Cardiovascular Complications in People With HIV

Updated: October 27, 2026
Reviewed: October 27, 2026

Key Considerations and Recommendations

HIV-Related ASCVD Risk and Antiretroviral Management

- All people with HIV should maintain viral suppression with continuous ART to reduce the risk of ASCVD **(AII).**
- Due to data supporting an increased risk of cardiovascular events, alternative ARV medications should be considered in place of abacavir and lopinavir/ritonavir in people with higher underlying ASCVD risk or known ASCVD **(AII).**

ASCVD Risk Estimation and Lipid Screening

- Lipid screening should be performed in people with HIV **(AIII),** with the following suggested frequency: (1) at entry into care or at the time of ART initiation, (2) 3–6 months after ART initiation or modification once viral suppression is achieved, (3) annually for those receiving statin therapy, (4) every 1–3 years for those not on statin therapy, and (5) with changes in ASCVD risk factors (see text for additional details on monitoring).
- For people with HIV aged 30–79 years, 10-year ASCVD risk should be estimated using the PREVENT-ASCVD equations. ASCVD risk should be calculated whenever lipids are measured, including changes in traditional CVD risk factors or ARV regimen (see text for additional details on risk estimation) **(BIII).**
  - Estimates can be generated using the ACC **ASCVD Risk Estimator**.
  - The PREVENT-ASCVD equations have been shown to underestimate ASCVD risk among people with HIV (see text for additional details).
- When compared to the general population, women with HIV have both a greater increase in relative risk of ASCVD and a greater underprediction of ASCVD by risk scores than men with HIV. For women with HIV, it is critical to ensure optimal ASCVD risk factor modification regardless of ASCVD risk score, implement ASCVD prevention strategies, and perform diagnostic evaluation for possible cardiac symptoms.

ASCVD Primary Prevention Strategies

- All people with HIV should be counselled about ways to reduce ASCVD risk through behavioral modifications, such as healthy diet, weight control, regular exercise, smoking cessation, and limiting alcohol consumption.
- Optimal management of comorbidities (e.g., hypertension, diabetes, obesity) remains important to further reduce ASCVD risk among people with HIV.
- The use of immunomodulatory therapy to reduce HIV-associated ASCVD risk is not recommended except in a clinical trial **(AII).**

Statin Therapy for Primary Prevention of ASCVD[a]

- For all people with HIV aged 40–75 years, the Panel concurs with the strong recommendation from the ACC/AHA/Multisociety Dyslipidemia Management Guideline (2026 Dyslipidemia Guideline) to initiate statin therapy regardless of LDL-C or estimated ASCVD risk.
- For people with HIV aged <40 or >75 years, an HIV-specific recommendation for statin therapy has not been issued by the 2026 Dyslipidemia Guideline nor the Panel. For these patients, decisions to initiate statin therapy should be informed by the 2026 Dyslipidemia Guideline with the following additional consideration:
  - HIV is an ASCVD risk enhancer. The presence of HIV can help support a decision to initiate statin therapy in addition to traditional ASCVD risk enhancers (as per the 2026 Dyslipidemia Guideline)[b].

- For people with HIV of any age who are hesitant to start statin therapy, the presence of HIV-related ASCVD risk enhancers[c] and/or traditional ASCVD risk enhancers (as per the 2026 Dyslipidemia Guideline)[b] that increase the risk of ASCVD events may further strengthen the rationale for initiating statin therapy.
- When statin therapy is initiated, the Panel recommends initiating at least moderate-intensity statin therapy **(AI).** Recommended options for moderate-intensity statin therapy based on evidence generated among people with HIV include the following:
  - Pitavastatin 4 mg once daily **(AI)**
  - Atorvastatin 20 mg once daily **(AII)**
  - Rosuvastatin 10 mg once daily **(AII)**
- Intensification of lipid-lowering therapy should be guided by LDL-C treatment goals as outlined in the 2026 Dyslipidemia Guideline.

Interactions Between Antiretroviral Drugs and Cardiovascular Drugs

- Some drugs prescribed to prevent or treat CVD (including certain statins) may have clinically significant interactions with certain ARV drugs. Before initiating or changing ARV drugs or concomitant medications, clinicians should carefully assess for potential interactions **(AIII)**. In some cases, the interaction may require statin dose adjustment, a switch to another statin, or increased monitoring for statin-related adverse effects (see the Liverpool Drug Interaction Checker).

Rating of Recommendations: *A = Strong; B = Moderate; C = Weak*

Rating of Evidence: *I = Data from randomized controlled trials; II = Data from well-designed nonrandomized trials or observational cohort studies with long-term clinical outcomes; III = Expert opinion*

[a] As in the general population, initiation of statin therapy should be deferred during pregnancy; statin therapy should be discontinued if pregnancy occurs, and breastfeeding is not recommended while using statin therapy.

[b] Traditional ASCVD risk enhancers, as reported in the ACC/AHA/Multisociety Dyslipidemia Management Guideline (Table 13), include: history of premature ASCVD in a parent or sibling (aged <55 years for men and <65 years for women), higher-risk ancestry (e.g., South Asian), high polygenic risk (if measured), chronic inflammatory diseases (e.g., systemic lupus), Lp(a) ≥125 nmol/L (or ≥50 mg/dL), hsCRP ≥2 mg/L on more than one occasion, CKM syndrome, TG persistently ≥150mg/dL (fasting), LDL-C persistently ≥160–189 mg/dL, non-HDL-C ≥190–219 mg/dL, or ApoB ≥120mg/dL.

[c] HIV-related ASCVD risk enhancers include factors that may increase ASCVD risk but are not considered in traditional risk estimation tools and may strengthen the rationale for initiating statin therapy in this population. Examples include prolonged duration of HIV infection, acquisition of HIV perinatally or during adolescence, history of prolonged HIV viremia, delay in ART initiation, and/or ART non-adherence, low current or nadir CD4 count (<350 cells/mm^3), exposure to older ARVs with cardiometabolic toxicity (e.g., abacavir), lipodystrophy/lipoatrophy, MASLD, and/or coinfection with hepatitis C.

Key: ACC = American College of Cardiology; AHA = American Heart Association; ApoB = apolipoprotein B; ART = antiretroviral therapy; ARV = antiretroviral; ASCVD = atherosclerotic cardiovascular disease; CD4 = CD4 T lymphocyte; CKM = cardiovascular-kidney-metabolic; CVD = cardiovascular disease; HDL-C = high-density lipoprotein cholesterol; hsCRP = high-sensitivity C-reactive protein; LDL-C = low-density lipoprotein cholesterol; Lp(a) = lipoprotein(a); MASLD = metabolic dysfunction-associated steatotic liver disease; PREVENT = Predicting Risk of cardiovascular disease EVENTs; TG = triglycerides; the Panel = U.S. Department of Health and Human Services Panel on Antiretroviral Guidelines for Adults and Adolescents

Overview

Most people with HIV who are using continuous antiretroviral therapy (ART) and achieve viral suppression have a life expectancy that is approaching that of people without HIV.[1] However, a gap in mortality remains due to comorbidities, such as atherosclerotic cardiovascular disease (ASCVD). As mortality from AIDS events among people with HIV has declined, the proportion of ASCVD-related deaths has increased. When compared to people without HIV, people with HIV have an

approximately twofold higher risk of developing ASCVD, even after accounting for traditional cardiometabolic risk factors,[2] with the age at incident ASCVD diagnosis among people with HIV younger than that of people without HIV. Factors influencing ASCVD risk among people with HIV include a higher prevalence of traditional cardiometabolic risk factors compared with the general population,[3] ongoing systemic inflammation associated with HIV despite viral suppression,[4] and cardiometabolic toxicity associated with certain antiretroviral (ARV) drugs.[5,6] In addition, differential access to screening and treatment of ASCVD risk factors also contributes to excess ASCVD risk among certain people with HIV in the United States.[7,8]

Data also suggest that ASCVD risk relative to the general population is greater among women with HIV than among age-matched men with HIV.[9,10] Although data indicating the absolute risk of myocardial infarction (MI) is similar—12.71 versus 10.48 per 1,000 person-years for women and men with HIV, respectively—women with HIV have exhibited a nearly 3- to 4-fold increase in relative MI risk versus a 1.5- to 2-fold increase for men compared to sex-specific rates among people without HIV.[10-13] A substudy from The Randomized Trial to Prevent Vascular Events in HIV (REPRIEVE) demonstrated the absence of an expected protective effect of female sex for the development of a major adverse cardiac event (MACE).[3] For more information on REPRIEVE, see Rationale for Statin Therapy and the REPRIEVE Trial Results.

While there has been extensive focus on the epidemiology and pathogenesis of ASCVD among people with HIV, there are also data demonstrating increased risk of other cardiac and vascular diseases. People with HIV have an increased risk of developing stroke (including ischemic and hemorrhagic), heart failure, arrhythmia or sudden cardiac death, and thrombosis.[14-17] Risk factors for these outcomes are thought to be similar to those for ASCVD, with an etiologic role for both traditional risk factors and HIV-related factors. Currently, this section of the guidelines is focused on primary prevention strategies for people with HIV, with an emphasis on ASCVD. As data emerge concerning secondary prevention strategies for ASCVD and primary and secondary prevention strategies for other cardiovascular outcomes specific to people with HIV, this section may be expanded.

