Open Your Heart: Optimizing Cardiovascular Disease Risk Factors Among People With HIV



Suman Srinivasa, MD, MS
Assistant Professor of Medicine
Division of Endocrinology
Harvard University
Massachusetts General Hospital
Boston, MA

IAS–USA

1

Financial Relationships With Ineligible Companies (Formerly Described as Commercial Interests by the ACCME) Within the Last 2 Years

Dr Srinivasa reported serving as a consultant to Parabilis Medicines, Inc, and receiving grants paid to her institution from Merida Biosciences, Biohaven Pharmaceuticals, Inc, and Viking Therapeutics, Inc. (Updated October 18, 2026)

Slide 2

2

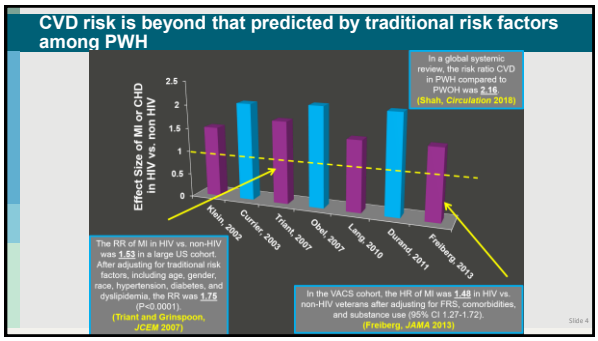
Learning Objectives

After attending this presentation, learners will be able to:

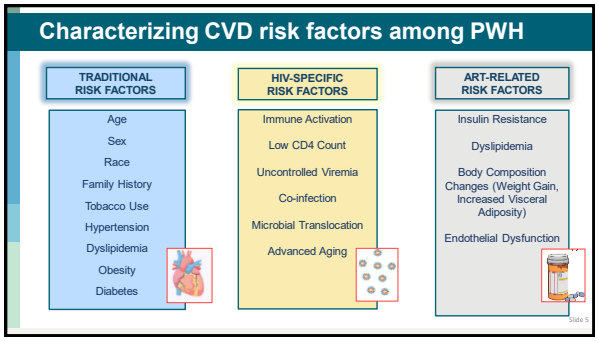
- Identify key risk factors for cardiovascular disease among people with HIV
- Assess cardiovascular risk profiles among people with HIV
- Implement the new 2026 American College of Cardiology/ American Heart Association dyslipidemia guidelines in clinical practice
- Manage therapies to reduce cardiovascular risk

Slide 3

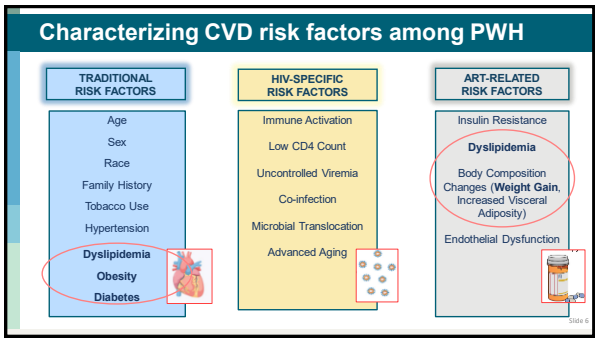
3


CVD risk is beyond that predicted by traditional risk factors among PWH
Effect Size of MI or CHD in HIV vs. non HIV
2.5
2
1.5
1
0.5
0
Klein, 2003
Currier, 2003
Triant, 2007
Obel, 2007
Lang, 2010
Durand, 2011
Freiberg, 2013
In a global systemic review, the risk ratio CVD in PWH compared to PWOH was 2.16 (Shah, Circulation 2018)
The RR of MI in HIV vs. non-HIV was 1.53 in a large US cohort. After adjusting for traditional risk factors, including age, gender, race, hypertension, diabetes, and dyslipidemia, the RR was 1.79 (P<0.0001). (Triant and Grinspoon, JCEM 2007)
In the VACS cohort, the HR of MI was 1.48 in HIV vs. non-HIV veterans after adjusting for FRS, comorbidities, and substance use (95% CI 1.27-1.72). (Freiberg, JAMA 2013)
Slide 4

