

FEDERAL BUREAU OF PRISONS
CLINICAL PRACTICE GUIDELINES FOR HIGH CHOLESTEROL
(Issued - JUNE, 2002)

PURPOSE

The Federal Bureau of Prisons Clinical Practice Guidelines for High Cholesterol provide recommendations for the medical management of inmates with elevated LDL cholesterol.

REFERENCES

National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), National Institutes of Health, Bethesda, MD, accessible at www.nhlbi.nih.gov.

Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), *JAMA* 2001;285:2486-2497.

Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II), Second Report of the Expert Panel, National Cholesterol Education Program (NCEP), National Institutes of Health, Bethesda, MD.

Surgeon General's Call To Action To Prevent and Decrease Overweight and Obesity 2001, U.S. Department of Health and Human Services, accessible at www.surgeongeneral.gov/library.

BOP Food Service Manual, P.S. 4700.04 and Food Service Technical Reference Manual, P.S. 4701.02.

DEFINITIONS

Body Mass Index (BMI) is a measure of weight in relation to height. The BMI equals weight in pounds divided by the square of the height in inches, multiplied by 703; or alternatively in kilograms divided by the square of the height in meters. BMI is highly correlated with total body fat and is used to assess overweight and obesity. A BMI of 30 kg/M² or greater indicates obesity; whereas a BMI between 25 kg/M² and 29.9 kg/M² identifies

overweight adults.

Cholesterol is a fat-like substance that is present in cell membranes and is a precursor to steroid hormones and bile acids.

Clinician is a physician or midlevel provider.

Coronary atherosclerosis is the deposition of cholesterol and fibrin complexes within the lumen of a coronary artery, narrowing the lumen and thereby limiting blood flow.

Coronary Heart Disease (CHD) is atherosclerosis of one or more coronary arteries which has resulted in symptomatic disease such as angina pectoris, myocardial infarction, congestive heart failure, or history of coronary artery surgery or coronary angioplasty.

CHD risk factors (that modify LDL goals) are factors that increase the likelihood of developing coronary atherosclerosis and associated CHD exclusive of LDL cholesterol itself. Factors include: **age** (men \geq 45 years of age and women \geq 55 years of age, cigarette **smoking**, **hypertension** (blood pressure $>$ 140/90 mm Hg or taking antihypertensive medications), **family history** of premature CHD or sudden death (in male first degree relative $<$ 55 years of age, or female first degree relative $<$ 65 years of age), and **low HDL-cholesterol** ($<$ 40 mg/dL).

- **NOTE:** A high HDL cholesterol value of \geq 60 mg/dL is considered a "negative" risk factor and reduces the risk factor "count" by one. Diabetes mellitus is considered a CHD risk equivalent and is not "counted" as a separate risk factor, i.e., patients with diabetes who have elevated LDL cholesterol are treated as if they had CHD. Obesity should be considered a target for intervention but is not considered a separate risk factor since it associated with multiple other risk factors.

CHD risk equivalents are factors or conditions that carry a risk for major coronary events equal to that of established CHD ($>$ 20% per 10 years). CHD risk equivalents include diabetes mellitus, peripheral arterial disease, abdominal aortic aneurysm, symptomatic carotid artery disease, renal artery disease, or the presence of multiple CHD risk factors that together confer a 10-year risk for coronary events $>$ 20%.

High density lipoproteins (HDL) are lipoproteins that contain 20-

30% of total serum cholesterol and are inversely correlated with CHD risk.

Lipoproteins are lipid containing proteins in the blood that transport cholesterol throughout the body.

Lipoprotein analysis is the measurement of fasting levels of total cholesterol, total triglyceride, LDL and HDL cholesterol.

Low Density Lipoproteins (LDL) are lipoproteins that contain 60-70% of the total serum cholesterol. $\text{LDL cholesterol} = \text{total cholesterol} - \text{HDL cholesterol} - \text{triglycerides}/5$ (invalid if triglycerides are $> 400 \text{ mg/dL}$).

Metabolic syndrome is a constellation of factors associated with insulin resistance and obesity that increase the risk of coronary events at every LDL level. Metabolic syndrome is diagnosed when 3 or more the following risk determinants are present: Fasting glucose between 110-126 mg/dL, blood pressure $\geq 130/\geq 85 \text{ mm Hg}$, triglycerides $\geq 150 \text{ mg/dL}$, HDL $< 40 \text{ mg/dL}$ for men and $< 50 \text{ mg/dL}$ for women, or abdominal obesity (waist circumference $> 40 \text{ inches}$ for men and $> 35 \text{ inches}$ for women).

Peripheral arterial disease (PAD) is the presence of atherosclerotic disease of the aorta, arteries to the limbs, or carotid arteries as evidenced by abdominal aortic aneurysms, clinical signs or symptoms of ischemia to the extremities or to the brain (transient ischemic attacks or stroke) documented by significant atherosclerosis on sonogram, angiogram or other diagnostic studies.

Very low density lipoproteins (VLDL) are lipoproteins that contain most of the triglycerides present in fasting serum as well as 10-15% of the total serum cholesterol.

PROCEDURES

Clinical information covered under "Procedures" is outlined as follows:

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- Periodic screening
- Methods

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LDL cholesterol
HDL cholesterol

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9. PERIODIC EVALUATIONS

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Diagnostic and laboratory evaluations
Inmate education

10. HEALTH CARE PROVIDER RESOURCES AND SELF ASSESSMENT

1. SCREENING

Intake evaluations: Screening for CHD risk factors and elevated LDL cholesterol should be considered in accordance with the following guidelines:

- All newly incarcerated inmates should be screened through the medical history for CHD risk factors and CHD risk equivalents. Inmates should be counseled on the benefits of a healthy diet, aerobic exercise, and reduction in modifiable CHD risk factors.

- Sentenced inmates with established CHD or PAD, any CHD risk equivalent such as diabetes, or hypertension should be screened for elevated LDL cholesterol by obtaining a fasting lipoprotein analysis.

- Sentenced inmates without CHD, PAD, a CHD risk equivalent, or hypertension, should be screened at intake on a case by case basis after review of other CHD risk factors and medical history by obtaining a nonfasting total cholesterol and HDL cholesterol or a fasting lipoprotein analysis. (If the nonfasting total cholesterol is ≥ 200 mg/dL or if the nonfasting HDL cholesterol is ≤ 40 mg/dL, then a follow-up fasting lipoprotein analysis is indicated).

- Screening of unsentenced inmates should be determined on a case by case basis after receiving a medical evaluation.

Periodic screening: A fasting lipoprotein analysis or a nonfasting total cholesterol and HDL cholesterol should be offered during periodic physical examinations that are scheduled in accordance with BOP policy or during routine health care encounters when clinically indicated. (If the nonfasting total cholesterol is ≥ 200 mg/dL or if the nonfasting HDL cholesterol is ≤ 40 mg/dL then a follow-up fasting lipoprotein analysis is indicated).

Methods: Lipid measurements should be obtained in accordance with the following guidelines:

- Total serum cholesterol and HDL cholesterol can be measured at any time of the day in the nonfasting state, since total cholesterol does not change significantly after a fat-containing meal, and HDL-C levels drop minimally. Venipuncture should be performed after 5 minutes in the sitting position, using the tourniquet as briefly as possible, to minimize the effect of plasma volume and posture on cholesterol levels. Recent surgery or trauma, acute infections, weight loss or changes in diet, and

pregnancy can all affect lipid metabolism and cholesterol levels.

