

EFLM Paper

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Survey on lipoprotein(a) testing in Europe. A report by the EFLM Committee on Vascular Risk Biomarkers

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Abstract

Objectives: Lipoprotein(a) [Lp(a)] is a lipoprotein particle similar to low-density lipoprotein with an additional apolipoprotein(a) [apo(a)] covalently linked to apoB-100. Apo(a) is highly polymorphic due to a conserved sequence motif which is repeated 10 to >50-times which is a challenge to immunoassays. Harmonization of diagnostic assays is so far unsatisfactory. This includes the units to report measurement results. This survey should provide information on the current practice of Lp(a) measurement and reporting in EFLM member countries.

Methods: A questionnaire entitled “Lipoprotein (a) – Biomarkers of Cardiovascular Risk” was developed. It consisted of 38 questions for laboratories which measure Lp(a) and 21 questions for laboratories which do not measure Lp(a). A call for the survey was sent out by the EFLM to all members in December 2024. The survey remained open until end of January 2025.

Results: There were 340 laboratories from 40 EFLM countries completing the survey of which 247 measured Lp(a) and 93 did not. There was significant heterogeneity in assay usage, reflected by the fact that the five most common assays made up for only 53 % of all laboratories. Approx. one half each of laboratories reported Lp(a) in mass units or in molar units which was largely dependent on the manufacturer’s application.

Conclusions: The survey findings demonstrate that variability in Lp(a) measurement spans all phases of the testing process and involves multiple stakeholders. Laboratories can optimize pre-analytical handling and result reporting. However, the analytical domain is the most critical part which requires coordinated action between standardization bodies and IVD-manufacturers, supported by aligned clinical guidelines.

Keywords: lipoprotein(a); survey; Europe

Introduction

Lipoprotein(a) [Lp(a)] was first described by Kare Berg in 1963 [1]. In the following quarter century the structure of Lp(a) and its main protein components apolipoprotein (apo) B-100 and apo(a) were determined. Lp(a) represents a low-density lipoprotein (LDL)-particle modified by the addition and covalent attachment of apo(a) to apoB-100. There are also some peculiarities of lipid-composition of Lp(a) compared to LDL [2–4].

Two features of apo(a) are remarkable: (1) apo(a) is highly homologous to human plasminogen, in particular its carboxyterminal half with kringles IV and V and the protease domain, and (2) due to a variable number of repeats of the kringle IV type-2 (KIV-2) motif, the molecular mass of apo(a) varies from approx. 300–800 kDa [2].

Lp(a) has been identified unequivocally as cardiovascular risk factor by traditional epidemiologic studies as well as by Mendelian randomization studies [5–6]. Unfortunately,

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dietary and drug treatment of elevated Lp(a) has been notoriously ineffective. Therefore, Lp(a) concentration has been measured primarily for cardiovascular risk assessment rather than treatment monitoring. Due to the small intraindividual variability in adult life repeated measurements for risk assessment have not been advocated. However, in recent years several drugs which lower Lp(a) have been developed and some of these are in advanced phase III clinical studies [7]. Thus, it is likely that treatment monitoring will become an indication of Lp(a) measurements.

There are two major issues when Lp(a) concentration is measured. First, most immunoassays are affected by the variable number of kringle IV-2 repeats, because available antibodies for immunoassays bind to epitopes in kringle IV-2. This causes systematic deviations in assay results depending on the number of kringle IV-2 repeats in a person's apo(a). Second, in the early literature about Lp(a) concentration was reported in mg/dL, i.e. total particle mass. Since neither the size of apo(a) nor the lipid composition of Lp(a) are known, estimation of particle mass is inherently inaccurate. It has been shown amply that assay calibration and reporting of Lp(a) in molar units of apo(a) concentration is much superior [8–10]. However, in spite of recommendations by several experts and societies to measure and report Lp(a) in molar units [11, 12], Lp(a) concentration is widely reported in mass units.

The aim of our survey was to get an overview of the practice of Lp(a) measurement in European countries. In addition, knowledge of laboratories regarding the specific issues related to Lp(a) and its diagnostic measurement should be analyzed.

