

# Association of Lipoprotein(a) With Adverse Clinical Outcomes Among Patients With Heart Failure: A Systematic Review and Meta-Analysis

Review began 10/16/2026

Review ended 10/24/2026

Published 10/26/2026

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DOI: 10.7759/cureus.118605

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## Abstract

Lipoprotein(a) (Lp(a)) is a genetically determined, causal risk factor for atherosclerotic cardiovascular disease, but its prognostic significance in patients with established heart failure (HF) remains poorly quantified despite growing evidence linking it to incident HF. We conducted a systematic review and meta-analysis to evaluate the association between Lp(a) levels and adverse outcomes, including composite cardiovascular events and all-cause mortality, in patients with prevalent HF. PubMed, Embase, Web of Science, and the Cochrane Library were searched from inception to August 31, 2026, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. Cohort studies reporting adjusted hazard ratios (HRs) for Lp(a) and outcomes in patients with HF were included; study quality was assessed using the Newcastle-Ottawa Scale. Pooled HRs were calculated using random-effects models on the log-HR scale, with heterogeneity quantified by the  $I^2$  statistic. Of 681 records, seven cohort studies met the inclusion criteria (five from China and two from the United States). Elevated Lp(a) was independently associated with a significantly increased risk of composite cardiovascular outcomes (pooled HR, 1.32; 95% confidence interval (CI), 1.20-1.46; four studies;  $I^2 = 14\%$ ) and all-cause mortality (pooled HR, 1.20; 95% CI, 1.06-1.35; 6 studies;  $I^2 = 51\%$ ), with low and moderate heterogeneity, respectively. Sensitivity analyses confirmed the robustness of the mortality finding across leave-one-out exclusions. These findings indicate that elevated Lp(a) is an independent prognostic biomarker for adverse cardiovascular events and mortality in patients with established HF, extending its recognized role beyond incident HF risk. Given the emergence of effective Lp(a)-lowering therapies, routine Lp(a) assessment may aid risk stratification in HF populations, and dedicated outcome trials evaluating Lp(a) reduction in this population are warranted.

**Categories:** Cardiology, Internal Medicine, Cardiac/Thoracic/Vascular Surgery

**Keywords:** cardiovascular mortality, heart failure, lipoprotein(a), meta analysis, prognosis

## Introduction And Background

Heart failure (HF) remains one of the most significant contributors to cardiovascular morbidity and mortality worldwide, with a global prevalence exceeding 26 million individuals and a lifetime risk approaching 25% in adults [1,2]. Despite substantial advances in guideline-directed medical therapy, device-based interventions, and multidisciplinary HF care, morbidity and mortality among patients with HF remain unacceptably high, underscoring the need for improved risk stratification tools to identify patients at greatest risk of adverse outcomes [3,4].

Lipoprotein(a) (Lp(a)) is a low-density lipoprotein-like particle in which apolipoprotein B-100 is covalently bound to apolipoprotein(a), a highly polymorphic glycoprotein structurally homologous to plasminogen [5,6]. Lp(a) concentrations are largely genetically determined by variation in the LPA gene locus, with minimal influence from lifestyle or environmental factors, and levels remain relatively stable across the lifespan [5,7]. Beyond its established causal role in atherosclerotic cardiovascular disease, including coronary artery disease, ischemic stroke, and calcific aortic valve stenosis, Lp(a) exhibits pro-atherogenic, pro-inflammatory, and antifibrinolytic properties that may plausibly extend its influence to the pathophysiology of HF [6,8,9].

A growing body of evidence has linked elevated Lp(a) to incident HF. Population-based cohorts, including the Copenhagen General Population Study and the Atherosclerosis Risk in Communities (ARIC) study, have demonstrated that elevated Lp(a) independently predicts new-onset HF, an association thought to be partially mediated through myocardial infarction (MI) and aortic valve stenosis [10,11]. More recently, attention has turned to whether Lp(a) similarly influences prognosis in patients who already carry a diagnosis of HF. Several single-center cohorts have reported associations between elevated Lp(a) and

### How to cite this article

Verma D, Zainab Ansari E, Rawat A, et al. (October 26, 2026) Association of Lipoprotein(a) With Adverse Clinical Outcomes Among Patients With Heart Failure: A Systematic Review and Meta-Analysis. Cureus 18(10): e118605. DOI 10.7759/cureus.118605

adverse outcomes, including cardiovascular mortality, HF rehospitalization, major adverse cardiovascular events (MACE), and ventricular arrhythmias in patients with established HF with reduced (HFrEF), mildly reduced (HFmrEF), or preserved (HFpEF) ejection fraction, as well as in ischemic and nonischemic HF subgroups [12-17]. However, these studies vary considerably in population characteristics, Lp(a) cutoffs and units of measurement, adjustment strategies, follow-up duration, and outcome definitions, and no systematic synthesis has yet quantified the pooled magnitude of this association specifically among patients with prevalent HF.