4


Characterizing CVD risk factors among PWH
TRADITIONAL RISK FACTORS
Age
Sex
Race
Family History
Tobacco Use
Hypertension
Dyslipidemia
Obesity
Diabetes
HIV-SPECIFIC RISK FACTORS
Immune Activation
Low CD4 Count
Uncontrolled Viremia
Co-infection
Microbial Translocation
Advanced Aging
ART-RELATED RISK FACTORS
Insulin Resistance
Dyslipidemia
Body Composition Changes (Weight Gain, Increased Visceral Adiposity)
Endothelial Dysfunction
Slide 5

5

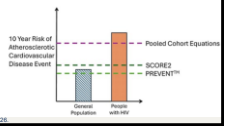

Characterizing CVD risk factors among PWH
TRADITIONAL RISK FACTORS
Age
Sex
Race
Family History
Tobacco Use
Hypertension
Dyslipidemia
Obesity
Diabetes
HIV-SPECIFIC RISK FACTORS
Immune Activation
Low CD4 Count
Uncontrolled Viremia
Co-infection
Microbial Translocation
Advanced Aging
ART-RELATED RISK FACTORS
Insulin Resistance
Dyslipidemia
Body Composition Changes (Weight Gain, Increased Visceral Adiposity)
Endothelial Dysfunction
Slide 6

6

Using the AHA/ACC PREVENT Calculator to assess ASCVD Risk

The American Heart Association PREVENT™ Online Calculator

Risk	PREVENT 10-year ASCVD risk	PCE 10-year ASCVD risk
Low	<3%	<5%
Borderline	3 to 5%	5 to 7.5%
Intermediate	5 to <10%	7.5 to 20%
High	≥10%	≥20%



The PREVENT ASCVD score reclassifies PWH into lower risk categories than PCE and underestimates risk even more!

Underprediction among women and Black people

https://professional.heart.org/en/guidelines-and-statements/prevent-calculator; Grimspoon, Zanni *Lancet HIV* 2025; Dunsterville *CID* 2026

7

REPRIEVE

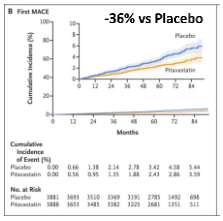
Randomized Trial to Prevent Vascular Events in HIV

ORIGINAL ARTICLE

Pitavastatin to Prevent Cardiovascular Disease in HIV Infection

PWH ages 40-75 on ART
Excluded for ASCVD
Low-to-moderate risk: mean ASCVD risk score 4.5% by PCE
Median screening LDL 108 mg/dL

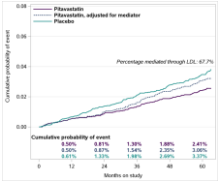
Pitavastatin was evaluated in the REPRIEVE trial because its lack of metabolism through P450 CYP3A4 reduces the risk of potential drug interactions compared with other statin therapies.



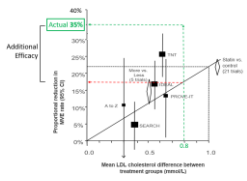
Grimspoon, *NEJM* 2024

8

CVD Protection from statins use likely extends beyond LDL-lowering among PWH



Pitavastatin effect on MACE was largely (40-68%), but not entirely, mediated through LDL-lowering



LDL lowering matters, and the statin effect is beyond what is expected for LDL lowering alone

?additional effects on inflammatory, metabolic, and vascular pathways
Ideal strategy: target lipid goals and inflammation,
no clear targets, but emerging REPRIEVE data suggests lowest incidence of MACE with LDL<70 and hsCRP<2

Hopkins *Lancet HIV* 2026; *Lancet* 2010; 376: 1670-81; REPRIEVE data in progress

