








American
Heart
Association®

Lipoprotein(a) & 2026 ACC/AHA Guideline on the Management of Dyslipidemia




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-  **Questions** – Click the Q&A button on the Zoom toolbar at the bottom of your screen to submit questions.

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Lipoprotein(a) Awareness Day 2026: What, Why, Who, When?

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Alexander Graham Bell Professor of Medicine
Director, Framingham Center for Population & Prevention Science
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Boston University Chobanian & Avedisian School of Medicine
Past President, American Heart Association

Boston University Chobanian & Avedisian School of Medicine



Disclosures

- Dr. Lloyd-Jones has no relationships with industry/conflicts of interest
 - Grant funding: NIH, CMS, AHA
- Dr. Lloyd-Jones has served as an unpaid fiduciary officer and director, and is currently a part-time employee, of the American Heart Association

Outline and Objectives

- What is Lp(a)?
- What does it do?
- Current prevention strategies
 - Lifestyle
 - LDL-C (and ApoB!) lowering
- Future prevention strategies?
 - Direct Lp(a) inhibition (stay tuned)
- Learning more about Lp(a)

2026 ACC/AHA/Multi-Specialty Dyslipidemia Guideline

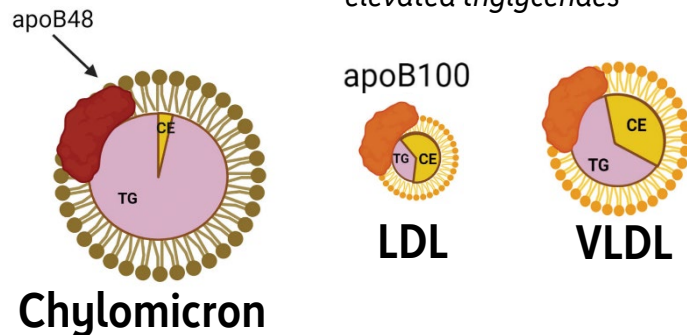
Measures all atherogenic particles
More accurate compared to LDL-C

Screening with Apolipoprotein B

In adults on LLT, particularly those with ASCVD, type 2 diabetes, and/or elevated TG, measurement of apoB is reasonable to guide decisions regarding further therapeutic intensification once LDL-C and/or Non-HDL-C goals are achieved. (2a)

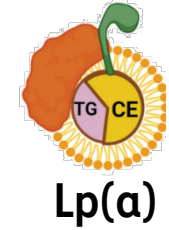
In adults not on LLT, measurement of apoB may be reasonable to enhance ASCVD risk assessment, guide initiation of LLT, and characterize inherited lipid disorders. (2b)

Particularly for secondary prevention, metabolic syndrome, diabetes, elevated triglycerides



New!

Screening with Lp(a)



Measurement of Lp(a) in all adults is recommended at least once for ASCVD risk assessment. (1)

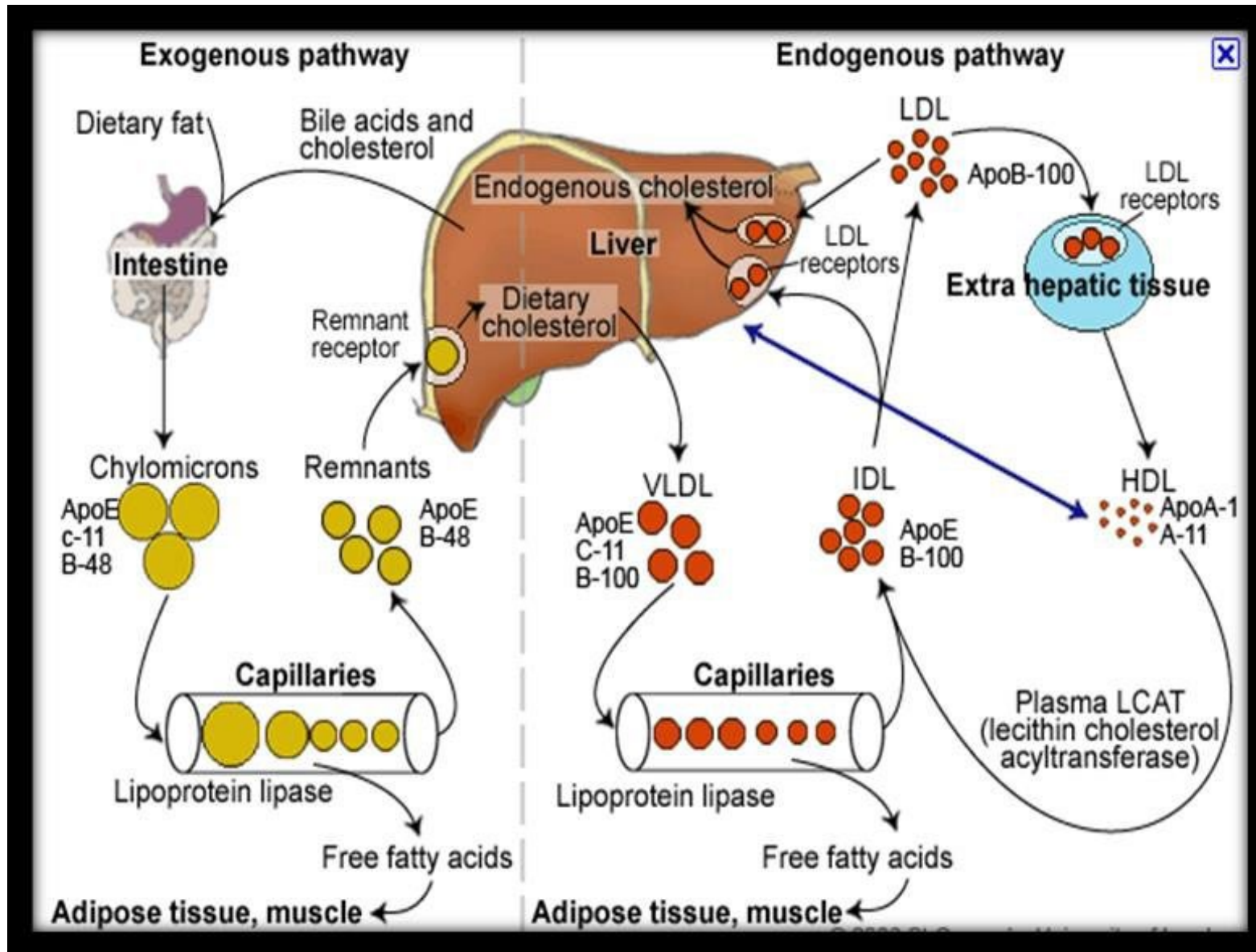
In those with FH, premature ASCVD, or high Lp(a), cascade testing of 1st-degree relatives is recommended. (1)

Lp (a) in nmol/L	ASCVD relative risk
450	4x
375	3x
250	2x
125	1.4x
75	Reference

Abbreviations: ApoB indicates apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; FH, familial hypercholesterolemia; LDL-C, low-density lipoprotein cholesterol; LLT, lipid-lowering treatment; Lp(a), lipoprotein(a); HDL-C, high-density lipoprotein cholesterol; and VLDL, very low-density lipoprotein.

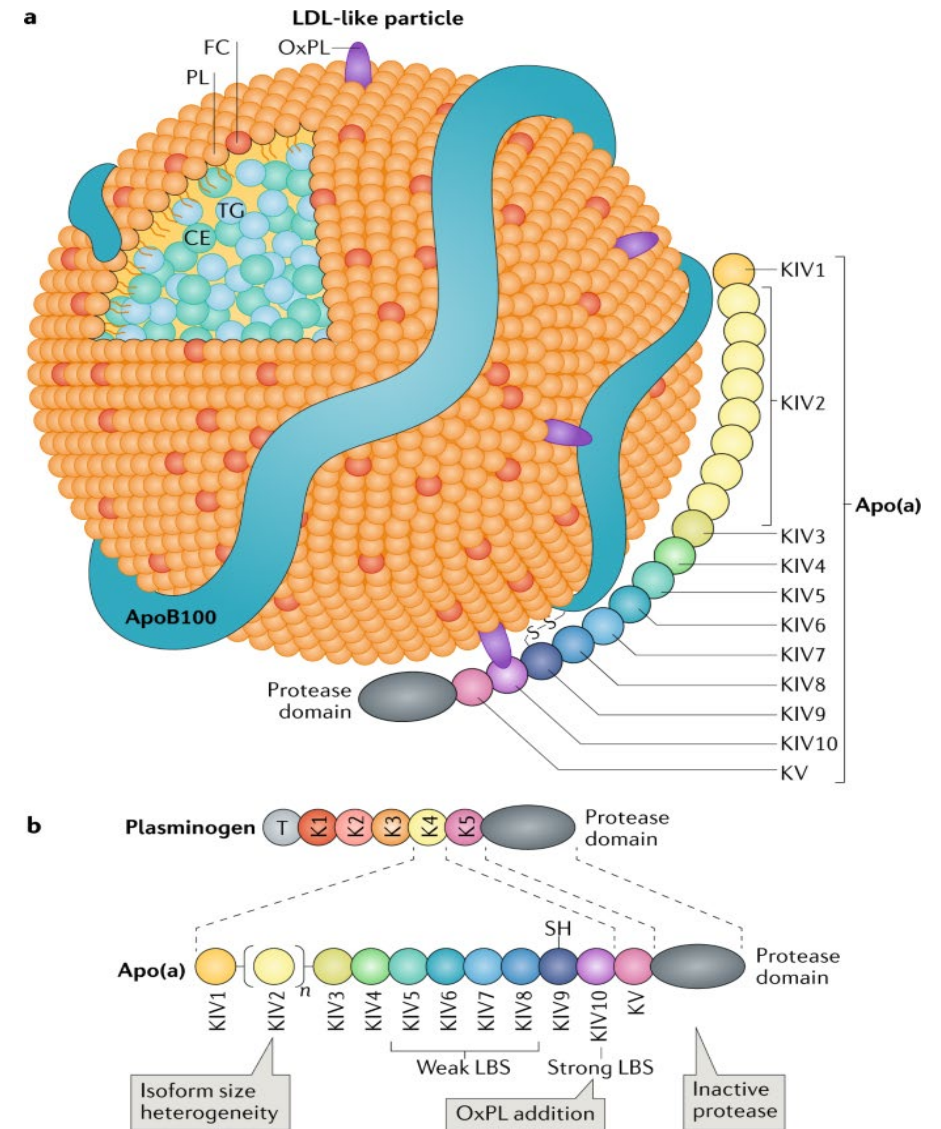
Graphics from De Oliveira-Gomes, Diana et al. "Apolipoprotein B: Bridging the Gap Between Evidence and Clinical Practice." *Circulation* vol. 150,1 (2024): 62-79. doi:10.1161/CIRCULATIONAHA.124.068885