- The lipoprotein analysis must be obtained in the **fasting** state (i.e. 9-12 hours without consuming any calories).
- If triglyceride levels are > 400 mg/dL, the LDL cholesterol cannot be accurately estimated from a routine lipoprotein analysis and special laboratory procedures are indicated.

2. CLASSIFICATION OF LIPID VALUES

Total cholesterol, LDL cholesterol, and HDL cholesterol are classified by the NCEP (ATP III) as follows:

Total cholesterol (mg/dL):

- **Desirable** (< 200)
- **Borderline high** (200-239)
- **High** (\geq 240)

LDL cholesterol (mg/dL):

- **Optimal** (< 100)
- **Near or above optimal** (100-129)
- **Borderline high** (130-159)
- **High** (160-189)
- **Very high** (\geq 190)

HDL cholesterol (mg/dL):

- **Low** (< 40)
- **High** (\geq 60)

3. RISK ASSESSMENT

LDL cholesterol results should be evaluated in conjunction with a cardiac risk assessment, since the relative risk of sustaining a new cardiac event helps establish both the target LDL cholesterol level and the appropriate treatment strategy. The risk assessment is based in part on the Framingham scoring system as outlined in **Appendix 1, Framingham Score for Men**, and **Appendix 2, Framingham Score for Women**. The Framingham Score is determined by summing designated points for age, cigarette smoking status, total cholesterol levels, HDL cholesterol levels, and systolic blood pressure. The following categories of risk are defined by NCEP (ATP III):

High risk category: presence of CHD or CHD equivalent (> 20% 10-year risk of coronary event, or as determined by the Framingham Risk Score); (target LDL cholesterol < 100 mg/dL, consider drug therapy if \geq 130 mg/dL)

Moderate risk category (a): multiple (2+) CHD risk factors with a 10-year risk of 10% - 20% (determined by assessing the Framingham Risk Score); (target LDL cholesterol < 130 mg/dL, consider drug therapy if \geq 130 mg/dL)

Moderate risk category (b): multiple (2+) CHD risk factors with a 10-year risk of < 10% (determined by assessing the Framingham Risk Score); (target LDL cholesterol < 130 mg/dL, consider drug therapy if \geq 160 mg/dL)

Low risk category: 1 or fewer CHD risk factors; (target LDL cholesterol < 160 mg/dL, consider drug therapy if \geq 190 mg/dL)

4. BASELINE EVALUATION

Inmates with CHD, or any CHD risk equivalent, or a screening LDL cholesterol of \geq 130 mg/dL with 2 or more CHD risk factors, or a screening LDL cholesterol of \geq 160 mg/dL with 1 or fewer CHD risk factors should be referred for a baseline clinician evaluation that ordinarily includes the following components:

Medical history: The baseline patient interview should focus on the following:

- Assessment and documentation of previously diagnosed CHD, PAD, and their complications
- Assessment and documentation of CHD risk factors, and CHD risk equivalents
- Establishment of risk category
- Review of lipid disorder(s), if previously diagnosed, and dietary and drug treatment history
- Investigation of family history for premature CHD, or severe dyslipidemias that may suggest a genetic disorder
- Assessment of current medications for drugs that may raise LDL cholesterol or lower HDL cholesterol, including progestins, anabolic steroids, corticosteroids, thiazide diuretics, retinoids (e.g. isotretinoin) and HIV protease inhibitors or other antiretroviral therapies
- Review of systems for symptoms of cardiovascular and peripheral vascular disease, and secondary causes of elevated LDL cholesterol, including hypothyroidism, diabetes, nephrotic syndrome, obstructive liver disease, and HIV infection treated with protease inhibitors or other antiretroviral therapies.

- Attention to relevant portions of the social history, including alcohol intake, illicit drug usage (including anabolic steroid usage), and factors that may affect the inmate's ability to understand or participate in treatment recommendations such as educational level, language and cultural barriers, and physical and mental disabilities.

Physical examination: The baseline physical examination should include a focused evaluation for evidence of CHD and PAD, hypertension and associated target organ damage, and secondary causes of lipid disorders. The examination should include the following:

- Vital signs and blood pressure measurement
- Height and weight
- Examination of the neck for carotid bruits, distended veins, and thyroid palpation
- Routine heart and lung examination
- Abdominal examination, including palpation for aortic aneurysm and auscultation for bruits
- Examination of the extremities for diminished or absent arterial pulses, femoral bruits, and edema
- Skin examination for xanthomas, (particularly in the Achilles tendons and extensor tendons of the hands)
- Screening neurologic evaluation

Diagnostic and laboratory evaluations: A baseline electrocardiogram should be obtained for all inmates with CHD or CHD risk equivalents and should be considered for other inmates undergoing evaluation for elevated LDL cholesterol. Diagnostic evaluations for diabetes mellitus, hypothyroidism, liver disease, renal insufficiency, or other co-morbid conditions should be pursued as clinically indicated.

5. TREATMENT - GENERAL STRATEGIES AND GOALS

NCEP (ATP III) LDL cholesterol goals and cut points for treatment interventions are based on the risk assessment and are outlined in **Appendix 3, LDL Cholesterol Goals and Cut Points for Treatment**. The two major treatment strategies for lowering LDL cholesterol are therapeutic lifestyle changes and drug therapy.

6. TREATMENT - THERAPEUTIC LIFESTYLE CHANGES

Therapeutic lifestyle changes include an improved diet, weight reduction in overweight patients, and increased physical activity. Therapeutic lifestyle changes alone can reduce LDL cholesterol to targeted levels and should be the initial treatment strategy for certain patients as outlined in **Appendix 3**.

Dietary guidance: The NCEP (ATP) recommends the following dietary goals for reducing LDL cholesterol:

- Reduce intake of saturated fat to < 7% of calories
- Reduce cholesterol intake to < 200 mg/d

Calculating saturated fat and cholesterol content of meals can be difficult for both the patient and their health care provider. The treating clinician should first determine the inmate's current eating habits to assess overall caloric intake and estimate how commonly foods high in saturated fat and cholesterol are consumed. Inmates should then be given the following general guidelines for consuming a healthier diet:

- Actively select foods that are lower in saturated fat, cholesterol, and calories in accordance with **Appendix 4, Patient Guide to Selecting a Fat/Cholesterol Controlled Diet**.

- Consume carbohydrate calories predominantly from whole grains, fruits, and vegetables (foods high in fiber and complex carbohydrates such as breads and cereals are great substitutes for foods high in fat)

- Increase consumption of dried beans, peas, and legumes

- Limit the consumption of trans fatty acids, the hydrogenated vegetable oil found in commercially prepared food and some hard margarines

Weight reduction: Overweight and obesity have reached epidemic proportions in the United States, increasing in both genders and all population groups. A gain of approximately 10 to 20 pounds results in an increased risk of CHD of 1.6 times for men, and 1.25 times for women. The degree of unhealthy weight gain can be assessed by determining the patient's Body Mass Index, BMI, that defines healthy weight, overweight, and obesity based on height and weight (See the Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity, accessible at www.surgeongeneral.gov/library). Obesity is defined as a BMI of

$\geq 30 \text{ kg/m}^2$ and overweight as $25\text{-}29 \text{ kg/m}^2$ (NOTE: Some persons with a large muscle mass may be inaccurately classified as overweight using this method). Abdominal obesity (waist circumference > 40 inches for men and > 35 inches for women) is specifically associated with the metabolic syndrome.