9

Featuring the new 2026 ACC/AHA multisociety dyslipidemia guidelines

CLINICAL PRACTICE GUIDELINE

2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Dyslipidemia

A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Developed in Collaboration With and Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, Association of Black Cardiologists, American College of Preventive Medicine, American Diabetes Association, American Geriatrics Society, American Pharmacists Association, American Society for Preventive Cardiology, National Lipid Association, and Preventive Cardiovascular Nurses Association

Slide 10

10

New insights from the 2026 ACC/AHA multisociety dyslipidemia guidelines

- Treat dyslipidemia earlier to reduce lifelong risk of prolonged exposure to atherogenic lipoproteins.
- LDL and non–HDL **treatment goals** are back to guide lipid-lowering therapy (LLT)
- **Apolipoprotein B (ApoB)** testing can be useful to improve risk assessment and guide therapy once LDL and non–HDL goals are met, particularly in those with ASCVD, elevated triglycerides (TG ≥150 mg/dL), diabetes, or CKM
- **Lipoprotein(a) [Lp(a)]** can be measured at least once to identify those individuals at higher risk of ASCVD.
- **Selective use of CV imaging for subclinical atherosclerosis** for men ≥ 40 and women ≥ 45 can improve risk assessment and guide LDL and non–HDL goals.

lower, sooner, and for longer

total *lifetime* burden of LDL cholesterol — how high it is, multiplied by how many years you carry it — drives atherosclerosis.

Slide 11

11

Statins are now recommended for primary prevention of CVD among PWH 40-75 years– regardless of ASCVD risk score

4.2.8.9. Persons Living With HIV

Recommendation for Persons Living With HIV
Referenced studies that support the recommendation are summarized in the Evidence Table.

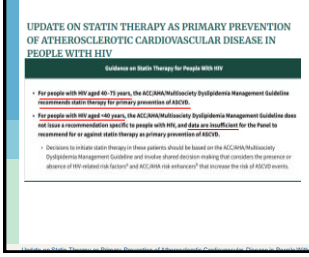
COR	LOE	Recommendation
1	B-R	1. In people living with HIV aged 40 to 75 y on stable combination antiretroviral therapy, statin therapy is recommended to reduce risk of a first ASCVD event and reduce the rate of coronary atherosclerosis progression.[1]

ASCVD: history of acute coronary syndromes (ACS), myocardial infarction (MI), Stable or unstable angina, coronary or other arterial revascularization, stroke, transient ischemic attack (TIA), or peripheral artery disease (PAD)

Slide 12

12

In PWH<40 years, decision to initiate statin may be based on HIV-related or general risk enhancers


UPDATE ON STATIN THERAPY AS PRIMARY PREVENTION OF ATHEROSCLEROTIC CARDIOVASCULAR DISEASE IN PEOPLE WITH HIV

Guidance on Statin Therapy for People With HIV

- For people with HIV aged 40–75 years, the ACC/AHA/Multisociety Dyslipidemia Management Guideline recommends statin therapy for primary prevention of ASCVD.
- For people with HIV aged >40 years, the ACC/AHA/Multisociety Dyslipidemia Management Guideline does not issue a recommendation specific to people with HIV, and data are insufficient for the Panel to recommend for or against statin therapy as primary prevention of ASCVD.
  - Decisions to initiate statin therapy in these patients should be based on the ACC/AHA/Multisociety Dyslipidemia Management Guideline and involve shared decision-making that considers the presence or absence of HIV-related risk factors[a] and ACC/AHA risk enhancers[b] that increase the risk of ASCVD events.

HIV-specific risk enhancers:
-prolonged duration of HIV infection
-delayed ART initiation
-long periods of HIV viremia and/or ART nonadherence
-low current or nadir CD4 (<350 cells/mm2)
-exposure to older ART with cardiometabolic toxicity
-HCV coinfection.