Lipid Metabolism

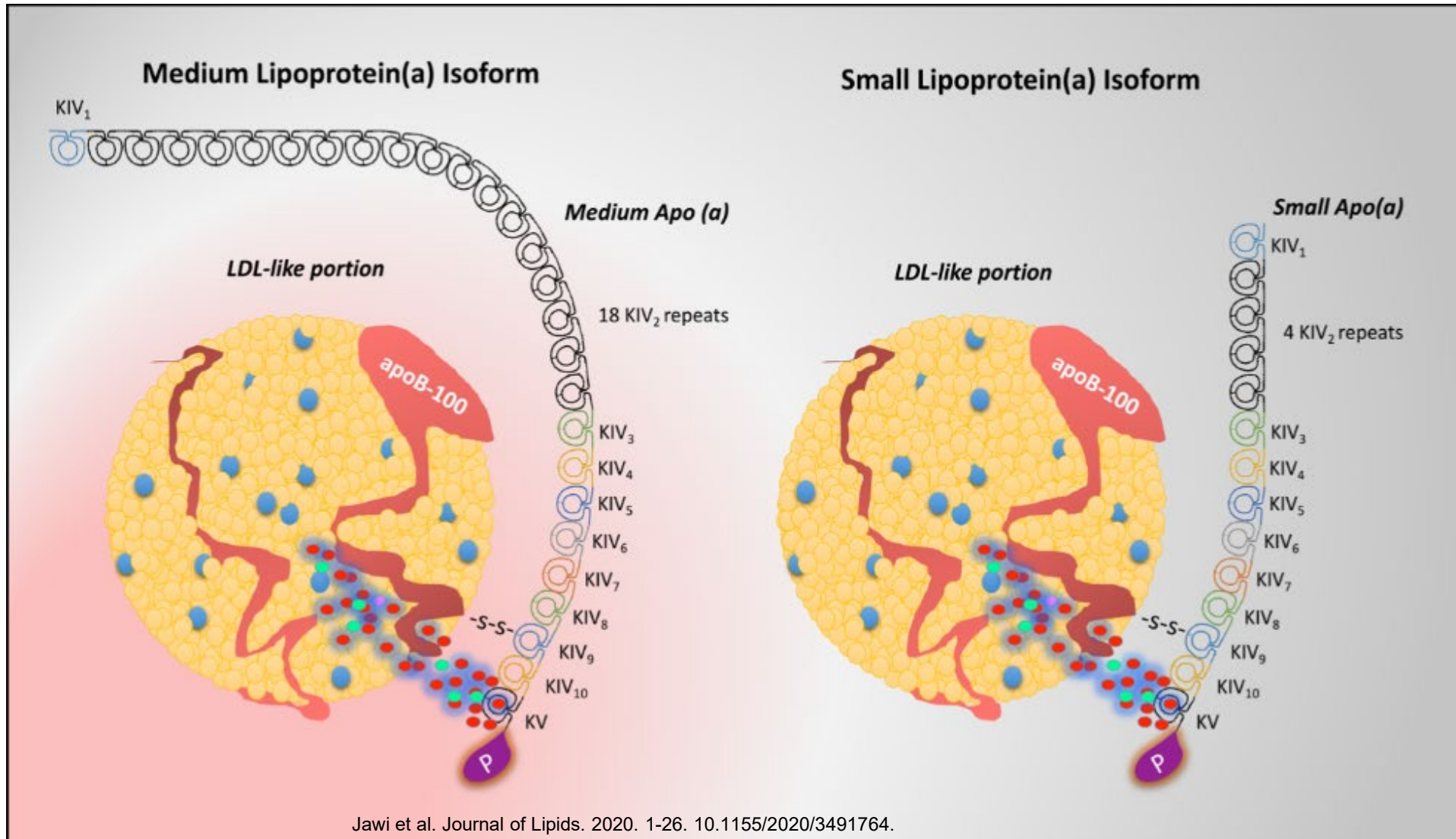


Lipoprotein(a) Particle

- ApoB100 containing Lipoprotein, covalently bound to apolipoprotein (a).
- LPA gene is one of the most potent monogenetic risk factors for CAD regardless of race and aortic stenosis
- Autosomal co-dominant inheritance, phenotype of both alleles is expressed.



Lipoprotein(a) Isoforms



Key Characteristics of KIV-2 Repeats

- **Copy Number Variation (CNV):** KIV-2 segment can repeat anywhere from **1 to over 40 times**
- **Inverse Relationship:** Strong inverse correlation between the number of KIV-2 repeats and plasma Lp(a) levels
 - **Small Isoforms (Fewer KIV-2 repeats):** Higher circulating Lp(a) concentration and increased risk of heart attack, stroke, and aortic stenosis
 - **Large Isoforms (More KIV-2 repeats):** Lower Lp(a) levels and more **neutral** for cardiovascular risk.
- **Inheritance:** Individuals inherit two different-sized isoforms (one from each parent), and their total Lp(a) level is often dominated by the smaller isoform

Measurement of Lp(a)

- Older assays measured concentrations in **mg/dL**
 - Tells you about the total mass of Lp(a) protein but not informative about isoforms (which is the important info!)
- Newer assays measure molar concentration (protein particles) in **nmol/L**
 - Increasingly standardized
 - Preferred for reliability of measurement and indication of risk
- Conversion possible but not reliable for individuals

Lp(a) and ASCVD Risk

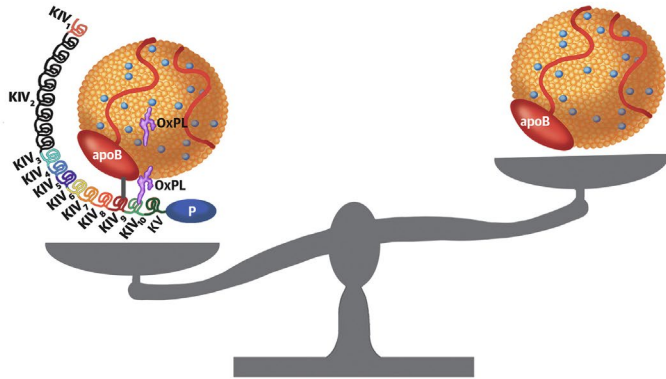
A Per-Particle ASCVD Risk

Lp(a)-apoB - 250 nmol/L

- Proinflammatory OxPL
- Proinflammatory proteome
- Smaller, more dense
- Antifibrinolytic

LDL-apoB - 250 nmol/L

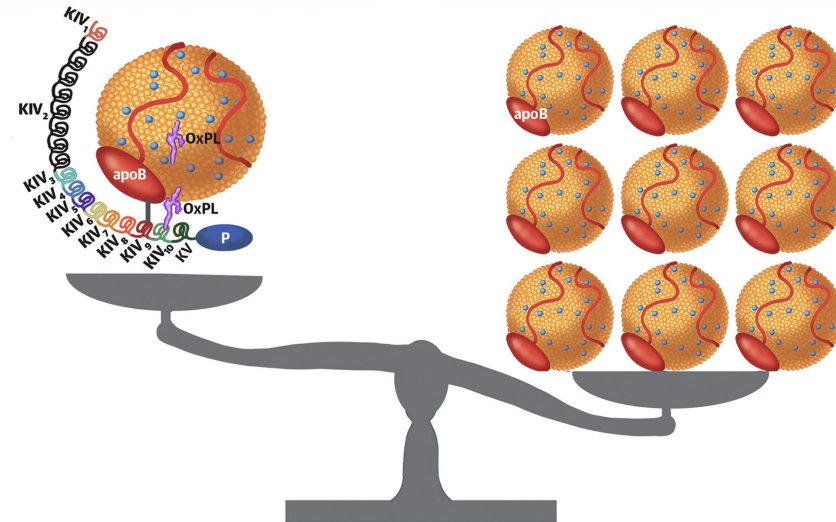
- Not inflammatory unless oxidized
- More benign proteome
- Larger, less dense
- Not antifibrinolytic



B Total Particle ASCVD Risk

Lp(a)-apoB, 250 nmol/L

LDL-apoB, 2,250 nmol/L



Tsimikas and Bittner. J Am Coll Cardiol. 2024;83(3):396-400.

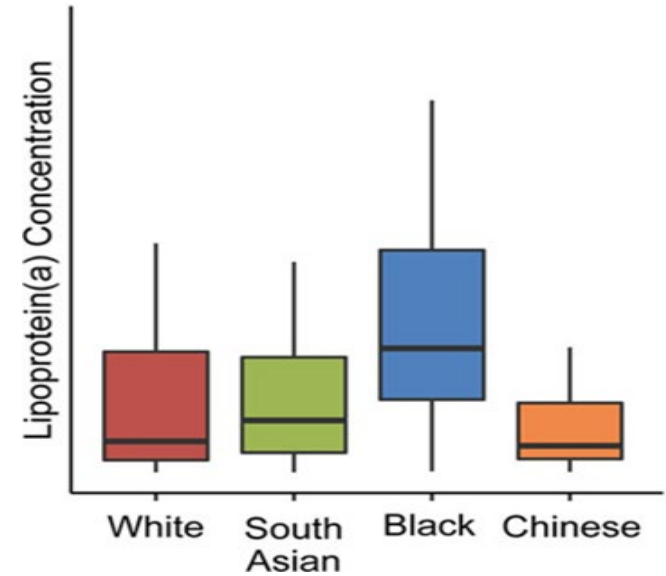
“ LDL particles are significantly more prevalent in most patients than Lp(a) particles. These observations put into clinical context the risk of ASCVD mediated by Lp(a) and LDL-C and suggest particle characteristics and particle number are both important variables in predicting ASCVD risk.”



