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v. 3

High Cholesterol and Dietetics, Part 1

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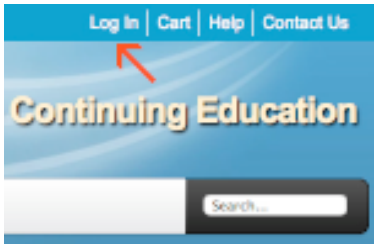

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High Cholesterol and Dietetics

A Three Part Series

Outlines of All Three Courses

High Cholesterol and Dietetics, Part One

This course covers the diagnosis of treatment of hyperlipidemia (high cholesterol and triglycerides) according to western biomedicine and presents the Chinese medicine theoretical foundations concerning hyperlipidemia including diagnostics and treatment principles. This course is approved for 6 acupuncturist continuing education credit hours.

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Obstruction of Spleen Transportation is a Key Component in Dyslipidemia
Spleen Deficiency and Dyslipidemia in Modern Research
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2.1.3 Kidney and Dyslipidemia
Kidney deficiency is a primary factor in the development of dyslipidemia
Kidney Yang Deficiency, Water Congealing and Becoming Phlegm
Kidney Yin Deficiency, Deficient Fire Scorching the Fluids
Kidney Essence Deficiency and Stagnation of Fat
Kidney Tonifying Medicinals and Dyslipidemia
Conclusion

Heart Section

High Cholesterol and Dietetics, Part Two

This course takes a detailed look at Chinese medicine dietetics and its relationship to the treatment of hyperlipidemia. Participants learn lifestyle changes important to reducing cholesterol and triglyceride levels according to Chinese Medicine principles. In addition, participants learn the Chinese medicinal properties of individual foods and their effects upon blood lipid levels. Also, participants learn how to apply Chinese Medicine lipid reducing food formulas to reduce cholesterol and triglyceride levels. This course is approved for 6 acupuncturist continuing education credit hours.

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3.2.1 Individual Foods
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2. Vegetables, Grains, Tofu

3.2.2 Lipid-Reducing Food Formulas

1. Teas
2. Conjee
3. Soups

High Cholesterol and Dietetics, Part Three

This course details the medicinal properties of Chinese herbs and their relationship to lowering serum lipid levels. Modern research is presented in addition to Chinese medicine theoretical principles. Individual herbs and herbal formulas are presented to make the clinical application of this material helpful to a licensed acupuncturist for the treatment of hyperlipidemia. Learn how to lower cholesterol and triglyceride levels with Chinese medicinal herbs. This course is approved for 13 acupuncturist continuing education credit hours.

3.3 Herbal Therapy

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High Cholesterol and Dietetics, Pt. 1

Introduction

Dyslipidemia affects millions of people worldwide, particularly in areas where fat-rich diets and sedentary lifestyles are prevalent, such as the United States, and is cause for concern due to the increased risk it poses for serious diseases such as atherosclerosis and coronary heart disease (CHD). While pharmaceutical drug-therapies to normalize serum lipid levels may be necessary in some cases, Chinese medicine offers preventative and remedial therapy options which are both safe and effective. This course aims to provide Chinese medicine practitioners with the fundamental knowledge needed to understand dyslipidemia from a biomedical perspective, and apply Chinese medicine herbal, dietary and lifestyle practices in the management of dyslipidemia patients.

1. Dyslipidemia in Western Medicine

1.1 Definition

Dyslipidemia is defined as disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. Dyslipidemia may include elevated plasma concentrations of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and triglyceride, and a decrease in high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein plasma concentrations. *Hyperlipidemia* refers to higher than normal levels of plasma lipids, including cholesterol, cholesterol esters, phospholipids, and triglycerides. All of these lipids are transported in the plasma bound to proteins, and these larger compound molecules are called *lipoproteins*. Lipoproteins have been classified on the basis of their densities into five major classes:

- chylomicrons
- very low-density lipoproteins (VLDL)
- intermediate-density lipoproteins (IDL)
- low-density lipoproteins (LDL)
- high-density lipoproteins (HDL)

When hyperlipidemia is defined in terms of elevated serum lipoproteins, the term *hyperlipoproteinemia* is used. Higher than normal serum cholesterol is termed *hypercholesterolemia*, and higher than normal serum triglyceride is termed *hypertriglyceridemia*. The main risk associated with dyslipidemia is atherosclerosis and subsequent increased risk for coronary heart disease (CHD) and other vascular diseases.

