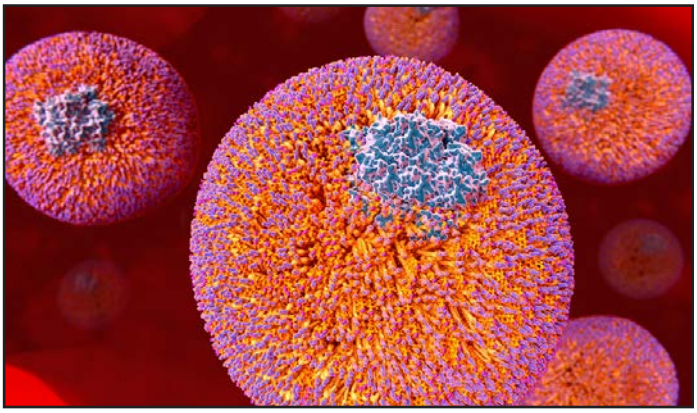
CE PRN

PHARMACY CONTINUING EDUCATION FROM WF PROFESSIONAL ASSOCIATES

"Update: Hyperlipidemia"

July 2016



So many patients are taking cholesterol medications that it's imperative to review this whole topic periodically. It reinforces one of the primary areas where we consult patients. The goals of this lesson are to: differentiate treatment options, and review newer medications. This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists & technicians in all practice settings. **The program ID # for this lesson is 707-000-16-007-H01-P for pharmacists & 707-000-16-007-H01-T for technicians.**

Participants completing this lesson by June 30, 2019 may receive full credit. Release date: July 1, 2016.

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call 1-847-945-8050. **Please write your name, NABP eProfile (CPE Monitor®) ID Number & birthdate (MM/DD) in the indicated space on the quiz page.**

The objectives of this lesson are such that upon completion participants will be able to:

Pharmacists:

1. Recognize the benefits of alirocumab & evolocumab.
2. Describe the function of lipids.
3. Comment upon effects associated with hyperlipidemia & cardiovascular diseases.
4. List risk factors associated with atherosclerotic vascular disease.
5. Describe MOA of statins & their adverse effects.
6. Discuss hyperlipidemic drugs other than statins.

Technicians:

1. Describe symptoms associated with hyperlipidemia.
2. List two of the newer antilipidemic drugs.
3. Comment upon MOA of statins.

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NOTE: ONE OF OUR FUTURE TOPICS THIS YEAR WILL BE ON THE OPIOID CRISIS OF ABUSE.

INTRODUCTION

Hyperlipidemia is a term used to describe the presence of an elevated blood level of lipids such as free fatty acids, triglycerides, sterols (cholesterol or cholesterol esters), or phospholipids (phosphoric acid esters of lipids). **Fatty acids**, which are usually derived from triglycerides or phospholipids, are either saturated or unsaturated and are an important source of energy. **Triglycerides** exist in nature as solids (fat) or liquids (oil). This depends on room temperature, length of the fatty acid chain, and extent of hydrogen ion saturation. **Sterols** occur naturally in plants and animals. Cholesterol is the best known animal sterol. Phytosterols are plant sterols.

Cholesterol is a waxy, fat-like sterol found in the bloodstream, and 75% of it is manufactured by the lining of the small intestines, adrenal glands, reproductive organs and other tissues. The remaining 25% is provided by the food we eat. Except for good cholesterol, high blood cholesterol and triglyceride levels are not healthy. However, cholesterol, especially at optimum blood levels, plays an essential role in the maintenance of cell membranes, formation of bile acids, steroid hormones, sex hormones such as progesterone and testosterone, cortisol and metabolism of fat soluble vitamins A, D, E and K. Additionally, it converts into Vitamin D when skin is exposed to sunlight. Finally, cholesterol and triglycerides serve as a good source of energy.

Hyperlipidemia is a silent, asymptomatic disorder that can be revealed only through blood tests. Over time it can increase the risk of the development of cardiovascular diseases including those that affect vessels supplying blood to the heart (coronary artery disease), brain (cerebrovascular disease), and limbs (peripheral vascular disease). These diseases are usually

triggered by the presence of atherosclerotic plaques within the arteries.

