiovascular Disease (ASCVD). While 20–30% of adults worldwide have elevated Lp(a), data from Indonesia remain scarce. Given reported differences by sex and metabolic profile, this cross-sectional study aimed to determine the prevalence of elevated Lp(a) and its associations with lipid parameters and Hemoglobin A1c (HbA1c) among adults undergoing routine health screening at a tertiary hospital in Indonesia.

Methods: This cross-sectional study analyzed data from 904 adults who underwent routine health screening at Siloam Hospitals Kebon Jeruk, Jakarta (2021–2024). Data on Lp(a), lipid profile, and HbA1c were extracted from the medical records. The normality of data was assessed using the Shapiro–Wilk test. Group comparisons were performed using the Mann–Whitney U test, and correlations between log-transformed Lp(a) and metabolic parameters were evaluated using Spearman correlation and simple linear regression.

Results: Among 904 participants, 18.8 % had elevated Lp(a) levels (≥ 30 mg/dL). Females showed significantly higher Lp(a) concentrations than males ($p = 0.008$). Low-Density Lipoprotein Cholesterol (LDL-C) levels were slightly higher in participants with elevated Lp(a) but did not differ significantly ($p = 0.14$). On linear regression, LDL-C was positively associated with log-transformed Lp(a) ($B = 0.002$, $p < 0.001$), whereas triglycerides were inversely associated ($B = -0.001$, $p = 0.019$); no robust associations were observed for High-Density Lipoprotein Cholesterol (HDL-C) or HbA1c.

Conclusions: Nearly one in five adults undergoing routine health screening in this Indonesian cohort had elevated Lp(a) levels. Females exhibited significantly higher Lp(a) concentrations than males, and LDL-C showed a modest positive association with Lp(a). These findings reinforce the relevance of Lp(a) as an important, genetically influenced cardiovascular risk marker and support routine Lp(a) screening for improved risk stratification in clinical practice.

(Indonesian J Cardiol, 2026;47)

Keywords: Lipoprotein (a), Dyslipidemia, Atherosclerosis, Cardiovascular risk factors, Screening.

[1]Advanced Cardiac Care Clinic, Siloam Hospitals Kebon Jeruk, Jakarta, Indonesia.
[2]Medical Check-Up Department, Siloam Hospitals Kebon Jeruk, Jakarta, Indonesia.
[3]Siloam Heart Institute, Siloam Hospitals Kebon Jeruk, Jakarta, Indonesia.

Correspondence:
Jason Wirandy Haryanto,

Advanced Cardiac Care Clinic, Siloam Hospitals Kebon Jeruk, Jakarta, Indonesia.

Email: jasonwirandy@gmail.com

Introduction

Lipoprotein(a) [Lp(a)] is a form of Low-Density Lipoprotein (LDL) consisting of an LDL particle covalently bonded to apolipoprotein(a) [apo(a)].[1] This unique composition differentiates Lp(a) from other lipoproteins, providing specific functional characteristics.[2] Lp(a) is recognized as a cardiovascular risk factor that is independent and contributes to the progression of Atherosclerotic Cardiovascular Disease (ASCVD) in several populations.[3-4] Unlike traditional lipid indicators such as LDL Cholesterol (LDL-C), Lp(a) levels are predominantly determined by genetic factors and remain stable throughout an individual's life.[5-6] This genetic stability indicates that Lp(a) is not readily altered by lifestyle modifications, dietary alterations, or pharmacological treatments that influence other lipid metrics.[7-9]

Notwithstanding its therapeutic importance, Lp(a) continues to be inadequately acknowledged and underused in standard cardiovascular risk evaluations.[10] Its impact on ASCVD risk is often overshadowed by more routinely measured lipid indicators, such as LDL-C. Consequently, numerous patients with heightened Lp(a) levels remain unrecognized or insufficiently treated. Moreover, routine screening for Lp(a) is seldom performed in clinical practice, as guidelines frequently omit its inclusion in standard lipid profiles. The issue is exacerbated by the lack of population-specific data on the incidence of elevated Lp(a) levels, particularly in certain communities.[11-12] Comprehending the distribution and prevalence of Lp(a) levels in these groups is essential for enhancing Cardiovascular Disease (CVD) prevention and therapy techniques. Nevertheless, information regarding the epidemiology of Lp(a) in local communities is limited, especially in our town.