Losing weight is very difficult for most patients. Health care providers should provide practical advice to patients and regularly reinforce attainable changes in daily dietary habits. Weight control guidance is outlined in **Appendix 5, Weight Control Information for Inmates**.

Increased physical activity: Aerobic exercise and increased physical activity can result in significant weight loss over time and improved cardiovascular capacity. Health care providers should encourage inmates to participate in aerobic recreational activities (unless contraindicated) and to increase their physical activity during their daily routine.

Monitoring: A fasting LDL cholesterol should be obtained 4-6 weeks after initiating dietary and other therapeutic lifestyle changes. If LDL cholesterol goals are not achieved at 6 weeks, further counseling should be provided and the LDL cholesterol reassessed at 12 weeks. Inmates who fail to achieve LDL cholesterol targets after 3 months of a therapeutic diet should be considered for drug therapy on a case by case basis depending on their medical history and CHD risk factors. NOTE: The vast majority of healthy patients with one or fewer risk factors for CHD with elevated LDL cholesterol can effectively be treated with therapeutic lifestyle changes alone.

7. TREATMENT - MEDICATION OPTIONS

Medication treatment options for lipid disorders are enumerated below and summarized in **Appendix 6, Drug Treatment Options for Lipid Disorders**. Drug therapy is indicated for patients who fail to respond to therapeutic lifestyle changes or who have significant CHD risk factors and LDL cholesterol elevations.

Bile acid-binding resins (bile acid sequestrants): Bile acid-binding resins such as cholestyramine and colestipol reduce LDL-C by 15%-30%. These agents are useful for patients with elevated LDL cholesterol with normal triglycerides, who have failed dietary therapy, particularly in young men and premenopausal women. Bile acid-binding resins can also be used in conjunction with HMG-CoA reductase inhibitors for the treatment of persons with severe hypercholesterolemia. These agents should not be used for inmates with elevated triglycerides, particularly if $> 400 \text{ mg/dL}$. Bile acid sequestrants decrease the absorption of

certain drugs and should usually be administered 2 hours before or 4-6 hours after administering other medications. The safety profile of these agents is excellent. Gastrointestinal side effects, such as bloating, nausea, and constipation occur commonly, but usually abate over time. Increasing dietary fiber or giving psyllium often relieves constipation and bloating.

Niacin (nicotinic acid): Niacin is effective in lowering LDL cholesterol (5%-25%), and triglycerides (20%-50%), and raising HDL cholesterol (15%-35%). This class of drugs is particularly effective in treating persons with low HDL cholesterol or both elevated LDL cholesterol and triglyceride levels. Niacin causes flushing, pruritus, gastrointestinal distress, blurred vision, fatigue, hyperuricemia, glucose intolerance, and exacerbations of peptic ulcer disease. Hepatic toxicity can be severe, particularly with the older sustained release niacin preparations. Nicotinic acid should be used with caution in persons with active liver disease, recent peptic ulcer, gout, and type 2 diabetes. Therapy should be initiated with low doses of crystalline nicotinic acid rather than sustained release preparations and gradually titrated with increasing doses as tolerated until dosage goals are achieved. Pretreatment with 325 mg of aspirin or 200 mg of ibuprofen can minimize cutaneous reactions that usually abate with continued treatment.

HMG-CoA reductase inhibitors (STATINS): HMG-CoA reductase inhibitors inhibit HMG-CoA reductase, the enzyme that catalyzes the rate limiting step in cholesterol synthesis. They reduce, but do not completely block cholesterol biosynthesis when given in therapeutic doses. Treatment with these agents decreases LDL cholesterol by (18%-55%) and decreases triglycerides by (7%-30%) and increases HDL cholesterol by (5%-15%). LDL cholesterol reductions are dose dependent. Single daily doses of any of these agents should be administered in the evening for maximal efficacy. Formulations of "statins" differ in their dosages and quantitative effects on lipids although all are very effective in lowering LDL cholesterol. The "statins" are compared in **Appendix 7, Comparison of HMG-CoA Reductase Inhibitors.**

HMG-CoA reductase inhibitors are well tolerated. Gastrointestinal side effects such as dyspepsia, flatus, and constipation are usually mild in severity and abate with time. Elevated hepatic transaminases (alanine aminotransferase, ALT, or aspartate aminotransferase, AST) occur in 1-2% of treated individuals when high doses are used, but elevations have also occurred at low dosages. Therapy should ordinarily be discontinued if transaminase levels increase to three times the upper limit of normal. The decision to either restart the same medication if transaminase levels normalize, try another HMG-CoA reductase

inhibitor, or try another lipid-lowering agent, should be made on a case by case basis.

A dose-related myopathy, associated with myalgias and marked elevations in creatine kinase levels is another potential toxicity associated with HMG-CoA reductase inhibitors. Rhabdomyolysis with renal failure occurs rarely. Routine screening of creatine kinase is not necessary, but inmates should be advised to report muscle pain, dark urine, or weakness.

Serum concentrations of HMG-CoA reductase inhibitors are significantly increased with concurrent administration of drugs similarly metabolized in the liver, including cyclosporine, gemfibrozil, itraconazole, ketoconazole, erythromycin, clarithromycin, nefazodone, or grapefruit juice. HMG-CoA reductase inhibitors can also potentiate the effect of oral anticoagulants, therefore inmates concurrently prescribed coumarin medications should have their prothrombin time monitored closely.

Fibric acid derivatives: Fibric acid containing medications such as gemfibrozil, fenofibrate, and clofibrate decrease the synthesis of VLDL triglycerides, and thus are primarily indicated for treating hypertriglyceridemia. These agents decrease triglycerides by (20%-50%), modestly increase HDL cholesterol by (10%-20%) and decrease LDL cholesterol by (5%-20%). These agents should not be used as first line treatment for hypercholesterolemia in persons with CHD because of their limited efficacy. Fibric acid derivatives are contraindicated in patients with severe renal or hepatic disease.

Fibric acid derivatives are generally well tolerated. Gastrointestinal side effects are the most common patient complaints. Gallstones are a well described complication. These agents potentiate the effects of oral anticoagulants and oral hypoglycemic agents.

Combination therapy (fibric acid derivatives plus HMG-CoA reductase inhibitors) is associated with a small but real risk of myopathy and rhabdomyolysis. Patients on combination therapy should be carefully monitored, ensuring that the inmate has normal renal function, that there are no drug interactions that could increase the blood levels of either drug, that creatine kinase levels are monitored at baseline and with symptoms, and that the inmate is counseled on the symptoms of myopathy. The long term use of fibric acids should generally be avoided since there is an ill defined, but potential risk of increased mortality and malignancy associated with these agents.

8. TREATMENT - MEDICATION STRATEGIES AND SPECIAL CONSIDERATIONS

Hypercholesterolemia (elevated LDL cholesterol): Lowering elevated LDL cholesterol to targeted levels is normally the primary goal of lipid-lowering therapy even when other abnormalities are present such as elevated triglycerides and depressed HDL cholesterol. The LDL threshold for initiating drug therapy is determined by the patient's risk assessment (**Appendix 3**). Patients with a history of CHD, PAD, or CHD equivalents such as diabetes mellitus should be aggressively treated with drug therapy if LDL is elevated above targeted goals. Patients with 1 or fewer CHD risk factors can ordinarily be managed with therapeutic lifestyle changes alone.