The liver of a healthy adult synthesizes about 1000 mg of cholesterol daily. The total cholesterol content of the body is approximately 35 grams. The average amount of cholesterol found in the dietary intake of an adult in the U.S. is from 200 mg to 300 mg per day. Once synthesized by the liver, cholesterol is transferred via the bile into the intestinal tract. About 50% of excreted cholesterol is reabsorbed by the digestive system and pumped back into the blood. This recycling cholesterol is a continuous process. Plant sterol, when included in the diet, tends to compete with cholesterol absorption resulting in reduction of cholesterol blood level. The higher the intake of cholesterol in the diet, the less production of cholesterol by the body, and vice versa. An elevated cholesterol in the blood, especially LDL, will allow cholesterol and other lipids to be deposited within the arterial walls of large and medium sized arteries as atherosclerotic plaque. This is usually composed of cholesterol, fats and calcium. Such plaques may cause obstruction of the arteries and may contribute to hypertension, reduction in the amount of oxygenated blood that reaches the heart and increased risk of coronary heart disease (CHD), myocardial infarction and cerebral atrial diseases.

Cholesterol is water insoluble, and as a result, it is immiscible with the aqueous blood plasma. Consequently, it does not travel in the free form in plasma. To make cholesterol mix with blood plasma and absorbable, the liver packages the cholesterol with protein and other compounds to form a carrier known as lipoprotein prior to its release into the bloodstream, thereby facilitating its transport to the cells. Lipoprotein is a package that consists of droplets of fat surrounded by a single layer of protein molecules (lipid on the inside and protein on the outside).

TYPES OF LIPOPROTEINS AND LIPIDS

There are four major types of lipoproteins and one triglyceride that are found in the bloodstream. They are:

1. **VLDL (Very Low Density Lipoprotein):** VLDL has the second highest triglyceride percentage of all lipoproteins. It consists of 50-60% triglycerides and 20-30% cholesterol, all of which are synthesized in the liver. VLDL is responsible for transporting triglycerides to the adipose and muscular tissues. What remains of VLDL is broken down to LDL.
2. **LDL (Low Density Cholesterol):** This lipoprotein is the remnant of VLDL metabolism and consists mainly of the cholesterol inner core. It is often referred to as "bad cholesterol." It consists of 51-58% cholesterol and 4-8% triglycerides. It makes up 60-75% of all plasma cholesterol. Its main function is to deliver cholesterol from liver cells to body tissues. If large quantities of LDL are carried and no new LDL receptors are found, then LDL absorption will be diminished and a harmful buildup of LDL occurs which may result in increased risk of CHD. A 25% reduction in plasma LDL level may reduce the incidence of CHD by 50%.
3. **HDL (High Density Lipoprotein):** HDL is the smallest and densest of all lipoproteins. This lipoprotein is referred to as "good cholesterol" because it lowers cholesterol by carrying it away from body tissues to the liver where it is broken down or excreted from the body as waste. Thus, accumulation of blood cholesterol and its deposition in the arteries is prevented. HDL constituents include 18-25% cholesterol and 2-7% triglycerides. It contributes approximately 20-30% of total cholesterol in the bloodstream. Unlike high LDL and VLDL blood levels, high HDL blood levels reduce the risk of the development of CHD.
4. **Chylomicrons:** Chylomicrons are lipoprotein particles that consist of 85-92% triglycerides,

6-12% phospholipids, 1-3% cholesterol and 1-2% protein. Their main function is to transfer dietary lipids and cholesterol from the intestinal lymphatics to the large veins and then to other locations in the body such as the liver, adipose, cardiac, and skeletomuscular tissues. The majority of chylomicrons are deactivated in the blood by the enzyme lipoprotein lipase within one day.

