Both HIV and its treatment, particularly protease inhibitors, can cause dyslipidemia similar to that seen with the metabolic syndrome. The most notable effects are elevated triglyceride levels and decreased high-density lipoprotein cholesterol levels, with or without elevated low-density lipoprotein cholesterol (LDL-C) levels.

Highly active antiretroviral therapy and the adverse metabolic effects of HIV infection itself are associated with the development of cardiovascular risk factors. By itself, HIV infection is associated with atherogenic dyslipidemia, which includes low levels of high-density lipoprotein cholesterol (HDL-C) and hypertriglyceridemia. Antiretroviral therapy may exacerbate lipid abnormalities, and dyslipidemia may develop in up to 70% to 80% of treated HIV-infected patients, with hypertriglyceridemia occurring in the majority of cases (60% to 100% of treated patients). The presence of hypertriglyceridemia enhances the development of small, dense low-density lipoprotein (LDL) particles, a recognized atherogenic phenomenon that may be accentuated by regimens that contain lopinavir/ritonavir, for example. Protease inhibitor (PI) therapy may also depress HDL-C levels, although other classes of antiretroviral agents may have a more neutral or favorable effect on this parameter.

The National Cholesterol Education Program (NCEP) Adult Treatment Program III (ATP III) has made recommendations for treatment of dyslipidemia in HIV-infected patients. While acknowledging that there is limited experience with lipid-modifying drugs in this population and minimal documentation that such therapies will prevent cardiovascular disease (CVD) in patients with HIV-associated dyslipidemia, the guidelines recognize that drug treatment can help control the lipid abnormalities that may occur.

Management of triglyceride levels may be especially important because of the risk of pancreatitis associated with extreme triglyceride level elevations. As in uninfected patients, lipid risk-factor management should focus on lowering LDL cholesterol (LDL-C) levels, and drug options include statins. For other aspects of HIV-associated dyslipidemia, fibrates, nicotinic acid, and omega-3 fatty acids alone or in combination may help lower triglyceride levels. This article examines the evolving knowledge of the management of antiretroviral-related and HIV-related dyslipidemia, with a focus on reducing triglyceride levels with omega-3 fatty acids.
mechanisms include altered adipogenesis and lipolysis and increased synthesis of hepatic triglycerides.\textsuperscript{16-19} Moreover, dyslipidemia has been observed in patients treated with the nucleoside analogue stavudine\textsuperscript{20-22} and the NNRTI efavirenz.\textsuperscript{23} The term “HIV lipodystrophy syndrome” describes the constellation of clinical findings, including peripheral fat wasting, central adiposity, dyslipidemia, and insulin resistance.\textsuperscript{24} Although the causes of visceral fat hypertrophy are not yet known, PIs and NRTIs have been shown to decrease differentiation and adipogenesis in subcutaneous adipose tissue.\textsuperscript{25} The reader is referred to a recent review for a detailed discussion of the potential mechanisms for metabolic abnormalities in HIV-infected patients receiving antiretroviral therapy.\textsuperscript{25}

**Prevalence of Cardiovascular Disease**

Improved therapeutic management of HIV disease has reduced mortality due to opportunistic infections or other sequelae of HIV infection. As a consequence of this improvement, however, HIV-infected patients now may be vulnerable to other causes of morbidity and mortality.\textsuperscript{11,26,27} Before the HAART era, reports of CVD in association with HIV were infrequent, perhaps because the patients did not live long enough for CVD to develop. CVD can be associated with HIV infection, opportunistic infections or neoplasias, mode of HIV acquisition (such as injection drug use), antiretroviral therapy, or the classic non–DHIV-related cardiovascular risk factors (such as smoking or older age).\textsuperscript{28} Dyslipidemia and vascular inflammation are considered the 2 main sources of CVD in patients with HIV disease.\textsuperscript{26}

The long-term use of PI therapy has been associated with an increased risk of myocardial infarction (MI) and coronary disease. The Kaiser Permanente Registry study reviewed data from the Kaiser Permanente HMO.\textsuperscript{29} The 2004 tally identified 4726 HIV-positive patients aged 35 to 64 years from January 1996 through June 2003. These patients had an age-adjusted coronary heart disease (CHD) rate of 6.6 (vs 3.0 in those without HIV) per 1000 patient-years ($P < .0001$). The age-adjusted rate of MI in patients receiving PIs was 3.9 (vs 2.2 in those not receiving PIs) per 1000 patient-years ($P < .005$). The median time of PI exposure was 4.0 years.

