Montana Geriatric Education Center

Instructions on Completing the Module

Screening for Lipid Disorders in Older Adults

*The results of the assessments and evaluations are confidential, and the data is used to meet requirements of our federally funded grant.

Please make sure to turn in Pre-Test, Post-Test, and Module Evaluation.

1. **Before** reading the module, and without looking at it, complete the Pre-Test.

   Record your answers on the examination form marked Pre-Test. *(Found at the start of the module.)* Keep the completed answer form to turn in at the completion of the module.

2. Complete the module as outlined.

3. **After** reading the module, please complete the Post-Test.

   Use the questions in Appendix H and record your answers on the examination form marked Post-Test. *(Appendix H.)* Keep the completed answer form to return with the pre-test at the completion of the module.

   Complete the Module Evaluation. *(Found after the post-test.)* Keep the completed module evaluation form to return with the pre-test and post-test at the completion of the module.

4. To obtain credit for the module you must:
   b. Turn in the Pre-Test, Post-Test, and Module Evaluation
   c. Obtain a score of 70% or better on the Post-Test

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Pre-test: Screening for Lipid Disorders in Older Adults

1) Lipids are involved in many physiologic roles with the exception of which of the following?
   a) Participate in the formation of bile salts, which assist in the emulsification of dietary fats and cholesterol for absorption.
   b) Source of amino acids needed for synthesis of proteins.
   c) Provide immediate and stored source of energy for the body’s physiologic needs.
   d) Assist in the biosynthetic formation of prostaglandins, steroids and cholesterol.

2) High density lipoproteins (HDL) composition includes all of the following except:
   a) High protein content
   b) High cholesterol content
   c) Low triglyceride content
   d) High phospholipid content

3) Which of the following lipoproteins is the primary carrier for plasma triglycerides?
   a) HDL
   b) LDL
   c) IDL
   d) Chylomicron

4) Which of the following statements is NOT true regarding the involvement of lipoproteins in lipid transport?
   a) HDL particles remove cholesterol from peripheral tissues and directly transport the cholesterol to the liver for clearance.
   b) Lipoproteins are needed to assist the transport of lipophilic molecules such as triglycerides and cholesterol.
   c) VLDL particles are involved in the transport of triglycerides, obtained from dietary absorption, to the peripheral tissues.
   d) LDL particles primarily carry cholesterol to the peripheral tissues.

5) Which of the following is NOT considered to be clinical atherosclerotic cardiovascular disease (ASCVD)?
   a) Unstable angina
   b) Hypertension
   c) Transient ischemic attack
   d) Peripheral artery disease

6) Initiating therapeutic lifestyle changes (TLC) may help lower LDL cholesterol. Which of the following is NOT a recommendation of the 2013 Lifestyle Guidelines?
   a) Increase physical activity to 40 min of moderate-to-vigorous activity at least 3 times weekly.
   b) Dietary cholesterol should be less than 200 mg per day
   c) Follow a dietary pattern (e.g., Mediterranean diet) may provide better results than changing specific diet components.
   d) Trans fatty acids should be increased in the diet and saturated fats should be decreased

7) Obtaining an adequate blood sample from a finger stick may be difficult at times. Which of the following should NOT be routinely performed during blood collection?
   a) Look for a fingertip with minimal calluses
   b) To increase blood flow, firmly massage the pricked finger starting from the base of the finger to the tip
   c) Warm up cold hands by placing the hands under warm, running water for about 60 seconds
   d) Inspect the fingertips by gently pressing them to see which ones have good blood return.
8) ABC is a 72 year old white female patient who appears to be in good health and is physically fit. She shows up for her lipid screening not having eaten since last night’s dinner. Her only medications are for low thyroid, a daily multivitamin, and occasional acetaminophen for arthritis. She states she has never smoked a cigarette in her life, and does not have any heart problems, nor does it run in her family. ABC is physically active (walks 2 miles/day) and eats “good” foods. Her lipid screening results are (see right):

Utilizing the ASCVD Risk Estimator, what is ABC’s 10-year risk of having an ASCVD event?

a) 5.8%
b) 8.8%
c) 11.2%
d) 26.8%

9) What counseling would you give to ABC?

a) She may benefit from low to moderate intensity statin therapy and should discuss drug therapy with her PCP.
b) **She may benefit from moderate to high intensity statin therapy and should follow up with her PCP.**
c) She does not need drug therapy and should continue her current lifestyle.
d) She should increase her walking to 4 miles/day and discuss non-statin drug therapy with her PCP.

10) Patient DEF, is a 68 year old white male who appears somewhat overweight. DEF is coming in for lipid testing at the insistence of his daughter who is accompanying him. DEF is a life-long rancher who took over his father’s business when his father died of a massive heart attack at age 59. His daughter states her father has not eaten since last night, which has made him grumpy. DEF claims to be as healthy as a horse and does not take any medications other than an occasional aspirin for a headache or backache. DEF’s older sister is alive and she takes medicine for her high blood pressure. DEF admits to smoking about ½ a pack a day of cigarettes, drinks 1-2 beers a day, and loves his meat and potatoes. DEF’s lipid screening provides the following results (see below):

<table>
<thead>
<tr>
<th>DEF’s Results:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>152/100 mmHg</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>226 mg/dL</td>
</tr>
<tr>
<td>Total chol.</td>
<td>241 mg/dL</td>
</tr>
<tr>
<td>HDL</td>
<td>36 mg/dL</td>
</tr>
<tr>
<td>LDL</td>
<td>185 mg/dL</td>
</tr>
<tr>
<td>VLDL</td>
<td>20 mg/dL</td>
</tr>
<tr>
<td>TC/HDL</td>
<td>6.7</td>
</tr>
</tbody>
</table>

DEF has multiple traditional risk factors for CHD. How many does he have?

a) 2
b) 3
c) **4**
d) 5
11) What is DEF’s 10-year risk assessment of developing ASCVD according to the ASCVD Risk Estimator?
   a) 12%
   b) 16%
   c) 25%
   d) 33%

12) Into which statin-benefit category should DEF be placed?
   a) Presence of Clinical ASCVD
   b) LDL > 190mg/dL
   c) Diabetes Type 1 or 2 and aged 40-75 years
   d) LDL 70-189mg/dL & estimated ASCVD risk > 7.5%

13) Appropriate counseling for DEF would include:
   a) Inform patient he is at great risk for developing ASCVD in the next 10 years and should be seen by a health care provider as soon as possible for follow-up assessment.
   b) Inform the patient that changes to his lifestyle (i.e., not smoking, increased physical activity, and eating foods lower in saturated fats), may reduce his cholesterol level.
   c) Inform the patient that individuals who have high LDL cholesterol have a higher risk of developing ASCVD and his primary goal is to lower his LDL cholesterol.
   d) All of the above should be included in patient counseling.

14) Patient HIJ, a 56 year old white female comes in for her lipid screening after not having eaten since last night about 9pm. HIJ is currently being followed by her physician for high blood pressure for which she takes the combination product lisinopril/hydrochlorothiazide. She takes no other medications on a regular basis. She is a non-smoker. Her parents are both deceased; father died from lung cancer and her mother died secondary to pneumonia. Her older brother is in good health and also takes medication for high blood pressure.

HIJ’s lipid screening gives the following results (see right):

Which of the following is NOT a positive risk factor for ASCVD? (Positive risk factor means having the risk factor increases the risk of developing ASCVD)
   a) Currently on medicine for blood pressure.
   b) Female ≥ 55 years old.
   c) HDL >60 mg/dL
   d) All are positive risk factors

15) Since HIJ has multiple risk factors, what is her calculated 10-year ASCVD risk of developing CHD?
   a) 3.2%
   b) 7.8%
   c) 14.2%
   d) 19.3%

16) Assuming HIJ is at low risk of developing ASCVD over the next 10 years, which of the following statements is the most appropriate action to be taken based on her LDL assessment?
   a) Nothing needs to be done; this patient is already at her LDL goal.
   b) This patient should be seen by her primary care provider as soon as possible to initiate drug therapy.
   c) This patient may benefit from initiating therapeutic lifestyle changes and should discuss the results of this screening with her health care provider at the next scheduled visit.
   d) This patient is doing just fine and should continue her current lifestyle despite an LDL above goal.
17) Patient KLM, a 58 year old, obese white male is being seen for lipid screening. KLM was recently diagnosed with type II diabetes and is currently taking metformin for glucose control. He takes no other prescription medications, but does take loratadine for seasonal allergies and occasionally acetaminophen for pain in his “bad” right knee. He does not smoke and does not get regular exercise due to his “bad” knee.

KLM is automatically considered to be at high risk for ASCVD because of:

a) His age
b) His obesity
\textbf{c)} His diabetes
d) His sedentary lifestyle

18) KLM states he ate breakfast this morning (which was about 4 hours ago), and he wants to know if he can still get his lipids tested. Which of the following actions is the most appropriate for this patient?

a) Turn the patient away; all lipid testing must be performed in fasting patients (9-12 hours).
b) Explain to the patient, fasting is preferred to get good results for HDL and LDL determination.
c) \textbf{Explain to the patient, fasting is necessary to get good results for LDL and triglyceride determination, and therefore, only this patient’s HDL and total cholesterol may be calculated.}
d) Tell the patient that not having fasted is OK, and that the lipid analysis will be fine.

19) You perform KLM’s lipid screening with the following results (see below). How would you counsel this patient?

<table>
<thead>
<tr>
<th>KLM’s Results:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
</tr>
<tr>
<td>Triglycerides</td>
</tr>
<tr>
<td>Total chol.</td>
</tr>
<tr>
<td>HDL</td>
</tr>
<tr>
<td>LDL</td>
</tr>
<tr>
<td>VLDL</td>
</tr>
<tr>
<td>TC/HDL</td>
</tr>
</tbody>
</table>

I KLM should be seen by his primary care provider in the near future to discuss lipid lowering therapy.

II KLM would benefit from initiating therapeutic lifestyle changes and may want to speak to a dietician since he has diabetes and potentially lipid abnormalities.

III KLM should not worry about his lipids; they are not too bad.

a) I only
b) II only
c) I & II
d) I, II, III

20) KLM is a candidate for high intensity statin therapy for the following reason:

a) \textbf{Presence of diabetes with ASCVD risk estimate > 7.5%}
b) Presence of diabetes with ASCVD risk estimate < 7.5%
c) Presence of clinical ASCVD
d) LDL > 100mg/dL
Screening for Lipid Disorders in Older Adults

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A 3-hour Geriatric Health Screening Module from the
Montana Geriatric Workforce Enhancement Program

A Consortium of:
University of Montana, Missoula
Mountain Pacific Health, Helena
RiverStone Health, Billings
St. Vincent Healthcare, Billings

Montana Geriatric Education Center Website

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Montana Geriatric Education Center (MTGEC)
Screening For Lipid Disorders in Older Adults
Disclosures

Montana Geriatric Workforce Enhancement Program Goals/Purpose
Improve health outcomes for older adults in rural Montana via increased knowledge of older adult care and treatment of health problems by health professionals.

Successful completion of this continuing education activity includes:
• Completion of the Pre-Test
• Reading of text
• Visiting websites as directed in module
• Completion of the Post-Test with at least 70% accuracy
• Completion of the module evaluation

Contact Hours: 3, including 1 Rx Hours for Nurses

Montana Nurses Association (MNA)
The Montana Geriatric Education Center is an approved provider of continuing nursing education by the Montana Nurses Association, an accredited approver by the American Nurses Credentialing Center’s Commission on Accreditation.
MNA Continuing Nursing Education Expiration Date: 1/19/2021

Conflicts of Interest
The planners and presenters of the CE activity have disclosed no relevant financial relationship with any commercial companies pertaining to this activity.

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Description of Module

Content:
This 3 hour module will discuss the basic issues which surround screening for lipid abnormalities in the geriatric population.

Module Purpose:
The purpose of this module is to enable the learner to improve his/her knowledge of screening and counseling for dyslipidemia in older adults and apply it in the professional setting.

Learning Objectives:
Specifically, the learner will be able to:

1. Describe the impact of lipid disorders on cardiovascular disease.
2. Examine the 2013 American College of Cardiology/American Heart Association (ACC/AHA) Guidelines for the assessment and treatment of elevated cholesterol levels.
3. Counsel on diet, lifestyle changes, and medication use (when appropriate), based on patient specific information, including risk factors for cardiovascular disease.
4. Describe techniques involved in performing point-of-care lipid testing using the CardioChek Plus®.
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Screening for Lipid Disorders in Older Adults

I. Overview

Heart disease is the leading cause of death in the United States, accounting for approximately 30.8% of all deaths in 2011. Each year, nearly 400,000 individuals die from coronary heart disease (CHD), while stroke accounts for almost 130,000 deaths. Additionally, stroke is the leading cause of serious disability in American adults.

The risk of developing heart disease increases with age. In the US, heart disease accounted for 26.5% of deaths in 2010 for those 65 years of age or older. Coupled with the increasingly older US population, screening for atherosclerotic cardiovascular disease (ASCVD) is imperative to decrease the mortality and morbidity associated with ASCVD.

The good news is that heart disease is declining in the US, as it accounted for 38% of deaths in 1980. It has been estimated that the proportion of preventable deaths related to cardiovascular disease in individuals aged less than 80 years is 30%. Abnormal lipid levels are associated with an increased risk of ASCVD; therefore, the lipid panel can be used as a screening tool to identify patients who would benefit from intervening therapies such as lifestyle modifications and medication management.