HIV-Related Risk Factors for Atherosclerotic Cardiovascular Disease

Factors related to HIV and its treatment have been shown to be important contributors to excess ASCVD risk. Specifically, HIV-related inflammation has been associated with elevated ASCVD risk. This inflammation is increased in the presence of active viremia and decreases with ART but still remains elevated when compared to those without HIV (see the Cardiovascular and Metabolic Complications Introduction). Data from the SMART trial demonstrated that continuous ART not only improves HIV-related outcomes but also significantly reduces non-AIDS events, including cardiovascular disease (CVD), an observation thought to be related to the beneficial effects of ART on inflammation.[18] In the large Veterans Aging Cohort Study (VACS), MI risk was independently associated with detectable or increased HIV RNA; compared to people without HIV, MI risk was increased by 39% in those with HIV RNA <500 copies/mL and by 75% in those with HIV RNA ≥500 copies/mL.[19] In REPRIEVE, even low-level HIV viremia (median 62 copies/mL) was linked to increased rates of MACE, as viremic individuals had a higher risk than those with viral load below the limit of detection (adjusted hazard ratio 1.4; 95% confidence interval, 1.00–1.97)[3,20]; however, another recent study did not show an association between HIV viremia and CVD.[21] Based on these findings, continuous ART is recommended for all people with HIV not only to maintain viral suppression but also to reduce ASCVD risk **(AII).** Immunomodulatory therapy as an intervention to reduce HIV-associated ASCVD risk **is not recommended** except in a clinical trial **(AII).** See Immune Activation and Inflammation Among People With HIV Receiving Antiretroviral Therapy for additional details.

Additional factors related to HIV or common among people with HIV may further impact ASCVD risk. Lower CD4 T lymphocyte (CD4) cell count and nadir CD4 count have been shown to be associated with ASCVD events independent of traditional ASCVD risk factors or specific ART,[22,23] with the VACS cohort demonstrating CD4 <200 cells/mm^3 as an independent predictor of MI with an increased risk of nearly 90%.[19,24] HIV-related ASCVD risk enhancers,[25] included in Table 11a below, may strengthen the rationale for ASCVD prevention strategies, including statin initiation in individuals <40 or >75 years (see Primary Prevention of ASCVD), and may be used to inform decisions to avoid certain ARV medications that have been associated with increased ASCVD risk.

Antiretroviral Therapy and Cardiometabolic Complications

Specific ARV medications and classes have been associated with potential cardiometabolic toxicity, which has informed the U.S. Department of Health and Human Services Panel on Antiretroviral Guidelines for Adults and Adolescents (the Panel)'s recommendations on ART regimen selection to reduce risk for cardiometabolic complications. See Interactions Between ARV Drugs and Drugs for Treatment or Prevention of Cardiovascular Diseases below and Antiretroviral Regimen Considerations for Initial Therapy Based on Specific Clinical Scenarios for more information. Specifically, for individuals with elevated traditional ASCVD risk or known ASCVD, the selection of an ART regimen should consider options that minimize the introduction of new cardiometabolic adverse effects.

Antiretroviral Therapy and Atherosclerotic Cardiovascular Disease Risk

Individual ARV medications and classes have been linked to increased risk of ASCVD events. An association between abacavir (ABC) use and MI was first reported in the Data Collection on Adverse Events of Anti-HIV Drugs (D:A:D) study, with the increased risk noted only with recent/current ABC exposure (i.e., within 6 months) and particularly among participants with pre-existing cardiac risk factors.[26] Since the D:A:D report, multiple studies, systematic reviews, and meta-analyses have evaluated the relationship between ABC therapy and cardiovascular events, with some confirming the initially observed association and others failing to identify an association.[26-31] Retrospective analyses from the REPRIEVE study corroborated the D:A:D results, showing an increased hazard of a first MACE with ART regimens, including ABC versus tenofovir-containing regimens,[32] and an increased MACE hazard of 62% in participants with previous use and 41% with current use of ABC compared with participants who were never exposed.[33] An association between ABC and ASCVD risk reported in multiple large studies provides a rationale to consider underlying ASCVD risk when using ABC as a component of ART. Based on these data, the Panel recommends considering alternative ARV medications in place of ABC in people with HIV and higher underlying ASCVD risk or known ASCVD **(AII).**

An early study from the D:A:D cohort demonstrated a 16% increased risk of MI per year of cumulative protease inhibitor (PI) exposure,[34] specifically with older PIs (e.g., indinavir and ritonavir-boosted lopinavir [LPV/r]). A case-control study within the French HIV ANRS cohort CO4 FHDH also found a 33% increase in MI risk with LPV/r use.[35] A later study from the D:A:D cohort showed a 59% increased risk of ASCVD per 5 additional years of use with ritonavir-boosted darunavir (DRV/r) but not ritonavir-boosted atazanavir (ATV/r),[36] yet subsequent studies did not observe an association between boosted darunavir and increased ASCVD risk.[37-39] Based on these data, the Panel recommends considering alternative ARV medications in place of LPV/r in people with HIV and higher underlying ASCVD risk or known ASCVD **(AII).** If use of DRV/r is indicated, clinicians should weigh the potential benefits of therapy versus the potential cardiovascular risk.

The effect of integrase strand transfer inhibitors (INSTIs) on the risk of ASCVD is an area of active research, with recent studies showing inconsistent results. In a multicenter observational study of the

international RESPOND consortium, INSTI exposure was associated with a nearly twofold greater incidence of a composite CVD endpoint within the first 6 months after exposure compared to no INSTI exposure,[40] and the increased risk persisted throughout the first 24 months. Subsequently, two target trial emulation studies found no association between INSTI use and a composite CVD outcome that included MI, stroke, or invasive cardiovascular procedures, regardless of previous ART experience.[41,42] Based on current data, there is insufficient evidence to recommend modifying suppressive INSTI-based ART regimens in people with weight gain, higher underlying ASCVD risk, or known ASCVD.

Antiretroviral Therapy and Dyslipidemia

Higher levels of low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), and triglycerides have been observed with tenofovir alafenamide (TAF), efavirenz (EFV), all boosted PIs, and elvitegravir/cobicistat (EVG/c).[43,44] Among the PIs, similar lipid changes have been shown in participants from a randomized trial of ATV/r- and DRV/r-based regimens.[45] Importantly, differences in lipid levels between two ART regimens can be due to a lipid-raising effect of a given medication and/or a lipid-lowering effect of the other medication. For example, levels of total cholesterol, LDL, HDL, and triglycerides are higher in participants who received TAF when compared with those who received tenofovir disoproxil fumarate (TDF) in multiple randomized controlled trials.[46,47] However, data from a placebo-controlled pre-exposure prophylaxis trial support that TDF exposure has an independent lipid-lowering effect (for total cholesterol, LDL, and HDL).[48] Further, data support that total cholesterol–to-HDL ratios did not differ between participants receiving TAF and those receiving TDF,[49,50] and the clinical significance of ASCVD risk from increases across all lipid indices without changes in total to HDL ratios is uncertain.

Antiretroviral Therapy and Diabetes/Insulin Resistance

Direct effects on diabetes mellitus (DM) and insulin resistance have been observed with older ARVs, including ZDV and LPV/r.[51] Several studies have demonstrated an increased risk of DM with INSTI use,[52-54] an association that may be mediated in part by weight gain.[55] An association between ART and DM has not been consistently observed with other contemporary ARVs, including those recommended as initial therapy in the guidelines.

Antiretroviral Therapy and Other Cardiac Complications

EFV, as well as higher-than-recommended doses of fostemsavir and rilpivirine, have been associated with QTc (QT corrected for heart rate) prolongation.[56-58] These ARV drugs should be used with caution in people with pre-existing heart disease, QTc prolongation, or concomitant use of medications that may increase drug concentration or prolong the QTc interval. Both ATV/r and LPV/r have been associated with PR prolongation, particularly among people with risk factors, including pre-existing heart disease and concomitant use of medications that may cause PR prolongation.[59,60]

Strategies for Atherosclerotic Cardiovascular Disease Risk Estimation and Screening for People With HIV

Cardiovascular risk assessment strategies developed for the general population do not reflect HIV-specific factors, such as inflammation and immune activation or potential contributions from certain ARV drugs, which are key mechanistic contributors to HIV-associated ASCVD. This section will address lipid screening and ASCVD risk estimation for people with HIV. ASCVD prevention strategies specific to people with HIV are further discussed in the Cardiovascular and Metabolic Complications in People With HIV Introduction section and the Primary Prevention of ASCVD subsection below.

Lipid Screening and Monitoring

Lipid screening should be performed in people with HIV **(AIII),** given the increased risk for ASCVD and the increased rate of dyslipidemia associated with certain ARV drugs. A fasting or random lipid profile should be obtained for all people with HIV at entry into care or at the time of ART initiation. If a random profile is abnormal, a fasting lipid profile should be obtained. Lipid levels should be rechecked 3 to 6 months after starting or modifying ART once viral suppression is achieved. Lipid levels measured after initiation of ART should then be used to calculate an ASCVD risk score and determine indications for statin therapy (see Primary Prevention of ASCVD). The 2026 American College of Cardiology (ACC)/American Heart Association (AHA)/Multisociety Dyslipidemia Management Guideline (2026 Dyslipidemia Guideline) additionally recommends checking lipoprotein(a) (Lp(a)) at least once for ASCVD risk assessment and considering testing for Apolipoprotein B (ApoB) in individuals on lipid-lowering therapy to guide decisions regarding further therapeutic intensification once LDL-C and/or non-HDL-C goals are achieved and in individuals not on lipid-lowering therapy to guide decisions about initiation of lipid-lowering therapy.

