# Management of Lipid Disorders

**Federal Bureau of Prisons**
**Clinical Practice Guidelines**

**April 2008**

# Management of Lipid Disorders

**Federal Bureau of Prisons**
**Clinical Practice Guidelines**

**April 2008**

## What's New in the Document?

The following changes have been made since the January 2006 version of these guidelines.

### Screening Recommendations

- Routine lipid screening is recommended for diabetics *annually*, beginning age 20 (see *Appendix 1*).

### Risk Assessment and LDL-C Goals

- The 10-year CHD risk calculation for women was corrected from the previous version (*Appendix 3b*).
- An LDL $<70$ mg/dl is now considered a "reasonable goal" for those who are "very high risk"(*Appendix 2*).

### Treatment

- Treatment should be considered for inmates who are "high risk" and have an LDL $\geq 100$ mg/dl (*Appendix 2*).
- It is clarified that for inmates treated for lipid disorders that they should have an LDL measured as clinically indicated, and at least annually.
- It is emphasized that inmates with hypercholesterolemia should be aggressively treated to achieve LDL goals, utilizing more than one agent as necessary. This is particularly true for inmates with cardiovascular disease and major cardiovascular risk factors, especially diabetes mellitus.
- It is emphasized that ezetimibe (Zetia®) is a third-line agent. Ezetimibe should only be utilized if LDL goals cannot be achieved by utilizing a statin in combination with niacin and/or a bile acid sequestrant, or if side effects or contraindications preclude the use of those drugs.

# Table of Contents

## Appendices

# 1. Purpose

The Federal Bureau of Prisons (BOP) Clinical Practice Guidelines for "Management of Lipid Disorders" provide recommendations for the screening and medical management of lipid disorders. These guidelines are based primarily on the recommendations of the National Cholesterol Education Program (NCEP) on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults - Adult Treatment Panel III (ATP III) and related NCEP updates.

# 2. Screening

Screening for lipid disorders should occur in conjunction with other preventive health care services as outlined in the BOP Clinical Practice Guideline for "Preventive Health Care." See *Appendix 1* for a summary of BOP screening recommendations.

## Screening frequency:

Each inmate's risk factors for lipid disorders should be assessed at the baseline prevention visit. Use the following criteria to determine when routine lipid screening should be offered.

- Beginning at age 20, inmates with any of the following risk factors should be screened *annually*:
  - coronary heart disease (CHD)
  - peripheral vascular disease (PVD)
  - diabetes mellitus (DM)

- Beginning at age 20, inmates with any of the following risk factors should be screened *every 5 years*:
  - family history of early myocardial infarction or death (first-degree relative--male before age 50 or female before age 60)
  - history of both hypertension and cigarette smoking

- Average risk inmates should be screened *every 5 years*, beginning at age 35 for males and at age 45 for females

## Screening methods:

Lipid measurements should be obtained in accordance with the following guidelines.

- **Total serum cholesterol and HDL cholesterol (HDL-C)** are the preferred screening tests for most inmates. Blood samples can be obtained at any time 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.

- **Lipoprotein analysis** should be performed as an *initial* screening test for inmates with CHD, diabetes, or other CHD risk equivalent, and as a *follow-up* test for anyone with abnormal total cholesterol or HDL. The table below outlines the criteria for follow-up testing.

| Criteria for Obtaining a Follow-up Lipoprotein Analysis |
|---|
| ▸ Total cholesterol ≥240 mg/dL and no identified CHD risk factors*<br>▸ Total cholesterol ≥200 mg/dL and presence of CHD risk factors*<br>▸ HDL cholesterol <40 mg/dL<br>* For list of cardiac risk factors see Definitions section. |

A 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-C cannot be accurately estimated from a routine lipoprotein analysis and special laboratory procedures are indicated.

Classification of lipoprotein analysis results is detailed below.

| Classification of Lipoprotein Analysis Results | | |
|---|---|---|
| **Total cholesterol** | <200<br>200-239<br>≥240 | Desirable<br>Borderline high<br>High |
| **LDL cholesterol** | <100<br>100-129<br>130-159<br>160-189<br>≥190 | Optimal<br>Near/above optimal<br>Borderline high<br>High<br>Very high |
| **HDL cholesterol** | <40<br>≥60 | Low<br>High |
| **Triglycerides** | <150<br>150-199<br>200-499<br>≥500 | Normal<br>Borderline high<br>High<br>Very high |

## 3. Risk Assessment

A risk assessment for lipid disorders should be conducted for any inmate for whom a fasting lipoprotein analysis is indicated. Because the relative risk of sustaining a new cardiac event helps to determine both the target LDL-C level and the appropriate treatment strategy, LDL-C

results are evaluated in conjunction with a cardiac risk assessment. Steps for conducting a risk assessment and developing treatment goals and decisions are described below. A risk assessment worksheet is provided in *Appendix 2*.

## Step 1. Obtain relevant baseline data.

Risk assessment data include total cholesterol, LDL-C, HDL-C, triglycerides, fasting blood glucose, waist circumference, and blood pressure.

## Step 2. Identify presence of major CHD risk factors.

These include: cigarette smoking; hypertension (BP $>$140/90 mm Hg or on anti-hypertensive medication); low HDL-C ($<$40 mg/dL); family history of premature CHD (in first degree relative, male under age 55 or female under age 65); and age (men $\geq$45 years and women $\geq$55 years). An HDL-C of $\geq$60 mg/dL counts as a "negative" risk factor; its presence removes one risk factor from the total count.

## Step 3. Identify presence of CHD or CHD risk equivalents.

CHD equivalents are risk factors which place a person at a similar risk for CHD events as a history of CHD itself. These include: diabetes mellitus; symptomatic carotid artery disease; peripheral arterial disease (PAD); abdominal aortic aneurysm; renal artery disease; and chronic renal insufficiency (serum creatinine concentrations exceeding 1.5 mg/dL or estimated glomerular filtration rates (creatinine clearance) less than 60 mL/min per 1.73 $m^2$).

## Step 4. Assess for metabolic syndrome.

The metabolic syndrome consists of a cluster of the most serious risk factors for CHD, including: diabetes or prediabetes; abdominal obesity; lipid abnormalities; and hypertension. Presence of three or more of these risk factors is diagnostic for metabolic syndrome. Metabolic syndrome confers a 3-fold increased risk for myocardial infarction or stroke.

## Step 5. If the individual has *no* CHD *and no* CHD risk equivalent condition, *but does have* two or more CHD risk factors, calculate the 10-year CHD risk.

It is unnecessary to calculate CHD risk for individuals with CHD or CHD risk equivalent conditions, because these conditions *automatically* confer "high risk" status. Those with less than two CHD risk factors *automatically* are considered "low risk". For the rest, calculate the CHD risk by utilizing the Framingham risk tables (*Appendix 3a* and *Appendix 3b*) or an internet-based CHD risk calculator. A 10-year CHD risk of 20% or greater constitutes a CHD risk equivalent.

## Step 6. Determine risk category, establish LDL-C goal and outline appropriate treatment plan.

Utilizing the risk assessment data obtained in steps 1 to 5, determine the appropriate risk category for the patient. Based on the risk category, identify the appropriate LDL-C goal and determine if therapeutic lifestyle changes and/or drug treatment are indicated as outlined in *Appendix 2*.

# 4. Baseline Clinician Evaluation

A baseline clinician evaluation for lipid disorders is indicated for all inmates who, based on the risk assessment for managing lipid disorders (*Appendix 2*), should be considered for therapeutic lifestyle changes and/or drug treatment.

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

- Review of lipid disorder(s), if previously diagnosed, and dietary and drug treatment history;
- Assessment of current medications that may raise LDL-C 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 arterial disease, as well as secondary causes of elevated LDL-C, including hypothyroidism, diabetes, nephrotic syndrome, obstructive liver disease, and HIV infection treated with protease inhibitors or other antiretroviral therapies; and
- Attention to relevant portions of the social history, including alcohol intake, and illicit drug usage (including anabolic steroid use), as well as factors that may affect the inmate's ability to understand or participate in treatment recommendations such as educational level, language and cultural barriers, or 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, weight, waist circumference, and body mass index (BMI). (To determine BMI, use the internet-based calculator at www.cdc.gov/nccdphp/dnpa/bmi/calc-bmi.htm or the chart in *Appendix 4 .);*
- Examination of the neck for carotid bruits and distended veins, and palpation of the thyroid;
- 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); and

- 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-C.