II. Impact of Dyslipidemias on Health

A. Role of Lipids in Cardiovascular Disease

Cardiovascular disease is a broad term which encompasses four main groups of diseases:

1. Coronary heart disease (CHD): myocardial infarction, acute coronary syndrome, and angina pectoris
2. Cerebrovascular disease: ischemic stroke and transient ischemic attacks (TIA)
3. Peripheral vascular disease with intermittent claudication
4. Aortic atherosclerosis and abdominal aortic aneurysm

Evidence supports the relationship between abnormal lipid levels (dyslipidemia) and cardiovascular risk for three main reasons.
1. **Elevated Cholesterol Increases Risk of Cardiovascular Deaths**

The assessment of risk factors has repeatedly demonstrated the direct relationship between elevated lipid levels and an increased risk of cardiovascular disease.⁸

A follow-up study was performed in over 80,000 men who were originally enrolled in three separate prospective studies back in the 1960's and 1970's when they were between the ages of 18-39 years old: Chicago Heart Association (CHA), People’s Gas Company Study (PG), and the Multiple Risk Factor Intervention Trial (MRFIT). These men were followed for 16-34 years to determine their long-term cardiovascular and mortality risk in association with their baseline cholesterol levels during the original studies. Figures 1 & 2 demonstrate the linear relationship associated with increased total serum cholesterol with cardiovascular-related deaths and all-cause mortality.⁹

![Cardiovascular Deaths By Cholesterol Level](image)

**Figure 1: Increasing Cardiovascular Death Rate with Increasing Cholesterol Level⁸**
2. Decreasing Lipid Levels Lowers Risk of Cardiovascular Disease

Many clinical trials involving lipid-lowering agents, primarily a group of medications commonly referred to as “statins” (e.g., lovastatin, pravastatin, atorvastatin, etc.), have consistently shown decreases in cardiovascular risk when cholesterol levels are lowered. Figure 3 demonstrates the range of coronary heart disease risk reduction among the classic multiple clinical trials in different populations.\textsuperscript{10-19}

Additionally, a meta-analysis of clinical trials using statin medications found that every 10% reduction in serum cholesterol correlates with a 15% and 11% reduction in CHD mortality and all-cause mortality,
respectively.\textsuperscript{16} A 2012 meta-analysis evaluated 22 trials comparing statin therapy to control (average follow-up 4.8 years), and 5 trials of higher versus lower intensity statin therapy (average follow-up 5.1 years). Results of this analysis found that for every 38mg/dL decrease in low density lipoprotein (LDL), the risk of major vascular events significantly decreased by 21% across the 27 trials.\textsuperscript{21}

**B. Role of Lipids and Different Lipoproteins**

Lipids are involved in many physiological roles including:\textsuperscript{22}

a. Energy source for immediate use or stored for future needs.

b. Structural support for cell membranes in the form of cholesterol and phospholipids.

c. Precursor to bile salts, which are used to emulsify dietary fat and cholesterol for absorption.

d. Production of substances, such as steroids, prostaglandins, thromboxanes, and leukotrienes, from the dietary intake of essential fatty acids.

Due to the highly lipophilic (fat-loving) nature of cholesterol and triglycerides, assistance is needed to transport these molecules throughout the highly hydrophilic (water-loving) circulatory system. This assistance is provided by lipid carriers called lipoproteins. Lipoproteins consist of a lipophilic core (cholesteryl esters and triglycerides), surrounded by hydrophilic lipids (phospholipids and non-esterified cholesterol), and proteins. The proteins, referred to as apolipoproteins, function as cofactors for enzymes and as recognition sites for receptors. The five main plasma lipoproteins, each of which is classified based on its density, are different in composition, size and function: high density lipoprotein (HDL), low density lipoprotein (LDL), intermediate density lipoprotein (IDL), very low density lipoprotein (VLDL), and chylomicrons (Figure 4 and Table 1). The density of the lipoprotein is based on the relative content of lipid and protein. Proteins are denser than lipids; therefore, the more protein contained in a carrier particle, the denser the lipoprotein. Thus HDL, while being a smaller particle, is denser than LDL due to the higher protein content.\textsuperscript{22-24}
1. **Lipoprotein Transport**

The different lipoproteins, while having different structural compositions, also have different functions in lipid transport. Depending upon the source of lipids, different lipoproteins are involved. Exogenous lipids or those found from dietary sources are primarily carried by chylomicrons, whereas endogenous lipids are carried by VLDL, IDL, LDL and HDL.\(^\text{22}\) Figure 5 provides a schematic overview of how the different lipids are transported.

---

### Table 1: Difference in Composition between Lipoproteins\(^\text{22,25}\)

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Composition (%)</th>
<th>Primary Apolipoproteins</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Protein</td>
<td>Cholesterol</td>
</tr>
<tr>
<td>Chylomicron</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>VLDL</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>IDL</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>LDL</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>HDL</td>
<td>50</td>
<td>4</td>
</tr>
</tbody>
</table>

---

**Figure 4: Comparison of Densities between Lipoproteins\(^\text{23,24}\)**

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**Chylomicrons**
Chylomicrons carry dietary lipids (primarily triglycerides) from the intestine, into the lymphatic system, and finally into the circulatory system to peripheral sites before reaching the liver. The chylomicrons release their triglycerides when apolipoprotein C-II (ApoC-II) interacts with lipoprotein lipase (LPL) found in the endothelial surfaces of capillaries, catalyzing the release of triglycerides into free fatty acids. The remaining chylomicron or remnant is taken up by the liver through an interaction with apolipoprotein E (ApoE) and the low density lipoprotein receptor (LDLR). Usually within 12-14 hours after eating, all chylomicrons are absent from circulation.\(^{22,23}\)

**Very Low Density Lipoproteins (VLDL)**
VLDL is formed in the liver and its primary function is to carry triglycerides manufactured in the liver from fatty acids and carbohydrates. VLDLs are hydrolyzed by the ApoC-II and LPL interaction and release most of their triglyceride content, especially in muscle and adipose tissue.\(^{22,23}\)

**Intermediate Density Lipoproteins (IDL)**
IDLs are formed from VLDLs once they have lost most of their triglyceride content. IDLs can either be cleared from circulation by the liver via the low density lipoprotein receptor (LDLR), or they can be modified by the hepatic lipase enzyme to form low density lipoprotein (LDL) particles.\(^{22,23}\)

**Low Density Lipoproteins (LDL)**
LDL is the main cholesterol-carrying lipoprotein and primarily contains only one apolipoprotein, ApoB-100, which is the receptor ligand for the LDL receptor (LDLR) found in peripheral tissues and on the liver. LDL is either taken back up by the liver via the LDLR to be used for bile acid production, or transported to peripheral tissues to be incorporated into cell membranes, utilized for hormone synthesis, or stored for future use.\(^{22,23}\) When excess LDL is present in the plasma and these processes are saturated, LDL can enter the intima of the blood vessels. Within the arterial wall, macrophages consume large amounts of oxidized LDL creating foam cells, an initial step in the formation of the fatty arterial streak.\(^{26}\)

**High Density Lipoproteins (HDL)**
HDL is involved in transporting cholesterol back from the tissues to the liver for excretion in the bile (Figure 6). HDL is produced in the liver and intestines and has apolipoprotein A-I (ApoA-I) on its
surface which is utilized to transfer cholesterol from the peripheral tissues to the HDL particle. Once in the plasma, the cholesterol carried on the HDL particle is converted to cholesteryl esters through reaction with lecithin-cholesterol acyltransferase (LCAT). The cholesteryl ester-laden HDL particle can either transfer its cholesteryl esters by mean of the cholesteryl ester transfer protein (CETP) enzyme to chylomicrons and VLDL which returns the cholesterol to the liver, or the HDL particle can be directly removed from circulation by the liver.\textsuperscript{22-24}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{lipid_transport_diagram}
\caption{Schematic of Lipid Transport to Peripheral Tissues\textsuperscript{22-24}}
\end{figure}
2. Lipoprotein Metabolism & Mechanisms to Lower Lipids

As mentioned earlier, the two main lipid sources are from the diet or by endogenous biosynthesis. On average, the liver synthesizes approximately 400 mg of bile salts, 11 grams of phospholipids, and 2 grams of cholesterol each day.27

One of the main functions of the liver is to produce bile salts which help emulsify dietary lipids for absorption. Bile salts are synthesized within the liver from cholesterol and stored in the gall bladder until needed. The total amount of the bile salt pool is about 3.5 grams of which 95-99% is reabsorbed in the lower small intestine to be re-circulated to the liver via the hepatic-portal system for future use. The small amount of bile acids which are not reabsorbed is secreted in the feces. Therefore, the amount of bile acids synthesized on a daily basis is essentially the amount which is lost. The bile acid sequestering medications [e.g., cholestyramine (Questran®) or colestipol (Colestid®)] are non-absorbable polymers which bind to the bile acids in the gastrointestinal tract decreasing the amount of bile acids re-circulated back to the liver and consequently decreasing the cholesterol pool.27

In regards to cholesterol, the liver synthesizes about 2 grams per day and the average daily dietary intake is about 400 mg. Of the total daily sum of cholesterol (2.4 gm), about half is excreted in the
feces daily, leaving a balance of 1.2 grams, which is about 3-4 times greater than what is needed to sustain physiologic functions.\textsuperscript{27} There is sufficient daily hepatic production of cholesterol, so dietary intake is above and beyond what is essential to maintain physiological processes.

Fat in the diet is primarily in the form of triglycerides (90%), and the remainder as cholesterol, phospholipids and fat-soluble vitamins A, D, E, and K.\textsuperscript{28} Pancreatic enzymes partially break down the triglycerides into fatty acids and monoglycerides, and cholesterol is enzymatically modified into cholesteryl esters to aid in the transport across the intestinal wall. Once inside the intestinal cell wall, the lipids are packaged into chylomicrons for transport to peripheral sites to deliver triglycerides.\textsuperscript{22, 23} Ezetimibe (Zetia™) inhibits the transport of cholesterol across the intestinal cell wall which decreases dietary absorption of cholesterol.\textsuperscript{29}

The body synthesizes cholesterol, primarily in the liver, through a series of biosynthetic steps starting with acetyl-CoA (Figure 7). One of the biosynthetic steps includes the production of the cholesterol precursor, mevalonic acid, through a modification step using the enzyme HMG-CoA reductase.\textsuperscript{22} The class of drugs known as the “statins”, (e.g., atorvastatin, lovastatin, pravastatin, etc.), inhibit this enzyme preventing the production of mevalonic acid and subsequent cholesterol formation. Additionally, due to decreased production of cholesterol, the liver increases the removal of LDL-cholesterol from the blood to maintain its cholesterol balance resulting in the lowering of circulating LDL-cholesterol available to contribute to atherosclerosis.\textsuperscript{8}

![Figure 7: Cholesterol Biosynthesis\textsuperscript{22}](image-url)
As stated above, the hepatic LDLRs are involved in the regulation of circulating LDL concentrations. When LDL binds to these receptors, the cholesterol particles are degraded by the liver. However, if LDLR number or activity is deficient, the result is an increase in LDL plasma levels. The enzyme, proprotein convertase subtilisin kexin 9 (PCSK9), binds to the LDLR, promoting receptor degradation. The PCSK9 enzyme has become a recent drug therapy target for cholesterol reduction. By inhibiting PCSK9, the LDLR remains functional and can bind LDL. Currently, two monoclonal antibody PCSK9 inhibitors have been approved by the FDA and are available in the US [alirocumab (Praluent®) and evolocumab (Repatha®)].

### 3. Different Types of Lipid Abnormalities

Abnormal levels of lipids or dyslipidemias are classified as primary or secondary disorders. Primary disorders are caused by an inherent dysfunction in lipid metabolism, whereas secondary disorders are related to dietary intake, lifestyle, medical conditions, and/or medications. There are multiple causes for primary lipid disorders, including genetic mutations causing dyslipidemias to occur within a family lineage. Table 2 lists the more common disorders based on the lipoprotein particle affected. Many diseases, lifestyle habits, and medications can alter lipoprotein levels, and Table 3 lists some of the more common secondary causes.

When assessing a patient for screening purposes, a clear determination of primary and secondary dyslipidemias may not be possible or practical, but rather the lipid results and patient history in conjunction with ASCVD risk assessment will determine therapeutic recommendations.

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Disorder</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL</td>
<td>Familial hypercholesterolemia (monogenic)</td>
<td>Defective LDL-receptor on liver decreases LDL clearance from circulation</td>
</tr>
<tr>
<td></td>
<td>Familial defective ApoB-100</td>
<td>Defective ApoB-100 on LDL decreases LDL-receptor binding &amp; decreases LDL clearance.</td>
</tr>
<tr>
<td></td>
<td>Polygenic hypercholesterolemia</td>
<td>Multiple mutations which collectively raise LDL</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>Lipoprotein lipase (LPL) deficiency</td>
<td>Low LPL levels decrease amount of triglycerides cleared from circulation</td>
</tr>
<tr>
<td></td>
<td>ApoC-II deficiency</td>
<td>Decreased ApoC-II leads to decreased activity of LPL leading to decreased triglyceride release into peripheral tissues</td>
</tr>
<tr>
<td></td>
<td>Familial hypertriglyceridemia</td>
<td>Overproduction of VLDL cholesterol with normal VLDL clearance leads to accumulation of VLDL</td>
</tr>
<tr>
<td>HDL</td>
<td>ApoA-I dysfunction</td>
<td>Mutations in ApoA-I can lead to decreased HDL production or increase HDL clearance from the circulation.</td>
</tr>
<tr>
<td>Lipoprotein Disorder</td>
<td>Effect</td>
<td></td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Familial dysalphalipoproteinemias</td>
<td>Increased or decreased production of HDL depending on genetic defect</td>
<td></td>
</tr>
<tr>
<td>LCAT deficiency</td>
<td>Low levels of LCAT prevent the conversion of cholesterol to the ester form, preventing cholesterol clearance</td>
<td></td>
</tr>
<tr>
<td>CETP deficiency</td>
<td>Low levels of CETP prevent the transfer of the cholesterol esters from HDL to VLDL and chylomicrons resulting in increased HDL</td>
<td></td>
</tr>
<tr>
<td>Combined TG and Cholesterol</td>
<td>An overproduction of apoprotein B particles which leads to an increase in HDL production</td>
<td></td>
</tr>
<tr>
<td>Familial dysbetaalipoproteinemia</td>
<td>Mutation involving apolipoprotein E which leads to reduced clearance of VLDL and chylomicrons</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2: Primary Causes of Dyslipidemias**

<table>
<thead>
<tr>
<th>Category</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine/Metabolic</td>
<td>• Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>• Lipodystrophy</td>
</tr>
<tr>
<td></td>
<td>• Cushing’s syndrome</td>
</tr>
<tr>
<td></td>
<td>• Hypothyroidism</td>
</tr>
<tr>
<td>Renal</td>
<td>• Chronic renal failure</td>
</tr>
<tr>
<td></td>
<td>• Glomerulonephritis</td>
</tr>
<tr>
<td></td>
<td>• Nephrotic syndrome</td>
</tr>
<tr>
<td>Hepatic</td>
<td>• Cirrhosis</td>
</tr>
<tr>
<td></td>
<td>• Obstructive liver diseases</td>
</tr>
<tr>
<td>Lifestyle</td>
<td>• Physical inactivity</td>
</tr>
<tr>
<td></td>
<td>• Obesity</td>
</tr>
<tr>
<td></td>
<td>• Smoking</td>
</tr>
<tr>
<td></td>
<td>• High fat diet (especially saturated fats)</td>
</tr>
<tr>
<td></td>
<td>• Alcohol intake</td>
</tr>
<tr>
<td>Medications</td>
<td>• Progestins, estrogens</td>
</tr>
<tr>
<td></td>
<td>• Thiazide diuretics</td>
</tr>
<tr>
<td></td>
<td>• Beta-blockers</td>
</tr>
<tr>
<td></td>
<td>• Glucocorticoids</td>
</tr>
<tr>
<td></td>
<td>• Isotretinoin</td>
</tr>
<tr>
<td></td>
<td>• Protease Inhibitors</td>
</tr>
<tr>
<td></td>
<td>• Atypical antipsychotics</td>
</tr>
<tr>
<td></td>
<td>• Cyclosporine</td>
</tr>
<tr>
<td></td>
<td>• Mirtazapine</td>
</tr>
<tr>
<td></td>
<td>• Anabolic steroids</td>
</tr>
</tbody>
</table>

**Table 3: Secondary Causes of Dyslipidemias**
C. Atherogenesis
The development of atherosclerosis is the precursor for atherosclerotic cardiovascular diseases; therefore, it is necessary to understand the pathogenesis and the role lipid abnormalities play in the progression of these conditions.