Drug therapy should be initiated with a single agent, either a (1) bile acid sequestrant, or a (2) HMG-CoA reductase inhibitor, or (3) nicotinic acid. Drug selection should be individualized based on the patient's medical history, potential drug interactions, and other clinical concerns. If LDL cholesterol does not decrease to the targeted goal after 6-12 weeks of therapy, therapy should be intensified with either (1) a higher dose of an HMG-CoA reductase inhibitor, or (2) addition of a bile acid sequestrant, or (3) addition of nicotinic acid. If intensified therapy is unsuccessful, then adherence to the treatment plan should be carefully reassessed and medications further intensified or altered in consultation with a physician specialist as necessary.

NOTE: Patients with baseline LDL cholesterol ≥ 190 mg/dL may have a hereditary lipid disorder or other cofactor affecting lipid metabolism. Such patients usually require combination therapy for adequate lipid control with referral to a specialist as necessary.

Once the targeted LDL cholesterol is achieved, LDL cholesterol levels should be monitored approximately every 6 months to determine if the treatment plan remains effective.

Metabolic syndrome: The risk factors associated with metabolic syndrome increase the risk of CHD at any given LDL cholesterol level. Patients with metabolic syndrome should be targeted for aggressive medical management. Weight reduction efforts should be consistently reinforced along with increased physical activity. Hypertension should be effectively treated. Elevated LDL cholesterol levels, hypertriglyceridemia, and decreased HDL cholesterol levels should be targeted for treatment.

Low HDL cholesterol: Low HDL cholesterol (< 40 mg/dL) is an independent risk factor for CHD. Current drug therapies do not significantly increase HDL cholesterol. Elevated LDL levels should be the primary goal of therapy; whereas increasing low HDL cholesterol levels is a secondary goal. Modest increases in HDL

cholesterol may be achieved by improving dietary habits, smoking cessation, and increased physical activity.

Hypertriglyceridemia: Elevated triglycerides may be an independent risk factor for CHD and are most commonly associated with metabolic syndrome. Triglycerides are classified as follows:

- **Normal:** < 150 mg/dL
- **Borderline high:** 150-199 mg/dL
- **High:** 200-499 mg/dL
- **Very high:** ≥ 500 mg/dL

The treatment strategy for hypertriglyceridemias depends on the etiology and the severity of the lipid disorder. Persons with very high triglyceride levels (≥ 500 mg/dL) are at increased risk of pancreatitis. If weight reduction, diabetic control, and the discontinuation of drugs that aggravate hypertriglyceridemia do not adequately lower very high triglyceride levels, then drug therapy with nicotinic acid or fibrates should be considered.

Patients with borderline high or high triglycerides should be managed with therapeutic lifestyle changes and should be considered for drug therapy on a case by case basis. Drug therapy should first target other associated lipid abnormalities such as elevated LDL cholesterol and depressed HDL cholesterol.

Diabetes mellitus: Diabetes is a CHD equivalent. Lowering elevated LDL cholesterol levels to < 100 mg/dL should be a priority for all diabetic patients. Drug therapy is usually required. Patients with diabetes frequently have low HDL cholesterol and elevated triglycerides which are secondary targets for treatment. **Maximizing control of elevated blood glucose is integral to the effective management of lipid disorders associated with diabetes.**

Gender considerations: Women with CHD or CHD equivalents should be treated similar to men when initiating drug therapy for elevated LDL cholesterol. Young healthy women with moderate elevations in LDL cholesterol may warrant a more conservative approach to initiating drug therapy for elevated LDL cholesterol, since CHD in women is delayed by 10 to 15 years, when compared to men.

Human immunodeficiency virus (HIV) infection: Persons with HIV infection may have significant changes in lipid metabolism. Hypertriglyceridemia can occur from increased VLDL production and

reduced triglyceride clearance. HDL cholesterol may decline to levels that increase CHD risk. Treatment with HIV protease inhibitors and other antiretroviral medications have been associated with further increases in triglycerides as well as hypercholesterolemia from increases in VLDL and LDL cholesterol. Changes in lipid metabolism may be associated with changes in body habitus and syndromes of fat redistribution such as fat loss or lipodystrophy, and fat accumulation such as increased abdominal girth, "buffalo hump," bilateral lipomatosis, and breast enlargement. These changes may or may not be unique to a specific antiretroviral medication or regimen.

Treatment of elevated LDL cholesterol and very high triglycerides (> 500 mg/dL) should be considered in persons with HIV infection, particularly if CHD, PAD, diabetes, or multiple CHD risk factors are present. Drug therapy is complicated by potential interactions with HIV medications and the frequent requirement for high dose or dual drug therapy. Treatment should be prescribed in consultation with a physician with experience in treating these complicated medical conditions. (Pravastatin is the only HMG-CoA reductase inhibitor that can be co-administered with most protease inhibitors).

9. PERIODIC EVALUATIONS

Periodic medical evaluations should be conducted as clinically indicated and in accordance with BOP policy for inmates with elevated LDL cholesterol.

Medical history: The periodic patient interview should focus on the following:

- Review of progress in modifying CHD risk factors
- Assessment of adherence to dietary therapy
- Assessment of adherence to drug therapy and presence of drug side effects (consult with the pharmacist to review adherence to the prescribed medication regimen)

Physical examination: The periodic examination should target the following:

- Measurement of vital signs, including blood pressure
- Examination of the heart, lungs, pulses, and extremities with auscultation over the carotid and femoral arteries for bruits
- Palpation and auscultation of the abdomen for evidence of an

aortic aneurysm

- Examination of the skin for xanthomas

Diagnostic and laboratory evaluations: The LDL cholesterol should be measured 4-6 weeks after beginning medication and again at 12 weeks. If LDL cholesterol goals are met, total cholesterol or lipoprotein analysis should be measured periodically during clinician evaluations. More frequent monitoring is indicated for inmates with poorly controlled hyperlipidemia, particularly when associated with underlying CHD and PAD. Drug side effects should be monitored by patient history and with laboratory evaluations as clinically indicated.

The management of inmates with high blood cholesterol requires a multidisciplinary effort of the entire health services staff. Pharmacists and nurses can assist clinicians by providing inmates information on diet modifications and medication use as appropriate and by monitoring inmate adherence to recommended treatments and adverse drug reactions. Pharmacists can order and review laboratory work and write treatment recommendations for the management of lipid disorders when privileged by the Clinical Director.

