KEY ELEMENTS

1. Base recommendations on high quality evidence with a focus on interventions that improve clinically significant patient-centered outcomes.


3. Use specific screening criteria to identify the patient with dyslipidemia who is most likely to benefit from appropriate intervention.


5. Use lipid lowering therapies to reduce cardiovascular risk and events that include:
   a. Evidence driven rationale for medication choices
   b. Lifestyle modification and diet with appropriate intensity


7. Define treatment goals.

8. Clarify contribution of triglycerides (TG) and HDL-C to cardiovascular disease (CVD) risk.
NON-LIPID CV RISK FACTORS [D]

Sidebar 1

Non-Lipid CV Risk Factors [D]
- Age
- Male Gender
- Family history of premature CVD
- Hypertension
- Smoking
- Diabetes mellitus
- Abdominal obesity

Sidebar 2

Lipid Profile Test [G]
- TC/HDL/TG can be measured directly, LDL-C value is calculated by formula and may be inaccurate in high TG.
- Consider direct LDL measurement if TG > 400 mg/dL and cannot be reduced with diet and exercise.

Sidebar 3

Screening Threshold Levels [H]
- Total cholesterol \( \geq 240 \text{ mg/dL} \)
- HDL cholesterol \(< 40 \text{ mg/dL} \)
- Triglycerides \( \geq 200 \text{ mg/dL} \)
- LDL cholesterol \( \geq 130 \text{ mg/dL} \)

Sidebar 4

Healthy Lifestyle Modification [I]
- Smoking cessation
- Healthy diet
- Increase physical activity
- Weight loss, if indicated
- Reduce excessive alcohol use

Total cholesterol
- HDL cholesterol
- Triglycerides
- LDL cholesterol

CVD - Cardiovascular Disease
TC - Total Cholesterol
HDL-C - High Density Lipoprotein Cholesterol
LDL-C - Low Density Lipoprotein Cholesterol
TG - Triglycerides
Module A - Management of Dyslipidemia - Screening

A LEVEL OF EVIDENCE

Annotation A
1. This guideline addresses adults (age 17 years or older) eligible for care in the Veterans Health Administration/Department of Defense (VHA/DoD) healthcare systems.

Annotation B
2. All patients with known CVD are considered high-risk and should be treated with aggressive lipid-lowering therapy to prevent acute vascular events. These include, but are not limited to, acute myocardial infarction (AMI) or cerebrovascular accident (CVA). [A]

Annotation C
3. Patients with Type 2 Diabetes Mellitus (DM) are at significant increased risk of CVD compared with non-diabetic patients of similar age and should, therefore, be treated more aggressively according to secondary prevention protocols. [A]

Annotation D
4. Patients screened for dyslipidemia should be assessed for risk factors for CVD. Assessment should include, but not be limited, to the following:
   4.1. Age (males 45 years or older and females age 55 or older).
   4.2. Family history of premature coronary artery disease; definite myocardial infarction (MI) or sudden death before age 55 in father or other male first-degree relative, or before age 65 in mother or other female first-degree relative.
   4.3. Current tobacco use/cigarette smoking (or within the last month).
   4.4. Hypertension (systolic blood pressure greater than or equal to 140 mmHg or diastolic blood pressure greater than or equal to 90 mmHg confirmed on more than one occasion, or current therapy with anti-hypertensive medications).
   4.5. Diabetes mellitus (DM) (elevated fasting blood sugar (greater than or equal to 126 mg/dL), or a random blood sugar (greater than or equal to 200 mg/dL) confirmed on more than one occasion, an abnormal glucose tolerance test or current therapy with anti-diabetic medications).
   4.6. Level of HDL-C (less than 40 mg/dL confirmed on more than one occasion).
5. In obese patients (BMI greater than or equal to 30), waist circumference measurement should be obtained to assist in the diagnosis of metabolic syndrome.
### Annotation E

6. **Fasting lipid profile testing should be obtained in the following:**

   6.1. All men age 35 years or older and women age 45 years or older every 5 years. [A]

   6.2. Adults with a family history or clinical evidence of familial hyperlipidemia. [A]

   6.3. Younger adults (men younger than age 35 and women younger than age 45) if they have one or more of the following risk factors: family history of premature CVD, hypertension (or under treatment for hypertension), or smoking. [B]

   6.4. Adults with abdominal obesity (waist circumference greater than 40 inches in men and greater than 35 inches in women) to aid in assessment of metabolic syndrome. [B]

   6.5. Elderly patients age 75 or older if they have multiple CVD risk factors, or a history of CVD and good quality of life with no other major life-limiting diseases. [I]

7. **Lipid profile should NOT be obtained in the following:**

   7.1. Within 8 weeks of acute hospitalization, surgery, trauma, or infection unless they are obtained within 12-24 hours of the event to ensure accuracy. [I]

   7.2. Pregnant women until three to four months post partum. [I]

### Annotation F

8. Clinical decisions should be based upon lipid profiles done 1 to 8 weeks apart (fasting) with an LDL-C or total cholesterol (TC) difference of less than 30 mg/dL. [I]

9. If level of TG is above 400 mg/dL, the calculated LDL-C level cannot be used for decision about treatment. In these cases:

   9.1. If TG levels can be brought to less than 400 mg/dL by dietary or other interventions, then Friedwald’s formula can be used to calculate a more exact LDL-C level. [C]

   9.2. If TGs cannot be brought to levels less than 400 mg/dL, then consider measuring LDL-C directly, or estimate the LDL-C using the following equation: [I]

     \[
     \text{Estimated LDL-C} = (\text{TC} - \text{HDL}) - 30
     \]

   9.3. Screen and treat common causes of elevated TGs: fatty diet, high carbohydrate diets, alcohol use, hypothyroidism, and hyperglycemia. [B]

   9.4. In the absence of secondary causes, the first-line therapy for elevated TGs should be therapeutic life-style changes. [C]
10. Patients with LDL greater than 130 mg/dL, HDL less than 40 mg/dL, or TG greater than 200 mg/dL should be assessed for further management of dyslipidemia. [ C ]

