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STATE-OF-THE-ART REVIEW

Lipoprotein(a) and Women's Cardiovascular Health

A Review

Erin D. Michos, MD, MHS,[a,*] Stephanie Saucier, MD,[b,*] Roxana Mehran, MD,[c] Marlys L. Koschinsky, PhD[d]

ABSTRACT

Elevated lipoprotein(a), or Lp(a), is a genetically determined risk factor for cardiovascular disease (CVD); however, knowledge relating to Lp(a) levels and contribution to CVD risk in women (defined herein as female sex at birth) is evolving and often conflicting. Female sex hormones are important modulators of lipoprotein metabolism, and Lp(a) levels are influenced by exogenous and endogenous estrogen levels. Thus, lifetime fluctuations of Lp(a) are observed, primarily during pregnancy and postmenopause. Although approved pharmacological Lp(a)-lowering therapies are not yet available, elevated Lp(a) is actionable now and strategies can be undertaken to reduce overall CVD risk. It is imperative that knowledge of the risks associated with elevated Lp(a) levels in women is appropriately conveyed to health care professionals to ensure optimal management of CVD risk in real-world practice. Moreover, further research in the field of Lp(a) and women's cardiovascular health is vital for the future of CVD prevention. (JACC Adv. 2026;■:102744)

Within the United States, cardiovascular (CV) disease (CVD) is the leading cause of mortality, accounting for 439,724 deaths in women in 2021.[1] Women are at an increased relative risk of CVD mortality compared with men, in part due to sex-specific CVD pathophysiology and risk factors such as adverse pregnancy outcomes (APOs) and early menopause.[1,2] Elevated lipoprotein(a) (Lp[a]) is a genetically determined risk factor for CVD in men and women.[3] A patient-level meta-analysis of 7 statin outcomes trials provided evidence suggesting that Lp(a) levels are an independent CVD risk factor; a linear association between higher levels of Lp(a) and increased risk of CV events was observed even after adjustment for age, sex, prior CVD, diabetes, smoking, blood pressure, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol.[4] Furthermore, evidence from Mendelian randomization studies suggest that elevated Lp(a) is likely causal in the development of atherosclerotic CVD (ASCVD).[5]

Lp(a) is an apolipoprotein B-containing lipoprotein that has proatherogenic, proinflammatory, and possible prothrombotic effects.[5] Lp(a) levels above approximately 125 nmol/L (~50 mg/dL) occur in 20% to 30% of the global population[6] and are associated with an increased risk of ASCVD, including myocardial infarction and ischemic stroke. Recent guidance from the European Society of Cardiology (ESC)/European Atherosclerosis Society (EAS) defines

From the [a]Division of Cardiology, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA; [b]Heart and Vascular Institute, Hartford Hospital, Hartford, Connecticut, USA; [c]Zena and Michael A. Wiener Cardiovascular Institute, Icahn School of Medicine at Mount Sinai, New York, New York, USA; and the [d]Robarts Research Institute, Schulich School of Medicine and Dentistry, Western University, London, Ontario, Canada. *These authors are co-first authors.

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ABBREVIATIONS AND ACRONYMS

APO = adverse pregnancy outcome

apo(a) = apolipoprotein(a)

ASCVD = atherosclerotic cardiovascular disease

CHD = coronary heart disease

CV(D) = cardiovascular (disease)

EAS = European Atherosclerosis Society

HRT = hormone replacement therapy

LDL-C = low-density lipoprotein cholesterol

Lp(a) = lipoprotein(a)

NLA = National Lipid Association

PCOS = polycystic ovary syndrome

elevated Lp(a) levels as ≥50 mg/dL, with a corresponding molar threshold of ~105 nmol/L, reflecting the specific conversion factor applied.[7] Lp(a) levels are ~90% genetically determined,[5] and before approximately 50 years of age, remain mostly consistent and are similar between men and women.[3] However, there is evidence of intraindividual variability in Lp(a) levels measured at different time points (approximately 20%-25%), with women exhibiting greater variability than men.[5,8,9]

Lp(a) levels increase significantly in women at around 50 years (coinciding with the average age of menopause),[3,10] approximately double during pregnancy (returning to prepregnancy levels postpartum),[11,12] and are influenced by exogenously administered estrogen (Central Illustration).[10,13,14] In adults older than 50 years of age, Lp(a) levels can be ~17% higher in women than in men, and the prevalence of Lp(a) ≥50 mg/dL is higher in women older than 50 years of age (~20%) compared with those younger than 50 years (15%).[3,15] Overall, evidence characterizing the impact of changes in Lp(a) levels on CV risk in women is conflicting and often of limited quality,[16-18] and the lack of a uniform standardized cutoff value for Lp(a) risk presents an additional challenge in evaluating CVD risk in women.[19]

In this review, the role of elevated Lp(a) levels in women's CVD risk is discussed. Research surrounding Lp(a) and adverse CVD outcomes and recommendations for Lp(a) testing in women is summarized, and areas of future research and knowledge consolidation required to more fully address Lp(a) risk in women are identified.