Inmate education: All inmates elevated blood cholesterol should receive education from a health care provider at the time of diagnosis and periodically during clinician evaluations and interactions with pharmacy and nursing staff. Inmates should be counseled on the risks of elevated cholesterol, the importance of modifying CHD risk factors, specific treatment recommendations and drug side effects. Inmates with CHD or severe or poorly controlled lipid disorders require more intensive personal or group educational efforts. Educational materials are attached in **Appendix 4, Guide to Selecting a Fat/Cholesterol Controlled Diet, Appendix 5, Weight Control Information for Inmates, Appendix 8, Patient Education (High Cholesterol), and Appendix 9, Inmate Fact Sheet (High Cholesterol).**

10. HEALTH CARE PROVIDER RESOURCES AND SELF-ASSESSMENT

Provider resources for managing lipid disorders are listed in **Appendix 10, Resources (Management of High Cholesterol).** An educational tool for health care providers is attached in **Appendix 11, Provider Self-Assessment (Management of High Cholesterol).**

ATTACHMENTS

Appendix 1: Framingham Score for Men

Appendix 2:	Framingham Score for Women
Appendix 3:	LDL Cholesterol Goals and Cut Points for Treatment
Appendix 4:	Patient Guide to Selecting a Fat/Cholesterol Controlled Diet
Appendix 5:	Weight Control Information for Inmates
Appendix 6:	Drug Treatment Options for Lipid Disorders
Appendix 7:	Comparison of HMG-CoA Reductase Inhibitors ("Statins")
Appendix 8:	Patient Education (Management of High Cholesterol)
Appendix 9:	Inmate Fact Sheet (High Cholesterol)
Appendix 10:	Resources (Management of High Cholesterol)
Appendix 11:	Provider Self-Assessment (Management of High Cholesterol)

FRAMINGHAM SCORE FOR MEN **(ESTIMATING 10-YEAR RISK FOR CHD)**

Age, y	Points
20-34	-9
35-39	-4
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	11
70-74	12
75-79	13

	Points				
	Age 20-39 y	Age 40-49 y	Age 50-59 y	Age 60-69 y	Age 70-79 y
Nonsmoker	0	0	0	0	0
Smoker	8	5	3	1	1

HDL, mg/dL	Points
≥60	-1
50-59	0
40-49	1
<40	2

Total Cholesterol, mg/dL	Points				
	Age 20-39 y	Age 40-49 y	Age 50-59 y	Age 60-69 y	Age 70-79 y
<160	0	0	0	0	0
160-199	4	3	2	1	0
200-239	7	5	3	1	0
240-279	9	6	4	2	1
≥280	11	8	5	3	1

Systolic BP, mm Hg	If Untreated	If Treated
<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥160	2	3

Point Total	10-Year Risk, %
<0	<1
0	1
1	1
2	1
3	1
4	1
5	2
6	2
7	3
8	4
9	5
10	6
11	8
12	10
13	12
14	16
15	20
16	25
≥17	≥30

FRAMINGHAM SCORE FOR WOMEN (ESTIMATING 10-YEAR RISK FOR CHD)

Appendix 2

Age, y	Points
20-34	-7
35-39	-3
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	12
70-74	14
75-79	16

Total Cholesterol, mg/dL	Points				
	Age 20-39 y	Age 40-49 y	Age 50-59 y	Age 60-69 y	Age 70-79 y
<160	0	0	0	0	0
160-199	4	3	2	1	1
200-239	8	6	4	2	1
240-279	11	8	5	3	2
≥280	13	10	7	4	2

Systolic BP, mm Hg	If Untreated	If Treated
<120	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥160	4	6

	Points				
	Age 20-39 y	Age 40-49 y	Age 50-59 y	Age 60-69 y	Age 70-79 y
Nonsmoker	0	0	0	0	0
Smoker	9	7	4	2	1

HDL, mg/dL	Points
≥60	-1
50-59	0
40-49	1
<40	2

Point Total	10-Year Risk, %
<9	<1
9	1
10	1
11	1
12	1
13	2
14	2
15	3
16	4
17	5
18	6
19	8
20	11
21	14
22	17
23	22
24	27
≥25	≥30

LDL CHOLESTEROL GOALS AND CUT POINTS FOR TREATMENT*

Risk Category	LDL Goal (mg/dL)	LDL Level for Therapeutic Changes (mg/dL)	Lifestyle	LDL Level at Which to Consider Drug Therapy (mg/dL)
High Risk CHD or CHD risk equivalents (10-year risk >20%)	<100	≥100		≥130 (100-129: drug optional)**
Moderate Risk (a) 2+ Risk factors (10-year risk 10- 20%)	<130	≥130		≥130
Moderate Risk (b) 2+ Risk factors (10-year risk < 10%)	<130	≥130		≥160
Low Risk 0-1 Risk factor	<160	≥160		≥190 (160-189: drug optional)

*Adapted from NCEP (ATP III), National Institutes of Health, 2001

**Some experts recommend use of LDL-lowering drugs in this category if an LDL cholesterol level of < 100 mg/dL cannot be attained by therapeutic lifestyle changes. Other experts prefer medications that primarily modify triglycerides and HDL cholesterol such as nicotinic acid and fibrate. Deferring drug treatment is also acceptable on a case by case basis.

PATIENT GUIDE TO SELECTING A FAT/CHOLESTEROL CONTROLLED DIET

	Choose	Go Easy On	Decrease
Meat, Poultry and Fish (up to 6 oz per day)	<ul style="list-style-type: none"> - Lean cuts of meat with fat trimmed - Baked unbreaded poultry without skin - Baked, unbreaded fish - Canned chicken, tuna, or sardines (water packed or rinsed) - Dried beans and peas as a meat substitute 		<ul style="list-style-type: none"> - Fried meat - Breaded meat - Organ meats, like liver - Sausage - Bacon - Lunch meats - Hot dogs - Fatty cuts of meat brisket, ribs
Eggs (no more than 4 egg yolks per week)	<ul style="list-style-type: none"> - Egg whites - Cholesterol-free egg substitutes 		<ul style="list-style-type: none"> - Egg yolks
Dairy Products (at least 2 servings per day)	<ul style="list-style-type: none"> - Skim milk, 1% milk, low fat buttermilk, or nonfat powdered milk - Low-fat yogurt (plain and frozen) - Low-fat cottage cheese 	<ul style="list-style-type: none"> - 2% milk - Yogurt - Part-skim cheeses like mozzarella, or string cheese 	<ul style="list-style-type: none"> - Whole milk cream, half-and-half, most nondairy creamers and products, real or nondairy whipped cream - Cream cheese - Sour cream - High-fat cheeses like Swiss, Cheddar, American
Fats and Oils (up to 6 teaspoonfuls per day)	<ul style="list-style-type: none"> - Low fat dressings 	<ul style="list-style-type: none"> - Unsaturated vegetable oils: olive, peanut, canola, safflower, soybean - Margarine - Nuts/seeds - Peanut butter - Olives - Avocados - Mayonnaise - salad dressings 	<ul style="list-style-type: none"> - Butter, lard, bacon fat - Coconut oil - Palm oil - Palm kernel oil - Bacon - Hydrogenated fat or oil
Fruits and Vegetables (2-4 servings of fruit and 3-5 servings of vegetables per day)	<ul style="list-style-type: none"> - Fresh, frozen, canned, or dried fruits and vegetables 		<ul style="list-style-type: none"> - Vegetables prepared in butter, cream, or sauce - Fried vegetables

