



Montana Geriatric Education Center

Instructions on Completing the Module

Screening for Lipid Disorders in Older Persons

**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 in the syllabus.
3. **After** reading the module, please complete the Post-Test.
Use the questions in **Appendix I** and record your answers on the examination form marked Post-Test. (*Found at the end of Appendix E.*) Keep the completed answer form to turn in 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 turn in with the pre-test and post-test at the completion of the module.

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

Rachael Zins
MTGEC/IPHARM
Skaggs Building Room 317
University of Montana
32 Campus Drive
Missoula MT, 59812-1522

Email: IPHARM@umontana.edu

Phone# (406) 243-2339 & Fax# (406) 243-4353

MONTANA GERIATRIC EDUCATION CENTER Required Disclosures to Participants

Goal/Purpose

Improve health outcomes for older adults in rural Montana via increased knowledge of geriatric care and treatment of health problems by health professionals.

Successful Completion of this Continuing Education Activity:

- Completion of MTGEC Participant Profile
- Completion of pre-test
- Reading of text
- Completion of post-test with at least 70% accuracy
- Completion of module evaluation

Contact Hours: 2

MT Nurses Association Continuing Education Expiration Date: 6/15/2013

Conflicts of Interest

A conflict of interest occurs when an individual has an opportunity to affect educational content about health-care products or services of a commercial company with which she/he has a financial relationship.

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

Commercial Company Support

There is no Commercial Company Support for this CE activity

Noncommercial Sponsor Support

This CE activity is supported 100% by a federally funded grant from the Health Resources and Services Administration (HRSA) Grant Number UB4HP19056 for \$2,136,009 (07/01/2010 – 06/30/2015).

Non-Endorsement of Products

Approved provider status does not imply that there is real or implied endorsement by MTGEC, ANCC, or MNA of any product, service, or company referred to in this activity nor of any company subsidizing costs related to the activity.

Off-label Product Use

This CE activity does not include any unannounced information about off-label use of a product for a purpose other than that for which it was approved by the Food and Drug Administration (FDA).

Pre-test: *Screening for Lipid Disorders in Older Persons*

Record responses on examination form.

- 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 phospholipids 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 a coronary heart disease (CHD) or a CHD-risk **equivalent**?
 - a) Unstable angina
 - b) Hypertension
 - c) Diabetes (type 1 or 2)
 - 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 TLC program?
 - a) Increase physical activity
 - b) Dietary cholesterol should be less than 200 mg per day
 - c) At least 20-30 g of fiber is recommended in the diet
 - 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?
- Look for a finger tip with minimal calluses
 - To increase blood flow, firmly massage the pricked finger starting from the base of the finger to the tip
 - Warm up cold hands by placing the hands under warm, running water for about 60 seconds
 - Inspect the fingertips by gently pressing them to see which ones have good blood return.
- 8) ABC is a 72 year old 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):

What would this patient's risk assessment be classified as?

- Low risk
- Moderate risk
- Moderately-high risk
- High risk

ABC's Results:	
Blood pressure	126/82 mmHg
Triglycerides	104 mg/dL
Total chol.	167 mg/dL
HDL	45 mg/dL
LDL	112 mg/dL
VLDL	10 mg/dL
TC/HDL	3.7

- 9) What counseling would you give to ABC?
- She is below her LDL goal and should consider eating more dietary fats.
 - She is below her LDL goal and should continue her current lifestyle.
 - She is above her LDL goal and should consider adding drug therapy.
 - She is above her LDL goal and should increase her walking to 4 miles/day.
- 10) Patient DEF, is a 68 year old 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 risk factors for CHD. How many does he have?

- 2
- 3
- 4
- 5

DEF's Results:	
Blood pressure	152/100 mmHg
Triglycerides	226 mg/dL
Total chol.	241 mg/dL
HDL	36 mg/dL
LDL	185 mg/dL
VLDL	20 mg/dL
TC/HDL	6.7

- 11) Since DEF has multiple risk factors, what is his 10-year risk assessment of developing CHD according to the Framingham risk assessment?
- 12%
 - 16%
 - 25%
 - ≥ 30%
- 12) Into which risk category should DEF be placed into to stratify his LDL goals?
- High risk
 - Moderately-high risk
 - Moderate risk
 - Low risk
- 13) Appropriate counseling for DEF would include:
- Inform patient he is at great risk for developing CHD in the next 10 years and should be seen by a health care provider as soon as possible for follow-up assessment.
 - 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.
 - Inform the patient that individuals who have high LDL cholesterol have a higher risk of developing CHD and his primary goal is to lower his LDL cholesterol.
 - All of the above should be included in patient counseling.

14) Patient HIJ, a 63 year old 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 currently smokes about 10 cigarettes a day and has failed multiple attempts to quit in the past. 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 CHD?
(Positive risk factor means having the risk factor increases the risk of developing CHD)

- Currently on medicine for blood pressure.
- Female ≥ 55 years old.
- HDL >60 mg/dL
- All are positive risk factors

HIJ's Results:

Blood pressure	126/82 mmHg
Triglycerides	158 mg/dL
Total chol.	248 mg/dL
HDL	62 mg/dL
LDL	176 mg/dL
VLDL	10 mg/dL
TC/HDL	4

- 15) Since HIJ has multiple risk factors, what is her calculated 10-year Framingham risk of developing CHD?
- 3%
 - 5%
 - 14%
 - 17%

- 16) Assuming HIJ is at moderate risk of developing CHD over the next 10 years, which of the following statements is the most appropriate action to be taken based on her LDL assessment?
- Nothing needs to be done; this patient is already at her LDL goal.
 - This patient should be seen by her primary care provider as soon as possible to initiate drug therapy.
 - 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.
 - This patient is doing just fine and should continue her current lifestyle.
- 17) Patient KLM, a 58 year old, obese 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’s risk classification would best be categorized as:
- High risk
 - Moderately-high risk
 - Moderate risk
 - Unable to determine (not enough information provided)
- 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?
- Turn the patient away; all lipid testing must be performed in fasting patients (9-12 hours).
 - Explain to the patient, fasting is preferred to get good results for HDL and LDL determination.
 - Explain to the patient, fasting is preferred to get good results for LDL and triglyceride determination, and therefore, only this patient’s HDL and total cholesterol may be calculated.
 - 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?
- KLM should be seen by his primary care provider as soon as possible to discuss lipid lowering.
 - KLM would benefit from initiating therapeutic lifestyle changes and may want to speak to a dietician since he has diabetes and potentially lipid abnormalities.
 - KLM should not worry about his lipids; they are not too bad.
- I only
 - II only
 - I & II
 - I, II, III
- 20) KLM is a strong candidate for metabolic syndrome.
- True
 - False

KLM’s Results:	
Blood pressure	150/100
Triglycerides	265 mg/dL
Total chol.	245 mg/dL
HDL	50 mg/dL
LDL	170 mg/dL
VLDL	25 mg/dL
TC/HDL	4.9

PRE-TEST: Examination Form

Screening for Lipids in Older Persons

Participant Information

1. Name: _____
2. Mailing address: _____

3. Date exam completed _____

Questions: (Please circle one response per question)

1	A	B	C	D
2	A	B	C	D
3	A	B	C	D
4	A	B	C	D
5	A	B	C	D
6	A	B	C	D
7	A	B	C	D
8	A	B	C	D
9	A	B	C	D
10	A	B	C	D
11	A	B	C	D
12	A	B	C	D
13	A	B	C	D
14	A	B	C	D
15	A	B	C	D
16	A	B	C	D
17	A	B	C	D
18	A	B	C	D
19	A	B	C	D
20	A	B	C	D

For credit, please return this completed page to:

Rachael Zins
MTGEC/IPHARM
 Skaggs Building Room 317
 University of Montana
 32 Campus Drive
 Missoula MT, 59812-1522
 Phone# (406) 243-2339 & Fax# (406) 243-4353



Montana Geriatric Education Center

Screening for Lipid Disorders in Older Persons

Developed by Kim Madsen, Pharm.D.

Revised by Larry Dent, Pharm. D., BCPS

Skaggs School of Pharmacy
University of Montana Missoula, MT

A 2-hour module from the

Montana Geriatric Education Center

A Consortium of:
The University of Montana, Missoula
St. Vincent Healthcare
Montana Tech

<http://mtgec.montana.edu>

May 2011 revised
Copyright 2008
Montana Geriatric Education Center

Description of Module

Content:

This 2-hour module will discuss the basic issues which surround screening for lipid abnormalities in the geriatric population.

Learning objectives:

- A. Describe the impact of lipid disorders on cardiovascular disease.
- B. Examine the 3rd edition of the Adult Treatment Plan (ATP III) guidelines and incorporate revised recommendations (August 2004).
- C. Discuss the role of risk factors in cardiovascular disease, and formulate a treatment plan based on patient specific information.
- D. Describe techniques needed to perform point-of-care lipid testing using the Cholestech LDX[®].

Table of Contents

I.	OVERVIEW	11
II.	IMPACT OF DYSLIPIDEMIAS ON HEALTH	11
A.	ROLE OF LIPIDS IN CARDIOVASCULAR DISEASE.....	11
1.	<i>Cholesterol Increases Risk of Cardiovascular Deaths</i>	11
2.	<i>Decreasing Lipid Levels Lower Risk of Cardiovascular Disease</i>	13
3.	<i>The Role of Lipids in Atherosclerosis</i>	14
B.	ROLE OF LIPIDS AND DIFFERENT LIPOPROTEINS.....	14
1.	<i>Lipoprotein Transport</i>	15
2.	<i>Lipoprotein Metabolism & Sites to Lower Lipids</i>	18
3.	<i>Different Types of Lipid Abnormalities</i>	20
C.	ATHEROGENESIS.....	22
III.	GUIDELINES FOR MANAGEMENT OF DYSLIPIDEMIAS	25
A.	ATP III GUIDELINES (UPDATED 2004).....	25
	STEP 1: DETERMINE LIPID LEVELS.....	25
	STEP 2: IDENTIFY PATIENTS WITH CORONARY HEART DISEASE OR EQUIVALENT.....	26
	STEP 3: DETERMINE PRESENCE FOR MAJOR RISK FACTORS ⁽⁴⁾	27
	STEP 4: DETERMINE 10-YEAR CORONARY HEART DISEASE RISK.....	28
	STEP 5: DETERMINE RISK CATEGORY AND LIPID THERAPEUTIC GOALS.....	29
	STEP 6: FEATURES OF THE THERAPEUTIC LIFESTYLE CHANGES (TLC).....	30
	STEP 7: DRUG THERAPY FOR ACHIEVEMENT OF THERAPEUTIC GOALS.....	34
	STEP 8: ASSESSMENT OF METABOLIC SYNDROME.....	35
	STEP 9: TREATMENT OF ELEVATED TRIGLYCERIDES & LOW HDL CHOLESTEROL.....	36
IV.	SCREENING FOR DYSLIPIDEMIAS	37
A.	WHO SHOULD BE TESTED?.....	37
B.	POINT-OF-CARE LIPID TESTING.....	38
1.	<i>Cholestech LDX[®]</i>	38
2.	<i>Components of the Cholestech LDX[®](47)</i>	39
3.	<i>Components of the Cholestech Test Cassette⁽⁴⁷⁾</i>	40
4.	<i>Setting Up the Cholestech LDX[®](47)</i>	40
5.	<i>Quality Assurance of the Cholestech LDX[®](47)</i>	41
6.	<i>Use of the Cholestech LDX[®](47)</i>	42
7.	<i>Performing a Finger Stick for Blood Collection</i>	42
8.	<i>Interpretation of Results</i>	44
V.	USEFUL WEBSITES	46
VI.	REFERENCES	47
APPENDIX A	IPHARM AUTHORIZATION TO TEST FORM	45
APPENDIX B	PROTECTION OF STAFF & PUBLIC FROM BLOOD-BORNE PATHOGENS	52
APPENDIX C	LIPID SCREENING: TOPICS FOR PATIENT COUNSELING	53
APPENDIX D	ATP III GUIDELINES. AT-A-GLANCE⁽³¹⁾	56
APPENDIX E	THERAPEUTIC LIFESTYLE CHANGES: DIETARY OPTIONS	62
APPENDIX F:	CHOLESTECH LDX[®] QUICK REFERENCE⁽⁴⁷⁾	64
APPENDIX G:	LIPID SCREENING: PATIENT ASSESSMENT FORM	69
APPENDIX H:	CHOLESTECH LDX LIPID PROFILE	71
APPENDIX I:	POST-TEST QUESTIONS	73
APPENDIX J:	EVALUATION FOR MTGEC MODULE	78

Screening for Lipid Disorders in Older Persons

I. Overview

Heart disease is the number one leading cause of death in the United States, accounting for 25% of all deaths in 2009.⁽¹⁾ Cardiovascular disease, which includes heart disease and stroke (4th leading cause of death), accounted for 30% of deaths in the US in 2009.⁽¹⁾ The good news is that heart disease is declining in the US, as it accounted for 38% of deaths in 1980.⁽¹⁾ The risk of developing heart disease increases with age. In the US, heart disease accounted for 44% of deaths in 2007 for those 65 years of age or older.⁽³⁾ Coupled with the increasingly older US population, screening for CVD is imperative to decrease the mortality and morbidity associated with CVD.

Abnormal lipid levels are associated with an increased risk of CVD; therefore, lipid levels can be used as a screening tool to identify patients who would benefit from intervening therapies such as lifestyle modifications and drug therapy.