Lp(a) IN WOMEN'S HEALTH AND CVD RISK

CVD RISK IN WOMEN. Sex (ie, to be biologically female) and sociocultural gender (ie, to identify as a woman) have complex and frequently intertwined impacts on CVD risk, CVD manifestation, health care utilization, and CV outcomes.[20,21] Multiple factors specific to women may contribute to CVD risk, including premature or late-onset menarche; endometriosis; polycystic ovary syndrome (PCOS); multiparity; use of assisted reproductive technology; miscarriage, gestational diabetes, pre-eclampsia, and other APOs; and premature menopause. In addition, women are more likely to have autoimmune and inflammatory conditions that increase CVD risk, such as systemic lupus erythematosus and rheumatoid arthritis.[2] Furthermore, the relative impact of traditional CVD risk factors, including diabetes, hypertension, obesity, and smoking, is greater in women than men.[22] Besides these established risk factors, there are under-recognized risks, including social, cultural, and economic factors, alongside a range of sex-specific biological influences, such as premature menopause and pregnancy, that may increase CVD risk in women.[23]

There are long-standing gender disparities in CV health care utilization, such that women are less likely to see a cardiologist[21] or to have access to timely and guideline-directed treatment,[24] and are under-represented in clinical research.[25] These disparities are amplified in non-White women,[25] and women from all racial or ethnic backgrounds are less likely to achieve lipid goals[26] and face increased mortality risk following acute CV events compared with men.[27] To address these disparities, several guidelines containing recommendations on the management of CVD in women have been released (Table 1).[2,17,28,29]

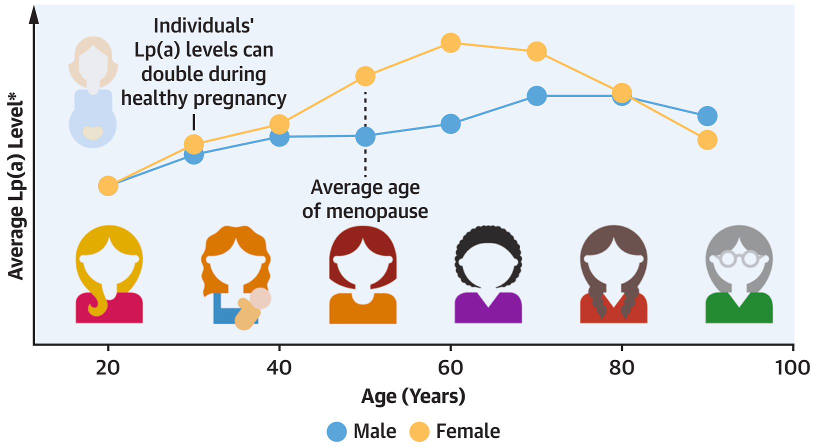
HIGHLIGHTS

- Female sex hormones modulate lipoprotein metabolism, and lifetime fluctuations of Lp(a) are observed in women.
- Evidence for the contribution of elevated Lp(a) to CV risk in women is evolving and conflicting.
- Routine testing for elevated Lp(a) in women is essential to guide clinical decision-making and reduce CV risk.
- The role of elevated Lp(a) levels in women's reproductive and CV health warrants further research.

Lp(a) AND SEX-RELATED CVD RISK. Although elevated Lp(a) is associated with similar overall CVD risk in men and women,[3,35] evidence suggests that the association with specific CV outcomes may differ.[36,37] For example, in 15,124 participants from the Atherosclerosis Risk in Communities cohort, elevated Lp(a) was associated with preclinical carotid atherosclerosis in men; in women, this association was only significant in the presence of CVD risk factors such as diabetes in Black women and smoking in White women.[36] A meta-analysis of 163,139 individuals showed that although elevated Lp(a) is associated with calcific aortic valve disease initiation and progression, the association is smaller for women compared with men.[38] The authors of this

CENTRAL ILLUSTRATION **Lipoprotein(a) Levels in Women Throughout Life and Remaining Evidence Gaps**