	Choose	Go Easy On	Decrease
Breads, Pasta, Cereals, Rice, Dried Beans, and Peas (6 to 11 servings per day)	<ul style="list-style-type: none"> - Breads, like white, whole wheat, rye, pita, pumpernickel - Bagels, English muffins, sandwich buns, rice cakes - Low-fat crackers, like matzo, bread sticks, rye krisp, saltines, zwieback - Rice, pasta, dried beans and peas prepared without fat 	<ul style="list-style-type: none"> - Pancakes - Waffles - Biscuits - Cornbread 	<ul style="list-style-type: none"> - Croissants, butter rolls, sweet rolls, Danish pastry, doughnuts - Cheese or butter crackers - Granola-type cereals - Pasta and rice prepared with cream, butter, or cheese sauce
Sweets and Snacks (avoid too many sweets)	<ul style="list-style-type: none"> - Fat-free desserts, like sherbet, Italian ice, frozen yogurt, popsicles - Fat-free cakes, like angel food cake - Fat-free candy, like jelly beans and hard candy - Very low-fat snacks, like popcorn, pretzels - Non-fat beverages, like carbonated drinks, juices, tea, coffee 	<ul style="list-style-type: none"> - Low-fat frozen desserts, like ice milk - Low-fat cookies, like fig bars, ginger snaps, animal crackers, graham crackers 	<ul style="list-style-type: none"> - High-fat frozen desserts, like ice cream - High-fat cakes - pound cake and frosted cakes - Pastries and cookies - Most candy, like chocolate bars and candies that contain chocolate - Potato chips, corn chips, and other snack chips - Buttered popcorn - High-fat beverages like milkshakes and egg nog

Label Ingredients

To avoid too much fat or saturated fat, read the ingredient labels and go easy on products that list any fat or oil first, or that list many fat and oil ingredients. (Ingredients are listed in order of how much is in the product. For example, if lard or coconut oil is listed as one of the first three ingredients, that food product has a very high fat content).

WEIGHT CONTROL INFORMATION FOR INMATES

- Cutting down on calorie intake (or eating less food) is the first step to losing weight. One pound of body fat is equal to 3,500 calories. A person must reduce calorie intake 500 calories per day to lose one pound in a week.
- Write down what you eat each day. This record will help you identify the amount of calories you are eating and potential “problem foods.”
- Note your pattern of eating and the time of day you are likely to overeat. Try and maintain a regular eating pattern. Avoid skipping meals.
- Ask the server to give you small portions. Leave off the gravy and high fat sauces.
- Avoid sweetened beverages such as lemonade, koolade, punch, and soft drinks. Fruit juices, although they contain vitamins, should be limited also. Diet drinks are an alternative choice.
- Limit the number of desserts on your tray. Cakes, pies, ice cream, and cookies are concentrated sources of calories. If you don’t put them on your tray, you won’t eat them. Consider sugar substitutes to sweeten food.
- Remove the breading/skin from fried meats. Most of the fat is found in the skin or absorbed in the outer breaded layer of fried foods. Avoid fried foods such as onion rings and fried potatoes.
- Try foods without adding butter, margarine, cream, or sugar.
- Don’t add creamy salad dressings to your salad (1 tablespoon of mayonnaise type salad dressing = 100 calories).
- Drink water with meals and between meals. Drink you tea or coffee black.
- Eat slowly. Eat your salad first.
- Learn to stop eating before you are “full” or “stuffed.” The slight hunger you feel will disappear about one-half hour after mealtime.
- Minimize idle time through recreational and work activities. Establish a regular schedule for exercise as much as possible so it becomes routine.
- Restrict your commissary items. What you don’t buy, you can’t eat. Avoid buying concentrated sweets, high fat crackers, cookies, and snack items.
- **NOTE:** If you eat just 100 extra calories a day, you will gain 10 pounds in the course of a year. If you eat just 100 fewer calories a day, you will lose 10 pounds in the course of a year. Small changes in your daily eating habits make a big difference!

DRUG TREATMENT OPTIONS FOR LIPID DISORDERS

	Medication	Dosage	Labs	Toxicities	Comments
HMG CoA - Reductase Inhibitors (“Statins”)	lovastatin (Mevacor™)	20-80 mg/day	ALT/AST (base-line & q6w x 2, repeat p dose ↑ & q6m)	rhabdomyolysis hepatotoxicity	- contraindicated in active liver disease, pregnancy, unexplained elevated LFT's - ↑ risk rhabdomyolysis when administered w/ fibrates or niacin - lower dose if creatinine clearance \leq 30 L/min
	simvastatin (Zocor™)	5-80 mg/day	ALT/AST (base-line and q6m)	rhabdomyolysis hepatotoxicity	- ↑ risk rhabdomyolysis when administered w/ fibrates or niacin - lower dose if renal insufficiency is severe
	fluvastatin (Lescol™)	20-80 mg/day	ALT/AST (base-line & @ 12 weeks, repeat p dose ↑, & q6m)	rhabdomyolysis hepatotoxicity pancreatitis hypersensitivity	- ↑ risk rhabdomyolysis when administered w/ fibrates or niacin - no adjustment for renal insufficiency
	pravastatin (Pravachol™)	10-40 mg/day	ALT/AST (base-line and dose ↑, & q6m)	rhabdomyolysis hepatotoxicity	- ↑ risk rhabdomyolysis when administered w/ fibrates or niacin - lower dose if creatinine clearance is \leq 60 L/min
	atorvastatin (Lipitor™)	10-80 mg/day	ALT/AST (base-line & @ 12 weeks, repeat p dose ↑, & q6m)	rhabdomyolysis hepatotoxicity	- ↑ risk rhabdomyolysis when administered w/ fibrates or niacin - no adjustment for renal insufficiency
	<p>Only one “Statin” at a time should be used, and titrated to the target LDL, side effects, or maximum dose before switching statins.</p> <p><u>Statin Drug Interactions:</u> cyclosporine, itraconazole, ketoconazole, gemfibrozil, niacin, erythromycin, clarithromycin, verapamil, diltiazem, nefazodone, fluvoxamine, and protease inhibitors (except pravastatin).</p>				

	Medication	Dosage	Labs	Toxicities	Comments
Bile Acid Sequestrants	cholestyramine (LoCholest™, Questran™, Prevalite™)	8-24 gm/day	LDL cholesterol and TG levels	fecal impaction	<ul style="list-style-type: none"> - dosed once to six times daily - take before meals - do not consume dry powder - may cause constipation - may prevent absorption of folic acid and fat soluble vitamins (A-D-E-K)
	colestipol (Colestid™)	10-30 gm/day	LDL cholesterol and TG levels	fecal impaction GI bleed	<ul style="list-style-type: none"> - dosed once or twice daily - do not consume dry powder - do not crush, cut, or chew - may cause constipation - may prevent absorption of folic acid and fat soluble vitamins (A-D-E-K)
	colesevelam (Welchol™)	2.5-4.375 gm/day	LDL cholesterol and TG levels	none reported	<ul style="list-style-type: none"> - take with water and meals - dosed once or twice daily - monotherapy or combination with HMG-CoARIs - may cause constipation - may prevent absorption of folic acid and fat soluble vitamins (A-D-E-K)
Niacin	niacin	1.5-6 gm/day	ALT/AST (baseline, q6-12w x 1 year, then q6m) Uric acid/fasting glucose - baseline, at 6 wks/annually	arrhythmias hepatotoxicity peptic ulcer fulminant hepatic necrosis	<ul style="list-style-type: none"> - contraindicated in active peptic ulcer, alcoholism, unexplained ↑ LFT's, severe liver dysfunction - use with caution w/ hx of PUD, DM, gout, ↓ renal fx - ASA ½ hour before administration to ↓ flushing - take with meals

