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Lipoprotein (a): A biomarker of cardiovascular risk detectable in childhood

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ABSTRACT

Lipoprotein (a) [Lp(a)] is a lipoprotein similar to low-density lipoprotein (LDL), which binds to a characteristic component: apolipoprotein (a). The plasma Lp(a) level is mainly determined by genetic factors, with variations across ethnic groups. In adults, various epidemiological and genetic studies have shown that elevated Lp(a) levels are an independent risk factor for atherosclerotic cardiovascular disease and aortic valve stenosis, associated with inflammatory, atherogenic, and thrombotic mechanisms.

Given that the distribution, variability, and prognostic value of this marker in the pediatric population have been less investigated, the objective of this review is to analyze the available evidence on the behavior of Lp(a) as a risk marker in children and adolescents, current recommendations for its measurement in pediatrics, and treatment prospects.

Keywords: *lipoprotein (a); biomarkers; risk factors for heart disease; pediatrics.*

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INTRODUCTION

Lipoprotein (a) [Lp(a)] is composed of a particle similar to low-density lipoprotein (LDL), in which apolipoprotein B100 is bound by a disulfide bond to apolipoprotein (a) [Apo(a)].[1,2] Genetic factors primarily determine plasma levels;[3] diet, body weight, and physical activity have minimal impact.[4]

There are important differences depending on the unit used for reporting; Lp(a) levels expressed in mg/dL reflect mass, while those reported in nmol/L reflect the number of particles, which are closely associated with cardiovascular risk. It is important to note that the two analytical methods are not interchangeable, as conversions between the two units are not accurate because they depend on Apo(a) isoforms. Although both forms of measurement are acceptable, it is preferable to express results in nmol/L to minimize measurement bias.[5,6]

In adults, multiple studies have shown that elevated Lp(a) levels are an independent risk factor for atherosclerotic cardiovascular disease and aortic valve stenosis, related to inflammatory, atherogenic, and thrombotic mechanisms (*Table 1* and *Figure 1*).[7-9] This association has been observed when values exceed 30 mg/dL (75 nmol/L),[10] while values > 50 mg/dL (or >105 nmol/L) are the cut-off points for identifying patients at higher risk.[5]

Given that the distribution, variability, and prognostic value of this marker in childhood have been less explored, the objective of this review is to analyze the available evidence on the behavior of Lp(a) as a risk marker in the pediatric population, current recommendations on its measurement, and treatment prospects.

CURRENT STATE OF KNOWLEDGE

The current information on Lp(a) is presented and analyzed in six items:

a) Lp(a) values in children and adolescents (studies in Europe, the United States, Asia, and Latin America); b) variability in the pediatric population and comparison with adults; c) prognostic marker in pediatrics related to surrogates of atherosclerosis; d) prognostic marker in pediatrics related to clinical events; e) recommendations for its measurement; and f) current treatment, perspectives, and conclusions.

VALUES IN CHILDREN AND ADOLESCENTS

At this point, we will briefly discuss the information obtained from the studies analyzed, conducted in Europe, the United States, Asia, and Latin America. The characteristics of each study, the results obtained, the distribution of Lp(a) levels in the pediatric population, and the main epidemiological findings are shown in *Table 2.*

Studies in Europe

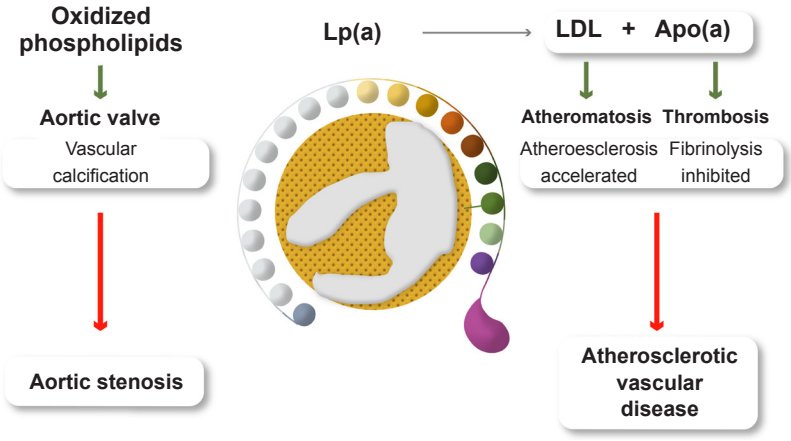
In Spain, the Rivas-Vaciamadrid study[11] analyzed Lp(a) levels and found that in 19.9% of cases, Lp (a) was >30 mg/dL, with an asymmetric distribution and no significant differences by sex. In another study conducted in the same country,[12]