11. All adults should be encouraged to adopt healthy lifestyles that may reduce the risk of cardiovascular disease, to include:
   
   11.1. Tobacco cessation interventions offered to all smokers [ A ]
   
   11.2. Healthy diet [ B ]
   
   11.3. Engage in 30 minutes or more of moderate intensity physical activity (to expend 200 Kcal) on most days of the week [ B ]

12. Patients with average or below average risk for atherosclerotic events should be screened for dyslipidemia every five years. [ B ]
Management of Dyslipidemia

Module B: Initiation of Therapy

1. Patient with abnormal lipid profile or history of CVD, DM [K]

2. Obtain history and physical examination, lab tests
   Assess for secondary causes, familial disorder and comorbidity [L1]
   Obtain baseline liver function test [L2]

3. Patient already on lipid lowering therapy?

4. History of ACS in past 6 months? [M1]

5. Ensure patient is on statin therapy and TLC (see VA/DoD IHD guideline)

6. History of CVD or DM and LDL-C above goal? [M2]

7. Initiate lipid lowering therapy: [O]
   Drug therapy [Q1]
   Therapeutic Lifestyle Changes [P]
   Check profile lipids 6-12 wks
   Assess for adverse effects

8. Calculate 10-year CVD risk score [M3]
   (or add up risk factors)
   Determine goal of therapy based on CVD risk [N]

9. Is LDL above goal, based on CV risk?

10. Initiate therapeutic lifestyle change (TLC), diet and exercise for 3-6 months [P]

11. Patient responds to therapy or LDL-C at goal?

12. Initiate drug therapy [Q1]
    Check lipid profile in 6-12 weeks
    Assess for adverse effects

13. Reinforce lifestyle education
    Smoking, MNT, and Exercise [I]
    Address CV risk or comorbidities

14. Repeat dyslipidemia evaluation in one to two years [R]

See sidebar 5

Secondary Prevention

Sidebar 5

Lipid Lowering Therapy in Patients with CVD or CVD Equivalent (Secondary Prevention)

<table>
<thead>
<tr>
<th>LDL-C (mg/dl)</th>
<th>TLC</th>
<th>Drug</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recent ACS</td>
<td>All</td>
<td>&gt;100</td>
<td>&lt;100</td>
</tr>
<tr>
<td>CVD</td>
<td>All</td>
<td>≥130</td>
<td>≤130</td>
</tr>
<tr>
<td>DM no RF</td>
<td>All</td>
<td>&gt;130</td>
<td>≤130</td>
</tr>
</tbody>
</table>

* Optional <70 mg/dL
** Optional 100-129 mg/dL

Primary Prevention

Sidebar 6

Lipid Lowering Therapy in patients based on 10-year CV risk (Primary Prevention)

<table>
<thead>
<tr>
<th>Risk</th>
<th>10-year Risk</th>
<th>TLC</th>
<th>Drug</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>High &gt;2+RF</td>
<td>&gt;20%</td>
<td>All</td>
<td>≥130</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Mod ≥2+RF</td>
<td>15-20%</td>
<td>All</td>
<td>≥130</td>
<td>&lt;130</td>
</tr>
<tr>
<td>Low 0-1 RF</td>
<td>&lt;10%</td>
<td>All</td>
<td>≥190</td>
<td>&lt;160</td>
</tr>
</tbody>
</table>

* (or HDL-C < 40) Optional LDL-C 100-129

Management of Dyslipidemia Follow-up continue on Algorithm C
MODULE B - MANAGEMENT OF DYSLIPIDEMIA – INITIATION OF THERAPY

ANNOTATION K

13. Patients managed by this guideline algorithm have abnormal lipid profiles (dyslipidemia) or evidence of cardiovascular disease (CVD) or diabetes.

ANNOTATION L

14. Adults with abnormal lipid profiles (dyslipidemia) should be assessed for secondary causes, familial disorders, and other underlying conditions that may influence lipid levels. [1]

15. Assessment for secondary causes should be based on medical history, physical examination and laboratory tests to include: (See Table 1)

15.1. Measurement of serum thyroid-stimulating hormone (TSH), BUN/creatinine, liver function tests (LFTs), and a dipstick urinalysis should be obtained to exclude hypothyroidism, chronic renal failure, obstructive liver disease, and nephrotic syndrome conditions. [1]

15.2. If dipstick urine protein is greater than 1+ (detected in two urine tests), nephrotic syndrome as a secondary cause of elevated LDL-C should be ruled out. [1]

15.3. Serum lipids should be assayed six to eight weeks post-TSH normalization to determine the need for additional treatment. [1]

15.4. Patients with hypertriglyceridemia should be evaluated for alcohol use, diabetes, and hypothyroidism. Addressing these underlying conditions can improve or normalize TG levels, and failure to address them can render therapy ineffective. [1]

15.5. Lipid levels in patients treated for secondary hyperlipidemia should be repeated 6-8 weeks post correction of the underlying disorder.

15.6. Family members of patients presenting with very severe hypercholesterolemia should be screened to detect other candidates for therapy.

15.7. Consider consulting with a specialist to assist the primary care clinician in co-managing patients with familial disorders who do not respond to therapy. [1]
SECONDARY PREVENTION (See Table 3)

ANNOTATION - M1

*Patients with History of Acute Coronary Syndrome in Past 6 Months*

16. A lipid panel should be drawn at the time of admission for all patients with suspected acute coronary syndrome (ACS). [ C ]

17. Initiation of moderate- to high-dose statin prior to hospital discharge may be considered in patients admitted with ACS irrespective of their lipid profile. [ B ]

18. Patients with recent ACS (within the past 6 months) should be on a moderate dose of statin therapy to reduce LDL-C level below 100 mg/dL. [ A ]

19. A lower target (less than 70 mg/dL) may be considered for high-risk patients. [ B ]

ANNOTATION M2 -

*Patients with History of CHD or CVD equivalent (DM with or without other risk factors)*

20. LDL should be lowered to less than 100 mg/dL for patients with previous documented CHD or CVD equivalent (DM with other major risk factors) for secondary prevention. [ A ]