Materials and methods

The European Federation of Clinical Chemistry and Laboratory Medicine (EFLM) Committee on Vascular Risk Biomarkers conducted an online survey to assess the practice of lipoprotein (a) [Lp(a)] measurement in member countries in December 2024 and January 2025. The aim was to assess the current situation regarding the availability, application, and interpretation of Lp(a) measurement. A call for the survey was sent out by the EFLM to all members in December 2024. The survey remained open until end of January 2025.

The questionnaire entitled “Lipoprotein (a) – Biomarkers of Cardiovascular Risk” was developed by the committee. It consisted of 38 questions for laboratories which measure Lp(a) and 21 questions for laboratories which do not measure Lp(a). Questions were divided into 5 sections. The first section consisted of 5 general questions about the laboratory, including whether Lp(a) is measured or not. The second section

consisted of 6 questions about the use of biomarkers of cardiovascular risk. In the third section 5 questions regarding the general practice of Lp(a) measurement in the respective country were asked. The fourth and fifth sections were specific for laboratories which do or do not measure Lp(a). Laboratories which measure Lp(a) were asked 22 questions concerning general knowledge concerning Lp(a) as well as preanalytical, analytical, and reporting practice. Laboratories which do not measure Lp(a) were asked 5 questions regarding handling of orders for Lp(a) and future plans for Lp(a) measurement.

The survey was hosted by LimeSurvey and is available at the following URL: <https://survey.eflm.eu/index.php/312122?lang=en>.

All responses were collected in a data base and analyzed by Microsoft Excel.

Results

There were 340 responses from 40 EFLM countries to the survey with relevant information (range 1–30 responses per country). Most laboratories belonged to hospitals (260–77 %) followed by private laboratories (63–19 %). Thirteen (4 %) laboratories belonged to other categories. Seventy-three percent (247) of laboratories measured Lp(a) and 27 % (93) did not. The proportion of laboratories that measure Lp(a) was quite different between EFLM countries ranging from 0 % to 100 % (Figure 1). If only the twelve countries with 10 or more responding laboratories were considered the range was from 30 % to 100 %.

With the exception of laboratories with less than 100,000 reported results per year there was a clear trend that larger laboratories were more likely to measure Lp(a) than smaller laboratories (52.4 % of laboratories with 100,000–1,000,000 reported results compared to 86 % of laboratories with >10,000,000 reported results per year). A majority of laboratories (approx. 60 %) indicated that Lp(a) is usually measured together with LDL-cholesterol as part of the standard lipid profile. However, more than 75 % of laboratories answer that they report at least 10-times more LDL-cholesterol results than Lp(a) results.

Type of assay and sample matrix

The five most commonly used assays made up for approx. 53 % of all laboratories with one assay alone accounting for approx. 27 % (Table 1). Only eight (3 %) laboratories used a laboratory developed test including one liquid chromatography-mass spectrometry (LC-MS) method. Surprisingly 37 % of responding