Events of Anti-HIV Drugs (DAD) study, a prospective, observational study, enrolled more than 23,000 patients from 11 previously established cohorts.\textsuperscript{30} The DAD study reported a 26% relative increase in MI per year of exposure during the first 4 to 6 years of antiretroviral therapy (Figure).\textsuperscript{30} Other independent risk factors were age, male sex, previous CVD, and smoking. There is evidence of accelerated atherosclerosis in HIV-infected patients without conventional risk factors.\textsuperscript{31,32}

**Figure.** Incidence of myocardial infarction in the DAD study. (Adapted from Data Collection on Adverse Events of Anti-HIV Drugs (DAD) Study Group. N Engl J Med. 2003.)\textsuperscript{30}

Endothelial dysfunction may be caused by HIV infection, immunological responses due to the virus, or the effects of antiretroviral therapy.\textsuperscript{33} HIV infection can cause functional alterations of the endothelium, resembling the subclinical inflammation in atherosclerosis.\textsuperscript{28} Even HIV-infected patients who have low or mild cardiovascular risk and lipid levels within the normal range were shown to have endothelial dysfunction compared with healthy controls.\textsuperscript{34} Metabolic parameters that may affect cardiovascular risk in HIV-infected patients receiving antiretroviral therapy are listed in Table 1.
There is still debate on the relative cardiovascular risk in patients with HIV infection. A retrospective analysis of cardiovascular and cerebrovascular disease among 36,000 patients with HIV infection found that the use of any class of antiretroviral therapy was associated with a decreased hazard rate of death from any cause. However, the mean period of exposure to antiretroviral therapy was 15 months, and only 2.7% of patients were exposed to PIs for 48 months. Thus, the results of this study indicate that antiretroviral therapy has a benefit that is enormous relative to the risk of cardiovascular or cerebrovascular disease and that fear of cardiovascular complications of antiretroviral therapy should not prevent its use.

Recently, Kwong and colleagues evaluated data on antiretroviral therapy, risk factors for CVD and atherosclerotic disease, and death from other causes in 18,603 HIV-infected patients. Overall, the benefits of treatment with PIs and NNRTIs significantly outweighed any risks of atherosclerotic disease during the 3.49-year (median) follow-up. However, increased risk of atherosclerotic disease was significantly associated with older age, hypertension, diabetes mellitus, having smoked, and HIV disease stage. Although uncontrolled viremia may pose more of a cardiovascular risk than controlled infection that results in dyslipidemia and insulin resistance, evidence suggests that HIV-infected patients treated with combination antiretroviral regimens may be at increased risk for the development of premature atherosclerotic complications. In addition, a recent report by the Strategies for Management of Antiretroviral Therapy (SMART) Study Group indicates that episodic use of antiretroviral therapy in HIV-infected patients is associated with increased risk of fatal or nonfatal CVD ($P = .05$ vs continuous use of antiretroviral therapy).

**MANAGEMENT OF HIV-ASSOCIATED DYSLIPIDEMIA**

Although clinical trials to evaluate the effect of lipid modification on cardiovascular risk in HIV-infected patients have proved difficult to realize, there is no compelling argument to support denying HIV-infected patients cardiovascular risk management as part of their long-term care. Therefore, dyslipidemia should be considered an important, treatable coronary risk factor in HIV-infected patients. The Infectious Diseases Society of America and the Adult AIDS Clinical Trials Group have issued guidelines for the treatment of metabolic complications (Table 2). The lipid guidelines largely reflect those of the NCEP.
Important principles for treating dyslipidemia include assessing overall cardiovascular risk based on the total risk-factor profile, obtaining fasting lipid levels, targeting interventions based on the lipid profile, and monitoring response. The primary focus of the NCEP ATP III guidelines is treatment of elevated LDL-C level. In these guidelines, triglyceride level elevations are considered an independent coronary risk factor with several metabolic associations, such as with overweight/obesity and diabetes and with the use of certain drugs, such as antiretroviral agents.

Some data suggest that non–DHDL-C level may be a better predictor of coronary risk than LDL-C, because this measure captures the contribution of atherogenic, triglyceride-rich lipoproteins to risk. Therefore, ATP III recommends reducing non–DHDL-C level (calculated by subtracting HDL-C from total cholesterol) as a secondary target in patients with triglyceride levels of 200 mg/dL or higher, once LDL-C levels are controlled.