- 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-C goals and cut points for treatment interventions are based on the risk assessment and are outlined in Step 6 of *Appendix 2*. The two major treatment strategies for lowering LDL-C are therapeutic lifestyle changes and drug therapy.

**Therapeutic lifestyle changes:** Therapeutic lifestyle changes include an improved diet, weight reduction in overweight patients, and increased physical activity. These lifestyle changes alone may reduce LDL-C to targeted levels and should be the initial treatment strategy for certain patients. Specific recommendations for therapeutic lifestyle changes are outlined in *Appendix 5 (Guide to Selecting a Fat/Cholesterol Controlled Diet)* and *Appendix 6 (Weight Control Information for Inmates)*.

- **Dietary guidance:** The NCEP (ATP III) recommends the following dietary goals for reducing LDL-C:
  - Reduce intake of saturated fat to $<7\%$ of calories; and
  - Reduce cholesterol intake to $<200$ mg/day.

  Calculating saturated fat and cholesterol content of meals can be difficult for both the inmate and the health care provider. The treating clinician should review the inmate's current eating habits and make dietary recommendations utilizing *Appendix 5*. General guidelines for a healthier diet to lower LDL-C include:
  - Actively select foods lower in saturated fat, cholesterol, and calories;
  - Consume carbohydrate calories predominantly from complex carbohydrates, which include grains (especially whole grains), fruits, vegetables, and dried beans and peas; and
  - Limit consumption of trans fatty acids, the hydrogenated vegetable oil found in most baked goods, fried foods, snack foods, and some hard margarines.

- **Weight reduction:** Overweight and obesity have reached epidemic proportions in the United States, increasing in both genders and in all population groups (see "The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity," accessible at http://www.surgeongeneral.gov/topics/obesity/). A gain of approximately 10 to 20 pounds results in an increased risk of CHD, at 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), which defines healthy weight, overweight, and obesity based on height and weight. Obesity is defined as a BMI of $\geq 30$ kg/m$^2$ and overweight, as a BMI of 25-29 kg/m$^2$.

  *NOTE: Some patients with a large muscle mass may be inaccurately classified as overweight when this method is used.*

  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 for inmates is outlined in *Appendix 6*.

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

- **Monitoring:** A fasting LDL-C should be obtained 12 weeks after initiating dietary and other therapeutic lifestyle changes. If LDL-C goals are not achieved at 12 weeks, further counseling should be provided and the LDL-C should be reassessed at 24 weeks. Inmates who fail to achieve LDL-C 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 no more than one risk factor for CHD and with modestly elevated LDL cholesterol can be treated effectively with therapeutic lifestyle changes alone.*

## Medication strategies and special considerations:

- **Hypercholesterolemia (elevated LDL cholesterol):** Lowering elevated LDL-C 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 (see *Appendix 2*). Patients with a history of CHD, PAD, or CHD equivalents such as diabetes mellitus, should be aggressively treated with drug therapy if LDL-C is elevated above targeted goals. Patients with one or no CHD risk factors can ordinarily be managed with therapeutic lifestyle changes alone. Drug therapy is usually not indicated for patients without risk factors unless the LDL-C is $\geq 190$ mg/dL.

In high-risk and moderate-risk patients, it is desirable to reduce LDL-C by 30-40% from baseline to achieve a significant reduction in risk for major coronary events. ***Prescribing minimal drug therapy to produce a small LDL-C reduction is not an effective use of LDL-lowering drugs.***

Drug therapy should be initiated with a single agent, usually an HMG-CoA reductase inhibitor ("statin"). Drug selection should be individualized, based on the patient's medical history, potential drug interactions, and other clinical concerns.

If LDL-C does not decrease to the targeted goal after 6-12 weeks of therapy, therapy should be intensified as follows. First, maximize the statin dose to achieve the target LDL-C (unless side effects preclude use of higher doses). If target LDL levels still are not achieved, then add one of the following agents: niacin or a bile acid sequestrant. Niacin should ordinarily be the first drug added to the regimen. Ezetimibe is a third-line agent. It should only be utilized if LDL goals cannot be achieved by utilizing a statin in combination with niacin and/or a bile acid sequestrant, or if side effects or contraindications preclude the use of those drugs.

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 lipid clinic or physician specialist. Once the targeted LDL-C is achieved, LDL-C levels should be monitored approximately every 6 months to determine if the treatment plan remains effective.

Patients at very high risk for CHD events (as defined in Step 6 of *Appendix 2*) may benefit from more intensive lipid lowering therapy. A target LDL-C of 70-80 mg/dL for individuals in this risk category has been proposed, using a statin alone. If these patients cannot achieve an LDL-C $<100$ mg/dL with a statin alone, a second drug such as niacin or a bile acid sequestrant should be considered.

*NOTE: Patients with baseline LDL-C $\geq$ 190 mg/dL may have a hereditary lipid disorder or other co-factor affecting lipid metabolism. Such patients usually require combination therapy for adequate lipid control.*

- **Metabolic syndrome:** The risk factors associated with metabolic syndrome increase the risk of CHD at any given LDL-C 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-C 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. Decreasing LDL-C levels should be the primary goal of therapy; 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. The treatment strategy for hypertriglyceridemias depends on the etiology and the severity of the lipid disorder. Patients with very high triglyceride levels ($\geq$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 fibrates or nicotinic acid 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-C and depressed HDL cholesterol. Patients with isolated elevated triglycerides on a fasting test should be screened for diabetes, pre-diabetes, and the metabolic syndrome.

  Treatment of isolated hypertriglyceridemia may be appropriate in a patient with overt CAD, a strong family history of CHD, or multiple coexisting risk factors. In addition, patients with very high triglyceride levels ($>$500 mg/dL) should be treated to avoid pancreatitis; patients with even higher triglyceride levels ($>$1000 mg/dL) should be treated to prevent the chylomicronemia syndrome.

- **Diabetes mellitus:** Diabetes is a CHD risk equivalent. Lowering elevated LDL-C levels to $<$100 mg/dL should be a priority for all diabetic patients. Diabetics with established cardiovascular disease are at very high risk for further cardiovascular events; for these patients, an LDL-C goal of $<$70-80 mg/dL is a reasonable clinical strategy. Drug therapy is usually required. Patients with diabetes frequently have low HDL cholesterol and elevated triglycerides, which are secondary targets for treatment.

  *NOTE: Maximizing control of elevated blood glucose is integral to the effective management of lipid disorders associated with diabetes.*

- **Gender considerations:** CHD in women is delayed by 10 to 15 years, as compared to men. Thus, young women without a CHD diagnosis who have moderate LDL-C elevations may warrant a more conservative approach to initiating drug therapy. Nevertheless, CHD remains the leading cause of death among women in the United States. Therefore, women with known CHD and elevations in LDL-C should be treated just as aggressively as men with similar high risk profiles.

- **Human immunodeficiency virus (HIV) infection:** Hypercholesterolemia and hypertriglyceridemia are potential complications of HIV infection which may be further exacerbated by antiretroviral therapy and can be associated with clinically significant cardiovascular disease. While protease inhibitors are the antiretroviral class of drugs most strongly correlated with dyslipidemias, other antiretroviral therapies may also affect lipid

metabolism. Risk assessments and treatment for lipid disorders for persons with HIV infection should be pursued consistent with the indications and recommendations of NCEP and as described in BOP clinical practice guidelines. Persons with co-existent HIV infection and lipid disorders should be treated for lipid disorders regardless of age due to the risk of cardiovascular complications; however, treatment should be no more aggressive than that for persons without HIV infection. Inmates with hypercholesterolemia who are prescribed protease inhibitors must be treated with an HMG-CoA reductase inhibitor that does not interfere with antiretroviral drug levels, such as pravastatin, fluvastatin, rosuvastatin, or atorvastatin. (Consult with the current BOP Formulary recommendations for preferred drug options). Inmates with hypertriglyceridemia and HIV infection should ordinarily be treated with gemfibrozil or fenofibrate if triglyceride levels exceed 500 mg/dL. Switching antiretroviral drug classes, as a strategy for improving the control of lipid abnormalities, may or may not be successful. Decisions about switching to alternate antiretroviral therapies should be considered on a case-by-case basis, while reviewing the potential treatment-related toxicities, drug interactions, and the risk of virologic relapse with the alternative antiretroviral regimen.