	Medication	Dosage	Labs	Toxicities	Comments
Fibric Acids	clofibrate (Atromid-S™)	2000 mg/day	monitor: - serum lipids - CBC - LFT's	anemia leukopenia hepatotoxicity cholelithiasis pancreatitis	- possible myalgia, myositis, myopathy, and rhabdomyolysis (w or w/o ↑ CPK) - ↑ risk rhabdomyolysis when administered w/ HMG CoARIs - contraindicated in significant hepatic or renal dysfunction, primary biliary cirrhosis, pregnancy, lactation - uncertain risk of malignancy -reserve for inmates refractory to other tx strategies
	gemfibrozil (Lopid™)	1200 mg/day	monitor: - serum lipids - CBC - LFT's - blood glucose	myositis myopathy thrombocytopenia rhabdomyolysis hepatotoxicity pancreatitis cholelithiasis hypersensitivity cholestatic jaundice	- ↑ risk rhabdomyolysis when administered w/ HMG CoARIs contraindicated in hepatic or severe renal dysfunction, primary biliary cirrhosis, preexisting gallbladder disease - take ½ hour before morning & evening meals
	fenofibrate (Tricor™)	200 mg/day	monitor: - serum lipids - CBC	pancreatitis cholelithiasis rhabdomyolysis hepatotoxicity hypersensitivity myopathy toxic epidermal necrolysis	- ↑ risk rhabdomyolysis when administered w/ HMG CoARIs - contraindicated in hepatic or severe renal dysfunction, primary biliary cirrhosis, unexplained persistent liver function abnormality; preexisting gallbladder disease - take with meals

COMPARISON OF HMG-CoA INHIBITORS (STATINS)

Drug	Equiv. Dose	Effect on Lipids (% change from baseline)					Drug Interactions
Atorvastatin <i>Lipitor</i>	10 mg	<u>LDL</u>	<u>TC</u>	<u>TG</u>	<u>HDL</u>	- interacts with drugs metabolized by CYP3A4 enzyme system*	
		10 mg	-34-39	-27-29	-13-19		+4-6
		20 mg	-41-46	-32-35	-20-26		+5-9
		40 mg	-48-51	-37-39	-29-32		+5-6
		80 mg	-54-60	-42-45	-25-37		+5
Fluvastatin <i>Lescol</i>	40 mg	20 mg	-17-22	-13	-5	+1	- metabolized by CYP2C9 - can increase cyclosporine and phenytoin levels
		40 mg	-23-27	-18-22	-10-20	+4-8	
		80 mg	-33-36	-27	-15-25	+4-8	
Lovastatin <i>Mevacor</i>	20 mg	20 mg	-25-29	-18-22	-12-13	+6-8	- interacts with drugs metabolized by CYP3A4 enzyme system*
		40 mg	-31-34	-23-27	-2-10	+5	
		80 mg	-41-48	-32-36	-13-15	+4-8	
Pravastatin <i>Pravachol</i>	20 mg	10 mg	-19-22	-13-16	-3-15	+7-10	- not metabolized by cytochrome P450, less likely to have drug interactions
		20 mg	-24-32	-18-24	-11-15	+2-3	
		40 mg	-33-34	-24-27	-10-24	+6-12	
Simvastatin <i>Zocor</i>	10 mg	10 mg	-28-30	-21-23	-12-15	+7-12	- metabolized by CYP3A4 enzyme system*
		20 mg	-35-38	-26-28	-15-17	+5-8	
		40 mg	-40-41	-30-31	-15-18	+9-10	
		80 mg	-47-48	-36	-24	+8	

*Erythromycin, clarithromycin, azole antifungals, verapamil, diltiazem, nefazodone, fluvoxamine, cyclosporine, protease inhibitors. (Adapted from *Pharmacist's Letter*, January 2002, *Pharmacotherapy* 2000;20(7):819-822, *Circulation* 2000;101:207-213)

PATIENT EDUCATION

(Management of High Cholesterol)

Objectives

- Describe how high cholesterol affects blood vessels
- Understand why high cholesterol levels should be controlled
- List actions to lower cholesterol levels and thereby reduce the risk of heart disease

Disease Description

Cholesterol is a waxy, fat-like substance present in every cell of the body. The body needs some cholesterol, but high levels of cholesterol in the blood increases the risk of developing coronary heart disease, the most common form of heart disease. Cholesterol build-up in arteries can happen so slowly that people are not even aware of it. Cholesterol plaques or blockages in the blood vessels slow down blood flow to the heart and other vital organs. When arteries become clogged with fat and cholesterol the heart must work harder to force the blood through those vessels to provide oxygen and nutrients to the tissues - most importantly the heart and brain. When not enough oxygen-filled blood reaches the heart, there may be chest pain (angina). Chest pain protects the heart by stopping whatever activity is causing the heart to work too hard. For some people, that may take barely any activity at all. If the blood supply is completely cut off to the blood vessels of the heart, the result is a heart attack. Heart disease is the number one killer of men and women in the United States.

Regardless of whether or not there is heart disease, lowering cholesterol levels NOW will decrease the risk of a heart attack and could prolong life. Cholesterol levels can be lowered with proper diet and medications.

Types of cholesterol

Cholesterol is present in the body in two primary forms: LDL and HDL.

- **LDL = low density lipoprotein**, is known as the "bad" cholesterol. LDL makes up the majority of the cholesterol in the blood and is also the type of cholesterol that can build up and block arteries. The higher the LDL level, the higher the risk for heart disease. Lowering LDL cholesterol can help prevent a heart attack. The LDL cholesterol is the target for treatment. The LDL cholesterol target is < 100 mg/dL for patients with heart disease and diabetes. The target LDL cholesterol for healthy persons is determined by their health care provider after assessing risk factors for heart disease and the patient's medical history.

- **HDL = high density lipoprotein**, is known as the "good" cholesterol because it helps remove the bad cholesterol from the blood. High levels of HDL can help reduce the chance of a heart attack. If HDL cholesterol blood levels are less than 40 mg/dL, there is a higher risk of developing heart disease.

- **Total cholesterol** is the cumulative amount of cholesterol carried in the blood. A desirable blood cholesterol is < 200 mg/dL.

- **Triglycerides** are a storage form of fat, so high levels are not normally in the blood. Elevated triglyceride levels are associated with heart disease and diabetes. A normal triglyceride level is < 200 mg/dL.

Treatment - lifestyle changes

The first approach to treating high cholesterol is changing certain habits where appropriate. Many people are able to control cholesterol levels just by changing diet, losing weight, and participating in an exercise program. Often drugs are not needed. In fact, physicians may not prescribe medication if there are no risk factors for heart disease other than mildly elevated cholesterol. Also, if there is another disease present that raises cholesterol, that disease needs to be addressed in order to decrease cholesterol levels. Diseases that may aggravate high cholesterol include diabetes, high blood pressure and hypothyroidism. Appropriate action steps include:

- **Quit smoking:** Smoking causes lung cancer, but is also strongly linked to heart disease. The risk of a heart attack is at least seven times greater for a smoker than for a non-smoker. Nicotine increases the blood pressure and causes blood vessels to constrict, or tighten up even further. This constriction is thought to cause microscopic tears in the lining of the arteries, which allows cholesterol to stick in these cracks more easily. Working hard to lower cholesterol without quitting smoking is doing things in the wrong order. Smoking is thought to cause the damage to blood vessels which allows cholesterol to stick to their walls.

- **Improve your diet:** Follow recommendations for a low fat/low cholesterol diet

- **Carefully manage your diabetes:** Out of control blood sugar can cause an increase in triglycerides and cholesterol levels.