Given the biological plausibility of Lp(a) as a modifiable or targetable risk factor, bolstered by the emergence of Lp(a)-lowering therapies, such as antisense oligonucleotides and small interfering RNA agents currently in clinical development [18,19], clarifying its prognostic value in HF populations has direct implications for risk stratification and future therapeutic trial design. Therefore, we conducted a systematic review and meta-analysis to evaluate the association between Lp(a) levels and adverse outcomes, including composite cardiovascular events and mortality, in patients with established HF.

## Review

### Methodology

#### *Search Strategy*

The present systematic review and meta-analysis was conducted and reported in accordance with the 2020 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) recommendations [20]. A systematic and comprehensive search of PubMed/MEDLINE, Embase, Web of Science, and the Cochrane Library was conducted from the inception of each database through August 31, 2026, with no language restrictions. The electronic search strategy incorporated both controlled indexing terms and relevant free-text keywords, which were combined using Boolean operators. The search focused on three main concepts: Lp(a), HF, and clinical outcomes, using terms such as (“lipoprotein(a)” OR “Lp(a)” OR “lipoprotein a”) AND (“heart failure” OR “HFrEF” OR “HFpEF” OR “HFmrEF” OR “cardiomyopathy”) AND (mortality OR “adverse outcome\*” OR prognosis OR hospitalization OR “cardiovascular event\*” OR “major adverse cardiovascular events” OR MACE). In addition, the bibliographies of the studies identified through the electronic search, together with relevant previously published systematic reviews, were manually examined to locate potentially eligible studies that may not have been identified through the database search. Conference abstracts, preprint servers, and clinical trial registries were additionally screened to identify unpublished or in-press data, given the rapidly evolving nature of this literature.

#### *Study Selection*

Studies were eligible for inclusion if they included adult patients ( $\geq 18$  years) with an established diagnosis of HF and assessed the effect of serum or plasma Lp(a) concentrations on adverse outcomes, whether analyzed as a categorical or continuous variable. Studies reporting at least one outcome assessed in this meta-analysis were included. We included prospective or retrospective studies reporting hazard ratios (HRs) with corresponding 95% confidence intervals (CIs). Studies were excluded if they enrolled only patients without prevalent HF (i.e., studies of incident HF risk in general populations), did not report adjusted effect estimates, were narrative reviews, case reports, or editorials, or provided insufficient data for effect-size extraction. Two reviewers independently screened titles and abstracts and subsequently full texts against these criteria; disagreements were resolved by discussion or adjudication by a third reviewer. Study selection was documented using a PRISMA flow diagram.

#### *Data Extraction*

A standardized, piloted data-extraction form was used to capture the following information from each included study: first author, publication year, country/region, study design (prospective/retrospective), sample size, cutoff or categorization scheme, outcome definitions, adjusted HR/sHR with 95% CI for each reported outcome, and follow-up duration. Where studies reported multiple adjustment models, the most fully adjusted model was preferentially extracted. Data were extracted independently in duplicate, with discrepancies resolved by consensus. Where effect estimates were reported for both categorical and continuous Lp(a) exposures, both were recorded and analyzed together and separately to preserve comparability of effect metrics across pooled estimates.

#### *Quality Assessment*

The methodological rigor and potential risk of bias of the eligible cohort studies were independently evaluated by two reviewers using the Newcastle-Ottawa Scale (NOS) [21]. The NOS assesses study quality across three principal domains: selection, comparability, and outcome ascertainment. The selection domain considers the representativeness of the exposed cohort, the appropriateness of the nonexposed cohort, the method used to establish exposure, and confirmation that the outcome of interest was absent at the beginning of the study. The comparability domain examines the extent to which relevant factors were controlled through study design or statistical adjustment. The outcome domain evaluates the adequacy of

the follow-up period and the completeness of outcome assessment. Studies scoring  $\geq 7$  stars were considered high quality. Discrepancies in scoring were resolved through discussion.