21. LDL should be lowered to less than 130 mg/dL for patients with DM without other major risk factors for secondary prevention. [ C ]

ANNOTATION M3 - Primary Prevention (See Table 3)

22. Goals of lipid lowering therapy should be tailored to risk level and based upon the balance between benefits, risks, and patient preferences. [ C ]

23. A global 10-year risk for CVD should be calculated to assess the short-term (10-year) absolute risk of a CVD event. [ A ]

24. The Framingham Risk Calculator should be used, as it is the most commonly used and readily available calculator validated in numerous populations. [ I ]


25. Other risk markers or measure of atherosclerotic burden may be useful to adjust the risk category, if they have been validated to have independent prognostic value. [ C ]

ANNOTATION N

26. LDL-C should be lowered to less than 100 mg/dL for patients with high 10-year risk greater than 20 percent. [ B ]

27. LDL-C should be lowered to less than 130 mg/dL for patients with intermediate 10-year risk (15-20 percent). [ B ]
28. LDL-C should be lowered to less than 130 mg/dL for patients with intermediate 10-year risk (10-14 percent). [ C ]

29. LDL-C should be lowered to less than 160 mg/dL for patients with low 10-year risk. [ I ]

30. LDL-C reduction of 30-40 percent from baseline may be considered an alternative therapeutic strategy for patients who can not meet the above goal.

ANNOTATION R

31. If the initial dyslipidemia screening reveals TC greater than 200 mg/dL, or fasting LDL-C greater than 130 mg/dL or HDL-C less than 40 mg/dL, but LDL-C level is under the recommended goal level based upon CV risk, the patient will be at low-risk for lipid-related events over a one to two-year period and thus, should be reevaluated for dyslipidemia in one to two years.

LIPID LOWERING THERAPY TO ACHIEVE GOAL (See Figure 1)

32. Appropriate lipid lowering therapy should be initiated based on LDL-C baseline level and other risk factors for CVD.

Non-Pharmacologic Therapy

ANNOTATION P

33. Therapeutic lifestyle changes (TLC) should be recommended for ALL patients with dyslipidemia, regardless of risk or baseline LDL-C level. [ C ]

34. For secondary prevention of recurrent CVD events, non-pharmacologic therapy is always indicated, but it should not delay appropriate pharmacotherapy.

35. Emphasis on TLC is an important component of primary prevention and is effective in reducing CVD risk by lowering LDL-C and blood pressure. [ B ]

36. Diet intervention should be the first step in lipid lowering therapy. [ B ]

37. Patients whose initial treatment is TLC should be given 3-6 months of dietary therapy prior to beginning medication and longer, if lipids are improving and nearing LDL thresholds. [ B ]

38. TLC is provided in a step-wise approach focused on initiating TLC components and followed by subsequent evaluation of the effect on LDL-C and moving to intensify MNT as indicated.
39. Medical Nutrition Therapy (MNT) should include: (See Table 4, Table 5)

39.1. Initial diet should focus on reduction of saturated fats to less than 7 percent of total calories and dietary cholesterol to less than 200 mg/day similar in composition to the TLC diet (formerly Step II diet). [B]

- The range of 25-35 percent of total calories from fat is to be paired with keeping saturated fats and trans-fatty acid percents of total calories low.
- Advise 10 percent monounsaturated fat, less than 7 percent saturated fat, and less than 200 mg cholesterol diet.
- If TGs are elevated ensure that blood glucose is under control, limit alcohol and simple sugars, and evaluate need for weight loss. Emphasis should be placed on weight reduction and physical activity.
- Limit foods with trans fatty acids (e.g., stick margarine, shortening, and commercially baked products and processed food).
- Select greater than 5-6 servings/day of fruits and vegetables and six servings/day of whole-grain products.

39.2. Patient’s specific diet should be individualized based on nutrition assessment, other CVD risk factors, other disease conditions, and patient’s lifestyle. [I]

39.3. Patients should be evaluated 4-6 weeks after their initial consultation. A lipid profile and anthropometric data should be analyzed. Further dietary intervention may include: [B]

- Increase soluble (viscous) fiber to 10-25 g/day to lower LDL-C
- Increase plant sterols/stanols to 2 g/day to lower LDL-C
- Include nuts such as walnuts and almonds (1 oz. ~5 times/week) and soy protein (25g/day or 8 oz. of tofu) to lower LDL-C
- Select fatty fish (average of 7 oz./week) (fish oil) to lower TG.

39.4. Weight management for overweight and obese patients should be encouraged to lower LDL-C and TG and to reduce CV risk. [B]

39.5. Patients in whom TGs greater than 500 mg/dL should receive strict diet therapy including avoidance of alcohol, restriction of dietary fat, and avoidance of concentrated carbohydrates (sweets). For TGs greater than 1000 mg/dL a very low fat diet should be instituted quickly to reduce chylomicronemia and risk of acute pancreatitis.

39.6. Patients with evidence of metabolic syndrome should receive MNT that incorporates the additional protocol for weight management with increased physical activity. [B]
40. Physical Activity / Exercise and Weight Control

40.1. Moderate intensity levels of physical activity should be performed for at least 30 minutes most, preferably all, days of the week. [B]

40.2. In patients with CVD, aerobic exercise should not precipitate angina.

40.3. Increased physical activity through lifestyle change should be encouraged, as it is equally as effective as structured exercise in reducing body fat, improving cardiorespiratory fitness and improving cardiovascular risk factors. [B]

40.4. Physical activity, through lifestyle change or structured exercise, should be encouraged to maintain weight control (or weight loss if overweight or obese), to improve insulin resistance, and increase HDL-C. [B]

PHARMACOTHERAPY (See Table 3, Table 7)

Drug Therapy for Secondary Prevention

ANNOTATION O

41. All patients with a recent ACS should be on at least a moderate dose of statin therapy. [A]

42. Statin drug therapy should be initiated for patients with previous documented CHD or CVD equivalent (diabetes with other major risk factors) if baseline LDL-C is greater than or equal to 100 mg/dL. [A]

43. Statin drug therapy should be initiated for patients with documented DM with no major risk factors if baseline LDL-C is greater than or equal to 130 mg/dL. [C]