5. **Triglycerides:** Triglycerides play a role in healthy arteries and are considered a good measure for the heart's health. The triglycerides that are formed in the bloodstream consist of glycerol and three different fatty acids. They are the main constituents of human, animal and plant fats, the main source in diet, and the carbohydrates stored in the body. The body converts the unused extra calories to triglycerides and then stores them as fat until such a time when food intake is reduced or is not enough to provide energy. Excess intake of triglycerides will result in an increase in blood triglycerides (hypertriglyceridemia). Since triglycerides are immiscible with water, they circulate in the bloodstream in combination with protein.

LIPOPROTEIN BLOOD LEVELS

Total cholesterol blood levels

A total of less than 200 mg/dL is normal; 200 mg/dL is borderline high; and 240 mg/dL and greater is high. LDL cholesterol level is more accurate than total cholesterol in determining the risk for the development of cardiovascular diseases and the potential for narrowing of blood vessels. The higher the level of LDL the higher the risk for atherosclerotic disease. An LDL level of less than 70 mg/dL is best for patients with heart disease or diabetes. A level of 100 mg/dL and below is considered optimal for patients who are at risk of heart disease; 100-129 mg/dL is near optimal if there is no heart disease, but above optimal for patients with heart disease; 130-159 mg/dL is borderline in the absence of heart disease, but high if there is heart disease; 160-189 mg/dL is high if there is no heart disease, but very high if there is heart disease; and 190 mg/dL and greater is very high.

HDL blood levels

The higher the blood level of HDL the less risk of developing atherosclerotic vascular diseases. Levels below 40 mg/dL are poor; 40 mg-59 mg/dL are considered good and 60 mg/dL is best. The guidelines in Canada and Europe differ slightly from those in the U.S.

CAUSES AND RISK FACTORS OF HYPERLIPIDEMIA

Hyperlipidemia is due to genetic, lifestyle and environmental factors that include:

1. **Heterozygous** (a pair of genes where one is dominant and the other is recessive) **familial hypercholesterolemia** which is an inherited condition that causes high levels of LDL beginning at birth, and heart attacks at an early age (55 for brother and father and under 65 years of age for mother and sister).
2. **Presence of diseases that tend to increase LDL blood level.** These may be diabetes, hypertension, hypertriglyceridemia, kidney and liver diseases.
3. **Gender:** Men have a greater chance of developing hyperlipidemia than women.
4. **Age:** As a patient becomes older, the chances for developing atherosclerosis and hyperlipidemia increase.
5. **Diet:** Many foods such as eggs, butter, liver, kidney and certain seafood contain cholesterol

in amounts that will not drastically change cholesterol blood level. Other foods, especially if consumed in relatively large quantities and frequently can detrimentally affect cholesterol and triglyceride blood levels. Red meat, bacon, sausages, many cheeses, creamy cakes and hot dogs have high contents of saturated fats and may affect the outcome of cholesterol blood level.

6. **Sedentary lifestyle:** It has been shown that non-vigorous physical activity tends to lower LDL and elevate HDL blood levels.
7. **Bodyweight:** Individuals who maintain normal bodyweight are less likely to have abnormal LDL or HDL levels.
8. **Smoking:** It has been reported that smoking contributes to about 400,000 deaths annually in the U.S. It detrimentally affects the levels of LDL and HDL.
9. **Alcohol beverages:** Patients who regularly consume large quantities of alcoholic beverages exhibit high LDL and low HDL levels.

COMPLICATIONS OF HYPERLIPIDEMIA

Atherosclerosis

Atherosclerosis is the leading cause of cardiovascular disease. It is also known as hardening of the arteries and is a common disorder that occurs when fat, cholesterol and calcium are deposited in the arterial lining to form multiple plaques. A plaque normally consists of three components:

1. atheroma which is a fatty, soft, yellowish nodular mass located in the center of the plaque and consists of macrophages (cells that play a role in immunity).
2. a layer of cholesterol, and
3. 3. calcified outer layer.

Coronary Artery Disease (CAD)

Narrowing of the arteries that supply blood to the myocardium results in limiting blood flow and insufficient amounts of oxygen to meet the needs of the heart. The narrowing may progress to the extent that the heart muscle sustains damage due to lack of blood supply and nutrients.