Therefore, this cross-sectional study aimed to determine the prevalence of elevated Lp(a) and its associations with lipid parameters (LDL-C, High-Density Lipoprotein Cholesterol [HDL-C], triglycerides) and Hemoglobin A1c (HbA1c) among adults undergoing routine health screening at Siloam Hospitals Kebon Jeruk, Jakarta.

Methods

Materials and Methods

This cross-sectional observational study evaluated the prevalence and distribution of elevated Lp(a) levels in adults undergoing routine health screening. The primary objective was to determine the prevalence of elevated Lp(a) and its associations with lipid parameters and HbA1c.

Data were obtained from the Medical Check-Up (MCU) unit of Siloam Hospitals Kebon Jeruk, Jakarta, for individuals who underwent Lp(a) testing between January 2021 and June 2024. A total of 904 participants were included. Adults aged ≥18 years who underwent routine medical check-up testing with available Lp(a) results were included. Participants with incomplete lipid profiles or HbA1c data were excluded. Because the dataset represented routine health screening, individuals with prior CVD (including ASCVD, coronary stent placement, or cardiac surgery) were not excluded; they were included and analyzed as part of the overall study population. This study received ethical approval from the Faculty of Medicine, Pelita Harapan University (No. 207/K-LKJ/ETIK/V/2025).

Data collection included measurements of Lp(a) levels, with elevated Lp(a) defined as levels of 30 mg/dL or greater, a threshold associated with increased cardiovascular risk.13,14 The medical records were analyzed to extract data on lipid profiles, which included total cholesterol, LDL-C, HDL-C, and triglycerides, in addition to Lp(a) levels. Body Mass Index (BMI) and renal function, evaluated by serum creatinine and estimated Glomerular Filtration Rate (eGFR), were documented. HbA1c levels of participants were included to assess glycemic control, especially among individuals with diabetes risk factors. Furthermore, participants' medical histories were gathered, including the presence of Hypertension (HTN), ASCVD, and Diabetes Mellitus (DM), as well as family histories of HTN, heart disease, and stroke. Demographic variables, including age and sex, were also noted for subgroup analyses.

This observational study used pre-existing data from routine clinical tests, so informed consent was not required for participation. The study adhered to ethical principles of confidentiality and data protection, ensuring that all personal identifiers were removed from the data before analysis.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26. Continuous variables are presented as mean ± Standard Deviation (SD) or median (Interquartile Range [IQR]) depending on data distribution, while categorical variables are summarized as frequencies and percentages. The Shapiro–Wilk test was applied to assess normality. Between-group comparisons were performed using the independent-samples t-test for normally distributed data and the Mann–Whitney U test for

non-normally distributed data. Categorical variables were compared using the chi-square test.

Correlations between Lp(a) and metabolic parameters were examined using Spearman correlation analysis. Simple linear regression was then conducted with $\log_{10}$-transformed Lp(a) as the dependent variable and LDL-C, triglycerides, HDL-C, and HbA1c as independent variables. A two-tailed $p < 0.05$ was considered statistically significant.

Results

The mean age of the participants was 48 ± 13 years, with 58.5% of the participants being male (N = 529). Regarding clinical parameters, the mean systolic blood pressure was 124.5 ± 16.5 mmHg, while the mean diastolic blood pressure was 78.7 ± 10.1 mmHg. The average BMI was 25.9 ± 4.6 kg/m². Regarding comorbidities, HTN was present in 18.7% of the participants (N = 169), diabetes mellitus in 5.1% (N = 46), and established ASCVD in 7.3% (N = 66).