Among people with HIV who are receiving statin therapy, lipid monitoring should be performed annually to reassess ASCVD risk and inform decisions on statin management and/or other risk modification strategies. Further, specific to people on statin therapy, the approach to monitor lipids at least annually to assess adherence and the need for treatment intensification is consistent with recommendations by the 2026 Dyslipidemia Guideline.[61] For people with HIV who are not receiving statin therapy, lipid levels should be monitored every 1 to 3 years to reassess ASCVD risk. For all people with HIV, more frequent lipid monitoring may be necessary based on lifestyle factors or clinical scenarios, including changes in traditional ASCVD risk factors (e.g., weight gain, new diagnosis of hypertension, concomitant medications that affect lipids) or a change to an ARV regimen with increased ASCVD risk.

Atherosclerotic Cardiovascular Disease Risk Estimation

ASCVD risk prediction algorithms enable clinicians to quantify the risk of a future cardiovascular event and are used in clinical practice to stratify risk and to guide the intensity of ASCVD preventive measures and risk factor management, including statin use and antihypertensive therapy. Yet established ASCVD risk prediction scores validated in the general population typically do not include risk factors specific to HIV, and they have been shown to systematically underestimate risk among people with HIV. Data from multiple studies and a meta-analysis have shown that the ACC/AHA Pooled Cohort Equations (PCEs)—the widely used U.S.-based CVD risk score since 2013—underpredict ASCVD risk in people with HIV in high-income countries.[62-67] Studies also suggest the accuracy of established ASCVD risk scores for people with HIV may vary by patient demographics and clinical characteristics, with underprediction of ASCVD risk demonstrated among women,[62,65,66] Black or African-American individuals,[62,66] and individuals with low or moderate predicted CVD risk versus high predicted risk.[62,64,65] ASCVD risk score accuracy may also differ by region globally, with a study of REPRIEVE data showing underprediction of risk in high-income countries yet overprediction in low- and middle-income countries.[66]

In 2023, the AHA developed a new set of ASCVD risk equations for the general population. The Predicting Risk of CVD EVENTs (PREVENT) equations differ from the PCEs on several levels, including the inclusion of a new derivation dataset of more than 6 million individuals; new variables reflecting a broader cardiovascular-kidney-metabolic framework; new outcomes, including heart failure; and the removal of race from the models.[68] However, like the PCEs, the PREVENT equations were developed for the general population and consequently do not incorporate HIV-specific risk factors. Predicted ASCVD risk has been shown to be lower using PREVENT-ASCVD in the general population[69] and in people with HIV,[70] with 97% of people with HIV having lower predicted risk and 58% fewer strongly recommended for statins using PREVENT-ASCVD (vs. PCEs). Similar to prior risk prediction scores, the PREVENT-ASCVD equations have been shown to

underestimate risk among people with HIV. In a U.S. cohort of more than 13,000 people with HIV, the PREVENT-ASCVD equations underestimated risk to a greater extent than the PCEs, with the degree of underprediction approximately twice as large with PREVENT.[71] In an analysis of the placebo arm of the REPRIEVE trial, the PREVENT-ASCVD equations similarly underestimated risk of MACE, including CVD-related death, MI, or stroke.[72] Importantly, the underestimation of ASCVD event risk by PREVENT, when compared to PCEs, is also present in the general population, and the 2026 Dyslipidemia Guideline provides risk categories that account for these differences in absolute risk between PREVENT and PCE scores.

The 2026 Dyslipidemia Guideline recommends using the PREVENT-ASCVD equations rather than the PCEs to estimate risk and guide lipid-lowering therapy for primary prevention in adults aged 30 to 79 years. For people with HIV aged 30 to 79 years, 10-year ASCVD risk should be estimated using the PREVENT-ASCVD equations whenever lipids are measured or in certain clinical scenarios that may affect ASCVD risk (e.g., changes in traditional CVD risk factors, such as weight gain, new diagnosis of hypertension, concomitant medications that affect lipids, or change to an ARV regimen with increased ASCVD risk) **(BIII).** Of note, although statin therapy is recommended for all people with HIV aged 40 to 75 years regardless of ASCVD risk estimate (see Primary Prevention of ASCVD below), routine estimation of ASCVD risk should be done when lipids are monitored among people with HIV (even outside of this age range) for statin intensification and/or additional lipid-lowering therapy, and for management of CVD risk factors other than dyslipidemia. Presently, there is insufficient evidence to recommend strategies to account for the underprediction of ASCVD risk by PREVENT-ASCVD and other established ASCVD risk scores among people with HIV, including the use of inflammatory biomarkers.

Primary Prevention of ASCVD

Traditional Cardiovascular Disease Risk Factor Modification

Although HIV-specific factors influence CVD risk, traditional risk factors also play a critical role in the development and prevention of CVD among people with HIV. Many traditional risk factors both predict risk for CVD events and occur at higher rates among people with HIV when compared with the general population.[3,25,73] A 2025 global meta-analysis assessed common CVD risk factors among people with HIV and found higher odds of CVD incidence associated with DM, hypertension, dyslipidemia, smoking, and a prior history of CVD.[74] A U.S. population-based study among people with HIV found conventional risk factors, such as tobacco use, hypertension, and obesity, to be persistently associated with CVD incidence over follow-up periods of as long as 15 years.[75] Nontraditional CVD risk factors that are also more common in people with HIV, but are not specific to HIV disease itself, include alcohol use disorders, depression, and hepatitis C virus coinfection.

These data highlight the importance of traditional risk factor management among people with HIV. While CVD risk factors such as advancing age and other demographic characteristics are non-modifiable, many risk factors may be further optimized with behavioral and/or pharmaceutical interventions. A longitudinal study leveraging health system data found that associations between CVD risk and HIV status were attenuated when dyslipidemia and DM were well controlled.[76] For women with HIV, ensuring optimal CVD risk factor management is critical given the greater relative risk of ASCVD and underperformance of ASCVD risk score algorithms.

Optimal management of comorbidities (e.g., hypertension, diabetes, obesity, smoking) and lifestyle factors remains important to further reduce CVD risk among people with HIV. The AHA has further outlined “Life’s Essential Eight” as modifiable risk factors that can be targeted to improve cardiovascular health for everyone (regardless of HIV infection), and these include: healthy diet; participation in physical activity; smoking cessation; healthy sleep; healthy weight; and healthy levels of blood lipids, blood glucose, and blood pressure.[77] Management strategies and treatment goals for

optimizing traditional risk factor profiles are outlined in guidelines established for the general population.[61,78,79]

Rationale for Statin Therapy and the REPRIEVE Trial Results

The increased ASCVD risk associated with HIV disease and/or ARV exposure, despite viral suppression and optimizing traditional risk factor management, has motivated research on lower thresholds for use of primary CVD prevention strategies. Statins may offer advantages for people with HIV over other prevention pharmacotherapy, given that statin therapy is associated with a well-described reduction in ASCVD events as well as treatment effects on inflammatory pathways.[80-82]

REPRIEVE provided evidence that the use of statin therapy as primary prevention for people with HIV who otherwise did not have an indication for statin reduced risk of ASCVD events. REPRIEVE was a Phase 3 global randomized placebo-controlled trial of oral daily pitavastatin 4 mg that enrolled 7,769 people with HIV who were aged 40 to 75 years and at low-to-intermediate risk based on 10-year ASCVD risk estimates (using PCEs) and LDL cholesterol levels.[20] People with known ASCVD were excluded. The primary outcome was the occurrence of MACE, defined as a composite of cardiovascular disease death; MI; hospitalization for unstable angina; stroke; transient ischemic attack; peripheral arterial ischemia; revascularization of coronary, carotid, or peripheral artery; or death of undetermined cause. Study participants were 31% female, had a median age of 50 years, had a median CD4 T cell count of 621 cells/mm^3, and 88% had an undetectable HIV RNA level. Compared to placebo, pitavastatin was associated with a 36% reduction in MACE (hazard ratio 0.64; 95% confidence interval [CI], 0.48–0.84), with event rates of 7.77 and 4.95 per 1,000 person-years, respectively, over a median follow-up of 5.6 years. In REPRIEVE, the absolute risk reduction was substantially greater for people with ASCVD risk estimates of ≥5% compared to those with risk of <5% (estimated using PCEs). Finally, incident diabetes (incident rate ratio [IRR] 1.29; 95% CI, 1.07–1.57) and Grade ≥3 or treatment-limiting muscle-related symptoms (IRR 1.58; 95% CI, 1.14–2.19) were also higher in the pitavastatin group.

Recommendations for Use of Statin Therapy Among People With HIV

The 2026 Dyslipidemia Guideline for the general population issues recommendations for people with HIV based on data from REPRIEVE.[61]

For People With HIV Aged 40–75 Years

Among all people with HIV aged 40–75 on stable ART, the Panel concurs with the strong recommendation from the 2026 Dyslipidemia Guideline (section 4.2.8.9) to initiate statin therapy regardless of LDL-C or estimated ASCVD risk to reduce the risk of a first ASCVD event and reduce the rate of coronary atherosclerosis progression. For these patients, additional testing is not specifically recommended for risk stratification for statin initiation. The full 2026 Dyslipidemia Guideline should be referenced for additional details, including LDL-C goals and thresholds for statin intensification.