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:^(4,5)

- (1) Coronary heart disease (CHD): myocardial infarction or 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. 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.⁽⁴⁾

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 4 & 5 demonstrate the linear relationship associated with increased total serum cholesterol with cardiovascular-related deaths and all-cause mortality.⁽⁶⁾

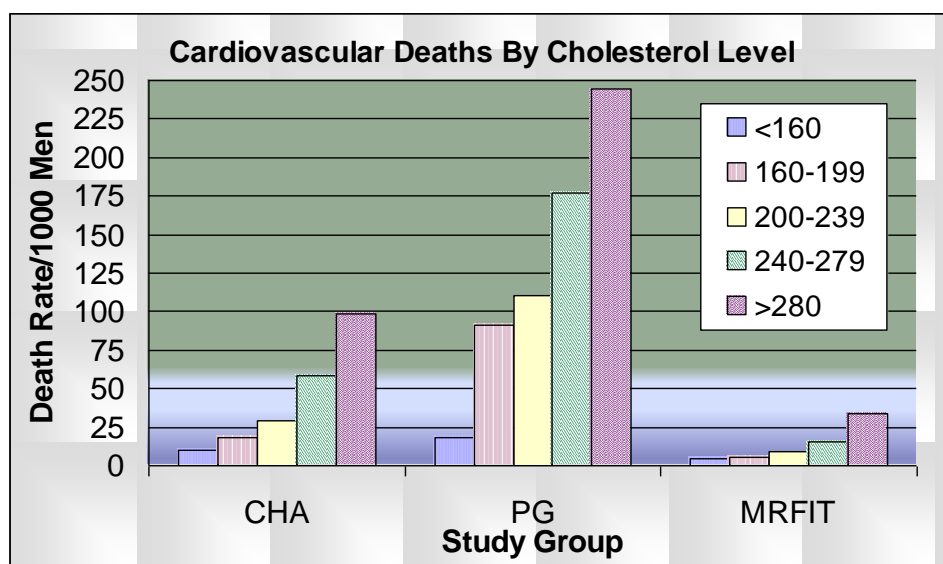
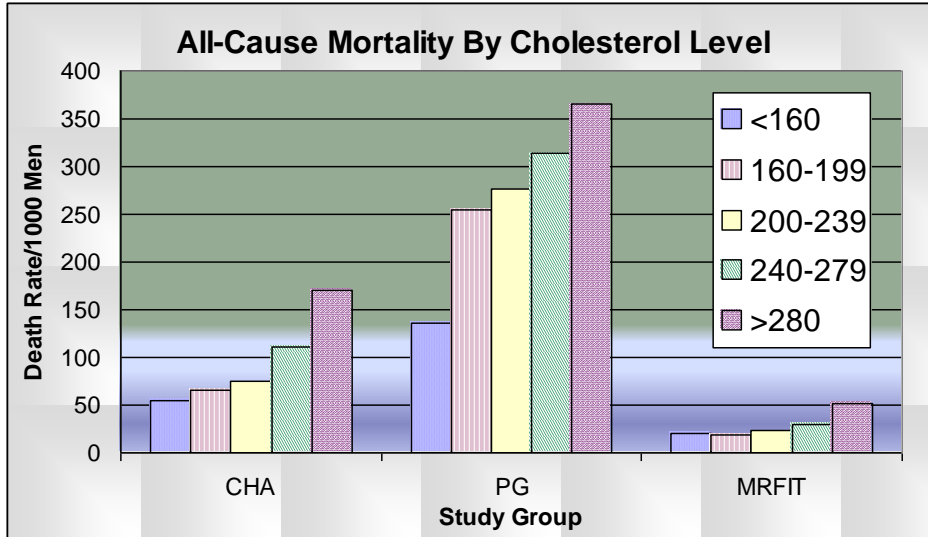


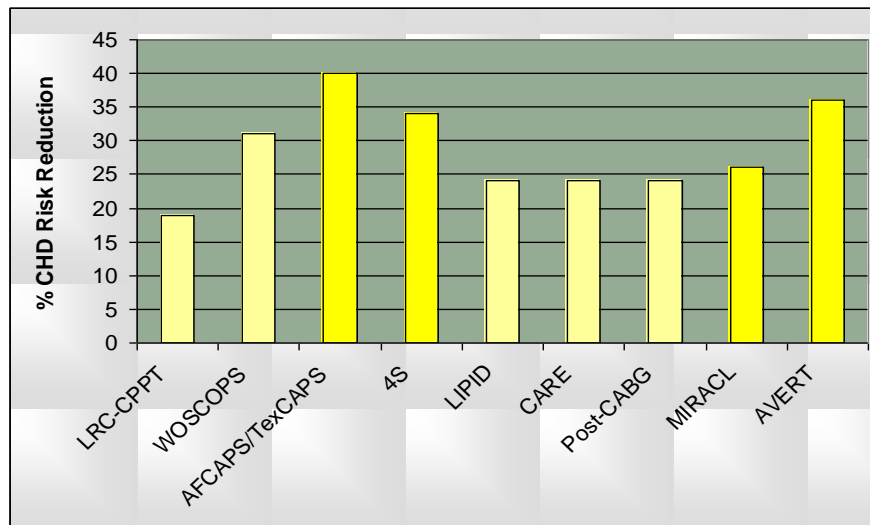
Figure 4: Increasing Cardiovascular Death Rate with Increasing Cholesterol Level⁽⁶⁾



**Figure 5: Death Rate for any Cause
Stratified by Baseline Cholesterol Level⁽⁶⁾**

2. Decreasing Lipid Levels Lower Risk of Cardiovascular Disease

Several 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 6 demonstrates the range of coronary heart disease risk reduction among multiple clinical trials in different populations.⁽⁷⁻¹⁵⁾



**Figure 6: Cardiovascular Risk Reduction
Associated with Lipid Lowering Trials⁽⁷⁻¹⁵⁾**

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.⁽¹⁶⁾

3. The Role of Lipids in Atherosclerosis

More knowledge is known about the role lipids play in the pathogenesis of atherosclerosis, and this topic is discussed in greater detail in Section II, Part C.

B. Role of Lipids and Different Lipoproteins

Lipids are involved in many physiological roles including:⁽¹⁷⁾

- 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, is 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 7 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.^(17,18,19)

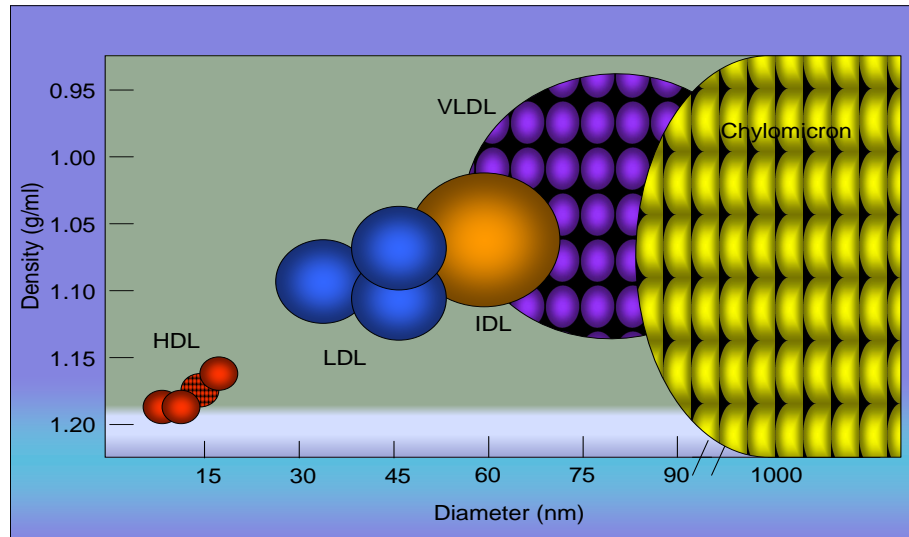


Figure 7: Comparison of Densities Between Lipoproteins^(18,19)

Lipoprotein	Composition (%)					Primary Apolipoproteins
	Protein	Cholesterol	Cholesteryl Ester	Triglyceride	Phospholipid	
Chylomicron	2	2	3	90	3	ApoC-II ApoE
VLDL	8	4	16	55	17	ApoC-II ApoB-100 ApoE
IDL	10	5	25	40	20	ApoB-100 ApoE
LDL	20	7	46	6	21	ApoB-100
HDL	50	4	16	5	25	ApoA-I

Table 1: Difference in Composition between Lipoproteins^(17,20)

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.⁽¹⁷⁾ Figure 8 provides a schematic overview of how the different lipids are transported.

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 ApoE and the low density lipoprotein receptor (LDLR). Usually within 12-14 hours after eating, all chylomicrons are absent from circulation.^(17,18)

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.^(17,18)

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.^(17,18)

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 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.^(17,18)

High Density Lipoproteins (HDL)

HDL is involved in transporting cholesterol back from the tissues to the liver for excretion in the bile (Figure 9). HDL is produced in the liver and intestines and has 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 means 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.^(17,18,19)

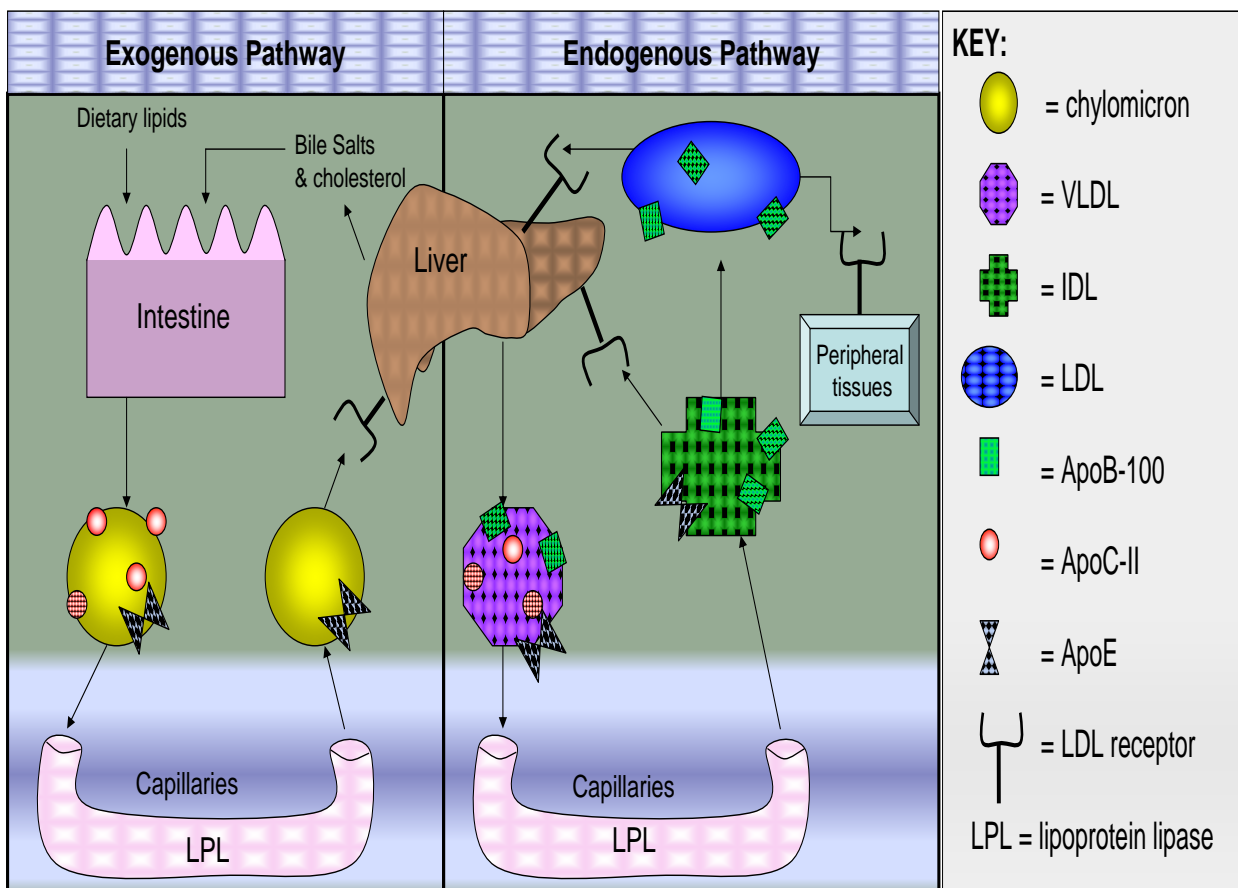


Figure 8: Schematic of Lipid Transport to Peripheral Tissues^(17,18,19)

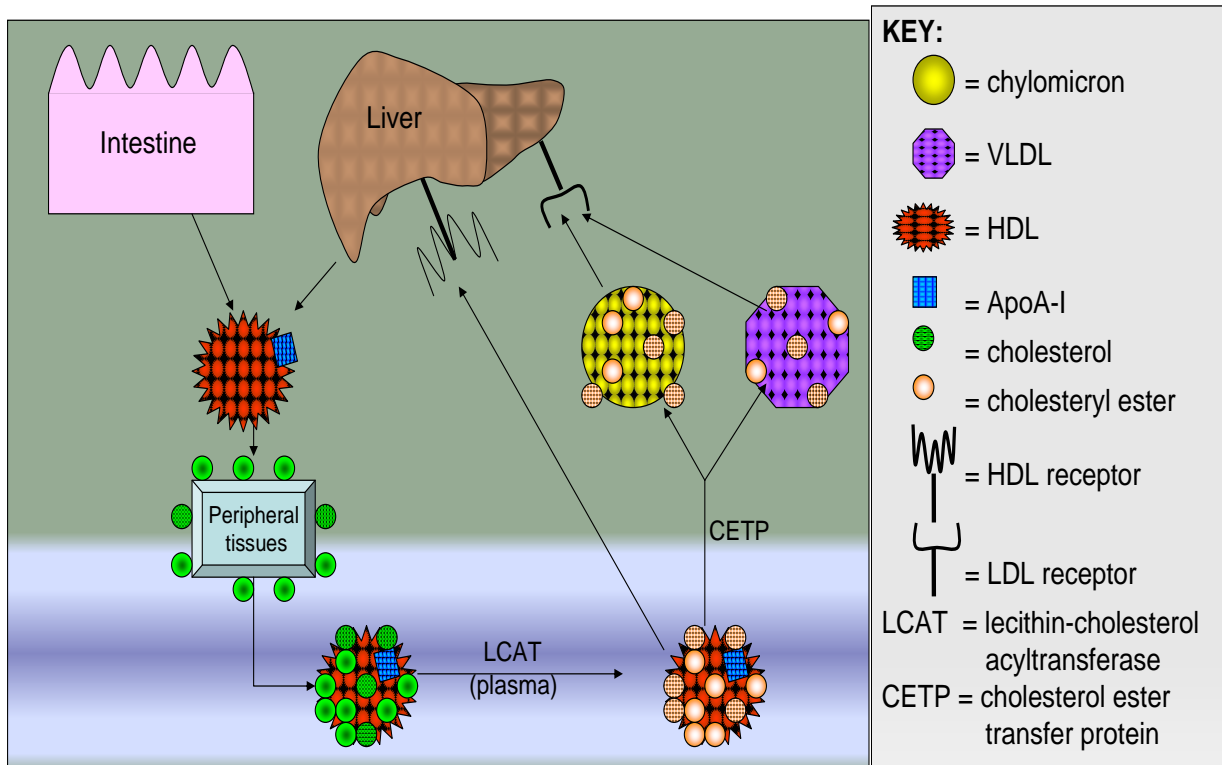


Figure 9: Schematic of HDL-Lipid Transport^(17,18,19)

2. Lipoprotein Metabolism & Sites to Lower Lipids

As mentioned earlier, the two main lipid sources are from the diet or by endogenous biosynthesis. On a daily basis, the liver synthesizes approximately 400 mg of bile salts, 11 grams of phospholipids, and 2 grams of cholesterol.⁽²¹⁾

One of the main functions of the liver is to produce bile salts to 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.⁽²¹⁾

In regards to cholesterol, the liver synthesizes about 2 grams per day and the average daily diet contains 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.⁽²¹⁾ Essentially, our bodies produce excess cholesterol daily so our dietary intake is above and beyond what is physiologically required.