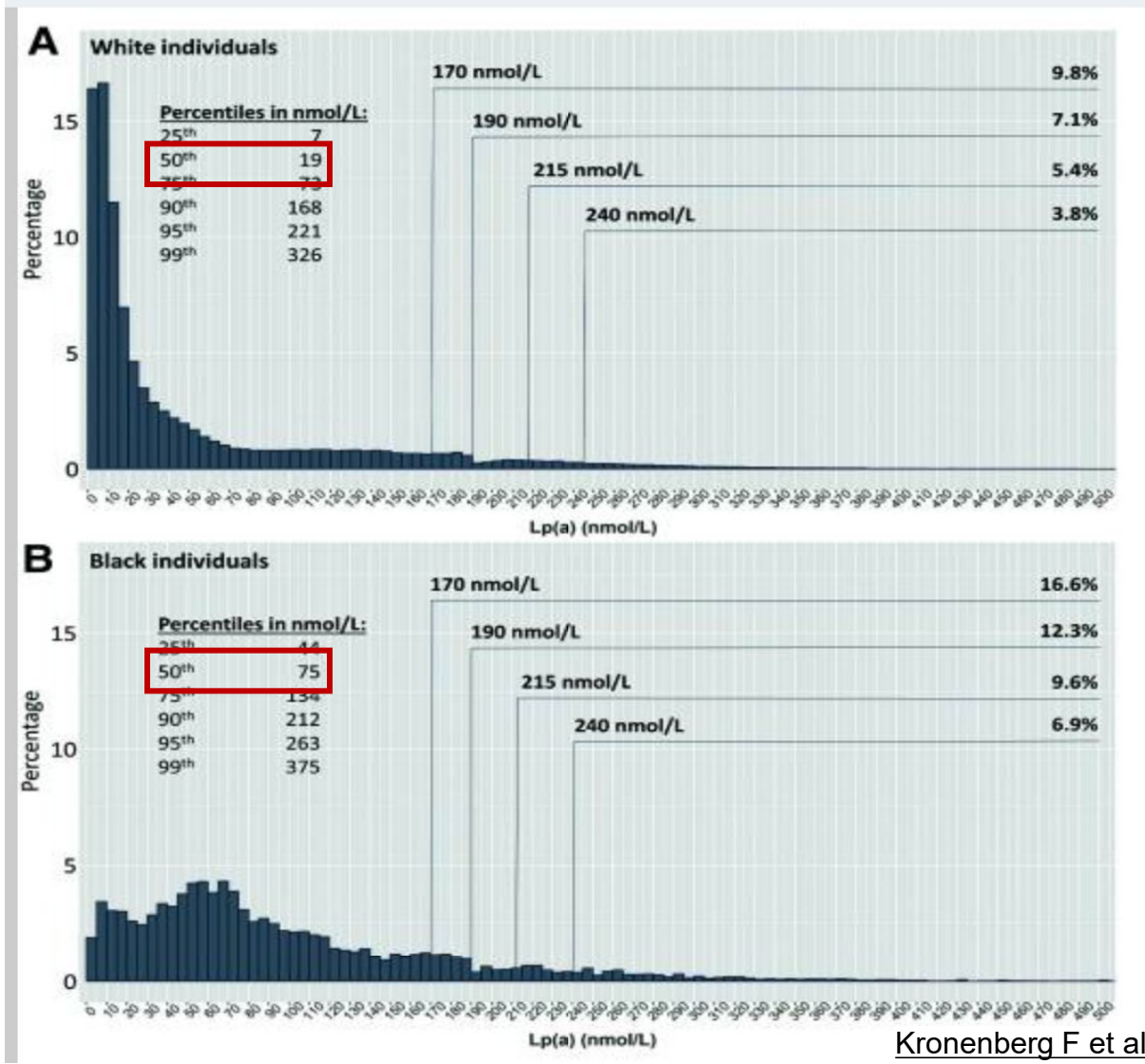
Lipoprotein(a) by Ancestry: UK Biobank

460,506 Adults
in UK Biobank

Significant differences in Lp(a) concentrations according to race

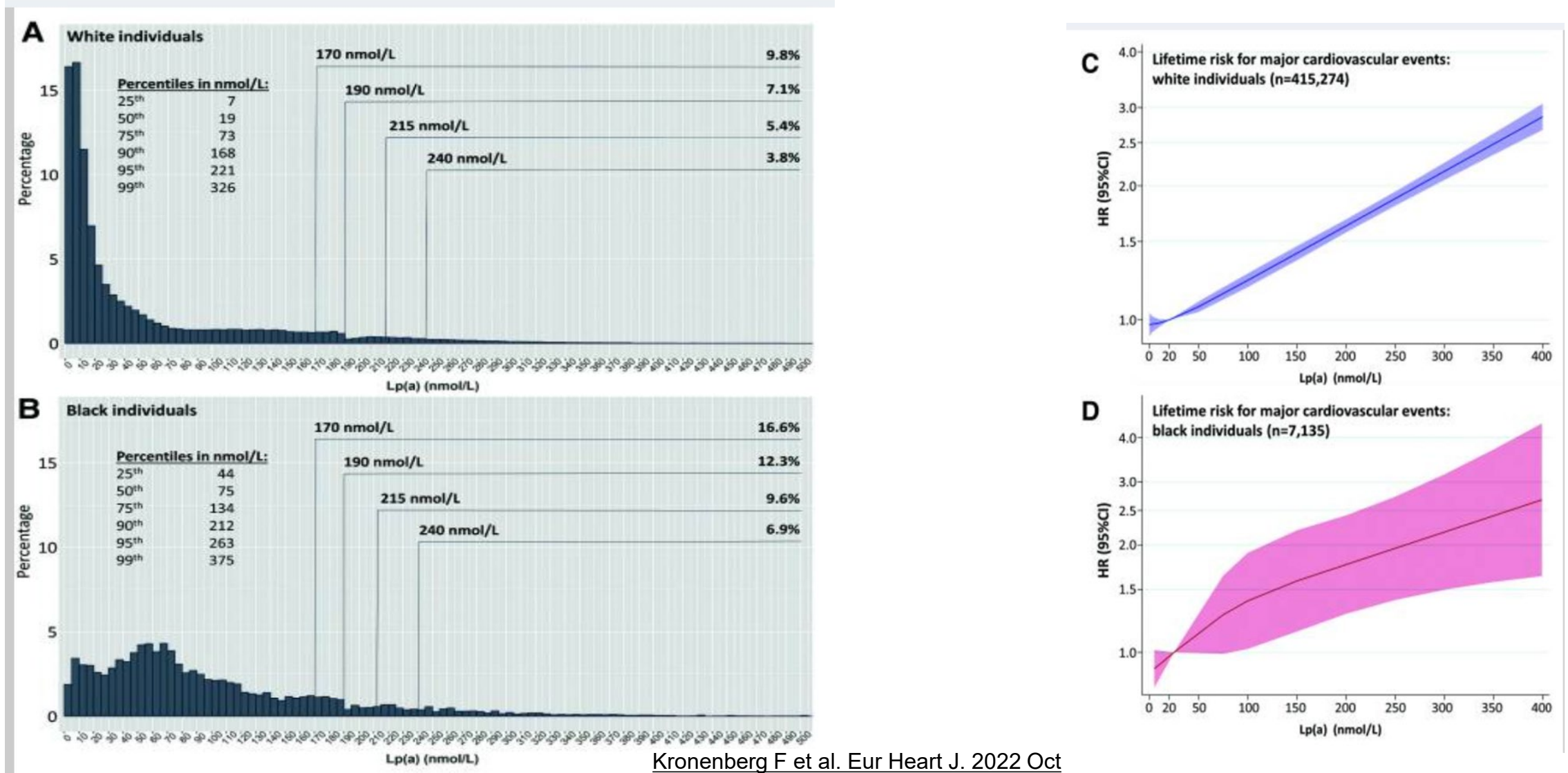


Patel et al. Arteriosclerosis, Thrombosis, and Vascular Biology. 2021;41:465–474



Kronenberg F et al. Eur Heart J. 2022 Oct 14; 43(39): 3925–3946.

Lipoprotein(a) by Ancestry: UK Biobank



Kronenberg F et al. Eur Heart J. 2022 Oct 14; 43(39): 3925–3946.

Lp(a) and Disease

- Pro-atherogenic effects:
 - Lp(a) can promote atherosclerosis by depositing in arterial walls
- Pro-thrombotic effects:
 - Lp(a) contains kringle IV and plasminogen-like domains, which can interfere with fibrinolysis, enhancing clot formation
- Clearance and degradation:
 - Lp(a) is cleared by the liver and possibly other tissues through mechanisms involving lipoprotein receptors

Why do we have Lp(a)?

Physiology

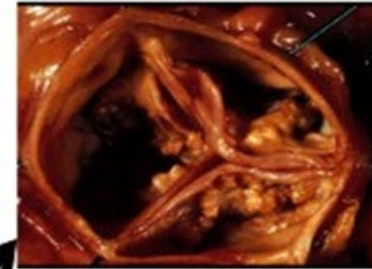
Improved survival due to reduced bleeding:
1) for mother and child during childbirth
2) during infectious diseases including tuberculosis
3) from injuries during war, from dangerous animals and insects, or from ordinary everyday life

Wound healing as apo(a)
KIV-2 slows fibrinolysis and promotes thrombosis



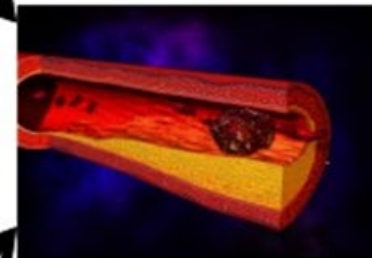
Pathophysiology

Thrombosis through fibrinolysis inhibition at turbulent flow



Aortic valve stenosis

Thrombosis through fibrinolysis inhibition at vulnerable plaques



Atherosclerotic stenosis



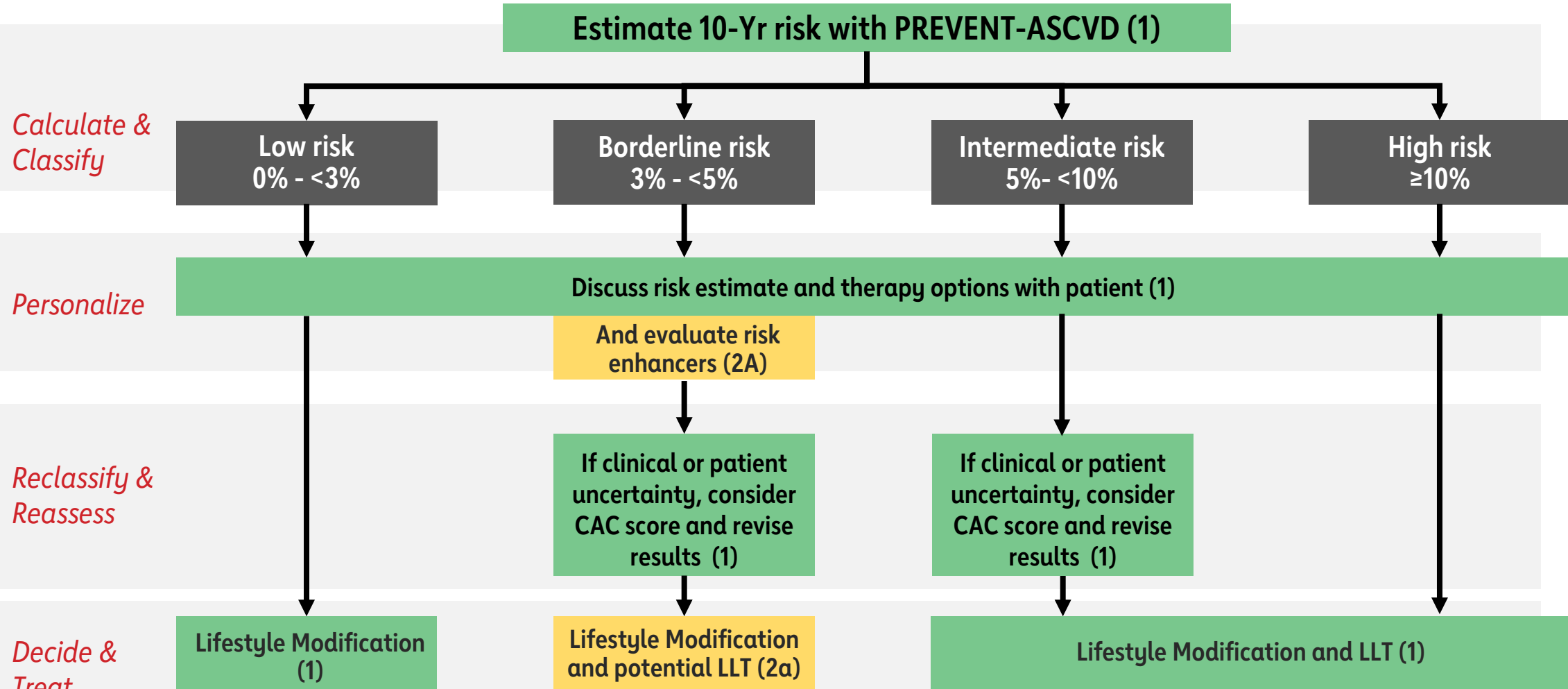
Myocardial infarction

Why measure Lp(a) if no current therapies?

- **Causality of Lp(a) in ASCVD has (largely) been established**
 - Mendelian randomization studies
 - Epidemiological studies
 - Clinical studies
- **Clinical presentations show strong associations of high Lp(a) and low isoform size with:**
 - Aortic valve stenosis
 - Peripheral vascular disease and venous thrombosis
 - Stroke
 - Myocardial infarction

Preventing CVD with Elevated Lp(a): 2026 ACC/AAHA/Multi-Specialty Guideline

Calculate, Personalize, Reclassify (CPR) Framework



Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; CAC, coronary artery calcium; LLT, lipid-lowering therapy; and PREVENT, Predicting Risk of cardiovascular disease EVENTS.

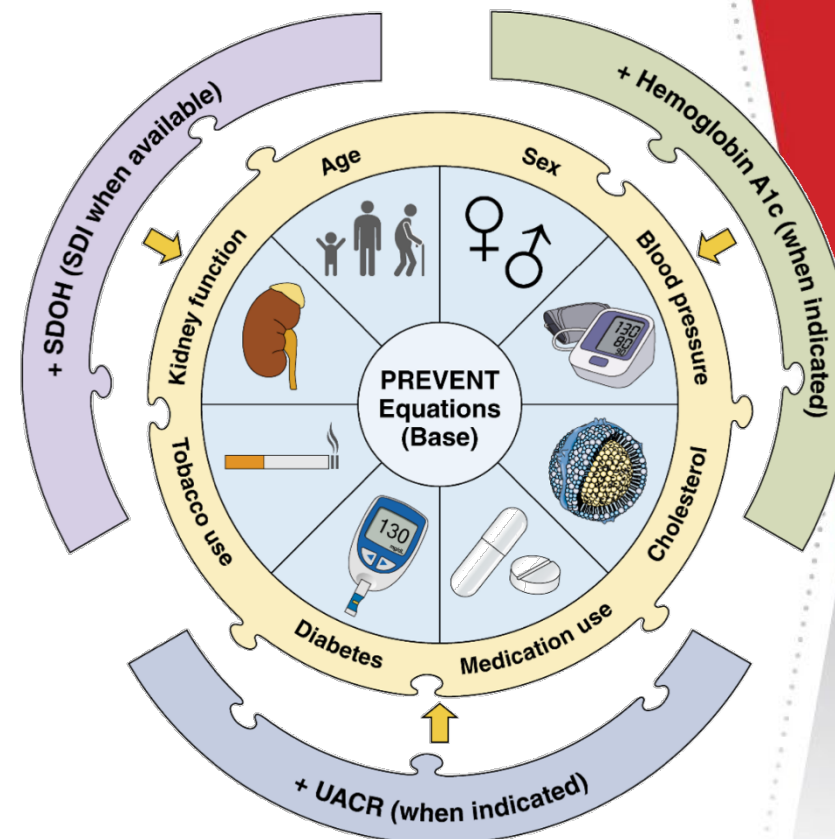


PREVENT-ASCVD Risk Calculator

COR	RECOMMENDATIONS
1	PREVENT-ASCVD equations should be used for risk assessment in adults aged 30-79 years without ASCVD or subclinical atherosclerosis, with LDL-C 70-189 mg/dL.