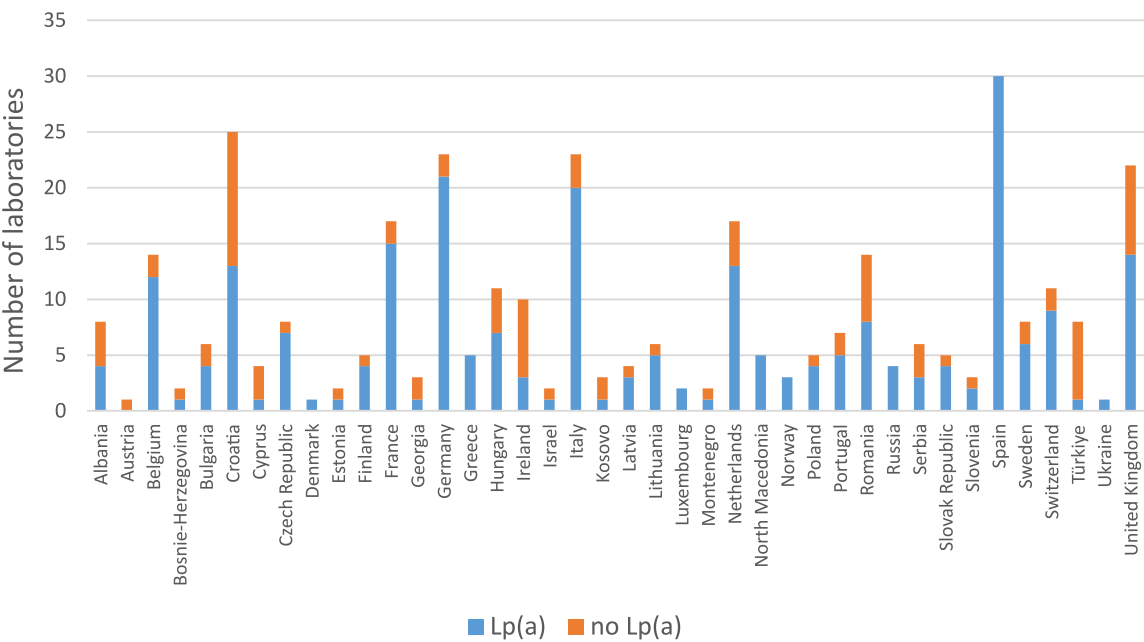


Figure 1: Distribution per EFLM member country of laboratories which measure Lp(a) and laboratories which do not (situation d.d. Jan 2025).

laboratories did or could not name the assay they used. The most common sample matrix was serum (81 %) followed by heparin-plasma (18 %) and EDTA-plasma (2 %). Of the 170 laboratories which answered whether they measured Lp(a) from fresh or frozen samples 86 % (147) used fresh samples while 14 % used frozen samples.

Laboratories were also asked on what criteria their decision for a specific assay was based. Five options were listed in the question and should be ranked. Of note, this question was only answered by 123 laboratories which

measure Lp(a), even though it was asked to all laboratories modified for laboratories which do not measure Lp(a) currently. For half of the laboratories the most important criterion was applicability to an available analyzer followed by accuracy of the method (Table 2).

Units to report Lp(a)

All laboratories reporting the assay type also specified the units to report Lp(a). The units reported correlated strongly with the commercial assay used and hence, the manufacturers recommendation. Overall, approx. one third each report Lp(a) in mass units (mg/dL or mg/L; 36 %) or molar units (nmol/L; 32 %). Another 32 % did not specify the units in

Table 1: Lp(a) assays applied.

Test	n	%	Principle	Design of the calibration curve
All	245	100.0 %		
Roche	67	27.3 %	ITA	1
Siemens	14	5.7 %	ITA	1
Siemens	12	4.9 %	INA	2
Abbott Alinity/Architect	19	7.8 %	ITA	1
Binding Site Optilite	10	4.1 %	ITA	
Randox	9	3.7 %	ITA	1
Beckman Coulter	6	2.4 %	INA	1
Sentinel	6	2.4 %	ITA	1
Other	4	1.6 %	Variable	Unknown
LDT	8	3.3 %	Variable	Unknown
Do not know	13	5.3 %	n.a.	
Not specified	77	31.4 %	n.a.	

INA, immunonephelometry; ITA, immunoturbidimetry; LDT, laboratory developed test. 1) Multipoint calibration with independent calibrators. 2) Serial dilution of high calibrator.

Table 2: Criteria for the choice of Lp(a) assay.

Criterion	#1	#2	#3	#4	#5
Applicable to analyzer available in the lab	62	36	17	6	2
Accuracy of method (e.g. traceability, measurement uncertainty)	38	40	36	8	1
Automated assay with high throughput	21	36	41	13	12
Price	2	10	21	57	73
Recommendation by colleague	0	1	8	39	75

The column headers indicate the order of the selection criteria. #1 is the most important criterion, and #5 is the least important criterion. The numbers indicate how often each criterion was mentioned. Bold numbers indicate that a criterion was most often given the respective importance.

which they report Lp(a) concentration. Conversion of mg/dL to nmol/L or *vice versa* is uncommon with only 9 % of laboratories. Interestingly, conversion is exclusively from molar units to mass units. The preferences for units varies between countries (Figure 2). Unexpectedly, the laboratory which uses LC-MS reports Lp(a) in mg/dL although it is a molar measurement.