Lp(a) Levels Fluctuate in Women in Response to Pregnancy and Menopause



Completing the Puzzle: Areas Requiring Attention and Further Research

Lp(a) and Women's Health		Lp(a) and Pregnancy	Lp(a) and Menopause
Define how sex-specific increases in Lp(a) levels contribute to CV risk, especially in women across ethnic groups	Integrate reproductive aging into study designs	Characterize the role of Lp(a) in healthy pregnancy and risk of APOs	Clarify whether Lp(a) levels rising at menopause increases CVD risk, or if risk is driven by lifetime exposure
Assess changes in Lp(a) with estrogen therapy in transgender women, and with testosterone therapy in transgender men	Study CVD risk in women with elevated Lp(a), particularly those using exogenous estrogen or with additional risk factors	Examine the impact of race/ethnicity on Lp(a) in pregnancy and APO risk	Determine whether menopausal HRT reduces or worsens Lp(a)-associated CVD risk
Stratify Lp(a)-associated CVD risk studies in women by age, menopause/estrogen status, and ethnicity	Determine whether Lp(a) predicts CVD risk in women independently of LDL-C	Investigate links between Lp(a) and infertility	Elucidate relationships between menopause, Lp(a) levels, and age

Michos ED, et al. JACC Adv. 2026;■(■):102744.

*Lp(a) trends (originally reported in mg/dL) based on Roeters van Lennep JE, Tokgözoğlu LS, Badimon L, et al. Women, lipids, and atherosclerotic cardiovascular disease: a call to action from the European Atherosclerosis Society. *Eur Heart J*. 2023;44(39):4157–4173. https://doi.org/10.1093/eurheartj/ehad472. APO = adverse pregnancy outcome; CV = cardiovascular; CVD = cardiovascular disease; HRT = hormone replacement therapy; LDL-C = low-density lipoprotein cholesterol; Lp(a) = lipoprotein(a).

study noted that this variation may be partially attributable to sex-related differences in the underlying pathophysiology of aortic valve calcification and stenosis.[38]

In a prospective study of 27,939 initially healthy American women, high-sensitivity C-reactive protein, LDL-C, and Lp(a) were predictors of incident CV events over a 30-year period, with each biomarker providing additive information and indicating the greatest risk when all 3 biomarkers were elevated.[39]

In a case-controlled study of women hospitalized for coronary heart disease (CHD), Lp(a) >30 mg/dL (~70 nmol/L) was associated with a 5-fold risk of CHD compared with low Lp(a) levels in premenopausal women, but with a 2-fold risk among postmenopausal women, suggesting that the risk

TABLE 1 Recommendations for Lp(a) Testing[5,7,29-34]

Organization	Year	Recommendations
AHA/ACC/multisociety	2026	• Lp(a) measurement is recommended at least once in all adults for ASCVD risk assessment
ESC/EAS	2025	• Consider Lp(a) testing at least once in every adult's lifetime, either at the first lipid profile or at the next one if lipid profiles have previously been performed without Lp(a) measurement • Repeat Lp(a) testing is reasonable to consider in women after menopause, particularly if the premenopausal Lp(a) level was borderline
NLA	2024	• Lp(a) testing is recommended at least once in all adults to refine assessment of ASCVD risk • Repeat measurement of Lp(a) may be warranted for postmenopausal women with intermediate risk Lp(a) level
AHA	2022	• Test Lp(a) in individuals with a family or personal history of ASCVD and consider cascade testing in appropriate individuals • International standards for measurement of Lp(a) must be established to allow consistent measurement. When standardization is achieved, reassessment of general population-based screening should be considered
EAS	2022	• Test Lp(a) concentration at least once in adults • Cascade screening may be valuable in patients with either FH, premature ASCVD, or a history of very high or high Lp(a)
CCS	2021	• Lp(a) should be tested once in a person's lifetime as a part of initial lipid screening
AACE/ACE	2020	Lp(a) should be tested in the following populations: • All individuals with clinical ASCVD or a family history of premature ASCVD and/or elevated Lp(a) • People with South Asian or African ancestry • Individuals with a 10-year ASCVD risk ≥10% (primary prevention) • People with a personal or family history of AVS or those with refractory elevations of LDL-C levels despite treatment with aggressive LDL-C-lowering therapy
HEART UK	2019	Serum Lp(a) levels should be measured in individuals with: • A personal or family history of premature ASCVD (<60 years of age) • First-degree relatives with raised serum Lp(a) (>200 nmol/L) • Calcific aortic valve stenosis • FH or other genetic dyslipidemias • Borderline increased (<15%) 10-year risk of a cardiovascular event

AACE = American Association of Clinical Endocrinology; ACC = American College of Cardiology; ACE = American College of Endocrinology; AHA = American Heart Association; ASCVD = atherosclerotic cardiovascular disease; AVS = aortic valve stenosis; CCS = Canadian Cardiovascular Society; EAS = European Atherosclerosis Society; ESC = European Society of Cardiology; FH = familial hypercholesterolemia; LDL-C = low-density lipoprotein cholesterol; Lp(a) = lipoprotein(a); NLA = National Lipid Association.