## Medication options:

Medications for lipid disorders are enumerated below and summarized in *Appendix 7* and *Appendix 8*. Clinicians should prescribe in accordance with the BOP National Formulary and ensure non-formulary use criteria are met for established non-formulary medications.

- **HMG-CoA reductase inhibitors (statins):** These agents inhibit HMG-CoA reductase, the enzyme that catalyzes the rate-limiting step in cholesterol synthesis. When given in therapeutic doses they reduce, but do not completely block, cholesterol biosynthesis. HMG-CoA reductase inhibitors are the only class of drugs that have been shown to decrease overall mortality in primary and secondary prevention. Treatment with these agents decreases LDL-C by 18-55% and triglycerides by 7-30%, and increases HDL cholesterol by 5-15%. LDL-C reductions are dose-dependent. Single daily doses of any of these agents should be administered for maximal efficacy. Doses should generally be administered with the evening meal.

  Formulations of statins differ in their dosages and quantitative effects on lipids although all are very effective in lowering LDL-C. The effectiveness of the statins is compared in *Appendix 9*.

  HMG-CoA reductase inhibitors are well tolerated. Gastrointestinal side effects such as dyspepsia, flatus, and constipation are usually mild 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 these 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 about restarting the same medication if transaminase levels normalize, trying another statin, or trying 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 statins. 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 and substances similarly metabolized in the liver, including cyclosporine, gemfibrozil, itraconazole, ketoconazole, erythromycin, clarithromycin, nefazodone, and grapefruit juice. Fluvastatin and rosuvastatin can also potentiate the effect of oral anticoagulants; therefore, inmates concurrently prescribed warfarin medications should have their prothrombin time monitored closely, or an alternative statin should be prescribed.

- **Niacin (nicotinic acid):** Niacin, a B-complex vitamin, is effective in lowering LDL-C (5-25%) and triglycerides (20-50%), and raising HDL cholesterol (15-35%). This class of drugs is particularly effective in treating patients with low HDL cholesterol or with both elevated LDL-C and triglyceride levels. Niacin may cause 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, which are contraindicated. 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, 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. The BOP National Formulary is restricted to Niaspan® brand, an extended-release formulation of niacin. It is a good option because it is dosed once daily (usually at bedtime), is associated with less initial flushing than immediate-release formulations, and appears to have a reduced incidence of hepatotoxicity as compared to long-acting niacin.

  Niacin is frequently administered as a second agent to intensify therapy in conjunction with a statin.

- **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-C and normal triglycerides who have failed dietary therapy, particularly young men and premenopausal women with one or no CHD risk factors. (If two or more CHD risk factors are present, use one of the statins unless they are contraindicated.) Bile acid-binding resins can also be used in conjunction with statins for the treatment of patients with severe hypercholesterolemia. These agents should not be used for inmates with elevated triglycerides, particularly if >400 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 use of psyllium should be recommended to relieve constipation and bloating.

- **Fibric acid derivatives:** Medications containing fibric acid, such as gemfibrozil and fenofibrate, decrease the synthesis of very low density lipoproteins (VLDL) triglycerides, and thus are primarily indicated for treating hypertriglyceridemia. These agents decrease triglycerides (20-50%), modestly increase HDL cholesterol (10-20%), and decrease LDL-C (5-20%). Because of their limited efficacy, these agents should not be used as first-line treatment for hypercholesterolemia in persons with CHD.

  *NOTE: 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 to ensure 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, potential risk of increased mortality and malignancy associated with these agents.

- **Cholesterol absorption inhibitor: Ezetimibe (Zetia®)** is the first in a relatively new class of lipid-lowering agents. It is indicated in combination with a statin for intensification of treatment of primary hypercholesterolemia. Ezetimibe has a unique mechanism of action compared to other classes of lipid-lowering agents. It inhibits the absorption of dietary and biliary cholesterol, decreasing the intestinal absorption by 54%. Ezetimibe does not, however, affect triglyceride absorption. In clinical trials, the only significant side effect of ezetimibe given as monotherapy was low back pain occurring in 4% of patients. No additional lab monitoring is needed when using ezetimibe.

  While ezetimibe has been shown to effectively lower LDL, a recent study showed no associated reduction in placque growth rate. Moreover there is no available outcome data to demonstrate the effect of ezetimibe on cardiovascular morbidity and mortality. Thus, ezetimibe is considered a third-line agent which should only be utilized if LDL goals cannot be achieved with statins in combination with niacin and/or a bile acid sequestrant, or if side effects or contraindications to those drugs preclude their use.

# 6. Periodic Evaluations

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

**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; and
- Assessment of adherence to drug therapy and the presence of drug side effects (consult with the pharmacist to review adherence to the prescribed medication regimen).

**Physical examinations:** Performed as clinically warranted, physical examinations should target the following: measurement of vital signs, including blood pressure; measurement of weight and waist circumference; and examination of the heart, lungs, pulses, and extremities, with auscultation over the carotid and femoral arteries for bruits.

**Diagnostic and laboratory evaluations:** The LDL-C should be measured 4-6 weeks after beginning medication and again at 12 weeks. If LDL-C goals are met, total cholesterol or lipoprotein analysis should be measured as clinically indicated and at least annually. 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 with information on diet modifications and medication use, and by monitoring for both adherence to treatment and occurrence of adverse drug reactions. Pharmacists, when privileged by the Clinical Director, can order and review laboratory work and provide medication management for lipid disorders per an approved protocol.

**Inmate education:** All inmates with 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 individual or group educational efforts. Educational materials are attached as *Appendix 5 (Guide to Selecting a Fat/Cholesterol Controlled Diet), Appendix 6 (Weight Control Information for Inmates), Appendix 10 (Health Education Guide on Lipid Disorders)* and *Appendix 11 (Inmate Fact Sheet on High Cholesterol)*.

# 7. Health Care Provider Resources

Provider resources for managing lipid disorders are listed in *Appendix 12*.

# Definitions

**Acute coronary syndrome** is a general term for clinical presentations of myocardial ischemia such as unstable angina and both Q-wave and non-Q-wave myocardial infarction.

**Body mass index (BMI)** is a measure of a person's weight in relation to his or her height. The BMI equals the weight in pounds divided by the square of the height in inches, multiplied by 703; alternatively the weight in kilograms divided by the square of the height in meters, multiplied by 703. BMI is highly correlated with total body fat and is used to assess overweight and obesity. A BMI of 30 $kg/m^2$ or greater indicates obesity; and a BMI between 25 and 29.9 $kg/m^2$ identifies overweight adults.

*NOTE: Some persons with large muscle mass may be inaccurately classified as overweight with this method.*

**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, a mid-level provider, or an appropriately credentialed pharmacist.

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

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

**CHD risk factors (that modify LDL-C goals)** are factors, exclusive of LDL-C itself, that increase the likelihood of developing coronary atherosclerosis and associated CHD. Factors include: **age** (men $\geq$45 years of age and women $\geq$55 years of age); **cigarette smoking**; **hypertension** (blood pressure $>$140/90 mm Hg or taking antihypertensive medications); **family history** of premature CHD or sudden death (in first-degree relative, male before age 55 or female before age 65); and **low HDL cholesterol** ($<$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 1. Diabetes mellitus is considered a CHD risk equivalent (see below) and is not counted as a separate risk factor, i.e., patients with diabetes who have elevated LDL-C 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 is 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 (PAD); abdominal aortic aneurysm; symptomatic carotid artery disease; renal artery disease; chronic renal insufficiency (serum creatinine $>$1.5

mg/dL or creatinine clearance <60 mL/min.), 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, and LDL and HDL cholesterol.