Fat in the diet consists primarily of triglycerides (90%), and the remainder as cholesterol, phospholipids and fat-soluble vitamins A, D, E, and K.⁽²²⁾ 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.^(17,18) Ezetimibe (Zetia[™]), inhibits the transport of cholesterol across the intestinal cell wall which decreases dietary absorption of cholesterol.⁽²³⁾

The body synthesizes cholesterol, primarily in the liver, through a series of biosynthetic steps starting with acetyl-CoA (Figure 10). One of the biosynthetic steps includes the production of the cholesterol precursor, mevalonic acid, through a modification step using the enzyme HMG-CoA reductase.⁽¹⁷⁾ 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.⁽⁴⁾

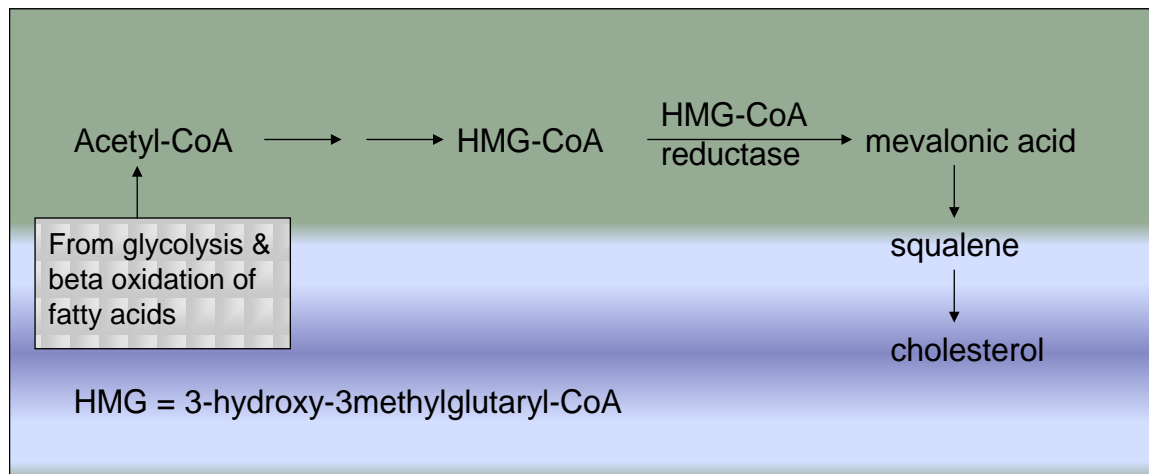


Figure 10: Cholesterol Biosynthesis⁽¹⁷⁾

3. Different Types of Lipid Abnormalities

Abnormal levels of lipids or dyslipidemias are classified as primary or secondary disorders. Primary disorders are caused by a dysfunction in lipid metabolism, whereas secondary disorders are related to an underlying disease or medication.⁽⁴⁾

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 or behaviors can alter lipoprotein levels, and Table 3 lists some of the more common secondary causes.^(4,24,25)

When assessing a patient for screening purposes, determination of primary and secondary dyslipidemias is not possible or practical, but rather the lipid levels themselves in conjunction with risk factor assessment will determine therapeutic recommendations.

Lipoprotein	Disorder	Effect
LDL	Familial hypercholesterolemia	Defective LDL-receptor on liver decreases LDL clearance from circulation
	Familial defective ApoB-100	Defective ApoB-100 on LDL decreases LDL-receptor binding & decreases LDL clearance.
Triglycerides	Lipoprotein lipase (LPL) deficiency	Low LPL levels decreases amount of triglycerides cleared from circulation
	ApoC-II deficiency	Decreased ApoC-II leads to decreased activity of LPL leading to decreased triglyceride release into peripheral tissues
	Familial hypertriglyceridemia	Over production of VLDL cholesterol with normal VLDL clearance leads to accumulation of VLDL
HDL	ApoA-I dysfunction	Mutations in ApoA-I can lead to decreased HDL production or increase HDL clearance from the circulation.
	Familial HDL deficiency	Low production of HDL
	LCAT deficiency	Low levels of LCAT prevents the conversion of cholesterol to the ester form, preventing cholesterol clearance
	CETP deficiency	Low levels of CETP prevents the transfer of the cholesterol esters from HDL to VLDL and chylomicrons

Table 2: Primary Causes of Dyslipidemias^(4,19,25)

Category	Cause
<i>Endocrine/Metabolic</i>	<ul style="list-style-type: none"> • Diabetes mellitus • Lipodystrophy • Cushing's syndrome • Hypothyroidism
<i>Renal</i>	<ul style="list-style-type: none"> • Chronic renal failure • Glomerulonephritis • Nephrotic syndrome
<i>Hepatic</i>	<ul style="list-style-type: none"> • Cirrhosis • Obstructive liver diseases
<i>Lifestyle</i>	<ul style="list-style-type: none"> • Physical inactivity • Obesity • Smoking • High fat diet (especially saturated fats) • Alcohol intake
<i>Medications</i>	<ul style="list-style-type: none"> • Progestins, estrogens • Thiazide diuretics • Beta-blockers • Glucocorticoids • Isotretinoin • Protease Inhibitors • Atypical antipsychotics • Cyclosporine • Mirtazapine • Anabolic steroids

Table 3: Secondary Causes of Dyslipidemias^(4,19,25)

C. Atherogenesis

The development of atherosclerosis is the foundation for cardiovascular disease; therefore, it is necessary to understand the pathogenesis of atherosclerosis and how lipid abnormalities play a significant role.

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).^(26,27,28) Figures 11 & 12 depict the normal and abnormal arterial cell wall composition, respectively.

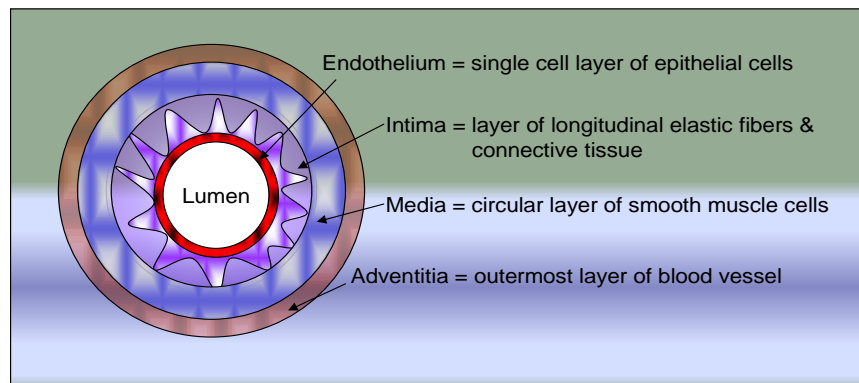


Figure 11: Normal Cross-Section of an Arterial Vessel

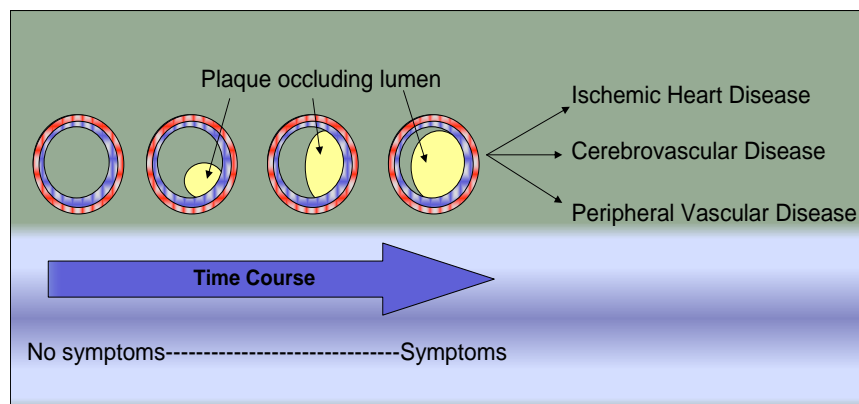


Figure 12: Disease Progression in an Atherosclerotic Vessel

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, leading to a chronic inflammatory response. Figure 13 portrays a hypothetical schematic of atherosclerosis.⁽²⁰⁾

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 is not clear but thought to be related to components found in cigarette smoke, elevated homocysteine levels, or infectious viruses or bacteria. 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.⁽²⁰⁾

Circulating white blood cells do not normally bind to the endothelial 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.⁽²⁰⁾ Also occurring during this stage of atherosclerosis, smooth muscle cells from the media layer of the arterial cell wall are attracted to and migrate into the intima layer where they proliferate.⁽²⁰⁾

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.⁽²⁰⁾

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.⁽²⁰⁾

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.⁽²⁰⁾

Atherosclerosis starts early in life with the development of fatty streaks as was noticed in autopsy studies of young men and women (ages 15-34) who died for non-cardiac reasons.⁽²⁹⁾ The asymptomatic progression may take years to develop before an adverse cardiovascular event occurs.

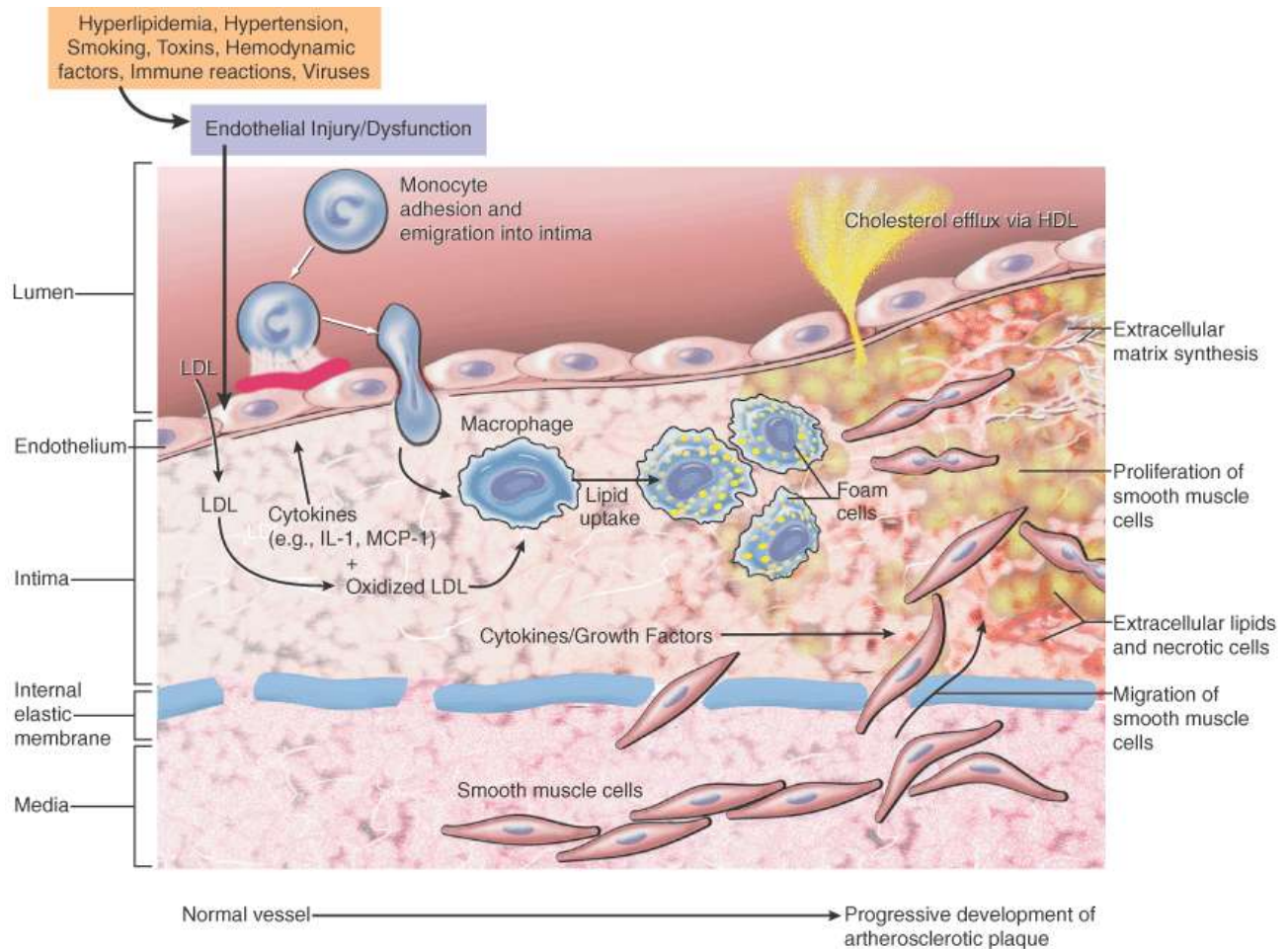


Figure 13: Hypothetical Mechanism of Atherosclerosis⁽²⁰⁾

III. Guidelines for Management of Dyslipidemias

In 1985, The National Heart, Lung and Blood Institute developed the National Cholesterol Education Program (NCEP) to reduce the number of Americans with high blood cholesterol and to decrease morbidity and mortality due to its association with coronary heart disease.⁽³⁰⁾ NCEP has published a sequential series of guidelines from the Adult Treatment Panel (ATP) with the last full guideline, ATP III, released in 2001. In August 2004, the ATP III guideline was revised to reflect more recent clinical trial data which provided better definition of lipid goals based on a patient's cardiovascular risk. The revised ATP-III goals added another risk stratification for a total of four groups: high risk, moderately-high risk, moderate risk, and low risk.⁽³¹⁾ Expected release date of ATP-IV is fall 2011.

A. ATP III Guidelines (Updated 2004)

Appendix D contains the ATP III Guidelines: *At-A-Glance*⁽³²⁾ which is a quick reference for determination of risk assessment and treatment recommendations, and can also be found at <http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm> The guide reflects the ATP III Guideline prior to the August 2004 update, as a new updated quick reference guide has not been published. This guide is still a valuable resource to determine a patient's risk, but Step 5 (Determine Risk Category) will need to be replaced with the new goals. For ease of discussion, the following sections will correlate with the 9 Steps in the ATP III Guidelines: *At-A-Glance* reference.

Step 1: Determine Lipid Levels

Utilizing point-of-care testing has allowed lipid testing to be performed outside of the physician's office or the hospital setting. (Detailed steps to perform lipid testing are discussed in Section IV.) Fasting conditions (9-12 hours) 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.⁽⁴⁾

As was previously discussed, increasing levels of LDL cholesterol have increasing detrimental effects on coronary heart disease; therefore, ATP has stratified cholesterol levels (LDL, HDL and total cholesterol) to assist with lipid management (Table 4).⁽⁴⁾

LDL Cholesterol	
<100	Optimal
100-129	Near optimal/ above optimal
130-159	Borderline high
160-189	High
≥ 190	Very high
Total Cholesterol	
<200	Desirable
200-239	Borderline high
≥ 240	High
HDL Cholesterol	
< 40	Low
≥ 60	High

Table 4: ATP III Classification of LDL, Total and HDL Cholesterol (mg/dL)⁽⁴⁾

Step 2: Identify Patients with Coronary Heart Disease or Equivalent

A fundamental concept with the ATP III Guidelines is that the intensity of lipid lowering is in relation to a patient's risk for CHD. As such, it is necessary to determine a patient's risk for CHD in the immediate future as well as their 10-year risk.⁽⁴⁾

The purpose of Step 2 is to aid in risk stratification by first identifying those patients at highest risk for CHD, such as patients with known CHD or CHD-risk equivalents. Patients with known CHD are at very high risk of having another CHD event,⁽⁴⁾ and multiple trials have demonstrated that lipid lowering (primarily LDL cholesterol) decreases the occurrence of major coronary events (ex., myocardial infarction), stroke and cardiovascular-related deaths.^(10,11,12) Some patients without diagnosed CHD may have comorbid diseases or multiple risk factors which classifies them as having a CHD-risk equivalent. These patients have a >20% chance of developing CHD over the next ten years, and therefore are categorized as high risk patients. Table 5 demonstrates the categories for known CHD and CHD risk equivalent. As of ATP III, diabetes is considered a CHD risk equivalent as patients with type 1 or type 2 diabetes are at increased risk for CHD.⁽⁴⁾

Coronary Heart Disease (any of following)
<ol style="list-style-type: none"> 1. History of myocardial infarction 2. Unstable angina 3. Stable angina 4. History of coronary artery procedures <ul style="list-style-type: none"> - angioplasty, stent placement, or bypass surgery
Coronary Heart Disease Risk Equivalent (any of the following)
<ol style="list-style-type: none"> 1. <u>Non-coronary atherosclerotic disease:</u> <ul style="list-style-type: none"> ▪ Peripheral artery disease ▪ Abdominal aortic aneurysm ▪ Symptomatic carotid artery disease <ul style="list-style-type: none"> - carotid origin of ischemic stroke or transient ischemic attacks 2. Diabetes (Type 1 or 2) 3. 2+ Risk factors with a 10-year risk >20% (See Steps 3 & 4 for determination)

Table 5: Categorization of Coronary Heart Disease and Risk Equivalents⁽⁴⁾

Step 3: Determine Presence of Major Risk Factors⁽⁴⁾

If a patient is not known to have CHD or CHD risk equivalents, then the sum of other known risk factors for CHD is utilized to determine a patient's future risk of CHD. Although elevated LDL, VLDL, and triglyceride levels are related to an increased risk of CHD, Step 3 excludes the incorporation of these lipid values into the risk assessment. Low HDL levels are considered a risk factor, while high HDL levels (>60 mg/dL) have a protective effect in preventing CHD and subsequently allows the subtraction of one risk factor.