Atherosclerosis is the progressive narrowing of the arteries characterized by development of plaques within the intima region of the arterial wall caused by cellular thickening and lipid accumulation. These plaques begin to press toward the center of the artery leading to a partial occlusion of the vessel lumen. When the lumen of a vessel is about 70-80% occluded, symptomatic evidence of disease can be seen as found in ischemic heart disease (e.g., acute coronary syndrome, myocardial infarction, or unstable angina), cerebrovascular disease (e.g., stroke), or peripheral vascular disease (e.g., intermittent claudication). \textsuperscript{32-34} Figures 8 & 9 depict the normal and abnormal arterial cell wall composition, respectively.

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure8.png}
\caption{Normal Cross-Section of an Arterial Vessel}
\end{figure}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure9.png}
\caption{Disease Progression in an Atherosclerotic Vessel}
\end{figure}
The mechanism of atherosclerosis is not fully understood, but the current theory hypothesizes changes in the arterial wall in response to injury to the endothelium lead to a chronic inflammatory response. Figure 10 portrays a hypothetical schematic of atherosclerosis.25

Chronic or repetitive injury to the arterial endothelium leading to endothelial dysfunction is thought to be the first step in atherosclerosis. The cause of the initial insult may be multifactorial and remains unclear. Factors such as oxidized LDL, direct injury to the endothelium, components found in cigarette smoke, elevated homocysteine levels, or infectious viruses or bacteria may be involved, leading to initial endothelial dysfunction. Arterial vessels which have areas of disturbed or turbulent flow, such as those found at branch points or in highly elastic vessels (e.g., aorta, carotid, or iliac arteries), are more susceptible to endothelial injury. Not only does the endothelial injury increase the permeability of lipoproteins in the intima region of the cell wall, but it also attracts monocytes. Monocytes are then transformed within the intima into macrophages.25

Circulating white blood cells do not normally bind to the arterial wall, but early in atherogenesis, endothelial cells begin to express adhesion factors on their luminal surface to facilitate binding and migration of monocytes into the intima.25 Also occurring during this stage of atherosclerosis, smooth muscle cells from the medial layer of the arterial cell wall are attracted to and migrate into the intimal layer where they proliferate.25

Isolated from plasma antioxidants, the increased levels of lipoproteins in the intima become susceptible to oxygen radicals produced by endothelial cells and macrophages resulting in oxidative modification of the LDL molecule. Primarily macrophages and some smooth muscle cells engulf the oxidized LDL-cholesterol forming foam cells. Oxidized LDL-cholesterol also stimulates the release of cytokines and growth factors which increase monocyte accumulation in the atherosclerotic lesion causing a fatty streak.25

Accumulation of foam cells and other cellular components can trigger some of the foam cells to die leaving a lipid core covered by a fibrous cap. This is the beginning of an atherosclerotic plaque. As the plaque continues to evolve with time, plaque calcification may occur.25

The fibrous cap of the plaque may erode, ulcerate or rupture into the lumen exposing the highly thrombogenic lipid core to the blood stream. This leads to arterial occlusion secondary to platelet aggregation and thrombus formation, or the plaque may break off causing an embolic threat to vessels downstream.25
It is known that atherosclerotic processes begin early in life. The development of fatty streaks are noted in autopsy studies of young men and women (ages 15-34) who died for non-cardiac reasons.\textsuperscript{35} Subsequent evaluation utilizing noninvasive methods such as carotid artery intima measurement and coronary artery calcification scores have confirmed that risk factors presenting in youth are strong predictors of atherosclerosis up to 20 years later.\textsuperscript{36} The asymptomatic progression may take decades to develop before an adverse cardiovascular event occurs.

\textbf{Figure 10: Hypothetical Mechanism of Atherosclerosis}\textsuperscript{25}

\textbf{III. Guidelines for Management of Dyslipidemias}

In 1985, the National Heart, Lung, and Blood Institute (NHLBI) developed the National Cholesterol Education Program (NCEP) with the aim to reduce the number of Americans with high blood cholesterol in order to decrease the morbidity and mortality associated with coronary heart disease.\textsuperscript{37} Guidelines
were released sequentially over the next two decades with the last full guideline from the Adult Treatment Panel (ATP) released in 2001. Newer clinical trial data providing better lipid goal definitions based on the patient’s cardiovascular (CV) risk were the basis for the updated ATP recommendations released in 2004.\textsuperscript{38, 39}

The process to update the ATP cholesterol guidelines began again in 2005. It had been several years since NHLBI published a comprehensive lipid management guideline. During that time, numerous trials had been published evaluating outcomes related to cardiovascular risk reduction and cholesterol management. The committee began to review available evidence to develop the revised guideline recommendations. The process proved to be complicated and after much delay, in June 2013, the NHLBI began collaborating with the American College of Cardiology (ACC) and the American Heart Association (AHA) to complete the guideline development process. In November 2013, the AHA/ACC released an updated set of guidelines addressing the topics of CV risk assessment, reduction of CV risk by lifestyle modifications, management of blood cholesterol, and adult obesity.\textsuperscript{40-42}

A. Guideline Comparison – Revised ATP III versus AHA/ACC 2013 Guidelines

The 2013 AHA/ACC series of guidelines represent a significant departure in the approach by which patients are assessed and treated as compared to the ATP III guidelines. Specific critical questions were identified by the expert panels and workgroups for inclusion in the final published guidelines.\textsuperscript{40} This shift was partly due to a 2011 Institute of Medicine (IOM) report which provided standards for trustworthy clinical guideline development.\textsuperscript{43} The significant changes in the new AHA/ACC guideline recommendations were based on available evidence from well-designed trials.

B. 2013 AHA/ACC Guideline on Assessment of Cardiovascular Risk

The primary charge of the workgroup was to develop or recommend a quantitative cardiovascular risk assessment approach to guide patient care. This selective approach was also to be used in the treatment guidelines and algorithms developed for cholesterol, hypertension, and obesity. Additionally, two critical questions were identified:
1. “What is the evidence regarding reclassification or contribution to risk assessment when high-sensitivity C-reactive protein (hs-CRP), apolipoprotein B (ApoB), glomerular filtration rate (GFR), microalbuminuria, family history, cardiorespiratory fitness, ankle-brachial index (ABI), carotid intima media thickness (CIMT), or coronary artery calcium (CAC) score are considered in addition to the variables that are in the traditional risk scores?”

2. “Are models constructed to assess the long-term (≥ 15 years or lifetime) risk for a first CVD event in adults effective in assessing variation in long-term risk among adults at low and/or intermediate short-term risk, whether analyzed separately or combined?”

**Critical Question 1 - Quantitative Risk Assessment**

The determination of 10-year risk of CHD in the ATP III guidelines utilized the Framingham study data, which was first reported in 1998. While the ATP III Framingham risk assessment was a useful tool, the data were derived from an all-white suburban population which limited the validity in ethnically diverse populations. Secondly, the Framingham risk assessment tool focused only on ‘hard coronary events’ including non-fatal MI and CHD death with no determination for stroke. For these reasons, the workgroup developed new Pooled Cohort ASCVD Risk Equations.

**Pooled Cohort ASCVD Risk Equations**

Similarly to the ATP III risk assessment tool, gender, age, systolic blood pressure, presence or absence of anti-hypertensive therapy, total cholesterol and HDL levels, smoking status, and history of diabetes are required patient variables. In comparison, the Pooled Cohort Equation risk calculator estimates 10-year risk for primary occurrence of nonfatal MI, CHD death, and nonfatal or fatal stroke. The tool is available on-line or smartphone. (http://my.americanheart.org/cvriskcalculator).

It should be noted that the Pooled Cohort Risk Equations have been validated only for the ages of 40 to 79 years. Additionally, the populations included in the studies from which the risk tool was derived were predominantly Caucasian and African-American. Therefore, it is recommended the race-specific option be used for non-Hispanic African Americans and Caucasians. For individuals from other racial or ethnic groups, the option for non-Hispanic whites is currently recommended. There is concern that this tool may underestimate risk in those with severe hypertension, diabetes and left ventricular dysfunction, while overestimating risk in younger and elderly populations.
Additional Risk Factors

Since ATP III was released in 2003, new CVD risk markers, such as apolipoprotein B (ApoB), high sensitivity C-reactive protein (hs-CRP), and many others have been identified. The goal of the workgroup was to determine if routine measurement of risk markers would improve risk assessment in the asymptomatic patients seen in general clinical practice.40

To assess this issue the workgroup approached the question in two formats. First, the risk of use, cost, availability, and reliability of the test in routine practice were evaluated. Secondly, as the PCE models were developed the risk markers in question were tested for inclusion. Risk markers subject to evaluation were hs-CRP, ApoB, creatinine or estimated GFR, microalbuminuria, coronary artery calcium (CAC), carotid intima media thickness (CIMT), ankle brachial index (ABI), family history, and cardiorespiratory fitness.

It was determined that 1) routine measurement of CIMT was not recommended in clinical practice, and 2) where ApoB, chronic kidney disease, albuminuria, or cardiorespiratory fitness fit into ASCVD risk assessment is unclear. Therefore, no recommendation for or against the routine measurement of these risk markers was made. It was the expert opinion of the workgroup that clinicians may 3) consider using family history, hs-CRP, CAC score, and ABI to revise risk assessment upward (if indicated) if treatment decisions are unclear after 10-year ASCVD risk is calculated and risk/benefit evaluated as described in Table 4.40

<table>
<thead>
<tr>
<th>Measure</th>
<th>Support Revising Risk Assessment Upward</th>
<th>Do Not Support Revising Risk Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family history of premature CVD</td>
<td>First degree relatives who are: Male &lt; 55 years old, Female &lt; 65 years old</td>
<td>Any events occurring at ages &gt; 55 years (male) or &gt; 65 years (female)</td>
</tr>
<tr>
<td>hs-CRP (high sensitivity C-reactive protein)</td>
<td>≥ 2 mg/L</td>
<td>&lt; 2 mg/L</td>
</tr>
<tr>
<td>CAC Score (Coronary Artery Calcium)</td>
<td>≥ 300 Agatston units or ≥ 75th percentile for age, sex, and ethnicity</td>
<td>&lt; 300 Agatston units and &lt; 75th percentile for age, sex, and ethnicity.</td>
</tr>
<tr>
<td>ABI (Ankle Brachial Index)</td>
<td>≤ 0.9</td>
<td>≥ 0.9</td>
</tr>
</tbody>
</table>

*Table 4: Additional Risk Factors that May Revise Risk Assessment Upward*
Critical Question 2 – Risk Assessment Long Term

PCEs estimating absolute 10-year risk assessment for first ASCVD events currently apply only to individuals who are aged 40 to 79 years. While the work group developing these guidelines identified and evaluated 10 studies published between 1999 to 2009, the data provided for other age groups was found to be observational. Therefore, assessment algorithms were not developed for age groups 39 years and younger or 80 years and older due to the limitations identified within the published literature. Rather, the recommendation is that ASCVD risk factors should be assessed for each patient and that for individuals aged < 39 years or ≥ 80 years, providers should refer to current clinical guidelines for adult primary prevention, obesity, and pediatric prevention.41 It should be noted that the majority of risk factors identified within these 10 studies were modifiable risk factors. Therefore, referral to the 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults and to the 2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk is quite applicable in younger populations.42,45

For adults without clinical ASCVD, assessment for the presence of traditional risk factors should occur every 4 to 6 years in any individual aged 20 to 79 years. Traditional risk factors include age, gender, total cholesterol, HDL, SBP, blood pressure medication use, presence of diabetes, and current smoking status. For those individuals aged 40 to 79 years without clinical ASCVD, the PCEs should then be used to estimate 10-year ASCVD risk every 4 to 6 years. The online and mobile app ASCVD risk calculators will only calculate risk for patients aged 40 to 79 years. It will not provide any numbers if an age <40 or >79 is plugged into the calculator due to the age limitation on the PCEs.41 To address risk assessment in the younger population, it is recommended that a 30-year or lifetime risk assessment for ASCVD be performed in those aged 20 to 59 years of age who do not have clinical ASCVD and who are not at high short-term risk based on traditional risk factor assessment.40 The majority of people > 79 will have an ASCVD risk score of > 10% when using the PCE calculator. A discussion between the health care provider and patient regarding CV disease risk and potential benefits/harm of medication therapy is recommended. Limited data on statin therapy for primary prevention exists in those > 75, individual factors are to be considered.

Pharmacotherapy
Where ATP III provided specific treatment cutpoints to determine parameters for implementation of lifestyle modifications and/or drug therapy, the 2013 AHA/ACC Guidelines on the Assessment of Cardiovascular Risk did not include any assessment recommendations or guidelines for drug therapy decisions. This guideline focused specifically on 10-year, 30-year, and lifetime ASCVD risk assessment parameters. Instead, pharmacologic treatment based on 10-year ASCVD risk estimates and/or the presence or absence of clinical ASCVD is provided in the new cholesterol management guidelines.

C. 2013 AHA/ACC Guidelines on the Treatment of Blood Cholesterol

The first guideline, described above, focuses on 10-year ASCVD risk assessments for those without clinical ASCVD. Further assessment is detailed within the cholesterol treatment guidelines which include recommendations regarding ASCVD risk reduction, LDL and non-HDL goals, and medication safety recommendations. Each of these components will present some opportunity for assessment at community based screenings events and in clinical practice. For the purposes of this module, only the assessment characteristics of the guideline will be detailed.

Since ATP III guidelines focused on treating patients with dyslipidemia to certain LDL and non-LDL goals, the workgroup searched the literature for evidence supporting this practice. What the work group found was that for both primary and secondary prevention, the trials used fixed-dose statin therapy to decrease LDL. The primary outcomes were to determine if specific doses of statin therapy would decrease LDL, but no studies looked to see if specific LDL targets reduced ASCVD events. Studies comparing superiority of one cutpoint over the other related to improved ASCVD risk reduction were non-existent. Additionally, no studies were identified that evaluated non-HDL treatment goals and ASCVD risk reduction.