For People With HIV Aged <40 or >75 Years

The REPRIEVE trial did not enroll people with HIV aged <40 years or >75 years. In this context, the 2026 Dyslipidemia Guideline does not issue a recommendation for statins specific to people with HIV aged <40 or >75 years and instead references additional sections that provide guidance on primary prevention for adults aged 19–39 years and adults with LDL-C <190 mg/dL (aged 30–79 years) regardless of HIV status. The Panel has also determined there is insufficient evidence to issue a recommendation for or against statin therapy as primary prevention for people with HIV aged <40 or >75 years. For these patients, decisions on use of statin therapy should be informed by

benefit–risk discussions with patients and apply decision algorithms based on 10-year risk estimates (using PREVENT-ASCVD), as outlined in the 2026 Dyslipidemia Guideline. For these decisions, HIV is an ASCVD risk enhancer that can help support the use of statin therapy if a statin is not already recommended (e.g., for those with a borderline 10-year risk as defined by PREVENT-ASCVD 3–5%), in addition to the ASCVD risk enhancers in the 2026 Dyslipidemia Guideline (see Table 11b below).

The 2026 Dyslipidemia Guideline recommends using the presence of coronary artery calcium (CAC) to improve risk assessment among those with "borderline" or "intermediate" risk (as estimated by PREVENT-ASCVD). There is no HIV-specific recommendation for using CAC scoring for risk stratification, beyond guidance provided for the general population within the 2026 Dyslipidemia Guideline. In people without HIV, the assessment of CAC is generally recommended for use in men aged ≥40 years and women aged ≥45 years; people with HIV in these age groups are already recommended to initiate statin therapy, making assessment of CAC to guide decision-making less relevant. Additionally, given the predominance of noncalcified plaque in people with HIV, it should be noted that the absence of CAC may not reflect lower risk in this context.

Additional Considerations for Decisions on Use of Statin Therapy

Statin therapy should be avoided for women with HIV who are pregnant or are planning to become pregnant. Specifically, initiation of statin therapy should be deferred until after pregnancy for those with HIV where statin therapy is recommended and should be discontinued if pregnancy occurs for those on statin therapy. As in the general population, breastfeeding is not recommended while taking statin therapy.

Some patients may be hesitant to start statin therapy despite recommendations and/or clinical scenarios in which statin therapy is favored. In these cases, clinicians should work to identify factors underlying a patient's concern and provide education or support to alleviate these concerns when feasible. In addition, the presence or absence of additional ASCVD risk enhancers may help inform and/or strengthen the rationale for statin therapy when discussing with patients through shared decision-making. Table 11a lists HIV-related factors that further increase ASCVD risk,[25] such as prolonged HIV viremia or low nadir CD4 count.

Table 11a. HIV-Related ASCVD Risk Enhancers

HIV-Related ASCVD Risk Enhancers
• Prolonged duration of HIV infection
• Acquisition of HIV perinatally or during adolescence
• History of prolonged HIV viremia
• Delay in ART initiation
• ART non-adherence
• Low current or nadir CD4 count (<350 cells/mm^3)
• Exposure to older ARVs with cardiometabolic toxicity, including ABC
• Lipodystrophy/lipoatrophy
• MASLD
• HCV coinfection

Key: ABC = abacavir; ART = antiretroviral therapy; CD4 = CD4 T lymphocyte; HCV = hepatitis C virus; MASLD = metabolic dysfunction-associated steatotic liver disease

Table 11b lists additional risk enhancers identified within the 2026 Dyslipidemia Guideline. For example, factors that are not specific to HIV, including clinical measurement of Lp(a), ApoB, and high-sensitivity C-reactive protein (hsCRP), may be helpful to further inform decisions on statin therapy in this context. Risk enhancers can help support initiation of statin therapy when decisions are based on risk assessments (e.g., for people <40 years old) and/or for people who are hesitant to start statin therapy.

Table 11b. ASCVD Risk Enhancers

ASCVD Risk Enhancers
• HIV infection
Additional ASCVD risk enhancers as reported in the 2026 Dyslipidemia Guideline[a]
• History of premature ASCVD in a parent or sibling (onset aged <55 years for men, <65 years for women)
• Higher-risk ancestry (e.g., South Asian, Filipino)
• High polygenic risk (if measured)
• Chronic inflammatory diseases (e.g., systemic lupus, rheumatoid arthritis, advanced psoriasis, inflammatory arthritis)
• Lp(a) ≥125 nmol/L or ≥50 mg/dL
• hsCRP ≥2 mg/L on more than one occasion (if measured)
• TG persistently ≥175 mg/dL (if nonfasting) and ≥150 mg/dL (if fasting)
• Cardiovascular-kidney-metabolic (CKM) syndrome
• LDL-C persistently ≥160–189 mg/dL, non–HDL-C ≥190–219 mg/dL, or ApoB ≥120 mg/dL
• Reproductive risk markers (premature menopause, preeclampsia, gestational diabetes, gestational hypertension, preterm delivery)

[a] Refer to the 2026 Dyslipidemia Guideline, Table 13, page e1188.

Key: ApoB = apolipoprotein B; ASCVD = atherosclerotic cardiovascular disease; CKM = cardiovascular-kidney-metabolic; HDL-C = high-density lipoprotein cholesterol; hsCRP = high-sensitivity C-reactive protein; LDL-C = low-density lipoprotein cholesterol; Lp(a) = lipoprotein(a); TG = triglycerides

Rationale for the Panel's Recommendation on Choice and Dose of Statin

Although there are no clinical outcome comparative effectiveness trials of different statins among people with HIV, additional studies beyond REPRIEVE support the treatment effect of certain other statins (other than pitavastatin) for lipid lowering, reductions in inflammation, and improvements in surrogate imaging measures. Pitavastatin, atorvastatin, and rosuvastatin are all associated with greater reductions in LDL-C among people with HIV than pravastatin. In addition, pitavastatin (4 mg daily), rosuvastatin (10 mg daily), and high-dose atorvastatin (80 mg daily) have each demonstrated reductions in inflammation among people with HIV.[83-85] Finally, in two separate Phase 2 placebo-controlled randomized trials among people with HIV, atorvastatin (20 mg daily) was associated with reductions in coronary noncalcified plaque, and rosuvastatin (10 mg daily) was associated with slower progression of carotid artery intima-media thickness.[86,87] Cumulatively, these data motivated the Panel's recommendation that moderate-intensity statin therapy include either pitavastatin 4 mg daily, atorvastatin 20 mg daily, or rosuvastatin 10 mg daily, with the level of evidence differing between statins (see Table 11c).

The 2026 Dyslipidemia Guideline classifies statin intensity as high, moderate, and low intensity.[61] Table 11c below outlines the statin agent and dose that qualify for each intensity, as well as the anticipated LDL-C lowering effect. Specifically, for every 39 mg/dL reduction in LDL-C, there is approximately a >20% reduction in ASCVD events and a 10% reduction in all-cause mortality.[88] However, it should also be noted that the magnitude of LDL-C lowering varies in clinical practice.

Table 11c. Intensity of Statin Therapy

Moderate Intensity (30% to 49% LDL-C Lowering Effect)	High Intensity (≥50% LDL-C Lowering Effect)
Pitavastatin 4 mg (AI)[a]	Atorvastatin[b] 40–80 mg
Atorvastatin 20 mg (AII)[a,b]	Rosuvastatin[b] 20–40 mg
Rosuvastatin 10 mg (AII)[a,b]	
Fluvastatin XL 80 mg	
Fluvastatin 40 mg twice daily	
Lovastatin[c] 40–80 mg	
Pravastatin 40–80 mg	
Simvastatin[c] 20–40 mg	

[a] Bolded statins are included in recommendations from the Panel for the Use of Antiretroviral Agents in Adults and Adolescents With HIV; see Rationale for the Panel's Recommendation on Choice and Dose of Statin above.

[b] Atorvastatin and rosuvastatin have known drug–drug interactions with ritonavir- and cobicistat-boosted antiretrovirals (ARVs); see the Liverpool HIV Drug Interaction Checker.

[c] Simvastatin and lovastatin are contraindicated with ritonavir- and cobicistat-boosted ARVs.

Key: LDL-C = low-density lipoprotein cholesterol; XL = extended-release

Common Adverse Effects of Statins

Statin therapy is well tolerated and safe; however, some side effects have been observed. The most frequent adverse events are statin-associated muscle symptoms, with reported occurrence in 5% to 25% of people who receive statins.[89]

All statins have been implicated in small increases in both relative and absolute risk of diabetes. The increased risk of diabetes with statin use has been associated with older age and the presence of at least two risk factors, such as elevated baseline fasting glucose level, elevated fasting triglycerides, elevated body mass index, or history of hypertension.[90,91] Mild increases in liver enzymes are seen in some individuals, although these are usually transient without clinical complications.

Interactions Between Antiretroviral Drugs and Drugs for Treatment or Prevention of Cardiovascular Diseases

People with HIV may receive medications for the treatment or prevention of CVD. Some of these drugs (especially antiarrhythmics, anticoagulants, calcium channel blockers, beta blockers, and statins) may have clinically significant interactions with some ARV drugs. Before initiating or changing these medications, clinicians should carefully assess for potential interactions **(AIII).** The Liverpool HIV Drug Interaction Checker provides guidance for managing these interactions. Major

mechanisms of pharmacokinetic interactions between ARV drugs and cardiac drugs are noted below.