Known risk factors for CHD include:

1. Cigarette smoking
2. Hypertension (systolic BP \geq 140/90 or currently on antihypertensive medication)
3. Low HDL cholesterol (< 40 mg/dL)
4. Age: men \geq 45 years old & women \geq 55 years old
5. Family history of premature CHD
 - CHD in a first degree male relative <55 years old.
 - CHD in a first degree female relative <65 years old.
(First degree is defined as parents or siblings.)

The sum of the above 5 risk factors is utilized in Step 4 to determine the patient's LDL goal. If a patient has zero or one risk factor, the LDL goal is <160 mg/dl, and it is

generally not necessary to calculate a 10-year risk assessment in Step 4, as these patients typically have a less than a 10% risk of developing CHD in the next ten years. If the patient has 2 or more risk factors, the 10-year risk assessment should be performed.

Other risk factors which are thought to increase the risk of CHD, but are not included in the ATP III risk assessment include:

1. Elevated blood glucose levels
2. Obesity
3. Atherogenic diet
4. Physical inactivity
5. Thrombogenic/hemostatic factors
6. Elevated homocysteine levels

There is evidence elevated homocysteine levels are positively correlated with an increased risk of CHD, but elevations are not seen consistently among all patients with known CHD; therefore, homocysteine levels are not routinely used as risk factor.⁽³³⁾

Step 4: Determine 10-year Coronary Heart Disease Risk

The purpose of determining a patient's 10-year risk of CHD is to determine the aggressiveness of treatment needed to prevent CHD. Appendix D provides a 10-year risk assessment tool (based on the Framingham risk assessment) for both men and women. Positive and negative points are assigned based on a patient's risk factors. Information needed to complete the tool are 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 points are tallied resulting in an estimated 10-year risk of developing CHD.⁽⁴⁾ An online 10-year risk calculator may be found at www.nhlbi.nih.gov/guidelines/cholesterol.

Another method used to predict the risk of developing CHD is the ratio of total cholesterol (TC) to HDL cholesterol. These values measure the opposite ends of the risk determination: TC infers atherogenic potential and an elevated HDL level provides CHD protection. A calculated TC/HDL ratio of >4.5 has been associated with an increased risk of CHD.⁽⁴⁾ The Framingham 10-year risk assessment, which incorporates both of these

factors in addition to other risk factors, is approximately 85% accurate in predicting future CHD risk compared to the TC/HDL ratio accuracy rate of 72%.⁽³⁴⁾

Step 5: Determine Risk Category and Lipid Therapeutic Goals

Once a 10-year CHD risk assessment has been calculated, it is then necessary to stratify the patient’s information into Table 6 which reflects the new recommendations for lipid goals and management. The ATP III Guideline was revised to reflect more aggressive management of higher risk groups, primarily based on new clinical trial evidence which demonstrated added benefit to achieving lower LDL goals than was previously recommended.⁽³¹⁾ In a trial in over 4,000 patients with a documented history of a myocardial infarction or unstable angina, patients were randomized to receive two different levels of aggressiveness in lipid lowering. Patients who received the more aggressive regimen were able to achieve a median LDL level of 62 mg/dL and had a statistically significant reduction in deaths from any cause or a major cardiovascular event.⁽³⁵⁾ Therefore, it is reasonable to aim for LDL levels <70 mg/dL for very high risk patients.⁽³¹⁾

Step 5a: Determine the risk category
(high, moderately high, moderate, and low)

Step 5b: Determine the LDL goal

Step 5c: Based on Steps 5a & 5b, the breakpoints of when to initiate therapeutic lifestyle changes (TLC) and drug therapy are determined.

Risk Category	LDL-C Goal (mg/dL)	Initiate TLC^a (mg/dL)	Initiate Drug Therapy (mg/dL)
High risk: CHD ^b or CHD equivalents ^c (10-year risk >20%)	< 100 (optional goal: <70) ^d	≥ 100	≥ 130 (<100 consider drug)
Moderately high risk: 2+ risk factors (10-year risk 10% to 20%)	< 130 (optional goal: <100) ^e	≥ 130	≥ 130 (100-129 consider drug)
Moderate risk: 2+ risk factors: (10-year risk < 10%)	< 130	≥ 130	≥ 160
Lower risk: 0-1 risk factors	< 160	≥ 160	≥ 190 (160-189 LDL-lowering drug optional)

- a. Dietary modification, weight reduction, exercise
- b. CHD includes history of myocardial infarction, unstable angina, stable angina, coronary artery procedures (angioplasty or bypass surgery), or evidence of clinically significant myocardial ischemia
- c. CHD risk equivalents include clinical manifestations of noncoronary forms of atherosclerotic disease (peripheral arterial disease, abdominal aortic aneurysm, and carotid artery disease [transient ischemic attacks or stroke of carotid origin or >50% obstruction of carotid artery]), diabetes, and 2+ risk factors with 10-year risk for hard CHD >20%.
- d. Consider an LDL-C goal <70 mg/dL in very high-risk patients (known CHD + multiple risk factors (especially diabetes), severe and poorly controlled risk factors (especially continued cigarette smoking), multiple risk factors of the metabolic syndrome (especially high triglycerides \geq 200 mg/dL plus non-HDL-C \geq 130 mg/dL with low HDL-C [$<$ 40 mg/dL]), and acute coronary syndromes.
- e. Consider LDL-C goal <100 mg/dL for patients of advancing age, more than 2 risk factors, severe risk factors (eg, continued cigarette smoking, a strongly positive family history of premature atherosclerotic CVD), high triglycerides (\geq 200 mg/dL) plus elevated non-HDL-C (\geq 160 mg/dL), low HDL-C (<40 mg/dL), the metabolic syndrome, and/or the presence of emerging risk factors (eg, serum high-sensitivity C-reactive protein >3 mg/L or coronary calcium >75th percentile for a person's age and sex).

Table 6: ATP III LDL-C Goals and Cutpoints for Therapeutic Lifestyle Change (TLC) and Drug Therapy in Different Risk Categories and Proposed Modifications Based on Recent Clinical Trial Evidence ⁽³¹⁾

ATP III recommends the reduction of LDL cholesterol as the primary goal of lipid management, as the achievement of LDL goals has been proven to maximize short-term and long-term reductions in CHD. Once LDL goals are achieved or close to being achieved, emphasis then shifts toward treatment of metabolic syndrome (see Step 8) and other lipid risk factors (i.e., low HDL or elevated triglycerides). To achieve LDL goals, two main areas are targeted for intervention: therapeutic lifestyle changes and pharmacologic therapy.⁽⁴⁾

Step 6: Features of the Therapeutic Lifestyle Changes (TLC)

Multiple components are involved in TLC and include the following:⁽⁴⁾

- Decrease dietary intake of saturated fats and cholesterol. Table 7 provides a complete list of nutritional components of the TLC Diet, and Table 8 describes characteristics of dietary fats;
- Increase intake of dietary options which enhance LDL lowering: plant sterols or stanols (Benacol™ and Take Control™) and viscous (soluble) fiber; (Plant sterols occur naturally in fruits and vegetables in small amounts. They can also be added to some foods as a supplement.)
- Weight reduction; and
- Increased physical activity.

Nutrient	Recommended Intake
Saturated fat	- < 7% of total calories - Trans fatty acids should be kept at a low intake
Polyunsaturated fat	- Up to 10% of total calories
Monounsaturated fat	- Up to 20% of total calories
Total fat	- 25-35% of total calories
Carbohydrate	- 50-60% of total calories - Predominantly from complex carbohydrates (i.e., whole grains, fruits and vegetables)
Fiber	- 20-30 grams daily
Protein	- Approximately 15% of total calories
Cholesterol	- < 200 mg daily
Total calories	- Balance energy intake and expenditure to maintain desirable body weight or to prevent weight gain

Table 7: Nutritional Composition of the TLC Diet⁽⁴⁾

	Good		Bad	Ugly
Type of fat	Mono-unsaturated	Poly-unsaturated	Saturated	Trans-fat
Physical appearance at room temp.	Liquid	Liquid	Solid	Solid (due to hydrogenation of liquid oils)
Dietary sources	- Nuts, avocados, & olives - Olive, peanut & canola oils	- Fish (ex. salmon) - Corn, soybean, safflower, sunflower & sesame oils	- Meats, poultry, & dairy - Coconut & palm oils - Commercially fried & baked goods	- Margarine & vegetable shortening - Commercially fried & baked goods
Effect on cholesterol				
LDL	↓	↓	↑	↑
HDL	No effect	↓	No effect	↓

Table 8: Characteristics of Dietary Fats^(36,37)

Just as a point of clarification, previously NCEP developed the Step I and Step II diets to assist patients achieve their target LDL goals. In 2001, when NCEP presented the ATP III Guidelines, the Step I and Step II diets were replaced by the TLC diet.⁽³⁸⁾ Appendix E provides a list of foods which are consistent with the TLC diet. Not all of the TLC components need to be started at the same time, but rather ATP III suggests a stepwise initiation (Figure 14.)⁽⁴⁾

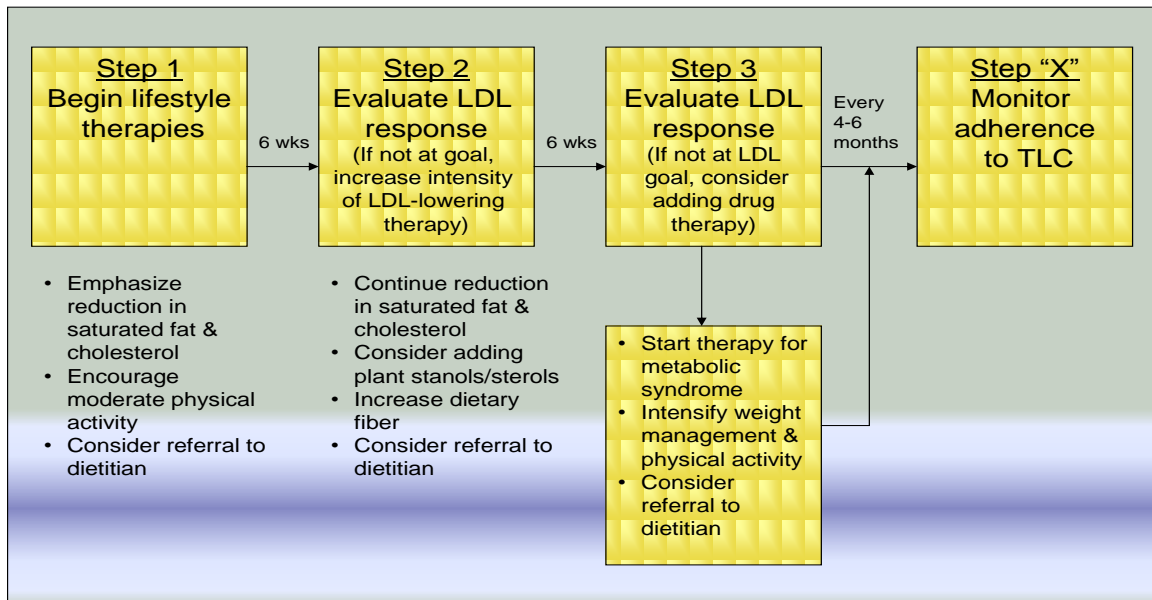


Figure 14: Steps to the Therapeutic Lifestyle Changes⁽⁴⁾

Use of Dietary Supplements

Dietary supplements have been a recent focus in decreasing the development of CHD with the main contributors being omega-3 fatty acids and antioxidants. Omega-3 fatty acids are thought to decrease hepatic production of triglycerides and subsequently VLDL, thereby decreasing the potential for atherogenesis.⁽³⁹⁾

Omega-3 fatty acids have two main sources:⁽³⁹⁾

1. Fish oils which contain eicosapentanoic acid (EPA) and docosahexanoic 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 dietary supplementation in patients with pre-existing heart disease and recommends patients increase omega-3 containing foods in the diet of patients without diagnosed CHD (Table 9).⁽⁴⁰⁾

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 reviewed 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 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 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. Patient 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 have a history of bleeding disorders. 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.

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

Table 9: American Heart Association Recommendations for Use of Omega-3 Fatty Acids⁽⁴⁰⁾

It is thought that the use of antioxidants, particularly vitamin C, vitamin E, beta-carotene, coenzyme Q10, and selenium, may decrease the negative effects of oxidized LDL cholesterol in atherogenesis. Individual clinical trials have shown mixed results in a variety of cardiovascular-related populations, but the long-term safety and efficacy of these treatments is not fully known.⁽⁴⁾ Recently a meta-analysis of vitamin E studies reported an increase in all-cause mortality in patients taking vitamin E, especially at higher doses. A dose-related effect was seen with a progressive increase in mortality. The authors concluded the use of vitamin E at doses ≥ 400 international units per day is not recommended.⁽⁴¹⁾

Step 7: Drug Therapy for Achievement of Therapeutic Goals

Some patients will require the use of pharmacologic intervention to achieve LDL goals. Currently the statins are the most powerful class of medications to lower LDL levels. Similar to the TLC implementation, a stepwise approach for initiating drug therapy is used (Figure 15). Once LDL goals are achieved or close to being achieved, focus can then shift on 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.⁽⁴⁾

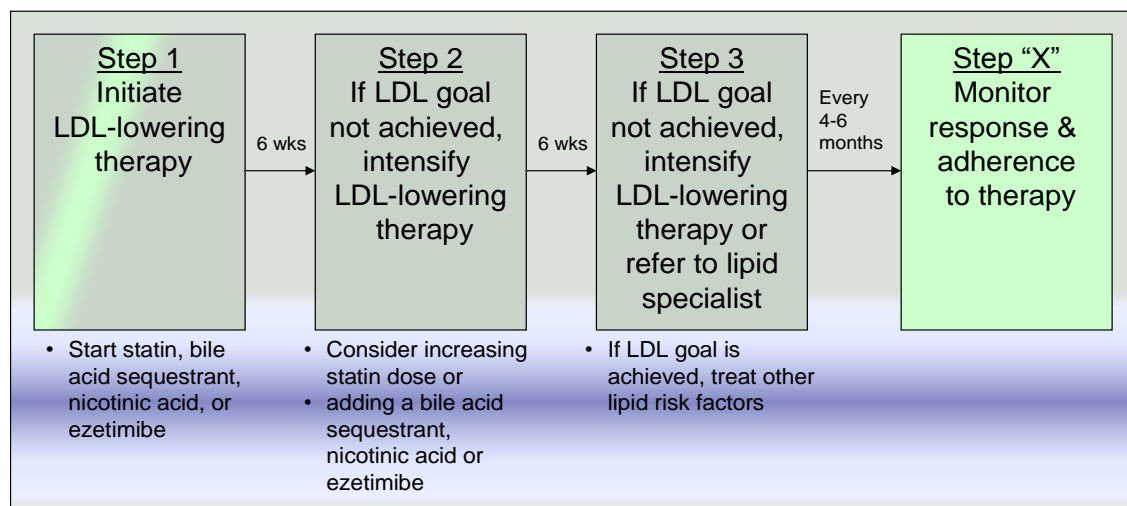


Figure 15: Steps of Drug Therapy⁽⁴⁾

Implementing drug therapy is beyond the scope of lipid screening and will not be discussed further in this module, but Table 10 briefly describes the common agents used in lipid lowering.^(4,42)