Table 13. Risk Enhancers

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) (Section 4.2.3.5, "Polygenic Risk Scores")
Chronic inflammatory diseases (eg, systemic lupus, rheumatoid arthritis, advanced psoriasis, inflammatory arthritis)
Lp(a) ≥195 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[a]
Reproductive risk markers (premature menopause, preeclampsia, gestational diabetes, gestational hypertension, preterm delivery; Section 4.3.3.4, "Reproductive Risk Markers")

Slide 13

13

What LDL and non-HDL to achieve—treatment goals are back for intensification

PREVENT 10yr Risk Category	Treatment Goals	Statin Intensity
Low <3%	LDL<160: counseling	n/a
Low <3%	LDL≥160: LDL<100 non-HDL<130	Moderate
Borderline 3 to 5%	LDL<100 non-HDL<130	Moderate
Intermediate 5 to <10%	LDL<100 non-HDL<130	Moderate to High
High ≥10%	LDL<70 non-HDL<100	High
Very High Risk	LDL<55 non-HDL<85	High

- Check lipids 4-12 weeks after initiation
- Then, every 6-12 months thereafter

****Reminder:**
PREVENT ASCVD score reclassifies PWH into lower risk categories than PCE and underestimates risk even more!

REPRIEVE data suggests lowest incidence of MACE with LDL<70 and hsCRP<2

Slide 14

14

Choosing statin therapy based on intensity



Slide 15

15

Drug-Drug interactions (DDI) with statins are relevant to HIV care

Preferred Statins for use with ART

Statin	ART Consideration
Pitavastatin	Not metabolized by CYP3A4 enzymes Least likely to interact with ART
Atorvastatin	Partially metabolized by CYP3A4 Max dose 20mg PI, Cobicistat, Lencapavir
Rosuvastatin	Not metabolized by CYP3A4 enzymes Max dose 20mg with PI, Cobicistat, Lencapavir
Pravastatin	Not metabolized by CYP3A4 enzymes, but has reduced efficacy
Lovastatin	Highly metabolized by CYP3A4, C/I use with PI, Cobicistat, Lencapavir
Simvastatin	Highly metabolized by CYP3A4, C/I use with PI, Cobicistat, Lencapavir

PI (including ritonavir containing ART) and cobicistat are CYP3A4 inhibitors and may decrease metabolism of statins. Start at lower statin dosing with these medications to avoid side effects.

Some NNRTI are CYP3A4 inducers and may increase the metabolism of statins.

Lencapavir is a moderate CYP3A4 inhibitor and may decrease metabolism of statins.

Slide 16

16

Carefully evaluate statin intolerance to determine whether an alternative strategy is needed

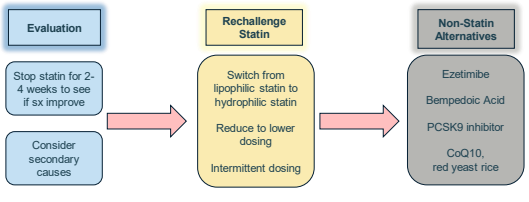
- ACC's Statin Intolerance Tool: https://tools.acc.org/LDL/StatinIntolerance/#!/
- Evaluate for secondary causes: TSH, ALT, 25OHD, electrolytes, ESR, other relevant medical history
- If severe myalgias or muscle weakness, measure CK and consider eval for rhabdomyolysis (Cr, UA)
- Verify statin-attributed muscle symptoms with side effects on ≥2 statins, 1 at lowest FDA-approved dose.

	Possibly Related	Unlikely Related
Symptom Type	Muscle ache, Weakness, Soreness, Stiffness, Cramping, Tenderness, or General Fatigue	Tingling, Twitching, Shooting Pain, Nocturnal Cramps, or Joint Pain
Symptom Area	Bilateral	Unilateral
Symptom Timing	Within 4 weeks of starting statin	Before starting statin After 4 weeks of starting statin

Slide 17

17

Consider statin rechallenge or non-statin alternatives when managing statin intolerance



Hydrophilic (less risk of myopathy): Rosuvastatin, Pravastatin
Lipophilic: Atorvastatin, Pitavastatin, Simvastatin, Lovastatin, Fluvastatin

Slide 18

18

Alternative LDL-lowering therapies to be considered if statin intolerance, drug–drug interactions, or inadequate LDL lowering