- **Lose weight, if overweight:** Overweight people tend to have higher cholesterol levels. Any weight loss, even 5-10 pounds, can help improve cholesterol levels. "Perfect" weight is not required to see a change in blood cholesterol levels. Watch the diet, especially fat intake and total calories for the day. Lose weight slowly (about ½ to 1 pound a week), rather than going on a drastic, starvation diet.

- **Exercise as permitted by your physician:** Inactive people are two times more likely to

develop heart disease than a physically active person. Increasing the level of activity can improve cholesterol levels by increasing the good cholesterol level and decreasing the bad cholesterol level. Exercise can help cause weight loss, lower blood pressure, reduce stress, and improve the fitness of the heart and blood vessels. All of these help lower the risk of heart disease. Participate in aerobic activity for 30 minutes at a time for three or more times a week whenever possible. Begin exercise gradually, but be persistent.

Treatment - medications

Medications are prescribed to treat high cholesterol if lifestyle changes are not completely effective. Several types of medications are available to treat high cholesterol.

- **"Statins"** (HMG CoA reductase inhibitors) act to lower LDL cholesterol by slowing down production of cholesterol in the liver and helping the body get rid of cholesterol. Potential side effects include muscle pain and liver inflammation.
- **Bile acid resins** lower LDL cholesterol by combining with bile acids in the gut. These drugs often cause constipation, therefore they need to be taken with plenty of water.
- **Niacin** is a vitamin that decreases fat production by the liver and lowers total cholesterol, LDL cholesterol, and triglycerides. The dose should be increased slowly to minimize side effects, which may include skin flushing and an upset stomach. These problems can often be avoided by taking niacin with food, or by taking an aspirin one hour before taking niacin.
- **Fibrates** lower triglyceride levels and raise HDL, but have little effect on lowering LDL cholesterol. They are taken twice a day before meals.

Knowing your medications and possible side effects is an important part of managing your high cholesterol.

Summary

Follow these important recommendations:

- Quit smoking. Smoking is an even worse risk factor than high cholesterol as a cause of heart disease and stroke.
- Work at keeping cholesterol levels down. Once cholesterol levels are brought under control, do not return to old patterns of poor eating habits and inactivity.
- Take your medications as directed and monitor your cholesterol levels along with your health care provider. Understand your risk factors for heart disease, take action, and play an active role in staying healthy.

INMATE FACT SHEET

(High Cholesterol)

- Anyone can have high cholesterol, regardless of gender, race, age or ethnic background.
- The higher your blood cholesterol, the greater your risk of heart disease. By lowering your cholesterol you can live longer.
- Overeating foods with high cholesterol and saturated fat can contribute to high cholesterol.
- Your total cholesterol level should be less than 200 mg/dL.
- LDL-cholesterol is "bad" cholesterol and can build up in your arteries, increasing your chance of heart disease. If you have heart disease or diabetes your LDL cholesterol should ideally be less than 100 mg/dL.
- HDL-cholesterol is "good" cholesterol and can prevent cholesterol build up in your arteries. HDL-cholesterol less than 40 mg/dL may increase your risk of heart disease.
- There are two ways to treat high cholesterol:
 - 1) Improve your lifestyle by dieting, exercising, quitting smoking, and controlling your high blood pressure or diabetes
 - 2) Take medications along with diet and exercise
- Exercise helps increase your HDL-cholesterol and decrease your LDL-cholesterol
- There is no cure for high cholesterol so controlling your cholesterol will be a lifelong process.

RESOURCES

(Management of High Blood Cholesterol)

National Institutes of Health

Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults

Third Report of the Expert Panel, NIH, 2001

National Heart, Lung, and Blood Institute Information Center

www.nhlbi.nih.gov/nhlbi/nhlbi.htm

American College of Cardiology

800 253-4636

www.acc.org

American Heart Association

800 242-8721

www.amhrt.org

**PROVIDER SELF-ASSESSMENT
(Management of High Cholesterol)**

1. Which of the following are major CHD risk factors that modify LDL cholesterol goals?
 - A. Cigarette smoking
 - B. Male age 45 years or greater
 - C. Female age 55 years or greater
 - D. Blood pressure of 140/90 mm Hg or greater
 - E. Family history of premature CHD
 - F. All of the above
2. A CHD risk equivalent is a factor or group of factors that confers a risk of a CHD event of 20% or greater during the next 10 years. True or False?
3. Which of the following lipid values represents a potential health risk for the patient?
 - A. LDL cholesterol of 90 mg/dL
 - B. HDL cholesterol of 79 mg/dL
 - C. HDL cholesterol of 30 mg/dL
 - D. Total cholesterol of 185 mg/dL
4. The target LDL cholesterol for a patient with known CHD or a CHD equivalent such as diabetes is which of the following?
 - A. < 70 mg/dL
 - B. < 100 mg/dL
 - C. < 130 mg/dL
 - D. < 160 mg/dL
 - E. < 190 mg/dL
5. The target LDL cholesterol for a young healthy patient with 1 or fewer CHD risk factors is which of the following?
 - A. < 70 mg/dL
 - B. < 100 mg/dL
 - C. < 130 mg/dL
 - D. < 160 mg/dL
 - E. < 190 mg/dL

6. Which of the following may be a secondary cause of lipid abnormalities?

- A. Hypothyroidism
- B. Thiazide diuretics
- C. Nephrotic syndrome
- D. Anabolic steroids
- E. All of the above

7. What is the Framingham score for a male inmate who is 41 years old, smokes cigarettes, with an HDL cholesterol of 30 mg/dL, a total cholesterol of 250 mg/dL and an untreated systolic blood pressure of 150 mm Hg?

- A. 4
- B. 8
- C. 14
- D. 20
- E. 28

8. What is the LDL cholesterol level at which the inmate in Question #7 should be considered for drug therapy?

9. Which of the following is not a good recommendation for an inmate trying to consume a low fat/cholesterol diet?

- A. Choose pretzels, not potato chips
- B. Choose jelly beans, not candy bars
- C. Choose popsicles, not ice cream
- D. Choose a bagel, not a doughnut
- E. Choose egg yolks, not egg whites

10. Which of the following drug categories does not potentially cause liver complications?

- A. Bile acid sequestrants
- B. HMG-CoA reductase inhibitors
- C. Sustained-release niacin
- D. Fibrates

11. All of the “STATINS” can effectively lower LDL cholesterol, True or False?

12. Match one of the following drug classes with the descriptions below: bile acid sequestrants, niacin, fibrate, or HMG-CoA reductase inhibitors (STATINS).

- A. Flushing/exacerbates gout
- B. Primarily to treat hypertriglyceridemia
- C. Myopathy/decreases LDL/increases HDL/decreases triglycerides
- D. Absorbs other drugs/not recommended if triglycerides are elevated/minimal toxicities

PROVIDER SELF-ASSESSMENT ANSWERS
(Management of Lipid Disorders)

1. Answer is E
2. Answer is TRUE
3. Answer is C
4. Answer is B
5. Answer is D
6. Answer is E
7. Answer is C
8. Answer is < 130 mg/dL
9. Answer is E
10. Answer is A
11. Answer is TRUE
12. Matching answers:
 - A. Niacin
 - B. Fibrates
 - C. STATINS
 - D. Bile acid sequestrants