Drug Class	Agents (Daily doses)	Effect on Lipids		Major Side Effects
<i>HMG CoA reductase inhibitors (statins)</i>	Atorvastatin (10-80 mg) Fluvastatin (20-80 mg) Lovastatin (20-80 mg) Pravastatin (20-40 mg) Rosuvastatin (5-40 mg) Simvastatin (20-80 mg)	LDL HDL TG	↓ 18-55% ↑ 5-15% ↓ 7-40%	- Myopathy - Increased liver enzymes
<i>Bile acid sequestrants</i>	Cholestyramine (4-16 g) Colesevelam (2.6-8.3 g) Colestipol (5-20 g)	LDL HDL TG	↓ 15-30% ↑ 3-5% no effect	- GI distress - Constipation - Decreases absorption of other drugs
<i>Nicotinic acid</i>	Immediate release (1.5-3 g) Extended release (1-2 g)	LDL HDL TG	↓ 2-25% ↑ 15-35% ↓ 20-50%	- Flushing - Hyperglycemia - Hyperuricemia - GI distress - Hepatotoxicity
<i>Fibric acid derivatives</i>	Fenofibrate (200 mg) Gemfibrozil (1200 mg)	LDL HDL TG	↓ 5-20% ↑ 10-20% ↓ 20-50%	- GI distress - Gallstones - Myopathy
<i>Antilipemics</i>	Ezetimibe (10 mg)	LDL HDL TG	↓ 18% ↑ 1% ↓ 8%	- Headache - Fatigue - Diarrhea

TG = triglycerides

Table 10: Pharmacologic Agents Used in Lipid Lowering^(4,42)

Step 8: Assessment of Metabolic Syndrome

Metabolic syndrome is a constellation of cardiovascular risk factors, including hypertension, dyslipidemias, abdominal obesity, insulin resistance, and hyperinsulinemia.⁽⁴⁾ According to the National Health and Nutrition Examination Survey (NHANES) 2003-2006, approximately 34% of U.S. adults met the criteria for metabolic syndrome.⁽⁴³⁾

According to ATP III, clinical identification of metabolic syndrome requires any three of the following:⁽⁴⁾

- (1) Abdominal obesity:
 - (a) Men > 102 cm (40 inches)
 - (b) Women > 88 cm (35 inches)

- (2) Triglycerides ≥ 150 mg/dL (or treated with medication)
- (3) HDL cholesterol:
 - (a) Men < 40 mg/dL
 - (b) Women < 50 mg/dL
- (4) Blood pressure $\geq 130/ \geq 85$ mmHg (or treated with medication)
- (5) Fasting glucose ≥ 100 mg/dL (or treated with medication)

NOTE: The National Heart, Lung and Blood Institute and the American Heart Institute have lowered the value of fasting glucose from the ATP III value of ≥ 110 mg/dL based on the recommendation of the American Diabetes Association which redefined impaired fasting glucose as ≥ 100 mg/dL.⁽⁴⁴⁾

Patients with metabolic syndrome are at higher risk of developing type 2 diabetes and/or CHD. Therefore, identifying patients with the above symptoms has important implications for their future health. The three main objectives for managing patients with metabolic syndrome are:⁽⁴⁾

- (1) Decrease the underlying causes (obesity and physical inactivity); and
- (2) Treat lipid and non-lipid risk factors
 - (a) Treat hypertension
 - (b) Use aspirin for CHD patients to reduce a prothrombotic state
 - (c) Treat elevated triglycerides and/or low HDL (See the next Step 9)
- (3) Treat hyperinsulinemia and insulin resistance through lifestyle changes and medication, if needed

Step 9: Treatment of Elevated Triglycerides & Low HDL Cholesterol

Elevated Triglycerides

Patients with elevated triglycerides are treated based on the level of elevation (Table 11). For patients with a triglyceride ≥ 200 mg/dL, ATP III utilizes the concept of evaluating the non-HDL cholesterol as a measure of the atherogenic apolipoprotein B containing particles. In essence, non-HDL cholesterol is the combination of LDL and VLDL cholesterol.^(4,42)

Total cholesterol (TC) = HDL + LDL + VLDL	
TC – HDL	= LDL + VLDL
Non-HDL	= LDL + VLDL

Value (mg/dL)	Classification	Treatment
< 150	Normal	None required
150-199	Borderline	<ul style="list-style-type: none"> - Primary aim is to reach LDL goal - Intensify weight management - Increase physical activity
200-499	High	<ul style="list-style-type: none"> - Primary aim is to reach LDL goal - Set secondary goal for non-HDL cholesterol to be 30 mg/dL higher than the patient's LDL goal. This may be accomplished by intensifying LDL-lowering medication or by adding either a fibrate or nicotinic acid medication.
≥ 500	Very High	<ul style="list-style-type: none"> - Primary aim is to prevent pancreatitis! First lower triglyceride level before addressing LDL goal. <ol style="list-style-type: none"> 1. Very low fat diet (≤ 15% calories from fat) 2. Weight management and increased physical activity 3. Add fibrate or nicotinic acid medication - Once triglycerides are below 500 mg/dL, return to the achievement of the LDL goal.

Table 11: Classification and Treatment for Elevated Triglycerides⁽⁴⁾

Low HDL Levels

ATP III does not specify a HDL goal but recognizes the clinical benefit of raising the HDL value. Treatment for a low HDL (< 40 mg/dL) is as follows:⁽⁴⁾

- The achievement of the LDL goal is first priority.
- Intensify weight management and increase physical activity.
- Smoking cessation.
- If triglycerides are between 200-499 mg/dL, achieve the non-HDL goal.
- If triglycerides are >200 mg/dL in patients with CHD or CHD risk equivalents, consider adding nicotinic acid, a fibrate, or a statin.

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, 2/3 of those treated with medications are not at their LDL goal.⁽⁴³⁾

According to the NCEP ATPIII Guidelines, the following recommendations reflect who should be tested for lipid abnormalities.⁽⁴⁾

- (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 5 years in adults age 20 and over.
- (2) More frequent measurements are required for persons with multiple risk factors or, in those with 0 – 1 risk factor, if the LDL level is only slightly below the goal level. Risk factors include:
 - (a) Cigarette smoking
 - (b) Hypertension
 - (c) Low HDL cholesterol
 - (d) Age: men \geq 45 years old & women \geq 55 years old
 - (e) Significant family history of cardiovascular disease; male relative before age 55 and female relative before age 65.

B. Point-of-Care Lipid Testing

1. Cholestech LDX[®]

The Cholestech LDX[®] is a CLIA-waived, portable device designed to provide point-of-care analysis of lipid levels obtained from capillary or venous blood samples.⁽⁴⁵⁾

The analyzer utilizes reflectance photometry, which 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 Cholestech LDX[®] optically detects the color change and quantifies the amounts of substances into mg/dL.⁽⁴⁵⁾

Accuracy & Precision

Recall from the “*Overview of Geriatric Health Screening*” module 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.⁽⁴⁵⁾

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

The National Cholesterol Education Program (NCEP) has issued guidelines specifying an acceptable upper limit of % bias, % CV, and TE for total cholesterol (TC), LDL, HDL, and triglycerides (TG). Multiple studies have demonstrated the use of the Cholestech LDX[®] to be precise and accurate in its measurement of TC, HDL, and TG. Pooled results from multiple studies did not always reveal % bias and % CV within the NCEP guidelines, but the pooled TE for TC, HDL, and TG always met the NCEP criteria. The estimated sensitivity and specificity for the Cholestech LDX[®] is 92.8% and 97.4%, respectively.

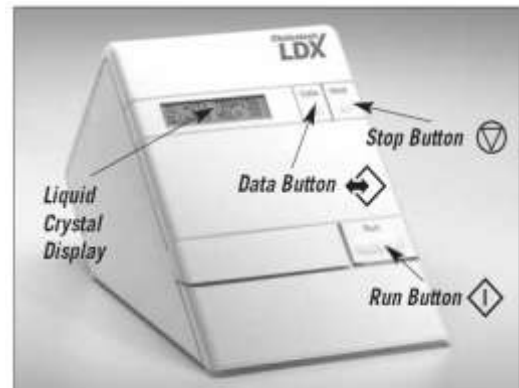
It should be noted that the Cholestech LDX[®] does not directly measure LDL or VLDL but derives them from the following Friedewald equation.⁽⁴⁶⁾

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

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

2. Components of the Cholestech LDX[®] (47)

- Cholestech LDX[®] analyzer
- Power supply
- Optics check cassette
- User manual
- Capillary tubes and plunger
- Lancets
- MiniPet pipette (for running control samples)
- Pipette tips
- Printer & cable assembly

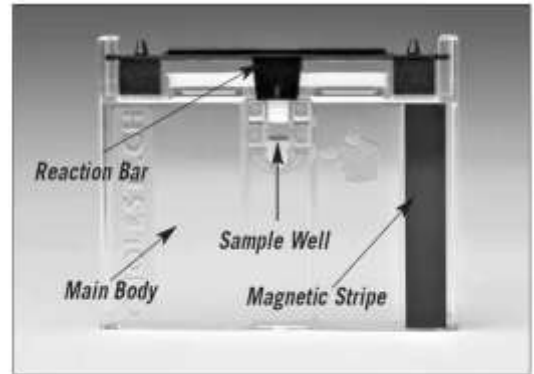


Additional materials needed to perform the testing are:

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

3. Components of the Cholestech Test Cassette⁽⁴⁷⁾

- 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.
- The reaction bar holds the reagent pads which contain the chemicals for each test.



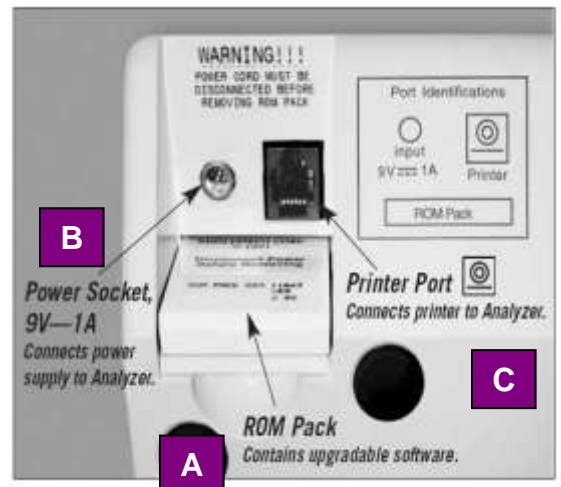
4. Setting Up the Cholestech LDX[®] (47)

Locational requirements for the Cholestech LDX[®] machine are:

- Room temperature 68-87° F (20-31°C)
- A stable work surface
- No direct heat (oven or room heater)
- No direct light (sunlight or spotlight)

Steps to set up Cholestech LDX[®]

- Ensure the ROM pack (software package) is completely inserted into the back of the analyzer
- Insert the power supply plug into the round socket
- Connect printer port to analyzer.
- Plug the power supply into a power source.



5. Quality Assurance of the Cholestech LDX[®] (47)

There are three methods to ensure accurate results from the Cholestech LDX[®] machine.

- a) **Optics check test cassette.** This cassette is used to check the optical system of the analyzer. Each day prior to using the analyzer, an optics test should be performed.

Performing the optics test:

- (1) Press RUN on analyzer; verify self-test OK message.
- (2) Place optics check cassette into the analyzer drawer right-side up and with the magnetic strip on the right. DO NOT place a blood sample on the cassette.
- (3) Press RUN; analyzer will automatically perform self test.
- (4) Results on the data analyzer will contain four numbers:
Example: 81 – 96 – 100 – 85

These numbers should be within the range specified on the optical test cassette. If they are not, the analyzer will display, “Optics test failed”. If the test fails, repeat the optics test. 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. Cholestech manufactures lipid control levels to be used with an actual test cassette: Lipid 1 for low levels and Lipid 2 for high levels. The control levels are to be kept refrigerated at 2-8°C (36-46°F), and are stable after opening for 30 days when stored at the same refrigerated temperatures. It is suggested to run control samples when a new lot of test cassettes are being used for the first time, or if repeated errors are occurring when running patient tests.

Running a control sample:

- (1) Press RUN on analyzer; verify self-test OK message. (Analyzer drawer will open.)
 - (2) Bring to room temperature (about 10 minutes) one of the two lipid control vials since both control tests cannot be run simultaneously.
 - (3) Gently mix the vial solution by inverting the vial 7-8 times.
 - (4) Using the MiniPet with attached pipette tip, draw up a sample of the control solution (35 μ l) and expel the contents into the sample well on a test cassette. Place the cassette into the analyzer and press RUN. Wipe off the top of the control lipid vial, replace the cap, and place in the refrigerator.
 - (5) Results will display on the analyzer and must be within the established reference ranges for the specific lipid control vial (which is provided with each control vial) prior to analyzing patient samples.
- c) **Visual examination of the test cassette.** If an error is noted after the analysis of a patient sample is performed, visually examining the edge of the test cassette may be warranted. The edge containing the reaction bar has four windows which allows the viewer to look for color changes in the underlying reaction pads. All the pads should change color in a fairly even manner. If the pads did not uniformly change, than the pads may not have completely reacted with the sample, possibly due to an insufficient sample. A new sample will need to be performed to get an accurate result.

6. Use of the Cholestech LDX^{®(48)}

The information found in Appendix F 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 onto 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 their non-dominant hand, as a band-aid will be placed on the finger utilized for the blood sample, and the less 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 finger tip 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 cassette for analysis. The collected sample needs to be expelled into the cassette 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 cassette immediately available.

- j. Place the cassette into the Cholestech LDX and press “Run”. The test will take about 5 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 Step 1 of the ATP At-A-Glance reference. Additionally, Appendix G provides a patient specific assessment tool to help guide the health screener through a patient's lipid assessment.