1.2 Prevalence

In recent years there are many studies showing direct correlation between incidence of coronary heart disease (CHD) and elevated total and LDL cholesterol levels. According to the third report of the Adult Treatment Panel (ATP III), issued by the National Cholesterol Education Program (NCEP) in May 2001, each year approximately 1.5 million Americans experience an acute myocardial infarction (MI), and one-third of them do not survive. Adoption of NCEP guidelines for the management of dyslipidemia means many more patients are now candidates for intensive lipid-lowering therapy. The NCEP estimated that under its 2001 guidelines, the number of Americans qualifying for dietary treatment would rise from 52 million to 65 million, and the number of candidates for drug therapy would nearly triple—from 13 million to 36 million.

1.3 Pathophysiology

The main risk of dyslipidemia is atherosclerosis (the leading cause of death and disability in the developed world) and the ensuing risk atherosclerosis poses for coronary heart disease (CHD), myocardial infarction (MI), cerebrovasular accident (CVA), peripheral arterial disease, carotid artery disease, abdominal aortic aneurysm (AAA), etc. Atherosclerosis is a subset of arteriosclerosis (hardening of the arteries), and is the formation of *atheromas* (fibrous fatty intimal plaques) in arterial walls. The exact pathogenesis of atherosclerosis is controversial and complex, but a simplified explanation is as follows:

Atherosclerosis begins when monocytes congregate on arterial walls in response to lipoprotein oxidation, creating a fatty streak (yellow streak of lipid-filled macrophage foam cells) on the arterial wall. The monocytes leave the bloodstream and enter the arterial intima where they become macrophages. The macrophages then phagocytize the oxidized LDL cholesterol and die, thus contributing to the lipid component of the fatty streak. Fatty streaks represent the initial lesion of atherosclerosis, are asymptomatic, and are present in the aorta and coronary arteries of most individuals by age 20.

As the fatty streak evolves into a more complicated atherosclerotic lesion, smooth-muscle cells accumulate within the expanding intima and the amount of extracellular matrix increases. Thus begins the formation of the fibrous cap, which separates a necrotic core of cellular debris, degenerating foam cells, and cholesterol crystals from the arterial lumen. The plaque eventually calcifies, thus stiffening and increasing the fragility of the blood vessel.

The endothelium plays a major role in the pathophysiology of atherosclerosis. Endothelial cells normally provide a permeability barrier, reduce clotting, and regulate vascular tone. In atherosclerosis, the intimal endothelium becomes dysfunctional, loses its ability to produce nitrous oxide (NO), and expresses selectins/integrins for leukocyte recruitment. NO is a vasoprotective gas released by the endothelium which has vasodilatory, anti-thrombotic, and anti-inflammatory effects. NO activates guanylate cyclase to generate cGMP, which causes smooth muscle dilation. NO also blocks

vascular inflammation by inhibiting endothelial release of inflammatory granules, and blocks platelet aggregation. Endothelial cells lose their ability to produce NO due to the presence of inflammation, toxins, atherosclerosis, or oxidized LDL.

Atherosclerosis is an inflammatory disease, which is why levels of C Reactive Protein closely correlate with and predict MI. LDL cholesterol, cigarettes and other toxins initiate vascular inflammation, damage the endothelium, and activate macrophages.