Myocardial Infarction (MI)

MI is a condition that occurs when blood and oxygen supplies are partially or completely blocked from flowing in one or more cardiac arteries, resulting in damage or death of heart cells. The blockage is usually due to the formation of a clot in the artery. The condition is commonly known as heart attack. The occlusion may be due to ruptured atherosclerotic plaque. If the restricted flow of blood through the arteries and the resulting limited supply of oxygen are left untreated for a period of time, the blockage can cause damage or death of the myocardium cells.

Angina Pectoris

Currently termed angina, this condition is not a disease but a symptom of an underlying heart problem. It is usually characterized by chest pain. The pain may also be felt in the shoulders, arms, neck and back. Angina occurs as a result of a reduction or a lack of blood supply to a part of the entire heart muscle, as well as impairment of waste removal. Poor blood

circulation is usually due to CHD when partial or complete obstruction of the coronary arteries is present. Angina attacks may be due to a spasm of the arteries. Angina may be a symptom of coronary microvascular disease (MVD), a condition that affects the heart's smallest arteries.

Stroke or Cardiovascular Accident (CVA)

Such events occur when blood circulation in part of the brain is blocked or diminished. When the blood supply, which carries oxygen, glucose and other nutrients, is disrupted, brain cells die and become dysfunctional. Usually strokes occur due to blockage of an artery by a blood clot or a fragment of a broken atherosclerotic plaque that lodges in a small vessel within the brain.

Hyperlipidemia is a silent asymptomatic disorder that is revealed only through blood tests and often during routine testing. An estimated 785,000 Americans experience a new coronary artery disease (CAD) and about 470,000 will suffer from a recent attack every year. In 2007 one out of every six deaths in the United States was due to CAD, whereas stroke was responsible for one in every 18 deaths. It has been estimated that 33,600,000 Americans ten years of age and older have a total cholesterol blood level of 240 mg/dL or greater and many are not aware of it.

CONTROL OF HYPERLIPIDEMIA

Lifestyle Modification and Changes

Depending on the general health of the individual, lipoprotein blood levels and presence of risk factors, a trial of lifestyle modification may be attempted prior to using drugs. Individuals, especially those with high LDL, should initiate new day-to-day habits. Diet modification such as reducing saturated fat in the diet, increasing daily intake of fruits and vegetables (at least 5 servings), weight reduction to an acceptable level, cessation of smoking and exercising are recommended for improving or maintaining physical fitness and lowering of lipid blood levels. Lifestyle modifications and changes can result in 10-15% reduction in cholesterol blood levels. Patients who suffer from hyperlipidemia should avoid or reduce consuming cholesterol rich foods like corned beef, ribs, steak, ground meat, hot dogs, sausages, organ meats, egg yolks, most cheeses, cream, fast food burgers, fried foods, most seafood, mayonnaise, butter, bacon, coconut oil and red meat in general. Foods with low fat content are recommended over foods with full fat content.

Medications

Before using medications, treating other present conditions, such as diabetes, should be attempted. Drugs that lower cholesterol blood levels should be implemented if lifestyle changes have failed to significantly reduce blood cholesterol within 6-12 months. Even when medications are used, lifestyle changes must not be abandoned. There are a number of medications available to lower elevated LDL, total cholesterol and triglycerides, and a few that increase HDL blood level. The **statins** are the most common and successful medications used in treating hyperlipidemia since September 1987, when the FDA approved the use of lovastatin as a cholesterol lowering drug. However, two antihyperlipidemia drugs that belong to a new class called proprotein convertase subtilisin/kexin type 9 inhibitors (PCSK9) were been approved by the FDA in July 2015 and August 2015, respectively.

Statins (HMG-CoA Reductase Inhibitors)

According to the CDC, the percentage of adult Americans 40 years of age and over who were taking antihyperlipidemic medications between 2003 and 2012 rose from 20 to 28%. Furthermore, the intake of statins has increased from 18 to 26% and the numbers keep rising. Three years ago over 20,000,000 Americans were taking statins. They are popular due to their effectiveness and tolerance. They are useful for high risk patients such as those with CHD and diabetes.