V. Useful Websites

- ★ Highly recommended websites for further understanding of key concepts related to geriatric screening.
 1. Governmental
 - a. National Heart, Lung and Blood Institute. <http://www.nhlbi.nih.gov/index.htm> ★
 - b. National Cholesterol Education Program (NCEP). <http://www.nhlbi.nih.gov/about/ncep/index.htm> ★
 - c. Administration on Aging. <http://www.aoa.gov>
 - d. Montana Cardiovascular Health Program. <http://www.dphhs.mt.gov/PHSD/cardiovascular/cardio-index.shtml>
 2. Cardiovascular Health
 - a. American Heart Association. <http://www.americanheart.org> ★
 - b. American College of Cardiology. <http://www.acc.org> ★
 - c. Adult Treatment Plan (ATP III) Guidelines <http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm> ★
 - d. Lipids online: an online resource for clinicians, researchers, & educators related to atherosclerosis, dyslipidemias, and lipid management. <http://www.lipidsonline.org>
 - e. The Heart: 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. <http://www.theheart.org>
 - f. National Lipid Education Council's cholesterol management resource. <http://www.lipidhealth.org>
 3. Miscellaneous
 - a. Clinical guidelines on obesity and overweight http://www.nhlbi.nih.gov/guidelines/obesity/ob_home.htm ★
 - b. Your Guide to Lowering Your Cholesterol with TLC: http://www.nhlbi.nih.gov/health/public/heart/cho/cho_tlc.pdf

VI. References

1. National Vital Statistics Report. Deaths: Final data for 2009,59(4), September 16, 2011. Available at http://www.cdc.gov/nchs/data/nvsr/nvsr59/nvsr59_04.pdf. Accessed on May 11, 2011.
2. Death rates for diseases of the heart, according to sex, race, Hispanic origin, and age: United States, selected years 1950-2007. Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System. Available at <http://www.cdc.gov/nchs/data/hus/hus10.pdf#030>. Accessed on May 11, 2011.
3. The Burden of Heart Disease and Stroke in the Big Sky State. Available at http://www.dphhs.mt.gov/PHSD/cardiovascular/pdf/146192LR_000.pdf. Accessed May 11, 2011.
4. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation* 2002;106:3143-21.
5. Cooper R, Cutler J, Desvigne-Nickens P, Fortmann SP, Friedman L, Havlik R, et al. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: Findings of the National Conference on cardiovascular disease prevention. *Circulation* 2000;102:3137-47.
6. Stamler J, Stamler R, Neaton JD, Wentworth D, Daviglius ML, Garside D, et al. Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: Findings for 4 large cohorts of young adult and middle-ages men and women. *JAMA* 1999;282:2012-18.
7. The lipid research Clinics coronary primary prevention trial results: Reduction in incidence of coronary heart disease. *JAMA* 1984;251:351-64.
8. Shepherd J, Cobbe SM, Ford I, Isles CG, Lorimer AR, MacFarlane PW, et al. Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia. West of Scotland Coronary Prevention Study Group. *N Engl J Med* 1995;333:1301-7.
9. Downs JR, Clearfield M, Weis S, Witney E, Shapiro DR, Beere PA, et al. Primary prevention of acute coronary events with lovastatin in men and women with average cholesterol levels: results of AFCAPS/TexCAPS. Air Force/Texas Coronary Atherosclerosis Prevention Study. *JAMA* 1998;279:1615-22.
10. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet* 1994;344:1383-9.
11. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group. *N Engl J Med* 1998;339:1349-57.
12. Ridker PM, Rifai N, Pfeffer MA, Sacks F, Braunwald E. Long-term effects of pravastatin on plasma concentration of C-reactive protein. The Cholesterol and Recurrent Events (CARE) Investigators. *Circulation* 1999;100:230-5.

13. The effect of aggressive lowering of low-density lipoprotein cholesterol levels and low-dose anticoagulation on obstructive changes in saphenous-vein coronary-artery bypass grafts. The Post Coronary Artery Bypass Graft Trial Investigators. *N Eng J Med* 1997;336:153-62. Erratum in: *N Engl J Med* 1997;337:1859.
14. Schwartz GG, Olsson AG, Ezekowitz MD, Ganz P, Oliver MF, Waters D, et al. Myocardial Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL) Study Investigators. Effects of atorvastatin on early recurrent ischemic events in acute coronary syndromes: the MIRACL study: a randomized controlled trial. *JAMA* 2001;285:1711-8.
15. Pitt B, Waters D, Brown WV, van Boven AJ, Schwartz L, Title LM, et al. Aggressive lipid-lowering therapy compared with angioplasty in stable coronary artery disease. Atorvastatin versus Revascularization Treatment Investigators. *N Engl J Med* 1999;341:70-6.
16. Gould AL, Rossouq JE, Santanello NC, Heyes JF, Furberg CD. Cholesterol reduction yields clinical benefit: Impact of statin trials. *Circulation* 1998;97:946-52.
17. Barrett KE, Barman SM, Boitano S, Brooks H. Energy balance, metabolism and nutrition. In: Ganong's . Review of medical physiology. 23th ed. New York (NY): McGraw-Hill;2010.
18. Rader DL, Hobbs HH. Lipoprotein metabolism. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, and Loscalzo J, Eds.. *Harrison's principles of internal medicine*. 17th ed. Online edition. McGraw-Hill;2008. Available at <http://www.merckmedicus.com>. Accessed May 11, 2011.
19. Ridker PM, Genest J, Libby P. Risk factor for atherosclerotic disease. In: Bonow RO, Mann DL, Zipes DP, Libby P, editors. *Braunwald: Heart Disease: A textbook of cardiovascular medicine*. 9th ed. St. Louis (MO):W.B. Saunders;2012.
20. Schoen FJ. Blood vessels. In: Kumar V, Abbas AK, Fausto N, editors. *Kumar: Robbins and Cotran: Pathologic Basis of Disease*, 7th ed. St. Louis (MO):W.B. Saunders;2005. p. 516-25.
21. Cohen DE. Cholesterol metabolism and the concept of dual inhibition. Online presentation. Available at <http://www.lipidsonline.org/slides/slide01.cfm?q=dual+inhibition>. Accessed May 11, 2011.
22. The gastrointestinal system. In: Rhodes R, Pflanzer R, editors. *Human Physiology*. 4rd ed. Forth Worth (TX): Saunders College Publishing; 2003.
23. Merck/Schering-Plough Pharmaceuticals. Zetia[®] package insert. Whitehouse Station, NJ: July 2009.
24. Nabel EG. Cardiovascular disease. *N Engl J Med* 2003;349:60-72.
25. The American Association of Clinical Endocrinologists medical guidelines for clinical practice for the diagnosis and treatment of dyslipidemia and prevention of atherosclerosis. *Endocr Pract* 2000;6:162-213.
26. Falk E, Shah PK, Fuster V. Coronary plaque disruption. *Circulation* 1995;92:657-71.
27. Little WC, Constantinescu M, Applegate RJ, Kutcher MA, Burrows MT, Kahl FR, Santamore JP. Can coronary angiography predict the site of subsequent myocardial infarction in patients with mild to moderate coronary artery disease? *Circulation* 1998;78:1157-66.

28. Ambrose JA, Tannenbaum MA, Alexopoulos D, Hjemdahl-Monsen CE, Leavy J, Weiss M, et al. Angiographic progression of coronary artery disease and the development of myocardial infarction. *J Am Coll Cardiol* 1988;12:56-62.
29. Strong JP, Malcom GT, McMahan CA, Tracey RE, Newman WP 3rd, Herderick EE, Cornhill JF. Prevalence and extent of atherosclerosis in adolescents and young adults. Implications for prevention from the pathobiological determinants of atherosclerosis in youth study. *JAMA* 1999;281:727-35.
30. About the National Cholesterol Education Program (NCEP), National Heart, Lung and Blood Institute. Available at <http://www.nhlbi.nih.gov/about/ncep/index.htm>. Accessed May 11, 2011.
31. Grundy SM, Cleeman JI, Bairey-Merz CN, Brewer HB, Clark LT, Hunninhake DB, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. *Circulation* 2004;110:227-39.
32. ATP III Guidelines At-A-Glance Quick Desk Reference. National Heart, Lung and Blood Institute; National Institutes of Health publication No. 01-3305, May 2001. Available at <http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.pdf>. Accessed May 11, 2011.
33. Hackman DG, Anand SS. Emerging risk factors for atherosclerotic vascular disease: a critical review of the evidence. *JAMA* 2003;290:932-40.
34. Pignone MP, Phillips CJ, Atkins D, Teutsch SM, Mulrow CD, Lohr KN. Screening and treating adults for lipid disorders. *Am J Prev Med* 2001;20:77-89.
35. Cannon CP, Braunwald E, McCabe CH, Rader DJ, Rouleau JL, Belder R, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. *N Engl J Med* 2004;350-:1495-504.
36. Know your fats. American Heart Association. Available at http://www.heart.org/HEARTORG/Conditions/Cholesterol/PreventionTreatmentofHighCholesterol/Know-Your-Fats_UCM_305628_Article.jsp. Accessed on May 11, 2011.
37. Nutrition Fact Sheet: Lipids. Northwestern Nutrition, Feinberg School of Medicine. Available at <http://nuinfo-prot04.northwestern.edu/nutrition/factsheets/lipids.html>. Accessed May 11, 2011.
38. Step I, Step II and TLC diets. American Heart Association. Available at <http://www.americanheart.org/presenter.jhtml?identifier=4764>. Accessed on May 11, 2011.
39. Covington MB. Omega-3 fatty acids. *Am Fam Physician* 2004;70:133-40.
40. Kris-Etherton PM, Harris WS, Appel LJ; American Heart Association. Nutrition Committee. Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. *Circulation* 2002;106:2747-57. Erratum in: *Circulation* 2003;107:512.
41. Miller ER 3rd, Pastor-Barriuso R, Dalal D, Riemersma RA, Appel LJ, Guallar E. Meta-analysis: high-dosage vitamin E supplementation may increase all-cause mortality. *Ann Intern Med* 2005;142:37-46.
42. Gotto AM, Guthrie R, Ito MK. Guide to dyslipidemia. *Pharmacy Practice News: Primary Care Special Edition* 2004;8:11-19.

43. Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS). National Health and Nutrition Examination Survey Data. Hyattsville, MD: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 2003-2006. Available at <http://www.cdc.gov/nchs/nhanes.htm>. Accessed on May 26, 2011.
44. Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Kahn R, et al. Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care* 2003;26:3160-7.
45. Evaluation of near patient cholesterol testing using the Cholestech LDX. Australia's Medical Services Advisory Committee application 1026: Assessment report; August 2001. Available at [http://www.health.gov.au/internet/msac/publishing.nsf/Content/1E0803AD53E44E39CA2575AD0082FD91/\\$File/1026%20-%20Near%20Patient%20Testing%20using%20Cholestech%20LDX%20Report.pdf](http://www.health.gov.au/internet/msac/publishing.nsf/Content/1E0803AD53E44E39CA2575AD0082FD91/$File/1026%20-%20Near%20Patient%20Testing%20using%20Cholestech%20LDX%20Report.pdf). Accessed May 11, 2011.
46. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without the preparative ultracentrifuge. *Clin Chem* 1972;18:449-502.
47. Cholestech LDX[®] System User Manual. Cholestech Corporation. Hayward (CA); 2002.
48. Cholestech LDX[®] Quick Reference Guide. Cholestech Corporation, Hayward (CA); 2010. Available at http://www.cholesteck.com/docs/ldx/LDX_QRG_EN.pdf. Accessed May 11, 2011.

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 before any tests are completed by an IPHARM Clinical Pharmacist Specialist (CPS), Principle Investigator (PI), or Project Coordinator (PC).

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, a Band-aid 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.

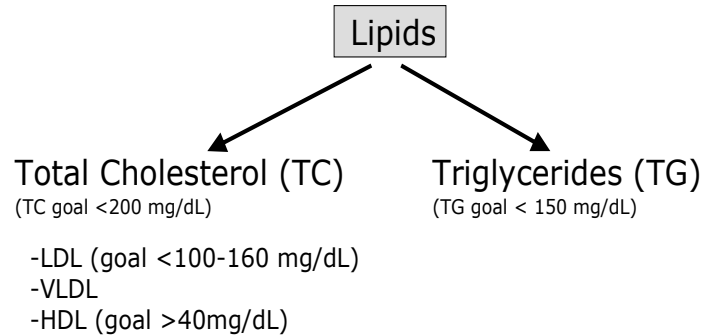
Appendix C Counseling

Lipid Screening: Topics for Patient

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. Recommended for every adult over 20 years of age to have routine lipid profile checks (at least once every 5 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 (ex., presence of heart disease, diabetes, peripheral artery disease, etc.) and risk factors.

Positive risk factors for coronary heart disease (CHD) which influence LDL goals;

- Cigarette smoking;
- Hypertension (BP \geq 140/90 or patient currently taking an antihypertensive medication);
- HDL < 40 mg/dL;

- Family history of premature CHD: 1st degree male relative < 55 years old, and 1st degree female relative <65 years old; and
- Men ≥ 45 years old and women ≥ 55 years old.

Using the ATP III Guidelines from the National Cholesterol Education Program, the patient's 10-year risk is determined which stratifies their LDL goals and subsequent treatment options.

5. Two main ways to lower LDL cholesterol and raise HDL cholesterol.

a. Therapeutic Lifestyle Changes (TLC)

- 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
- Increase physical activity – 30 minutes of regular exercise on most days of the week.

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 can have 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 ATP III Guidelines. At-A-Glance⁽³¹⁾

National Cholesterol Education Program

ATP III Guidelines At-A-Glance Quick Desk Reference

1

Step 1 Determine lipoprotein levels—obtain complete lipoprotein profile after 9- to 12-hour fast.

ATP III Classification of LDL, Total, and HDL Cholesterol (mg/dL)

LDL Cholesterol – Primary Target of Therapy

<100	Optimal
100-129	Near optimal/above optimal
130-159	Borderline high
160-189	High
≥190	Very high

Total Cholesterol

<200	Desirable
200-239	Borderline high
≥240	High

HDL Cholesterol

<40	Low
≥60	High

2

Step 2 Identify presence of clinical atherosclerotic disease that confers high risk for coronary heart disease (CHD) events (CHD risk equivalent):

- Clinical CHD
- Symptomatic carotid artery disease
- Peripheral arterial disease
- Abdominal aortic aneurysm.

3

Step 3 Determine presence of major risk factors (other than LDL):

Major Risk Factors (Exclusive of LDL Cholesterol) That Modify LDL Goals

Cigarette smoking
Hypertension (BP ≥140/90 mmHg or on antihypertensive medication)
Low HDL cholesterol (<40 mg/dL)*
Family history of premature CHD (CHD in male first degree relative <55 years;
CHD in female first degree relative <65 years)
Age (men ≥45 years; women ≥55 years)

* HDL cholesterol ≥60 mg/dL counts as a "negative" risk factor; its presence removes one risk factor from the total count.

- Note: in ATP III, diabetes is regarded as a CHD risk equivalent.

NATIONAL INSTITUTES OF HEALTH
NATIONAL HEART, LUNG, AND BLOOD INSTITUTE



4
Step 4

If 2+ risk factors (other than LDL) are present without CHD or CHD risk equivalent, assess 10-year (short-term) CHD risk (see Framingham tables).

Three levels of 10-year risk:

- >20% — CHD risk equivalent
- 10-20%
- <10%

5
Step 5

Determine risk category:

- Establish LDL goal of therapy
- Determine need for therapeutic lifestyle changes (TLC)
- Determine level for drug consideration

LDL Cholesterol Goals and Cutpoints for Therapeutic Lifestyle Changes (TLC) and Drug Therapy in Different Risk Categories.

Risk Category	LDL Goal	LDL Level at Which to Initiate Therapeutic Lifestyle Changes (TLC)	LDL Level at Which to Consider Drug Therapy
CHD or CHD Risk Equivalents (10-year risk >20%)	<100 mg/dL	≥100 mg/dL	≥130 mg/dL (100-129 mg/dL: drug optional)*
2+ Risk Factors (10-year risk ≤20%)	<130 mg/dL	≥130 mg/dL	10-year risk 10-20%: ≥130 mg/dL
			10-year risk <10%: ≥160 mg/dL
0-1 Risk Factor†	<160 mg/dL	≥160 mg/dL	≥190 mg/dL (160-189 mg/dL: LDL-lowering drug optional)

* Some authorities recommend use of LDL-lowering drugs in this category if an LDL cholesterol <100 mg/dL cannot be achieved by therapeutic lifestyle changes. Others prefer use of drugs that primarily modify triglycerides and HDL, e.g., nicotinic acid or fibrates. Clinical judgment also may call for deferring drug therapy in this subcategory.

† Almost all people with 0-1 risk factor have a 10-year risk <10%, thus 10-year risk assessment in people with 0-1 risk factor is not necessary.