Further baseline data included creatinine levels of 0.9 ± 0.2 mg/dL and an average eGFR of 96.4 ± 17 ml/kg/min. The mean HbA1c was 5.7 ± 1.1%. Lipid profiles showed a mean total cholesterol of 204.2 ± 42.4 mg/dL and a mean LDL-C of 138.7 ± 40.5 mg/dL. The average Lp(a) level was 19.2 ± 28.3 mg/dL. Family history data indicated that 37.8% had a family history of HTN (N = 342), 16.3% had a family history of heart disease (N = 147), and 10% had a family history of stroke (N = 90). Additionally, 15.6% of participants were current smokers (N = 141) (Table 1).

Table 1. Baseline characteristics.

Variables	Value
Age (years)	48 ± 13
Male (%)	58.5
Systolic Blood Pressure (mmHg)	124.5 ± 16.5
Diastolic Blood Pressure (mmHg)	78.7 ± 10.1
Body Height (cm)	165.7 ± 9.0
Body Weight (kg)	71.6 ± 15.6
BMI (kg/m2)	25.9 ± 4.6
Laboratory values	
Creatinine (mg/dL)	0.9 ± 0.2
eGFR (ml/min/1.73m²)	96.4 ± 17
HbA1c (%)	5.7 ± 1.1
Total cholesterol (mg/dL)	204.2 ± 42.4
Triglycerides (mg/dL)	123.6 ± 66.6
High-density lipoprotein (mg/dL)	52.0 ± 13.3
Low-density lipoprotein (mg/dL)	138.57 ± 40.75
Lipoprotein a (mg/dL)	19.2 ± 28.3
Comorbid	
Hypertension (%)	18.7
Diabetes Mellitus (%)	5.1
Established Atherosclerosis CVD (%)	7.3
Family history	
Stroke (%)	10
Heart disease (%)	16.3
Hypertension (%)	37.8
Smoking status (%)	15.6

Abbreviation: BMI, Body Mass Index; CVD, Cardiovascular Disease; eGFR, estimated Glomerular Filtration Rate.

Of the study participants, 81.2% exhibited non-elevated Lp(a) levels (< 30 mg/dL), whereas 18.8% had elevated Lp(a) levels (≥ 30 mg/dL) (Fig 1). Sex differences in Lp(a) levels were observed, with males presenting a mean of 17.3 ± 24.7 mg/dL and females a higher mean of 22.0 ± 32.7 mg/dL. Lp(a) levels were significantly higher in females than in males (Mann–Whitney U = 88 888, p = 0.008; Fig 2).

While the mean LDL-C level in the normal Lp(a) group (Lp(a) < 30 mg/dL) was 137.4 ± 1.46 mg/dL, the elevated Lp(a) group (Lp(a) ≥ 30 mg/dL) had a mean LDL-C level of 144.4 ± 3.41 mg/dL. This suggests that individuals with elevated Lp(a) typically have higher LDL-C levels than those with normal Lp(a) (Fig 3). Median LDL-C levels did not differ significantly between participants with normal and elevated Lp(a) (Mann–Whitney U = 57 865, p = 0.14; Fig 3).

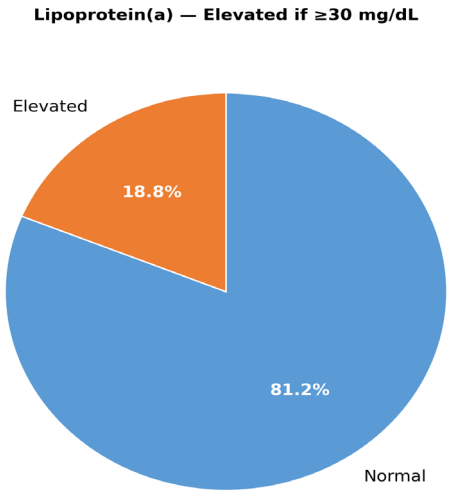

Figure 1. Percentages of elevated Lipoprotein(a).