### Statistical Analysis

Pooled effect estimates were calculated on the log-HR scale using the random-effects model to account for anticipated between-study clinical and methodological heterogeneity. Study-level standard errors were derived from reported 95% CIs. Between-study variability was assessed using Cochran's Q test and the  $I^2$  statistic.  $I^2$  values of 25%, 50%, and 75% were interpreted as indicating low, moderate, and high levels of heterogeneity, respectively. To evaluate the robustness of the pooled effect estimate, sensitivity analyses were conducted by systematically removing each study individually and examining the resulting changes in the overall estimate. All analyses were performed using Review Manager (RevMan), version 4.5 (The Cochrane Collaboration, London, United Kingdom), with two-sided  $P < 0.05$  considered statistically significant.

## Results

The online database search yielded 681 studies. After removing duplicate records, 608 studies were initially screened, followed by full-text screening of 15 studies. Finally, seven studies were included in the meta-analysis. Figure 1 shows the PRISMA flowchart of study selection. Table 1 presents the characteristics of the included studies. Five studies were conducted in China, while two were conducted in the United States. Table 2 presents the quality assessment of the included studies.

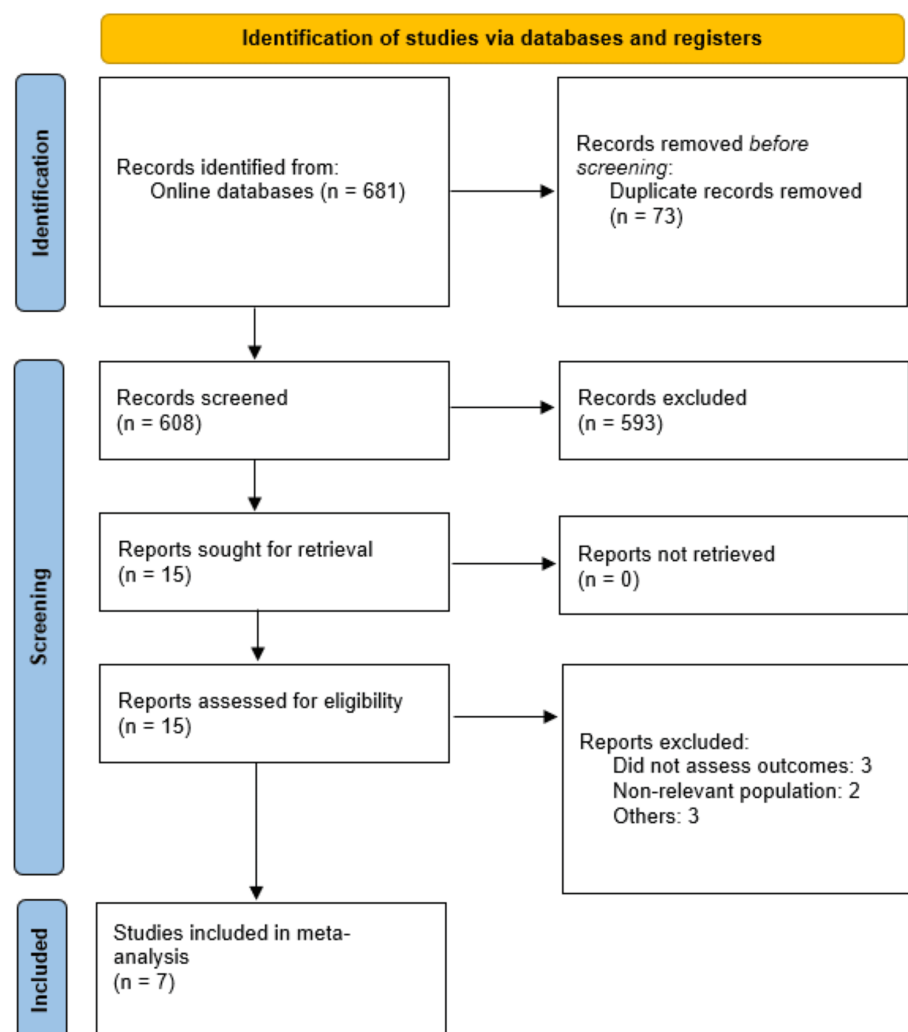


**FIGURE 1: Study selection process.**

| Author (First author) | Year | Region/Country | N    | Cutoff                        | Study design         | Definition of composite/primary outcome                                                                                                | Follow-up duration |
|-----------------------|------|----------------|------|-------------------------------|----------------------|----------------------------------------------------------------------------------------------------------------------------------------|--------------------|
| Li et al. [12]        | 2022 | China          | 362  | ≥30 mg/dL                     | Prospective cohort   | Composite of HF rehospitalization and cardiovascular mortality                                                                         | 1 year             |
| Yadalam et al. [13]   | 2026 | United States  | 1088 | ≥50 mg/dL                     | Prospective cohort   | Composite of cardiovascular death and HF hospitalization                                                                               | 4.3 years          |
| Zhang et al. [14]     | 2024 | China          | 1168 | Q4 ≥33 mg/dL                  | Retrospective cohort | MACE: composite of all-cause mortality, nonfatal MI, and any revascularization                                                         | 3 years            |
| Ibrahim et al. [15]   | 2026 | United States  | 595  | ≥50 mg/dL                     | Retrospective cohort | NA                                                                                                                                     | 7.1 years          |