Statins act by interfering with the biosynthesis of cholesterol in the liver. In addition, the statins have an antiatherosclerotic activity. They enhance the stability of atherosclerotic plaques. Clinical studies have shown that statins have significantly reduced the total cardiovascular mortality and morbidity; however, they have a number of limitations. At the standard dosage used, they are capable of reducing LDL levels by 30-40%. However, when such doses are used to reduce LDL levels by 40%, high risk patients failed to reach the desired level of 100 mg/dL. Increasing the dose of statins has its disadvantages. Doubling, tripling or quadrupling the dose will result in only 6%, 12% or 18% lowering of LDL, respectively. The higher the dose of statins, the greater the potential for toxicity, the most serious of which is myopathy. Some patients have low tolerance to high doses.

Adverse effects of statins include elevation of liver enzymes and muscle problems such as rhabdomyolysis (an acute but maybe fatal disorder characterized by destruction of skeletal muscle), myalgia, muscle cramps and diabetes at high doses. That being said, the incidence of statins adverse effects is low. Monitoring of liver enzymes has been abandoned by many practitioners following FDA recommendations. The intake of grapefruit or grapefruit juice may inhibit the metabolism of statins. In February 2012, the FDA required making changes to the safety information to the label of statins, especially that the intake of such medications may raise the blood level of sugar that leads to memory loss and confusion. However, the FDA indicated that such information should not prevent patients from taking statins due to their benefits. In 2012 the FDA informed health professionals that a drug interaction may occur when statins and protease inhibitors such as ritonavir, indinavir, nelfinavir, etc. are taken concurrently. Such interactions may lead to elevation of statin blood levels and an increase in the risk of myopathy.

In September 1987 the FDA approved the use of lovastatin as the first cholesterol lowering statin. Since then, six statins, two of which are semi-synthetic (simvastatin and pravastatin) and four are synthetic (fluvastatin, atorvastatin, rosuvastatin, and pitavastatin) have entered the market. Prior to 1987, nicotinic acid and cholestyramine were the primary antihyperlipidemic medications. Not only were the therapeutic effects of these compounds minimal to moderate, as in the case of cholestyramine, they caused adverse effects that many patients could not tolerate. Thus the discovery of the statins has opened a new era in the treatment of hyperlipidemia. In late 2015, the FDA approved the use of a new class of cholesterol lowering drugs called PCSK9 (Protein Convertase Subtilisin/Kexin type 9) inhibitors.

The statins in use

1. **Simvastatin:** The use of simvastatin resulted in a reduction of 36% of the LDL blood level. In 2011 the FDA announced safety label changes for simvastatin which include limiting the use of the highest approved dose of 80 mg due to increased risk of myopathy, particularly during the first 20 months. The most frequently encountered adverse effects include abdominal

distress, constipation, flatulence, nausea, heartburn and headache. Myalgia and/or muscle weakness are rare. The initial adult dose is 20 mg daily, and this may be increased at intervals of no less than four weeks until a maximum dosage of 80 mg is reached. Simvastatin was approved by the FDA in 1991.