Class	Name	Mechanism of Action	Expected LDL Reduction	CV Outcomes Data
Cholesterol absorption inhibitor	Ezetimibe	inhibit intestinal and biliary sterol absorption	18%	Reductions in CV events in very high-risk secondary prevention in combination with statin (IMPROVE-IT)
PCSK9 Inhibitor: Monoclonal Ab	SQ: Alirocumab Evolocumab PO: enlicitide	binds to PCSK9 in the circulation and decreases degradation of LDL receptors	45-64%	Evolocumab/Alirocumab led to reductions in CV events in very high-risk secondary prevention in combination with statin (FOURIER, ODYSSEY). Evolocumab led to a lower risk of first CV events in atherosclerosis or diabetes and without a previous MI or stroke (VESALIUS-CV).
PCSK9 siRNA	Inclisiran	directs catalytic breakdown of mRNA for PCSK9 and decreases degradation of the LDL receptor	48–52%	Limited data shows no difference with placebo; other studies in progress
ATP citrate lyase inhibitor	Bempedoic acid	Inhibits ATP citrate lyase in the liver to decrease cholesterol production	21-24%	Reductions in CV events in individuals treated for high-risk primary and secondary prevention with statin-attributed side effects.

Cannon, NEJM 2015; CVDreamline Consortium, 2022; Schmidt, NEJM 2018; Ridker, NEJM 2025; Nissen, JAMA, 2023; Nissen, JAMA, 2023

19

There are limited studies of alternative LLT among PWH

PCSK9 inhibitors

BEIJERINCK trial
PWH on maximally tolerated statin
Evolucumab **reduced LDL by 56.9%** vs. placebo after 6 months
Achieved LDL<70 mg/dL in 73% vs 8%

TriNetX (retrospective)
PCSK9 inhibitor use was linked to **9% lower odds of all-cause mortality** compared to non-use
No association to MI or CVA

GLAGOV Trial
PWH on statin and angiographic CAD
Evolucumab **achieved plaque regression in 64%** vs. 47% in placebo
Lower mean time weighted LDL 36.6 mg/dL

Bempedoic Acid

CLEAR HIV Trial *(RCT in progress)*
PWH≥40 with CVD or 1+ CVD risk factor
Most statin use permitted
Assess inflammation, lipids, plaque

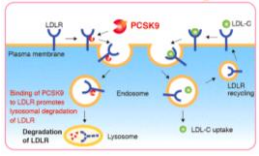
Outpatient experience in European cardiology clinic:
PWH high risk on statin-ezetimibe, not at ESC goal LDL 55
Reduced LDL by 39% after 3 months

Slide 20

Data: EHJ 2024 Review; JACC 2020; Nichols, JAMA 2018/J EAR Test (R); Priscilla Heart; Curtis, EHJ 2024

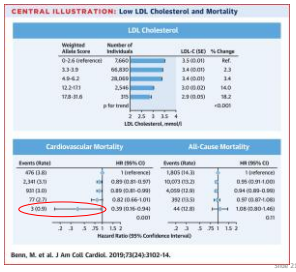
20

Insights derived from genetics: Low LDL expected with PCSK9 variants reduces cardiovascular mortality



Gain of Function PCSK9 mutations
↑ increased ASCVD

Loss of Function PCSK9 mutations
↓ decreased ASCVD



Benn, M. et al. J Am Coll Cardiol. 2019;73(24):3102-14.

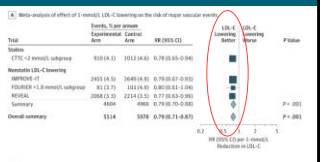
Slide 21

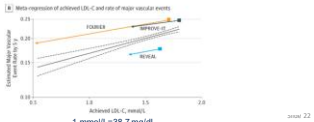
21

LDL: How low can you go? There is no too low.

Efficacy and Safety of Further Lowering of Low-Density Lipoprotein Cholesterol in Patients Starting With Very Low Levels
A Meta-analysis

lower, sooner, and for longer
total *lifetime* burden of LDL cholesterol — how high it is, multiplied by how many years you carry it — drives atherosclerosis.