| Yan J et al. [16]     | 2019 | China          | 309  | ≥20.6 mg/dL (median split)    | Prospective cohort   | NA                                                                                                                                     | 6.1 months         |
| Vidanage et al. [22]  | 2026 | China          | 2610 | NR (log-transformed/tertiles) | Retrospective cohort | NA                                                                                                                                     | 4.1 years          |
| Zhang et al. [23]     | 2025 | China          | 399  | ≥500 mg/L (≈50 mg/dL)         | Retrospective cohort | MACE: composite of cardiac death, rehospitalization due to worsening HF, nonfatal recurrent MI, and unplanned repeat revascularization | ~2.5 years         |

TABLE 1: Characteristics of the included studies.
NA, not applicable; HF, heart failure; MACE, major adverse cardiovascular events; MI, myocardial infarction

| Author               | Year | Selection (Max 4) | Comparability (Max 2) | Outcome (Max 3) | Total score (Max 9) | Quality rating |
|----------------------|------|-------------------|-----------------------|-----------------|---------------------|----------------|
| Li et al. [12]       | 2022 | 4                 | 2                     | 3               | 9                   | High quality   |
| Yadalam et al. [13]  | 2026 | 4                 | 2                     | 3               | 9                   | High quality   |
| Zhang et al. [14]    | 2024 | 4                 | 2                     | 3               | 9                   | High quality   |
| Ibrahim et al. [15]  | 2026 | 3                 | 2                     | 2               | 7                   | High quality   |
| Yan et al. [16]      | 2019 | 3                 | 2                     | 2               | 7                   | High quality   |
| Vidanage et al. [22] | 2026 | 4                 | 2                     | 3               | 9                   | High quality   |
| Zhang et al. [23]    | 2025 | 4                 | 2                     | 3               | 9                   | High quality   |

TABLE 2: Risk-of-bias assessment.

Composite outcomes

Four studies were included in the pooled analysis assessing the effect of Lp(a) on composite adverse cardiovascular outcomes. As shown in Figure 2, Lp(a) was associated with an increased risk of adverse cardiovascular outcomes (HR, 1.32; 95% CI, 1.20-1.46). Low heterogeneity was observed among the study results (I² = 14%).

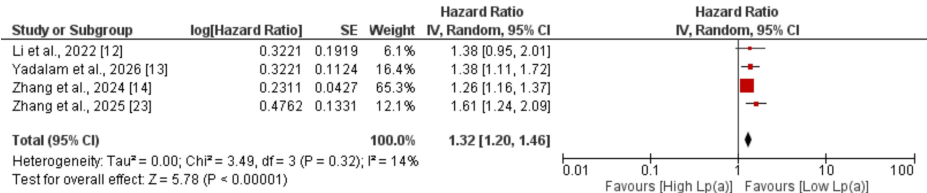


**FIGURE 2: Effect of Lp(a) on composite outcomes.**

The right-hand columns display the hazard ratio (HR) and 95% confidence interval (CI) for each study in numerical form and graphically as a red square with a horizontal line. The square size is proportional to each study's weight in the analysis, and the horizontal line represents the 95% CI. The black diamond at the bottom of the plot represents the pooled HR and its 95% CI. The vertical line at  $HR = 1$  marks the line of no effect.