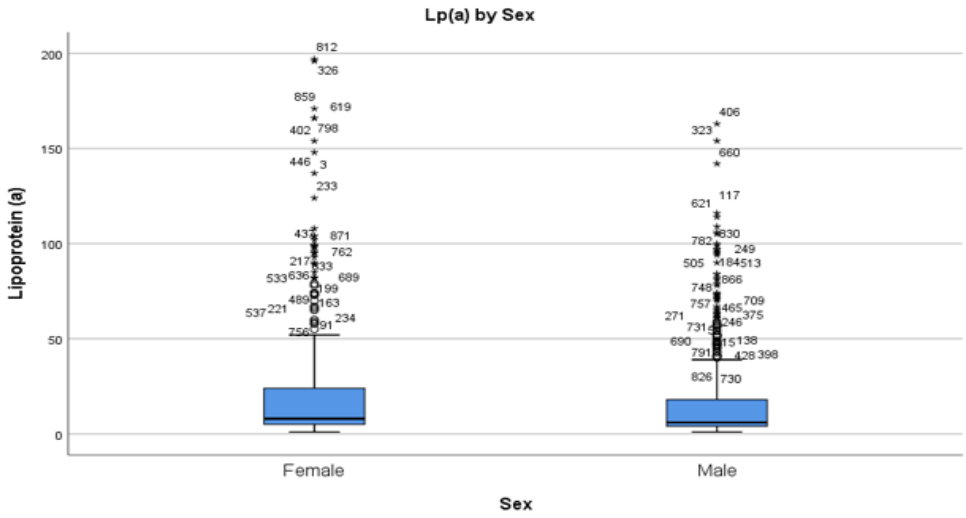

Figure 2. Sex differences in mean Lp(a).

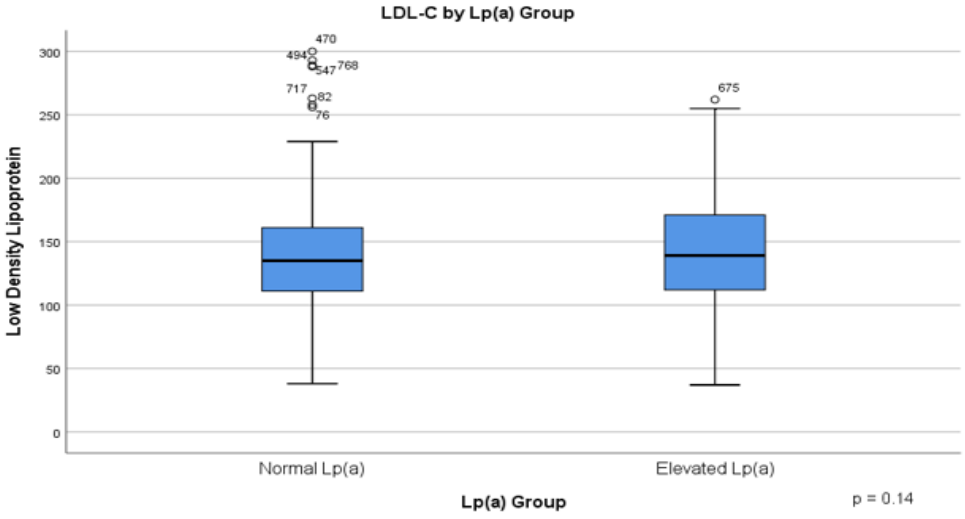

Figure 3. LDL-C differences in elevated vs normal Lp(a).

After comparing LDL-C levels between normal and elevated Lp(a) groups, we further analyzed the relationship between Lp(a) and key metabolic parameters using correlation and regression analyses. Spearman correlation analysis (Table 2) demonstrated a weak positive association between log-transformed Lp(a) and LDL-C ($\varrho = 0.134$, $p < 0.001$). A very weak positive correlation was also observed between log-transformed Lp(a) and HDL-C ($\varrho = 0.088$, $p = 0.008$). No significant correlations were found between log-transformed Lp(a) and triglycerides ($\varrho = -0.056$, $p = 0.094$) or HbA1c ($\varrho = 0.017$, $p = 0.606$).