Approximate Equivalent Ranges of 10-yr ASCVD Risk Estimates

RISK	POOLED COHORT EQUATIONS	PREVENT-ASCVD
Low	<5%	<3%
Borderline	5 - <7.5%	3 to <5%
Intermediate	7.5 - <20%	5 to <10%
High	≥20%	≥10%



Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; LDL-C, low-density lipoprotein cholesterol; PREVENT, Predicting Risk of cardiovascular disease EVENTS; SDI, social deprivation index; SDOH, social determinants of health; and UACR, urine albumin-to-creatinine ratio.

Role of Individualized Benefit-Risk Discussion



Information

Clinician provides the best available evidence for treatment options, including the risks & benefits of each option



Patient-Centered Care

Treatment & care options take into consideration individual values & preferences



Shared Decision-Making

A collaborative decision about treatment or care is documented and shared with relevant stakeholders

COR	RECOMMENDATIONS
1	In individuals with dyslipidemia, clinicians and their patients should engage in a discussion of the patient's ASCVD risk, healthy lifestyle as the foundation of risk reduction, expected risk reduction benefits from LLT, possible harms and DDI, costs, and patient preferences to make individualized treatment decisions and/or consider additional options for evaluation to aid in decision-making.

Discussion should emphasize

- Patient's ASCVD risk
- Consideration of additional options for evaluation
- Healthy lifestyle as the foundation of risk reduction
- Expected risk reduction benefits from lipid-lowering therapies
- Possible harms and drug-drug interactions
- Costs
- Administration frequency
- Patient preferences

Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; DDI, drug-drug interaction; and LLT, lipid-lowering therapy.

Blumenthal, R.S., Morris, P.B., et al. 2026 ACC/AHA Guideline on the Management of Dyslipidemia. *Circulation*.

Risk Enhancers

- History of premature ASCVD in a parent or sibling (onset age <55 y for men, <65 y for women)
- Higher risk ancestry (eg, South Asian, Filipino)
- High polygenic risk (if measured)
- Chronic inflammatory diseases (eg, systemic lupus, rheumatoid arthritis, advanced psoriasis, inflammatory arthritis)
- Lp(a) ≥ 125 nmol/L or ≥ 50 mg/dL
- hsCRP ≥ 2 mg/L on >1 occasion (if measured)
- TG persistently ≥ 175 mg/dL (2 mmol/L) (if nonfasting) and ≥ 150 mg/dL (1.7 mmol/L) (if fasting)
- CKM syndrome
- LDL-C persistently ≥ 160 -189 mg/dL (4.1-4.9 mmol/L), non-HDL-C ≥ 190 -219 mg/dL or apoB ≥ 120 mg/dL
- Reproductive risk markers (premature menopause, preeclampsia, gestational diabetes, gestational hypertension, preterm delivery)

COR	RECOMMENDATIONS
2a	In adults without ASCVD with a borderline 10-year ASCVD risk estimate (3% to <5%) by the PREVENT-ASCVD equations, consideration of risk-enhancers is reasonable to personalize risk assessment and the potential benefit of initiating LLT as an adjunct to lifestyle management to reduce ASCVD risk.

Abbreviations: ApoB indicates apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; HDL-C, high-density-lipoprotein cholesterol; hsCRP, high-sensitivity C-reactive protein, LDL-C, low-density lipoprotein cholesterol; LLT, lipid-lowering therapy; and PREVENT, Predicting Risk of CVD Events.

Lp(a) as a risk-enhancing factor: How to quantify?

- Log-linear relationship of Lp(a) level with risk

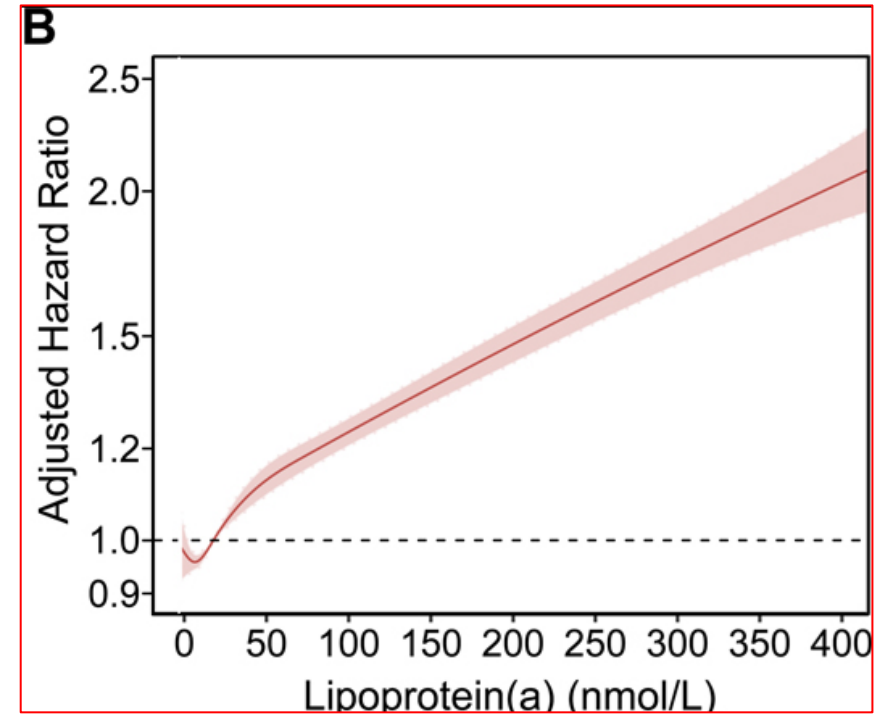
- Approximate updated 10-y risk estimate:

Predicted 10-y risk $\times [1.11^{(\text{patient's Lp(a) level in nmol/L}/50)}]$

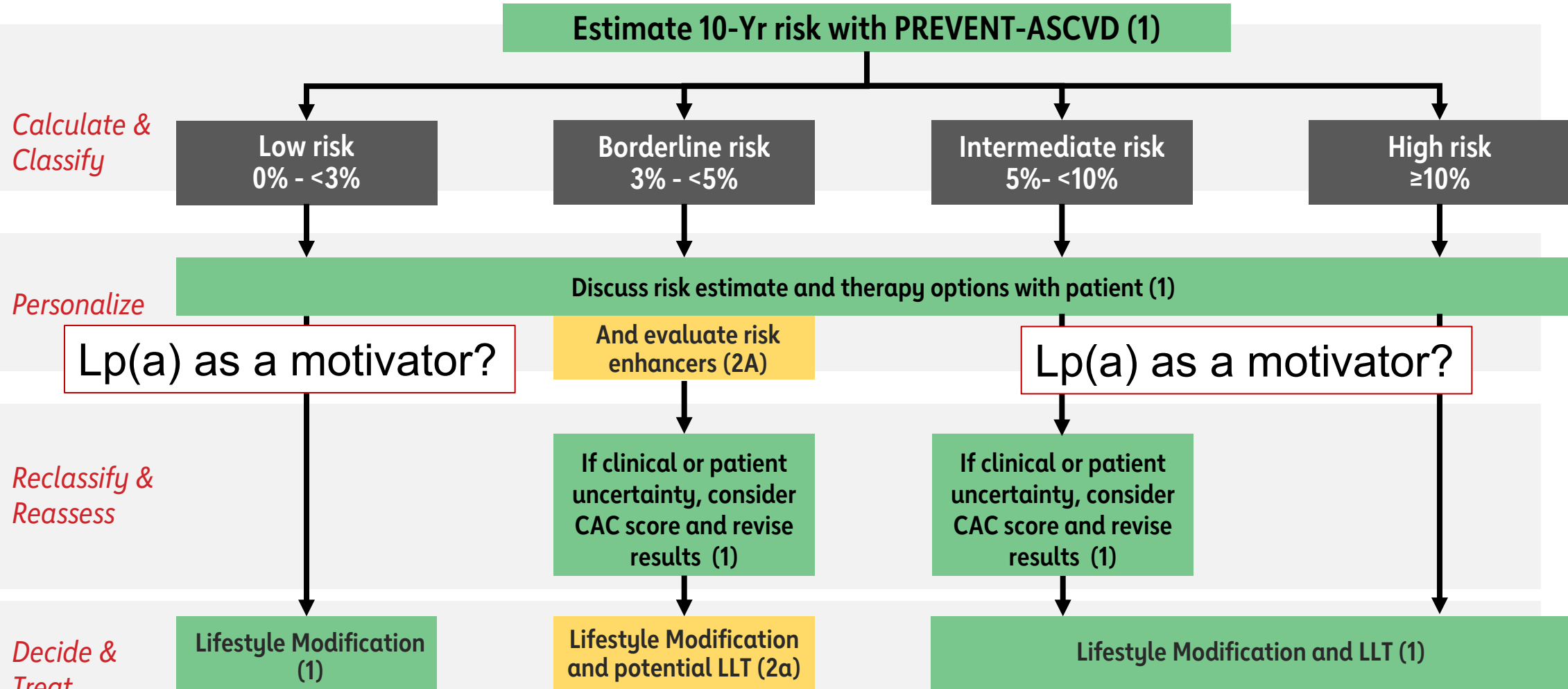
Example: Patient with 10-yr risk of 10% and

Lp(a) = 250 nmol/L

$$10.0\% \times 1.11^{(250/50)} = 10.0\% \times 1.11^5 = 10.0\% \times 1.69 \\ = 16.9\%$$

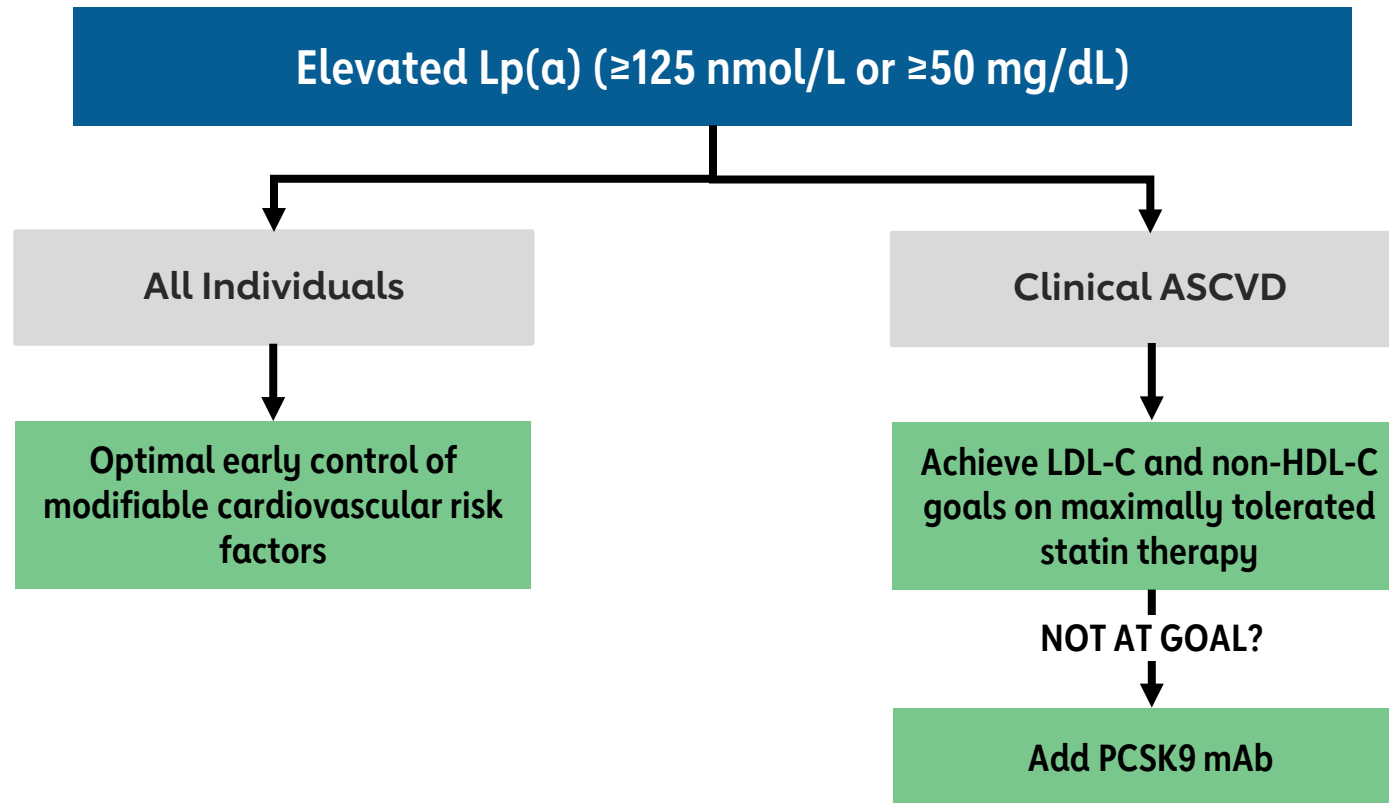


Calculate, Personalize, Reclassify (CPR) Framework



Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; CAC, coronary artery calcium; LLT, lipid-lowering therapy; and PREVENT, Predicting Risk of cardiovascular disease EVENTS.