6
Step 6

Initiate therapeutic lifestyle changes (TLC) if LDL is above goal.

TLC Features

- TLC Diet:
 - Saturated fat <7% of calories, cholesterol <200 mg/day
 - Consider increased viscous (soluble) fiber (10-25 g/day) and plant stanols/sterols (2g/day) as therapeutic options to enhance LDL lowering
- Weight management
- Increased physical activity.

7

Step 7

Consider adding drug therapy if LDL exceeds levels shown in Step 5 table:

- Consider drug simultaneously with TLC for CHD and CHD equivalents
- Consider adding drug to TLC after 3 months for other risk categories.

Drugs Affecting Lipoprotein Metabolism

Drug Class	Agents and Daily Doses	Lipid/Lipoprotein Effects	Side Effects	Contraindications	
HMG CoA reductase inhibitors (statins)	Lovastatin (20-80 mg)	LDL	↓18-55%	Myopathy Increased liver enzymes	Absolute: • Active or chronic liver disease Relative: • Concomitant use of certain drugs*
	Pravastatin (20-40 mg)	HDL	↑5-15%		
	Simvastatin (20-80 mg)	TG	↓7-30%		
	Fluvastatin (20-80 mg)				
	Atorvastatin (10-80 mg) Cerivastatin (0.4-0.8 mg)				
Bile acid sequestrants	Cholestyramine (4-16 g)	LDL	↓15-30%	Gastrointestinal distress Constipation Decreased absorption of other drugs	Absolute: • dysbeta-lipoproteinemia • TG >400 mg/dL Relative: • TG >200 mg/dL
	Colestipol (5-20 g)	HDL	↑3-5%		
	Colesevelam (2.6-3.8 g)	TG	No change or increase		
Nicotinic acid	Immediate release (crystalline) nicotinic acid (1.5-3 gm), extended release nicotinic acid (Niaspan®) (1-2 g), sustained release nicotinic acid (1-2 g)	LDL	↓5-25%	Flushing Hyperglycemia Hyperuricemia (or gout) Upper GI distress Hepatotoxicity	Absolute: • Chronic liver disease • Severe gout Relative: • Diabetes • Hyperuricemia • Peptic ulcer disease
		HDL	↑15-35%		
		TG	↓20-50%		
Fibric acids	Gemfibrozil (600 mg BID) Fenofibrate (200 mg) Clofibrate (1000 mg BID)	LDL	↓5-20%	Dyspepsia Gallstones Myopathy	Absolute: • Severe renal disease • Severe hepatic disease
			<i>(may be increased in patients with high TG)</i>		
		HDL	↑10-20%		
		TG	↓20-50%		

* Cyclosporine, macrolide antibiotics, various anti-fungal agents, and cytochrome P-450 inhibitors (fibrates and niacin should be used with appropriate caution).

Identify metabolic syndrome and treat, if present, after 3 months of TLC.**Clinical Identification of the Metabolic Syndrome – Any 3 of the Following:**

Risk Factor	Defining Level
Abdominal obesity*	Waist circumference [†]
Men	>102 cm (>40 in)
Women	>88 cm (>35 in)
Triglycerides	≥150 mg/dL
HDL cholesterol	
Men	<40 mg/dL
Women	<50 mg/dL
Blood pressure	≥130/≥85 mmHg
Fasting glucose	≥110 mg/dL

* *Overweight and obesity are associated with insulin resistance and the metabolic syndrome. However, the presence of abdominal obesity is more highly correlated with the metabolic risk factors than is an elevated body mass index (BMI). Therefore, the simple measure of waist circumference is recommended to identify the body weight component of the metabolic syndrome.*

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

Treatment of the metabolic syndrome

- Treat underlying causes (overweight/obesity and physical inactivity):
 - Intensify weight management
 - Increase physical activity.
- Treat lipid and non-lipid risk factors if they persist despite these lifestyle therapies:
 - Treat hypertension
 - Use aspirin for CHD patients to reduce prothrombotic state
 - Treat elevated triglycerides and/or low HDL (as shown in Step 9).

Treat elevated triglycerides.**ATP III Classification of Serum Triglycerides (mg/dL)**

<150	Normal
150-199	Borderline high
200-499	High
≥500	Very high

Treatment of elevated triglycerides (≥150 mg/dL)

- Primary aim of therapy is to reach LDL goal
- Intensify weight management
- Increase physical activity
- If triglycerides are ≥200 mg/dL after LDL goal is reached, set secondary goal for non-HDL cholesterol (total – HDL) 30 mg/dL higher than LDL goal.

Comparison of LDL Cholesterol and Non-HDL Cholesterol Goals for Three Risk Categories

Risk Category	LDL Goal (mg/dL)	Non-HDL Goal (mg/dL)
CHD and CHD Risk Equivalent (10-year risk for CHD >20%)	<100	<130
Multiple (2+) Risk Factors and 10-year risk ≤20%	<130	<160
0-1 Risk Factor	<160	<190

If triglycerides 200-499 mg/dL after LDL goal is reached, consider adding drug if needed to reach non-HDL goal:

- intensify therapy with LDL-lowering drug, or
- add nicotinic acid or fibrate to further lower VLDL.

If triglycerides ≥500 mg/dL, first lower triglycerides to prevent pancreatitis:

- very low-fat diet (≤15% of calories from fat)
- weight management and physical activity
- fibrate or nicotinic acid
- when triglycerides <500 mg/dL, turn to LDL-lowering therapy.

Treatment of low HDL cholesterol (<40 mg/dL)

- First reach LDL goal, then:
- Intensify weight management and increase physical activity
- If triglycerides 200-499 mg/dL, achieve non-HDL goal
- If triglycerides <200 mg/dL (isolated low HDL) in CHD or CHD equivalent consider nicotinic acid or fibrate.

Men

Estimate of 10-Year Risk for Men

(Framingham Point Scores)

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

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

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

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

Systolic BP (mmHg)	If Untreated	If Treated
<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥160	2	3

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

10-Year risk _____%

Women

Estimate of 10-Year Risk for Women

(Framingham Point Scores)

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

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

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

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

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

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

10-Year risk _____%

Appendix E Therapeutic Lifestyle Changes: Dietary Options

▶ ▶ ▶ ▶ **Tip sheet** ◀ ◀ ◀ ◀

What to Look for When Grocery Shopping

Courtesy of the National Heart, Lung and Blood Institute.

<http://nhlbisupport.com/chd1/Tipsheets/lookfor.htm>

Breads, Cereals, Rice, and Pasta Group

- Breads** (like whole wheat, rye, pumpernickel, or white)
- Buns, dinner rolls, bagels, English muffins, pita breads**
- Low fat crackers (like bread sticks, saltines, rice crackers)**
- Soft tortillas, corn or whole wheat
- Hot and cold cereals** (except granola or muesli)
- Pasta (like plain noodles, spaghetti, or macaroni)
- Rice (white, brown, wild, basmati, or jasmine)
- Grains (bulgur, cous cous, quinoa, barley, hominy, millet, aramanth)

Fruit and Vegetable Group

- Fruits: any fresh, canned, dried, frozen, without added sugar
- Vegetables: any fresh, frozen, or canned** without cream or cheese sauce
- Fresh or frozen juices, without added sugar

Milk, Yogurt and Cheese Group

- Fat free or 1 % milk
- Cheese** (3 grams of fat or less per serving)
- Lowfat or nonfat yogurt

Meat, Poultry, Fish, Dry Beans, Eggs, and Nuts Group

- Lean cuts of meat:
- Beef: eye of round, top round, sirloin
- Pork: tenderloin, sirloin, top loin
- Veal: shoulder, ground veal, cutlets, sirloin
- Lamb: leg-shank
- Lean or extra lean ground beef
- Chicken or turkey, white or light meat (remove skin)
- Luncheon meats, 95% to 99% fat free
- Fish (most white meat fish is very low in fat, saturated fat and cholesterol)
- Tuna, light meat canned in water
- Shellfish* (shrimp, scallops, crab)
- Dry peas and beans (black-eyed peas, chick peas, kidney beans, lentils, navy beans, soybeans, split peas)
- Peanut butter, reduced fat
- Tofu (soy bean)
- Eggs, egg whites, egg substitutes

*Shellfish is very high in cholesterol. Limit the amount you eat so you don't consume a total of more than 300 milligrams of cholesterol per day if you are following the Heart Healthy Diet or 200 mg per day if you are following the TLC Diet.

**** SODIUM ALERT:**

If you are watching your sodium intake, be sure to check the label to find low-sodium varieties.

Sweets and snacks

- Low fat cookie: animal crackers, devil's food cookies, fig and other fruit bars, ginger snaps, graham crackers, vanilla or lemon wafers
- Angel food cake or other lowfat cakes
- Low fat frozen yogurt, ice milk, fruit ices, sorbet, sherbet
- Pudding (make it with fat free or 1% milk), gelatin desserts
- Popcorn without butter or oil, pretzels, baked tortilla chips**

Fats and Oils

- Margarine** (soft, diet, tub, or liquid)
- Vegetable oil (canola, olive, corn, peanut, sunflower, safflower, or sesame oil)

Miscellaneous/Condiments

- Herbs
- Spices
- Non-stick cooking spray
- Imitation butter (flakes or buds)
- Reduced calorie or fat-free salad dressing
- Reduced fat or non-fat sour cream
- Reduced fat or non-fat mayonnaise
- Mustard (Dijon, etc.)
- Horseradish
- Ginger
- Garlic
- Catsup
- Vinegar
- Lemon juice
- Lime juice
- Jelly or jam
- Sodium-free salt substitute
- Salsa or picante sauce
- Low-fat soup** (broth or tomato-based, bean soup, vegetable, minestrone)
- Spaghetti sauce

**** SODIUM ALERT:**

If you are watching your sodium intake, be sure to check the label to find low-sodium varieties.

Beverages

- Water
- Sparkling water
- Milk, fat free or low fat (1%)
- 100% fruit juice, regular or low calorie
- Lemonade, regular or low calorie
- Iced tea, regular or low calorie
- Tea

Tips for Buying Frozen Foods

Many prepared and frozen foods are high in saturated fat and cholesterol. Look for frozen food packages that say, "Light," "Lean," "Reduced Fat," "Reduced calorie," "Healthy," or "Diet." These version will be lower in saturated fat, cholesterol, calories, and/or sodium than the regular versions. Check out the Reading Food Labels tip sheet for more information.

Tips for Buying Prepared Foods

When choosing prepared foods, choose vegetables, pasta and grain salads and side dishes made without high fat mayonnaise and oil. Steer clear from high saturated fat meats, dressing and other spreads, and dishes with creams and other sauces. Fruit salad is usually available and is always a great choice.

Appendix F: Cholestech LDX[®] Quick Reference⁽⁴⁸⁾



Introduction

The Cholestech LDX is a small, portable analyzer and test cassette system. It is easy to use, and gives fast and reliable results. To run a test, just add a sample to the cassette and place it in the Analyzer. In a few minutes, the results will appear on the screen.

The Cholestech LDX System is for *in vitro* diagnostic use only. This guide contains information intended to familiarize and instruct the user in the proper use and care of the Cholestech LDX System.

System Components

Cholestech LDX Analyzer



Printer



Results from each cassette will print out on the Cholestech Thermal Printer. One or two labels may be printed.

Test Cassette



Blood from a fingerstick sample is placed into a single-use, disposable cassette. Multiple test results are available in 5 minutes.

Capillary Tubes/Plungers



Blood from a fingerstick sample is collected into a capillary tube with heparin anticoagulant. The black plunger dispenses the sample into the sample well in the cassette.

Lancets



A single-use lancet is used to perform a fingerstick to collect a blood sample for testing on the Cholestech LDX.

PLEASE NOTE: Even though the Cholestech LDX is a "CLIA-waived" test system, each laboratory or testing site using the Cholestech LDX must have a CLIA Certificate of Waiver. To obtain a Certificate of Waiver, call your state department of health or Cholestech for an application. Failure to operate the Cholestech LDX in accordance with the manufacturer's instructions will void the waived status. The tests will then be considered highly complex.

Setup

Take the time to examine and familiarize yourself with the contents of the Cholestech LDX System.

1 Connecting the Cholestech LDX to the Printer

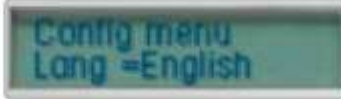


Plug the printer power supply **A** into the printer and the wall or power strip. Connect the end of the printer cable **B**, labeled "To Printer," to the printer.



Plug the power supply **A** into the Cholestech LDX and the wall or power strip. Insert the end of the white printer cable **B**, labeled "To Cholestech LDX," into the Analyzer.

2 Configuration Menu



Before starting to test, press and hold **STOP** to enter the Configuration Menu and select options for language, units, sample type, printer, risk assessment, date, and time.

The **DATA** button allows you to select choices for each of the menu items in the Configuration Menu. The **RUN** button enters your choices and advances you to the next menu item.

3 Optics Check

Run the Optics Check Cassette

Press the **RUN** button. The drawer will open. Place the Optics Check Cassette into the cassette drawer. Press the **RUN** button again to close the drawer.



Record Results

Check to see that the four numbers are within the acceptable range printed on the Optics Check Cassette. Record the results on the Optics Check Log. If the numbers are outside the range, the Analyzer will be temporarily disabled until another optics check is run that falls within range.



The Optics Check Cassette is used to check the optical system of the Analyzer. Run the Optics Check Cassette: once each day before patient samples are tested and after the Cholestech LDX has been moved or serviced.

4 Running Controls

Cholestech Level 1 and 2 Controls

Controls must be run following manufacturer's recommendations. Controls must be at room temperature before testing. Verify that the lot number on the control vial and the assay sheet are the same. Mix each vial by gently inverting 7-8 times immediately before use. Store controls in the refrigerator when not in use.



Testing QC Material

Use the MiniPet™ Pipette and tips to measure and dispense the control sample into the cassette. Place the cassette in the drawer *immediately* and press **RUN**. Record results on the Quality Control Log. Results must be within established ranges before patient samples can be tested.



Test Procedure

1 Perform a Fingerstick



After cleaning the selected site with alcohol and drying it thoroughly, firmly prick the site with a lancet. Gently squeeze the finger to obtain a large drop of blood. *Wipe off the first large drop of blood.*

2 Collect the Sample



Squeeze the finger gently again until a large drop of blood forms. Hold the capillary tube horizontally by the end with the plunger. Touch it to the drop of blood without touching the skin. Fill the capillary tube within 10 seconds. Do not allow any bubbles to enter the capillary tube.

3 Dispense the Sample



Place the end of the capillary tube into the sample well and dispense the sample by pushing down on the plunger. Keep the cassette level after the sample has been applied.

4 Open the Drawer



Press the **RUN** button on the Analyzer. The cassette drawer will open, and the screen will display: **Load Cassette and Press RUN.**

5 Insert the Cassette



Immediately place the cassette into the Analyzer drawer with the black reaction bar toward the Analyzer and the brown magnetic stripe on the right.

6 Begin the Test



Press **RUN**. The drawer will close. Test run time is 5 minutes. When the test is finished, the Analyzer will beep and the drawer will open. **Put everything that touched the blood sample into a biohazardous waste container.**

7 View the Results



Press the **DATA** button on the Analyzer to view the calculated results.

8 Print the Results



The results will print when the test is finished. If the Framingham Risk is **ON**, press the **DATA** button twice and push **RUN** to run the risk assessment, or **STOP** to print the results.

FAQ

Frequently asked questions about the use of the Cholestech LDX

How often do I have to run the controls?