**Low density lipoproteins (LDL)** are lipoproteins that contain 60-70% of the total serum cholesterol. *LDL cholesterol = Total cholesterol - HDL cholesterol - (Triglycerides/5)*. (This calculation is invalid if triglycerides are >400 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-C level. Metabolic syndrome is diagnosed when three or more of the following risk determinants are present: fasting glucose between 100-110 mg/dL; blood pressure ≥130/≥85 mm Hg; triglycerides ≥150 mg/dL; HDL <40 mg/dL for men and <50 mg/dL for women; or abdominal obesity (waist circumference >40 inches for men and >35 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), and 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.

# References

American Heart Association, American College of Cardiology. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update. *Circulation*. 2006;113:2363-2372. Available from: http://circ.ahajournals.org/cgi/content/full/113/19/2363#TBL3

Dube MP, Stein JH, Alberg JA, et al. Guidelines for the evaluation and management of dyslypidemia in human immunodeficiency virus-infected adults receiving antiretroviral therapy: Recommendations of the HIV Medicine Association of the Infectious Disease Society of America and the Adult AIDS Clinical Trials Group. *Clin Inf Dis* 2003;37:613-27.

Federal Bureau of Prisons. Food service manual (P.S. 4700.04). Washington, D.C.: Department of Justice; 1996. Available from: http://www.bop.gov/policy/progstat/4761_004.pdf

Federal Bureau of Prisons, Division of Health Services. National formulary. Available from: http://www.bop.gov/news/medresources.jsp

Federal Bureau of Prisons. *Clinical Practice Guideline: Preventive Health Care*. Washington, DC: Federal Bureau of Prisons; 2004. Available from: http://www.bop.gov/news/medresources.jsp

Grundy SM, Cleeman JI, Merz CNB, et al. Implications of recent clinical trials for the National Cholesterol Education Project Adult Treatment Panel III guidelines. *Circulation* 2004; 110:227-239. Available from: http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3upd04.pdf

National Cholesterol Education Program (NCEP) Expert Panel. Detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III). Bethesda, MD: National Institutes of Health; 2002. Available from: http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm

Rosenson RS. Overview of treatment of hypercholesterolemia. *Up To Date*. May 13, 2005.

U.S. Department of Health and Human Services. *The Surgeon General's call to action to prevent and decrease overweight and obesity*. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001. Available from: http://www.surgeongeneral.gov/topics/obesity/calltoaction/toc.htm

## Appendix 1. Screening Criteria for Lipid Disorders

| Risk Status | Age for Screening | Frequency | Initial Screening Test |
|---|---|---|---|
| **Average Risk Inmates** | | | |
| **Males** | age 35 | every 5 yrs | HDL-C & total cholesterol[1] |
| **Females** | age 45 | | |
| **Inmates with Risk Factors** | | | |
| **First-degree relative with history of myocardial infarction or cardiac death** (male before age 50 or female before age 60) | age 20 | every 5 yrs | HDL-C & total cholesterol[1] |
| **History of both cigarette smoking and hypertension** (BP > 140/90 or on antihypertensives) | | | |
| **Diabetes mellitus** | | every year | fasting lipoprotein analysis |
| **Coronary heart disease** or **peripheral vascular disease** | | every year | |

**[1] Criteria for Obtaining a Follow-up Lipoprotein Analysis**

A follow-up, fasting lipoprotein analysis should be performed for inmates with abnormal total cholesterol or HDL, as indicated below:

- Total cholesterol ≥ 240 mg/dL and no identified cardiac risk factors*
- Total cholesterol ≥ 200 mg/dL and presence of cardiac risk factors*
- HDL cholesterol < 40 mg/dL

* Cardiac risk factors include:
- cigarette smoking
- hypertension (BP > 140/90 or on antihypertensive medication)
- low HDL cholesterol (< 40 mg/dL)
- family history of premature CHD
  (CHD in first-degree relative, male before age 55 or female before age 65)
- age (male ≥ 45 years and female ≥ 55 years)

## Appendix 2. Risk Assessment for Managing Lipid Disorders

**(page 1 of 2)** (Adapted from NCEP. ATP III Guidelines At-A-Glance: Quick Desk Reference)

**Step 1. Obtain relevant baseline data.**

| | |
|---|---|
| Total cholesterol | ____mg/dL |
| LDL cholesterol | ____mg/dL |
| HDL cholesterol | ____mg/dL |
| Triglycerides | ____mg/dL |
| Fasting blood glucose | ____mg/dL |
| Waist circumference | ____ inches |
| Blood pressure | _____/_____mm Hg |

**Step 2. Identify presence of major CHD risk factors.**

- ☐ Cigarette smoking
- ☐ Hypertension (BP >140/90 or on antihypertensive medication)
- ☐ Low HDL cholesterol (<40 mg/dL)
- ☐ Family history of premature CHD
  (CHD in first-degree relative, either male before age 55 or female before age 65)
- ☐ Age (men ≥45 years; women ≥55 years)
- ☐ **None**

---

- ☐ High HDL cholesterol (>60 mg/dL)=negative risk factor. Remove 1 risk factor from count.

**Step 3. Identify presence of CHD or CHD risk equivalents**, which confer "high risk" status for occurrence of CHD events.

- ☐ Clinical CHD
- ☐ Diabetes mellitus
- ☐ Peripheral arterial disease
- ☐ Renal artery disease
- ☐ Abdominal aortic aneurysm
- ☐ Symptomatic carotid artery disease
- ☐ Chronic renal insufficiency (serum creatinine >1.5 mg/dL or GFR <60 ml/min per1.73 $m^2$)
- ☐ **None**

**Step 4. Assess for metabolic syndrome.** The diagnostic criteria for metabolic syndrome requires presence of three or more of the following conditions:

- ☐ Abdominal obesity (males >40 inches; females >35 inches)
- ☐ Triglycerides ≥150 mg/dL
- ☐ HDL cholesterol (males <40 mg/dL; females <50 mg/dL)
- ☐ Blood pressure ≥130/≥85
- ☐ Fasting blood glucose ≥100-110 mg/dL

**Step 5. If ☐ no CHD *and* ☐ no CHD risk equivalent *and* ☐ two or more CHD risk factors are present, then calculate 10-year CHD risk.**

Use Framingham tables (*Appendix 3a* and *Appendix 3b*) or on-line risk calculator at: http://hin.nhlbi.nih.gov/atpiii/calculator.asp?usertype=prof

| **10-yr risk score= ____%** | ☐ >20% = high risk<br>☐ 10-20% = moderate risk (a)<br>☐ <10% = moderate risk (b) |
|---|---|

## Appendix 2. Risk Assessment for Managing Lipid Disorders
**(page 2 of 2)**

**Step 6. Use Risk Criteria to determine Risk Category. Then determine LDL Goal and treatment plan.**

| Risk Category | Risk Criteria | LDL Goal | LDL Level (mg/dL) at which to … | |
|---|---|---|---|---|
| | | | Initiate Lifestyle Changes | Consider Drug Therapy |
| **Very High Risk** | ☐ Established CHD *plus*:<br>☐ major risk factors, esp. diabetes; **or**<br>☐ severe and poorly controlled risk factors (esp. continued cigarette smoking); **or**<br>☐ metabolic syndrome risk factors (esp. triglyc ≥200 and HDL-C ≤40); **or**<br>☐ acute coronary syndrome | <100 (with <70 considered a reasonable goal) | ≥100 | ≥100<br>(<100 ⇒ optional[1]) |
| **High Risk** | ☐ CHD or CHD risk equivalents (Step 3)<br>**or**<br>☐ 10-year risk >20% (Step 5) | <100 | ≥100 | ≥100 |
| **Moderate Risk (a)** | ☐ 2 or more CHD risk factors (Step 2)<br>**and**<br>☐ 10-year risk 10-20% (Step 5) | <130 | ≥130 | ≥130 |
| **Moderate Risk (b)** | ☐ 2 or more CHD risk factors (Step 2)<br>**and**<br>☐ 10-year risk <10% (Step 5) | <130 | ≥130 | ≥160 |
| **Low Risk** | ☐ 0-1 CHD risk factor (Step 2) | <160 | ≥160 | ≥190<br>(160-189 ⇒ optional) |

[1] Initiating drug therapy in very high-risk patients with LDL-C <100 mg/dL is an option that has clinical support.