44. Statin drug therapy may be considered optional for all patients with CHD or CVD equivalent (diabetes with other major risk factors) regardless of LDL-C baseline. [B]

Drug Therapy for Primary Prevention

45. Drug therapy should be initiated for high-risk patients (10-year risk greater than 20%) if baseline LDL is greater than or equal to 130 mg/dL. [B]

46. Drug therapy is optional to consider in high-risk patients (10-year risk greater than 20%) if baseline LDL is 100-129 mg/dL. [B]

47. Drug therapy may be offered to patients with high-intermediate risk (10-year risk 15-20%) if baseline LDL is greater than or equal to 130 mg/dL. [B]

48. Drug therapy may be offered to patients with low-intermediate risk (10-year risk 10-14%) if baseline LDL is greater than or equal to 160 mg/dL. [C]
49. Drug therapy may be offered to low-risk patients (10-year risk less than 10%) if baseline LDL is greater than or equal to 190 mg/dL. [1]

**Elevated LDL-C - Monotherapy**

**ANNOTATION Q1**

50. Statins are first line agents in primary and secondary prevention of CVD regardless of HDL-C or TG level. [A]

51. Moderate doses of formulary statins (to achieve an LDL-C reduction of 25% or greater) should be initiated unless a patient is considered to be at greater than usual risk for adverse events from statins (e.g., myopathy). [A]

52. For patients who cannot tolerate statins, niacin or resins should be considered for treatment. [A]

53. There is insufficient clinical outcome evidence to recommend ezetimibe monotherapy for reduction of CV risk. [I]

54. Ezetimibe can be considered for lowering LDL-C in patients who are unable to tolerate other lipid-lowering drugs. [A]

55. The dose of statin should be adjusted at 6 to 12 week intervals until individual LDL-C goals are achieved or statin doses have been maximized. [I]

**Isolated Hypertriglyceridemia**

56. Niacin, fibrates, or fish oil supplements may be used in treatment of isolated hypertriglyceridemia. [B]

**Isolated Low HDL-C**

57. For secondary prevention Gemfibrozil or niacin may be used in patients with isolated low HDL-C and normal LDL-C. [A-Gemfibrozil; B-Niacin]

**SAFETY AND FOLLOW-UP**

**ANNOTATION L2**

58. Baseline serum transaminase (ALT/AST) should be obtained prior to starting lipid-lowering therapy. [I]

59. Level of serum transaminase (ALT/AST) should be obtained after 6-12 weeks following initiation of treatment, any elevation in dose, and combination, and annually thereafter in patients receiving statins, fibrates, or niacin. [I]
60. Creatine kinase (CK) levels should be obtained in patients who develop muscle pain, weakness, or tenderness after institution of statin or fibrate therapy. [1]

61. Patients treated with statins or fibrates should be educated regarding the importance of recognizing and reporting any unexplained muscle tenderness, pain, or weakness. [1]

62. Lipid profiles should be repeated 6-12 weeks after initiation of therapy and/or change in dose and/or combination therapy. [B]
Follow-up patient for lipid lowering therapy

15. LDL-C at goal?
   Y
   - Patient responds to therapy or LDL-C at goal?
     Y
     - Is TG level elevated, or low HDL-C or metabolic syndrome? (T)
       Y
       - Intensify TLC and consider drug therapy for Elevated TG [U]
       - Low HDL-C [V]
       - Metabolic Syndrome [W]
     N
     - Reschedule lipids evaluation at appropriate frequency and follow-up to maintain goals [X]
   N
   - Address adherence [S]
     - 1. Intensify MNT & exercise [P]
     - 2. Modify drug/dose, consider combination therapy [Q2]
     - 3. Consider consultation/referral
     - Reschedule lipids evaluation at appropriate frequency and follow-up until goals are met [X]

Sidebar 4

Healthy Lifestyle Modification

- Smoking cessation
- Healthy diet
- Exercise/physical activity
- Weight loss, if indicated
- Reduce excessive alcohol use

CVD - Cardiovascular Disease
DM - Diabetes Mellitus
MNT - Medical Nutrition Therapy
TLC - Therapeutic Lifestyle Changes
HDL-C - High Density Lipoprotein Cholesterol
LDL-C - Low Density Lipoprotein Cholesterol
TG - Triglycerides
**Module C - Management of Dyslipidemia – Follow-up Therapy Recommendations**

*Adherence to Therapy*

**Annotation S**

63. The causes for inadequate response to therapy following dose or stepwise titration should be identified.

64. Adherence to therapy should be assessed at every visit, through history, pill count, and/or administrative records especially if therapeutic goals have not been reached. [I]

65. Adherence to lipid-lowering medication regimens may be improved by a multi-pronged approach [I] including:
   
   a. Evaluation of medication side effects
   
   b. Simplifying medication regimens to incorporate patient preference
   
   c. Addressing barriers for obtaining the medications (administrative, economic, etc.)
   
   d. Coordination with other healthcare team members to improve monitoring of adherence with prescriptions of pharmacological and lifestyle modification
   
   e. Patient and family education about their disease/treatment regimens
   
   f. Evaluation for depression

**LDL-C Lowering Combination Therapy**

[Only for secondary prevention] (See Table 8)

**Annotation Q2**

66. For patients not at goal, monotherapy should be titrated until goal is achieved or maximum tolerable dose has been reached. [C]

67. Combination therapy to achieve LDL-C goal may be considered for carefully selected patients who do not achieve the LDL-C goal with maximally tolerated monotherapy. [I]

68. Combination lipid-lowering therapy should include a statin unless the patient is unable to tolerate statins. [A]

69. Addition of a resin to the statin can be considered for secondary prevention in patients not meeting their LDL-C goals on maximally tolerated doses of statins. [B]

70. Addition of niacin or a resin to the statin can be considered in patients not meeting their LDL-C goals to further reduce the LDL-C level. [B]
71. Addition of ezetimibe to the statin can be considered in patients not meeting their LDL-C goals on maximally tolerated doses of statins and unable to tolerate niacin or a resin to reduce the LDL-C level. [I]