If you are testing under CLIA-waived status, you must follow the manufacturer's recommendations for running controls. Cholestech recommends that you run controls:

- With each new shipment of cassettes (even if cassettes are from the same lot previously received).
- With each new lot of cassettes.
- As otherwise required by your laboratory's standard quality control procedures.
- If you are not running the Cholestech LDX under CLIA-waived status, or if your local or state regulations require more frequent testing of quality control material, then quality control must be performed in compliance with those regulations.

How should I store my cassettes?

When stored refrigerated at 36–46°F (2–8°C), the cassettes are stable until the expiration date printed on the cassette pouch and box. Cassettes can be stored at room temperature for 30 days. **Do not use a cassette past the expiration date.** Do not return cassettes to the refrigerator once they have been stored at room temperature.

Appendix G: Lipid Screening: Patient Assessment Form

- Patient Sex: Male or Female
- Patient's Age: _____
- Patient's BP: _____ / _____

1. Currently on medications: If yes, list

- a. Cholesterol: _____
- b. Diabetes _____
- c. Hypertension _____

2. Does patient have a history of coronary heart disease? Check all that apply.

- Told by health care provider they have heart disease
- Heart attack (myocardial infarction)
- Angina, stable or unstable (chest pain due to insufficient blood flow to heart)
- History of coronary procedures (stents, angioplasty, bypass surgery)
- Blocked arteries in the legs (peripheral artery disease)
- Blocked carotid artery
- Small strokes (transient ischemic attacks)

If any item in #2 is checked, the patient is considered to be HIGH RISK.

3. Does patient have any of the following risk factors? Circle all that apply.

- + 1. Current smoker
- + 1. High blood pressure (BP \geq 140/90 mmHg)
- + 1. Currently on medication for hypertension (see Question #1c)
- + 1. Female \geq 55 years old
- + 1. Male \geq 45 years old
- + 1. Female family history = parents or siblings with CHD before 65 y.o.
- + 1. Male family history = parents or siblings with CHD before 55 y.o.
- + 1. HDL <40 mg/dL
- 1. HDL >60 (a high HDL allows the subtraction of one risk factor)



= SUM of risk factors from Question #3

If 0 - 1 risk factor → patient is LOW RISK

If 2+ risk factors → use Framingham Table to determine risk category

LDL Goal Classification and Treatment Initiation

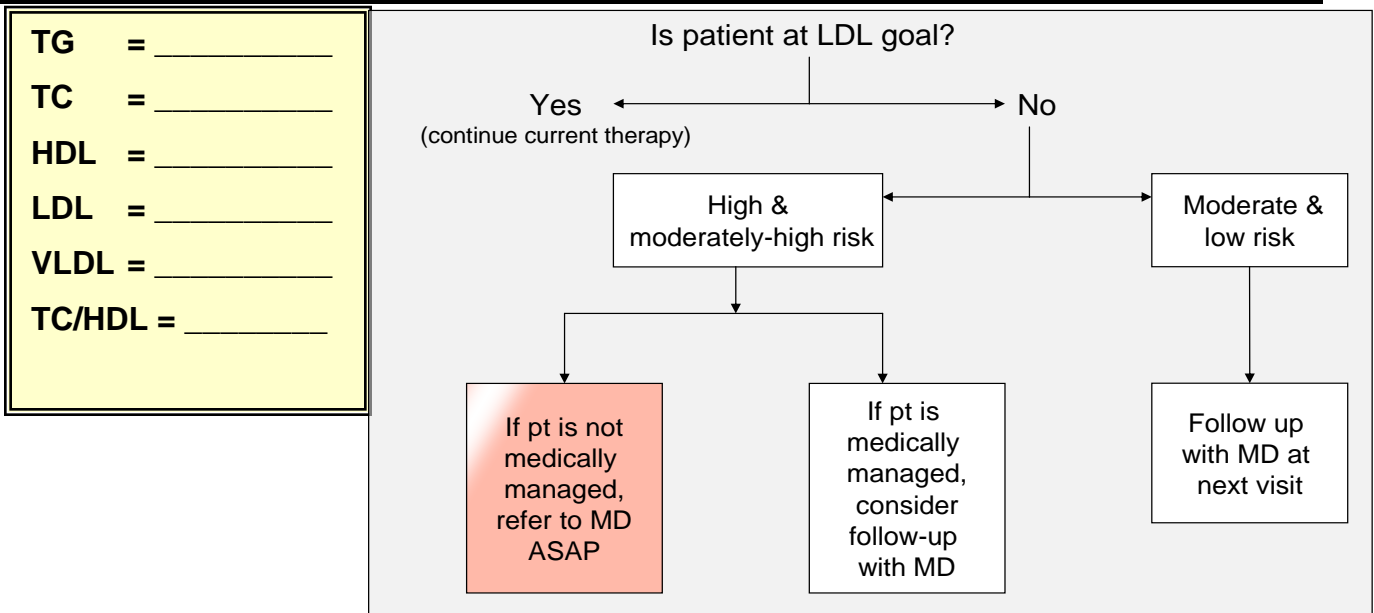
Check the patient's risk category and determine LDL goal

✓ Patient Risk	Risk Category	LDL Goal (mg/dL)	LDL Level (mg/dL) of when to Initiate TLC	LDL Level (mg/dL) of when to Consider Drug Therapy ^a
	High risk CHD or CHD risk equivalents (10-year risk >20%)	< 100 (< 70 optional)	≥ 100 ^b	≥ 130 (100-129: LDL-lowering drug optional)
	Moderately high risk 2+ risk factors with a 10-year risk 10-20%	< 130 (< 100 optional)	≥ 130 ^b	≥ 130 (100-129: LDL-lowering drug optional)
	Moderate risk 2+ risk factors with a 10-year risk <10%	< 130	≥ 130	≥ 160
	Lower risk 0 – 1 risk factor	< 160	≥ 160	≥ 190 (160-189: LDL-lowering drug optional)

a = Target LDL-lowering regimen should achieve 30-40% LDL reductions

b = High risk and moderately high risk patients should initiate TLC when lifestyle-related risk factors are also present (i.e., obesity, physical inactivity, elevated triglycerides, smoking)

Patient's Recommendations for Referrals



APPENDIX H: Cholestech LDX Lipid Profile

Cholesterol testing is recommended at least every 5 years for all adults over 20 years. The test you are taking today is designed to determine how much of each of the five 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 (the lower the better). LDL can be thought of as **L**ousy 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 **H**ighly **D**esirable or **H**ealthy 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. **VLDL** (very low density lipoprotein) cholesterol: 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. **TRG** (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.
6. **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, TRG, and HDL components are measured simultaneously from a single drop of blood (obtained by a finger stick) using rapid, accurate technology. A TC/HDL ratio and estimated values for LDL and VLDL cholesterol are calculated using the other values.

Current National Cholesterol Education Program ATPIII Guidelines:

TC :	<200	Desirable	HDL:	<40	Low
	200-239	Borderline high		>60	High
	>239	High			
					Males: > 40 mg/dL
					Females: > 50 mg/dL

TRG - Your goal if you fasted is less than **150mg/dL**.

TC/HDL ratio - 4.5 or less is considered optimal.

To Determine individualized LDL and Non-HDL goal:

Step 1: Determine presence of clinical ASVD or risk equivalent: CHD, CAD, PAD, AAA or DM (If present, LDL goal is 100mg/dL or with an optional goal of 70mg/dL)

Step 2: Determine presence of major risk factors that modify LDL goals.

Calculate the risk status by giving yourself “1” risk point for any true statement below:

___ HDL less than 40mg/dL.

___ Cigarette smoker

___ High blood pressure ($\geq 140/90$) or take medication for hypertension.

___ Age (female age 55 or older or male age 45 or older)

___ Family history of **premature** coronary heart disease (CHD), defined as:

CHD in a first degree female relative before age 65.

CHD in a first degree male relative before age 55.

*(If HDL is greater than 60mg/dL, then subtract 1 risk point)

Total risk points: _____

Step 3: If 2 or more risk factors are present (without the presence of CHD or risk equivalent), calculate Framingham score using the tables.

LDL and Non-HDL Goals:

0-1 risk factor: **LDL less than 160mg/dL (non-HDL goal <190mg/dL)**

2 or more risk factors AND 10-year risk score 20% or less: **LDL less than 130mg/dL (non-HDL goal 160mg/dL)**

Have CHD OR CHD risk equivalent (DM, PVD, symptomatic CAD, etc.) OR 10-year risk score of more than 20%: **LDL less than 100mg/dL (non-HDL goal < 130)**

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 TRG, the increase in TRG will affect the results obtained for LDL. **For TRG, 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.**

Appendix I: Post-test: Screening for Lipid Disorders in Older Persons

(Record responses on examination form)

- 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 phospholipids 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 a coronary heart disease (CHD) or a CHD-risk **equivalent**?
 - a) Unstable angina
 - b) Hypertension
 - c) Diabetes (type 1 or 2)
 - 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 TLC program?
 - a) Increase physical activity
 - b) Dietary cholesterol should be less than 200 mg per day
 - c) At least 20-30 g of fiber is recommended in the diet
 - 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?
- Look for a finger tip with minimal calluses
 - To increase blood flow, firmly massage the pricked finger starting from the base of the finger to the tip
 - Warm up cold hands by placing the hands under warm, running water for about 60 seconds
 - Inspect the fingertips by gently pressing them to see which ones have good blood return.
- 8) ABC is a 72 year old 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):

What would this patient's risk assessment be classified as?

- Low risk
- Moderate risk
- Moderately-high risk
- High risk

ABC's Results:

Blood pressure	126/82 mmHg
Triglycerides	104 mg/dL
Total chol.	167 mg/dL
HDL	45 mg/dL
LDL	112 mg/dL
VLDL	10 mg/dL
TC/HDL	3.7

- 9) What counseling would you give to ABC?
- She is below her LDL goal and should consider eating more dietary fats.
 - She is below her LDL goal and should continue her current lifestyle.
 - She is above her LDL goal and should consider adding drug therapy.
 - She is above her LDL goal and should increase her walking to 4 miles/day.
- 10) Patient DEF, is a 68 year old 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 risk factors for CHD. How many does he have?

- 2
- 3
- 4
- 5

DEF's Results:

Blood pressure	152/100 mmHg
Triglycerides	226 mg/dL
Total chol.	241 mg/dL
HDL	36 mg/dL
LDL	185 mg/dL
VLDL	20 mg/dL
TC/HDL	6.7

- 11) Since DEF has multiple risk factors, what is his 10-year risk assessment of developing CHD according to the Framingham risk assessment?
- 12%
 - 16%
 - 25%
 - ≥ 30%
- 12) Into which risk category should DEF be placed into to stratify his LDL goals?
- High risk
 - Moderately-high risk
 - Moderate risk
 - Low risk
- 13) Appropriate counseling for DEF would include:
- Inform patient he is at great risk for developing CHD in the next 10 years and should be seen by a health care provider as soon as possible for follow-up assessment.
 - 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.
 - Inform the patient that individuals who have high LDL cholesterol have a higher risk of developing CHD and his primary goal is to lower his LDL cholesterol.
 - All of the above should be included in patient counseling.

14) Patient HIJ, a 63 year old 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 currently smokes about 10 cigarettes a day and has failed multiple attempts to quit in the past. 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 CHD?
(Positive risk factor means having the risk factor increases the risk of developing CHD)

- Currently on medicine for blood pressure.
- Female ≥ 55 years old.
- HDL >60 mg/dL
- All are positive risk factors

HIJ's Results:

Blood pressure	126/82 mmHg
Triglycerides	158 mg/dL
Total chol.	248 mg/dL
HDL	62 mg/dL
LDL	176 mg/dL
VLDL	10 mg/dL
TC/HDL	4

- 15) Since HIJ has multiple risk factors, what is her calculated 10-year Framingham risk of developing CHD?
- 3%
 - 5%
 - 14%
 - 17%

- 16) Assuming HIJ is at moderate risk of developing CHD over the next 10 years, which of the following statements is the most appropriate action to be taken based on her LDL assessment?
- Nothing needs to be done; this patient is already at her LDL goal.
 - This patient should be seen by her primary care provider as soon as possible to initiate drug therapy.
 - 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.
 - This patient is doing just fine and should continue her current lifestyle.
- 17) Patient KLM, a 58 year old, obese 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’s risk classification would best be categorized as:
- High risk
 - Moderately-high risk
 - Moderate risk
 - Unable to determine (not enough information provided)
- 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?
- Turn the patient away; all lipid testing must be performed in fasting patients (9-12 hours).
 - Explain to the patient, fasting is preferred to get good results for HDL and LDL determination.
 - Explain to the patient, fasting is preferred to get good results for LDL and triglyceride determination, and therefore, only this patient’s HDL and total cholesterol may be calculated.
 - 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?
- KLM should be seen by his primary care provider as soon as possible to discuss lipid lowering.
 - KLM would benefit from initiating therapeutic lifestyle changes and may want to speak to a dietician since he has diabetes and potentially lipid abnormalities.
 - KLM should not worry about his lipids; they are not too bad.
- I only
 - II only
 - I & II
 - I, II, III
- 20) KLM is a strong candidate for metabolic syndrome.
- True
 - False

KLM’s Results:	
Blood pressure	150/100
Triglycerides	265 mg/dL
Total chol.	245 mg/dL
HDL	50 mg/dL
LDL	170 mg/dL
VLDL	25 mg/dL
TC/HDL	4.9

POST-TEST: Examination Form

Screening for Lipids in Older Persons

Participant Information

1. Name: _____

2. Mailing address: _____

3. Date exam completed _____

Questions: (Please circle one response per question)

1	A	B	C	D
2	A	B	C	D
3	A	B	C	D
4	A	B	C	D
5	A	B	C	D
6	A	B	C	D
7	A	B	C	D
8	A	B	C	D
9	A	B	C	D
10	A	B	C	D
11	A	B	C	D
12	A	B	C	D
13	A	B	C	D
14	A	B	C	D
15	A	B	C	D
16	A	B	C	D
17	A	B	C	D
18	A	B	C	D
19	A	B	C	D
20	A	B	C	D

For credit, please return this completed page to ⇨ ⇨ ⇨

Rachael Zins
MTGEC/IPHARM
 Skaggs Building Room 317
 University of Montana
 32 Campus Drive
 Missoula MT, 59812-1522
 Phone# (406) 243-2339 & Fax# (406) 243-4353

APPENDIX J: Evaluation for MTGEC Module

Screening for Lipid Disorders in Older Persons

1. Please indicate your profession: _____

	Please circle or underline appropriate number.	Strongly Agree	Agree	Neither Agree or Disagree	Disagree	Strongly Disagree	Don't Know
2	The overall visual presentation of the material enhanced my learning.	5	4	3	2	1	X
3	The module content was understandable.	5	4	3	2	1	X
4	The content was presented without bias.	5	4	3	2	1	X
5	The content will be useful for health-care professionals working with the elderly.	5	4	3	2	1	X
6	The objectives were clear.	5	4	3	2	1	X
7	This approach met my learning objectives.	5	4	3	2	1	X
8	The objectives of the module were achieved.	5	4	3	2	1	X
9	The module objectives related well to the overall purpose/goal of the web-based curriculum.	5	4	3	2	1	X
10	The assignments were appropriate.	5	4	3	2	1	X
11	The test questions were unambiguous.	5	4	3	2	1	X
12	The test questions were appropriate to the module content.	5	4	3	2	1	X
13	The teaching method was appropriate and used effectively.	5	4	3	2	1	X