Atherosclerosis leads to vascular occlusion when the plaque ruptures or ulcerates, exposing thrombogenic material and leading to the formation of a blood clot, or thrombus. The thrombus may block the artery, causing MI or CVA, or it may become incorporated into the plaque, enlarging its size. Atherosclerotic dysfunction of the endothelium prevents NO production, inhibiting vasodilation and further aggravating the occlusion. Additionally, embolization may occur if pieces of the plaque, called atheroma, break off and become lodged in distal sites.

In the heart, increased plaque volume causes arterial remodeling, which results in an outward expansion of the coronary arteries. The arteries expand in an effort to overcome the effects of the blockage, allowing blood to flow through the stenosed arterial segment. This expansion continues until the artery reaches its maximum point of flexibility and can no longer accommodate the continued growth of the plaque. This threshold generally occurs when the arterial stenosis reaches 40%. As the plaque ages, an increasing amount of fibrous tissue accumulates, leading to the formation of a fibrous cap, which is vulnerable to rupture. Progressive arterial stenoses eventually lead to ischemic vascular disease, and the rupture of a plaque can cause a myocardial infarction.

1.4 Signs and Symptoms

The primary clinical manifestations of dyslipidemia are ischemic vascular disease, pancreatitis, and xanthomatosis. The major ischemic vascular diseases are atherosclerosis, coronary heart disease, peripheral vascular disease, and cerebrovascular disease. Pancreatitis may be associated with hypertriglyceridemia. Xanthomas are tumor-like collections of lipids (triglycerides and cholesteryl esters) that can arise in the tendons, points of continued trauma, legs, knees and elbows, as well as palms.

1.5 Description of Lipoproteins

Lipoprotein Structure
Lipoproteins are spherical particles made up of hundreds of lipid and protein molecules, yet they are smaller than red blood cells. The major lipids of the lipoproteins are cholesterol, triglycerides, and phospholipids. Triglycerides and cholesterol esters are nonpolar, hydrophobic lipids that make up the core of the lipoproteins. Phosphoplipids and a small amount of unesterified cholesterol cover the surface of the lipoproteins, where they act as the interface between the plasma and core components. A family of

proteins, known as apolipoproteins, also occupies the surface of the lipoproteins and plays an important role in the regulation of lipid transport and lipoprotein metabolism.

LDL Cholesterol (LDL-C)
LDL cholesterol typically accounts for 60-70% of total serum cholesterol. LDL is the main atherogenic lipoprotein and is the primary target of cholesterol-lowering therapy. The focus on LDL reduction has been strongly validated in recent years by clinical trials that show the efficacy of LDL-lowering therapy in reducing CHD risk. However, recent studies have also shown the significance of LDL particle size in relation to atherosclerosis and heart disease. Smaller, denser LDL carries more atherogenic potential than larger, less dense LDL particles.

HDL Cholesterol (HDL-C)
HDL cholesterol typically accounts for 20-30% of total serum cholesterol. HDL cholesterol levels are inversely correlated to CHD risk. However, recent studies[i] have shown that very high levels of HDL (≥70 mg/dL), and very large HDL particle size, are both associated with increase CHD risk. Furthermore, while some evidence suggests that HDL protects against development of atherosclerosis, a low HDL level often reflects the presence of other atherogenic factors.

VLDL Cholesterol (VLDL-C)
VLDL are triglyceride-rich lipoproteins which contain between 10-15% of total serum cholesterol. VLDL are produced by the liver and are precursors of LDL. Some forms of VLDL appear to be atherogenic, particularly VLDL remnants, which consist of partially degraded VLDL and are relatively enriched in cholesterol ester. Technically, IDL belong to these remnant lipoproteins, but IDL is included in the measurement of serum LDL.

Chylomicrons
Chylomicrons are triglyceride-rich proteins formed in the intestine from dietary fat, and which appear in the blood after a fat-containing meal. Partially degraded chylomicrons, known as chylomicron remnants, may also be atherogenic.