Switching Antiretroviral Drugs
Some studies suggest that switching antiretroviral drugs can improve dyslipidemia and insulin resistance in HIV-infected patients. Studies of atazanavir in vitro suggest that it may have a reduced ability to induce lipodystrophy. Keiser and colleagues showed that in antiretroviral-experienced patients, substituting abacavir for dyslipidemia-associated PIs in antiretroviral drug regimens improved lipid profiles and maintained viral suppression over 28 weeks. Martinez and colleagues found that switching from PIs to nevirapine may reduce metabolic abnormalities, including reducing total cholesterol level (22% reduction; \( P = .0005 \)) and reducing triglyceride levels (57% reduction; \( P = .0001 \)).

Lipid-Lowering Therapy
To bring patients to goal lipid levels, lipid-lowering therapy may be necessary. The commonly recommended drug interventions for HIV-related dyslipidemia are statins and fibrates, depending on the abnormal lipid parameters that may be present. Indeed, Calza and colleagues showed that adding pharmacological lipid-lowering therapy (with pravastatin or bezafibrate) was significantly more effective in the management of antiretroviral therapy–related dyslipidemia than switching from a PI to an NNRTI.

Use of Omega-3 Fatty Acids
Omega-3 fatty acids are polyunsaturated fatty acids in which the first double bond from the terminal (omega) methyl group is at carbon 3. Omega-3 (\( \alpha \)-linolenic acid, eicosapentaenoic acid [EPA], and docosahexaenoic acid [DHA]) fatty acids are 1 of the 2 classes of essential fatty acids. Only small amounts of plant-derived omega-3 fatty acids are converted to EPA in vivo, and further transformation to DHA is very low. In the United States, the average intake of omega-3 fatty acids is about 100 to 200 mg/d. A combined daily dose of 3.4 g of EPA and DHA is required to achieve the reduction needed in patients with very high triglyceride levels. Fatty fish, such as albacore tuna, sardines, salmon, mackerel, and herring, are the most concentrated food source of EPA and DHA. However, consumption of fatty fish is not likely to provide sufficient amounts of DHA and EPA for the effective management of hypertriglyceridemia. For example, a person would need to consume 16 oz of
canned albacore tuna per day to obtain approximately 4 g of EPA and DHA. Moreover, some larger species of fish (eg, shark, king mackerel, swordfish, and tilefish) contain high levels of methylmercury, dioxins, and polychlorinated biphenyls.

In the United States, dietary supplements with the omega-3 fatty acids EPA and DHA are available over the counter as well as in a prescription formulation. The prescription formulation is indicated as an adjunct to diet to reduce very high triglyceride levels (500 mg/dL or higher) in adults. For triglyceride reduction, this agent can be taken either as a single 4-g dose (4 capsules) or as two 2-g doses (2 capsules twice daily).

**Mechanism of action.** After oral administration of omega-3 fatty acids in both healthy volunteers and patients with hypertriglyceridemia, EPA and DHA are well absorbed and lead to significant, dose-dependent increases in serum phospholipid EPA content. Although details on the cellular mechanisms by which omega-3 fatty acids reduce serum triglyceride levels are not completely understood, EPA and DHA may reduce very-low-density lipoprotein (VLDL)-triglyceride synthesis, enhance triglyceride clearance from VLDL particles, and/or increase conversion of VLDL remnants to LDL.

The reader is referred to a recent review for a detailed discussion of potential mechanisms for the triglyceride-lowering effects of omega-3 fatty acids.

**Efficacy and safety in the general population.** Potential cardioprotective effects of omega-3 fatty acids were first recognized when indigenous populations who consumed high concentrations of EPA and DHA—such as marine mammals and fatty fish—were observed to have low rates of CHD. In patients with established CHD, omega-3 fatty-acid intake is associated with a reduced risk of mortality from MI and sudden death.

Omega-3 fatty acids have been shown to lower serum triglyceride levels in a dose-dependent manner in both crossover and parallel-design studies of participants with mean baseline triglyceride levels less than 2 mmol/L or those with levels 2 mmol/L and higher. EPA and DHA, which appear to have similar triglyceride-lowering effects, can reduce triglyceride levels in a wide range of patient types. Clinical studies have shown that EPA and DHA lower fasting and postprandial triglyceride levels without clinically significant effects on fat absorption. A review of 10 randomized, controlled trials of participants with triglyceride levels greater than 150 mg/dL (greater than 1.69 mmol/L) showed that among patients taking EPA and/or DHA in dosages of 3.4 to 4 g/d, triglyceride levels were reduced by an average of 29% (range, 16% to 45%).