Table 1. Pathophysiological mechanisms explaining the increased cardiovascular risk associated with elevated levels elevated levels of lipoprotein (a)

Action	Mechanisms
Proatherosclerotic	↑ endothelial permeability ↑ smooth muscle cell proliferation and migration ↑ foam cell formation ↑ calcification
Proinflammatory	↑ endothelial dysfunction ↑ supply of oxidized phospholipids ↑ monocyte migration to the subendothelium ↑ IL-8 expression in macrophages ↑ macrophage apoptosis
Procoagulant/antifibrinolytic	↑ PAI-1 expression ↑ platelet activation ↓ tissue factor pathway inhibitor ↓ clot permeability ↓ plasminogen activation

IL: interleukin; PAI-1: plasminogen activator inhibitor-1.

Figura 1. Schematic representation of the structure and main functions of lipoprotein (a)



Lp(a): lipoprotein (a); LDL: low-density lipoprotein; Apo(a): apolipoprotein (a).

the percentage of individuals with an elevation >30 mg/dL was much lower: 9.4% (7.4% in males and 11.4% in females). The highest values were observed in those with a family history of cardiovascular disease, compared to the group without a history. In both situations, healthy children were studied, and the differences were not significant.[13]

In Italy, however, the two studies analyzed pediatric patients with familial dyslipidemia and, in the other, patients with risk factors.[14] In the first study, elevated Lp(a) values were higher in those with a family history of cardiovascular disease compared to the group without a family history (21% of children with a family history and 9.2% without a family history had values ≥ 85th percentile). The second study[15] included patients with obesity, dyslipidemia, and hypertension, 22.6% of whom had elevated Lp(a) values ≥75 nmol/L; in 5.6% of these patients, the elevated values were associated with high LDL cholesterol (LDL-C) levels.

In Germany,[16] healthy children were analyzed, and only 12.5% had levels ≥50 mg/dL, with no significant differences by sex. In Poland,[17] patients with obesity, hypertension, and/or diabetes were evaluated, and the mean Lp(a) was 30 mg/dL, double that of the control group. In Wales,[18] healthy children were also evaluated, and 26% had Lp(a) levels ≥30 mg/dL (60% had a family history of cardiovascular disease).

A study conducted in Norway[19] on children with heterozygous familial hypercholesterolemia showed that 20.3% of the total had Lp(a) values >50 mg/dl or >105 nmol/l, with a statistically significant difference between females and males.

Studies in the United States

Wang et al.[20] evaluated a group of healthy pediatric patients from the Cherokee community and observed lower values in this population than in the aforementioned studies (mean and median <15 mg/dL), with no significant differences between the sexes. The Third National Health and Nutrition Examination Survey in children aged 4 to 19 years revealed higher lipid levels in African Americans.[21] An association was reported between Lp(a) levels and family history of cardiovascular disease. Another study in patients with diabetes[22] also showed higher levels in the African American population.

Studies in Asia

In Kuwait, Alsaeid et al.[23] analyzed Lp(a) values in healthy children, with an asymmetric distribution of the mean, with higher concentrations at lower levels, and ≥30 mg/dL in only 8.7%. Similar results were reported in Taiwan,[24] where healthy patients were also evaluated, and the mean and median did not exceed 20 mg/dL.

In Lebanon,[25] 14.4% had Lp(a) levels ≥75 nmol/L, with a median of 20 nmol/L, with no significant differences between sexes. In Korea, Choi et al.[26] observed that 11.3% of patients had values ≥ 100 nmol/L and reported a 90th percentile of 107.8 nmol/L. In Turkey,[27] healthy children with a family history of premature cardiovascular disease were analyzed in comparison with a control group, and no significant differences were observed.

Studies in Latin America

In Brazil, 43.8% of school-age children had Lp(a) values >30 mg/dL, with no significant