Consistent relative risk reduction in major vascular events (even as low as 21 mg/dL)

LDL lowering was not associated with an increased risk of serious adverse event.

1-mmol/L=38.7 mg/dL

22

Selective use of CV imaging for subclinical atherosclerosis to manage lipid lowering therapy—general population

CALCIFIED PLAQUE?

- If initiation uncertain

In intermediate risk and select borderline risk with no prior ASCVD:

CAC score=0, reasonable to defer therapy and reassess in 3-7yrs

CAC score>0, recommended to initiate LLT, particularly if the CAC score is ≥100

- If intensification uncertain:

In intermediate or high risk with no prior ASCVD:

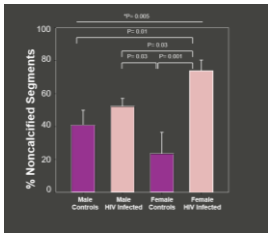
CAC score can be useful to refine treatment goals and decide whether to intensify LLT

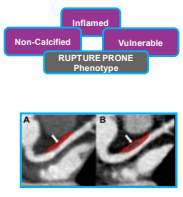
CAC score is not for routine monitoring after statin therapy

CAC Score	Treatment Goals	Statin Intensity
0 Absent	LDL<160: counseling	n/a
1-99 Mild	LDL<100 non-HDL<130	Moderate
100-299 Moderate	LDL<70 non-HDL<100	Moderate to High
300-999 Severe	LDL<70 non-HDL<100 Consider: LDL<55 non-HDL<85	Moderate to High
>1000 Very Severe	LDL<55 non-HDL<85	High

23

Non-calcified plaque, a highly inflamed and rupture prone phenotype, is increased among persons with HIV





24

Selective use of CV imaging for subclinical atherosclerosis to manage lipid lowering therapy—specific to HIV

NON-CALCIFIED PLAQUE?

Recommendations for Selective Imaging of Subclinical Atherosclerosis (Men ≥40 or Women ≥45 Years) (Continued)

COR	LOE	Recommendations
2b	B-NR	6. In adults with no prior ASCVD who are likely to have a high burden of noncalcified plaque (eg, inflammatory disorders, persons living with HIV, and diabetes), selective use of coronary computed tomography angiography (CCTA) may be useful to inform risk assessment and guide decisions regarding treatment intensity of LLT.[13–17]

Slide 25

25

ApoB: Features that Promote CVD Risk

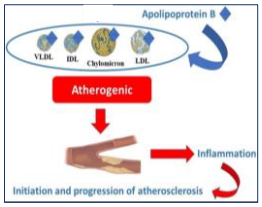
ApoB: structural protein found on all atherogenic (plaque-forming) lipoprotein particles in the blood.

May predict CVD risk better

ApoB ≥130 mg/dL is considered a risk-enhancing factor for ASCVD

Recommendations for Measurement of ApoB
Referenced studies that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
2a	B-NR	1. In adults on LLT, particularly those with ASCVD, DM, symptoms, type 2 diabetes, and/or elevated LDL measurement of apoB is reasonable to guide decisions regarding further therapeutic intensification once LDL-C and/or non–HDL-C goals are achieved.[1–4]
2b	B-NR	2. In adults not on LLT, measurement of apoB may be reasonable to enhance ASCVD risk assessment, guide decisions about initiation of LLT, and characterize inherited lipid disorders.[5a–5d]

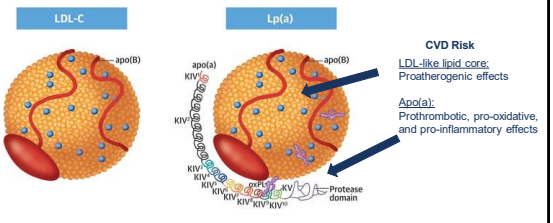


Slide 26

Figure adapted from Ouweneel AB, et al. J Mol Biol. 2022.