Because of the lack of literature supporting titration of drug therapy to specific LDL and/or non-HDL cutpoints, which was a mainstay of ATP III, it is now recommended not to treat to a specific LDL and/or non-HDL goal, but rather to treat a patient to a specific statin-intensity. This means that the widely recognized ATP III goals of LDL < 100, 130, or 160 mg/dL (or <70mg/dL) are out and appropriate statin dosing and drug choice is in.
While there were fixed dose statin trials that evaluated the effect of statin therapy on ASCVD risk and event reduction, trials of non-statin agents generally did not. Instead many of these trials (e.g., fibrates, ezetimibe) evaluated whether or not the drug in question could achieve a specific LDL cutpoint. Because these trials did not evaluate ASCVD reduction, they did not meet the inclusion criteria for guideline development. Therefore, the recommendations provided within the 2013 AHA/ACC Cholesterol Treatment Guidelines focus almost entirely on statin therapy, although some recommendations for non-statin therapy in patients who cannot tolerate statins are provided.\textsuperscript{41}

**Who will benefit from drug therapy?**

Literature review by the expert panel identified four groups who would benefit most from statin therapy. For these groups, adverse event risk is outweighed by reduction of ASCVD risk.\textsuperscript{41} The four statin benefit groups are listed in Table 5:

<table>
<thead>
<tr>
<th>Presence of Clinical ASCVD</th>
<th>LDL $\geq$ 190mg/dL</th>
<th>Persons with Diabetes Types 1 or 2 who are 40-75 years old with LDL 70 – 189 mg/dL &amp; without clinical ASCVD</th>
<th>Persons with LDL 70-189 mg/dL AND estimated 10-year ASCVD risk $&gt; 7.5%$ (using PCEs)</th>
</tr>
</thead>
</table>

*Table 5: Statin Benefit Groups\textsuperscript{41}*

Clinical ASCVD is defined as acute coronary syndromes, history of MI, stable or unstable angina, coronary or other arterial revascularization (e.g., stent), history of stroke, history of transient ischemic attack (TIA), and atherosclerotic peripheral arterial disease.\textsuperscript{41}

**What drug therapy is the best choice?**

As previously stated, trials included for review in the new cholesterol treatment guidelines used fixed doses of statin medications to achieve specific ASCVD event reduction. Therefore, when determining optimal drug therapy for treatment it was not a question of which drug to initiate, but rather at what intensity. Based on the doses used in clinical trials, three statin intensity groups were identified for primary and secondary ASCVD prevention.\textsuperscript{41} These statin intensity groups are identified in Table 6.

Defining the intensity of statin therapy was based on the percent LDL reduction achieved at specific statin dosing. For instance, high intensity statin therapy should reduce LDL by 50% or more when used
appropriately. Because no recommendation is made within the 2013 cholesterol guidelines to treat to a specific LDL and/or non-HDL cutpoint, the expected LDL reduction identified with a statin intensity can be used to monitor for adherence to therapy and therapy efficacy, while also monitoring for side effects or adverse events.

<table>
<thead>
<tr>
<th>Intensity of Therapy</th>
<th>Statin Dose</th>
<th>Expected % LDL Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High</strong></td>
<td>Atorvastatin 40 – 80mg, Rosuvastatin 20 – 40mg</td>
<td>≥ 50%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Moderate</strong></td>
<td>Atorvastatin 10 – 20mg, Rosuvastatin 5 – 10mg, Simvastatin 20 – 40mg, Pravastatin 40 – 80mg, Lovastatin 40mg, Fluvastatin XL 80mg, Fluvastatin 40mg twice daily, Pitavastatin 2 – 4mg</td>
<td>&lt; 50%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Low</strong></td>
<td>Simvastatin 10mg, Pravastatin 10 – 20mg, Lovastatin 20mg, Fluvastatin 20 – 40mg, Pitavastatin 1mg</td>
<td>&lt; 30%</td>
</tr>
</tbody>
</table>

*Table 6: Intensity of Statin Therapy and Recommended Doses*

**D. General Assessment of the Patient**

Assessment of a patient for cardiovascular risk and presence or absence of potential dyslipidemia is described over the course of the two guidelines described previously. To simplify the process, a step-wise approach may be used in community based screenings and in clinical practice.

1. Determine lipid levels.
2. Determine if the patient falls into one of the 4 statin benefit groups.
3. Determine 10-year ASCVD risk using the PCE calculator.
4. Determine the presence of additional risk factors.
5. Determine appropriate statin intensity based on ASCVD risk and/or statin benefit group.
6. Initiate statin drug therapy where appropriate.
Step 1: Determine Lipid Levels

Point-of-care testing has allowed lipid testing to be performed in settings outside of the typical office, hospital, or worksite wellness screening. (Detailed steps to perform lipid testing are discussed in Section IV.) Fasting conditions are necessary to perform lipid analysis for those parameters influenced by food intake (i.e., LDL, VLDL, and triglycerides). Therefore, if a patient has not fasted, only the total cholesterol (TC) and HDL can be accurately measured. As a general rule, a fasting state is considered one in which a person has not consumed any food or liquids other than water for 9 to 12 hours. Consumption of black coffee (e.g., no cream or sugar) and medications is also considered fasting.

As was previously discussed, increasing levels of LDL cholesterol have increasing detrimental effects on coronary heart disease. The 2013 AHA/ACC Cholesterol Guidelines did not address lipid classification. Therefore, classification continues to be based on studies reported by ATP I through III, which stratified cholesterol levels (LDL, HDL and total cholesterol) to assist with lipid management (Table 6).

<table>
<thead>
<tr>
<th><strong>LDL Cholesterol</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;100</td>
<td>Optimal</td>
</tr>
<tr>
<td>100-129</td>
<td>Near optimal/ above optimal</td>
</tr>
<tr>
<td>130-159</td>
<td>Borderline high</td>
</tr>
<tr>
<td>160-189</td>
<td>High</td>
</tr>
<tr>
<td>≥ 190</td>
<td>Very high</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Total Cholesterol</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;200</td>
<td>Desirable</td>
</tr>
<tr>
<td>200-239</td>
<td>Borderline high</td>
</tr>
<tr>
<td>≥ 240</td>
<td>High</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>HDL Cholesterol</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40</td>
<td>Low</td>
</tr>
<tr>
<td>≥ 60</td>
<td>High</td>
</tr>
</tbody>
</table>

Table 7: ATP Classification of LDL, Total and HDL Cholesterol (mg/dL)

Step 2: Determine Statin Benefit Group

The fundamental concept of the 2013 Cholesterol Guidelines is that there are specific groups of individuals where treatment with a statin achieves prevention of primary or secondary ASCVD events. This prevention benefit far outweighs the risk of adverse events. Therefore, the purpose of this second step is to aid in risk stratification by first identifying those patients at highest risk for clinical ASCVD, such as patients with known ASCVD. Patients with known ASCVD are at very high risk of having another ASCVD event, and multiple trials have demonstrated that lipid lowering (primarily LDL cholesterol)
decreases the occurrence of major coronary events, stroke, and cardiovascular related deaths. Patients without diagnosed ASCVD may have specific risk factors that would place them in a higher intensity treatment group. To review, the four statin benefit groups are listed in Table 8.

<table>
<thead>
<tr>
<th>Patients with clinical ASCVD</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Acute coronary syndromes (ACS)</td>
</tr>
<tr>
<td>- Myocardial infarction (MI)</td>
</tr>
<tr>
<td>- Angina – stable or unstable</td>
</tr>
<tr>
<td>- Coronary (or other) arterial revascularization</td>
</tr>
<tr>
<td>- Stroke</td>
</tr>
<tr>
<td>- TIA</td>
</tr>
<tr>
<td>- Atherosclerotic peripheral artery disease</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patients with LDL &gt; 190mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with Diabetes (Type 1 or 2), who are 40 to 75 years old, who do not have clinical ASCVD, but have LDL 70 – 189mg/dL.</td>
</tr>
</tbody>
</table>

| Patients with LDL 70 – 189mg/dL who also have an estimated 10-year ASCVD risk of 7.5% or more. |

**Table 8: The Four Statin Benefit Groups**

**Step 3: Determine 10-year ASCVD Risk**
The purpose of determining a patient’s 10-year risk of ASCVD is to determine the aggressiveness of treatment needed to prevent ASCVD. Information needed to complete the risk assessment includes patient’s sex, age, total cholesterol and HDL values, smoking status, systolic blood pressure, and whether the patient is currently being treated with an antihypertensive agent. The online ASCVD-Risk-Estimator is a useful tool.

**Step 4: Determine Presence of Additional Risk Factors**
When assessing a patient for ASCVD risk, other known risk factors for ASCVD may be used to titrate the risk assessment upward to determine if a higher intensity of treatment is warranted. The only risk factor identified in the 2013 assessment guidelines that may be identified in a screening setting is family history of premature CHD. Other risk factors, such as hs-CRP, CAC score, and ABI may only be measured by venipuncture (hs-CRP, CAC) or by a blood pressure cuff and Doppler device (ABI). Known risk factors for ASCVD also include cigarette smoking, hypertension (systolic BP ≥ 140mmHg or currently on antihypertensive medication), low HDL cholesterol, and age. These risk factors are addressed using the Pooled Cohort Equations for 10-year ASCVD risk in primary prevention.
Other risk factors which are thought to increase the risk of ASCVD, but are not included in the 2013 AHA/ACC risk assessment or treatment guidelines, include:

1. Elevated blood glucose levels
2. Obesity
3. Atherogenic diet
4. Physical inactivity
5. Thrombogenic/hemostatic factors
6. Chronic kidney disease
7. HIV/AIDS

Factors 1-4 are addressed in the 2013 AHA/ACC Guidelines on Lifestyle Management to Reduce Cardiovascular Risk.42

**Step 5: Determine Appropriate Statin Intensity Based on ASCVD Risk and/or Statin Benefit Group.**

Once a 10-year ASCVD risk assessment has been calculated, it is then necessary to stratify the patient’s information into Figure 11 (next two pages) which reflects the new recommendations for lipid management.40, 41
Heart-healthy lifestyle habits are the foundation of ASCVD prevention
(See 2013 AHA/ACC Lifestyle Management Guideline)

Clinical ASCVD

Age ≥21 y and a candidate for statin therapy

Yes

Age ≤75 y
High-intensity statin
(Moderate-intensity statin if not candidate for high-intensity statin)

Age >75 y OR if not candidate for high-intensity statin
Moderate-intensity statin

No

High
Daily dose lowers LDL-C by approx. ≥50%

Moderate
Daily dose lowers LDL-C by approx. 30% to <50%

Definitions of High- and Moderate-Intensity Statin Therapy
(See Table 5)

LDL-C ≥190 mg/dL

Yes

High-intensity statin
(Moderate-intensity statin if not candidate for high-intensity statin)

No

Diabetes
LDL-C 70-189 mg/dL
Age 40-75 y

Yes

Moderate-intensity statin

No

Estimated 10-y ASCVD risk ≥7.5%

High-intensity statin

No

DM age <40 or >75 y or LDL-C <70 mg/dL

Primary prevention
(No diabetes, LDL-C 70 to 189 mg/dL, and not receiving statin therapy)
Estimate 10-y ASCVD risk every 4-6 y using Pooled Cohort Equations

<5%
10-y ASCVD risk

Yes

Age <40 or >75 y and LDL-C <190 mg/dL

≥7.5%
10-y ASCVD risk
(Moderate- or high-intensity statin)

5% to <7.5%
10-y ASCVD risk
(Moderate-intensity statin)

No

In selected individuals, additional factors may be considered to inform treatment decision making

Clinician-Patient Discussion
Prior to initiating statin therapy, discuss:
1. Potential for ASCVD risk-reduction benefits
2. Potential for adverse effects and drug–drug interactions
3. Heart-healthy lifestyle
4. Management of other risk factors
5. Patient preferences
6. If decision is unclear, consider primary LDL-C ≥160 mg/dL, family history of premature ASCVD, lifetime ASCVD risk, abnormal CAC score or ABI, or hs-CRP ≥2 mg/L

Emphasize adherence to lifestyle
Manage other risk factors
Monitor adherence

No to statin

Yes to statin

Encourage adherence to lifestyle
Initiate statin at appropriate intensity
Manage other risk factors
Monitor adherence
(See Fig 5)
Figure 11: Lipid Assessment/Management Algorithms from the 2013 ACC/AHA Guidelines on the Treatment of Blood Cholesterol⁴¹
Step 6: Initiate Statin Drug Therapy Where Appropriate

Many patients will require the use of pharmacologic intervention to achieve ASCVD risk reduction. Similarly to the Therapeutic Lifestyle Changes (TLC) implementation, a stepwise approach for initiating statin therapy is used (Figure 12). Once the appropriate statin intensity is achieved or close to being achieved, focus can then shift to treating non-LDL dyslipidemias. It is important to note that continued use of TLC in conjunction with drug therapy is essential to achieving patient goals.40-41

Implementing drug therapy is beyond the scope of lipid screening and will not be discussed further in this module, but Table 9 briefly describes the common agents used in lipid lowering.41 Once statin intensity treatment goals are achieved or close to being achieved, emphasis then shifts toward treatment of other lipid risk factors (e.g., significantly elevated triglycerides).
<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Agents (Daily doses)</th>
<th>Effect on Lipids</th>
<th>Major Side Effects</th>
</tr>
</thead>
</table>
| **HMG CoA reductase inhibitors (statins)** | Atorvastatin (10-80 mg)  
Fluvastatin (20-80 mg)  
Lovastatin (20-80 mg)  
Pravastatin (20-40 mg)  
Rosuvastatin (5-40 mg)  
Simvastatin (20-40 mg) | LDL ↓ 18-55%  
HDL ↑ 5-15%  
TG ↓ 7-40% | - Myopathy  
- Increased liver enzymes |
| **Bile acid sequestrants** | Cholestyramine (4-16 g)  
Colesevelam (2.6-8.3 g)  
Colestipol (5-20 g) | LDL ↓ 15-30%  
HDL ↑ 3-5%  
TG ↑ 0-12% | - GI distress  
- Constipation  
- Decreases absorption of other drugs |
| **Nicotinic acid**         | Immediate release (1.5-3 g)  
Extended release (1-2 g) | LDL ↓ 2-25%  
HDL ↑ 15-35%  
TG ↓ 20-50% | - Flushing  
- Hyperglycemia  
- Hyperuricemia  
- GI distress  
- Hepatotoxicity |
| **Fibric acid derivatives** | Fenofibrate (200 mg)  
Gemfibrozil (1200 mg) | LDL ↓ 5-20%  
HDL ↑ 10-20%  
TG ↓ 20-50% | - GI distress  
- Gallstones  
- Myopathy |
| **Antilipemics**           | Ezetimibe (10 mg) | LDL ↓ 18%  
HDL ↑ 1%  
TG ↓ 8% | - Headache  
- Fatigue  
- Diarrhea |
| **PCSK9 Inhibitors**       | Alirocumab (75-150mg SQ every 2 weeks)  
Evolocumab (140mg SQ every 2 weeks or 420mg every month) | LDL ↓ 40-64%  
HDL no effect  
TG no effect | - Injection site reactions  
- Arthralgias  
- Myalgias |