Approach to Patients with Elevated Lp(a)



Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein-cholesterol; Lp(a), lipoprotein(a); mAb, monoclonal antibody; and PCSK9, Proprotein Convertase Subtilisin/Kexin type 9.

Lifestyle for All!

Particularly important for those with family history/genetic risk

Goals with lifestyle therapy

- Goal is to reduce underlying risk – not to reduce Lp(a) levels directly
- Diet, PA, etc. have minimal effects on Lp(a) levels *per se*
- But they can additively reduce risk for CVD events, reducing the background risk through direct effects and indirect effects on other RFs

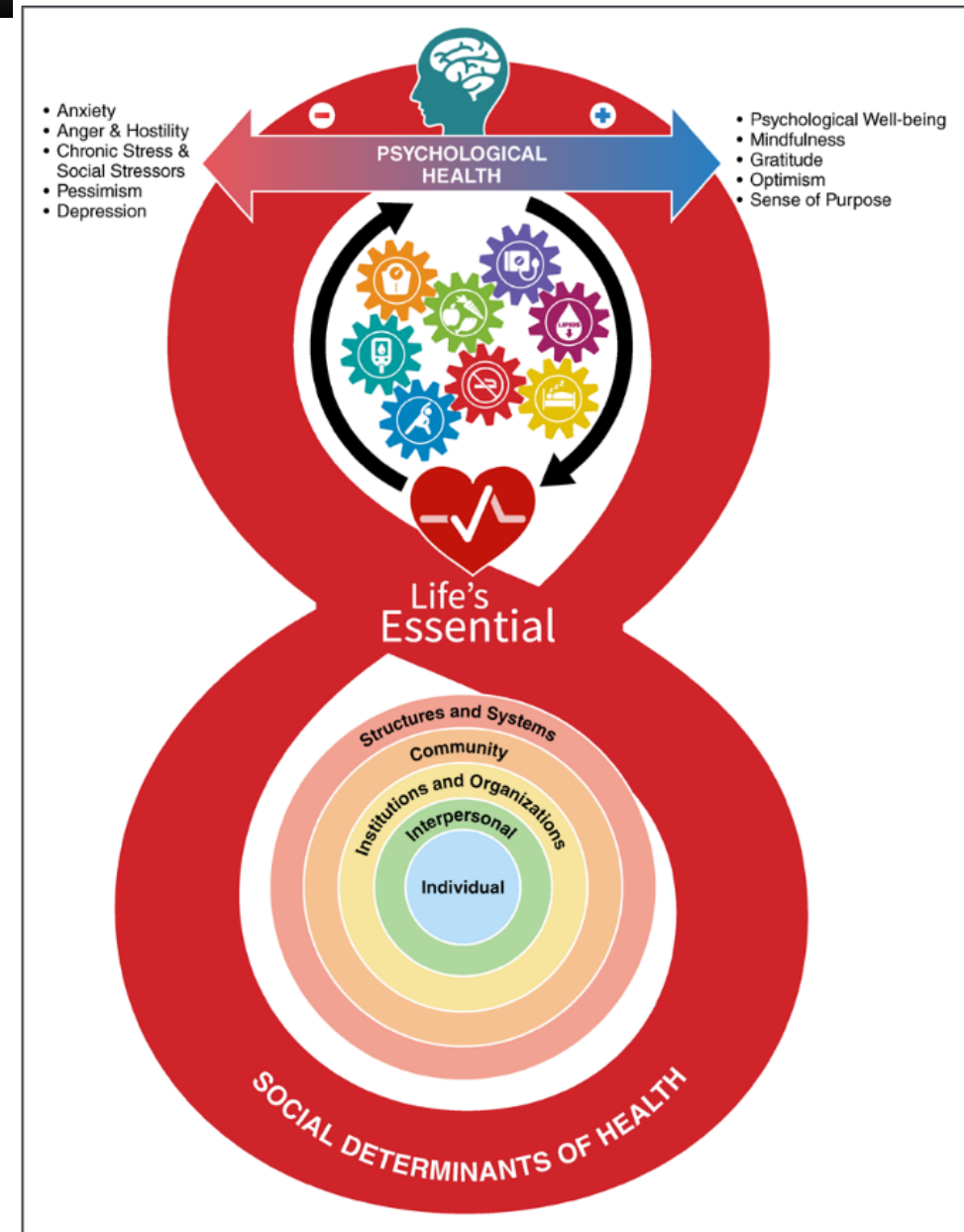
Prevention with Elevated Lp(a)

Circulation

AHA PRESIDENTIAL ADVISORY

Life's Essential 8: Updating and Enhancing the American Heart Association's Construct of Cardiovascular Health: A Presidential Advisory From the American Heart Association

Donald M. Lloyd-Jones, MD, ScM, FAHA, Chair; Norrina B. Allen, PhD, MPH, FAHA; Cheryl A.M. Anderson, PhD, MPH, MS, FAHA; Terrie Black, DNP, MBA, CRRN, FAHA; LaPrincess C. Brewer, MD, MPH; Randi E. Foraker, PhD, MA, FAHA; Michael A. Grandner, PhD, MTR, FAHA; Helen Lavretsky, MD, MS; Amanda Marma Perak, MD, MS, FAHA; Garima Sharma, MD; Wayne Rosamond, PhD, MS, FAHA; on behalf of the American Heart Association



Lloyd-Jones, D.M. *et al.* (2022) 'Life's essential 8: Updating and enhancing the American Heart Association's construct of Cardiovascular Health: A presidential advisory from the American Heart Association', *Circulation*, 146(5). doi:10.1161/cir.0000000000001078.

My Life Check

 American Heart Association.
My Life Check

**GOOD HABITS BUILD
BETTER HEALTH**

We've helped millions of people make healthier choices.

The AHA is the nation's oldest and largest voluntary organization dedicated to fighting heart disease and stroke. For nearly 100 years, we've been helping people like you live longer, healthier lives.

[Get Started](#)

My Life Check – Improving LE8

The screenshot displays the 'My Life Check' app interface. At the top left is the American Heart Association logo and 'My Life Check' text. A settings gear icon is at the top right. Below the header are two tabs: 'Assessment' and 'Heart Health Score', with the latter being selected. The main content area is titled 'HEART HEALTH SCORE' and features a large circular gauge showing a score of 90 out of 100. A red button labeled 'Update your score' is positioned below the score. To the right of the gauge, there are two sections: 'IMPROVE' (indicated by an orange dot) and 'CELEBRATE' (indicated by a green dot). The 'IMPROVE' section includes three items: 'Eat Better' (green icon), 'Lose Weight' (orange icon), and 'Manage Blood Pressure' (purple icon). The 'CELEBRATE' section includes four items: 'Get Active' (blue icon), 'Stop Smoking' (red icon), 'Reduce Blood Sugar' (teal icon), and 'Control Cholesterol' (purple icon). Each item has a right-pointing arrow icon.