26

Lp(a): Features that Promote CVD Risk



Slide 27

Figure adapted from https://b100method.com/blogs/heart-health-lipoprotein-a

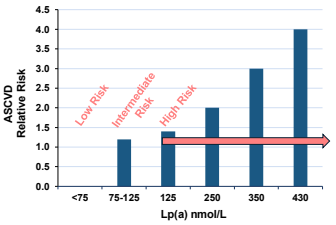
27

Increased Lp(a) contributes to ASCVD relative risk

Recommendations for Measurement of Lp(a)
Referenced studies that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	B-NR	1. In all adults, measurement of Lp(a) concentration is recommended at least once for ASCVD risk assessment.[1-4]

An Lp(a) concentration of 125 nmol/L (50mg/dL) is considered high and is associated with approximately a 40% relative risk increase in ASCVD



Slide 28

28

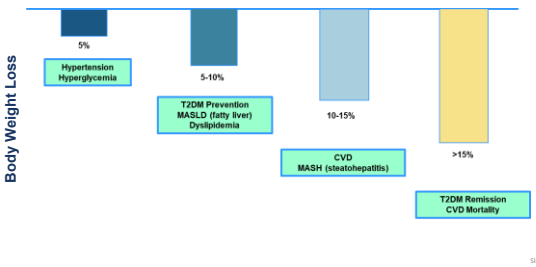
Effects of Available Lipid-Lowering Therapies on Lp(a)

Class	Name	Mechanism of Action	Expected Lp(a) Effects	CV Outcomes Data
Statin			Increase 8-24% overridden by LDL lowering benefit	
PCSK9 Inhibitor: Monoclonal Ab *not FDA approved indication	SQ: Alirocumab Evolocumab	Increases Lp(a) clearance	Decrease 14-35%	FOURIER and ODYSSEY trials demonstrate ASCVD risk reduction in recent/established ASCVD with LDL≥70 on statin
Lp(a) Apheresis		Extracorporeal binding of Lp(a)	Decrease 64%	
Antisense Oligonucleotide	SQ: Pelacarsen	Targets the LPA gene, which encodes Lp(a)	Decrease 64%	No reduction in MACE ***Lowering Lp(a) alone, even with potent reduction, did not translate into reduced CVD risk in this high-risk population*

For now, COUNSEL, consider universal Lp(a) testing and continue aggressive management of other ASCVD risk factors.
Limited in management as no approved targeted Lp(a)-lowering therapy yet.

29

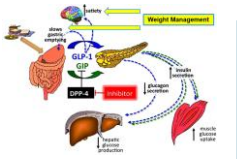
Greater weight loss leads to increased metabolic benefits



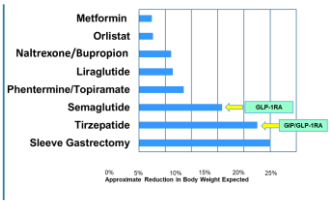
Slide 30

30

Comparison of efficacy of weight loss therapies



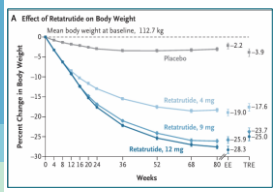
Incretin hormones: GLP-1, GIP
Secreted after nutrient intake.
Act to decrease glucose, suppress appetite, and delay gastric emptying



Slide 31

31

TRIUMPH-1 Trial: Retatrutide in Obesity (Not FDA Approved)



Retatrutide is a triple-receptor agonist of GIP, GLP-1, and glucagon receptors.