*NOTE: For more information on the risk assessment process, see Sections 2 and 3 of these guidelines.*

## Appendix 3a. Framingham Score for Men (Estimating 10-Year Risk for CHD)

**1. HDL Cholesterol (mg/dL)** **Points: ___**

| HDL | Points |
|---|---|
| ≥60 | -1 |
| 50-59 | 0 |
| 40-49 | 1 |
| <40 | 2 |

**2. Total Cholesterol (mg/dL)** **Points:___**

| Total Cholesterol | Age 20-39 | Age 40-49 | Age 50-59 | Age 60-69 | Age 70-79 |
|---|---|---|---|---|---|
| <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 |

**3. Smoking** **Points:___**

| | Age 20-39 | Age 40-49 | Age 50-59 | Age 60-69 | Age 70-79 |
|---|---|---|---|---|---|
| Non-smoker | 0 | 0 | 0 | 0 | 0 |
| Smoker | 8 | 5 | 3 | 1 | 1 |

**4. Age** **Points:___**

| Age | 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 |

**5. Systolic BP** **Points:____**

| Systolic BP | Untreated | Treated |
|---|---|---|
| <120 | 0 | 0 |
| 120-129 | 0 | 1 |
| 130-139 | 1 | 2 |
| 140-159 | 1 | 2 |
| ≥160 | 2 | 3 |

**Calculate Total Points**

1. HDL Cholesterol ___
2. Total Cholesterol ___
3. Smoking ___
4. Age ___
5. Systolic BP ___

**Total Points** ___

**10-Year CHD Risk** ____%

| Total Points | 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 |

Internet-based 10-year CHD risk assessment tool:
http://hin.nhlbi.nih.gov/atpiii/calculator.asp?usertype=prof

## Appendix 3b. Framingham Score for Women (Estimating 10-Year Risk for CHD)

### 1. HDL Cholesterol (mg/dL) Points: ___

| HDL | Points |
|---|---|
| ≥60 | -1 |
| 50-59 | 0 |
| 40-49 | 1 |
| <40 | 2 |

### 2. Total Cholesterol (mg/dL) Points:___

| Total Cholesterol | Age 20-39 | Age 40-49 | Age 50-59 | Age 60-69 | Age 70-79 |
|---|---|---|---|---|---|
| <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 |

### 3. Smoking Points:___

| | Age 20-39 | Age 40-49 | Age 50-59 | Age 60-69 | Age 70-79 |
|---|---|---|---|---|---|
| Non-smoker | 0 | 0 | 0 | 0 | 0 |
| Smoker | 9 | 7 | 4 | 2 | 1 |

### 4. Age Points ___

| Age | 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 |

### 5. Systolic BP Points:____

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

### Calculate Total Points

1. HDL Cholesterol ____
2. Total Cholesterol ____
3. Smoking ____
4. Age ____
5. Systolic BP ____

Add Total Points ____

### 10-Year CHD Risk ____%

| Total Points | 10-Year Risk (%) |
|---|---|
| <9 | <1 |
| 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 |

Internet-based 10-year CHD risk assessment tool:
http://hin.nhlbi.nih.gov/atpiii/calculator.asp?usertype=prof

## Appendix 4. Body Mass Index (BMI) Table

| BMI (kg/m²) | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 32 | 33 | 34 | 35 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Height (inches) | Body Weight (Pounds) | | | | | | | | | | | | | | | | |
| | Normal | | | | | | Overweight | | | | | Obese | | | | | |
| 58 | 91 | 96 | 100 | 105 | 110 | 115 | 119 | 124 | 129 | 134 | 138 | 143 | 148 | 153 | 158 | 162 | 167 |
| 59 | 94 | 99 | 104 | 109 | 114 | 119 | 124 | 128 | 133 | 138 | 143 | 148 | 153 | 158 | 163 | 168 | 173 |
| 60 | 97 | 102 | 107 | 112 | 118 | 123 | 128 | 133 | 138 | 143 | 148 | 153 | 158 | 163 | 168 | 174 | 179 |
| 61 | 100 | 106 | 111 | 116 | 122 | 127 | 132 | 137 | 143 | 148 | 153 | 158 | 164 | 169 | 174 | 180 | 185 |
| 62 | 104 | 109 | 115 | 120 | 126 | 131 | 136 | 142 | 147 | 153 | 158 | 164 | 169 | 175 | 180 | 186 | 191 |
| 63 | 107 | 113 | 118 | 124 | 130 | 135 | 141 | 146 | 152 | 158 | 163 | 169 | 175 | 180 | 186 | 191 | 197 |
| 64 | 110 | 116 | 122 | 128 | 134 | 140 | 145 | 151 | 157 | 163 | 169 | 174 | 180 | 186 | 192 | 197 | 204 |
| 65 | 114 | 120 | 126 | 132 | 138 | 144 | 150 | 156 | 162 | 168 | 174 | 180 | 186 | 192 | 198 | 204 | 210 |
| 66 | 118 | 124 | 130 | 136 | 142 | 148 | 155 | 161 | 167 | 173 | 179 | 186 | 192 | 198 | 204 | 210 | 216 |
| 67 | 121 | 127 | 134 | 140 | 146 | 153 | 159 | 166 | 172 | 178 | 185 | 191 | 198 | 204 | 211 | 217 | 223 |
| 68 | 125 | 131 | 138 | 144 | 151 | 158 | 164 | 171 | 177 | 184 | 190 | 197 | 203 | 210 | 216 | 223 | 230 |
| 69 | 128 | 135 | 142 | 149 | 155 | 162 | 169 | 176 | 182 | 189 | 196 | 203 | 209 | 216 | 223 | 230 | 236 |
| 70 | 132 | 139 | 146 | 153 | 160 | 167 | 174 | 181 | 188 | 195 | 202 | 209 | 216 | 222 | 229 | 236 | 243 |
| 71 | 136 | 143 | 150 | 157 | 165 | 172 | 179 | 186 | 193 | 200 | 208 | 215 | 222 | 229 | 236 | 243 | 250 |
| 72 | 140 | 147 | 154 | 162 | 169 | 177 | 184 | 191 | 199 | 206 | 213 | 221 | 228 | 235 | 242 | 250 | 258 |
| 73 | 144 | 151 | 159 | 166 | 174 | 182 | 189 | 197 | 204 | 212 | 219 | 227 | 235 | 242 | 250 | 257 | 265 |
| 74 | 148 | 155 | 163 | 171 | 179 | 186 | 194 | 202 | 210 | 218 | 225 | 233 | 241 | 249 | 256 | 264 | 272 |
| 75 | 152 | 160 | 168 | 176 | 184 | 192 | 200 | 208 | 216 | 224 | 232 | 240 | 248 | 256 | 264 | 272 | 279 |
| 76 | 156 | 164 | 172 | 180 | 189 | 197 | 205 | 213 | 221 | 230 | 238 | 246 | 254 | 263 | 271 | 279 | 287 |

**Body mass index calculator:** www.cdc.gov/nccdphp/dnpa/bmi/calc-bmi.htm

## Appendix 5. Guide to Selecting a Fat/Cholesterol Controlled Diet
**(page 1 of 2)**

| Daily Goals | Choose | Go Easy On | Decrease |
|---|---|---|---|
| **Meat, Poultry, and Fish** (Up to 6 oz. per day) | ▸ Lean cuts of meat with fat trimmed<br>▸ Baked, unbreaded poultry without skin<br>▸ Baked, unbreaded fish<br>▸ Canned chicken, tuna, or sardines (water packed or rinsed)<br>▸ Dried beans and peas as a meat substitute | | ▸ Fried meat<br>▸ Breaded meat<br>▸ Organ meats, like liver<br>▸ Sausage<br>▸ Bacon<br>▸ Lunch meats<br>▸ Hot dogs<br>▸ Fatty cuts of meat, like brisket or ribs |
| **Eggs** (No more than 2 egg yolks *per week*) | ▸ Egg whites<br>▸ Cholesterol-free egg substitutes | | ▸ Egg yolks |
| **Dairy Products** (2-3 servings per day) | ▸ Skim milk, 1% milk, low fat buttermilk, or nonfat powdered milk<br>▸ Low-fat yogurt (plain and frozen)<br>▸ Low-fat cottage cheese | ▸ 2% milk<br>▸ Yogurt<br>▸ Part-skim cheeses like mozzarella, or string cheese | ▸ Whole milk<br>▸ Cream, half-and-half, most non-dairy products (like creamers), real or non-dairy whipped cream<br>▸ Cream cheese<br>▸ Sour cream<br>▸ High-fat cheeses like Swiss, cheddar, and American |
| **Fats and Oils** (Amount adjusted to caloric level) | ▸ Low-fat dressings | ▸ Unsaturated vegetable oils: olive, peanut, canola, safflower, soybean<br>▸ Margarine<br>▸ Nuts/seeds<br>▸ Peanut butter<br>▸ Olives<br>▸ Avocados<br>▸ Mayonnaise salad dressings | ▸ Butter, lard, bacon fat (animal fats)<br>▸ Coconut oil<br>▸ Palm oil<br>▸ Palm kernel oil<br>▸ Bacon<br>▸ Hydrogenated fat or oil |

