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CURRENT ADVANCES IN LIPOPROTEIN(A) MANAGEMENT: CLINICAL SIGNIFICANCE AND EMERGING THERAPIES

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ABSTRACT

Background: Lipoprotein(a) [Lp(a)] is an independent, genetically determined risk factor for atherosclerotic cardiovascular disease (ASCVD) and aortic stenosis. Despite its clinical importance, Lp(a) remains largely unaffected by traditional lipid-lowering therapies, contributing to significant residual cardiovascular risk.
Aim: This review aims to synthesize current knowledge regarding the clinical significance of elevated lipoprotein(a) levels and to explore the landscape of emerging therapeutic strategies designed to specifically address this challenging cardiovascular risk factor.
Materials and Methods: An extensive synthesis of the current literature, prioritizing comprehensive review articles and meta-analyses published within the last five years, was conducted using multiple electronic databases (PubMed, Scopus, Google Scholar).
Results: Epidemiological data indicate that 20–30% of the global population has elevated Lp(a) levels (>30–50 mg/dL). While lifestyle and statins show negligible effects on Lp(a) concentration, novel nucleic acid-based therapies - specifically antisense oligonucleotides (pelacarsen) and small interfering RNAs (olpasiran, lepodisiran, zerlasiran) have demonstrated the ability to reduce Lp(a) levels by 70% to over 95%. Furthermore, oral small-molecule inhibitors like muvalaplin offer promising alternatives by disrupting the assembly of the Lp(a) particle.
Conclusion: The emergence of targeted RNA-based and small-molecule therapies represents a paradigm shift in cardiovascular prevention. As phase 3 clinical trials progress, these interventions may provide the first definitive means of mitigating the residual risk associated with elevated Lp(a).

KEYWORDS

Lipoprotein(a), Cardiovascular Disease, Cardiovascular Risk, RNA Therapeutics, Pelacarsen, Olpasiran, Muvalaplin

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1. Introduction

Cardiovascular disease continues to be a leading cause of mortality globally, despite significant advancements in diagnostic tools and therapeutic interventions (Tsimikas, 2017). A significant residual risk of atherosclerotic cardiovascular disease persists, even among patients receiving optimal conventional treatments, highlighting the need for novel therapeutic strategies (Reyes-Soffer et al., 2021). Among the myriad risk factors, lipoprotein(a) has emerged as an increasingly recognized, yet often undertreated, independent genetic risk factor for cardiovascular disease (Fujino et al., 2024). Its strong causal association with atherosclerotic cardiovascular disease and aortic valve stenosis underscores the critical importance of understanding its pathophysiology and developing targeted interventions (Kronenberg et al., 2022).

2. Methodology

This review is based on an exhaustive analysis of scientific publications retrieved from multiple electronic databases: PubMed, PubMed Central, Google Scholar, and Scopus. To ensure the provision of the most current and evidence-based knowledge on the topic, the analysis primarily focused on comprehensive review articles and meta-analyses published within the last five years.

2.1. Inclusion criteria: The following types of scientific literature were considered for inclusion:

- Articles focusing on the epidemiology, pathophysiology, and pharmacological management of elevated Lp(a).
- Publications retrieved from the primary databases: PubMed, PubMed Central, Google Scholar, and Scopus.

- High-level evidence, including comprehensive review articles, meta-analyses, and clinical practice guidelines from major international scientific societies.
- Studies published between 2020 and 2025 to ensure the currency of the evidence.

2.2. Exclusion criteria: The following types of literature were generally excluded:

- Primary research papers (e.g., small cohort studies, case reports) unless they provided unique, highly relevant data on novel diagnostic or therapeutic modalities not yet covered in systematic reviews.
- Publications not peer-reviewed or were not retrieved from the predetermined academic databases.
- Non-English language articles, unless they provided foundational data critical to the topic.
- Literature published before 2020, unless considered essential for historical context or foundational knowledge.

2.3. AI

Artificial intelligence tools were employed in this study solely as assistive instruments under human supervision. They were used for translation of selected sections of the manuscript, refinement of academic English (grammar,style,and clarity), and to improve efficiency in data processing. At no stage did AI replace human judgment - all data interpretation, classification, and formulation of conclusions were performed exclusively by the authors.