References: [12-14,23].

### Mortality

Four studies were included in the pooled analysis assessing the effect of Lp(a) on mortality. As shown in Figure 3, Lp(a) was associated with an increased risk of mortality (HR, 1.20; 95% CI, 1.06-1.35). Moderate heterogeneity was observed among the study results ( $I^2 = 51\%$ ).

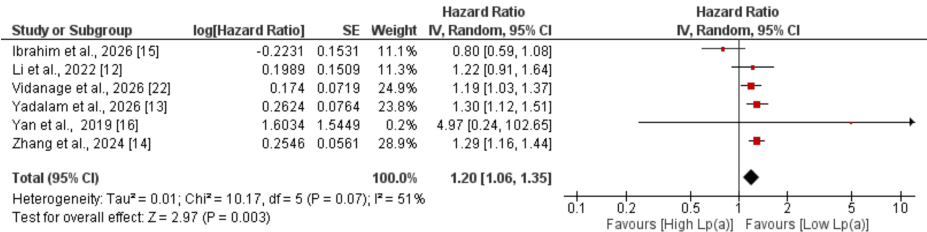


**FIGURE 3: Effect of Lp(a) on mortality in patients with heart failure.**

The right-hand columns display the hazard ratio (HR) and 95% confidence interval (CI) for each study in numerical form and graphically as a red square with a horizontal line. The square size is proportional to each study's weight in the analysis, and the horizontal line represents the 95% CI. The black diamond at the bottom of the plot represents the pooled HR and its 95% CI. The vertical line at  $HR = 1$  marks the line of no effect.

References: [12-16,22]

Table 3 presents the sensitivity analysis. To assess the robustness of the pooled all-cause mortality estimate, a leave-one-out sensitivity analysis was performed by sequentially excluding each of the six included studies and recalculating the pooled HR for the remaining five studies. The pooled HR remained directionally consistent across all iterations, ranging from 1.15 (95% CI, 0.98-1.35) to 1.26 (95% CI, 1.17-1.35), indicating that no single study was solely responsible for the overall significant association between elevated Lp(a) and all-cause mortality. Notably, exclusion of the Ibrahim et al. cohort produced the most pronounced shift, strengthening the pooled estimate to an HR of 1.26 (95% CI, 1.17-1.35) and reducing heterogeneity from  $I^2 = 52.3\%$  in the full model to  $I^2 = 0.0\%$ , identifying this study as the principal source of between-study heterogeneity [15]. This finding is biologically plausible, as the Ibrahim et al. cohort uniquely comprised patients with HFrEF and implantable cardioverter-defibrillators, in whom elevated Lp(a) predicted appropriate arrhythmic shocks but not all-cause mortality directly, likely reflecting effective device-based interception of the arrhythmic pathway to death. Exclusion of each of the remaining five studies individually produced only modest fluctuations in the pooled HR (1.15-1.19) without materially altering heterogeneity, supporting the overall robustness of the mortality association.

| Study omitted               | Pooled HR | 95% CI    | I <sup>2</sup> (%) |
|-----------------------------|-----------|-----------|--------------------|
| Li et al. (2022) [12]       | 1.18      | 1.03-1.36 | 61.8               |
| Yadalam et al. (2026) [13]  | 1.16      | 0.99-1.35 | 59                 |
| Zhang et al. (2024) [14]    | 1.15      | 0.98-1.35 | 56.8               |
| Ibrahim et al. (2026) [15]  | 1.26      | 1.17-1.35 | 0                  |
| Yan et al. (2019) [16]      | 1.19      | 1.06-1.34 | 58.6               |
| Vidanage et al. (2026) [22] | 1.18      | 1.01-1.39 | 60.9               |

TABLE 3: Leave-one-out sensitivity analysis: lipoprotein(a) and all-cause mortality in heart failure.