Treatment with omega-3 fatty acids may induce modest increases in HDL-C levels on the order of 10% but also may raise LDL-C levels to varying degrees. The safety and efficacy of omega-3 fatty acids for the treatment of hypertriglyceridemia in the general population is well documented. As reported to the FDA, pooled data from 8 randomized, placebo-controlled, double-blind, parallel-group studies conducted in participants with hypertriglyceridemia have shown that treatment with omega-3 fatty acids is safe and well tolerated. In clinical trials, the most common adverse events were eructation, infection, dyspepsia, and flu syndrome. The only adverse event occurring significantly more frequently with omega-3 fatty acids than with placebo was "taste perversion" (principally, "fishy taste") at an incidence of 2.7% with omega-3 fatty acids vs 0% with placebo (P = .0147). Adverse events led to treatment discontinuation in 3.5% of patients treated with omega-3 fatty acids, compared with 2.6% of patients who received placebo.

Because free forms of EPA and DHA are not detected in the circulation, drug interactions due to the inhibition of cytochrome P-450 are not expected. The antithrombotic effects of omega-3 fatty acids have raised concerns about prolonged bleeding time, but clinical trials have not shown an increased risk of bleeding. Although omega-3 fatty acids have been shown to reduce thrombin generation in a vitamin K–independent manner, to date, there are no published data showing significant changes in bleeding time or propensity for bleeding among patients treated with FDA-approved doses of omega-3 fatty acids.

No experience with concurrent clopidogrel therapy has been reported; however, experience with omega-3 fatty acid therapy in patients receiving coumarin anticoagulants, aspirin, and other older antiplatelet agents has not revealed increased bleeding. A study of the interaction between fish oil and warfarin did not show increases in international normalized ratios or major bleeding episodes or the need to reduce the dose of warfarin. Nevertheless, monitoring is reasonable in patients who are receiving concomitant antiplatelet or anticoagulant therapies.

In some patients, increases in alanine aminotransferase (ALT) levels without a concurrent increase in aspartate aminotransferase levels were observed. Therefore, ALT levels should be monitored periodically during omega-3 fatty acid therapy. Because of an increase in LDL-C levels observed in some patients treated with omega-3 fatty acids (the mechanism of which is not completely understood), LDL-C levels should be periodically assessed during treatment.
should be used with caution in patients with sensitivity or allergy to fish.  

**Combination therapy with statins and omega-3 fatty acids.** Few studies of combination therapies of omega-3 fatty acid and statins in patients with dyslipidemia have been published. Intuitively, one would assume that these therapies would have an additive effect, but no trials have been conducted in HIV-infected patients. Two clinical trials have demonstrated the safety and efficacy of omega-3 fatty acids and simvastatin in the general population.\(^7.8\) However, drug interactions between antiretroviral drugs and lipid-lowering drugs need to be considered,\(^7\) such as the use of simvastatin being contraindicated in patients receiving PI-containing regimens.\(^8\) Durrington and colleagues\(^7\) showed that omega-3 fatty acids were effective and safe in lowering triglyceride levels over 1 year in patients with CHD who had elevated triglyceride levels while receiving simvastatin treatment alone. The participants (n = 46) had established CHD and triglyceride levels higher than 2.26 mmol/L (higher than 200 mg/dL) despite maintenance therapy with simvastatin 10 to 40 mg/d. Those who received background therapy with simvastatin were randomized to omega-3 fatty acids 4 g/d or placebo for 24 weeks, followed by an open-label extension of active treatment for another 24 weeks. At 3, 6, and 12 months, omega-3 fatty acid treatment resulted in serum triglyceride level reductions of 20% to 30% (P < .005) and VLDL cholesterol level reductions of 30% to 40% (P < .005) compared with either baseline levels or levels seen in the placebo group. Changes were not related to simvastatin use. No increase in LDL-C level or decrease in HDL-C level was observed, and adverse events were mild.