3. Results

3.1 Epidemiology

Dyslipidemia, broadly characterized by abnormal plasma lipid levels including elevated total cholesterol, low-density lipoprotein cholesterol, and triglycerides, affects approximately 30–40% of the adult population globally, with significant regional and sex-based differences (Ballena-Caicedo et al., 2025). The global prevalence of elevated lipoprotein(a) levels exhibits considerable variation across different populations and ethnic groups, underscoring the influence of both genetic predisposition and environmental factors on its distribution (Wang et al., 2024). Specifically, elevated Lp(a) levels, often defined as above 30 mg/dL or 75 nmol/L, are found in 20–30% of the population, establishing it as a common, yet frequently unrecognized, genetic risk factor for cardiovascular disease (Kronenberg et al., 2022). This high prevalence, combined with its strong causal association with atherosclerotic cardiovascular disease and aortic valve stenosis, positions elevated Lp(a) as a significant public health challenge.

3.2 Genetic Determinants of Lp(a) Levels

The genetic basis of dyslipidemias, including elevated Lp(a), is increasingly understood to involve both monogenic and polygenic contributions, necessitating advanced genetic research and bioinformatics for comprehensive risk assessment (Kalwick & Roth, 2025; Lee, 2021). Approximately 70% to 90% of the interindividual variability in Lp(a) levels is genetically determined, primarily dictated by the *LPA* locus. The two major protein components of Lp(a) particles are apolipoprotein B-100 and apolipoprotein(a) (Reyes-Soffer et al., 2021).

This strong genetic influence means that, unlike other modifiable risk factors, Lp(a) levels are largely unaffected by lifestyle modifications or conventional lipid-lowering therapies such as statins (Nissen et al., 2023). This predisposition highlights the critical need for genetic testing, especially when traditional clinical diagnoses are ambiguous due to unclear family histories (Lee, 2021). The concentration of Lp(a) is predominantly determined by genetic variability at the *LPA* locus, with levels ranging widely and influencing the plasma pool of apolipoprotein B (apoB) (Kronenberg et al., 2022). Intriguingly, Lp(a) concentrations are independent of conventional dietary patterns and lifestyle interventions, distinguishing it from other lipid parameters (Thau et al., 2024). Adult Lp(a) levels typically stabilize by the age of five years, underscoring the early onset and lifelong persistence of this risk factor (Kozárová et al., 2023).

3.3 Pathophysiological Mechanisms of Lipoprotein(a) in Atherosclerosis

The relationship between Lp(a) and atherosclerosis is mediated by its pro-atherogenic, pro-inflammatory, and pro-thrombotic properties. Structurally, Lp(a) consists of an LDL-like particle with apoB-100 disulfide-bonded to apolipoprotein(a) (Koutsogianni et al., 2022). Its structural homology with plasminogen allows it to interfere with fibrinolysis and promote a procoagulant state (Corte et al., 2023). Furthermore, the apo(a) component exacerbates cardiovascular risk by impairing fibrinolysis and promoting a prothrombotic environment (Noothi et al., 2023).

Lp(a)’s pro-inflammatory effects are primarily mediated by its capacity to transport oxidized phospholipids (OxPL), which activate inflammatory pathways within the endothelium and facilitate the recruitment of immune cells to atherosclerotic plaques (Kronenberg et al., 2022). These OxPLs are recognized

as endogenous danger-associated molecular patterns, triggering sterile inflammation and calcification, thus contributing to the pathogenesis of both ASCVD and calcific aortic valve disease (Reyes-Soffer et al., 2021).

3.4 Current Guidelines and Screening Strategies

Professional medical organizations have established guidelines for screening, though recommendations remain heterogeneous. Despite the evidence linking Lp(a) to cardiovascular risk, there is no universal consensus on the definition of an "abnormal" concentration, largely due to ethnic variations (O'Donoghue et al., 2022). Nevertheless, the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) advocate for at least a one-time measurement of Lp(a) in all adults to identify high-risk individuals (Szarek et al., 2020; Mach et al., 2025). Clinical significance is typically noted at levels above 30 mg/dL (or 75 nmol/L), with markedly increased risk at levels exceeding 50 mg/dL (Šuran et al., 2022).