For the prevention of ASCVD, among the statins, pitavastatin, pravastatin, and rosuvastatin are not significantly metabolized by cytochrome P450 (CYP) enzymes. Atorvastatin, lovastatin, and simvastatin are primarily metabolized by CYP3A4, whereas fluvastatin is a major substrate of CYP2C9. Lovastatin and simvastatin are **contraindicated** when coadministered with cobicistat- or ritonavir-boosted ARVs. All statins are susceptible to interactions related to drug transporters (e.g., OATP1B1).[92]

Other Management Considerations and Unanswered Questions

Estrogen, Progestins, or Testosterone Therapy and Cardiovascular Risk

Studies have produced conflicting results on associations between estrogen, progestins, or testosterone therapy and CVD risk, varying by study design, study population, duration and timing of therapy, and other clinical factors.[93-96] Research on relationships between these drugs and CVD among people with HIV is even more limited.[97-100] Data on testosterone use among people with HIV have been limited to the treatment of testosterone deficiency and lack longitudinal information on CVD-related outcomes.[101,102] Studies of estrogen use suggest a potential increase in CVD risk; however, this association may vary by dose, mode of delivery, and underlying physiological stress.[95-97,103] A longitudinal study of combined estrogen and progestin use for oral contraception among people with HIV suggested increased CVD risk, although the association was not statistically significant.[104]

As the population with HIV ages, a growing number of women with HIV are entering menopause. CVD is the leading cause of morbidity and mortality in menopausal women, and this risk is increased in women with HIV.[105,106] Studies of the cardiovascular risk associated with menopausal hormone therapy have led to changes in guidelines over the years.[106] Systemic hormone therapy is not currently recommended for CVD prevention or treatment.[107] However, hormone therapy remains the most effective treatment for vasomotor symptoms.[108] Some research suggests that women with HIV are undertreated for menopausal symptoms.[109] However, data are lacking on menopause and hormone therapy in women with HIV, including whether menopause amplifies the effects of HIV on CVD risk.[110] Current HIV primary care guidelines recommend that menopause-related hormone therapy for women with HIV should not differ from the care of women without HIV.[111]

Cardiovascular Risks of Adults Who Acquired HIV Perinatally or During Adolescence

An increasing number of adults acquired HIV perinatally or during adolescence, but the cardiovascular effects of perinatal or adolescent HIV acquisition and long-term ART use are unclear. For individuals who acquired HIV perinatally, it is hypothesized that exposure to HIV and ART *in utero* while the cardiovascular system is developing, use of older ART drugs with cardiometabolic and mitochondrial toxicity, lifelong duration of infection, and a longer period of chronic inflammation may further exacerbate the already heightened CVD risk observed in people with HIV. Investigation of the cardiovascular complications of perinatal HIV infection has largely focused on youth and adolescents. Abnormalities in cardiac structure and function have been well documented and include systolic and diastolic dysfunction, left ventricular hypertrophy and dilatation, and myocardial fibrosis.[112-117] Studies of perinatally acquired HIV have also demonstrated elevated rates of surrogate markers of atherosclerosis (including carotid intima-media thickness, arterial stiffness, and endothelial dysfunction),[118,119] impaired glucose metabolism and insulin resistance,[120] lipodystrophy and metabolic syndrome,[121] and increased Pathobiological Determinants of Atherosclerosis in Youth

(or PDAY) cardiovascular risk scores.[122,123] Adults with perinatally acquired HIV also appear to be at risk for cardiometabolic comorbidities, with a large North American study of young adults aged 18–30 years demonstrating a high incidence of DM, hypercholesterolemia, hypertriglyceridemia, hypertension, and chronic kidney disease.[124] Current gaps in knowledge regarding CVD risk among those who acquired HIV perinatally or during adolescence include (1) the clinical significance of cardiac structural and functional abnormalities and surrogate markers of atherosclerosis with respect to coronary plaque and cardiac endpoints; (2) whether the epidemiology and pathophysiology of CVD differ for people with similar durations of perinatally versus horizontally acquired HIV, including HIV acquired during adolescence; (3) whether the epidemiology and pathophysiology of CVD differ for people in low- and middle-income versus high-income countries; (4) the optimal approach to estimate ASCVD risk and frequency of monitoring in adults who acquired HIV perinatally or during adolescence; and (5) whether primary prevention approaches (e.g., statins) for adults with HIV aged 40 and older apply to younger adults who acquired HIV perinatally or during adolescence.

Knowledge Gaps

- More research is needed on strategies to improve ASCVD risk stratification among people with HIV, including the utility of using inflammatory biomarkers, other non-traditional risk factors, Lp(a) and ApoB levels, and CAC estimates.
- More research is needed on recommended strategies for primary prevention of ASCVD among people with HIV aged <40 or >75 years.
- More research is needed on the role of immunomodulatory therapy to reduce HIV-associated ASCVD risk.
- More research is needed on the epidemiology, pathophysiology, risk estimation, prevention, and management strategies for ASCVD in key subgroups, including—
    - Women with HIV
    - People who acquired HIV perinatally or during adolescence (see Cardiovascular Risks of Adults Who Acquired HIV Perinatally or During Adolescence above for current knowledge gaps)
    - People with HIV across different geographic regions globally
- More research is needed on the role of HIV in non-ASCVD manifestations, including stroke, heart failure, arrhythmia, sudden cardiac death, and thrombosis.
- More research is needed on the association of CVD risk with INSTI-based ART, as well as newer regimens entering clinical practice.
- More research is needed on HIV-specific recommendations for pharmacotherapy targeting traditional risk factors (e.g., dyslipidemia, hypertension, diabetes, chronic kidney disease), such as lower thresholds for primary prevention, treatment goals and medication intensification, non-statin lipid-lowering therapy (including PCSK-9 inhibitors), and secondary prevention.
- More research is needed on implementation strategies for primary prevention of ASCVD among people with HIV.

References

1. Marcus JL, Leyden WA, Alexeeff SE, et al. Comparison of overall and comorbidity-free life expectancy between insured adults with and without HIV infection, 2000–2016. *JAMA Netw Open*. 2020;3(6):e207954. Available at: https://pubmed.ncbi.nlm.nih.gov/32539152.

2. Shah ASV, Stelzle D, Lee KK, et al. Global burden of atherosclerotic cardiovascular disease in people living with HIV: systematic review and meta-analysis. *Circulation*. 2018;138(11):1100-1112. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29967196.

3. Grinspoon SK, Watanabe M, Ribaudo HJ, et al. Factors affecting risk of cardiovascular disease (CVD) events in a global CVD prevention cohort of people with HIV. *Clin Infect Dis*. 2026;81(6):e548-e557. Available at: https://www.ncbi.nlm.nih.gov/pubmed/40281648.

4. Tenorio AR, Zheng Y, Bosch RJ, et al. Soluble markers of inflammation and coagulation but not T-cell activation predict non-AIDS-defining morbid events during suppressive antiretroviral treatment. *J Infect Dis*. 2014;210(8):1248-1259. Available at: https://www.ncbi.nlm.nih.gov/pubmed/24795473.

5. D:A:D Study Group, Sabin CA, Worm SW, et al. Use of nucleoside reverse transcriptase inhibitors and risk of myocardial infarction in HIV-infected patients enrolled in the D:A:D study: a multi-cohort collaboration. *Lancet*. 2008;371(9622):1417-1426. Available at: https://www.ncbi.nlm.nih.gov/pubmed/18387667.

6. Worm SW, Sabin C, Weber R, et al. Risk of myocardial infarction in patients with HIV infection exposed to specific individual antiretroviral drugs from the 3 major drug classes: the Data Collection on Adverse Events of Anti-HIV Drugs (D:A:D) study. *J Infect Dis*. 2010;201(3):318-330. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20039804.

7. Richardson KK, Bokhour B, McInnes DK, et al. Racial disparities in HIV care extend to common comorbidities: implications for implementation of interventions to reduce disparities in HIV care. *J Natl Med Assoc*. 2016;108(4):201-210.e3. Available at: https://www.ncbi.nlm.nih.gov/pubmed/27979005.

8. Muiruri C, Longenecker CT, Meissner EG, et al. Prevention of cardiovascular disease for historically marginalized racial and ethnic groups living with HIV: a narrative review of the literature. *Prog Cardiovasc Dis*. 2020;63(2):142-148. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32057785.

9. Triant VA, Lee H, Hadigan C, Grinspoon SK. Increased acute myocardial infarction rates and cardiovascular risk factors among patients with human immunodeficiency virus disease. *J Clin Endocrinol Metab*. 2007;92(7):2506-2512. Available at: https://www.ncbi.nlm.nih.gov/pubmed/17456578.

10. Lang S, Mary-Krause M, Cotte L, et al. Increased risk of myocardial infarction in HIV-infected patients in France, relative to the general population. *AIDS*. 2010;24(8):1228-1230. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20400883.

11. Balde A, Lang S, Wagner A, et al. Trends in the risk of myocardial infarction among HIV-1-infected individuals relative to the general population in France: impact of gender and immune status. *PLoS One*. 2019;14(1):e0210253. Available at: https://www.ncbi.nlm.nih.gov/pubmed/30650119.

12. Womack JA, Chang CC, So-Armah KA, et al. HIV infection and cardiovascular disease in women. *J Am Heart Assoc*. 2014;3(5):e001035. Available at: https://www.ncbi.nlm.nih.gov/pubmed/25324353.

13. Durand M, Sheehy O, Baril JG, Lelorier J, Tremblay CL. Association between HIV infection, antiretroviral therapy, and risk of acute myocardial infarction: a cohort and nested case-control study using Quebec's public health insurance database. *J Acquir Immune Defic Syndr*. 2011;57(3):245-253. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21499115.

14. Rokx C, Borjas Howard JF, Smit C, et al. Risk of recurrent venous thromboembolism in patients with HIV infection: a nationwide cohort study. *PLoS Med*. 2020;17(5):e1003101. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32407386.

15. Park DY, An S, Romero ME, et al. Incidence and risk factors of atrial fibrillation and atrial arrhythmias in people living with HIV: a systematic review and meta-analysis. *J Interv Card Electrophysiol*. 2022;65(1):183-191. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35610524.