The linear regression model (Table 3) similarly identified LDL-C as a modest but statistically significant predictor of log-transformed Lp(a) ($B = 0.002$, $p < 0.001$), while triglycerides showed a weak inverse association ($B = -0.001$, $p = 0.019$). HDL-C showed no significant association. HbA1c reached borderline significance in the regression model ($B = 0.027$, $p = 0.043$) but was not significantly correlated with Lp(a) ($\varrho = 0.017$, $p = 0.61$) and was not robust to sensitivity analysis, indicating no meaningful independent association.

Table 2. Spearman correlation between log-transformed Lp(a) and metabolic parameters.

Variable	Spearman's rho (ϱ)	p-value
LDL-C (mg/dL)	0.134	<0.001
Triglycerides (mg/dL)	-0.056	0.094
HDL-C (mg/dL)	0.088	0.008
HbA1c (%)	0.017	0.606

Spearman's rho was used due to non-normal distribution of Lp(a). logLP_A = $\log_{10}$[Lp(a)+1].

Table 3. Linear regression analysis of lipid and metabolic parameters associated with log-transformed Lp(a).

Predictor	B	Std. Error	β (Standardized)	p-value
LDL-C (mg/dL)	0.002	0.000	0.157	< 0.001
Triglycerides (mg/dL)	−0.001	0.000	−0.088	0.019
HDL-C (mg/dL)	0.002	0.001	0.058	0.115
HbA1c (%)	0.027	0.013	0.067	0.043

Dependent variable = $\log_{10}$[Lp(a)+1]; Model $R^2 = 0.037$, $F = 8.67$, $p < 0.001$.

Discussion

In our study, 18.8% of participants had elevated Lp(a) levels, aligning with global prevalence estimates of 20–30% for elevated Lp(a).[1,15-17] This finding supports previous research suggesting that Lp(a) is an under-recognized yet significant cardiovascular risk factor and highlights the potential value of routine screening, especially in populations at risk for ASCVD.

Female participants had higher Lp(a) levels than males, mirroring large cohorts in which women—particularly after menopause—show approximately 15–30% higher concentrations than men.[18] This sex difference is thought to be driven predominantly by genetic variation in the Lipoprotein(a) Gene (*LPA*), which determines apo(a) isoform size and, consequently, Lp(a) secretion rate; individuals carrying smaller apo(a) isoforms (fewer kringle IV type-2 repeats) have higher plasma Lp(a) levels. A greater prevalence of these smaller isoforms or subtle differences in hepatic lipoprotein handling in women may therefore underlie higher Lp(a) independent of hormonal status.[19-21] Estrogen does modulate *LPA* expression. Its decline after menopause is associated with a moderate rise in Lp(a) that can be partly attenuated by hormone therapy. Still, these hormonal influences appear secondary to the strong genetic determination of Lp(a).[18] Consistent with these mechanisms, female sex was significantly associated with elevated Lp(a) in this population.