72. In patients unable to tolerate statins and not achieving their LDL-C goals with niacin or resins, a combination of both resin and niacin may be considered. [B]

73. In any combination therapy the lowest possible dose of statin should be used to achieve lipid goals. When combined with fibrates (greatest risk), niacin or possibly ezetimibe, the risk of adverse events with statins (e.g., muscle toxicity) appears to increase with increasing statin doses. [C]

74. Combination therapy with statins and niacin, fish oils or fibrates can be considered for the secondary prevention of CVD in patients with elevated LDL-C and very high TGs. [C]

75. Combination therapy with niacin and fibrates can be considered for the secondary prevention of CVD in patients with elevated LDL-C and very high TGs in patients unable to tolerate statins. [C]

76. For secondary prevention of CVD in patients with either low HDL-C or very high TGs and no elevation of LDL-C levels, combination therapy with statin plus niacin, fibrate or fish oil may be considered. [C]

77. Combination therapy with niacin and fibrates and/or fish oils can be considered in patients unable to tolerate statins. [C]

Patient with Elevated TG Level, or Low HDL-C Level, or Metabolic Syndrome

ANNOTATION T -

78. Once the LDL-C goal has been reached, treatment attention may shift to obtain optimal lipoprotein profiles.

High Level of Triglycerides (See Table 9, See Box 7)

ANNOTATION U

79. Patients with elevated TG (greater than or equal to 200 mg/dL) should have a repeat fasting lipid profile, and if persistent receive intensive MNT, an appropriate exercise program, and be screened for underlying causes. [B]

80. Drug therapy may be considered in patients with very high TG levels (greater than or equal to 500 mg/dL) that do not respond to lifestyle interventions and the treatment of underlying causes of elevated TG, for the purpose of preventing pancreatitis. [I]
81. Effective drugs for lowering hypertriglyceridemia include: fibrates, niacin, and fish oil. [B]

Low Level of HDL-C (See Table 10)

ANNOTATION V

82. Patients with CVD who have low HDL-C (less than 40 mg/dL), TG greater than 200 mg/dL and normal levels of LDL-C may benefit from gemfibrozil therapy. [A]

83. Lifestyle modifications, including weight loss, exercise, and smoking cessation should be given high priority in the therapeutic plan for patients with low HDL-C. [B]

84. CVD patients with low HDL-C (less than 40 mg/dL) may be considered for treatment with niacin. [B]

Evaluation and Treatment of Metabolic Syndrome (See Box 8)

ANNOTATION W

85. TLC should be initiated for patients diagnosed with metabolic syndrome. [B]

86. Lifestyle modification for weight reduction through diet and increased physical activity is indicated for patients diagnosed with metabolic syndrome. [B]

87. Drug therapy to alter insulin resistance or low HDL-C or elevated TG has not been demonstrated to improve CVD outcomes in patients with metabolic syndrome and as such, clinicians will have to individualize therapy. [I]

Follow-up

ANNOTATION X

88. Lipid profiles should be reevaluated after at least 6-12 weeks of drug therapy or change in dose or after at least three to six months of dietary therapy to document efficacy, identify adverse effects, and to titrate medication dose. [I]

89. Follow-up visits should include [I]:

- Patient history
- Physical exam
- Laboratory tests
- Documentation of adverse events
90. Once the goal is achieved, therapy for dyslipidemia should be continued to maintain the goal. Treatment of dyslipidemia is a lifelong process; however, adjustments may be necessary if the patient develops medical conditions that affect the severity of comorbidity or life expectancy.

ANNOTATION Y

91. Lipid evaluations should be repeated at least annually. [1]
## TABLES

Table 1. Secondary Causes of Lipid Abnormalities

Table 2. Lipid Screening Criteria

Table 3. Summary of Dyslipidemia Therapy Thresholds and Goals

Table 4. Essential Components of Therapeutic Lifestyle Changes (TLC)

Table 5. Macronutrients Recommendations for TLC Diet

Table 6. Dyslipidemia Drug Therapy

Table 7. Doses of Currently Available Statins Required to Attain an Approximate 30% to 40% Reduction of LDL-C Levels (Standard Doses)*

Table 8. Potential Combination Pharmacological Treatments for Dyslipidemia

Table 9. Drug Treatment for Hypertriglyceridemia

Table 10. Drug Treatment for Isolated Low HDL-C
### Table 1. Secondary Causes of Lipid Abnormalities

<table>
<thead>
<tr>
<th>Disorder/Patient Characteristic</th>
<th>Effect on Lipids</th>
<th>Laboratory Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic renal failure/post renal transplantation</td>
<td>↑ TG, ↑ TC, ↓ HDL-C</td>
<td>Scr</td>
</tr>
<tr>
<td>DM</td>
<td>↑ TG, ↑ TC, ↓ HDL-C</td>
<td>Glucose, HbA1c</td>
</tr>
<tr>
<td>Ethanol use</td>
<td>↑ TG, ↑ HDL-C</td>
<td>—</td>
</tr>
<tr>
<td>HIV/AIDS Wasting</td>
<td>↑ TG, ↓ TC, ↓ HDL-C, ↓ LDL-C</td>
<td>—</td>
</tr>
<tr>
<td>HIV/AIDS (HAART)</td>
<td>↑ TG, ↑ TC, ↑ LDL-C</td>
<td>—</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>↑ TG, ↑ TC, ↑ LDL-C</td>
<td>TSH</td>
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<td>Inactivity</td>
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<td>↑ TC, ↑ LDL-C</td>
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<tr>
<td>Obesity</td>
<td>↑ TG, ↓ HDL-C</td>
<td>—</td>
</tr>
<tr>
<td>Obstructive liver disease</td>
<td>↑ TC</td>
<td>LFTs (Alkaline phosphatase, total bilirubin)</td>
</tr>
<tr>
<td>Estrogen therapy</td>
<td>↑ TG, ↓ LDL-C, ↑ HDL-C</td>
<td>—</td>
</tr>
<tr>
<td>Medications</td>
<td>Variable</td>
<td>—</td>
</tr>
</tbody>
</table>

AIDS = acquired immune deficiency syndrome; DM = diabetes mellitus; HAART = Highly Active AntiRetroviral Therapy; HbA1c = glycosylated hemoglobin; HDL-C = high-density lipoprotein cholesterol; HIV = human immunodeficiency virus; LDL-C = low-density lipoprotein cholesterol; LFTs = liver function tests; Scr = serum creatinine; TC = total cholesterol; TG = triglycerides; TSH = thyroid-stimulating hormone.