16. Rasmussen LD, Engsig FN, Christensen H, et al. Risk of cerebrovascular events in persons with and without HIV: a Danish nationwide population-based cohort study. *AIDS*. 2011;25(13):1637-1646. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21646903.

17. Tseng ZH, Moffatt E, Kim A, et al. Sudden cardiac death and myocardial fibrosis, determined by autopsy, in persons with HIV. *N Engl J Med*. 2021;384(24):2306-2316. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34133860.

18. El-Sadr WM, Lundgren JD, Neaton JD, et al. CD4+ count-guided interruption of antiretroviral treatment. *N Engl J Med*. 2006;355(22):2283-2296. Available at: https://www.ncbi.nlm.nih.gov/pubmed/17135583.

19. Freiberg MS, Chang CC, Kuller LH, et al. HIV infection and the risk of acute myocardial infarction. *JAMA Intern Med*. 2013;173(8):614-622. Available at: https://www.ncbi.nlm.nih.gov/pubmed/23459863.

20. Grinspoon SK, Fitch KV, Zanni MV, et al. Pitavastatin to prevent cardiovascular disease in HIV infection. *N Engl J Med*. 2023;389(8):687-699. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37486775.

21. Elvstam O, Ryom L, Neesgaard B, et al. Viremia does not independently predict cardiovascular disease in people with HIV: a RESPOND cohort study. *Open Forum Infect Dis*. 2025;12(2):ofaf016. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39896984.

22. Mogadam E, King K, Shriner K, et al. The association of nadir CD4-t cell count and endothelial dysfunction in a healthy HIV cohort without major cardiovascular risk factors. *SAGE Open Med*. 2020;8:2050312120924892. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32537154.

23. Ho JE, Scherzer R, Hecht FM, et al. The association of CD4+ T-cell counts and cardiovascular risk in treated HIV disease. *AIDS*. 2012;26(9):1115-1120. Available at: https://www.ncbi.nlm.nih.gov/pubmed/22382147.

24. Lichtenstein KA, Armon C, Buchacz K, et al. Low CD4+ T cell count is a risk factor for cardiovascular disease events in the HIV Outpatient Study. *Clin Infect Dis*. 2010;51(4):435-447. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20597691.

25. Feinstein MJ, Hsue PY, Benjamin LA, et al. Characteristics, prevention, and management of cardiovascular disease in people living with HIV: a scientific statement from the American Heart Association. *Circulation*. 2019;140(2):e98-e124. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31154814.

26. Strategies for Management of Anti-Retroviral Therapy/INSIGHT, DAD Study Groups. Use of nucleoside reverse transcriptase inhibitors and risk of myocardial infarction in HIV-infected patients. *AIDS*. 2008;22(14):F17-F24. Available at: https://www.ncbi.nlm.nih.gov/pubmed/18753925.

27. Obel N, Farkas DK, Kronborg G, et al. Abacavir and risk of myocardial infarction in HIV-infected patients on highly active antiretroviral therapy: a population-based nationwide cohort study. *HIV Med*. 2010;11(2):130-136. Available at: https://www.ncbi.nlm.nih.gov/pubmed/19682101.

28. Choi AI, Vittinghoff E, Deeks SG, Weekley CC, Li Y, Shlipak MG. Cardiovascular risks associated with abacavir and tenofovir exposure in HIV-infected persons. *AIDS*. 2011;25(10):1289-1298. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21516027.

29. Young J, Xiao Y, Moodie EE, et al. Effect of cumulating exposure to abacavir on the risk of cardiovascular disease events in patients from the Swiss HIV cohort study. *J Acquir Immune Defic Syndr*. 2015;69(4):413-421. Available at: https://www.ncbi.nlm.nih.gov/pubmed/25932884.

30. Elion RA, Althoff KN, Zhang J, et al. Recent abacavir use increases risk of type 1 and type 2 myocardial infarctions among adults with HIV. *J Acquir Immune Defic Syndr*. 2018;78(1):62-72. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29419568.

31. Jaschinski N, Greenberg L, Neesgaard B, et al. Recent abacavir use and incident cardiovascular disease in contemporary-treated people with HIV. *AIDS*. 2023;37(3):467-475. Available at: https://www.ncbi.nlm.nih.gov/pubmed/36001525.

32. Davies Smith E, Malvestutto C, Ribaudo HJ, et al. Cardiovascular hazards of abacavir- versus tenofovir-containing antiretroviral therapies: insights from an analysis of the REPRIEVE trial cohort. *Open Forum Infect Dis*. 2025;12(4):ofaf177. Available at: https://www.ncbi.nlm.nih.gov/pubmed/40207047.

33. Fichtenbaum CJ, Malvestutto CD, Watanabe MG, et al. Effects of antiretrovirals on major adverse cardiovascular events in the REPRIEVE trial: a longitudinal cohort analysis. *Lancet HIV*. 2025;12(7):e496-e505. Available at: https://pubmed.ncbi.nlm.nih.gov/40482662.

34. DAD Study Group, Friis-Moller N, Reiss P, et al. Class of antiretroviral drugs and the risk of myocardial infarction. *N Engl J Med*. 2007;356(17):1723-1735. Available at: https://www.ncbi.nlm.nih.gov/pubmed/17460226.

35. Lang S, Mary-Krause M, Cotte L, et al. Impact of individual antiretroviral drugs on the risk of myocardial infarction in human immunodeficiency virus-infected patients: a case-control study nested within the French hospital database on HIV ANRS cohort CO4. *Arch Intern Med*. 2010;170(14):1228-1238. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20660842.

36. Ryom L, Lundgren JD, El-Sadr W, et al. Cardiovascular disease and use of contemporary protease inhibitors: the D:A:D international prospective multicohort study. *Lancet HIV*. 2018;5(6):e291-e300. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29731407.

37. Costagliola D, Potard V, Lang S, et al. Is the risk of myocardial infarction in people with human immunodeficiency virus (HIV) associated with atazanavir or darunavir? A nested case-control study within the French hospital database on HIV. *J Infect Dis*. 2020;221(4):516-522. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31828320.

38. Diaz CM, Segura ER, Luz PM, et al. Traditional and HIV-specific risk factors for cardiovascular morbidity and mortality among HIV-infected adults in Brazil: a retrospective cohort study. *BMC Infect Dis*. 2016;16:376. Available at: https://www.ncbi.nlm.nih.gov/pubmed/27503230.

39. Antinori A, Rusconi S, Gianotti N, Bini T, Mancusi D, Termini R. Cardiovascular adverse events during treatment with darunavir-based regimens in an Italian observational study. *Drug Des Devel Ther*. 2019;13:1667-1685. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31190745.

40. Neesgaard B, Greenberg L, Miró JM, et al. Associations between integrase strand-transfer inhibitors and cardiovascular disease in people living with HIV: a multicentre prospective study from the respond cohort consortium. *Lancet HIV*. 2022;9(7):e474-e485. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35688166.

41. Surial B, Chammartin F, Damas J, et al. Impact of integrase inhibitors on cardiovascular disease events in people with human immunodeficiency virus starting antiretroviral therapy. *Clin Infect Dis*. 2023;77(5):729-737. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37157869.

42. Rein SM, Lodi S, Logan RW, et al. Integrase strand-transfer inhibitor use and cardiovascular events in adults with HIV: an emulation of target trials in the HIV-causal collaboration and the antiretroviral therapy cohort collaboration. *Lancet HIV*. 2023;10(11):e723-e732. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37923486.

43. The RESPOND Study Group. Incidence of dyslipidemia in people with HIV who are treated with integrase inhibitors versus other antiretroviral agents. *AIDS*. 2021;35(6):869-882. Available at: https://www.ncbi.nlm.nih.gov/pubmed/33443370.

44. Oliveira DPR, Macêdo APA, Dos Santos Gonçalves M, Couto RD. Dyslipidemia secondary to the use of HAART in adults: a systematic review. *Curr Drug Res Rev*. 2022;14(3):239-246. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35619289.

45. Ofotokun I, Na LH, Landovitz RJ, et al. Comparison of the metabolic effects of ritonavir-boosted darunavir or atazanavir versus raltegravir, and the impact of ritonavir plasma exposure: ACTG 5257. *Clin Infect Dis*. 2015;60(12):1842-1851. Available at: https://www.ncbi.nlm.nih.gov/pubmed/25767256.

46. Gallant JE, Daar ES, Raffi F, et al. Efficacy and safety of tenofovir alafenamide versus tenofovir disoproxil fumarate given as fixed-dose combinations containing emtricitabine as backbones for treatment of HIV-1 infection in virologically suppressed adults: a randomised, double-blind, active-controlled phase 3 trial. *Lancet HIV*. 2016;3(4):e158-e165. Available at: https://www.ncbi.nlm.nih.gov/pubmed/27036991.

47. Ogbuagu O, Ruane PJ, Podzamczer D, et al. Long-term safety and efficacy of emtricitabine and tenofovir alafenamide vs emtricitabine and tenofovir disoproxil fumarate for HIV-1 pre-exposure prophylaxis: week 96 results from a randomised, double-blind, placebo-controlled, phase 3 trial. *Lancet HIV*. 2021;8(7):e397-e407. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34197772.

48. Glidden DV, Mulligan K, McMahan V, et al. Metabolic effects of preexposure prophylaxis with coformulated tenofovir disoproxil fumarate and emtricitabine. *Clin Infect Dis*. 2018;67(3):411-419. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29415175.