## Appendix 5. Guide to Selecting a Fat/Cholesterol Controlled Diet
**(page 2 of 2)**

| Daily Goals | Choose | Go Easy On | Decrease |
|---|---|---|---|
| **Fruits and Vegetables** (2-4 servings of fruit and 3-5 servings of vegetables per day) | ▸ Fresh, frozen, canned, or dried fruits and vegetables | | ▸ Vegetables prepared in butter, cream, or sauce<br>▸ Fried vegetables |
| **Breads, Pasta, Cereals, Rice, Dried Beans, and Peas** (6 or more servings per day, adjusted to caloric needs) | ▸ Breads like white, whole wheat, rye, pita, pumpernickel, bagels, English muffins, sandwich buns, rice cakes<br>▸ Low-fat crackers, like matzo, bread sticks, rye krisp, saltines, zwieback<br>▸ Rice, pasta, dried beans and peas prepared without fat | ▸ Pancakes<br>▸ Waffles<br>▸ Biscuits<br>▸ Cornbread | ▸ Croissants, butter rolls, sweet rolls, Danish pastry, doughnuts<br>▸ Cheese or butter crackers<br>▸ Granola-type cereals<br>▸ Pasta and rice prepared with cream, butter, or cheese sauce |
| **Sweets and Snacks** (avoid too many sweets, even when low- fat) | ▸ Fat-free desserts, like sherbet, Italian ice, frozen yogurt, popsicles<br>▸ Fat-free cakes, like angel food cake<br>▸ Fat-free candy, like jelly beans and hard candy<br>▸ Very low-fat snacks, like popcorn and pretzels<br>▸ Non-fat beverages, like carbonated drinks, juices, tea, coffee | ▸ Low-fat frozen desserts, like ice milk<br>▸ Low-fat cookies, like fig bars, ginger snaps, animal crackers, graham crackers | ▸ High-fat frozen desserts, like ice cream<br>▸ High-fat cakes, like pound cake and frosted cakes<br>▸ Pastries and cookies<br>▸ Most candy, especially chocolate<br>▸ Potato chips, corn chips, and other snack chips<br>▸ Buttered popcorn<br>▸ High-fat beverages like milkshakes and egg nog |

**Very Low Cholesterol Diet**

To further reduce your saturated fat and cholesterol intake, follow these suggestions:

- Eat only foods from the "choose" category.
- Limit egg yolks to no more than one per week.
- Choose dried beans as a meat substitute, when possible.

## Appendix 6. Weight Control Information for Inmates

- **Cutting down on calories is the first step to losing weight.** One pound of body fat is equal to 3,500 calories. A person must reduce calorie intake by 500 calories per day to lose one pound in a week. You can reduce calories by eating less and by eating foods that are lower in calories.
- **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 to 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, Kool-aid, 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 other 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 your tea or coffee black, or limit what you add to them.
- **Eat slowly. Eat your salad first:** It helps to fill you up.
- **Learn to stop eating *before* you are "full" or "stuffed."** The slight hunger you feel will disappear about one-half hour after mealtime.
- **Stay active. 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!*

## Appendix 7. Overview of Drugs Effecting Lipid Metabolism

(See Appendix 8 for more detailed drug treatment information.)

Drug therapy should be initiated with a single agent, usually an HMG-CoA reductase inhibitor ("statin"). If LDL-C does not decrease to the targeted goal after 6-12 weeks of therapy, therapy should be intensified as follows. First, maximize the statin dose to achieve the target LDL-C (unless side effects preclude use of higher doses). If target LDL-C levels still are not achieved, then add one of the following agents: niacin or a bile acid sequestrant. Niacin is usually the first drug added.

For "high risk" and "moderate risk" persons a reduction of LDL-C by 30-40% is needed to reduce the risk of major coronary events.

| Drug Class | Agents | Lipid Effect | Side Effects | Contraindication |
|---|---|---|---|---|
| **HMG CoA reductase inhibitors (statins)** | ▸ Lovastatin<br>▸ Pravastatin<br>▸ Simvastatin<br>▸ Fluvastatin<br>▸ Atorvastatin<br>▸ Rosuvastatin | LDL ⇓18-55%<br>HDL ⇑ 5-15%<br>TG ⇓7-30% | ▸ myopathy<br>▸ increased liver enzymes | **Absolute:**<br>▸ active or chronic liver disease<br>**Relative:**<br>▸ concomitant use of certain drugs |
| **Bile acid sequestrants** | ▸ Cholestyramine<br>▸ Colestipol<br>▸ Colesevelam | LDL ⇓15-30%<br>HDL ⇑ 3-5%<br>TG No change or increase | ▸ GI distress<br>▸ constipation<br>▸ decreased absorption of other drugs | **Absolute:**<br>▸ dysbeta-lipoproteinemia<br>▸ TG >400 mg/dL<br>**Relative:**<br>▸ TG >200 mg/dL |
| **Nicotinic acid** | ▸ Extended release niacin | LDL ⇓5-25%<br>HDL ⇑15-35%<br>TG ⇓20-50% | ▸ flushing<br>▸ hyperglycemia<br>▸ hyperuricemia (or gout)<br>▸ upper GI distress<br>▸ hepatotoxicity | **Absolute:**<br>▸ chronic liver disease<br>▸ severe gout<br>**Relative:**<br>▸ diabetes<br>▸ hyperuricemia<br>▸ peptic ulcer disease |
| **Fibric acids** | ▸ Gemfibrozil<br>▸ Fenofibrate | LDL ⇓ 5-20% (may increase in patients with high TG)<br>HDL ⇑10-20%<br>TG ⇓20-50% | ▸ dyspepsia<br>▸ gallstones<br>▸ myopathy | **Absolute:**<br>▸ severe renal disease<br>▸ severe hepatic disease |
| **Cholesterol absorption inhibitor** | ▸ Ezetimibe | LDL ⇓15-19%<br>HDL ⇑ 3-4%<br>TG No change | ▸ low back pain | None |