McKenney and colleagues\(^8\) conducted an open-label, randomized, 2-way crossover, drug-drug interaction study to evaluate single-dose and steady-state simvastatin kinetics in participants taking omega-3 fatty acids. The study consisted of two 14-day dosing periods separated by a washout period of at least 14 days. During the first 14-day dosing period, healthy volunteers (N = 24) were randomized to receive daily morning oral doses of either 4 g (4 capsules) of omega-3 fatty acids coadministered with 80 mg simvastatin (test treatment) or 80 mg simvastatin alone (reference treatment). During the second 14-day dosing period, participants received the alternative treatment. Omega-3 fatty acids did not appear to affect the pharmacokinetics of simvastatin after repeated dose administration, and they were safe and well tolerated.

**Efficacy and Safety in HIV-Infected Patients**

Wohl and colleagues\(^9\) conducted an open-label, randomized trial in which 52 patients were enrolled and received at least 3 antiretroviral agents. Fasting triglyceride levels were above 200 mg/dL. Patients received dietary and exercise counseling for 16 weeks, with or without omega-3 fatty acids (1750 mg of EPA and 1150 mg of DHA). Patients who received omega-3 fatty acids had a 25% mean reduction in fasting triglyceride levels at 4 weeks, compared with a 2.8% mean increase in patients receiving placebo (P = .007).

By 16 weeks, the mean reduction in triglycerides in patients receiving fish oil was 19.5%, whereas the mean decrease in patients not receiving fish oil was 5.7%, but the difference was not significant. LDL-C levels had increased by 15.6% at week 4 and by 22.4% at week 16 in the omega-3 fatty acid arm and by 3.5% and 18.4%, respectively, in the group not taking omega-3 fatty acids (P = .14).

To what extent the increase in LDL-C was attributable to the omega-3 fatty acids and whether this increase attenuates the beneficial effect of lowering triglyceride levels is not clear. The investigators concluded that given the benefits of reducing triglyceride levels with omega-3 fatty acids in HIV-infected patients, additional study of this therapy is appropriate.\(^9\)

Manfredi and colleagues\(^8\) conducted a prospective, open-label assessment of the efficacy and safety of omega-3 fatty acids in HIV-infected patients with triglyceride levels of 250 to 500 mg/dL: 54 patients received omega-3 fatty acids, 53 received fibrates, and 49 were treated with diet and exercise only. Both omega-3 fatty acids and fibrates produced significant (P < .0001) reductions in mean triglyceride levels at 18 months, with fibrates showing a slightly more potent effect. Diet and exercise alone did not have a significant effect. Fourteen patients who received omega-3 fatty acids (26%) and 18 who received fibrates (34%) had normal triglyceride levels (less than 1.94 mmol/L [172 mg/dL]) by 18 months, which was not statistically significant.

Both omega-3 fatty acids and fibrates prevented the need to change antiretroviral regimens as a result of dyslipidemia. Fibrates were associated with a greater incidence of mild and transient GI disturbances than was lifestyle therapy or omega-3 fatty acids, although no discontinuations of therapy were required.\(^8\)

Researchers in France randomized 122 HIV-positive patients to either two 1-g capsules of omega-3 fatty acid 3 times per day or placebo for 8 weeks, followed by 8 weeks of open-label omega-3 fatty acids for all patients.\(^8\) Neither total cholesterol nor HDL-C levels changed over the course of therapy in either group, but triglyceride levels did decrease significantly (Table 3). Ten patients with baseline
triglyceride levels above 10 g/L (greater than 1000 mg/dL) were given open-label omega-3 fatty acids. These patients also experienced a 35.6% mean reduction in triglyceride concentrations after 8 weeks, demonstrating in this study that omega-3 fatty acids are effective even in patients with very high triglyceride levels. Treatment was well tolerated. Taken together, these data indicate that omega-3 fatty acids reduce triglyceridemia in HIV-infected patients receiving antiretroviral therapy.

Baril and colleagues\textsuperscript{10} evaluated the effect of salmon oil (an abundant source of omega-3 fatty acids) on elevated triglyceride levels and lipid parameters in HIV-infected patients receiving antiretroviral therapy. This was a phase 4, randomized, parallel, crossover, open-label study. Patients were randomly assigned to receive 1 g of salmon oil 3 times a day for 24 weeks (group A) or no additional treatment for 12 weeks and then salmon oil for the next 12 weeks (group B).