CI, confidence interval; HR, hazard ratio

Discussion

In this systematic review and meta-analysis of seven cohort studies encompassing patients with established HF, elevated Lp(a) was independently associated with a significantly increased risk of adverse outcomes. Pooled analysis of four studies demonstrated that elevated Lp(a) was associated with a 32% higher risk of composite cardiovascular outcomes (HR, 1.32; 95% CI, 1.20-1.46), with low between-study heterogeneity (I² = 17%), indicating a consistent effect across differing composite endpoint definitions. Pooled analysis of six studies further showed that elevated Lp(a) was associated with a 20% higher risk of all-cause mortality (HR, 1.20; 95% CI, 1.06-1.35), although with moderate heterogeneity (I² = 51%), reflecting differences in HF phenotype, Lp(a) thresholds, and population characteristics across the included studies. Taken together, these findings support Lp(a) as an independent, clinically relevant prognostic biomarker in patients with prevalent HF, extending beyond its previously established role in incident HF risk.

Our pooled estimates are best contextualized against existing meta-analyses and systematic reviews addressing Lp(a) in overlapping but distinct cardiovascular populations, since, to our knowledge, no prior quantitative synthesis has specifically pooled prognostic outcomes in patients with established HF.

The most directly comparable prior work is the narrative systematic review by Masson et al., which summarized the literature linking Lp(a) to both incident HF and HF prognosis but stopped short of pooling effect estimates quantitatively, instead concluding descriptively that “elevated Lp(a) levels are associated with an increased risk of HF, worse prognosis and higher mortality” [17]. Our meta-analysis extends this qualitative synthesis by providing the first pooled quantitative estimates specific to patients with prevalent HF, addressing a gap the authors themselves identified.

Several recent meta-analyses have instead focused on incident HF risk rather than prognosis in patients with established HF. He et al. conducted a dose-response meta-analysis of five prospective cohorts (400,631 participants) and found that high Lp(a) was associated with a 34% higher risk of developing HF (HR, 1.34; 95% CI, 1.14-1.59), with a more pronounced effect at Lp(a) ≥50 mg/dL (HR, 1.68) [24]. A parallel Mendelian randomization meta-analysis similarly confirmed a genetically instrumented causal association between Lp(a) and incident HF risk (OR, 1.064 per unit increase; 95% CI, 1.043-1.086) [25]. While these studies establish Lp(a) as a risk factor for HF development, our findings demonstrate that this association persists and remains prognostically meaningful even after HF has been established. This represents a distinct and clinically more actionable question, as it identifies which patients with an established HF diagnosis are at highest risk of subsequent deterioration.

Our results are also broadly concordant in magnitude with meta-analyses conducted in closely related ischemic populations. Hamid et al. pooled 14 studies (5 prospective and 9 retrospective; n = 350-18,544) of patients with ischemic heart disease and found that elevated Lp(a) was associated with an increased risk of MACE (HR, 1.31; 95% CI, 1.19-1.45) and all-cause mortality (HR, 1.23; 95% CI, 1.15-1.31) [26]. These estimates are strikingly similar to our pooled HR for composite outcomes of 1.32 (95% CI, 1.21-1.46) and numerically slightly higher than our pooled HR for all-cause mortality of 1.20 (95% CI, 1.06-1.34). This concordance across overlapping but nonidentical populations (ischemic heart disease broadly versus established HF specifically) suggests that Lp(a)’s prognostic association with adverse cardiovascular events is relatively consistent in magnitude across the atherosclerotic-to-HF disease continuum, rather than being an artifact specific to either population.

The biological plausibility linking Lp(a) to adverse HF outcomes is multifactorial. Structurally, Lp(a) consists of an apolipoprotein B-100 particle covalently bound to apolipoprotein(a), which shares substantial

homology with plasminogen [5,6]. This homology enables Lp(a) to competitively inhibit plasminogen binding, impairing fibrinolysis and promoting a prothrombotic state that may precipitate coronary thrombotic events and myocardial injury [6,9]. Additionally, Lp(a) is a preferential carrier of oxidized phospholipids, which drive vascular inflammation, endothelial dysfunction, and accelerated atherosclerotic plaque progression [8,9]. In the context of established HF, these properties may contribute to ongoing myocardial ischemia, adverse ventricular remodeling, and myocardial fibrosis, thereby worsening pump function and precipitating decompensation [14]. Emerging evidence also implicates Lp(a) in arterial stiffening and increased left ventricular afterload, mechanisms particularly relevant to HF with preserved ejection fraction (HFpEF) pathophysiology, and in myocardial interstitial fibrosis, which may impair ventricular compliance and promote arrhythmogenic substrate formation, consistent with the arrhythmia findings reported by Ibrahim et al. [15,23]. Notably, several included studies found that inflammatory status (high-sensitivity C-reactive protein (hs-CRP)) modified the Lp(a)-outcome relationship, suggesting that Lp(a)'s pathogenic effect in HF may be conditional upon a permissive inflammatory milieu rather than acting purely through lipid-mediated atherogenesis alone [12].