## Appendix 8. Specific Drug Treatment Options for Lipid Disorders[1,2]
**(page 1 of 4)**

| Medication/ Dose Range | Toxicities/ Labs | Comments/ Drug Interactions |
|---|---|---|
| **HMG CoA Reductase Inhibitors (Statins)** | | |
| **lovastatin (Mevacor®) Immediate Release 10-80 mg/day** | ▸ rhabdomyolysis<br>▸ hepatotoxicity<br>**Monitor:** ALT/AST (baseline, every 6 wks x 2 yrs; repeat after dose increased, then q 6 mos) | Starting dose 20 mg at bedtime. Maximum dose 80 mg daily. Take with food. Contraindicated in active liver disease, pregnancy, unexplained elevated LFTs. Lower dose if creatinine clearance ≤30 L/min. ***Drug interactions:*** Interacts with drugs metabolized by CYP3A4 enzyme system[3]. Increased risk of rhabdomyolysis when administered with fibrates or niacin. |
| **simvastatin (Zocor®) 5-80 mg/day** | ▸ rhabdomyolysis<br>▸ hepatotoxicity<br>**Monitor:** ALT/AST (baseline, then every 6 months) | Starting dose 20 mg at bedtime. Maximum dose 80 mg at bedtime. Lower dose if renal insufficiency is severe (initial dose 5 mg). For patients titrated to 80 mg, obtain ALT/AST before titration, 3 months afterwards and every 6 months x 2 years. ***Drug interactions:*** Interacts with drugs metabolized by CYP3A4 enzyme system[3]. Increased risk of rhabdomyolysis when administered with fibrates or niacin. |
| **fluvastatin (Lescol®) Immediate Release 20-80 mg/day** | ▸ rhabdomyolysis<br>▸ hepatotoxicity<br>▸ pancreatitis<br>▸ hypersensitivity<br>**Monitor:** ALT/AST (baseline & at 12 weeks; repeat after dose is increased, then q 6 mos) | Starting dose 20 mg at bedtime. Maximum dose 40 mg twice daily. (Twice daily requires non-formulary approval.) No dose adjustment for renal insufficiency. ***Drug interactions:*** Metabolized by CYP2C9, *not* CYP3A4, and may be less likely to be involved in drug interactions. Can increase warfarin, phenytoin, and NSAID levels. Rifampin can lower fluvastatin levels. Increased risk of rhabdomyolysis when administered with fibrates or niacin. Can be used with protease inhibitors. |
| **pravastatin (Pravachol®) 10-80 mg/day** | ▸ rhabdomyolysis<br>▸ hepatotoxicity<br>**Monitor:** ALT/AST (baseline and after dose is increased, then every 6 months) | Starting dose 40 mg once daily. Maximum dose 80 mg once daily. Lower dose if creatinine clearance is ≤ 60 L/min (initial dose 10 mg daily). ***Drug interactions:*** Not metabolized by cytochrome P450 system, so less likely to have drug interactions. Cyclosporine can increase pravastatin levels. Can be used with protease inhibitors using low dose (consult with HIV pharmacist). Risk of rhabdomyolysis increased when given with fibrates or niacin. |
| **atorvastatin (Lipitor®) 10-80 mg/day** | ▸ rhabdomyolysis<br>▸ hepatotoxicity<br>**Monitor:** ALT/AST (baseline & at 12 wks; repeat after dose increases, then q 6 mos) | Starting dose 10 mg at bedtime. Maximum dose 80 mg at bedtime. No adjustment for renal insufficiency. May be given without regard to meals. ***Drug interactions:*** Interacts with drugs metabolized by CYP3A4 enzyme system[3], but less than lovastatin and simvastatin. Increased risk of rhabdomyolysis when administered with fibrates or niacin. |
| **rosuvastatin (Crestor®) 5-40 mg/day** | ▸ rhabdomyolysis<br>▸ hepatotoxicity<br>**Monitor:** ALT/AST (baseline & at 12 weeks; repeat after dose is increased, then q 6 mos) | Starting dose 10 mg daily. Maximum dose 40 mg once daily. Lower dose if creatinine clearance ≤30 L/min. ***Drug Interactions:*** Not metabolized by cytochrome P450 system, so less likely to have drug interactions; however, may increase INR with warfarin. Increased risk of rhabdomyolysis when administered with fibrates or niacin. |

[1] Prescribe according to BOP formulary. [2] Use only one "statin" at a time, titrating to target LDL-C, side effects, or maximum dose before switching. [3] Watch for drug interactions which inhibit this enzyme including: diltiazem, erythromycin, clarithromycin, ketoconazole, verapamil, nefazodone, fluvoxamine, cyclosporine, protease inhibitors

## Appendix 8. Specific Drug Treatment Options for Lipid Disorders
**(page 2 of 4)**

| Medication/ Dose Range | Toxicities/ Labs | Comments/ Drug Interactions |
|---|---|---|
| **Bile Acid Sequestrants** | | |
| **cholestyramine (LoCholest®, Questran®, Prevalite®) 4-24 gm/day** | ▸ fecal impaction<br><br>**Monitor** LDL-C and TG levels. | Initially, 4 g 1 or 2 times daily. Increase dose at 4-week intervals as tolerated. Maximum dose: 24 g daily. 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®) 5-30 gm/day granules**<br><br>**2-16 gm/day tablets** | ▸ fecal impaction<br>▸ GI bleed<br><br>**Monitor** LDL-C and TG levels. | *Granules:* Initially, 5 g 1 or 2 times daily. Increase dose in 5 gm intervals at 4-8 week intervals as tolerated. *Tablets:* Initially, 2 gms 1 to 2 times daily. Increase dose in 2 gm increments at 1 to 2 month intervals. Do not consume dry powder. Do not crush, cut, or chew. May cause constipation. May prevent absorption of folic acid & fat soluble vitamins (A-D-E-K). |
| **colesevelam (Welchol®) 2.5-4.375 gm/day** | ▸ none reported<br><br>**Monitor** LDL-C and TG levels. | The recommended starting dose is 3 (625 mg) tablets taken twice per day or 6 tablets once per day. Dose can be increased to 7 tablets, depending upon the desired therapeutic effect. Take with water and meals. Dosed once or twice daily. Monotherapy or combination with a statin. May cause constipation. May prevent absorption of folic acid & fat soluble vitamins (A-D-E-K). |
| **Niacin** | | |
| **niacin extended release tablets (Niaspan®)**<br><br>**500-2000 mg/day**<br><br>The BOP National Formulary is restricted to Niaspan® brand only. | ▸ arrhythmias<br>▸ hepatotoxicity<br>▸ peptic ulcer<br>▸ fulminant hepatic necrosis<br><br>**Monitor** ALT/AST (baseline, every 6-12 wks x 1 year; then every 6 months); Obtain uric acid and fasting glucose (baseline, at 6 wks, then annually). | Initiate at 500 mg at bedtime with a snack for one month and then increased to 1000 mg for one month. Subsequently the dose can be further titrated upward by 500 mg increments at a minimum of one-month intervals according to patient response. A maximum daily dose of 2000 mg should not be exceeded. Consult prescribing information. Escalate dose slowly and take aspirin or ibuprofen ½ hour before administration to decrease flushing. Take with meals. Avoid drinking hot drinks at time of dosing. Contraindicated with active peptic ulcer, alcoholism, unexplained increased LFT's, severe liver dysfunction. Use with caution with when there is a history of PUD, DM, gout, or decreased renal function. |

## Appendix 8. Specific Drug Treatment Options for Lipid Disorders[1,2]
**(page 3 of 4)**

| Medication/ Dosing Range | Toxicities/ Labs | Comments/ Drug Interactions |
|---|---|---|
| **Fibric Acids (continued)** | | |
| **gemfibrozil (Lopid®)**<br><br>**1200 mg/day** | ▸ myositis<br>▸ myopathy<br>▸ thrombocytopenia<br>▸ rhabdomyolysis<br>▸ hepatotoxicity<br>▸ pancreatitis<br>▸ cholelithiasis<br>▸ hypersensitivity<br>▸ cholestatic jaundice<br><br>**Monitor** serum lipids, CBC, LFT's, blood glucose. | Initial dose 600 mg twice daily, 30 minutes before morning and evening meals. Maximum dose 600 mg twice daily.<br>***Contraindications:*** hepatic or severe renal dysfunction, primary biliary cirrhosis, pre-existing gallbladder disease. Benefit must clearly outweigh risk if used during pregnancy.<br>***Drug interactions:*** Statins generally contraindicated due to increased risk of rhabdomyolosis and myopathy, which may occur acutely a few weeks to several months after combined therapy. (Periodic monitoring of creatinine kinase is not helpful in monitoring risk.) Anticoagulants may increase risk of bleeding (frequent monitoring needed). |
| **fenofibrate (Tricor®) (Antara®) (Lofibra®) (Triglide®)**<br><br>Dose range depends on brand. | ▸ pancreatitis<br>▸ cholelithiasis<br>▸ rhabdomyolysis<br>▸ hepatotoxicity<br>▸ hypersensitivity<br>▸ myopathy<br>▸ toxic epidermal necrolysis<br><br>**Monitor** serum lipids, CBC. | Dosing depends upon brand utilized. Refer to prescribing information. Recommended starting dose 43 or 67 mg depending on brand. Increase dose at 4 to 8 week intervals and only after evaluating effects on renal function and lipid levels. Take once daily with main meal. Adjust dose in patients with renal function impairment and the elderly. Reduce dose if lipids fall below target range. Discontinue after 2 months of maximum dose if inadequate response.<br>***Contraindications:*** hepatic or severe renal dysfunction, primary biliary cirrhosis, unexplained persistent liver function abnormality, pre-existing gallbladder disease. Benefit must clearly outweigh risk if used during pregnancy.<br>***Drug Interactions:*** Statins generally contraindicated due to increased risk of rhabdomyolosis and myopathy, that may occur acutely a few weeks to several months after combined therapy. (Periodic monitoring of creatinine kinase is not helpful in monitoring risk). Anticoagulants may increase risk of bleeding (frequent monitoring needed). Cyclosporine increases risk of nephrotoxicity (benefits must outweigh risks; use lowest possible dose). Fenofibrate should be administered at least 1 hour before or 4-6 hours after bile acid sequestrant due to potential blocking of absorption. |