### Box 2. Lipid Screening Criteria

a. Male age 35 or older OR
b. Female age 45 or older OR
c. Young adults with more than one of the following:
   - Family history of premature CVD
   - Patient is smoking
   - Patient has or is being treated for hypertension
d. Consider obtaining lipid profile for young adults with abdominal obesity
### Table 3. Summary of Dyslipidemia Therapy Thresholds and Goals

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>Disease Status or Risk Factors</th>
<th>Calculated 10-Year Risk</th>
<th>TLC</th>
<th>LDL-C Level for Considering Statin Drug Therapy</th>
<th>LDL Goal of Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Secondary Prevention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very high</td>
<td>Recent ACS</td>
<td>N/A</td>
<td>All</td>
<td>All</td>
<td>&lt;100 mg/dL</td>
</tr>
<tr>
<td></td>
<td>CHD or DM with other risk factors</td>
<td>N/A</td>
<td>All</td>
<td>≥100 mg/dL</td>
<td>&lt;100 mg/dL</td>
</tr>
<tr>
<td></td>
<td>DM with no other risk factors</td>
<td>N/A</td>
<td>All</td>
<td>≥130 mg/dL (or HDL &lt;40) 100-129 optional</td>
<td>&lt;130 mg/dL</td>
</tr>
<tr>
<td><strong>Primary Prevention</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>More than 2 RF</td>
<td>≥20%</td>
<td>All</td>
<td>≥130 mg/dL</td>
<td>&lt;100 mg/dL</td>
</tr>
<tr>
<td>Intermediate</td>
<td>More than 2 RF</td>
<td>15-20%</td>
<td>All</td>
<td>≥130 mg/dL</td>
<td>&lt;130 mg/dL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10-14% **</td>
<td>All</td>
<td>≥160 mg/dL</td>
<td>&lt;130 mg/dL</td>
</tr>
<tr>
<td>Low</td>
<td>0 or 1 RF</td>
<td>N/A</td>
<td>All</td>
<td>≥190 mg/dL</td>
<td>&lt;160 mg/dL</td>
</tr>
</tbody>
</table>

LDL-C reduction of 30-40% from baseline may be considered an alternative therapeutic strategy for patients who can not meet the above goals.

* N/A = Not applicable; TLC = Therapeutic Lifestyle Changes; RF = Risk Factor

* Lowering absolute risk involves modification of multiple risk factors/co-morbidities, not only LDL-C levels. Therefore, these goals should serve as a general guide and clinical judgment should be used to modify the goals as appropriate for each patient.

** There is insufficient evidence at this time to recommend routine screening for other risk markers not included in the risk index (e.g., FH, hsCRP, metabolic syndrome, depression), or evidence of significant atherosclerotic burden (e.g., high coronary artery calcification scores, intima medial thickness, abnormal brachial reactivity, or abnormal ankle-brachial index). These risk markers may be useful in the intermediate risk patient for whom it is less convincing that drug therapy would have a meaningful impact on outcomes.

### Table 4. Essential Components of Therapeutic Lifestyle Changes (TLC)

<table>
<thead>
<tr>
<th>Component</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL-raising nutrients</td>
<td></td>
</tr>
<tr>
<td>Saturated fats*</td>
<td>Less than 7% of total calories</td>
</tr>
<tr>
<td>Dietary cholesterol</td>
<td>Less than 200 mg/day</td>
</tr>
<tr>
<td>Therapeutic options for LDL lowering</td>
<td></td>
</tr>
<tr>
<td>Plant sterols/sterols</td>
<td>2 grams per day</td>
</tr>
<tr>
<td>Increased viscous (soluble) fiber</td>
<td>10—25 grams per day</td>
</tr>
<tr>
<td>Total calories (energy)</td>
<td>Adjust total caloric intake to maintain desirable body weight/prevent weight gain</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Include enough moderate exercise to expend at least 200 kcal per day</td>
</tr>
</tbody>
</table>

*Trans fatty acids are another LDL-raising fat that should be kept at a low intake.

### Table 5. Macronutrient Recommendations for the TLC Diet

<table>
<thead>
<tr>
<th>Component</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyunsaturated fat</td>
<td>Up to 10% of total calories</td>
</tr>
<tr>
<td>Monounsaturated fat</td>
<td>Up to 20% of total calories</td>
</tr>
<tr>
<td>Total fat</td>
<td>25—35% of total calories*</td>
</tr>
<tr>
<td>Carbohydrate†</td>
<td>50—60% of total calories*</td>
</tr>
<tr>
<td>Dietary fiber</td>
<td>20—30 grams per day</td>
</tr>
<tr>
<td>Protein</td>
<td>Approximately 15% of total calories</td>
</tr>
</tbody>
</table>

* ATP-III allows an increase of total fat to 35 percent of total calories and a reduction in carbohydrates to 50 percent for persons with the metabolic syndrome. Any increase in fat intake should be in the form of either polyunsaturated or monounsaturated fat.