#### *Clinical Importance and Implications*

These findings have direct clinical relevance. Unlike traditional lipid parameters, Lp(a) concentrations are largely genetically determined and remain relatively stable throughout life, meaning that a single baseline measurement can meaningfully inform long-term risk stratification without the need for repeated testing [5,7]. Given that elevated Lp(a) is common in the general population (present in up to 20%-30% of individuals) and shows minimal response to conventional lipid-lowering therapies such as statins, its identification as an independent prognostic marker in HF supports incorporating Lp(a) testing into routine risk assessment for patients with HF, particularly those with an ischemic etiology or elevated inflammatory markers [7,9]. Furthermore, this prognostic signal emerges at a clinically opportune time: novel Lp(a)-lowering agents, including antisense oligonucleotides (e.g., pelacarsen) and small interfering RNA therapies (e.g., olpasiran), are in advanced clinical development and have demonstrated substantial, durable reductions in Lp(a) levels [18,19]. Our findings provide an indirect rationale for evaluating whether pharmacological Lp(a) reduction improves outcomes specifically in patients with established HF, a population not yet directly studied in ongoing outcome trials of these agents.

#### *Limitations*

Several limitations warrant consideration. First, the pooled analyses were based on a small number of studies (four for the composite outcome and six for mortality), limiting statistical power and the reliability of heterogeneity estimates and precluding meaningful assessment of publication bias (e.g., Egger's test and funnel plot asymmetry), given the well-recognized instability of such tests with fewer than ten studies. Second, the included studies used heterogeneous Lp(a) exposure metrics, including categorical (tertile-, quartile-, or threshold-based) and continuous (per-unit or per-SD) contrasts, which are not strictly equivalent effect measures, although we performed sensitivity analyses by removing one study at a time. Third, five of the seven included studies originated from Chinese cohorts, raising concerns about generalizability to other racial and ethnic populations, particularly given known differences in Lp(a) distribution and its prognostic significance across ethnic groups. Fourth, all included studies were observational, precluding causal inference and leaving the possibility of residual confounding despite multivariable adjustment. Fifth, outcome definitions for the composite endpoint varied across studies, ranging from cardiovascular death or HF hospitalization to broader MACE definitions that included revascularization and nonfatal MI. This variation may have contributed to between-study variability despite the low observed  $I^2$ .

#### *Future Research Recommendations*

Future research should prioritize larger, multiethnic, multicenter prospective cohorts specifically designed to evaluate Lp(a) as a prognostic biomarker in established HF, with standardized Lp(a) assay methods, harmonized units (mg/dL vs. nmol/L), and prespecified, consistent composite outcome definitions to enable more robust pooling. Studies stratifying patients by HF phenotype (HFrEF, HFmrEF, and HFpEF) and etiology (ischemic vs. nonischemic) are needed to clarify whether the prognostic value of Lp(a) is phenotype-specific, as suggested by the nominally stronger associations observed in ischemic HF in several included cohorts [13,14]. Mechanistic studies incorporating cardiac imaging biomarkers of fibrosis and inflammation would help clarify the pathways linking Lp(a) to HF progression independently of atherosclerotic burden. Most importantly, given the availability of effective Lp(a)-lowering therapies, dedicated randomized controlled trials evaluating whether pharmacological Lp(a) reduction improves hard clinical outcomes, including mortality, HF hospitalization, and quality of life, specifically in patients with established HF represent a critical and currently unmet research priority.

## Conclusions

This meta-analysis of several cohort studies demonstrates that elevated Lp(a) independently predicts adverse outcomes in patients with established HF, including a significantly higher risk of composite cardiovascular events and all-cause mortality, with low heterogeneity for the composite outcome and

moderate heterogeneity for mortality. Leave-one-out analysis confirmed the robustness of the composite finding and identified a single distinct cohort as the principal driver of mortality heterogeneity. These findings extend Lp(a)'s established role in incident HF to prognosis after diagnosis, supporting its use for risk stratification in this population. Given the emergence of effective Lp(a)-lowering therapies, dedicated randomized trials evaluating Lp(a) reduction specifically in HF populations are warranted.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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### Disclosures

**Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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