We then asked what is measured and reported when Lp(a) is measured. Intriguingly, only 61 % of laboratories reporting mass units answered that they determined Lp(a) mass, while 24 % answered particle count, and 15 % did not know. For laboratories reporting molar units the proportion of correct answers was significantly higher (Table 3).

Apo(a) size polymorphism

When we asked about the awareness of the apo(a) size polymorphism there was a significant difference between laboratories which measure Lp(a) and laboratories which do not measure Lp(a). Approx. three quarters of the former were aware of the polymorphism, while this was only true for approx. 40 % of the latter. However, half of the laboratories which measure Lp(a) did not know, if calibration of their assay compensates for the size polymorphism. Of the

Table 3: Responses regarding reported Lp(a) units and measurand.

	mg/dL or L	nmol/L	
Mass	54	10	64
Particle count	21	62	83
Do not know	13	7	20
	88	79	

Column headings indicate reported units; line headings indicate the laboratories response regarding what was measured.

other half approx. 40 % answered that their assay does not compensate for the size polymorphism, i.e. reduce measurement uncertainty due to the size polymorphism during calibration.

Cardiovascular risk thresholds

We then asked for the risk thresholds indicated by the laboratories. In this questionnaire, risk thresholds 75 and 125 nmol/L and the EAS consensus of <75 nmol/L for low risk, >125 for increased risk and 75–125 nmol/L grey zone as the most common thresholds were given. For mg/dL 30 and 50 mg/dL and again the EAS consensus of <30 mg/dL for low risk, >50 mg/dL for increased risk and 30–50 mg/dL grey zone

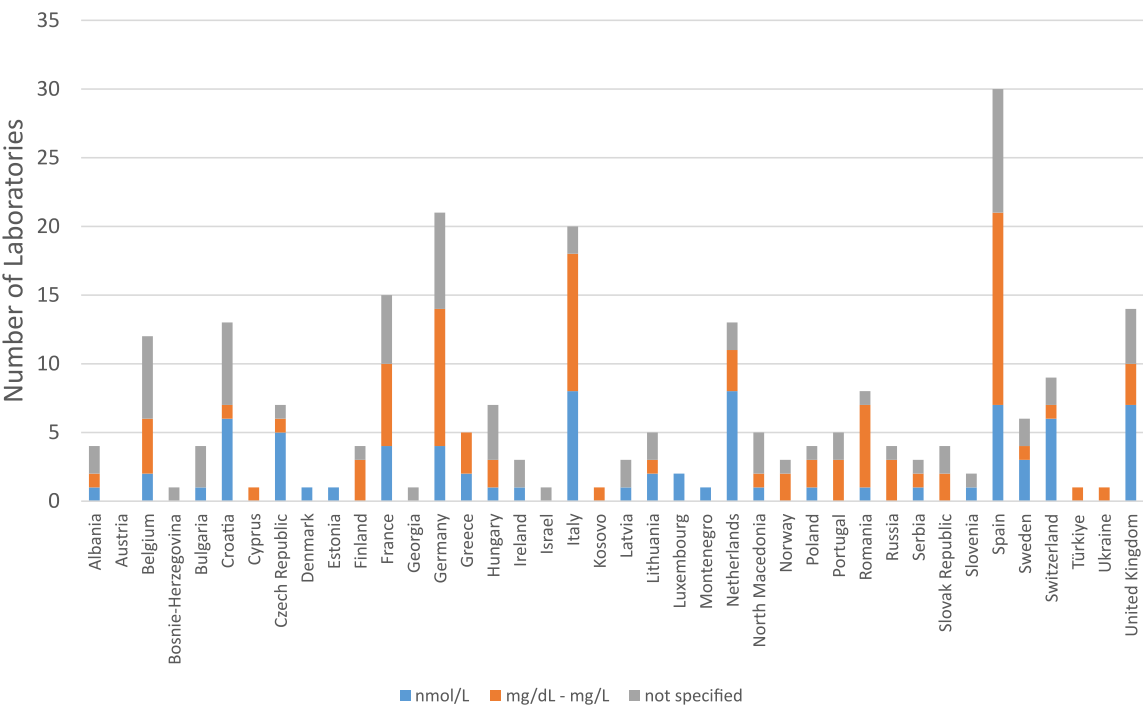


Figure 2: Distribution of reported units for Lp(a) concentration per EFLM member country.

thresholds were given. It was also possible to indicate that another threshold was used. Only laboratories which used a specified unit for reporting were evaluated. Results are summarized in Table 4. Of note, some laboratories reporting Lp(a) in mass units indicated risk thresholds in molar units and vice versa. If this was just an error or real practice remains unknown. Most laboratories did not comment on concentrations of Lp(a) beyond their respective risk thresholds.