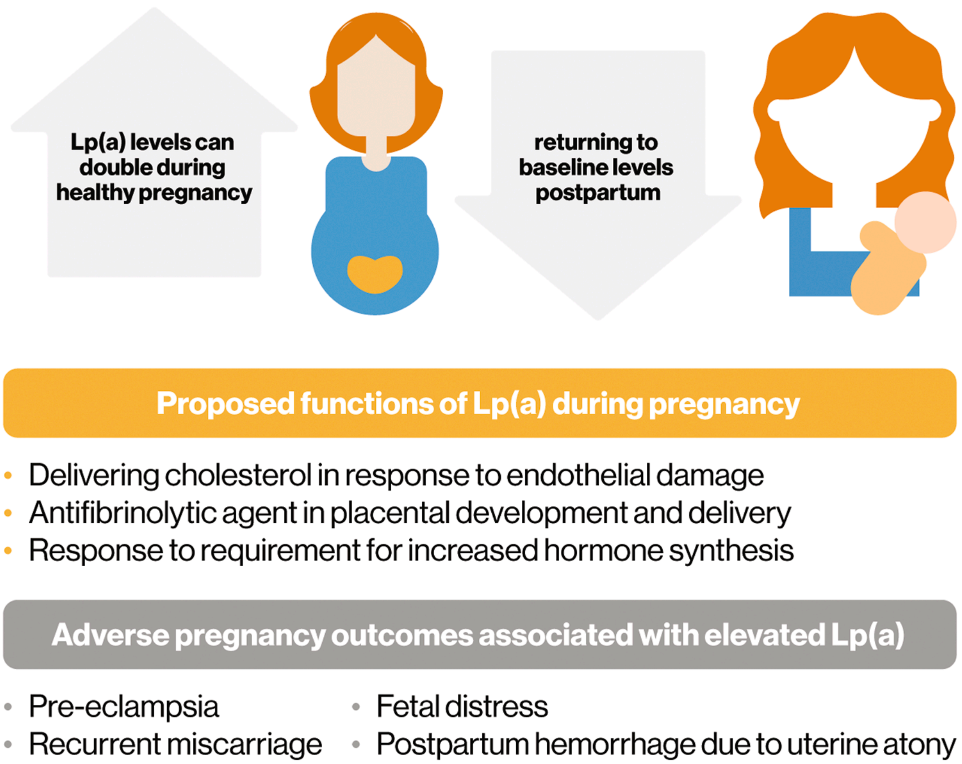
attributable to elevated Lp(a) may be greater among younger women.[40] In the Women's Health Study (WHS) of initially healthy individuals, using an adjusted analysis that accounted for total cholesterol levels, women in the highest quintile of Lp(a) had a 47% increased risk of a subsequent CVD event (HR: 1.47; 95% CI: 1.21-1.79), compared with women in the lowest quintile.[41] This suggests that the risk attributable to Lp(a) may be independent of total cholesterol. In the Nurses' Health Study, there was an interaction of Lp(a) with LDL-C, such that the relative risk of incident CVD was nearly 4-fold when both LDL-C (≥160 mg/dL) and Lp(a) (≥30 mg/dL) were elevated, but only 2-fold when Lp(a) alone was elevated, compared with women who had lower levels of both lipid parameters.[42]

In contrast, some data suggest that Lp(a) does not confer CVD risk in women with low total cholesterol[41,43] or that a risk association is unclear.[10,44] In the WHS analysis, it was observed that only women above the 90th percentile of Lp(a) levels were at an increased risk of major CV events and that associations were the strongest among women with high LDL-C.[41] A 2018 analysis examined the association of Lp(a) with incident CVD in women from 3 large cohorts (the WHS, the Women's Health Initiative [WHI], and the Justification for Use of Statins in Prevention [JUPITER] trial), concluding that in the primary prevention setting, Lp(a) is a CVD risk factor in women only when total cholesterol is elevated.[43] In addition, an analysis of the Framingham Heart Study concluded that increased Lp(a) was an independent risk factor for CHD in men but that the data were inconclusive in women.[10] However, the authors noted that their findings may be confounded by the relationship between estrogen status and Lp(a) levels.[10] Similarly, in a prospective study of 5,888 healthy elderly participants in the United States, elevated Lp(a) was reported to be an independent predictor of stroke, death from vascular disease, and death from any cause in men but not in women with or without estrogen use.[44]