American Heart Association.
My Life Check

Assessment Heart Health Score

HEART HEALTH SCORE

90
out of 100
Update your score

0 100

● IMPROVE

- Eat Better
- Lose Weight
- Manage Blood Pressure

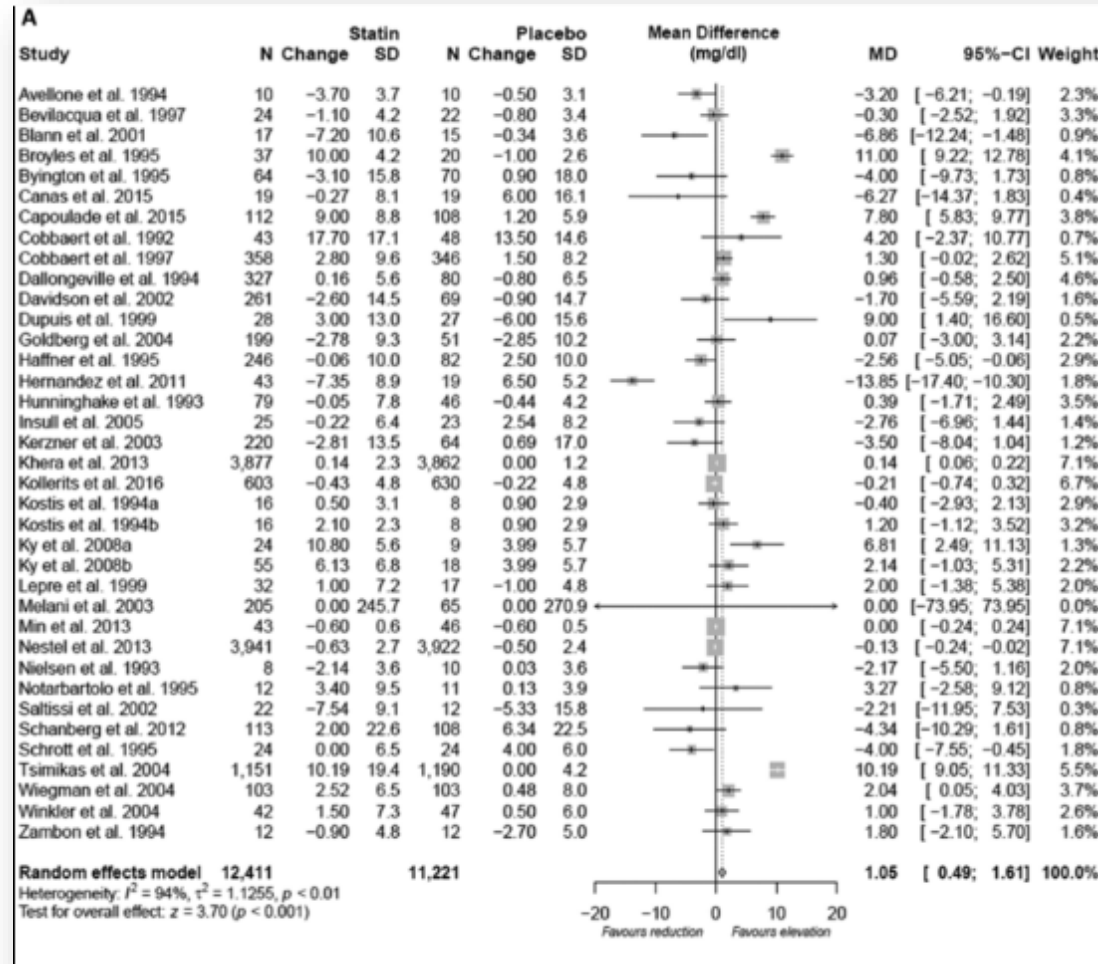
● CELEBRATE

- Get Active
- Stop Smoking
- Reduce Blood Sugar
- Control Cholesterol
- Sleep

Medical therapy

LDL-C reduction remains the cornerstone of therapy for now

Statins do not alter Lp(a) levels substantially

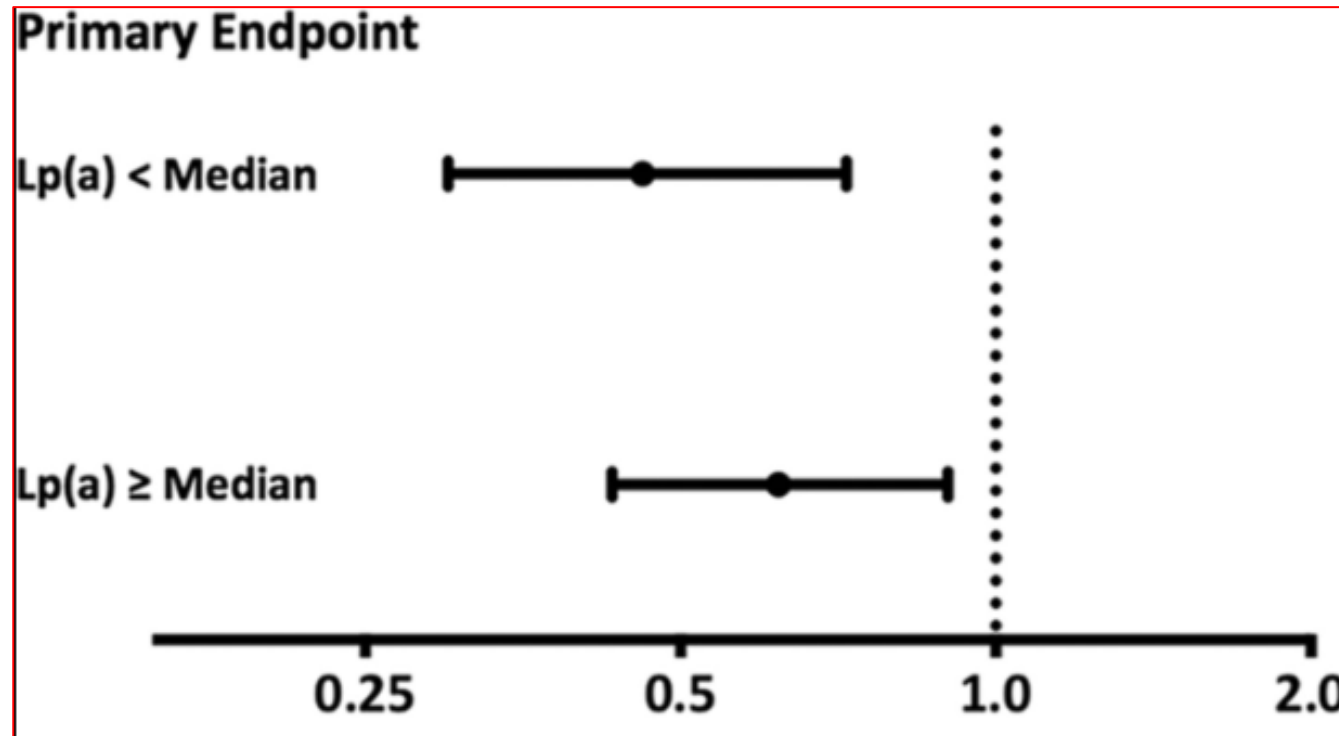


Boston University Chobanian & Avedisian School of Medicine



Statins reduce ASCVD events – even in people with Lp(a)

- JUPITER trial: Similar RRR, greater ARR with elevated Lp(a)



Boston University Chobanian & Avedisian School of Medicine



Personal approach to therapy in patients with elevated Lp(a)

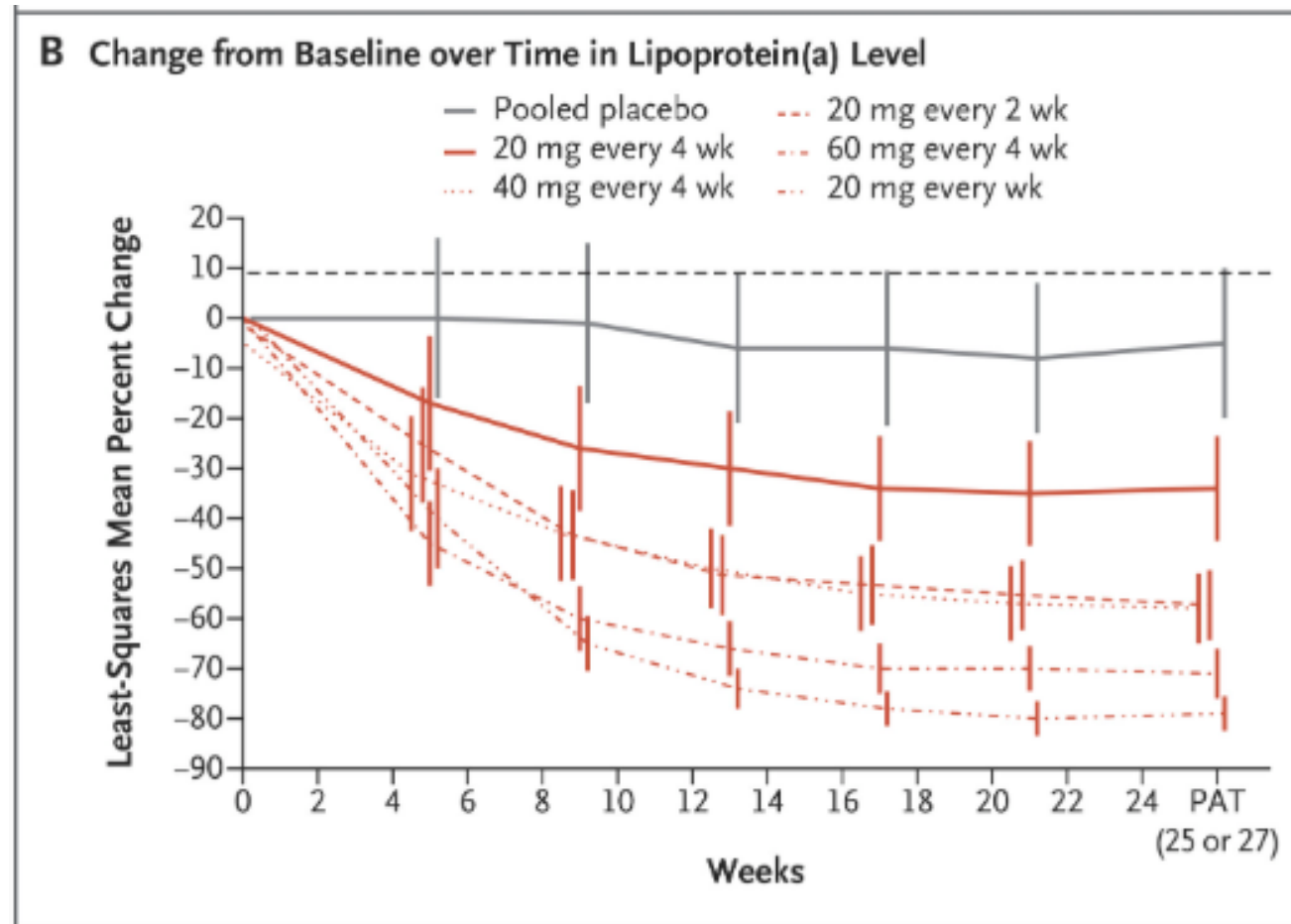
- Remove the vector (LDL-C particles)
- NLA and ESC GL recommendations:
 - LDL-C <100 mg/dL for primary prevention (ApoB <90)
 - LDL-C <70 mg/dL for high-risk primary and secondary prevention (ApoB <70)
 - LDL-C <55 mg/dL for very high-risk secondary prevention (ApoB <55)
- Checking ApoB can help avoid residual risk related to LDL-C particles
- Consider aspirin in higher-risk patients (this appears to be one group with net benefit)

PCSK9 mAb and inclisiran reduce Lp(a) modestly; bempedoic acid does not

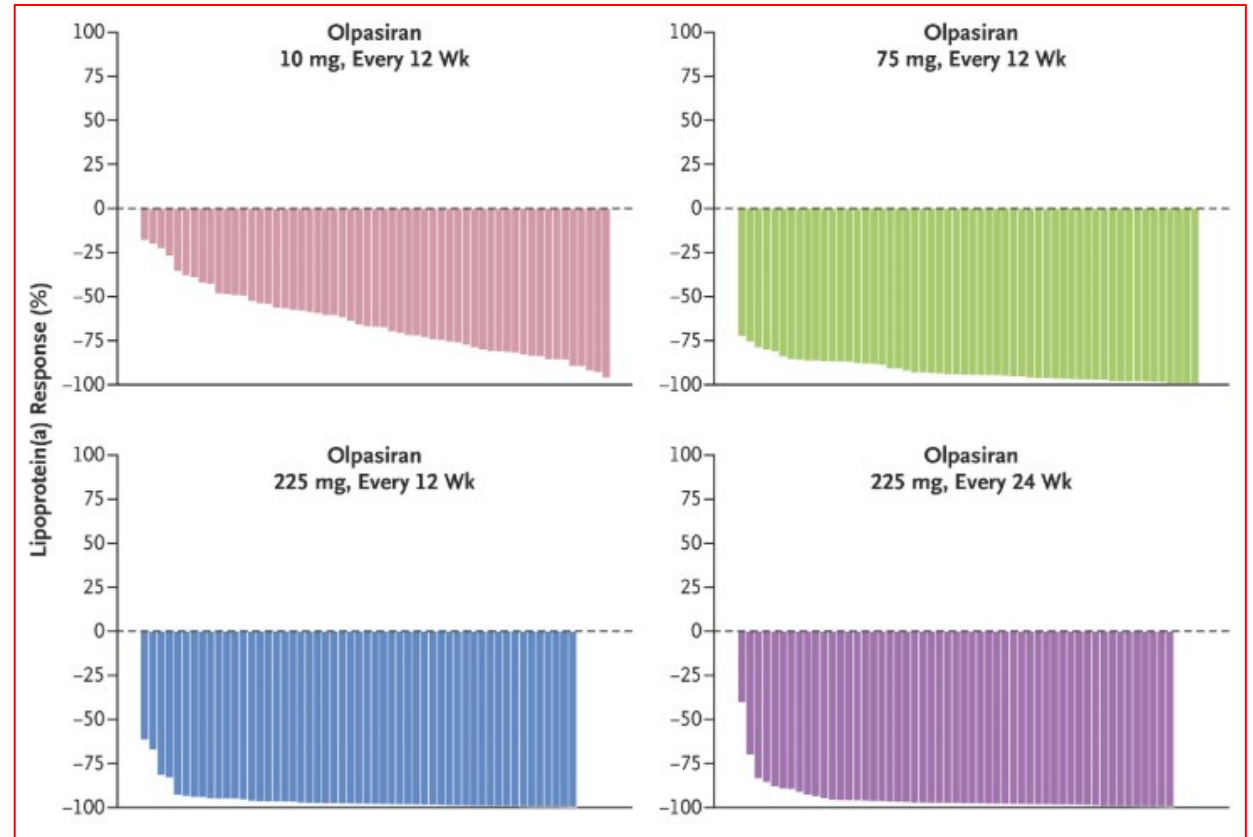
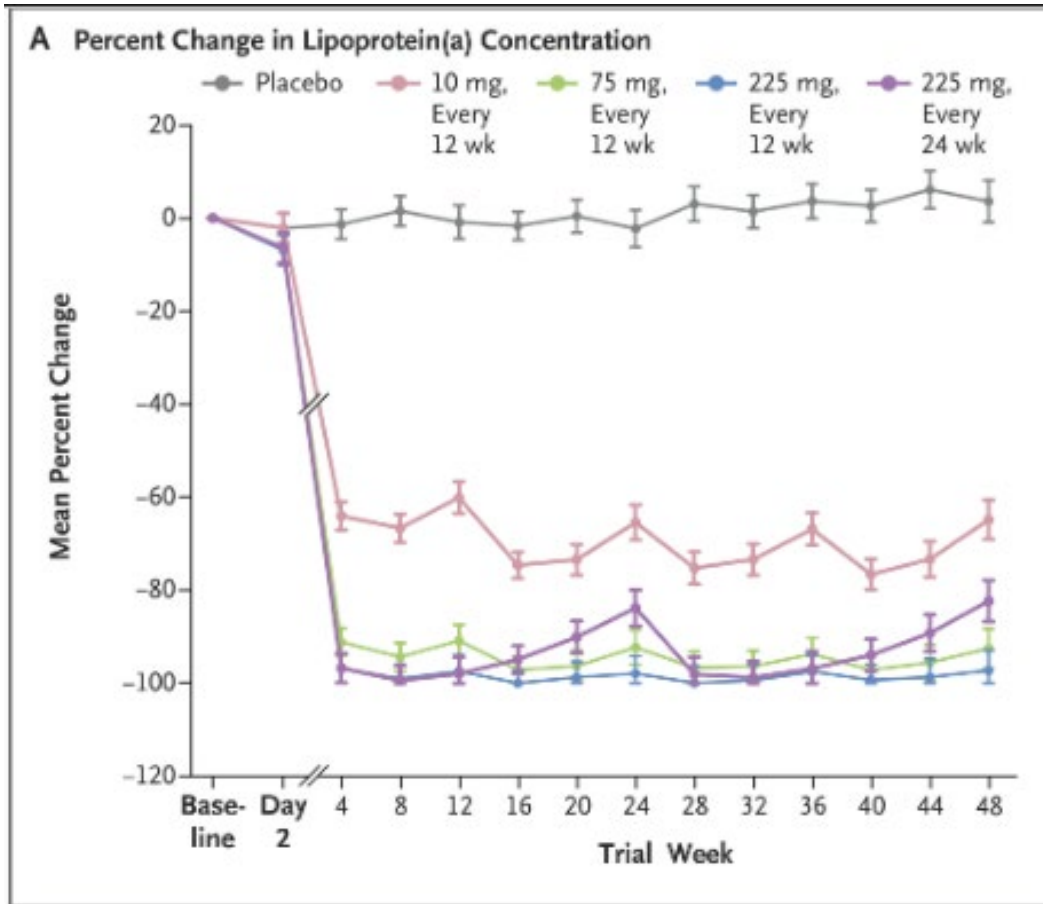
- Both evolocumab and alirocumab reduce Lp(a) levels modestly
 - Reduction of Lp(a) in those with elevated Lp(a) appears to provide greater benefit and explain some of the efficacy
- Inclisiran also associated with modest Lp(a) reduction – large outcomes trial awaited
- Bempedoic acid associated with no significant change in Lp(a)