Laboratories that do not measure Lp(a)

We asked laboratories that do not measure Lp(a), if they send samples to another laboratory, when they get a request for Lp(a). Seventy-four of 93 laboratories answered this question. The majority (78.4%) answered this question in the affirmative. Forty-two laboratories do not plan to implement an Lp(a) assay in the future while 30 laboratories plan to do so.

Discussion

The present survey highlights persistent heterogeneity in lipoprotein(a) [Lp(a)] measurement practices across laboratories and gaps of knowledge, underscoring critical challenges throughout the pre-analytical, analytical, and post-analytical phases. Importantly, these challenges are not solely technical but reflect a distributed responsibility across multiple stakeholders, including standardization bodies, reference material providers and metrology institutes, assay manufacturers, clinical guideline organizations, and laboratories. The lack of alignment among these actors continues to limit progress toward full harmonization.

Table 4: Cardiovascular risk threshold indicated by laboratories.

	Reported units	
	mg/dL – mg/L	nmol/L
All	88	79
30 mg/dL	34	
50 mg/dL	26	1
EAS consensus, mg/dL	13	1
Other, mg/dL	2	
75 nmol/L	3	42
125 nmol/L	3	9
EAS-consensus, nmol/L	3	13
Other, nmol/L		11
No response	4	2

Pre-analytical considerations

Pre-analytical variability remains an important and often underestimated source of uncertainty in Lp(a) measurement. Although Lp(a) is generally considered stable, it is not. Freeze–thaw cycles may affect apo(a) structure and epitope accessibility, introducing assay-dependent variability. This may be relevant when samples are analyzed in batches or sent to another laboratory. In addition, matrix effects – particularly differences between serum and plasma or the influence of anticoagulants – may lead to systematic bias and/or confounding in individual specimens [13, 14].

Responsibility for managing these pre-analytical variables lies primarily with clinical laboratories, which define sample handling, storage, and processing protocols. However, this responsibility is constrained by incomplete or inconsistent guidance from manufacturers and standardization bodies. Greater leadership from professional organizations (e.g., EFLM, IFCC) is needed to establish clear, evidence-based pre-analytical standards, while manufacturers should provide more explicit validation data regarding sample stability and matrix effects. Without coordinated guidance, local practices will continue to introduce avoidable variability.

Analytical challenges

Analytical variability remains the dominant limitation in Lp(a) measurement. A recent study showed differences of up to 5-times in Lp(a) concentration between commercially available assays, even though calculated correlation between assays was excellent [15]. It is obvious that such differences are of major clinical relevance as they affect individual risk prediction and treatment decisions. Thus, standardization of Lp(a) assays by IVD-manufacturers to internationally agreed, IFCC-endorsed reference methods and material is urgently needed. A road map for apo(a)/Lp(a) test standardization, with preservation of traceability of test results to the first WHO-IFCC reference material SRM2B, has been described recently [10].

However, despite recommendations to calibrate and report Lp(a) in molar units (nmol/L) for making test results traceable to the former higher order reference measurement system developed by Santica Marcovina in the nineties [16], most assays remained calibrated in mass units. It should be noted that *by design* no assay available is able to measure Lp(a) mass. Thus, for fundamental metrological reasons Lp(a) particle number must be estimated from a molar signal. Since neither the mass of apo(a) (variable number of KIV-2 repeats) nor the lipid composition of individual Lp(a) particles are known precisely, estimation of Lp(a) mass is

always flawed and uncertain. For test standardization according to ISO 17511:2020 based on reference measurement procedures and primary/secondary reference materials the measurand should be well defined first. Due to the extreme heterogeneity of apo(a), the exact molecular proteoform (in the case of MS-based tests) or epitopes (in the case of IA-based tests) of interest for apo(a) quantitation should remain unchanged across the calibration hierarchy. Thus, standardization of Lp(a) assays necessitates detection and quantification in moles/molar units and assays calibrated to mass units are by design confounded and lack traceability to SRM2B.