## Appendix 8. Specific Drug Treatment Options for Lipid Disorders[1,2]
**(page 4 of 4)**

| Medication/ Dosing Range | Toxicities/ Labs | Drug Interactions / Comments |
|---|---|---|
| **Cholesterol Absorption Inhibitor** | | |
| **ezetimibe (Zetia®)**<br><br>**10 mg/day** | ▸ low back pain<br>Follow monitoring recommendations for the statin being prescribed with it. | Dose 10 mg once daily. Inhibits the absorption of dietary and biliary cholesterol decreasing the intestinal absorption by >50%; does not affect triglyceride absorption. Ezetimibe is considered a third-line agent, to be utilized only if a statin in combination with niacin or bile acid sequestrants does not achieve treatment goals or if those drugs are not tolerated or are contraindicated. It is indicated for treatment intensification in combination with a statin for primary hypercholesterolemia. Not recommended in moderate to severe hepatic insufficiency. Administer at least 2 hours before or 4 hours after bile acid sequestrant. May administer without regard to meals.<br>***Drug Interactions***: Cyclosporine may significantly increase ezetimibe levels (~12-fold). (Monitor carefully.) Gemfibrozil and fenofibrate increase ezetimibe bioavailability. Use with caution with fibrates (may lead to cholelithiasis). Cholestyramine decreases ezetimibe AUC >50%. |

## Appendix 9. Comparison of HMG-CoA Reductase Inhibitors (Statins)

| Drug/ Equivalent Dose | Doses of Each Drug | Effect on Lipids (% change from baseline) | | | |
|---|---|---|---|---|---|
| | | LDL | TC | TG | HDL |
| **Atorvastatin** *Lipitor®* **10 mg** | 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** *Lescol®* **40 mg** | 20 mg | ⇓17-22 | ⇓13 | ⇓5 | ⇑1 |
| | 40 mg | ⇓23-27 | ⇓18-22 | ⇓10-20 | ⇑4-8 |
| | 80 mg | ⇓33-36 | ⇓27 | ⇓15-25 | ⇑4-8 |
| **Lovastatin** *Mevacor®* **20 mg** | 20 mg | ⇓25-29 | ⇓18-22 | ⇓12-13 | ⇑6-8 |
| | 40 mg | ⇓31-34 | ⇓23-27 | ⇓2-10 | ⇑5 |
| | 80 mg | ⇓41-48 | ⇓32-36 | ⇓13-15 | ⇑4-8 |
| **Pravastatin** *Pravachol®* **20 mg** | 10 mg | ⇓19-22 | ⇓13-16 | ⇓3-15 | ⇑7-10 |
| | 20 mg | ⇓24-32 | ⇓18-24 | ⇓11-15 | ⇑2-3 |
| | 40 mg | ⇓33-34 | ⇓24-27 | ⇓10-24 | ⇑6-12 |
| | 80 mg | ⇓37 | ⇓27 | ⇓19 | ⇑3 |
| **Simvastatin** *Zocor®* **10 mg** | 10 mg | ⇓28-30 | ⇓21-23 | ⇓12-15 | ⇑7-12 |
| | 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 | ⇑7-16 |
| **Rosuvastatin** *Crestor®* **5 mg** | 5mg | ⇓43 | ⇓24-33 | ⇓21-35 | ⇑3-13 |
| | 10mg | ⇓50 | ⇓36-40 | ⇓10-37 | ⇑8-14 |
| | 20mg | ⇓53 | ⇓34-40 | ⇓23-37 | ⇑8-22 |
| | 40 mg | ⇓62 | ⇓40-46 | ⇓28-43 | ⇑10-17 |

Adapted from Pharmacist's Letter August 2003; Vol. 19: Detail document #190801. 2004. Wolters Kluwer Health, Inc. Drug Facts and Comparisons. St. Louis, MO. Facts and Comparisons Inc, online efacts; accessed 1-09-06.

# Appendix 10. Health Education Guide on Lipid Disorders
**(page 1 of 3)**

## 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 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 these 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 a person already has 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 less than 100 mg/dL for patients with heart disease and diabetes, with some experts recommending of goal of less than 70 mg/dL. The target LDL cholesterol for healthy people 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 less than 200 mg/dL.
- **Triglycerides** are a storage form of fat, so high levels of triglycerides are not normally in the blood. Elevated triglyceride levels are associated with heart disease and diabetes. A normal triglyceride level is less than 200 mg/dL.

## Appendix 10. Health Education Guide on Lipid Disorders
**(page 2 of 3)**

### Treatment: Lifestyle changes

The first approach to treating high cholesterol is changing certain habits. Many people are able to control cholesterol levels just by changing diet, losing weight, and participating in an exercise program. Quite 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 a person has another disease 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. Below are some actions you can take to address high cholesterol levels.

**Quit smoking.**

Smoking causes lung cancer, but it's 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. Smoking is thought to cause the damage to blood vessels which allows cholesterol to stick to their walls. 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.

**Improve your diet.**

Follow recommendations for a low fat/low cholesterol diet.

**Carefully manage your diabetes.**

Out-of-control blood sugar can increase triglycerides and cholesterol levels.

**Lose weight (if you are 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 you lose weight, 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.

## Appendix 10. Health Education Guide on Lipid Disorders
**(page 3 of 3)**

### Treatment: Medications

If lifestyle changes are not completely effective, medications are prescribed to treat high cholesterol. 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.

- **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 30-60 minutes before taking niacin. Avoid drinking hot drinks around the time you take niacin.

- **Bile acid resins** lower LDL cholesterol by combining with bile acids in the gut. These drugs often cause constipation, so they need to be taken with plenty of water.

- **Fibrates** lower triglyceride levels and raise HDL, but have little effect on lowering LDL cholesterol. They are taken twice a day before meals.

- **Ezetimibe** lowers cholesterol by interfering with the absorption of dietary cholesterol. It is rarely prescribed and is usually given when other medications do not effectively lower LDL.

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 the leading cause of heart disease.

- **Work at keeping your cholesterol levels down.** Once cholesterol levels are brought under control, resist returning to any 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 positive action, and play an active role in staying healthy.

## Appendix 11. Inmate Fact Sheet on High Cholesterol

- **Anyone can have high cholesterol**, regardless of sex, 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.**
- **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:**
  - Improve your lifestyle by changing your diet, exercising regularly, quitting smoking, and controlling high blood pressure or diabetes, if you have them.
  - 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.**

## Appendix 12. Management of Lipid Disorders: Resources

### National Institutes of Health

**Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III).** Available at:
http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm

**National Cholesterol Education Project. ATP III Guidelines At-A-Glance Quick Desk Reference.** Available at:
http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.pdf

**Risk Assessment Tool for Estimating 10-Year Risk of Developing Hard CHD**
Available at:
http://hin.nhlbi.nih.gov/atpiii/calculator.asp?usertype=prof

### Centers for Disease Control and Prevention

**Body Mass Index Calculator.** Available at:
www.cdc.gov/nccdphp/dnpa/bmi/calc-bmi.htm

### American College of Cardiology

www.acc.org

### American Heart Association

www.americanheart.org