In our regression model, LDL-C was the only lipid parameter positively associated with Lp(a) ($B = 0.002$, $p < 0.001$), and triglycerides were inversely associated with Lp(a) ($B = -0.001$, $p = 0.019$), whereas HDL-C showed no significant relationship. Although HbA1c reached borderline significance in the multivariable model, this was not corroborated by correlation analysis and was sensitive to extreme values; we therefore did not interpret HbA1c as an independent determinant of Lp(a). Notably, these variables together explained only ~3.7% of the variance in Lp(a) ($R^2 \approx 0.037$), underscoring that Lp(a) is largely independent of traditional metabolic factors. Spearman correlation analysis supported these findings, demonstrating a weak positive association between log-transformed Lp(a) and LDL-C, a very weak but statistically significant correlation with HDL-C, and no significant correlations with triglycerides or HbA1c. Minor differences between Spearman coefficients and regression estimates likely reflect the non-normal distribution of Lp(a) and the differing assumptions of these analytical approaches. The modest Lp(a)–LDL correlation can be explained by shared structure and clearance pathways: each Lp(a) particle contains an LDL-like apolipoprotein B100 core, and both Lp(a) and LDL are cleared via the LDL receptor (which is downregulated by Proprotein Convertase Subtilisin/Kexin type 9 [PCSK9]).[22-23] Consistent with this, therapies that up-regulate LDL receptors, such as PCSK9 inhibitors, substantially reduce Lp(a) levels, supporting an intertwined catabolism of Lp(a) and LDL.[24] In contrast, the inverse Lp(a)–triglyceride relationship observed in our regression analysis may reflect competition for apolipoprotein B (apoB) in the liver. During hypertriglyceridemia, increased Very-Low-Density Lipoprotein (VLDL) output (each VLDL particle requires an apoB) could limit the availability of apoB for Lp(a) assembly.[25] Supporting this hypothesis, insulin-resistant states with high VLDL–triglyceride production tend to show lower Lp(a) levels.[26] Taken together, these findings support Lp(a) as an essentially fixed, heritable cardiovascular risk factor, predominantly genetically determined with only minor modulation by LDL-related and triglyceride pathways.

The results underscore the importance of assessing Lp(a) levels in all adults at least once, in accordance with the American College of Cardiology (ACC)/American Heart Association (AHA) 2018 and the European Society of Cardiology (ESC)/European Atherosclerosis Society (EAS) 2019 guideline recommendations.[27-28] Routine Lp(a) testing may enhance cardiovascular risk stratification, particularly for individuals with elevated Lp(a) but low ASCVD risk based on traditional lipid measures.[29] Incorporating Lp(a) into standard cardiovascular evaluations could facilitate more targeted prevention strategies and earlier management of at-risk individuals not identified by conventional cholesterol testing.

Although our study provides valuable insights into the prevalence and distribution of elevated Lp(a) in an Indonesian population, several limitations should be acknowledged. This cross-sectional, single-center design precludes causal inference and limits generalizability. Despite adjustment for key lipid and metabolic variables, residual confounding cannot be excluded. Larger, multi-center prospective studies are needed to validate these findings and further explore the clinical implications of elevated Lp(a) as a cardiovascular risk factor. Future studies should also assess the prognostic impact of elevated Lp(a) on long-term cardiovascular outcomes and evaluate potential therapeutic strategies once effective agents

become available.

Conclusion

In conclusion, 18.8% of participants in this study had elevated Lp(a) levels, consistent with global prevalence estimates. Females demonstrated significantly higher Lp(a) concentrations than males, and LDL-C showed a modest but statistically significant positive association with log-transformed Lp(a), independent of other lipid parameters. These findings highlight the growing relevance of Lp(a) as a critical cardiovascular risk marker in our community. Routine Lp(a) screening, particularly among individuals with high or borderline cardiovascular risk, may improve risk stratification and enable earlier, more targeted preventive interventions.

List of Abbreviations

ACC American College of Cardiology
AHA American Heart Association
apo(a) Apolipoprotein(a)
apoB Apolipoprotein B
ASCVD Atherosclerotic Cardiovascular Disease
BMI Body Mass Index
CVD Cardiovascular Disease
eGFR Estimated Glomerular Filtration Rate
EAS European Atherosclerosis Society
ESC European Society of Cardiology
HbA1c Hemoglobin A1c
HDL-C High-Density Lipoprotein Cholesterol
HTN Hypertension
LDL-C: Low-Density Lipoprotein Cholesterol
LPA Lipoprotein(a) Gene
Lp(a) Lipoprotein(a)
PCSK9 Proprotein Convertase Subtilisin/Kexin Type 9
VLDL Very-Low-Density Lipoprotein

Ethical Clearance

This study received ethical approval from the Faculty of Medicine, Pelita Harapan University (No. 207/K-LKJ/ETIK/V/2025).