A relevant problem for immunoassays is the presence of variable numbers of KIV-2 repeats in apo(a). Since most antibodies used in diagnostics bind in this region, signal intensity is not only affected by apo(a) concentration, but also by the number of accessible epitopes within the apo(a) molecule. It has been shown that the use of multiple independent calibrators, rather than relying solely on serial dilution, represents a more robust approach, as it better captures biological variability and reduces systematic isoform-related confounding. Calibration models incorporating such multi-point strategies, combined with traceability to international reference material such as SRM2B, are essential for advancing analytical harmonization [11]. Notably, also other genetic variation, N- and O-glycosylation and posttranslational modifications, conformational changes, proteolysis, etc. may affect Lp(a) immunoassay recovery [11, 17, 18].

Accountability for resolving these analytical limitations is shared but asymmetrical. International standardization bodies (IFCC, WHO, EFLM) bear primary responsibility for establishing robust reference systems and traceability frameworks. However, *in vitro* diagnostic (IVD) manufacturers are operationally accountable, as they control assay design, antibody specificity, calibration strategy, and reporting units. Without active implementation of the agreed international higher order reference measurement procedure and material by IVD-manufacturers – including transition to molar reporting and adoption of calibration hierarchies – standardization efforts cannot succeed. Academic and reference laboratories contribute critical evidence and method evaluation but lack the authority to enforce change.

Post-analytical interpretation

Post-analytical variability further complicates the clinical application of Lp(a). Differences in reporting units, interpretative comments, and decision thresholds contribute to inconsistent risk assessment. While Lp(a) concentrations expressed in nmol/L more accurately reflect particle number – and are

analytically equivalent to apo(a) molar concentration, given that each particle contains a single apo(a) molecule – clinical reporting continues to rely on the term Lp(a), consistent with the existing evidence base and guideline frameworks.

A major challenge remains the lack of globally harmonized clinical decision limits. European guidance from the EAS and ESC typically recommends thresholds around 50 mg/dL (≈ 105 nmol/L; 80th percentile), with increasing recognition of a continuous risk relationship [5]. Also AHA/ACC guidelines emphasize optimal control of cardiovascular risk factors if Lp(a) concentration is >50 mg/dL (which is assumed to be equivalent to 125 nmol/L) within a broader risk assessment strategy [19]. These differences reflect divergent clinical philosophies but create ambiguity in laboratory reporting and interpretation. Furthermore, it is obvious that the European and American societies use different factors to convert molar units to mass units underscoring once more the need for uniform use of the metrologically correct molar units.

In this post-analytical domain, clinical guideline organizations (e.g., EAS/ESC; AHA/ACC) bear primary accountability for harmonizing interpretative frameworks and promoting consistent use of units and thresholds. Regulatory authorities may further support this process by encouraging alignment between analytical performance and clinical recommendations. Laboratories, while responsible for accurate reporting, are largely dependent on upstream standardization and guidance.

Implications and future directions

The survey findings demonstrate that variability in Lp(a) measurement is a systemic issue spanning all phases of the testing process and involving multiple stakeholders. While laboratories can optimize pre-analytical handling and reporting practices, the most critical barriers reside in the analytical domain and require coordinated action between standardization bodies and IVD-manufacturers, supported by aligned clinical guidelines.

Progress toward harmonization will depend on:

- standardized pre-analytical protocols supported by professional organizations and manufacturers;
- adoption of KIV-2 isoform-independent molar-based tests with improved calibration strategies (including multiple calibrators);
- global alignment of clinical interpretation and decision limits across guideline bodies.

Only through coordinated efforts across these stakeholder groups the longstanding challenges of Lp(a) standardization

can be effectively addressed and its full clinical utility realized.