Despite conflicting evidence regarding the role of Lp(a) in CV risk in women, the current view, supported by multiple medical societies, suggests that Lp(a) is an independent genetic risk factor across all populations.[32,34] Importantly, it is possible that measurement of Lp(a) levels in early studies was not accurate, as prolonged storage and repeated freeze-thawing of samples can affect the accuracy of measurement. Furthermore, measuring Lp(a) using methods that are not apolipoprotein(a) (apo[a]) isoform-independent can be inaccurate.[45]

Elevated Lp(a) levels are associated with multiple disease states that may affect CVD risk in women. In women with endometriosis, Lp(a) levels correlate

FIGURE 1 Changes in Lp(a) Levels During Pregnancy



The various functions of Lp(a) during pregnancy and the relationship between pregnancy-associated Lp(a) fluctuations and adverse pregnancy outcomes. Lp(a) = lipoprotein(a).

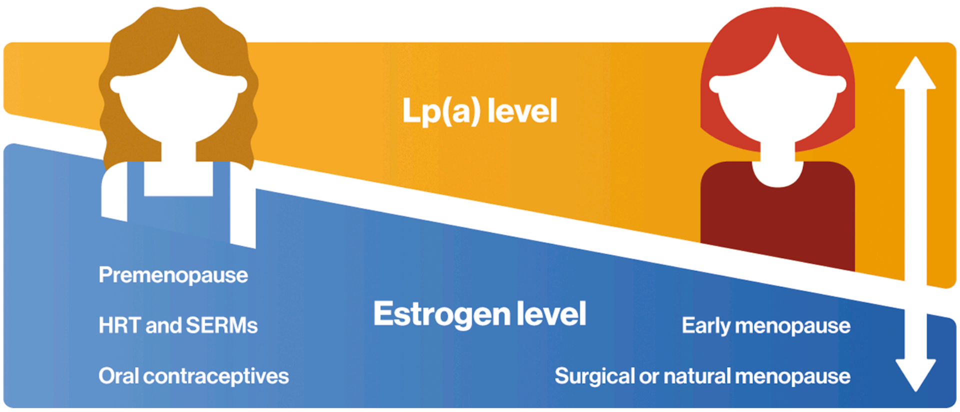
with the severity of disease.[46] In addition, studies report higher Lp(a) levels in women with PCOS,[47] which may be related to nongenetic modulators of Lp(a) levels, the proinflammatory state in PCOS, or alterations in hormone levels due to hyperandrogenism.[47] In the Nurses' Health Study, an interaction between Lp(a) and high-sensitivity C-reactive protein was identified, where CVD risk was greatest for women who had elevated levels of both biomarkers compared with elevation of either one of these factors.[42]

Lp(a) IN PREGNANCY. Serum lipids and lipoproteins (including Lp[a]) increase during pregnancy, with levels returning to baseline postpartum.[11,12] Although these increases alone are generally not considered atherogenic, many at-risk women are re-exposed to atherogenic lipid levels during their reproductive years due to avoidance of many lipid-lowering therapies in pregnancy and breastfeeding.[48] Notably, CV health during pregnancy and the presence of APOs can inform future CV risk for mother and child,[1,2] and women who have prepregnancy dyslipidemia are at a greater risk of an APO.[49]

The role of Lp(a) in healthy pregnancy is poorly characterized, despite Lp(a) levels doubling throughout (Figure 1).[16,17,33] Lp(a) levels are postulated to rise in pregnancy in response to increased endothelial dysfunction, which occurs in healthy pregnancy due to hyperlipidemia and/or hormonal changes.[50,51] This rise in Lp(a) levels may impair placental perfusion, which is implicated in the pathophysiology of pre-eclampsia.[12,16,50,52,53] Additional physiological roles for Lp(a) in pregnancy have also been proposed (Figure 1).[11,12,53]

Higher Lp(a) levels have been reported in women who experience APOs compared with healthy pregnant women; however, evidence for an association of Lp(a) with APOs remains conflicting.[16,50,53-63] Literature reviews conducted in 2005 and 2013 examined the association of Lp(a) with pre-eclampsia, intrauterine growth restriction, and recurrent miscarriage, noting that diverse results and study designs precluded firm conclusions, with conflicting data

FIGURE 2 Changes in Lp(a) Levels in Women in Response to Changes in Estrogen



How Lp(a) levels change in women due to fluctuating estrogen levels at different life stages and in response to exogenous factors. HRT = hormone replacement therapy; SERM = selective estrogen receptor modulator; other abbreviation as in Figure 1.