† Carbohydrate should derive predominantly from foods rich in complex carbohydrates including grains—especially whole grains—fruits, and vegetables.
### Table 6. Dyslipidemia Drug Therapy

<table>
<thead>
<tr>
<th>Drug</th>
<th>Expected % Change in Lipoprotein (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>↑LDL-C</strong></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>Statins</td>
</tr>
<tr>
<td>Alternate</td>
<td>Niacin</td>
</tr>
<tr>
<td></td>
<td>Bile acid resin</td>
</tr>
<tr>
<td></td>
<td>Ezetimibe</td>
</tr>
<tr>
<td><strong>↑LDL-C and ↑TG</strong></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>Statins</td>
</tr>
<tr>
<td></td>
<td>Niacin</td>
</tr>
<tr>
<td>Alternate</td>
<td>Fibrates</td>
</tr>
<tr>
<td><strong>↑LDL-C and ↓HDL-C</strong></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>Statins</td>
</tr>
<tr>
<td></td>
<td>Niacin</td>
</tr>
<tr>
<td>Alternate</td>
<td>Fibrates</td>
</tr>
</tbody>
</table>

* Considerations:

- **Statins**: Statins are contraindicated in active liver disease, in those persons with persistent elevation of liver transaminases, and in pregnancy.
- **Niacin**: Niacin is contraindicated in hepatic disease and relatively contraindicated in gout or history of complicated/active peptic ulcer disease (PUD). Use niacin with caution in diabetics, since it may alter glucose control.
- **Resins**: Resins may increase TG and can reduce the absorption of many drugs. Therefore, other drugs should be administered 1 hour before or 4-6 hours after administration of the resin.
- **Fibrates**: Fibrates are contraindicated in severe renal or hepatic disease, including primary biliary cirrhosis and preexisting gallbladder disease.
- **Ezetimibe**: Maximum LDL-C lowering effect should be apparent within 2 weeks of initiation of treatment.

### Table 7. Doses of Currently Available Statins Required to Attain an Approximate 30% to 40% Reduction of LDL-C Levels (Standard Doses)*

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose, mg/day</th>
<th>LDL Reduction, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atorvastatin</td>
<td>10†</td>
<td>39</td>
</tr>
<tr>
<td>Lovastatin</td>
<td>40†</td>
<td>31</td>
</tr>
<tr>
<td>Pravastatin</td>
<td>40†</td>
<td>34</td>
</tr>
<tr>
<td>Simvastatin</td>
<td>20—40†</td>
<td>35—41</td>
</tr>
<tr>
<td>Fluvastatin</td>
<td>40—80</td>
<td>25—35</td>
</tr>
<tr>
<td>Rosuvastatin</td>
<td>5—10‡</td>
<td>39—45</td>
</tr>
</tbody>
</table>

* Estimated LDL reductions were obtained from U.S. Food and Drug Administration (FDA) package inserts for each drug.

† All of these are available at doses up to 80 mg. For every doubling of the dose above standard dose, an approximate 6% decrease in LDL-C level can be obtained.

‡ For rosuvastatin, doses available up to 40 mg; the efficacy for 5 mg is estimated by subtracting 6% from the FDA–reported efficacy at 10 mg. (Jones et al., 1998)
### Table 8. Potential Combination Pharmacological Treatments for Dyslipidemia

<table>
<thead>
<tr>
<th>Drug Combination</th>
<th>Expected % Change in Lipoproteins (Range)</th>
<th>Outcome DATA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>↑ LDL-C When Monotherapy is Inadequate</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statin + Resin</td>
<td>-30 to -60, -</td>
<td>+10</td>
</tr>
<tr>
<td>Statin + Niacin</td>
<td>-25 to -57, -13 to -36, -19 to -38</td>
<td>No Data</td>
</tr>
<tr>
<td>Statin + Ezetimibe</td>
<td>-34 to -60, -3 to -9, -11 to -24</td>
<td>No Data</td>
</tr>
<tr>
<td>Niacin + Resin</td>
<td>-32 to -43, -37 to -43, -27 to -29</td>
<td>No Data</td>
</tr>
<tr>
<td>Statin + Resin or Ezetimibe + Niacin</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td><strong>↑ LDL-C and ↑ TG (≥500 mg/dL)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+Statin + Niacin***</td>
<td>-25 to -57, -13 to -36, -19 to -38</td>
<td>No Data</td>
</tr>
<tr>
<td>Statin + Fibrate</td>
<td>-</td>
<td>-19 to -22, -41 to -53</td>
</tr>
<tr>
<td>Statin + Fish Oil</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Niacin + Fibrate</td>
<td>No Data - TC -13, -45</td>
<td>-20</td>
</tr>
<tr>
<td>Ezetimibe + Niacin</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Ezetimibe + Fish Oil</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td><strong>Very High TG and/or Low HDL-C Without Elevated LDL-C</strong>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statin + Niacin***</td>
<td>See above for effect on lipids.</td>
<td>No Data</td>
</tr>
<tr>
<td>Statin + Fibrate</td>
<td>See above</td>
<td>No Data</td>
</tr>
<tr>
<td>Statin + Fish Oil</td>
<td>See above</td>
<td>No Data</td>
</tr>
<tr>
<td>Fibrate + Niacin</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Fibrate or Niacin + Fish Oil</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Fibrate + Niacin + Fish Oil</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td><strong>Low HDL-C, high LDL-C and high TG)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statin + Niacin</td>
<td>See Above</td>
<td>No Data</td>
</tr>
<tr>
<td>Statin + Fibrate</td>
<td>See Above</td>
<td>No Data</td>
</tr>
<tr>
<td>Fibrate + Niacin + Resin</td>
<td>26, 36, 50</td>
<td>No Data</td>
</tr>
<tr>
<td>Ezetimibe + Niacin</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Ezetimibe + Fish Oil</td>
<td>No Data</td>
<td>No Data</td>
</tr>
</tbody>
</table>

(Guyton 1999, Worz & Bottorff, 2003, NCEP ATP-III, 2002). *Combination studies did not include patients with very high TG (≥500 mg/dL).

- =No additional benefit with combination, N=niacin, NR=not reported, R=resin, S=statin, TC=total cholesterol. The manufacturers of ezetimibe recommend avoiding the combination of ezetimibe plus fibrates (Fibrates can increase cholesterol excretion into the bile). In a dog study, ezetimibe also increased cholesterol excretion into the bile. There is no data on the combination of ezetimibe plus fish oils.