2. **Pravastatin:** It is capable of reducing LDL and triglyceride blood levels and at the same time raises blood levels of HDL. The usual maintenance dose is 10-40 mg daily. Side effects of pravastatin include diarrhea, abdominal cramps, flatulence and headache. Pravastatin was approved by the FDA in 1996.
3. **Fluvastatin:** It is available in 20 and 40 mg capsules, and extended release 80 mg tablets. At the recommended dosage, fluvastatin possesses a low incidence of side effects. They include abdominal discomfort, nausea, flatulence, back pain and rash. The drug was approved by the FDA in 1993.
4. **Atorvastatin:** It is used to reduce LDL and triglyceride blood levels. It is usually taken once daily with or without food. Adult dosages should be adjusted within 2-4 weeks after the initial dose of 10 mg daily. The maintenance adult dose is between 10-80 mg daily. Atorvastatin was approved by the FDA in 1996.
5. **Rosuvastatin:** As with all statins, there is a common concern for the development of rhabdomyolysis. However, the FDA indicated that the risk of this disorder is greater with rosuvastatin than with other statins. The FDA indicated that the risk of myopathy during rosuvastatin therapy may increase in Asian Americans. Physicians should start Asian patients at a lower dose level of 5 mg once daily and a maximum daily dose of 40 mg. Rosuvastatin was approved by the FDA in 2003.
6. **Pitavastatin:** It is capable of reducing total cholesterol, LDL and triglycerides while increasing HDL levels. It was approved by the FDA in 2009.
7. **Lovastatin:** Lovastatin was the first statin to be approved by the FDA in 1987. It occurs naturally and is found in foods such as oysters and mushrooms. As with other statins, the dosage varies from one patient to another and should be determined in accordance with the requirement and response of the patient. The usual maintenance dose is 10-80 mg daily.

NEW HYPERLIPIDEMIA LOWERING DRUGS

In late 2015 the FDA approved the use of a new class of hyperlipidemic lowering drugs known as PCSK9 inhibitors.

PCSK9 (Proprotein Convertase Subtilisin/Kexin Type 9) Inhibitors

PCSK9 is a specific protein that reduces the number of receptors on the liver that remove LDL from circulation. Inhibition of PCSK9 results in availing more receptors on the liver that eliminate LDL from the blood. **Alirocumab and evolocumab** are two PCSK9 inhibitors that are available. Both are human monoclonal antibodies (IgG2) that bind to PCSK9, thereby inhibiting its action. These drugs are indicated in the treatment of adult patients with high LDL caused by inherited heterozygous familial hyperlipidemia, a hyperlipidemia with a history of atherosclerotic cardiovascular disease in which there is a need for further lowering of LDL. It is also recommended for patients whose LDL blood levels remain high in spite of lifestyle modifications and treatment with statins alone. Studies have shown that the majority of patients who failed to respond to standard hyperlipidemic lowering drugs alone have achieved their goals with 75 mg of a subcutaneous dose of alirocumab in combination with diet and a maximally tolerated

dose of a statin. It is estimated that 8 - 10 million Americans suffer from inherited heterozygous familial hyperlipidemia and atherosclerotic cardiovascular disease. The PCSK9 inhibitors are administered by injection. Alirocumab is available in 75 mg and 150 mg doses in a single, one ml injection delivered in a pre-filled pen or syringe, that may be administered every two weeks. The dose of evolocumab is 140 mg/ml in a subcutaneous dose every two weeks. It is available as one single-use prefilled syringe, or single-use prefilled autoinjector. It is administered along with diet and maximally tolerated statin doses. Side effects include redness, swelling, itching and tenderness at the injection site, confusion, abnormalities in liver enzyme levels and allergic reactions, some of which may require hospitalization.

OTHER MEDICATIONS

Ezetimibe: This drug was approved by the FDA in 2002 for patients with low risk of CHD and inability to tolerate statins. Its mechanism of action is different than that of statins. It is considered a selective cholesterol absorption inhibitor. It acts by blocking the absorption of dietary and biliary cholesterol. In 2008, a panel of experts recommended that it should be used as a last resort. The recommended dose is 10 mg daily. Side effects include GI disturbances, headache, fatigue and rash.

Bile Acid Sequestrants: The bile acid binding resins (cholestyramine, colestipol and colesevelam), combined with bile acids present in the intestine form an insoluble complex. Because the complex is not absorbed from the GI tract, they do not possess systemic adverse effects that can cause GI disturbances.

Niacin: Niacin is capable of reducing LDL (15 - 25%), VLDL (25 - 35%), as well as triglycerides, and at the same time results in elevation of HDL (15 - 25%). The mechanism of action is not fully understood. At an effective dose, niacin can cause side effects such as flushing of skin, skin rash, hepatotoxicity and others that many patients cannot tolerate. The dose is 1-2 g three times daily.