reported for all APOs studied.[16,53] However, several studies have since reported a link between elevated Lp(a) levels and pre-eclampsia in women across different ethnic groups.[50,54-60] A study of 360 women with placenta-mediated pregnancy complications reported that Lp(a) levels were elevated in women who experienced pre-eclampsia and stillbirth but not low birth weight.[54]

Most research to date supports an association between elevated Lp(a) levels and recurrent miscarriage.[16,54,61-63] One study has linked elevated Lp(a) levels with fetal distress and postpartum hemorrhage due to uterine atony,[55] although data on the relationship of elevated Lp(a) with gestational diabetes[55,59] and with intrauterine growth restriction[16,53] remain conflicting. Finally, elevated Lp(a) is also associated with diseases that may cause infertility, such as endometriosis and PCOS[46,47]; however, evidence for a direct association of Lp(a) with infertility is lacking. Notwithstanding Lp(a) elevation during pregnancy,[51] the relationship between elevated Lp(a) and APOs, which increase CVD risk, remains unclear. Future research should aim to distinguish between physiological elevations of Lp(a) during pregnancy and pathophysiological increases in Lp(a) that are associated with APOs and CVD risk.

Lp(a), MENOPAUSE, AND ESTROGEN

MENOPAUSE, ESTROGEN, AND CVD RISK. As women undergo menopause, the cardioprotective functions of estrogen are lost, leading to increases in total cholesterol, LDL-C, and triglyceride levels and a decrease in high-density lipoprotein cholesterol.[64] Premature (occurring before 40 years of age) and early (occurring between ages 40-45 years) menopause, both natural and surgical, have been shown to increase the relative risk of incident ASCVD and major CV events compared with similarly aged women who have not experienced premature menopause.[34,65,66]

MENOPAUSE AND Lp(a). Female sex hormones are major modulators of lipoprotein metabolism, and both endogenous and exogenous estrogens appear to modulate plasma Lp(a) levels.[67] Estrogen is an established negative regulator of the gene encoding apo(a) (*LPA*).[68] Significant increases in Lp(a) levels have been observed in postmenopausal women from diverse ethnic groups, which may be due to the associated decline in estrogen (Figure 2),[3,18,36,69] such that the overall prevalence of elevated Lp(a) increases in women aged >50 years compared with younger age groups.[3] In an analysis of 70,000 participants in the Copenhagen General Population Study, Lp(a) levels were, on average, 18% higher in women aged 50 to 59 years compared with those aged <50 years; in a subanalysis of women with premenopausal and postmenopausal Lp(a) measurements, levels were 27% higher postmenopause.[3]

Identifying discrete roles for menopause and aging on the modulation of Lp(a) levels has proven difficult.[18] A 2023 meta-analysis concluded that Lp(a) levels are significantly increased following menopause but that the effect of aging cannot be excluded.[18] However, in the Copenhagen General Population Study, only women experienced a significant increase in Lp(a) levels after the age of 50 years, but males did not, suggesting other factors beyond aging alone.[3] The impact of bilateral oophorectomy (surgical menopause) on Lp(a) levels offers valuable insight. Lp(a) levels significantly increase immediately following this procedure, with levels reduced to presurgical levels following initiation of estrogen hormone replacement therapy (HRT).[70,71] In several studies that reported no change in Lp(a) levels following bilateral oophorectomy, the induction of HRT could have masked the impact of endogenous estrogen deficiency on Lp(a).[72,73] Similarly, due to the inclusion of Lp(a) measurements from women who initiated HRT following oophorectomy, Anagnostis et al[18] concluded that bilateral oophorectomy does not affect Lp(a) levels, which is in contrast to 2 of the 3 studies reporting significantly increased Lp(a) levels following the procedure.[70-72]

EXOGENOUS ESTROGEN AND Lp(a). Considerable evidence supports a role for exogenous estrogen, such as oral contraceptives, HRT, and selective estrogen receptor modulators, in the fluctuation of Lp(a) levels (**Figure 2**).[18,74,75] Evidence from 24 randomized controlled trials demonstrated that HRT significantly reduced Lp(a) levels in postmenopausal women (mean relative difference: −20.4%) compared with those not receiving HRT.[76] A 2015 meta-analysis of 12 studies investigating the effect of the oral selective estrogen receptor modulator tibolone on Lp(a) levels in postmenopausal women reported a dose-dependent 25% reduction in circulating Lp(a) following treatment.[74] Estrogen-based oral contraceptives are also associated with decreases in Lp(a) levels[75] and may have a net benefit in reducing the incidence of CVD events.[77] Few studies have been conducted in transgender women receiving estrogen therapy (and in transgender men receiving testosterone therapy) to examine the impact of gender-affirming hormone therapy on Lp(a) levels.[78]