### Table 9. Drug Treatment for Hypertriglyceridemia

<table>
<thead>
<tr>
<th>TG 500-1000 mg/dL</th>
<th>Drug</th>
<th>Efficacy (Expected % Reduction in TG)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Initial</strong></td>
<td>Fibrates</td>
<td>-20 to -50</td>
</tr>
<tr>
<td><strong>Alternate</strong></td>
<td>Niacin</td>
<td>-20 to -35</td>
</tr>
<tr>
<td>n-3 PUFA Supplements, Omega-3 Fatty Acids/Fish Oils</td>
<td>-20 to -30</td>
<td></td>
</tr>
</tbody>
</table>

### Table 10. Drug Treatment for Isolated Low HDL-C

<table>
<thead>
<tr>
<th>LDL-C &lt;130 and Low HDL-C</th>
<th>Drug</th>
<th>Efficacy (Expected % Reduction in TG)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gemfibrozil</td>
<td>LDL-C</td>
<td>+10 to −35</td>
</tr>
<tr>
<td></td>
<td>HDL-C</td>
<td>+2 to 34</td>
</tr>
</tbody>
</table>
**Figure 1: Step Wise Care Approach (NCEP ATP-III, 2002)**

**Visit 1**
- Begin TLC
- Emphasize reduction in saturated fat and cholesterol
- Emphasize need for regular physical activity
- Consider referral to a dietitian for MNT

**Visit 2**
- Evaluate LDL-C response; If goal not met, intensify treatment
- Reinforce reduction in saturated fat and cholesterol
- Consider adding plant stanols/sterols
- Increase fiber intake
- Consider referral to a dietitian for MNT

**Visit 3**
- Evaluate LDL-C response If LDL goal not met, consider drug therapy
- Intensify weight management and physical activity
- Focus on treatment for metabolic syndrome
- Consider referral to a dietitian for MNT

---

**Box 5. Classification of Serum Lipids**

<table>
<thead>
<tr>
<th>Total Cholesterol (TC) mg/dl (mmol/L)</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 200 (&lt; 5.2)</td>
<td>Normal</td>
</tr>
<tr>
<td>200 - 239 (5.2 - 6.1)</td>
<td>Borderline high</td>
</tr>
<tr>
<td>≥ 240 (≥ 6.2)</td>
<td>High</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LDL-Cholesterol mg/dl (mmol/L)</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 100 (&lt; 2.6)</td>
<td>Normal</td>
</tr>
<tr>
<td>100 - 129 (2.6 - 3.3)</td>
<td>Above, near optimal</td>
</tr>
<tr>
<td>130 - 159 (3.4 - 4.0)</td>
<td>Borderline high</td>
</tr>
<tr>
<td>160 - 189 (4.1 - 4.8)</td>
<td>High</td>
</tr>
<tr>
<td>≥ 190 (≥ 4.9)</td>
<td>Very high</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HDL-Cholesterol mg/dl (mmol/L)</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40 (&lt;1.0)</td>
<td>Low</td>
</tr>
<tr>
<td>≥ 60 (≥ 1.6)</td>
<td>High</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Triglycerides (TG) mg/dL (mmol/L)</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150 mg/dL (&lt; 1.7)</td>
<td>Normal</td>
</tr>
<tr>
<td>150 - 199 mg/dL (1.7 - 2.2)</td>
<td>Borderline High</td>
</tr>
<tr>
<td>200 – 499 mg/dL (2.3 - 5.6)</td>
<td>High</td>
</tr>
<tr>
<td>≥ 500 mg/dL (≥ 5.6)</td>
<td>Very High</td>
</tr>
</tbody>
</table>

---

**Box 6. Key Elements in Management of Combination Therapy**

1. Treatment of LDL and non-HDL should focus on statin therapy alone.
2. Reserve combination therapy for high-risk patients (secondary prevention or familial hypercholesterolemia)
3. Discuss the risks and unproven clinical benefits of statin-fibrate therapy with the patient and document it in the patient’s medical record.
4. Prescribe the lowest effective dosages of the statin and fibrate to achieve treatment goals.
5. Use caution in patients with the following characteristics: advanced age, female gender, compromised renal function, heavy alcohol use, frailty and hyperthyroidism.
6. Be cautious about use of drugs that could interfere with the metabolism of the statin, or are known potent CYP 3A4 inhibiting medications (e.g., macrolides, azole antifungals, protease inhibitors, cyclosporine, etc.)
7. Obtain a baseline CK level and repeat the measurement during therapy, if the patient reports symptoms consistent with myopathy.
8. Teach patients to recognize and report generalized muscle weakness, tenderness, or pain; be prepared to evaluate those who experience these symptoms. (Evaluate CK and UA.)
9. Discontinue therapy for myopathic symptoms and elevated CK
10. If TG-lowering drug is added to a statin, caution is required due to particularly higher risk of myopathy. Fibrate and niacin combinations with statin may be more toxic than combination with fish oil.
Box 8. Criteria for Identifying Metabolic Syndrome

The metabolic syndrome is identified by the presence of three or more of the following criteria:

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Defining Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal Obesity</td>
<td>Waist Circumference</td>
</tr>
<tr>
<td>Men†</td>
<td>&gt;40 in (&gt;102 cm)</td>
</tr>
<tr>
<td>Women</td>
<td>&gt;35 in (&gt;88 cm)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>&gt;150 mg/dl</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>&lt;40 mg/dL</td>
</tr>
<tr>
<td>Women</td>
<td>&lt;50 mg/dL</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>≥130/85 mmHg</td>
</tr>
<tr>
<td>Fasting Glucose</td>
<td>≥110 mg/dL</td>
</tr>
</tbody>
</table>

NCEP ATP-III, 2002

† Some male persons can develop multiple metabolic risk factors when the waist circumference is only marginally increased, e.g., 94–102 cm (37–39 in). Such persons may have a strong genetic contribution to insulin resistance. They should benefit from changes in life habits, similarly to men with categorical increases in waist circumference.

AIDS = acquired immune deficiency syndrome  
DM = diabetes mellitus  
HAART = highly active antiretroviral therapy  
HbA1c = glycosylated hemoglobin  
HDL-C = high-density lipoprotein cholesterol  
HIV = human immunodeficiency virus  
LDL-C = low-density lipoprotein cholesterol  
LFTs = liver function tests  
SCr = serum creatinine  
TC = total cholesterol  
TG = triglycerides  
TSH = thyroid-stimulating hormone.