3.5 Risk Stratification and Clinical Actionability

While genetic predisposition dictates Lp(a) levels, the associated cardiovascular risk is modulated by traditional risk factors (Kronenberg et al., 2022). A comprehensive assessment integrating both Lp(a) and established metrics is essential for accurate management (Koschinsky et al., 2024). Recent studies suggest that individuals with elevated Lp(a) but favorable cardiovascular health metrics exhibit lower incident CVD risk compared to those with similar Lp(a) elevations and poor health metrics (Gary et al., 2023).Lp(a) can also be very useful in identifying patients who will benefit from acetylsalicylic acid [ASA] as a primary prevention strategy, especially given that its levels exhibit a curvilinear relationship with myocardial infarction and ischemic stroke risk, with a more linear association observed in primary prevention settings (Nicholls, 2024).

3.6 Pharmacological Approaches to Lipoprotein(a) Reduction

Conventional therapies have demonstrated limited efficacy in lowering Lp(a). However, nucleic acid-based interventions have recently shown substantial promise (Nissen et al., 2024). These therapies, including antisense oligonucleotides (ASOs) and small interfering RNAs (siRNAs), target the hepatic synthesis of apolipoprotein(a) by degrading the *LPA* messenger RNA (Nissen et al., 2022).

- Pelacarsen: An ASO that inhibits apolipoprotein(a) translation. Phase 2 trials demonstrated mean decreases of 72% to 80% (Kronenberg et al., 2022; Reyes-Soffer et al., 2021).
- Olpasiran and Zerlasiran: These siRNA therapies have shown potent and sustained reductions, with some studies reporting decreases of 71% to 98% that persist for months after a single dose (Krychtiuk et al., 2022; Nissen et al., 2024).
- Lepodisiran: An siRNA that has achieved dose-dependent reductions exceeding 90% (Nissen et al., 2023).
- Muvalaplin: A novel oral small-molecule inhibitor that prevents the association of apo(a) with apoB, disrupting the assembly of the Lp(a) particle (Cui et al., 2025; Saniewski et al., 2025).

3.7 Non-Pharmacological Interventions

Lifestyle modifications, while vital for overall health, have a negligible impact on Lp(a) levels (Wierzbowska et al., 2024). Dietary interventions typically result in only minimal changes (10–15%), which are clinically insufficient compared to the 80–95% reductions achieved by emerging pharmacological agents (Kronenberg et al., 2022).

4. Discussion

The clinical recognition of Lp(a) as a key driver of residual cardiovascular risk has transformed the therapeutic landscape of lipidology. As demonstrated in this review, the structural unique properties of Lp(a) - specifically its apo(a) component - confer a triple threat of atherogenesis, thrombosis, and inflammation that is largely resistant to conventional statin therapy. The most significant finding from current literature is the unprecedented efficacy of nucleic acid-based therapies. While earlier attempts at Lp(a) reduction using niacin or apheresis were limited by side effects or logistical burdens, agents like pelacarsen and olpasiran offer a high-potency, targeted approach with the potential for infrequent dosing. However, a critical question remains: does the massive biochemical reduction in Lp(a) translate directly into a proportional reduction in cardiovascular events? Ongoing phase 3 trials are expected to provide this answer. Furthermore, the development of muvalaplin addresses the need for oral alternatives, which could significantly improve long-term patient compliance. Until these therapies become standard clinical practice, the management of individuals with elevated Lp(a) should focus on aggressive control of all other modifiable risk factors to minimize total ASCVD burden.

5. Conclusions

Lipoprotein(a) is a highly prevalent, genetically determined risk factor that contributes significantly to the global burden of cardiovascular disease. This review highlights that traditional lifestyle and pharmacological interventions are insufficient for meaningful Lp(a) reduction. However, the emergence of RNA-targeting therapies (ASOs and siRNAs) and novel oral inhibitors like muvalaplin represents a major advancement, capable of reducing Lp(a) levels by up to 98%. These therapies offer hope for mitigating residual risk in patients who remain at high risk despite optimal LDL-C management. Future clinical practice should integrate universal Lp(a) screening to identify at-risk populations and facilitate the transition toward personalized, targeted cardiovascular prevention.

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