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References

- Berg K. A new serum type system in man: the Lp-system. *Acta Pathol Microbiol Scand* 1963;59:369–82.
- McLean JW, Tomlinson JE, Kuang WJ, Eaton DL, Chen EY, Fless GM, et al. cDNA sequence of human apolipoprotein(a) is homologous to plasminogen. *Nature* 1987;330:132–7.
- Utermann G. The mysteries of lipoprotein(a). *Science* 1989;246:904–10.
- Leibundgut G, Scipione C, Yin H, Schneider M, Boffa MB, Green S, et al. Determinants of binding of oxidized phospholipids on apolipoprotein (a) and lipoprotein (a). *J Lipid Res* 2013;54:2815–30.
- Kronenberg F, Mora S, Stroes ESG, Ference BA, Arsenault BJ, Berglund L, et al. Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European Atherosclerosis Society consensus statement. *Eur Heart J* 2022;43:3925–46.
- Kronenberg F, Utermann G. Lipoprotein(a): resurrected by genetics. *J Intern Med* 2013;273:6–30.
- Anchouche K, Thanassoulis G. Lp(a): a rapidly evolving therapeutic landscape. *Curr Atheroscler Rep* 2024;27:7.
- Albers JJ, Marcovina SM, Lodge MS. The unique lipoprotein(a): properties and immunochemical measurement. *Clin Chem* 1990;36:2019–26.
- Marcovina SM, Albers JJ. Lipoprotein (a) measurements for clinical application. *J Lipid Res* 2016;57:526–37.
- Cobbaert CM, van Neer NJM, Diederiks NM, Leijnse EJJ, Ruhaak LR. On the cusp of global lipoprotein(a) standardization. *Clin Chem Lab Med* 2026;64:1064–73.
- Ruhaak LR, Cobbaert CM. Quantifying apolipoprotein(a) in the era of proteoforms and precision medicine. *Clin Chim Acta* 2020;511:260–8.
- White-Al Habeeb NMA, Higgins V, Venner AA, Bailey D, Beriault DR, Collier C, et al. Canadian society of clinical chemists working group on reference interval harmonization. Canadian society of clinical chemists harmonized clinical laboratory lipid reporting recommendations on the basis of the 2021 Canadian cardiovascular society lipid guidelines. *Can J Cardiol* 2022;38:1180–8.
- Sgoutas DS, Tuten T. Effect of freezing and thawing of serum on the immunoassay of lipoprotein(a). *Clin Chem* 1992;38:1873–7.
- Amesz JH, Cobbaert CM, Kusters E. Effect of heparin plasma storage at –20 °C or 4 °C on lipoprotein(a) risk stratification. *Clin Chem Lab Med* 2026;64:e240–2.
- Yamashita K, Wakita M, Niizeki N, Ito S, Ito Y, Sato A, et al. JSLM, JSCC, JCCLS and JACRI joint working group on Japan research project on the global standardization/harmonization of lipoprotein (a) measurement. Harmonization of lipoprotein(a) immunoassays with the IFCC- endorsed reference measurement procedure validated in patient samples and multiple routine testing as a second step towards apolipoprotein standardization. *J Atheroscler Thromb* 2026; 33. <https://doi.org/10.5551/jat.66345> [Epub ahead of print].
- Marcovina SM, Albers JJ, Scanu AM, Kennedy H, Giaculli F, Berg K, et al. Use of a reference material proposed by the International Federation of Clinical Chemistry and Laboratory Medicine to evaluate analytical methods for the determination of plasma lipoprotein(a). *Clin Chem* 2000;46:1956–67.
- Coassin S, Schönherr S, Weissensteiner H, Erhart G, Forer L, Losso JL, et al. A comprehensive map of single-base polymorphisms in the hypervariable LPA kringle IV type 2 copy number variation region. *J Lipid Res* 2019;60:186–99.
- Coassin S, Kronenberg F. Lipoprotein(a) beyond the kringle IV repeat polymorphism: the complexity of genetic variation in the LPA gene. *Atherosclerosis* 2022;349:17–35.
- Blumenthal RS, Morris PB, Gaudino M, Johnson HM, Anderson TS, Bittner VA, et al. 2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Dyslipidemia: a Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol* 2026;87:2624–757.