49. Martinez-Sanz J, Serrano-Villar S, Muriel A, et al. Metabolic-related outcomes after switching from tenofovir disoproxil fumarate to tenofovir alafenamide in adults with human immunodeficiency virus (HIV): a multicenter prospective cohort study. *Clin Infect Dis*. 2023;76(3):e652-e660. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35903910.

50. Plum PE, Maes N, Sauvage AS, et al. Impact of switch from tenofovir disoproxil fumarate-based regimens to tenofovir alafenamide-based regimens on lipid profile, weight gain and cardiovascular risk score in people living with HIV. *BMC Infect Dis*. 2021;21(1):910. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34488664.

51. De Wit S, Sabin CA, Weber R, et al. Incidence and risk factors for new-onset diabetes in HIV-infected patients: the Data Collection on Adverse Events of Anti-HIV Drugs (D:A:D) study. *Diabetes Care*. 2008;31(6):1224-1229. Available at: https://www.ncbi.nlm.nih.gov/pubmed/18268071.

52. O'Halloran JA, Sahrmann J, Parra-Rodriguez L, et al. Integrase strand transfer inhibitors are associated with incident diabetes mellitus in people with human immunodeficiency virus. *Clin Infect Dis*. 2022;75(12):2060-2065. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35521785.

53. Hwang YJ, Lesko CR, Brown TT, et al. Association between switching to integrase strand transfer inhibitors and incident diabetes in people with HIV. *AIDS*. 2024;38(11):1696-1702. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38864578.

54. Rupasinghe D, Bansi-Matharu L, Law M, et al. Integrase strand transfer inhibitor-related changes in body mass index and risk of diabetes: a prospective study from the RESPOND cohort consortium. *Clin Infect Dis*. 2025;80(2):404-416. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39117341.

55. Gutierrez F. "Editorial comment on: Increased incidence of diabetes in people living with HIV treated with first-line integrase strand transfer inhibitors: a French multicentre retrospective study". *HIV Med*. 2025;26(1):4-5. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39340145.

56. Food and Drug Administration. Symfi [package insert]. 2019. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2019/022142s037lbl.pdf.

57. Food and Drug Administration. Rukobia [package insert]. 2024. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/212950s004lbl.pdf.

58. Food and Drug Administration. Edurant [package insert]. 2024. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/202022Orig1s020;%20s022lbl.pdf.

59. Food and Drug Administration. Reyataz [package insert]. 2024. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/021567s049,206352s011lbl.pdf.

60. Food and Drug Administration. Kaletra [package insert]. 2020. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/021251s059,021906s054lbl.pdf.

61. Blumenthal RS, Morris PB, Gaudino M, et al. 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. *J Am Coll Cardiol*. 2026;87(19):2624-2757. Available at: https://www.ncbi.nlm.nih.gov/pubmed/41824590.

62. Feinstein MJ, Nance RM, Drozd DR, et al. Assessing and refining myocardial infarction risk estimation among patients with human immunodeficiency virus: a study by the Centers for AIDS Research Network of Integrated Clinical Systems. *JAMA Cardiol*. 2017;2(2):155-162. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28002550.

63. Thompson-Paul AM, Lichtenstein KA, Armon C, et al. Cardiovascular disease risk prediction in the HIV Outpatient Study. *Clin Infect Dis*. 2016;63(11):1508-1516. Available at: https://www.ncbi.nlm.nih.gov/pubmed/27613562.

64. van Zoest RA, Law M, Sabin CA, et al. Predictive performance of cardiovascular disease risk prediction algorithms in people living with HIV. *J Acquir Immune Defic Syndr*. 2019;81(5):562-571. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31045648.

65. Triant VA, Perez J, Regan S, et al. Cardiovascular risk prediction functions underestimate risk in HIV infection. *Circulation*. 2018;137(21):2203-2214. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29444987.

66. Grinspoon SK, Zanni MV, Triant VA, et al. Performance of the pooled cohort equations and D:A:D risk scores among individuals with HIV in a global cardiovascular disease prevention trial: a cohort study leveraging data from REPRIEVE. *Lancet HIV*. 2025;12(2):e118-e129. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39832519.

67. Soares C, Kwok M, Boucher KA, et al. Performance of cardiovascular risk prediction models among people living with HIV: a systematic review and meta-analysis. *JAMA Cardiol*. 2023;8(2):139-149. Available at: https://www.ncbi.nlm.nih.gov/pubmed/36576812.

68. Khan SS, Coresh J, Pencina MJ, et al. Novel prediction equations for absolute risk assessment of total cardiovascular disease incorporating cardiovascular-kidney-metabolic health: a scientific statement from the American Heart Association. *Circulation*. 2023;148(24):1982-2004. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37947094.

69. Diao JA, Shi I, Murthy VL, et al. Projected changes in statin and antihypertensive therapy eligibility with the AHA PREVENT cardiovascular risk equations. *JAMA*. 2024;332(12):989-1000. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39073797.

70. Durstenfeld MS, McLaughlin MM, Gandhi M, Kornak J, Beatty AL, Hsue PY. Prediction of cardiovascular risk among people with HIV using the PREVENT equations compared to the pooled cohort equations. *J Gen Intern Med*. 2026;41(1):64-72. Available at: https://www.ncbi.nlm.nih.gov/pubmed/40481382.

71. Durstenfeld MS, Nance RM, Jones R, et al. Predicting Risk of Cardiovascular Disease Events (PREVENT) score accuracy among people with HIV in the multicenter CNICS cohort. *AIDS*. 2025;39(13):F5-F11. Available at: https://www.ncbi.nlm.nih.gov/pubmed/40905667.

72. Grinspoon SK, Zhao S, Martinez E, et al. Risk assessment in a global CVD prevention cohort of people with HIV by PCE, PREVENT, and SCORE2. *Clin Infect Dis*. 2026;82(2):e248-e257. Available at: https://www.ncbi.nlm.nih.gov/pubmed/41004413.

73. Althoff KN, Gebo KA, Moore RD, et al. Contributions of traditional and HIV-related risk factors on non-AIDS-defining cancer, myocardial infarction, and end-stage liver and renal diseases in adults with HIV in the USA and Canada: a collaboration of cohort studies. *Lancet HIV*. 2019;6(2):e93-e104. Available at: https://www.ncbi.nlm.nih.gov/pubmed/30683625.

74. Du M, Zhang S, Liu M, Liu J. Cardiovascular disease and its risk factors among people living with HIV: a systematic review and meta-analysis. *J Infect Public Health*. 2025;18(2):102654. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39793126.

75. Liu Z, Zhang J, Yang X, et al. The dynamic risk factors of cardiovascular disease among people living with HIV: a real-world data study. *BMC Public Health*. 2024;24(1):1162. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38664682.

76. Silverberg MJ, Levine TM, Lea AN, et al. Cardiovascular disease risk factor control in people with and without HIV. *Clin Infect Dis*. 2024;78(5):1264-1271. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38227614.

77. Lloyd-Jones DM, Allen NB, Anderson CAM, et al. 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*. 2022;146(5):e18-e43. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35766027.

78. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2019;140(11):e563-e595. Available at: https://www.ncbi.nlm.nih.gov/pubmed/30879339.

79. Writing Committee Members, Jones DW, Ferdinand KC, et al. 2025 AHA/ACC/AANP/AAPA/ABC/ACCP/ACPM/AGS/AMA/ASPC/NMA/PCNA/SGIM guideline for the prevention, detection, evaluation and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Hypertension*. 2025;82(10):e212-e316. Available at: https://www.ncbi.nlm.nih.gov/pubmed/40811516.

80. Albert MA, Danielson E, Rifai N, Ridker PM, PRINCE Investigators. Effect of statin therapy on C-reactive protein levels: the pravastatin inflammation/CRP evaluation (PRINCE): a randomized trial and cohort study. *JAMA*. 2001;286(1):64-70. Available at: https://www.ncbi.nlm.nih.gov/pubmed/11434828.

81. Ridker PM, Danielson E, Fonseca FA, et al. Reduction in C-reactive protein and LDL cholesterol and cardiovascular event rates after initiation of rosuvastatin: a prospective study of the JUPITER trial. *Lancet.* 2009;373(9670):1175-1182. Available at: https://www.ncbi.nlm.nih.gov/pubmed/19329177.

82. Ridker PM, Rifai N, Clearfield M, et al. Measurement of C-reactive protein for the targeting of statin therapy in the primary prevention of acute coronary events. *N Engl J Med*. 2001;344(26):1959-1965. Available at: https://www.ncbi.nlm.nih.gov/pubmed/11430324.

83. Ganesan A, Crum-Cianflone N, Higgins J, et al. High dose atorvastatin decreases cellular markers of immune activation without affecting HIV-1 RNA levels: results of a double-blind randomized placebo controlled clinical trial. *J Infect Dis*. 2011;203(6):756-764. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21325137.

84. Funderburg NT, Jiang Y, Debanne SM, et al. Rosuvastatin reduces vascular inflammation and T-cell and monocyte activation in HIV-infected subjects on antiretroviral therapy. *J Acquir Immune Defic Syndr*. 2015;68(4):396-404. Available at: https://www.ncbi.nlm.nih.gov/pubmed/25514794.

85. Toribio M, Fitch KV, Sanchez L, et al. Effects of pitavastatin and pravastatin on markers of immune activation and arterial inflammation in HIV. *AIDS*. 2017;31(6):797-806. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28252528.

86. Lo J, Lu MT, Ihenachor EJ, et al. Effects of statin therapy on coronary artery plaque volume and high-risk plaque morphology in HIV-infected patients with subclinical atherosclerosis: a randomised, double-blind, placebo-controlled trial. *Lancet HIV*. 2015;2(2):e52-e63. Available at: https://www.ncbi.nlm.nih.gov/pubmed/26424461.