LDL = low density lipoprotein cholesterol, HDL = high density lipoprotein cholesterol, TG = triglycerides

**Table 9: Pharmacologic Agents Used in Lipid Lowering**

### E. Therapeutic Lifestyle Changes (TLC)

Therapeutic lifestyle changes should be initiated alone or in addition to pharmacologic therapy to reduce the risk of ASCVD. The 2013 cholesterol treatment guidelines identify lifestyle modification as an integral component of ASCVD risk reduction and cite guidance from the 2013 AHA/ACC Lifestyle Management Guideline. This guideline, which addresses lifestyle changes to reduce both blood pressure and cholesterol, was also released in November 2013 and should be used in conjunction with the cholesterol assessment and treatment guidelines. As were the others, the lifestyle management guideline was developed using the IOM best practices in guideline development. It also addressed specific critical questions, focusing on dietary patterns (not specific dietary components), sodium and potassium intake, and the effect of physical activity. These three foci are in line with previous dietary recommendations provided as Step I and II diets from ATP I and II, the TLC diet from ATP III, and the...
2010 Dietary Guidelines from the Department of Agriculture and Department of Health and Human Services.\textsuperscript{8, 42, 57}

1. Dietary Patterns
The Lifestyle Guideline evaluated randomized controlled trials reporting ASCVD outcomes based on the Mediterranean diet, DASH diet (Dietary Approaches to Stop Hypertension), and combinations of diets based on glycemic index and DASH variations. Specifically, the lifestyle guideline recommends following specific dietary patterns versus focusing on one specific component of the diet.\textsuperscript{42} Additionally, AHA/ACC/TOS guidelines on the management of overweight and obese adults (also published in November 2013) provided recommendations regarding specific diets, nutrients, and recommended intakes. These are summarized in Table 10 with the TLC diet summarized in Table 11. Characteristics of dietary fats, which are quite useful when counseling patients on fat intake to reduce ASCVD risk, are summarized in Table 12. Appendix D provides a list of foods which fit many of these dietary patterns.\textsuperscript{42, 45}

<table>
<thead>
<tr>
<th>Diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>High protein diet</td>
</tr>
<tr>
<td>• % calories from: protein (25%); fat (30%); carbohydrate (45%)</td>
</tr>
<tr>
<td>• Use of foods that achieve a calorie deficit.</td>
</tr>
<tr>
<td>Zone-type high protein</td>
</tr>
<tr>
<td>• 5 meals per day</td>
</tr>
<tr>
<td>• % calories from: protein (30%); fat (30%); carbohydrate (30%)</td>
</tr>
<tr>
<td>• No formal calorie restriction but achieved a calorie deficit.</td>
</tr>
<tr>
<td>Lacto-ovo-vegetarian</td>
</tr>
<tr>
<td>• Calorie restriction</td>
</tr>
<tr>
<td>Low-calorie</td>
</tr>
<tr>
<td>• Calorie restriction</td>
</tr>
<tr>
<td>Low-carbohydrate</td>
</tr>
<tr>
<td>• Less than 20g per day carbohydrate to start.</td>
</tr>
<tr>
<td>• No calorie restriction but achieved a calorie deficit.</td>
</tr>
<tr>
<td>Low fat, vegan style</td>
</tr>
<tr>
<td>• 10-20% total calories from fat</td>
</tr>
<tr>
<td>• No calorie restriction but achieved a calorie deficit.</td>
</tr>
<tr>
<td>Low-glycemic load</td>
</tr>
<tr>
<td>• With or without calorie restriction, but achieved a calorie deficit.</td>
</tr>
<tr>
<td>High dairy, lower fat, with or without increased fiber and/or low glycemic load foods.</td>
</tr>
<tr>
<td>• 30% or less of calories from fat</td>
</tr>
<tr>
<td>• 4 servings per day of dairy</td>
</tr>
<tr>
<td>• Calorie restriction</td>
</tr>
</tbody>
</table>
Macronutrient-targeted
- Protein – 15% or 25% total calories
- Fat – 20% or 40% total calories
- Carbohydrate – 35%, 45%, 55%, or 65% total calories.
- Calorie restriction

European Association for the Study of Diabetes Guidelines
- Targets food groups versus formal calorie restriction.

Table 10: Dietary Patterns for Weight Management\textsuperscript{42, 45}

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Recommended Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated fat</td>
<td>- &lt; 7% of total calories &lt;br&gt; - Trans fatty acids should be kept at a low intake</td>
</tr>
<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 &lt;br&gt; - Predominantly from complex carbohydrates (i.e., whole grains, fruits and vegetables)</td>
</tr>
<tr>
<td>Fiber</td>
<td>- 20-30 grams daily</td>
</tr>
<tr>
<td>Protein</td>
<td>- Approximately 15% of total calories</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>- &lt; 200 mg daily</td>
</tr>
<tr>
<td>Total calories</td>
<td>- Balance energy intake and expenditure to maintain desirable body weight or to prevent weight gain</td>
</tr>
</tbody>
</table>

Table 11: Nutritional Composition of the TLC Diet\textsuperscript{8}

<table>
<thead>
<tr>
<th>Type of fat</th>
<th>Good</th>
<th>Bad</th>
<th>Ugly</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical appearance at room temp.</td>
<td>Mono-unsaturated</td>
<td>Poly-unsaturated</td>
<td>Saturated</td>
</tr>
<tr>
<td>Liquid</td>
<td>Liquid</td>
<td>Solid</td>
<td>Solid (due to hydrogenation of liquid oils)</td>
</tr>
<tr>
<td>Dietary sources</td>
<td>- Nuts, avocados, &amp; olives &lt;br&gt; - Olive, peanut &amp; canola oils</td>
<td>- Fish (ex., salmon) &lt;br&gt; - Corn, soybean, safflower, sunflower &amp; sesame oils</td>
<td>- Meats, poultry, &amp; dairy &lt;br&gt; - Coconut &amp; palm oils &lt;br&gt; - Commercially fried &amp; baked goods</td>
</tr>
<tr>
<td>Effect on cholesterol</td>
<td>LDL</td>
<td>HDL</td>
<td></td>
</tr>
<tr>
<td>↓</td>
<td>No effect</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>↑</td>
<td>No effect</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

Table 12: Characteristics of Dietary Fats\textsuperscript{57, 58}
Sodium and Potassium Intake
The focus on sodium and potassium intake is to reduce blood pressure and, therefore, reduce ASCVD events. Because this is a blood pressure issue primarily, and does not affect cholesterol levels, it will not be discussed in this module.

2. Physical Activity
The effect of physical activity on chronic disease reduction has been well reported, with ASCVD occurring less frequently when individuals have higher levels of activity. The Lifestyle Guideline workgroup evaluated systematic reviews and meta-analyses and determined the following:

- Aerobic physical activity decreased LDL by 3 to 6 mg/dL and non-HDL an average of 6mg/dL, but no consistent effect on TG or HDL was found.
- Resistance training had no effect on HDL, but decreased LDL, non-HDL, and TG an average of 6 to 9mg/dL.

The recommendation provided by the Lifestyle Guideline to decrease LDL and non-HDL is that adults should participate in moderate-to-vigorous physical activity at least 3 to 4 times per week with each session lasting 40 minutes.

3. Use of Dietary Supplements
Dietary supplements have been a recent focus in decreasing the development of CHD with the main agents being omega-3 fatty acids, niacin, and red yeast rice.

Omega-3 Fatty Acids
Omega-3 fatty acids are polyunsaturated fatty acids thought to decrease hepatic production of triglycerides and subsequently VLDL, thereby decreasing the potential for atherogenesis.

Omega-3 fatty acids have two main sources:
1. Fish oils which contain eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and are found in oily fish
2. Alpha-linolenic acid (ALA) found in soybeans, flaxseed and canola oils, green leafy vegetables and walnuts

Clinical evidence has shown benefit to increasing omega-3 content in the diet as a method of primary and secondary prevention of CHD. The American Heart Association (AHA) endorses the use of omega-3...
When recommending a fish oil supplement, a few points regarding labeling and side effects should be reviewed with patients. For appropriate dosing, a fish oil product should be evaluated for its content of EPA and DHA, which is what the AHA recommendations are based upon. For example, if the patient wishes to take 1,000mg of EPA and DHA daily based on the AHA recommendations for diagnosed heart disease, the labeling may be confusing. The product may be labeled as 1,200mg of fish oil concentrate. However, it contains less EPA and DHA, which are only a portion of the omega-3 fatty acids in the product. The content labeling, usually listed on the back of the product bottle, will list the amount of EPA and DHA contained in 1,200mg of fish oil. Therefore, if the label states it contains 236mg of EPA plus 276mg of DHA, the product contains 512mg of EPA and DHA and the person would need to take 2 capsules daily in order to get 1,000mg daily of EPA and DHA.

There are a few precautions with fish oil supplements. Patients with a fish allergy should not take these supplements. Additionally, omega-3 acids appear to have a “blood-thinning” effect and should be used with caution in patients taking anticoagulants or who have a history of bleeding disorders. Omega-3 fatty acid supplementation can raise LDL cholesterol which may be more pronounced in people with significantly elevated TG levels. Cod liver oil should be avoided as a source of omega-3 fatty acids due to the high vitamin A content, which can lead to toxicity.

<table>
<thead>
<tr>
<th>Patient Population</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosed heart disease</td>
<td>By means of diet ± supplements, consume 1 gram of EPA plus DHA daily.</td>
</tr>
<tr>
<td>No diagnosed heart disease</td>
<td>Increase dietary intake of omega-3 containing foods</td>
</tr>
<tr>
<td>Patients who would benefit from triglyceride lowering</td>
<td>Consume 2-4 grams of EPA plus DHA daily. (At these doses, dyspepsia and bad breath may occur.)</td>
</tr>
</tbody>
</table>

*Table 13: American Heart Association Recommendations for Use of Omega-3 Fatty Acids*
Niacin (nicotinic acid)
Niacin, a form of vitamin B3, has long been a dietary supplement added to statin and non-statin treatment regimens for dyslipidemias. Niacin reduces LDL cholesterol and triglycerides while raising HDL cholesterol.

Recently, several non-statin drug therapies have fallen out of favor due to lower efficacy rates and concerns over adverse drug effects. In 2011, the AIM-HIGH trial reported results regarding the use of niacin to reduce CV events if added to simvastatin ± ezetimibe. About 3400 participants received extended release niacin or placebo in addition to current statin therapy. An increase of HDL was seen in the niacin group (25%) versus the placebo group (10%). However, while CV events were reduced in the niacin group, when compared to placebo, the reduction was not clinically significant. Based on this summary and previously published evidence for niacin, the 2013 AHA/ACC cholesterol treatment guidelines recommended the addition of niacin to current statin therapy only if an insufficient response to statin therapy was found and the benefit outweighed the risk.

Higher doses of niacin are typically required to positively benefit lipid serum concentrations. Although over-the-counter niacin is available as both immediate and extended-release products, it is not currently recommended to take as a dietary supplement for cholesterol management due to a higher risk of dermatologic side effects (IR) and hepatotoxicity (SR) compared to the prescription product (Niaspan ER). Several adverse effects have been found with niacin, including flushing, itching, and headache (immediate release); liver toxicity and GI side effects (extended-release); peptic ulcer activation (both); increased uric acid (both); and transient increases in blood sugar and reduced insulin sensitivity (both). Niacin is contraindicated in active liver disease, active peptic ulcer disease, and gout.

Red Yeast Rice
Red yeast rice is made by fermenting rice grains with red yeast (Monascus purpureus). This product is available as a dietary supplement and contains monacolin K, which contains 'statin'-like compounds such as lovastatin. Red yeast rice can lower LDL and some products may lower LDL similarly to lovastatin 10mg daily dose.

Due to the constituents of red yeast rice, this supplement may have side effects and drug interactions similar to statin medications. Red yeast rice may also contain citrinin, a mycotoxin, which may lead to
liver and kidney damage. Currently there is no CV outcome data for red yeast rice products and limited long-term safety data. This product is not currently recommended in treatment guidelines for the management of dyslipidemias.

**F. Treatment of Elevated Triglycerides & Low HDL Cholesterol**

**Elevated Triglycerides**

Currently, the 2013 cholesterol treatment guidelines refer to the 2011 AHA Scientific Statement on Triglycerides and Cardiovascular Disease for triglyceride treatment recommendations. Table 14 summarizes triglyceride classification and treatment recommendations based on this statement.

<table>
<thead>
<tr>
<th>Value (mg/dL)</th>
<th>Classification</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 150</td>
<td>Normal</td>
<td>None required</td>
</tr>
</tbody>
</table>
| 150-199      | Borderline     | - Intensify weight management  
- Increase physical activity  
- Avoid/limit alcohol |
| 200-499      | High           | - Intensify weight management  
- Increase physical activity  
- Consider intensifying statin therapy or adding a fibrate, fish oils or niacin |
| ≥ 500        | Very High      | - Primary aim is to prevent pancreatitis. Goal is to lower triglycerides to < 500mg/dL  
1. Very low fat diet (≤ 15% calories from fat)  
2. Weight management and increased physical activity  
3. Add fibrate or niacin |

*Table 14: Classification and Treatment for Elevated Triglycerides*

**Low HDL Levels**

HDL levels were not addressed in the 2013 cholesterol treatment guidelines. In general; lifestyle modifications have the most effect on HDL increases, although statin and non-statin therapies do increase HDL a small percentage. Medication therapy targeted to raise HDL levels has not proven to be an effective strategy to reduce CV events and improve overall outcomes. Currently, drug therapy is not recommended for the sole purpose of increasing low HDL levels.
IV. Screening for Dyslipidemias

A. Who should be tested?

It is estimated that more than 50% of Americans are unaware they have high cholesterol levels. Additionally, one third of those treated with medications are not at their LDL goal. According to the 2013 AHA/ACC Assessment Guidelines, the following recommendations reflect who should be tested for lipid abnormalities.40

1. A fasting lipoprotein profile including major blood lipid fractions, i.e., total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride, should be obtained at least once every 4 to 6 years in adults age 20 and over.