Adults with obesity excluded for DM with
BMI ≥30 kg/m^2 or
BMI 27-29.9 kg/m^2 + weight-related comorbidities

Mean %change in body weight:
- −17.6% (4-mg dose)
- −23.7% (9-mg dose)
- **−25.0% (12-mg dose)**
- −3.9% (placebo group)

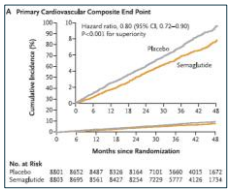
Most common side effect remains GI

Slide 32

32

SELECT Trial: GLP-1RA and cardiovascular outcomes in obesity

Participants had BMI≥27, **Pre-existing CVD (secondary prevention),** and were excluded for DM



Primary CV endpoint:
composite death from CV causes, nonfatal MI, or nonfatal CVA

6.5% in the semaglutide SQ group
8.0% in the placebo group

***20% risk reduction with semaglutide SQ**

Consider addition of semaglutide to adults with overweight/obesity and established CVD to reduce the risk of cardiovascular death, non-fatal heart attack, and non-fatal stroke

33

Using GLP-1RAs for Overweight/Obesity

Chronic Weight Management Indications

- BMI ≥30 kg/m² **OR**
- BMI 27-29.9 kg/m² with weight-related comorbidities (CVD, dyslipidemia, HTN, OSA, T2DM)

+

Have not met weight-loss goals (5% loss of total body weight over 3-6 months) with comprehensive lifestyle intervention alone

GLP-1RA

Semaglutide SQ weekly or oral daily
obesity (Weygovy, max dose 7.2mg SQ or 25mg PO)
also FDA approved for established CVD or MASH

Liraglutide SQ daily
obesity (Saxenda, max dose 3.0mg SQ)

Orforglipron oral daily (max dose 17.2mg PO)

GIP/GLP-1RA

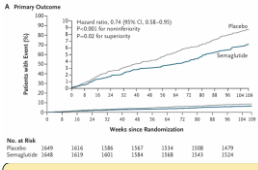
Tirzepatide SQ weekly
obesity (Zepbound, max dose 15mg)
also FDA approved for moderate/severe OSA

Slide 34

34

SUSTAIN-6 Trial: Semaglutide and cardiovascular outcomes in DM

Participants had T2DM, **Pre-existing CVD (secondary prevention) or age≥60+subclinical CVD**



Primary CV endpoint:
composite death from CV causes, nonfatal MI, or nonfatal CVA

6.6% in the semaglutide group
8.9% in the placebo group

***26% risk reduction with semaglutide**

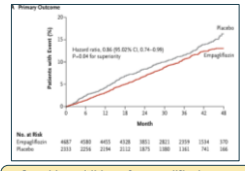
Consider addition of dulaglutide, liraglutide, semaglutide (subcutaneous or oral), or tirzepatide in patients with ASCVD + T2DM given demonstrated cardiovascular benefits

Slide 35

35

EMPA-REG OUTCOME Trial: SGLT2i and cardiovascular outcomes in DM

Participants had T2DM, **Pre-existing CVD (secondary prevention)**



Primary CV endpoint:
composite death from CV causes, nonfatal MI, or nonfatal CVA

10.5% in the empagliflozin group
12.1% in the placebo group

***14% risk reduction with empagliflozin**

Consider addition of empagliflozin or canagliflozin or dapagliflozin (SGLT2i) in patients with ASCVD + T2DM, chronic kidney disease, or heart failure given demonstrated cardiovascular and renal benefits

Slide 36

36

Take Aways

- CVD in increased 1.5-2 fold among PWH even after adjusting for traditional CVD risk factors.
- Management of CVD in HIV requires addressing traditional, HIV-specific, and ART-specific risk factors.
- Per new 2026 guidelines, statin therapy is recommended for PWH ages 40-75 yrs for primary prevention to reduce the risk of an ASCVD event regardless of ASCVD risk score. This identifies a new subgroup for whom statin therapy would have not have previously been recommended.
- Newest message for LDL-lowering: **lower, sooner, and for longer**
- In statin intolerant patients who may warrant LLT, consider use of PCSK9 inhibitor or bempedoic acid for either primary or secondary prevention
- Apo(B), Lp(a) and selective use of cardiac imaging (CCTA) may be considered to help risk stratify further. PWH have a higher burden of non-calcified plaque, which is highly inflamed and more likes to rupture.
- Consider use of GLP-1RA for use in obesity or diabetes and SGLT2i for use in diabetes to reduce CVD risk (secondary prevention)

Slide 37

37

Q and A Session

IAS–USA

38