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

Conceptualization and methodology: J.W.H., K.J.S., S.S.I.; Investigation: J.W.H., K.J.S., A.C.R.; Formal analysis: J.W.H., K.J.S., S.S.I.; Visualization and writing – original draft: J.W.H., K.J.S., A.C.R.; Writing – review and editing: S.S.I., L.P.S.; Supervision: L.P.S. All authors have read and approved the final version of the manuscript.

Acknowledgments

The authors are grateful to all colleagues from Siloam Hospital Kebon Jeruk for their support and contributions.

Conflict of Interest

All authors declare no conflicts of interest.

Availability of Data and Materials

The data that support the study's findings are available in the article.

Funding

No designated financial assistance was obtained for this work's research, authorship, or publishing.

Copyright/Permissions for Figures

Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

No artificial intelligence (AI) tools were used at any stage of this study, including data collection, analysis, visualization, or manuscript preparation. All aspects of the work were performed manually by the authors.

References

1. Vavuranakis M, Jones S, Cardoso R, Gerstenblith G, Leucker T. The role of lipoprotein(a) in cardiovascular disease: current concepts and future perspectives. Hellenic Journal of Cardiology 2020;61:398-403.
2. Alhomoud I, Talasaz A, Mehta A, Kelly M, Sisson E, Bucheit J, et al. Role of lipoprotein(a) in atherosclerotic cardiovascular disease: a review of current and emerging therapies. Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy 2023;43:1051-63.