2. More frequent measurements are required for persons with multiple risk factors, including:
   (a) Cigarette smoking
   (b) Hypertension
   (c) Low HDL cholesterol
   (d) Age: men ≥ 45 years old & women ≥ 55 years old
   (e) Significant family history of cardiovascular disease: male relative before age 55 and female relative before age 65.

3. For adults aged 40 to 79 years, who do not have clinical ASCVD, calculate 10-year ASCVD risk every 4 to 6 years.

B. Point-of-Care Lipid Testing

1. CardioChek Plus®

The CardioChek Plus® is a CLIA-waived, portable device designed to provide point-of-care analysis of lipid levels obtained from capillary or venous blood samples.68

The analyzer uses reflectance photometry and electrochemical biosensor technology. Both methods measure the enzymatic reaction that occurs when whole blood is placed into the sample well. Reflectance photometry is a method to measure the amount of light reflected from a solid surface, to determine cholesterol levels. Once a blood sample is dispersed into the sample well, the cassette separates the blood cells from plasma allowing the plasma to interact with reagent pads causing a color change to the pads. The resulting current from blood contacting the electrochemical strip is measured and then converted. The CardioChek Plus® detects the color change and current and quantifies the amounts of
substances into mg/dL. The reflectance and electrochemical tests may be run separately or simultaneously.

**Precision and Accuracy**

Precision is the ability of a test to consistently reproduce results and accuracy reflects the ability of a test to correctly detect what it should be detecting. The total error (TE) of an analytical device accounts for both precision [represented by % coefficient of variation (CV)] and accuracy (represented by % bias). The following formula is used to calculate total error.

\[
\text{Total Error (TE)} = \% \text{ bias} + (1.96 \times \% \text{ CV})
\]

The National Cholesterol Education Program (NCEP) has issued guidelines specifying an acceptable upper limit of % bias, % CV, and TE for total cholesterol (TC), LDL, and HDL. NCEP has identified the following acceptable percent total errors: ≤ 8.9% for TC; ≤ 13% for HDL; and ≤ 12% for LDL. Multiple studies have demonstrated the use of the CardioChek Plus® to be precise and accurate in its measurement of TC (4.7% TE), HDL (5.9% TE), and LDL (5.3% TE).

It should be noted that the CardioChek Plus® does not directly measure LDL or VLDL but derives them from the following Friedewald equation.

\[
\begin{align*}
\text{Total cholesterol (TC)} &= \text{HDL} + \text{LDL} + \text{VLDL} \\
\text{TC - HDL} &= \text{LDL} + \text{VLDL} \\
\text{(VLDL is 20% of the triglyceride level)}
\end{align*}
\]

Therefore, \( \text{LDL} = \text{TC} - \text{HDL} - \text{TG (0.2)} \)

**2. Components of the CardioChek Plus® (68)**

- CardioChek Plus® analyzer
- 4 AA Batteries
- Analyzer check strip
- User manual
- Capillary tubes and plunger
- Lancets
- MiniPet pipette (for running control samples)
- Pipette tips
- Printer & cable assembly
Additional materials needed to perform the testing are:

- Biohazard waste container (See Appendix B for the policy & procedure utilized by IPHARM for the safe handling of blood products.)
- Alcohol swabs
- Disposable gloves (preferably latex free, in case of latex-allergic patients)
- Gauze or tissues
- Adhesive bandages

3. **Components of the PTS Panels Tests Strips & MEMo Chip**
   - Sample well is the area in which the blood sample is placed for analysis.
   - Magnetic strip contains test information for the analyzer to determine what type of tests to perform.
   - Various reagents which are the chemicals for each test.
   - The MEMo Chip is lot-specific. A new MEMo Chip is included with each package of test strips and should be used for those test strips only.
     - Contains the lot number, expiration date, test name, and calibration curve.
4. **Setting Up the CardioChek Plus®**

Locational requirements for the CardioChek Plus® machine are:

- Room temperature 68-80°F (20-27°C)
- A stable work surface
- No direct heat (oven or room heater)
- No direct light (sunlight or spotlight)

**Steps to set up CardioChek Plus®**

1) Ensure the batteries have power or have been freshly changed.
2) Connect printer port to analyzer.
3) Use Section III of the CardioChek Plus User Guide to navigate through the analyzer set-up menus to set date/time, units, sound, Wi-Fi, and printer.

5. **Quality Assurance of the CardioChek Plus®**

To ensure accurate results from the CardioChek Plus®, an analyzer check strip has been provided with the machine. Additionally, ChekMate Quality Control is an available kit that may be used with PTS Panels test strips quality control materials. The ChekMate kit also checks optics, power, MEMo chip functionality, and endpoint algorithm processing. This kit is recommended for daily use.

a) **Analyzer check strip.** This gray strip reads a specific calibrated reflectance and is used to check the optical and electronic systems of the analyzer. Each day prior to using the
Analyzer, a check test should be performed. A check should also be performed if the analyzer is ever dropped or if the results of tests are not consistent with those expected.

To perform the analyzer test:

1. Turn machine on and bypass “Install MEMo Chip” or “Run Test” by pressing the Next button until “Utility” is displayed. Press Enter.

2. When “Check Strip” is displayed, press Enter.

3. When “Insert Strip” is displayed, insert the analyzer check strip into the reflectance strip slot ribbed side up.

   The analyzer will display either “Passed” or “Failed”. If the test fails, repeat the test after cleaning the machine. If repeated tests fail, contact the manufacturer.

b) Running a test using control samples is a method to determine if the analyzer is reading lipid samples accurately. CardioChek manufactures quality control solutions to be used with an actual test strip: Level 1 for low lipid levels and Level 2 for high lipid levels. The control solutions may be kept refrigerated at 2-8°C (35-46°F), or may be stored in a cool dry place at 68-86°F (20-30°C). They are stable after opening for 10 months when stored at room temperature. It is suggested to run control samples when a new lot of test cassettes is being used for the first time, or if repeated errors are occurring when running patient tests.

Running a control sample:

1. Turn on the analyzer and insert MEMo Chip for the current test strip lot in use.

2. Press Next until “Utility” is displayed and press Enter. Then, press “Next” again until “Run Control” is displayed and press Enter.

3. Insert the reflectance strip and wait for “Apply Sample” to be displayed.

4. Apply a large drop of control solution to the application window without touching the bottle to the test strip.

5. “Control” should be displayed on the analyzer while it is testing. Results should be available within 1-2 minutes.

6. Compare the results to the Quality Control Range Card that was provided with the control solution. The analyzer will also store the control result in “Memory”. Control range cards for specific lot numbers are also available at:

   http://www.cardiochek.com/products/professional-strips/control-range-values
6. **Use of the CardioChek Plus**

The information found in Appendix E provides a quick reference on the materials included in the testing, how to set up the testing device, and how to run a sample.

7. **Performing a Finger Stick for Blood Collection**

Obtaining an adequate quantity of blood from a finger stick is sometimes the most challenging component of lipid screening. Among the following steps below are some suggestions to assist with minimizing collection difficulties.

   a. Patients with thick calluses or poor peripheral circulation may be the most difficult. Warming the hands under warm water or holding hand warmers can substantially help with obtaining an adequate blood supply down to the finger tips. For patients with thick calluses, try to look for a finger with the least amount of callus.

   b. It is generally a better idea to obtain the blood sample from the non-dominant hand, as an adhesive bandage will be placed on the finger utilized for the blood sample, and the non-dominant hand may be less callused.

   c. Inspect the patient’s fingers and gently press on the tips of the finger tips to assess which fingertip has good blood return. (The tip should become a rosy-red color compared to the other finger areas.) The middle (3rd) finger or the ring (4th) finger is generally a good choice to perform the finger stick.

   d. Cleaning of the finger area with an alcohol swab prepares the site for penetration with the lancet. The fingertip needs to be completely dry prior to performing the finger stick.

   e. The recommended placement of the lancet on the finger is on the outside edge of the fingertip. (About the 2 o’clock position when looking at the fingertip.) Place the lancet firmly on the tip and push downward AND hold in place for two seconds. Holding the lancet in place prevents the reflexive impulse to pull the lancet away from the skin before it has fully penetrated.

   f. When trying to get the area which was pricked to bleed, keep in mind the lancet provides a slit-like cut which responds better to gently opening the cut as opposed to squeezing the cut (which pushes the cut edges together.) A drop of blood should appear with gentle massaging. Try to avoid “milking” the finger (which is squeezing along the finger towards the tip), as this may lead to an inaccurate result.
g. The first drop of blood needs to be removed with a gauze pad or tissue. Gently repeat the above step to get the finger to produce a second blood droplet.

h. Placing the collection capillary tube at a slight downward angle to the blood droplet and gently touching the side of the droplet with the capillary tube should allow the blood to enter the tube. Avoid getting air bubbles in the tube, as this may interfere with the results of the test.

i. Once the blood reaches the designated black line on the capillary tube, provide the patient with a tissue or gauze pad to press against the bleeding finger. This should allow the person performing the test the time to place the blood sample in the test strip well for analysis. The collected sample needs to be expelled into the test strip within 5 minutes of collection (preferably sooner) to prevent clotting. Pressing on the plunger of the capillary tube pushes the blood out of the tube; therefore, it is necessary to have the test strip immediately available.

j. Insert the test strip into the CardioChek Plus analyzer. When “Apply Sample” is displayed, apply the blood to the test strip. It will take about 1 to 2 minutes to analyze. When the test is done, a printout will be provided on the printer. (See sample at right.)

k. If an error message occurs, the user manual provides a comprehensive table of actions to take for problems that arise.

8. Interpretation of Results
The interpretation of the lipid results needs to be in context with patient-specific risk factors. Therefore, once the results are obtained, they should be incorporated into the 2013 AHA/ACC Assessment and Treatment Guidelines for Cholesterol. Additionally, Appendix F provides a patient specific assessment tool to help guide the health screener through a patient’s lipid assessment.

V. Video of IPHARM Screening Event
The MTGEC/IPHARM program provides wellness screening for people throughout Montana that might otherwise be unable to access service. Additionally, the program provides patient care experience for students in their last professional year in the study of pharmacy, physical therapy, nursing and other health care fields.
The following video illustrates a typical screening for lipids. The video shows how to set up, prepare and run the CardioChek Plus®. The results obtained during this screening should be used to counsel the patient highlighting diet, exercise and lifestyle modifications. Watching the video is a component of the contact hours for this module and should be completed at this time.

**CardioChekPlus® Video**

**VI. Useful Websites**

🌟 Highly recommended websites for further understanding of key concepts related to geriatric screening for lipids.

1. Governmental
   a. [National Heart, Lung and Blood Institute](https://www.nhlbi.nih.gov) 🌟
   b. [National Cholesterol Education Program (NCEP)](https://www.nhlbi.nih.gov) 🌟
   c. [Administration on Aging](https://www.aoa.gov)
   d. [Montana Cardiovascular Health Program](https://www.montanaheart.org)

2. Cardiovascular Health
   a. [American Heart Association](https://www.heart.org) 🌟
   b. [American College of Cardiology](https://www.acc.org) 🌟
   c. [Cardiosource (American College of Cardiology)](https://www.cardiosource.org) 🌟
   d. [Lipids online](https://www.LipidExpert.org): an online resource for clinicians, researchers, & educators related to atherosclerosis, dyslipidemias, and lipid management.
   e. [The Heart](https://www.theheart.org): an online resource for healthcare professionals which provides information on caring for people with disorders of the heart and circulation, and on preventing such disorders.
   f. [National Lipid Education Council’s cholesterol management resource](https://www.ncep清算) 🌟

3. Miscellaneous
   a. [Clinical guidelines on obesity and overweight](https://www.clinicalguidelines.org) 🌟
   b. [Your Guide to Lowering Your Cholesterol with TLC](https://www.yourguide.com)
VII. References


Appendix A:  IPHARM AUTHORIZATION TO TEST FORM

IPHARM will provide SCREENING test(s) to you today at your request and for the specific purpose of providing you with information that may relate to your health. An explanation of the results and how they may relate to your health will be provided by IPHARM personnel.

What will happen today?
IPHARM personnel will conduct the test(s) you have requested, obtain the results, and explain the results to you. You will receive the original and only copy of your complete test results. IPHARM personnel will record your results for statistical purposes on a data sheet that does NOT include your name. The results will be used in IPHARM reports compiled with all other test results and your results will never be individually identified or connected to you without your written permission. IPHARM will keep your agreement to be tested and the results sheets confidential, separated, and secure. IPHARM further agrees to use personnel trained to provide these tests and to follow general methods approved for these tests. IPHARM agrees to exercise due caution in those areas associated with the tests provided.

What do I agree to when I sign below?
By signing below, you indicate you have read and understand this form. You agree that IPHARM has no responsibility to contact your health care provider. You agree to receive testing from IPHARM for the test(s) you have requested. Finally, you agree to hold harmless IPHARM personnel for acts beyond their control or outside their responsibility in providing you these tests. A copy of this form is available upon request.

Do I need to give these results to my health care provider?
IPHARM strongly encourages you to take your results to your provider when you next schedule an appointment. In some cases, IPHARM may suggest you schedule an appointment with your provider. IPHARM reminds you that a single screening test result, whether abnormal or normal, does not provide you or your provider enough information on which to make therapeutic decisions about your health. However, the tests may indicate that you should have further tests done or undertake changes in your life that could improve your health.

____________________________________________     _____________
Client Signature                                      Date
__________________________________________________
Printed name of client                                Daytime phone number

_____ Initial here if you will allow IPHARM to take a picture of you during testing to be used for publicity of the IPHARM program.

____________________ Client record number (record on results sheet also)
Appendix B: Protection of Staff & Public from Blood-Borne Pathogens

IPHARM will follow procedures outlined below in order to protect individuals administering finger-stick tests and individuals exposed to finger-stick test waste that might cause injury. In all cases, IPHARM’s intent is to protect staff and the public from potential injury.

**Procedure 1**
All IPHARM workers will be instructed by an IPHARM Clinical Pharmacist Specialist (CPS), Principle Investigator (PI), or Project Coordinator (PC) before any tests are completed.

**Procedure 2**
All IPHARM workers administering finger-sticks must wear non-latex gloves on both hands prior to administering any finger-stick.

**Procedure 3**
All IPHARM workers will administer finger-stick tests only after training on the proper method for doing this procedure and only after observation of an instructor (PI, CPS, or PC) administering this test.

**Procedure 4**
The following items must be placed in a “Sharps” container after use:

- Lancets (closed, open, or retractable), pipettes or other collection tubes, any other devices or potentially sharp objects that are used and come into contact with blood or body fluids. Items that may be discarded in a plastic garbage bag include the following: alcohol swabs, tissues including tissue with blood, used Band-Aids, and gloves that are properly removed and folded inside out into one another (gloves with blood may be handled in this manner also).