Table 2. Distribution of lipoprotein levels (a) in the pediatric population

Study (country, year)	N	Population	Lp(a) cutoff point used	Lp(a) values Mean (SD) / median (IQR)
González et al. (Spain, 2003)[11]	673	Healthy 6-year-old children.	Lp(a) ≥30 mg/dL: 19.9%	
Meabe et al. (Spain, 2006)[12]	98	Healthy children aged 6 to 7 years.	Lp(a) ≥30 mg/dl: 9.4% (females: 7.4%, males: 11.4%)	Mean (SD): 13.1 mg/dL (19.8) Median (IQR): 5.6 mg/dl (2.4-13.5)
Guardamagna et al. (Italy, 2011)[14]	231	Children and adolescents aged 2 to 18 years with familial dyslipidemia.	>85th percentile: 21% (with FH) and 9% (without FH)	Median (IQR) With FH: 35.1 mg/dL (1-302) Without FH: 31.9 mg/dL (1-156)
Giussani et al. (Italy, 2024)[15]	195	Children and adolescents (mean age 11.5 years) with other risk factors.	Lp(a) >75 nmol/L: 21.6%	Median (IQR): 22 nmol/L (7.8-68.6)
Stürzebechery col. (Germany, 2024)[16]	512	Healthy children and adolescents aged 5 to 18 years.	Lp(a) 30-50 mg/dL: 11.5% Lp(a) ≥50 mg/dL: 12.5%	Median (IQR): 9.7 mg/dl (4-28.3)
Glowinska et al. (Poland, 2003)[17]	285	Children and adolescents aged 6 to 20 years) with other risk factors.		Mean (SD): 30 mg/dL (34)
Thomas et al. (Wales, 2009)[18]	208	Healthy children aged 12 to 13 years	Lp(a) ≥30 mg/dL: 26%	
Johansen et al. (Norway, 2024)[19]	386	Children and adolescents (mean age 13.8 years) with HFHe.	Lp(a) ≥50 mg/dL or ≥75 nmol/L: 20.3%	Median (IQR): 9.7 mg/dl (4-28.3)
Wang et al. (US, 2005)[20]	975	Healthy children and adolescents aged 5 to 19 years Cherokee community.		Mean* Males: ages 5–9, 8.3 mg/dL; 10-19 years old, 12.1 mg/dL. Women: 5-9 years old, 9.8 mg/dL; 10-19 years, 14.7 mg/dl. Median* Men: 5-9 years, 4 mg/dL; 10-19 years, 5 mg/dL. Women: 5-9 years, 5 mg/dL; 10-19 years, 6 mg/dL.
Obisesan et al. (US, 2004)[21]	3,585	Healthy children and adolescents aged 4 to 19 years.	Lp(a) ≥30 mg/dL: Black, 54.4%; White, 20.4%.	Median* White: 4-5 years, 7 mg/dL; 6-11 years old, 12 mg/dl; 12-15 years old, 10 mg/dL; 16-19 years old, 9 mg/dL. Black race: 4-5 years, 31 mg/dl; 6-11 years, 32 mg/dL; 12-15 years, 33 mg/dL; 16-19 years, 31 mg/dL.
Foster et al. (US, 2021)[22]	700	Children and adolescents aged 12 to 19 years with diabetes.		Median (IQR): 8 (5-12) mg/dL
Alsaeid et al. (Kuwait, 1998)[23]	103	Healthy children aged 2 and 156 months of age.	Lp(a) ≥30 mg/dL: 8.7%	Mean (SD): 14.4 mg/dL (14.2) Median: 9.5 mg/dl*
Chu et al. (Taiwan, 2000)[24]	1,283	Children and adolescents aged 12 to 16 years.		Means Boys: 16.8 mg/dL; girls: 20.8 mg/dL* Median Men: 8.8 mg/dL; women: 11.9 mg/dL*
Gannagé-Yared et al. (Lebanon, 2020)[25]	961	Healthy children and adolescents aged 8 to 18 years.	Lp(a) >75 nmol/L: 14.4%	Median (IQR): 20 (10-50) nmol/L
Choi et al. (South Korea, 2022)[26]	416	Healthy children and adolescents (median age 11.1 years).	Lp(a) >100 nmol/L: 11.3%	Median (IQR): 21.5 nmol/L (8.2-51.7)
Bornaun et al. (Turkey, 2017)[27]	45	Children and adolescents aged 6 to 18 with and without FH of premature cardiovascular disease.		Means (SD) FH: 12.2 mg/dL
Cândido et al. (Brazil, 2021)[28]	320	Healthy children and adolescents.	Lp(a) ≥30 mg/dL: 43.8%	Mean (SD): 33.7 mg/dL (27.6)

FH: family history; SD: standard deviation; HFHe: heterozygous familial hypercholesterolemia; Lp(a): lipoprotein (a); IQR: interquartile range.
**Dispersion values not reported.*

difference between sexes, and an asymmetric distribution was observed (mean: 33.7 mg/dL; median: 25.5 mg/dL).[28]

As shown, the studies reviewed exhibit marked diversity across population groups (age range, healthy children, children with primary dyslipidemia, children with risk factors such as diabetes, family history, etc.). In addition, wide variability in the mean and median values of Lp(a) is observed in different geographic regions. These differences could reflect both genetic factors, linked to the inheritance of the *LPA* gene, and ethnic and/or environmental influences that modulate its expression. Methodological heterogeneity in measurement techniques, units, and cut-off points across cohorts could contribute to the observed dispersion. In this context, it is necessary to harmonize quantification methods and establish specific reference ranges for age and population, thereby facilitating clinical interpretation in the pediatric population.