3. Lampsas S, Xenou M, Oikonomou E, Pantelidis P, Lysandrou A, Sarantos S, et al. Lipoprotein(a) in atherosclerotic diseases: from pathophysiology to diagnosis and treatment. Molecules 2023;28:969.
4. Fusco S, Arca M, Scicchitano P, Alonzo A, Perone F, Gulizia M, et al. Lipoprotein(a): a risk factor for atherosclerosis and an emerging therapeutic target. Heart 2022;109:18-25.
5. Reyes-Soffer G, Ginsberg H, Berglund L, Duell P, Heffron S, Kamstrup P, et al. Lipoprotein(a): a genetically determined, causal, and prevalent risk factor for atherosclerotic cardiovascular disease: a scientific statement from the American Heart Association. Arteriosclerosis, Thrombosis, and Vascular Biology 2022;42.
6. Doherty S, Hernandez S, Rikhi R, Mirzai S, Reyes C, McIntosh S, et al. Lipoprotein(a) as a causal risk factor for cardiovascular disease. Current Cardiovascular Risk Reports 2025;1.
7. Pearson K, Rodríguez F. Lipoprotein(a) and cardiovascular disease prevention across diverse populations. Cardiology and Therapy 2020;9:275-92.
8. Francis G. Lipoprotein (a) in cardiovascular risk assessment and management in diabetes mellitus. Canadian Diabetes &Amp Endocrinology Today 2023.
9. Rhee E. The prognostic role of lipoprotein(a) in cardiovascular disease. Cardiovascular Prevention and Pharmacotherapy 2025;7:9-12.
10. Kronenberg F, Mora S, Stroes E, Ference B, Arsenault B, Berglund L, et al. Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European atherosclerosis society consensus statement. European Heart Journal 2022;43:3925-46.
11. Annink M, Janssen E, Reeskamp L. Effectiveness of cascade screening for elevated lipoprotein(a), an underdiagnosed family disorder. Current Opinion in Lipidology 2024;35:290-6
12. Jubran A, Zetser A, Zafrir B. Lipoprotein(a) screening in young and middle-aged patients presenting with acute coronary syndrome. Cardiology Journal 2019;26:511-8.
13. Ruscica M, Rizzuto A, Corsini A. Role of lipoprotein(a) in plaque progression. European Heart Journal Supplements 2022;24(Supplement_I):I72-5.
14. Tsimikas S, Fazio S, Ferdinand K, Ginsberg H, Koschinsky M, Marcovina S, et al. Nhlbi working group recommendations to reduce lipoprotein(a)-mediated risk of cardiovascular disease and aortic stenosis. Journal of the American College of Cardiology 2018;71:177-92.
15. Nordestgaard B, Langsted A. Lipoprotein(a) and cardiovascular disease. The Lancet 2024;404:1255-64.
16. Dyrbuś K, Mędrala Z, Konsek K, Nowowiejska-Wiewióra A, Trzeciak P, Skrzypek M, et al. Study of lipoprotein(a) and its impact on atherosclerotic cardiovascular disease: design, rationale, and first results of the zabrze-lip(a)r registry. European Heart Journal 2024;45(Supplement_1).
17. Mascarenhas M, Garcez J, Silva B, Ferreira I, Oliveira J, Palma I. prevalence of lp(a) in a real-world Portuguese cohort: implications for cardiovascular risk assessment. Lipids in Health and Disease 2025;24.
18. Simony S, Mortensen M, Langsted A, Afzal S, Kamstrup P, Nordestgaard B. Sex differences of lipoprotein(a) levels and associated risk of morbidity and mortality by age: the Copenhagen general population study. Atherosclerosis 2022;355:76-82.
19. Sosnowska B, Toth PP, Razavi AC, Remaley AT, Blumenthal RS, Banach M. 2024: the year in cardiovascular disease – the year of lipoprotein(a). Research advances and new findings. Archives of Medical Science. 2025;21:355.
20. Kostner KM, Marz W, Kostner GM. When should we measure lipoprotein(a)? European Heart Journal. 2013;34:3268-76.
21. Kronenberg F, Utermann G. Lipoprotein(a): resurrected by genetics. Journal of Internal Medicine. 2013;273:6-30.
22. Schmidt K, Noureen A, Kronenberg F, Utermann G. Structure, function, and genetics of lipoprotein(a). Journal of Lipid Research 2016;57:1339-59.
23. Romagnuolo R, Scipione CA, Marcovina SM, Gemin M, Seidah NG, Boffa MB, Koschinsky ML. Roles of the low-density lipoprotein receptor and related receptors in inhibition of lipoprotein(a) internalization by proprotein convertase subtilisin/kexin type 9. PLoS One 2017;12:e0180869.
24. Raal FJ, Giugliano RP, Sabatine MS, Koren MJ, Blom D, Seidah NG, Honarpour N, Lira A, Xue A, Chiruvolu P, Jackson S. PCSK9 inhibition-mediated reduction in Lp(a) with evolocumab: an analysis of 10 clinical trials and the LDL receptor's role. Journal of Lipid Research 2016;57:1086-96.
25. Sheashaa H, Mousa H, Abbas MT, Farina JM,

Awad K, Pereyra M, Scalia IG, Ali NB, Javadi N, Bismee NN, Esfahani SA. Interaction between lipoprotein(a) and other lipid molecules: a review of the current literature. Biomolecules 2025;15:162.
26. Corral P, Matta MG, Schreier L. Are lipoprotein(a) levels decreased in insulin resistance and type 2 diabetes? Current Opinion in Lipidology 2025;36:179-84.
27. Grundy S, Stone N, Bailey A, Beam C, Birtcher K, Blumenthal R, et al. 2018 aha/acc/aacvpr/aapa/abc/acpm/ada/ags/apha/aspc/nla/pcna guideline on the management of blood cholesterol: a report of the american college of cardiology/american heart association task force on clinical practice guidelines. Circulation 2019;139.
28. Mach F, Baigent C, Catapano A, Landmesser U, Drexel H, Corsini A, et al.. 2019 esc/eas guidelines for managing dyslipidaemias: lipid modification to reduce cardiovascular risk. European Heart Journal 2019;41:111-88.
29. Nurmohamed N, Moriarty P, Stroes E. Considerations for routinely testing for high lipoprotein(a). Current Opinion in Lipidology 2023.