**Procedure 5**
After a person has a finger-stick test, they should be told to compress the site for at least 3-5 minutes with gentle but firm pressure. The IPHARM staff member working the station should inspect the site after this in order to determine if the person’s lancet wound has stopped bleeding. If not, an adhesive bandage shall be applied.

**Procedure 6**
In the event any worker believes they have come into contact with blood or body fluid and such contact has consisted of contact with an open sore or mucous membrane, the worker should immediately contact the IPHARM Clinical Pharmacist Specialist at the event.
Appendix C: Lipid Screening: Topics for Patient Counseling

Heart disease (1st) and stroke (3rd) are leading causes of death in the USA. The risk of these events can be reduced with proper management of lipids.

1. It is recommended that every adult over 20 years of age have routine lipid profile checks at least once every 4 - 6 years.

2. Terms used in lipid profiles.

3. Variable lipid results may occur if:
   a. Patient has eaten within 9-12 hours of the blood sample collection. Fasting for 9-12 hours before testing will give more accurate results, as recent food consumption will elevate levels of triglycerides and LDL. Therefore, non-fasting patients can only reliably determine their TC and HDL levels and not their LDL or TG.
   b. Patient is pregnant. Cholesterol levels may increase 20-35% due to increases in LDL and VLDL.
   c. There have been recent changes in diet. Variations in diet up to two weeks prior to testing can alter results.
   d. The patient’s position changes from standing to sitting. Cholesterol levels can decrease significantly (~ 6%) when a person goes from a standing to a sitting position. Therefore, it is recommended the patient sits for about 5 minutes prior to blood sample collection.

4. Lipid goals are based on the person’s current health conditions (i.e., presence of heart disease, diabetes, peripheral artery disease, etc.) and risk factors. Currently there is no recommendation for a specific LDL goal, but rather a target statin intensity if an individual is determined to benefit from statin therapy.

Positive risk factors for coronary heart disease (CHD) which influence ASCVD risk:

- Cigarette smoking
- Hypertension (BP ≥140/90 or patient currently taking an antihypertensive medication)
- HDL < 40 mg/dL
- Men ≥ 45 years old and women ≥ 55 years old
• Family history of premature CHD: 1st degree male relative < 55 years old, and 1st degree female relative <65 years old

Using the 2013 AHA/ACC Cholesterol Guidelines, the patient’s 10-year risk is determined which further stratifies their statin benefit group and subsequent treatment options.

5. Two main ways to lower LDL cholesterol and raise HDL cholesterol.
   a. **Therapeutic Lifestyle Changes** (TLC)
      • Dietary Patterns (e.g., TLC diet)
        1. Less than 7% of daily calories are from saturated fat.
        2. Less than 200 mg of dietary cholesterol per day.
        3. Between 25-35% of daily calories should be from fat.
        4. Restrict sodium to < 2400 mg per day.
      • Weight management (see 2013 Obesity Guidelines)
      • Increase physical activity – moderate-to-vigorous physical activity at least 3 to 4 times per week with each session lasting 40 minutes
   b. **Drug Therapy**
      • There are many therapeutic options to help lower LDL and raise HDL cholesterol. These options will need to be discussed with the patient’s health care provider.

6. Dietary suggestions
   a. **Fats**
      • Reduce saturated or hydrogenated fats (solid at room temperature).
        Ex., butter, lard, shortening.
      • Choose mono- or polyunsaturated fats (liquid at room temperature).
        Ex., olive oil, canola oil, safflower.
      • Avoid margarines with trans-fatty acids (TFAs), which can increase LDL cholesterol. There are some margarine products without TFAs.
   b. **Meats, poultry, & fish**
      • Trim fat off meats and poultry.
      • Remove skin from poultry before eating.
      • White meat on chicken and turkey has less cholesterol than dark meat.
      • Goose and duck are high in saturated fats, even with the skin removed.
      • Shellfish contain cholesterol. Ex., 3 oz. of shrimp has ~ 165 mg cholesterol.
c. **Dairy**
   - Use fat-free or low-fat milks and cheeses. Use varieties of cheese with < 3gm of fat/oz.
   - Each egg yolk contains ~ 212 mg of cholesterol. Recommend 2 egg yolks per week.
   - Use egg substitute or egg whites. (2 egg whites equal 1 egg yolk in cholesterol content)

d. **Fruits and vegetables**
   - Diets high in fruits and vegetables may help decrease cholesterol levels.
   - Recommended to have at least 3-5 servings per day.

e. **Breads, grains and beans**
   - Carbohydrates do not contain cholesterol, but should be limited in quantity as they are high in calories.
   - Whole grains and unprocessed foods are preferred.
Appendix D: Therapeutic Lifestyle Changes: Dietary Options

Tips for Dining Out!

ASK! – Will the restaurant:
• Serve margarine rather than butter with the meal?
• Serve fat-free (skim) milk rather than whole milk or cream?
• Trim visible fat from poultry or meat?
• Leave all butter, gravy, or cream sauces off a dish?
• Serve salad dressing on the side?
• Accommodate special requests?
• Use less cooking oil when cooking?

ACT! – Choose foods that are:
• Steamed in their own juice (au jus)
• Broiled
• Baked
• Roasted
• Poached
• Lightly sautéed

Healthy Choices for Stocking the Pantry!

Herbs and Spices (fresh and dried)
• Basil
• Ground black pepper
• Cayenne pepper
• Chili powder
• Cilantro
• Cinnamon
• Coriander
• Crushed red pepper
• Cumin
• Garlic
• Ginger
• Mint
• Nutmeg
• Oregano
• Paprika/smoked paprika
• Parsley
• Rosemary
• Salt-free seasoning mix
• Tarragon
• Thyme

Condiments, Sauces, and Other Seasonings (and, a little goes a long way!)
• Canned tomato paste, no salt added
• Canned tomatoes, no salt added
• Capers
• Dijon mustard
• Fish sauce
• Honey
• Lemon juice
• Lime juice
• Low-sodium broth or stock (chicken, beef, vegetable)
• Lite soy sauce
• Light teriyaki sauce
• Salsa or reduced-sodium taco sauce
• Spaghetti sauce, no salt added
• Vinegar (apple cider, balsamic, red wine, rice)

Oils and Fats: Low in Saturated Fat and Trans Fat
• Cooking spray
• Nut oil (peanut, sesame)
• Soft tub margarine
• Vegetable oil (safflower, canola, corn, olive)
Nuts, Seeds, and Beans: Low in Saturated Fat and High in Protein and Fiber
- Low-sodium canned beans (black, kidney, pinto, chick peas, cannellini)
- Dried lentils
- Unsalted nuts (almonds, pine nuts, walnuts)

Whole Grains: Add Fiber and Other Nutrients to Side Dishes and Main-Dish Meals
- Whole wheat bread
- Brown rice
- Whole-wheat couscous
- Quinoa
- Whole-wheat pasta
- Whole-wheat tortillas
- Popcorn with little or no added salt or butter

Fruits and Vegetables
- Fresh fruits and veggies
- Vegetables rich in color
- Frozen veggies without cream or sauce
- Canned veggies with “reduced sodium,” “low sodium,” or “no salt added”
- Canned fruits in “water” or “100% fruit juice” – avoid “heavy syrup”
- 100% fruit juice with no added sugar

Calcium Rich Dairy
- Fat free or 1% milk
- Cheese with 3 grams of fat or less per serving
- Low-fat or nonfat yogurt
- Plain yogurt as a substitute for sour cream

Go Lean with Protein
- Lean cuts of meat at the deli (90% lean)
- Seafood twice weekly in place of meat/poultry
- Alternate protein sources:
  - Legumes (black-eyed peas, chick peas, kidney beans, lentils, navy beans, soybeans, split peas)
  - Eggs (limit to one egg yolk per day and as many egg whites as you want)
  - Nuts and seeds

Sweets and snacks
- Low fat cookies: animal crackers, devil's food cookies, fig and other fruit bars, ginger snaps, graham crackers, vanilla or lemon wafers
- Angel food cake or other low-fat cakes
- Low fat frozen yogurt, ice milk, fruit ices, sorbet, sherbet
- Pudding (make it with fat free or 1% milk), gelatin desserts
- Pretzels, baked tortilla chips

Beverages
- Water/Sparkling water
- Milk, fat free or low fat (1%)
- 100% fruit juice, no sugar added
- Coffee
- Lemonade, regular or low calorie
- Iced tea, regular or low calorie
- Tea
Appendix E: CardioChek Plus® Quick Reference

Preparing to Test

Testing Supplies

- CardioChek P-A Analyzer (A) with matching Lipid Panel MEMo Chip (B) (shown inserted)
- PTS Panels® Lipid Panel Test Strips (C)
- Gloves*
- Sterile lancets (D)
- Capillary tubes (E)
- Plungers (F)
- Gauze*
- Alcohol wipes (G)
- Bandages*
- Biohazardous waster container*
- 4-AA batteries*

*Not pictured
The CardioChek P-A® is similar to the CardioChek Plus® and uses the same procedure for testing.
APPENDIX F: Cholesterol Profile

Cholesterol testing is recommended every 4-6 years for all adults 20-79 years of age.\textsuperscript{40} The test you are taking today is designed to determine how much of each of the most commonly measured types of cholesterol is in your blood. You will receive numbers for:

1. **TC** (total cholesterol): The sum of all cholesterol types in your blood (LDL, HDL, and VLDL). Your body needs some cholesterol for normal function. The cholesterol in your body comes from two sources: fats in the foods that you eat and from your liver.
2. **LDL** (low density lipoprotein) cholesterol: Known as bad cholesterol. LDL can also be thought of as Lousy cholesterol. LDL cholesterol builds up in artery walls. High LDL cholesterol levels have been shown to increase a person’s risk of heart disease.
3. **HDL** (high density lipoprotein) cholesterol: Known as good cholesterol. HDL can be thought of as the Highly Desirable or Healthy cholesterol. HDL helps carry LDL cholesterol away from the walls of the arteries and returns it to the bloodstream. This prevents the buildup of cholesterol in the artery walls.
4. **TG** (triglycerides): The chemical form of fat being transported through the blood to and from fat cells. Much of the body’s fat is stored in the form of triglycerides for later use as energy. **VLDL** (very low density lipoprotein) cholesterol is a carrier for triglycerides. VLDL carries triglycerides away from the liver to the fat cells. High levels of VLDL have been associated with increased risk of heart disease.
5. **TC/HDL ratio.** The TC/HDL ratio compares the amount of beneficial HDL cholesterol to your total cholesterol level. The lower the ratio, the less risk you have of developing heart disease.

The TC, TG, and HDL components are measured simultaneously from a single drop of blood (obtained by a finger stick) using rapid, accurate technology. The following guidelines are used to interpret your lipid profile results.

|-------------------------|------------------------------------------|
| TC:  
200-239 mg/dL          | Lowering your LDL cholesterol lowers the risk of having a heart attack or stroke, but there is no target LDL goal. |
| > 239 mg/dL             | Patients with **TG \geq 500\text{mg/dL}** or **LDL \geq 190\text{mg/dL}** should follow up with their provider. |
| HDL: < 40 mg/dL         | |
| > 60 mg/dL Males: > 40 | |
| Females: > 50           | |
| TG: < 150 mg/dL (fasting) | |
| TC/HDL ratio: < 4.5 is considered good | |
1. Is the patient taking a statin?

☐ Yes  ☐ No

2. Is the patient taking cholesterol lowering medications (non-statin)?

☐ Yes  ☐ No

3. The ACC/AHA guideline recommends statin therapy for the following groups:

☐ 10-year risk ≥ 7.5% for having a heart attack or stroke in people without cardiovascular disease or DM.
☐ History of a cardiovascular event (heart attack, stroke, stable or unstable angina, peripheral artery disease, transient ischemic attack, or coronary or other arterial revascularization).
☐ Age 21 years and older with LDL cholesterol ≥ 190 mg/dL or higher.
☐ Type 1 or Type 2 diabetes in people ages 40 to 75 years old and LDL 70-189 mg/dL.

Some patients who do not fall into the four categories may also benefit from statins, a decision that should be discussed with your provider.

Things to consider:

☐ Only TC and HDL can be measured accurately in non-fasting individuals. Triglycerides can increase markedly after eating. Since LDL is calculated using TC and TG, the increase in TG will affect the results obtained for LDL. For TG, LDL, and VLDL, only results obtained after a 12 hour fast (other than water and prescribed medications) are valid.
☐ Cholesterol values can vary by about 2-3% within the same day.
☐ Variations in diet up to two weeks prior to testing can alter results.
☐ Certain drugs, besides cholesterol lowering agents, can affect levels.
☐ Cholesterol levels can increase by as much as 20-35% during pregnancy because of increases in LDL and VLDL.
☐ Cholesterol values are higher in the winter and may vary with hard exercise.
☐ Cholesterol levels can decrease significantly when a person goes from a standing to a sitting position (up to a 6% decrease after sitting for 10-15 minutes) It is recommended you sit quietly for about 5 minutes before the blood sample is obtained.
Sample Shopping List

**Breads and Cereals**
- Whole grain bread
- Whole grain pasta
- Brown rice
- Oat bran

**Vegetables/Dry Beans/Peas/Fruits**
- 2-3 servings daily
- All vegetables & fruits without added salt or sugar are acceptable
- Broccoli
- Celery
- Black beans
- Kidney beans
- Strawberries
- Bananas
- Apples

**Dairy Products**
- 1% or skim milk
- Low-fat yogurt
- Low-fat cream cheese
- Low-fat sour cream

**Meat/Poultry/Fish**
- 3 ounces or less daily
- Trimmed lean meats
- Skinless chicken
- Salmon

**Oils/Fats**
- Amount depends on daily calorie level
- Canola oil
- Olive oil
- Fat-free salad dressing
- Liquid margarine or butter

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**CHOLESTEROL & YOUR LIFESTYLE**

University of Montana
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32 Campus Drive
Missoula, MT 59812-1522
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Email: IPHARM@umontana.edu

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**What is cholesterol?**
Cholesterol is a white, waxy, fat-like substance found in foods that come from animal sources such as meat and dairy products. It is also made by your body because it is essential for the normal function of your body.

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**So why does it matter?**

- **Risk factors you cannot change:**
  - Age - Men 45 or older and women 55 or older.
  - Family history - If your dad or brother had a heart attack before age 55 or if your mother or sister had a heart attack before age 65.

- **Risk factors you can change:**
  - Social habits - Smoking and other tobacco use, excessive alcohol use, physical inactivity or being overweight.
  - Other disease states - High blood pressure, diabetes, high LDL and triglycerides, and low HDL.