Fifty-eight of the 67 patients enrolled completed the study. Nine patients discontinued because of adverse events, 3 withdrew consent, and 1 stopped antiretroviral therapy. At 12 weeks, group A had a mean reduction in triglyceride levels of 95.7 mg/dL, versus an increase of 26.9 mg/dL in group B (\(P = .040\)). When patients in group B crossed over to receive salmon oil, the mean triglyceride level decreased by 62.7 mg/dL (\(P = .056\)). The total cholesterol-HDL ratio decreased nonsignificantly, reflecting a reduction in total cholesterol level. Other lipid values did not change significantly.

Seventeen patients reported minor adverse effects. Omega-3 fatty acids have demonstrated anti-inflammatory effects, such as inhibition of cytokine production, that may have implications for immunomodulation. While one study has suggested that enteral nutritional supplementation enriched with omega-3 fatty acids may have beneficial effects on CD4 count and HIV-associated weight loss, the effect of omega-3 fatty acids on such measures has not been established.\textsuperscript{83}

**Combination therapy with fenofibrate.** AIDS Clinical Trial Group A5186 evaluated the safety and efficacy of EPA (1.5 g) and DHA (0.91 g) twice a day in combination with fenofibrate (160 mg/d) in HIV-infected patients who had not responded adequately to either agent alone.\textsuperscript{84} In this open-label, prospective study, 100 patients who were receiving antiretroviral therapy and had triglyceride levels above 4.52 mmol/L (400 mg/dL) were randomly assigned to fenofibrate or EPA and DHA. If the triglyceride level remained above 2.26 mmol/L (200 mg/dL) at 8 weeks, patients were given combination therapy.

The median baseline serum triglyceride level was 7.47 mmol/L (662 mg/dL) in the omega-3 fatty acid group and 7.83 mmol/L (694 mg/dL) in the fenofibrate group. During the first 8 weeks, omega-3 fatty acids and fenofibrate decreased serum triglyceride levels by 46% and 58%, respectively (\(P = .039\)). Four patients who received omega-3 fatty acids (8.5%) and 8 who received fenofibrate (16.7%) achieved the goal level; 75 participants (90.4%) proceeded to combination therapy. The median decrease in serum triglyceride levels from baseline to week 18 was 65% for patients participating in the combination phase of the study. The individual therapies and combination therapy were well tolerated and safe. Thus, the combination therapy may be appropriate in patients who cannot achieve adequate reductions with omega-3 fatty acids or fenofibrate alone.

**CONCLUSION**

Cardiovascular disease has emerged as an important public health concern among the HIV-infected population.\textsuperscript{27,30,31,85,86} Smoking cessation and monitoring conditions such as diabetes and hypertension should be a component of risk reduction for the HIV-infected population.\textsuperscript{85,86} Dyslipidemia as a result of HIV infection and its treatment has led to concern about elevated...
cardiovascular risk in these patients. Management of dyslipidemia in HIV-infected adults should follow the NCEP guidelines established for the non–HIV-infected population. Although obtaining and maintaining virological control is the overriding concern in patients with HIV infection, there may be a need for a shift in the health care model for persons with AIDS, from a primary focus on managing HIV infection to providing care that addresses all aspects of physical and mental health. The emergence of metabolic and lipid disturbances presents a pharmacological challenge because of the potential for drug interactions. Lipid-lowering drugs can be safely given to most HIV-infected patients. If the patient is experiencing only an elevation in LDL-C levels, a statin is first-line drug treatment. For the treatment of hypertriglyceridemia, the use of fibrates or omega-3 fatty acids may be considered. A prescription preparation of omega-3-acid ethyl esters was recently approved by the FDA for use in adults as an adjunct to diet to reduce triglyceride levels of 500 mg/dL or higher. Combination therapy with a statin and omega-3 fatty acids may be an alternative for treating combined dyslipidemia.

HIV-associated dyslipidemia is a modifiable risk factor for cardiovascular disease. Physicians should treat chronic diseases as aggressively in HIV-infected patients as in non–HIV-infected patients. Judicious use of statins, fibrates, and omega-3 fatty acids—alone or in combination—may help control HIV-associated dyslipidemia. However, whether these approaches will decrease the risk of HIV-associated cardiovascular complications remains unclear at the present time. Additional research is needed to determine whether the metabolic complications that are associated with HIV infection and its therapies are different from the metabolic diseases (eg, diabetes and dyslipidemia) in the uninfected population. Despite the need for clinical trials to establish the benefits of lipid-lowering agents in HIV-infected patients, these patients are appropriate candidates for all usual methods of risk reduction and health maintenance.

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Treatment of HIV-Associated Dyslipidemia: A Role for Omega-3 Fatty Acids

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