The Future – Direct inhibition of apo(a) production

Pelacarsen – ASO dose-ranging study



Olpasiran – siRNA dose-ranging trial

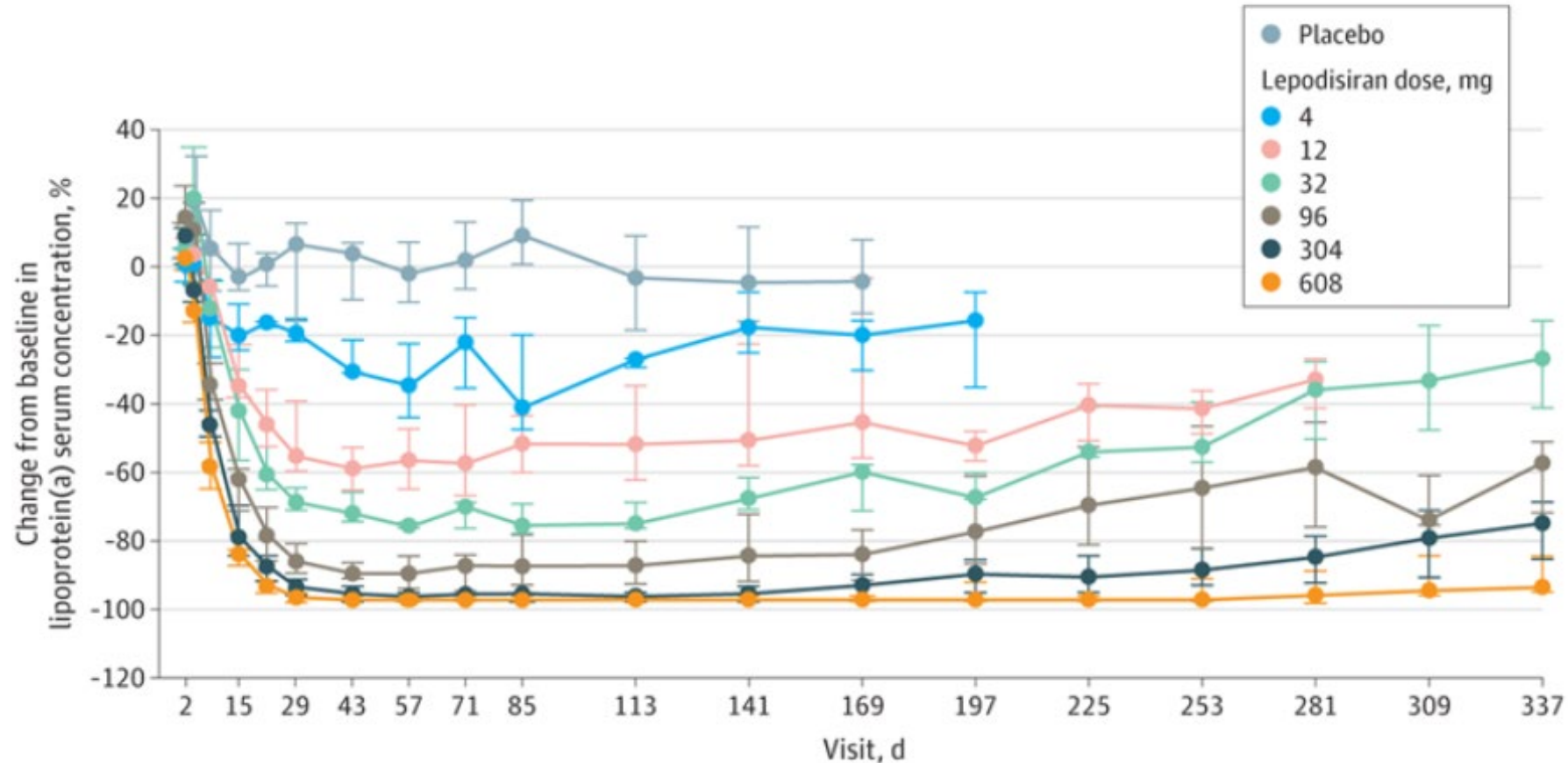


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Lepodisiran – siRNA dose-ranging trial

Figure 3. Percentage Change in Levels of Lipoprotein(a) From Baseline to 336 Days (48 Weeks) After Administration

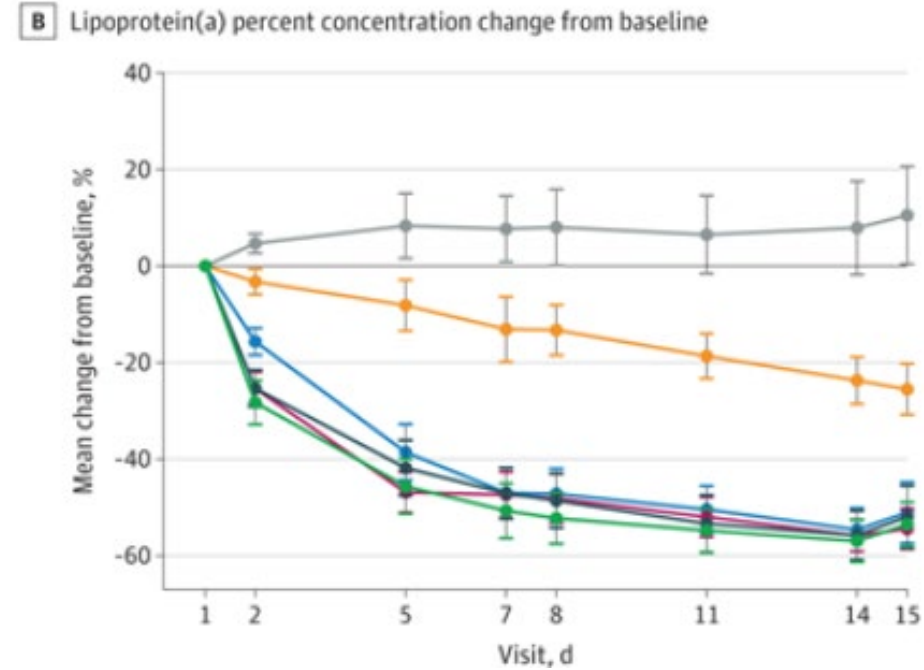


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Muvalaplin – oral agent

- Blocks covalent binding of apo(a) with apoB-100



Boston University Chobanian & Avedisian School of Medicine



Nicholls SJ, Nissen SE, Fleming C, et al. Muvalaplin, an Oral Small Molecule Inhibitor of Lipoprotein(a) Formation: A Randomized Clinical Trial. *JAMA*. 2023;330(11):1042–1053. doi:10.1001/jama.2023.16503

Take Home Points

- Lp(a) is likely a causal risk factor for ASCVD, enhances risk for ASCVD, and context of Lp(a) matters
- Managing traditional risk factors with lifestyle and medication remains paramount
- Statins, ezetimibe, and PCSK9mAb can reduce risk with variable effects on Lp(a) itself
- The future of direct Lp(a) therapy looks bright
 - We will answer the “causal question”
 - Direct Lp(a) inhibition may become an important adjunct to LDL-C lowering therapy, if compounds prove safe and efficacious in larger trials (ongoing)

Learn More About Lp(a)

Heart Attack and Stroke Symptoms Volunteer Learn CPR ShopHeart

American Heart Association

Type to search... [Donate Once](#) [Donate Monthly](#)

Healthy Living Health Topics Professionals Get Involved Ways to Give About Us Learn CPR In Your Community

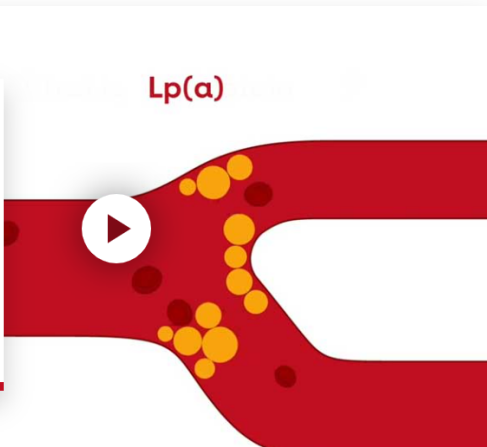
Home / Health Topics / Cholesterol / Genetic Conditions / **Lipoprotein (a)**

Lipoprotein (a)

Lp(a) is a genetic independent risk factor for heart disease. Knowing your Lp(a) levels can help you reduce your risk of heart disease for you or someone you love.

[Video: What is Lp\(a\)?](#)
[Watch the video in Spanish](#)

Lp(a)



Learn about Lp(a)

Many people don't have symptoms. You could have a [high Lp\(a\)](#) even if you have a healthy lifestyle and all other heart disease risk factors are controlled. Talk with your health care professional about screening if you have:


- Known family history of high Lp(a)
- Family or personal history of heart disease or premature cardiovascular disease
- Diagnosis of familial hypercholesterolemia (FH), an inherited condition that causes the body to poorly recycle LDL or bad cholesterol

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Home / Lp(a) in Focus: Breakthrough Therapies and Clinical Implications



Lp(a) in Focus: Breakthrough Therapies and Clinical Implications

Elevated Lipoprotein(a) or Lp(a) is increasingly recognized as a significant, independent risk enhancer for cardiovascular disease (CVD). This course explores the evolving landscape of Lp(a) in clinical practice, emphasizing the importance of Lp(a) testing in comprehensive cardiovascular risk assessment. The safety and efficacy of current and emerging lipid-lowering therapies, with a focus on novel agents targeting Lp(a) reduction will be examined.