VARIABILITY IN THE PEDIATRIC POPULATION AND COMPARISON WITH ADULTS

In adults, given that concentrations are mainly genetically determined, significant intra-individual variability is not expected. However, some recent studies suggest that Lp(a) levels may vary, especially in patients with intermediate levels.[29,30]

In children, data is scarcer. Lp(a) levels are low at birth and increase significantly between 0 and 7 days after delivery, continuing to rise until 180 days.[31] The studies conducted in Germany and South Korea, mentioned above, also analyzed this aspect.[16,26] In the first case, measurements were repeated in 154 children (follow-up from 1 to 4 years), and it was shown that levels remained stable in 94%. In the second study, 122 children repeated the measurement (with an average follow-up of 6.7 months); 5.7% showed an increase of ≥100 nmol/L. The small percentage of patients who did not maintain stable Lp(a) values suggests that, in most cases, routine repeat testing would not be necessary.

Puberty appears to influence Lp(a) values. Chen *et al*.[32] analyzed 314 pairs of same-sex Chinese twins aged 5 to 18 years and observed that values increased after the onset of puberty and were significantly higher in girls. However, the magnitude of the total variation observed in lipid levels decreased after puberty. From ages 10 to 12, total cholesterol (TC) and LDL-C levels decrease by 5% to 10% in both sexes (more evident in males) and reach average adult levels from age 20 onwards.[33] It is important to note that Lp(a) is a fraction of LDL-C; this small study invites reflection on the most appropriate time to measure Lp(a) in the pediatric population, given that hormonal changes could alter the values. However, the available evidence remains limited and warrants further investigation.

A study in the Netherlands analyzed Lp(a) variation in 2740 children compared to adults.[34] From the age of 8, the mean increased by 22% in those who reached adulthood without receiving lipid-lowering drugs, while in patients medicated with statins (n = 418) and those who additionally used ezetimibe (n = 65), Lp(a) increased by 43% and 9%, respectively, with an intra-individual variation of 70%. This study provides additional information by comparing populations (pediatric and adult) with the use of lipid-lowering treatments (especially statins) that could modify baseline Lp(a) values.

PROGNOSTIC MARKER IN PEDIATRICS RELATED TO ATHEROSCLEROSIS SUBROGATES

Lapinleimu et al. studied 193 11-year-old children who had followed a dietary counseling program to reduce cholesterol levels since early childhood and compared them with a control group (n = 198). They assessed endothelial dysfunction using ultrasound to measure flow-mediated dilation (FMD) in the brachial artery.[35] In the control group, FMD showed an inverse association with Lp(a) concentration (p = 0.007), while no such association was observed in the intervention group (p >0.5). The authors concluded that elevated concentrations were associated with impaired endothelial function, a condition that could be mitigated by early lifestyle intervention.

One study compared carotid intima-media thickness and pulse wave velocity in 27 healthy children (mean age 9.9 years) with Lp(a) levels >30 mg/dL without other lipid abnormalities, compared with 27 controls. Individuals with elevated levels did not have altered vascular indices compared to controls.[36] Another study analyzed 200 children aged 8 to 18 years (mean: 13 years) with heterozygous familial hypercholesterolemia over 20 years of follow-up.[37] Higher Lp(a) levels were significantly associated with greater progression of carotid intima-media thickness (adjusted β = 0.0073 mm per 50 nmol/L of Lp(a); p = 0.017). However, this association

was not confirmed in the analysis of the 88 siblings without overt disease.[38]

PROGNOSTIC MARKER FOR CLINICAL EVENTS

A meta-analysis (4 studies included) reported a significant association between elevated Lp(a) values and the risk of ischemic stroke (OR: 6.27; 95% CI: 4.52-8.69).[39] Another systematic review, which included 341 pediatric patients and 729 controls, reported the same association with Lp(a) levels >30 mg/dL (OR: 4.24; 95% CI: 2.94-6.11).[40]

A case-control study analyzed 12 newborns and 23 children, with a mean age of 6.2 (between 1 month and 18 years), who had suffered a stroke and found that Lp(a) levels >50 mg/dl were more frequent in comparison with controls (21.7% vs. 3.2%, $p = 0.02$). A significant association was reported for every 10 mg/dL increase in Lp(a) with the risk of stroke (adjusted OR: 1.36; 95% CI: 1.02-1.82; $p = 0.041$).[41]