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**Are you at risk?**

Using Therapeutic Lifestyle Changes (TLC) to reduce your risk:

The TLC program, which has three parts, can be used to improve your cholesterol and decrease your risks of heart disease.

Part 1: Diet
Part 2: Physical Activity
Part 3: Weight Management
**Part 1: Adopt a healthy diet!**

Adopting a healthy diet can decrease your triglycerides and LDL cholesterol and raise your HDL cholesterol. One way to incorporate a healthy diet into your lifestyle is to follow the TLC diet.

The TLC diet is defined as:
1. Limiting cholesterol to less than 200 mg per day,
2. Eliminating trans fat from your diet (less than 1% of daily calorie intake),
3. 25-35% of daily calories from total fat (includes saturated fat calories),
4. Having less than 7% of your daily calories come from saturated fat.

**Practical ways to decrease fat in your diet:****

- Trim visible fat from meats; do not eat skin on poultry.
- Use lean ground beef or sirloin instead of ground chuck or regular ground hamburger; replace meat with fish, tofu, beans, or vegetable patties.
- Use olive oil or canola oil; avoid butter, hard margarine, hydrogenated oils, palm oil or coconut oil.
- Limit red meat to 4 ounce portions (about the size of a deck of cards), 3 times per week.
- Use low-fat or fat-free dairy products, condiments, and salad dressings.
- Eat more fruits and vegetables.

**Smoking cessation:** Smoking lowers HDLs and raises LDLs and triglycerides. Tobacco cessation can improve your whole cholesterol panel and is the best thing you can do for your health.

If you have **high LDLs:**
- Stop smoking.
- Weight loss decreases LDLs by 15%.
- Exercise 30 minutes most days of the week.
- Adopt the TLC diet—low amount of saturated fat, limiting cholesterol and increasing the amount of soluble fiber and plant stanois/sterols in your diet.
- Eat soluble fiber—found in oatmeal, psyllium.
- Eat plant stanois/sterols—found in Benecol® and Take Control® margarine.

If you have **low HDLs:**
- Smoking cessation increases HDLs by 5%.
- Weight loss increases HDLs by 5-20%.
- Aerobic exercise increases HDLs by 30-50%.
- Adopt the TLC diet—low saturated fat, limiting cholesterol, and increasing the amount of soluble fiber and plant stanols/sterols in your diet.
- If you are diabetic, control your blood sugars.

If you have **high triglycerides:**
- Stop smoking.
- Weight loss decreases triglycerides by 30%.
- Exercise 30 minutes most days of the week.
- Decrease the amount of alcohol you drink.
- Decrease the amount of carbohydrates in your diet.
- If you are diabetic, control your blood sugars.

If you have **Omega-3 fatty acids:**

**Part 2: Physical Activity**

- **Aim for moderate-intensity exercise for at least 30 minutes a day, most, if not all, days of the week.**
- Lack of physical activity is a major risk factor for heart disease. Regular physical activity can help you manage your weight.
- **Physical activity can also decrease your risk for other conditions such as high blood pressure and diabetes.**

**How to start getting active:**

- **Beginning activity:** Try increasing standing activities and chores such as painting a room, pushing a wheelchair, doing yard work, ironing, or cooking.
- **Light Activity:** Try something light such as walking slowly (a 24-minute mile), house cleaning, child care, or golf.
- **Moderate-Intensity Activity:** Walking a 15-minute mile, weeding a garden, cycling, playing tennis, or dancing.
- **High-Intensity Activity:** Try walking a 10-minute mile, playing basketball or soccer, kick ball, climbing, or swimming.

**Part 3: Weight Management**

- Maintaining a healthy body mass index (BMI) is important for keeping your risk for heart disease low. A BMI between 18.5-24.9 means you are at a healthy weight.
- There are no quick fixes to lose weight. You need to change your lifestyle—follow the TLC diet, reduce calories, and become physically active.
- An overweight person can achieve great improvements in their health with as little as a 10 pound weight loss.

**A 10% reduction in weight can:**

- Decrease total mortality > 20%.
- Decrease blood pressure (10mmHg systolic/20mmHg diastolic).
- Decrease fasting blood sugars by 30-50%.
- Decrease the risk of developing diabetes by 50%.
**Post-test: Screening for Lipid Disorders in Older Adults**

1) Lipids are involved in many physiologic roles with the **exception** of which of the following?
   a) Participate in the formation of bile salts, which assist in the emulsification of dietary fats and cholesterol for absorption.
   b) **Source of amino acids needed for synthesis of proteins.**
   c) Provide immediate and stored source of energy for the body’s physiologic needs.
   d) Assist in the biosynthetic formation of prostaglandins, steroids and cholesterol.

2) High density lipoproteins (HDL) composition includes all of the following except:
   a) High protein content
   b) **High cholesterol content**
   c) Low triglyceride content
   d) High phospholipid content

3) Which of the following lipoproteins is the primary carrier for plasma triglycerides?
   a) HDL
   b) LDL
   c) IDL
   d) Chylomicron

4) Which of the following statements is **NOT** true regarding the involvement of lipoproteins in lipid transport?
   a) HDL particles remove cholesterol from peripheral tissues and directly transport the cholesterol to the liver for clearance.
   b) Lipoproteins are needed to assist the transport of lipophilic molecules such as triglycerides and cholesterol.
   c) VLDL particles are involved in the transport of triglycerides, obtained from dietary absorption, to the peripheral tissues.
   d) LDL particles primarily carry cholesterol to the peripheral tissues.

5) Which of the following is **NOT** considered to be clinical atherosclerotic cardiovascular disease (ASCVD)?
   a) Unstable angina
   b) **Hypertension**
   c) Transient ischemic attack
   d) Peripheral artery disease

6) Initiating therapeutic lifestyle changes (TLC) may help lower LDL cholesterol. Which of the following is **NOT** a recommendation of the 2013 Lifestyle Guidelines?
   a) Increase physical activity to 40 min of moderate-to-vigorous activity at least 3 times weekly.
   b) Dietary cholesterol should be less than 200 mg per day
   c) Follow a dietary pattern (e.g., Mediterranean diet) may provide better results than changing specific diet components.
   d) **Trans fatty acids should be increased in the diet and saturated fats should be decreased**

7) Obtaining an adequate blood sample from a finger stick may be difficult at times. Which of the following should **NOT** be routinely performed during blood collection?
   a) Look for a fingertip with minimal calluses
   b) **To increase blood flow, firmly massage the pricked finger starting from the base of the finger to the tip**
   c) Warm up cold hands by placing the hands under warm, running water for about 60 seconds
   d) Inspect the fingertips by gently pressing them to see which ones have good blood return.
8) ABC is a 72 year old white female patient who appears to be in good health and is physically fit. She shows up for her lipid screening not having eaten since last night’s dinner. Her only medications are for low thyroid, a daily multivitamin, and occasional acetaminophen for arthritis. She states she has never smoked a cigarette in her life, and does not have any heart problems, nor does it run in her family. ABC is physically active (walks 2 miles/day) and eats “good” foods. Her lipid screening results are (see right):

Utilizing the ASCVD Risk Estimator, what is ABC’s 10-year risk of having an ASCVD event?

- a) 5.8%
- b) 8.6%
- c) 11.2%
- d) 26.8%

9) What counseling would you give to ABC?

- a) She may benefit from low to moderate intensity statin therapy and should discuss drug therapy with her PCP.
- b) She may benefit from moderate to high intensity statin therapy and should follow up with her PCP.
- c) She does not need drug therapy and should continue her current lifestyle.
- d) She should increase her walking to 4 miles/day and discuss non-statin drug therapy with her PCP.

10) Patient DEF, is a 68 year old white male who appears somewhat overweight. DEF is coming in for lipid testing at the insistence of his daughter who is accompanying him. DEF is a life-long rancher who took over his father’s business when his father died of a massive heart attack at age 59. His daughter states her father has not eaten since last night, which has made him grumpy. DEF claims to be as healthy as a horse and does not take any medications other than an occasional aspirin for a headache or backache. DEF’s older sister is alive and she takes medicine for her high blood pressure. DEF admits to smoking about ½ a pack a day of cigarettes, drinks 1-2 beers a day, and loves his meat and potatoes. DEF’s lipid screening provides the following results (see below):

DEF has multiple traditional risk factors for CHD. How many does he have?

- a) 2
- b) 3
- c) 4
- d) 5
11) What is DEF’s 10-year risk assessment of developing ASCVD according to the ASCVD Risk Estimator?
   a) 12%
   b) 16%
   c) 25%
   d) 33%

12) Into which statin-benefit category should DEF be placed?
   a) Presence of Clinical ASCVD
   b) LDL > 190mg/dL
   c) Diabetes Type 1 or 2 and aged 40-75 years
   d) LDL 70-189mg/dL & estimated ASCVD risk > 7.5%

13) Appropriate counseling for DEF would include:
   a) Inform patient he is at great risk for developing ASCVD in the next 10 years and should be seen by a health care provider as soon as possible for follow-up assessment.
   b) Inform the patient that changes to his lifestyle (i.e., not smoking, increased physical activity, and eating foods lower in saturated fats), may reduce his cholesterol level.
   c) Inform the patient that individuals who have high LDL cholesterol have a higher risk of developing ASCVD and his primary goal is to lower his LDL cholesterol.
   d) All of the above should be included in patient counseling.

14) Patient HIJ, a 56 year old white female comes in for her lipid screening after not having eaten since last night about 9pm. HIJ is currently being followed by her physician for high blood pressure for which she takes the combination product lisinopril/hydrochlorothiazide. She takes no other medications on a regular basis. She is a non-smoker. Her parents are both deceased; father died from lung cancer and her mother died secondary to pneumonia. Her older brother is in good health and also takes medication for high blood pressure.

HIJ’s lipid screening gives the following results (see right):

Which of the following is NOT a positive risk factor for ASCVD? (Positive risk factor means having the risk factor increases the risk of developing ASCVD)
   a) Currently on medicine for blood pressure.
   b) Female ≥ 55 years old.
   c) HDL >60 mg/dL
   d) All are positive risk factors

15) Since HIJ has multiple risk factors, what is her calculated 10-year ASCVD risk of developing CHD?
   a) 3.2%
   b) 7.8%
   c) 14.2%
   d) 19.3%

16) Assuming HIJ is at low risk of developing ASCVD over the next 10 years, which of the following statements is the most appropriate action to be taken based on her LDL assessment?
   a) Nothing needs to be done; this patient is already at her LDL goal.
   b) This patient should be seen by her primary care provider as soon as possible to initiate drug therapy.
   c) This patient may benefit from initiating therapeutic lifestyle changes and should discuss the results of this screening with her health care provider at the next scheduled visit.
   d) This patient is doing just fine and should continue her current lifestyle despite an LDL above goal.

HIJ’s Results:
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<th>Parameter</th>
<th>Value</th>
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<tr>
<td>Blood pressure</td>
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<td>Triglycerides</td>
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<td>Total chol.</td>
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<tr>
<td>HDL</td>
<td>62 mg/dL</td>
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<td>LDL</td>
<td>176 mg/dL</td>
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<td>VLDL</td>
<td>10 mg/dL</td>
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<td>TC/HDL</td>
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</table>
17) Patient KLM, a 58 year old, obese white male is being seen for lipid screening. KLM was recently diagnosed with type II diabetes and is currently taking metformin for glucose control. He takes no other prescription medications, but does take loratadine for seasonal allergies and occasionally acetaminophen for pain in his “bad” right knee. He does not smoke and does not get regular exercise due to his “bad” knee.

KLM is automatically considered to be at high risk for ASCVD because of:
   a) His age
   b) His obesity
   c) His diabetes
   d) His sedentary lifestyle

18) KLM states he ate breakfast this morning (which was about 4 hours ago), and he wants to know if he can still get his lipids tested. Which of the following actions is the most appropriate for this patient?
   a) Turn the patient away; all lipid testing must be performed in fasting patients (9-12 hours).
   b) Explain to the patient, fasting is preferred to get good results for HDL and LDL determination.
   c) Explain to the patient, fasting is necessary to get good results for LDL and triglyceride determination, and therefore, only this patient’s HDL and total cholesterol may be calculated.
   d) Tell the patient that not having fasted is OK, and that the lipid analysis will be fine.

19) You perform KLM’s lipid screening with the following results (see below). How would you counsel this patient?
   I KLM should be seen by his primary care provider in the near future to discuss lipid lowering therapy.
   II KLM would benefit from initiating therapeutic lifestyle changes and may want to speak to a dietician since he has diabetes and potentially lipid abnormalities.
   III KLM should not worry about his lipids; they are not too bad.

   a) I only
   b) II only
   c) I & II
   d) I, II, III

20) KLM is a candidate for high intensity statin therapy for the following reason:
   a) Presence of diabetes with ASCVD risk estimate > 7.5%
   b) Presence of diabetes with ASCVD risk estimate < 7.5%
   c) Presence of clinical ASCVD
   d) LDL > 100mg/dL

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POST-TEST: Examination Form
Screening for Lipids in Older Adults

Participant Information

1. Name: ______________________________________

2. Mailing address: ______________________________

   ______________________________

   ______________________________

   ______________________________

3. Date exam completed _________________________

Questions: (Please circle one response per question)

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Appendix I: Evaluation: Screening for Lipid Disorders in Older Adults

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<th>Please indicate your major</th>
<th>Strongly Agree</th>
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<th>Disagree</th>
<th>Strongly Disagree</th>
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<td>1. Based on the module description and stated objectives, this module met my expectations of the content it would deliver.</td>
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<td>2. How effective were the following in helping you understand the material?</td>
<td>Very Effective</td>
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<td>3. I learned something I can use in my practice/employment or personal setting.</td>
<td>Strongly Agree</td>
<td>Agree</td>
<td>Neutral</td>
<td>Disagree</td>
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4. How do you plan to implement the information from this module to strengthen your practice, employment or personal goals? (check any that apply)

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<tr>
<th>Provide new information to patients/clients</th>
<th>Adjust practices with geriatric patients/clients</th>
<th>New program development or program enhancement</th>
<th>Provide new information to family/friends/co-workers</th>
<th>Train staff or provider</th>
<th>Other implementation*</th>
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* Describe 'other' implementation plan here:

5. How long did it take you to complete the module? (including pre-test, module review, post-test and evaluation)

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6. The test questions were relevant to the module content.

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7. Please provide suggestions to improve the online learning experience to meet your needs.

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8. Please offer ideas or suggestions for new modules.

For credit, please return this completed page to:

MTGEC/IPHARM
Skaggs Building Room 318
University of Montana
32 Campus Drive
Missoula MT, 59812-1522
Phone# (406) 243-2339 & Fax# (406) 243-4353