87. Longenecker CT, Sattar A, Gilkeson R, McComsey GA. Rosuvastatin slows progression of subclinical atherosclerosis in patients with treated HIV infection. *AIDS*. 2016;30(14):2195-2203. Available at: https://www.ncbi.nlm.nih.gov/pubmed/27203715.

88. Cholesterol Treatment Trialists (CTT) Collaboration, Baigent C, Blackwell L, et al. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170,000 participants in 26 randomised trials. *Lancet*. 2010;376(9753):1670-1681. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21067804.

89. Warden BA, Guyton JR, Kovacs AC, et al. Assessment and management of statin-associated muscle symptoms (SAMS): a clinical perspective from the National Lipid Association. *J Clin Lipidol*. 2023;17(1):19-39. Available at: https://www.ncbi.nlm.nih.gov/pubmed/36115813.

90. Sattar N, Preiss D, Murray HM, et al. Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials. *Lancet*. 2010;375(9716):735-742. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20167359.

91. Waters DD, Ho JE, Boekholdt SM, et al. Cardiovascular event reduction versus new-onset diabetes during atorvastatin therapy: effect of baseline risk factors for diabetes. *J Am Coll Cardiol*. 2013;61(2):148-152. Available at: https://www.ncbi.nlm.nih.gov/pubmed/23219296.

92. Balasubramanian R, Maideen NMP. HMG-CoA reductase inhibitors (statins) and their drug interactions involving CYP enzymes, P-glycoprotein, and OATP transporters—an overview. *Curr Drug Metab*. 2021;22(5):328-341. Available at: https://www.ncbi.nlm.nih.gov/pubmed/33459228.

93. Willemars MMA, Nabben M, Verdonschot JAJ, Hoes MF. Evaluation of the interaction of sex hormones and cardiovascular function and health. *Curr Heart Fail Rep*. 2022;19(4):200-212. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35624387.

94. Guldan M, Unlu S, Abdel-Rahman SM, et al. Understanding the role of sex hormones in cardiovascular kidney metabolic syndrome: toward personalized therapeutic approaches. *J Clin Med*. 2024;13(15):4354. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39124622.

95. Turino Miranda K, Kalenga CZ, Saad N, et al. Gender-affirming estrogen therapy route of administration and cardiovascular risk: a systematic review and narrative synthesis. *Am J Physiol Heart Circ Physiol*. 2022;323(5):H861-H868. Available at: https://www.ncbi.nlm.nih.gov/pubmed/36053748.

96. Rytz CL, Miranda KT, Ronksley PE, et al. Association between serum estradiol and cardiovascular health among transgender adults using gender-affirming estrogen therapy. *Am J Physiol Heart Circ Physiol*. 2024;327(2):H340-H348. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38578239.

97. Poteat TC, Ehrig M, Ahmadi H, et al. Hormones, stress, and heart disease in transgender women with HIV in LITE Plus. *Am J Prev Med*. 2025;68(2):245-256. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39389223.

98. Lake JE, Wang R, Barrett BW, et al. Trans women have worse cardiovascular biomarker profiles than cisgender men independent of hormone use and HIV serostatus. *AIDS*. 2022;36(13):1801-1809. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35950945.

99. Martinez CA, Rikhi R, Fonseca Nogueira N, et al. Estrogen-based gender-affirming hormone therapy and subclinical cardiovascular disease in transgender women with HIV. *LGBT Health*. 2023;10(8):576-585. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37459150.

100. Poteat TC, Rich AJ, Jiang H, et al. Cardiovascular disease risk estimation for transgender and gender-diverse patients: cross-sectional analysis of baseline data from the LITE Plus cohort study. *AJPM Focus*. 2023;2(3):100096. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37790660.

101. Grant JK, Loyd Q, Martinez C. Testosterone, HIV, and cardiovascular disease risk. *Cardiovasc Endocrinol Metab*. 2020;10(2):72-79. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34124602.

102. Garcia M, Londoño V, Martinez C. Updates on testosterone deficiency in men living with HIV and the cardiovascular implications. *Current Infectious Disease Reports*. 2025;27(1):3. Available at: https://doi.org/10.1007/s11908-024-00851-x.

103. Masumori N, Nakatsuka M. Cardiovascular risk in transgender people with gender-affirming hormone treatment. *Circ Rep*. 2023;5(4):105-113. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37025940.

104. Norwood J, Jenkins CA, Bhatta M, et al. Hormonal contraception and risk of cardiometabolic disease in women with HIV. *J Womens Health (Larchmt)*. 2024;33(7):966-974. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38484324.

105. King EM, Prior JC, Pick N, et al. Menopausal hormone therapy for women living with HIV. *Lancet HIV*. 2021;8(9):e591-e598. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34384545.

106. Uddenberg ER, Safwan N, Saadedine M, Hurtado MD, Faubion SS, Shufelt CL. Menopause transition and cardiovascular disease risk. *Maturitas*. 2024;185:107974. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38555760.

107. Cho L, Kaunitz AM, Faubion SS, et al. Rethinking menopausal hormone therapy: for whom, what, when, and how long? *Circulation*. 2023;147(7):597-610. Available at: https://www.ncbi.nlm.nih.gov/pubmed/36780393.

108. Cameron CR, Cohen S, Sewell K, Lee M. The art of hormone replacement therapy (HRT) in menopause management. *J Pharm Pract*. 2024;37(3):736-740. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37002679.

109. Bullington BW, Edmonds A, Ramirez C, et al. Premature and early menopause among US women with or at risk for HIV. *Menopause*. 2022;29(6):741-747. Available at: https://pubmed.ncbi.nlm.nih.gov/35324546.

110. Dragovic B, Rymer J, Nwokolo N. Menopause care in women living with HIV in the UK - a review. *J Virus Erad*. 2022;8(1):100064. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35242357.

111. Horberg M, Thompson M, Agwu A, et al. Primary care guidance for providers who care for persons with human immunodeficiency virus: 2024 update by the HIV Medicine Association of the Infectious Diseases Society of America. *Clinical Infectious Diseases*. 2024:ciae479. Available at: https://doi.org/10.1093/cid/ciae479.

112. Majonga ED, Henderson M, Ferrand RA. Cardiovascular health in people with perinatally acquired HIV - where do we stand? *Curr Opin HIV AIDS*. 2024;19(6):348-354. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38935060.

113. McCrary AW, Nyandiko WM, Ellis AM, et al. Early cardiac dysfunction in children and young adults with perinatally acquired HIV. *AIDS*. 2020;34(4):539-548. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31794518.

114. Jao J, Zar HJ, Kahts M, et al. Subclinical myocardial fibrosis in South African youth with HIV: results from the CTAAC-HEART study. *Open Forum Infect Dis*. 2024;11(10):ofae555. Available at: https://www.ncbi.nlm.nih.gov/pubmed/39416995.

115. Wilkinson JD, Williams PL, Yu W, et al. Cardiac and inflammatory biomarkers in perinatally HIV-infected and HIV-exposed uninfected children. *AIDS*. 2018;32(10):1267-1277. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29596110.

116. Lipshultz SE, Wilkinson JD, Thompson B, et al. Cardiac effects of highly active antiretroviral therapy in perinatally HIV-infected children: the CHAART-2 study. *J Am Coll Cardiol*. 2017;70(18):2240-2247. Available at: https://pubmed.ncbi.nlm.nih.gov/29073951.

117. Lipshultz SE, Wilkinson JD, Thompson B, et al. Cardiac effects of highly active antiretroviral therapy in perinatally HIV-infected children: The CHAART-2 study. *J Am Coll Cardiol*. 2017;70(18):2240-2247. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29073951.

118. Dirajlal-Fargo S, Sattar A, Kulkarni M, Bowman E, Funderburg N, McComsey GA. HIV-positive youth who are perinatally infected have impaired endothelial function. *AIDS*. 2017;31(14):1917-1924. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28590332.

119. Majonga ED, Ferrand RA, Deanfield JE, Chiesa ST. The effect of perinatal HIV and antiretroviral therapy on vascular structure and function in young people: a systematic review and meta-analysis. *Atherosclerosis*. 2022;352:53-61. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35688118.

120. Dirajlal-Fargo S, Strah M, Ailstock K, et al. Factors associated with insulin resistance in a longitudinal study of Ugandan youth with and without HIV. *AIDS*. 2024;38(2):177-184. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37788113.

121. Dirajlal-Fargo S, Jacobson DL, Yu W, et al. Longitudinal changes in body fat and metabolic complications in young people with perinatally acquired HIV. *HIV Med*. 2024;25(2):233-244. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37845017.

122. Mahtab S, Frigati LJ, Ntusi NAB, et al. The determinants of elevated pathobiological determination of atherosclerosis in youth risk score in perinatally HIV-infected adolescents in South Africa. *J Acquir Immune Defic Syndr*. 2024;95(1):82-89. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37851954.

123. Greybe L, Barnabas S, Cotton M, Rose P, Rabie H, Frigati L. Cardiometabolic risk profiles of adolescents living with perinatally acquired HIV in South Africa. *Pediatr Infect Dis J*. 2024;43(7):669-674. Available at: https://www.ncbi.nlm.nih.gov/pubmed/38535131.

124. Haw NJL, Lesko CR, Ng DK, et al. Incidence of non-AIDS defining comorbidities among young adults with perinatally acquired HIV in North America. *AIDS*. 2024;38(9):1366-1374. Available at: https://pubmed.ncbi.nlm.nih.gov/38507583.