ARE ESTROGEN DEFICIENCY-ASSOCIATED ELEVATIONS IN Lp(a) LINKED TO INCREASED CVD RISK? Several analyses highlight a reduced correlation between Lp(a) levels and CV outcomes in women aged >50 years.[10,79] In a Japanese population undergoing coronary angiography, elevated Lp(a) was a risk factor for premature CHD in both men and women aged <55 years, and in men but not women aged 55 to 65 years.[79]

In contrast, many studies point to CV risk in postmenopausal women with elevated Lp(a) levels.[14,43,80] Simony et al[3] demonstrated that the morbidity and mortality risk associated with elevated Lp(a) is similar in women and men aged >50 years. In an analysis of postmenopausal women with hypercholesterolemia from the WHS and the WHI Observational Study, the risk of a 10-year first CVD event was 89% higher when Lp(a) level was >50 mg/dL (∼125 nmol/L) vs a reference group with Lp(a) <10 mg/dL.[43] Finally, in a study by Yan et al[80] including 783 postmenopausal women with a first episode of angina-like chest pain, Lp(a) was found to be an independent risk factor for predicting the presence and severity of new-onset CHD.

HRT use in postmenopausal women may confound the CV risk associated with elevated Lp(a). Furthermore, the question of whether HRT can reduce the CVD risk associated with elevated Lp(a) is controversial. The Heart and Estrogen/Progestin Replacement Study assessed the effects of HRT on the reduction of Lp(a) levels and CHD events in 2,763 postmenopausal women. The authors concluded that Lp(a) was an independent risk factor for recurrent CHD and that HRT lowered Lp(a) levels.[14] Reductions in Lp(a) level greater than -8.8 mg/dL significantly lowered the risk of CHD events compared with women who had smaller reductions in Lp(a).[14] An analysis of 27,736 women from the WHS found that among women not receiving HRT, elevated Lp(a) was significantly associated with CVD risk, whereas among women receiving HRT, there was no statistically significant association with risk.[13] In contrast, in the UK Biobank cohort, the opposite relationship was seen, with the risk of CHD associated with elevated Lp(a) being greater among HRT users compared with nonusers ($P = 0.04$).[81] Differences in patterns of HRT use over time may contribute to contrasting results between studies.

It is important to note that the use of HRT for the sole purpose of management of Lp(a) is not recommended by the National Lipid Association (NLA).[32] Although estrogen therapy can lower Lp(a) levels, the prothrombotic and proinflammatory effects of HRT may outweigh any benefits.

WHEN TO TEST FOR ELEVATED Lp(a)

Several medical societies recommend Lp(a) testing in individuals with a personal or family history of premature ASCVD and/or elevated Lp(a) (**Table 1**).[33,34] Notably, the NLA, the ESC/EAS, the American Heart Association/American College of Cardiology, and the Canadian Cardiovascular Society all recommend ≥1 measurement of Lp(a) in all adults, regardless of family history, to identify individuals at elevated CVD risk (**Table 1**).[7,29,32-34] However, the measurement of Lp(a) remains low in clinical practice. The global Lp(a)HERITAGE study reported that only 14% of patients with clinical ASCVD had their Lp(a) measured before enrollment in the study.[82] Furthermore, in the United States, the rate of Lp(a) testing has been reported to be <1%.[83] Importantly, knowledge of elevated Lp(a) levels can help to identify individuals at higher risk of adverse CV outcomes who may benefit from taking steps to lower risk, such as implementing a heart-healthy lifestyle, managing other CV risk factors including hypertension, and initiation of lipid-lowering therapies for more intensive LDL-C management. In the NLA's recent recommendation,[32] the authors advocate for universal Lp(a) screening in both men and women as part of the first lipid panel. In addition, cascade testing in relatives of patients found to have high Lp(a) is recommended.[5,33]