Fibric Acid Derivatives: Gemfibrozil and fenofibrate are minimally effective. Adverse effects include rash and GI disturbances.

Plant Sterols: Sterols are capable of reducing LDL by about 10%. Their mechanism of action involves blocking cholesterol absorption from the intestine.

SUMMARY

Hyperlipidemia, a disorder characterized by increased blood levels of lipids and/or lipoproteins, plays an important role in causing coronary artery disease, cerebrovascular disease and peripheral vascular disease. Such diseases are triggered by the presence of atherosclerotic plaques within the arteries. Cholesterol is a soft, waxy steroid that is found in the blood as a result of synthesis in the liver and ingested foods. Cholesterol is required by the body to maintain crucial functions such as the formation of bile salts, steroid hormones, sex hormones and metabolism of fat soluble vitamins A, D, E and K. In 1987 the FDA approved the use of lovastatin, an agent for lowering lipid blood levels. The drug, which belongs to the statin class, opened the door for additional medications within this group. In 2015 a new class of antihyperlipidemic drugs that act as PCSK9 inhibitors was approved. Currently, there are two drugs in this class: alirocumab and evlocumab.

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UPCOMING TOPICS

- New – Approved Drugs
- Validation of Pain Medication Rxs
- Pharmacogenetics
- Hyperlipidemia
- Pharmacy Considerations Regarding the Opioid Crisis of Abuse
- Vaccines—Truths, Myths, Hesitancy, Controversies
- Update C. *diff*—do probiotics and/or yogurt help?

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LESSON EVALUATION

Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?
   - Recognize benefits of alirocumab & evolocumab — YES NO
   - Describe the function of lipids — YES NO
   - Comment upon effects of hyperlipidemia & CVD — YES NO
   - List risk factors associated with atherosclerosis — YES NO
   - Describe MOA of statins — YES NO
2. Was the program independent & non-commercial? — YES NO

	Low Relevance						Very Relevant
3. Relevance of topic	1	2	3	4	5	6	7

4. What did you like most about this lesson?_____
5. What did you like least about this lesson?_____

Please Mark the Correct Answer(s)

1. **Synthesis of cholesterol in the body takes place mainly in the:**
   A. Pancreas B. Liver
   C. Spleen D. Stomach
2. **Which of these lipoproteins is known as "bad cholesterol?"**
   A. HDL B. VLDL
   C. Chylomicron D. LDL
3. **Which statement is TRUE about atherosclerosis?**
   A. Enhances blood flow through arteries
   B. Occurs when fat, cholesterol & calcium deposit in the arterial lining to form plaques
   C. Occurs only in families
   D. The main cause of hyperlipidemia
4. **Which of these is NOT a risk factor for hyperlipidemia?**
   A. Consumption of fiber-rich diet
   B. Family history
   C. Obesity
   D. Smoking
5. **Statins achieve their action via:**
   A. Interaction with antibodies in the blood
   B. Formation of an unabsorbable complex with lipoprotein
   C. Inhibition of the enzyme HMG-CoA reductase
   D. Stimulation of bile secretion
6. **Patients taking large doses of statins for a prolonged time may risk development of:**
   A. Rhabdomyolysis
   B. Peptic ulcer
   C. Osteoporosis
   D. Atherosclerosis
7. **What is the function of PCSK9?**
   A. Suppression of IgE
   B. Potentiation of the production of triglycerides
   C. Breakdown of insulin
   D. Reduction of number of receptors on the liver that remove LDL
8. **PCSK9 inhibitors are indicated in the treatment of the following:**
   A. High HDL blood level
   B. Hypertriglyceridemia
   C. Heterozygous familial hyperlipidemia
   D. Breakdown of atherosclerotic plaques
9. **Which of these act by inhibiting cholesterol absorption?**
   A. Niacin B. Pitavastatin
   C. Ezetimibe D. Lovastatin
10. **The average amount of cholesterol found in the dietary intake of an adult in the U.S. is:**
    A. 100 mg B. 600 mg
    C. 75 mg D. From 200 – 300 mg

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