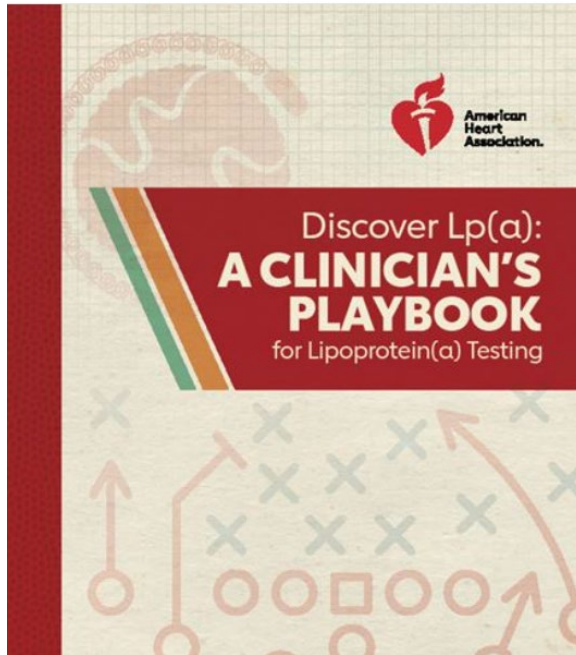
Complimentary  Training Program

[Access Now](#)

Activity Summary

Product Number	28-1957
Expiration Date	Nov 14, 2028

Lp(a) Resources



Lp(a) in Special Populations: Aortic Stenosis and Peripheral Arterial Disease

Dr. Nishant P. Shah, MD
Associate Professor of Medicine in Cardiology
Duke Clinical Research Institute
Duke Cardiometabolic Prevention

Dr. Marc Bonaca, MD
Professor of Medicine
Director of Vascular Research
William R. Hiatt Endowed Chair in Cardiovascular Research
University of Colorado School of Medicine

Lp(a) in Practice: Challenges in Testing and Opportunities in Care

Leslie J. Donato, Ph.D., D (ABCC)
Co-Director Clinical Specialty Laboratory
Co-Director Hospital Clinical Laboratory and Point of Care
Associate Professor, Laboratory Medicine and Pathology
Department of Laboratory Medicine and Pathology at Mayo Clinic

Howard S. Weintraub, MD, FACC, FAHA, FASPC
Clinical Professor of Medicine
NYU Grossman School of Medicine
Clinical Director, NYU Center for the Prevention of Cardiovascular Disease



Lipoprotein (a): Myths Vs. Facts

Myth 1: If I know my LDL "bad" cholesterol number, I don't need to have my Lp(a) tested.

Fact: Lipoprotein (a), commonly abbreviated as Lp(a), and LDL cholesterol are not the same. While both can contribute to heart disease, they are different in their composition and potential impact on increasing the risk of heart disease. LDL primarily consists of cholesterol esters and apolipoprotein B on its surface. Lp(a) shares a similar composition with LDL but contains an additional protein called apolipoprotein (apo) attached to its core. This difference in their structures introduces unique properties to Lp(a), potentially leading to increased plaque buildup, inflammation and blood clotting in the arteries due to the similarity of Lp(a) to plasminogen, a protein involved in blood clotting regulation.

You could have a normal LDL number and a high Lp(a) level. Since the regular cholesterol test doesn't include Lp(a), ask your doctor about getting an Lp(a) test.

Myth 2: I don't need to know my Lp(a) level because it doesn't affect my health.

Fact: Too much Lp(a) in your arteries can cause the accumulation of fatty deposits, known as plaques, which narrow arteries and reduce blood flow. If a piece of the plaque breaks free, it can block blood flow to vital organs such as the heart, brain, kidneys, lungs, and other parts of the body. This can lead to various conditions including heart disease, coronary artery disease, aortic aneurysm, peripheral artery disease (PAD), and stroke. Therefore, having high Lp(a) levels can significantly impact your health.

Myth 3: I don't have any symptoms, so I don't need to get my Lp(a) tested.

Fact: Many people don't have symptoms until they experience a serious event such as heart attack or stroke. Since Lp(a) levels are mostly determined by genetics, you could have high Lp(a) even if you maintain a healthy lifestyle and control all other heart disease risk factors. Talk to your doctor if you have:

- Known family history of high Lp(a)
- Family or personal history of heart disease or premature cardiovascular disease
- Diagnosis of familial hypercholesterolemia (FH) - an inherited condition where the body poorly regulates LDL or bad cholesterol

Myth 4: Just because a close relative has high Lp(a), it doesn't mean my...

Fact: Lp(a) is a common risk factor in your genes, and tested as well. The risk of heart screening and specific needs.

Understanding the Lp(a) Test

- What should prompt a talk with my health care professional about a screening?**
 - Known family history of high Lp(a)
 - Family or personal history of heart disease or premature cardiovascular disease
 - Diagnosis of familial hypercholesterolemia (FH) - inherited condition that causes the body to poorly regulate LDL or bad cholesterol
- How do I get screened?**
 - The standard cholesterol test, also known as a lipid panel, does not include Lp(a). Talk to your health care professional about screening for Lp(a).
 - Next, get a simple blood test that can be done at your doctor's office or diagnostic lab center.
- What do the results mean?**
 - Levels higher than 50 mg/dL (5.2 mmol/L) are considered to be high.
 - A high Lp(a) level increases the risk of heart attack, stroke, peripheral artery disease (PAD), and aortic aneurysm.
- Will my health insurance cover the test?**
 - Lp(a) is a genetic risk factor. If you have high Lp(a), encourage your family members to get tested. Ask your doctor about genetic screening. Other genetic testing done for your specific risks.
 - Will insurance plans cover Lp(a) testing? If you're unsure about your insurance coverage, finding your insurance provider them with PDF code 8886 for test can help clarify.
 - Your health insurance will cover the Lp(a) test. Your health care professional may be able to assist you in finding suitable options.


Thoughtful Talks with My Health Care Professional: Understanding My Lp(a) Risk

Bring this sheet to your appointment and discuss the following questions.

REVIEW MY PERSONAL & FAMILY HISTORY	UNDERSTANDING MY NUMBER	ASSESS MY HEART DISEASE RISK
<p>Lp(a) stands for lipoprotein (a) and is a genetically inherited independent risk factor for heart disease. Discuss with your health care professional if you have any of the following:</p> <ul style="list-style-type: none"> <input type="checkbox"/> Known family history of high Lp(a) <input type="checkbox"/> Family or personal history of heart disease or premature coronary artery disease (defined as younger than 45 for men and 55 for women) <input type="checkbox"/> Diagnosis of familial hypercholesterolemia (inherited condition that causes the body to poorly regulate LDL or bad cholesterol) <p>Notes:</p>	<p>Once you've been screened, ask your health care professional:</p> <p>My Lp(a) number: _____</p> <p>What does my Lp(a) number mean? _____</p> <p>What level is considered to be high? _____</p> <p>Does anything contribute to a high Lp(a) number? _____</p> <p>Should I encourage my family members to get screened? _____</p>	<p>Do you think I'm at risk for a heart attack or stroke? _____</p> <p>What else contributes to my risk? _____</p> <p>EXPLORE TREATMENTS</p> <p>Although Lp(a) is not affected by lifestyle changes, it is still important to lower your overall risk of heart attack, stroke, and peripheral artery disease.</p> <p>What lifestyle changes can I make to lower my risk for heart disease? _____</p> <p>What resources can help me learn more about Lp(a) and heart disease? _____</p>
<p><input checked="" type="checkbox"/> If so, ask if you should be screened for Lp(a).</p>		

Remember if you have a high Lp(a), you didn't do anything to cause it, and now that you know, take control and reduce your overall heart disease risk! Learn more at heart.org/lpa

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Lipoprotein(a) Awareness Day Webinar Case Discussion

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Disclosures

Donald M. Lloyd-Jones, MD, ScM

FINANCIAL DISCLOSURE:

No relationship with industry. Part-time employee of AHA

UNLABELED/UNAPPROVED USES DISCLOSURE:

None

Frank Qian, MD, MPH

FINANCIAL DISCLOSURE:

None

UNLABELED/UNAPPROVED USES DISCLOSURE:

None

Case 1

- Ms. L is a 65-year-old woman who presents to clinic after a **recent hospitalization for acute stroke**
- She developed sudden-onset right arm weakness and expressive aphasia while eating dinner with her family, which prompted her to call for an ambulance. Symptoms resolved shortly before her arrival in the emergency room.
- MRI brain showed a focal **acute left MCA territory infarct** without hemorrhagic conversion
- CTA head and neck showed **an 80% stenosis of the left internal carotid artery**, and she underwent an uncomplicated carotid endarterectomy
- Transthoracic echocardiogram showed mild left ventricular hypertrophy and mild aortic valve calcification without stenosis
- In-hospital and post-discharge cardiac monitoring did not demonstrate evidence of atrial fibrillation

Case 1 (cont.)

➤ Past Medical History:

- Hypertension, treated
- Hyperlipidemia, treated
- Former smoker (20 pack-years, quit 10 years ago)
- Family history of heart attack and stroke on her father's side (all after the age of 65)

➤ Home Medications:

- Aspirin 81 mg daily
- Atorvastatin 10 mg daily (myalgias with higher doses)
- Lisinopril 20 mg daily

➤ Physical Exam:

- BP: 135/85
- BMI: 29 kg/m²

➤ Laboratory Values:

- HbA1c: 5.3%

➤ Lipid panel:

- Total cholesterol: 194 mg/dL
- LDL cholesterol (calculated): 128 mg/dL
- HDL cholesterol: 50 mg/dL
- Triglyceride: 80 mg/dL
- **Lp(a): 299 nmol/L**

Case 1 Management Discussion

- At hospital discharge, in addition to aspirin, she was initiated on clopidogrel 75 mg daily and atorvastatin was switched to rosuvastatin 40 mg daily
- At the 3-month follow-up, her LDL cholesterol is now 65 mg/dL
- What would you recommend for her lipid-lowering strategy given her elevated Lp(a)?
- What other therapies would you consider?
- What would you do about her aortic valve calcification?

Case 2

- Mr. P is a 40-year-old man who is referred to lipid clinic for an elevated Lp(a)
- He was recommended for testing after his brother had a MI at the age of 45 years
- He exercises 3-4 times/week (running and stationary bike) with no exertional symptoms
- He is interested in learning about ways to reduce his cardiovascular disease risk

Case 2 (cont.)

➤ Past Medical History:

- Seasonal allergies
- Never smoker

➤ Home Medications:

- No regular medications

➤ Physical Exam:

- BP: 122/76
- BMI: 27 kg/m²

➤ Laboratory Values:

- HbA1c: 4.7%
- Cr 1.01 (eGFR: 96 ml/min/1.73 m²)

➤ Lipid panel:

- Total cholesterol: 201 mg/dL
- LDL cholesterol (calculated): 120 mg/dL
- HDL cholesterol: 59 mg/dL
- Triglyceride: 110 mg/dL
- **Lp(a): 193 nmol/L**

➤ PREVENT:

- 10-year total CVD: 1.1%
- 10-year ASCVD: 0.8%
- 30-year total CVD: 8.3%
- 30-year ASCVD: 5.3%

Case 2 Management Discussion

- Given his low 10 and 30-year CVD risk, would you consider any other testing to aid in risk stratification?
- What should his LDL goal be and would you consider initiating statin therapy now, particularly given his strong family history?
- Would you recommend cascade testing for his children?

Case 3

- Ms. A is a 25-year-old woman who presents to primary care clinic to establish care
- She inquires about cardiovascular risk assessment as she has had multiple family members on her father's side who had cardiovascular events in their early 50s
- She exercises 5 times/week (swimming and light weights) with no exertional symptoms
- She inquired about Lp(a) testing, as she read in an online article about its ability to help predict who will develop cardiovascular disease

Case 3 (cont.)

➤ **Past Medical History:**

- Never smoker

➤ **Home Medications:**

- No regular medications

➤ **Physical Exam:**

- BP: 110/70
- BMI: 23 kg/m²

➤ **Laboratory Values:**

- HbA1c: 4.8%
- Cr 0.75 (eGFR: 113 ml/min/1.73 m²)

➤ **Lipid panel:**

- Total cholesterol: 185 mg/dL
- LDL cholesterol (calculated): 107 mg/dL
- HDL cholesterol: 62 mg/dL
- Triglyceride: 80 mg/dL

➤ **PREVENT:**

- 10-year total CVD: 0.3%
- 10-year ASCVD: 0.3%
- 30-year total CVD: 2.2%
- 30-year ASCVD: 1.5%

Case 3 Management Discussion

- Would you recommend Lp(a) testing for her and how should you counsel her on the results?
- If her Lp(a) was found to be elevated, what should her LDL goal be and would you consider any preventative therapies now, particularly given her strong family history?
- What if her LDL was elevated to 170 mg/dL?
- Would you consider other risk assessment approaches?



Thank you for joining American Heart Association's Lipoprotein(a) Awareness Day Webinar!



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Questions and Discussion