The Cardiovascular Risk in Young Finns Study revealed that Lp(a) levels measured in individuals aged 9 to 24 years were associated with a higher incidence of cardiovascular events in adulthood (47-year follow-up).[42,43] The age- and sex-adjusted hazard ratios for fatal and non-fatal events were 1.96 (95% CI: 1.35-2.57) and 1.25 (95% CI: 1.03-1.47), respectively, considering a cutoff point for Lp(a) >30 mg/dL. The results observed in this study were replicated in the Bogalusa Heart Study database. In this case, in an age- and sex-adjusted model, subjects aged 8 to 17 years exposed to elevated Lp(a) levels had a 2.5-fold increased risk of developing cardiovascular disease compared to unexposed individuals.[43]

RECOMMENDATIONS FOR MEASUREMENT

To date, there is no universal recommendation for requesting Lp(a) testing in pediatrics. The European Atherosclerosis Society recommends screening in young people with stroke, a family history of premature cardiovascular disease, or high Lp(a) without other identifiable risk factors.[5] A recent report by the National Lipid Association (NLA) suggests that Lp(a) testing could be performed from the age of 5 years to estimate the risk of developing cardiovascular disease and aortic stenosis throughout life, as part of a universal screening program.[44] This could be done simultaneously with the traditional lipid profile between the ages of 6 and 11, as suggested by the Consensus of the Sociedad Argentina de Pediatría for the management of dyslipidemia.[45] Early identification could be especially useful in families with a history of premature cardiovascular disease.

Although cardiovascular disease manifests clinically in adulthood, the underlying atherosclerotic process is already evident during the first decade of life. Therefore, prevention should begin at a very early stage. The questions that arise are whether pediatric Lp(a) testing is useful, cost-effective, and appropriate for screening all children, and at what age, or whether it should be reserved only for individuals at particularly high risk.[46,47] The limited evidence available today makes it difficult to provide definitive answers. The questions relate to the absence of a specific treatment capable of reducing Lp(a), the risk of causing anxiety in families or inducing excessive dietary restrictions in young children, uncertainty regarding risk thresholds based on Lp(a) values in different ethnic groups, and, finally, the lack of evidence on the course of action to take once children with high Lp(a) values reach adulthood.

Some authors propose that including Lp(a) testing in the panel of pediatric cardiometabolic risk factors could be clinically relevant.[48] This proposal is based mainly on the fact that early identification of individual risk would allow for the implementation of early and potentially more effective preventive strategies. However, other experts believe that this assessment should focus on children and adolescents with additional risk factors, especially those with a family history (*Table 3*).[47] The discussion remains open, and the available evidence continues to expand.

CURRENT TREATMENT, PERSPECTIVES, AND CONCLUSIONS

Currently, there are no specific treatments available to reduce Lp(a) levels. Several ongoing studies are evaluating different therapies: antisense oligonucleotides or small interfering RNAs that selectively bind to the mRNA encoding Apo(a), blocking its synthesis and reducing its levels.[49] It will be necessary to wait for results in adults and to conduct further research in pediatric populations.

While we await new studies and guidelines for pediatrics, the current recommendation is to address modifiable risk factors to compensate for the elevated risk. Maintaining a balanced diet, maintaining a healthy weight, engaging in physical activity, and avoiding smoking are essential

Table 3. Key concepts regarding the clinical applicability of lipoprotein (a) measurement

When to order Lp(a)?	Clinically suspected or genetically confirmed FHC. First-degree relatives with history of premature ASCVD (age <55 years in men and <65 years in women). Ischemic stroke of unknown cause. First-degree relatives with elevated Lp(a).
What is its purpose?	It requires starting a cascade screening of immediate family members. It helps identify children and adolescents with increased atherogenic risk. It allows us to understand the importance of properly managing other risk factors during childhood and ensures continuity of follow-up into adulthood.
What should be done when Lp(a) is elevated?	Monitor LDL-C levels and other associated and modifiable risk factors, such as overweight or blood pressure. Encourage adherence to a balanced, heart-healthy diet and regular physical activity from childhood onwards. Avoid smoking.

ASCVD: atherosclerotic cardiovascular disease; FHC: familial hypercholesterolemia; Lp(a): lipoprotein (a).

measures. Until effective and safe therapeutic tools are available to reduce Lp(a) levels, efforts should focus on controlling and treating classic risk factors, primarily adequate LDL-C levels. ■

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