Specific guidance from medical societies on testing and management of Lp(a) levels in women is limited (**Table 1**). The EAS notes that current guideline recommendations, based on family history or a once-in-a-lifetime approach, may not capture the fluctuations in Lp(a) levels that occur in women throughout their lives, and that repeat testing may be necessary.[17] Women with low Lp(a) levels (ie, <75 nmol/L) premenopause are unlikely to exceed the risk-enhancing threshold of Lp(a) levels postmenopause (>125 nmol/L) and may not require additional Lp(a) measurements. However, in the 2024 statement from the NLA, it is recommended that for premenopausal women with Lp(a) levels in the intermediate risk category (75-125 nmol/L), remeasuring Lp(a) may be warranted after menopause.[32] The American Heart Association and EAS recommend additional CV monitoring for women reaching menopausal age;[17,84] the 2022 EAS statement incorporates Lp(a) measurement into this strategy for women with borderline premenopausal Lp(a) levels of 75 to 125 nmol/L to better stratify those at increased CVD risk and allow for timely intervention for treating modifiable risk factors.[17] Moreover, monitoring of Lp(a) levels in pregnant women may offer an early opportunity to identify individuals at an increased risk of APOs and implement preventive strategies, and young women with a history of APOs or PCOS may be considered candidates for Lp(a) testing.[47] If initial Lp(a) testing is performed during pregnancy and the level is noted to be elevated (>125 nmol/L), practitioners may consider repeat Lp(a) testing postpartum to identify the patient's baseline Lp(a) level.[85]

Although Lp(a) measurement reproducibility in commercial laboratories has improved substantially, and most contemporary assays show good correlation with reference methods, clinically relevant interassay variability persists, with reported differences ranging from 3% to 69% across individual samples.[86,87] This assay variability has important implications for clinical practice, particularly for risk stratification and longitudinal monitoring in individuals whose Lp(a) levels are in the intermediate risk range as outlined previously. Nevertheless, most modern assays demonstrate good correlation with reference methods,[86] and Lp(a) testing is strongly encouraged to facilitate risk stratification.

MANAGEMENT OF ELEVATED Lp(a) IN WOMEN

Although CV outcome trials of Lp(a)-targeted therapies, including pelacarsen, olpasiran, and lepodisiran, are underway,[88] approved pharmacological therapies for specifically and effectively lowering Lp(a) are not yet available. Nevertheless, elevated Lp(a) is actionable now, as strategies can be undertaken to reduce overall CVD risk.[89] Although elevated Lp(a) is present in 20% to 30% of the global population,[6] the majority of CVD events are preceded by suboptimal control of traditional risk factors,[90] underscoring the need for a holistic approach to CVD prevention. Guidelines recommend early, intensive management of modifiable risk factors such as smoking, diet, hypertension, diabetes, and LDL-C levels.[29,33,34] There are currently no sex-specific recommendations for lipid or Lp(a) management; therefore, patient education is needed to raise awareness of CVD risk factors in women,[2] including elevated Lp(a).

MULTIDISCIPLINARY MANAGEMENT OF CVD IN WOMEN. A multidisciplinary approach to CVD management in women is essential to help lower CVD-associated morbidity and mortality.[91] For instance, cardio-obstetrics involves collaboration between several disciplines, including cardiology, obstetrics/gynecology, maternal-fetal medicine, anesthesiology, and pharmacy, with the aim of optimizing maternal CVD outcomes.[91] In practice, gynecologists and obstetricians are at the forefront of patient management during pregnancy, delivery, and/or menopause, while primary care physicians are often the first point of contact and should be aware of CVD prevention strategies, particularly during menopause and after delivery.[92] Although a baseline lipid panel before pregnancy may be beneficial to identify women with severe lipid disorders, specific guidance for testing Lp(a) levels in women is limited. Ongoing communication between specialties is an important component of a holistic risk mitigation strategy. In line with this, a Delphi consensus panel of experts spanning cardiology, gynecology and obstetrics, and primary care, reported recommendations that may improve CVD management in women, including specific CVD risk follow-up in women with preterm labor, pre-eclampsia, and/or gestational hypertension.[92]

CONCLUSIONS

More work is needed to determine the role of elevated Lp(a) levels in women's reproductive and CV health. Potentially confounding factors such as race and ethnicity, apo(a) isoform distribution, reproductive age, and the use of HRT are not adequately characterized, or considered, in current research.[16,18,52,53,67] The authors provide a call to action on areas that require attention and areas for future research, which are summarized in the **Central Illustration**. Sex-specific, nongenetic influences on Lp(a) levels challenge the recommendation that one single lifetime measurement of Lp(a) is adequate in women;[16-18] however, evidence-based guidance on the management of increases in Lp(a) levels postmenopause is lacking. Although Lp(a) elevations above those considered to be normal during pregnancy are associated with APOs, data are conflicting, with little research into the underlying pathologic mechanisms. Nevertheless, it is crucial that the risks associated with elevated Lp(a) levels in women are appropriately recognized by health care professionals to ensure the effective management of CVD risk and optimal CVD prevention in clinical practice.

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ADDRESS FOR CORRESPONDENCE: Dr Marlys L. Koschinsky, Robarts Research Institute, Schulich School of Medicine and Dentistry, Western University, London, Ontario N6A 5B7, Canada. E-mail: mlk@robarts.ca. X handle: @MarlysLPA.

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