Characteristics of the Major Lipoprotein Classes

Lipoprotein	Density (g/dL)	Diameter (nm)	Lipid (%) *		
			Triglyceride	Cholesterol	Phospholipid
Chylomicrons	0.95	75-1200	80-95	2-7	3-9
VLDL	0.95-1.006	30-80	55-80	5-15	10-20
IDL	1.006-1.019	25-35	20-50	20-40	15-25
LDL	1.019-1.063	18-25	5-15	40-50	20-25
HDL	1.063-1.210	5-12	5-10	15-25	20-30

* The remaining percent composition is made up of the apoproteins.

Characteristics of Lipoproteins:

Lipoprotein	Normal % of Total Cholesterol	Desirable Plasma Concentration	Atherogenic Potential
Total Cholesterol	-	< 200 mg/dL	Yes
LDL	60-70%	< 100 mg/dL	High
HDL	20-30%	> 40 mg/dL < 70 mg/dL	Yes, at high levels. Moderate levels may protect against atherogenesis
VLDL	10-15%	10-31 mg/dL	Likely

1.6 Serum Lipid Levels

ATP III classified levels of serum lipids as described below. However, the actual desirable level (goal) for each individual varies depending on risk factors for CHD, and is calculated using ATP III guidelines and the Framingham Point Score tables. Therefore, the following levels are merely a rough guideline and not applicable to all individuals.

Total Cholesterol
Elevated total cholesterol is associated with increased risk of CHD. A total cholesterol level under 200 mg/dL is desirable, while a level of 200 to 239 mg/dL is borderline high, and greater than or equal to 240 mg/dL is high. However, most decisions about treatment are based upon levels of LDL or HDL cholesterol rather than total cholesterol.

LDL Cholesterol
Low-density lipoprotein (LDL) cholesterol is a more accurate predictor of CHD risk than total cholesterol, and higher LDL cholesterol concentrations are associated with increased incidence of CHD in a large number of studies.

LDL concentrations as low as 25 to 60mg/dL are physiologically sufficient. Baseline LDL levels under 100mg/dL are considered acceptable and not requiring of further LDL lowering therapies. However, individuals in this category should still follow dietary and lifestyle recommendations (TLC- Therapeutic Lifestyle Changes) that will allow for maintenance of low LDL levels.

According to ATPIII, baseline LDL cholesterol levels between 100-129 mg/dL require therapeutic intervention. Several options are available, but as in all cases of elevated cholesterol, TLC is the initial therapy. If healthy LDL levels are attained on dietary therapy alone, the use of lipid-lowering drugs may be unnecessary. If LDL remains above 100 mg/dL after 3 months of maximum dietary therapy, an LDL-lowering drug may be considered. If elevated triglyceride levels or low HDL levels are also present, another lipid-lowering drug may be considered, such as nicotinic acid or fibric acid.

For baseline LDL cholesterol levels ≥130 mg/dL, use of an LDL-lowering drug is generally considered necessary to achieve LDL cholesterol levels <100 mg/dL.

Increased LDL levels may result from the following:

1. Primary hyperlipoproteinemia
2. High fat diet
3. Acute myocardial infarction
4. Obstructive liver disease (primary biliary cirrhosis)
5. Hypothyroidism
6. Nephrotic syndrome
7. Diabetes mellitus
8. Anabolic steroid use
9. Medications
    1. Progestins
    2. Thiazide diuretics

Decreased LDL cholesterol levels may result from any of the following:

1. Abetalipoproteinemia
2. Advanced liver disease
3. Malnutrition

HDL cholesterol

It is generally accepted that elevated serum levels of HDL and larger HDL particle size lower the risk of CHD, and according to NCEP guidelines, an HDL level greater than or equal to 60 mg/dL is a negative risk factor for CHD. It is also generally accepted that low levels of HDL (<40 mg/dL) may require treatment, particularly in cases with established CHD. New research, however, shows that very high levels of HDL (≥70 mg/dL), and very large HDL particle size, are both associated with increase CHD risk. This gives rise to the hypothesis that very large HDL, which is cholesterol enriched, may at some point become a cholesterol donor instead of an acceptor. Furthermore, although it has widely been acknowledged that the anti-inflammatory capacity of HDL contributes to its antiatherogenic potency, several studies have demonstrated that HDL can also turn into a proinflammatory particle. Therefore, it is not simply a matter of higher HDL levels being better. Rather, both HDL particle size and serum levels must be considered.

Positive cardiac risk factors (atherogenic):

1. HDL < 35 mg/dL or ≥70 mg/dL
2. Total Cholesterol (TC) to HDL ratio
    1. Men > 5.0
    2. Women > 4.5

Negative cardiac risk factors according to NCEP (protective):

1. HDL > 60 mg/dL

The following may lead to increased serum HDL levels:

1. Medications
    1. Gemfibrozil
    2. Niacin
    3. Exogenous estrogens

2. Moderate alcohol intake (1 ounce per day)
3. Regular aerobic exercise
4. Weight loss (for obese patients)
    1. HDL increases 2 mg/dl for each 4.5 kg of weight loss

The following may lead to decreased serum HDL levels:

1. Tobacco abuse
2. Diabetes Mellitus
3. Hypertriglyceridemia
4. Menopause
5. Obesity
6. Puberty in males
7. Uremia
8. Anabolic Steroids
9. Apolipoprotein deficiency
10. Liver disease
11. Tangier disease
12. Medications
    1. Progestins
    2. Probucol

Triglycerides

Elevated triglycerides are also associated with an increased risk of CHD. Although LDL levels are the primary focus of therapy, ATP III also recommends aggressive treatment of elevated triglyceride levels through weight management and increased physical activity. Patients whose triglyceride levels remain at 200 mg/dL or higher after they have reached their LDL goal are recommended to achieve a secondary goal based on their "non-HDL" level (total cholesterol level minus HDL level). The non-HDL goal should be 30 mg/dL higher than the LDL goal. The basis for attempting to reach this secondary goal is that all non-HDL particles are potentially atherogenic. This can be achieved through activities of daily living or the use of niacin and fibrates.

ATP III classifies triglyceride concentrations as follows:

- Normal: less than 150 mg/dL
- Borderline high: 150 to 199 mg/dL
- High: 200 to 499 mg/dL
- Very high: greater than 500 mg/dL

The following may lead to increased serum triglyceride levels:

1. Hyperlipoproteinemia (types I, IIb, III, IV, and V)
2. Pregnancy
3. Obesity
4. Alcohol abuse
5. Acute myocardial infarction
6. Pancreatitis
7. Nephrotic syndrome

8. Chronic renal insufficiency
9. Glycogen storage disease
10. Acute intermittent porphyria
11. Endocrine disease
    1. Diabetes mellitus
    2. Hypothyroidism
    3. Cushing's syndrome
    4. Hypopituitarism
12. Medications
    1. Exogenous estrogens
    2. Diuretics
    3. Glucocorticoids
    4. Ticlopidine

The following may lead to decreased serum triglyceride levels:

1. Malnutrition
2. Abetalipoproteinemia
3. Medications
    1. Gemfibrozil
    2. Nicotinic Acid
    3. Clofibrate

VLDL Cholesterol

Serum VLDL levels should ideally be below 30 mg/dL. The following may lead to increased serum VLDL levels:

1. Idiopathic increased hepatic secretion of VLDL
2. Diabetes mellitus
3. Obesity

ATPIII Serum Lipid Level Classification* (mg/dL)*:

Desirable/Optimal	Near/Above Optimal	Borderline High	High	Very High
Total Cholesterol				
< 200	-	200-239	≥ 240	-
LDL Cholesterol				
< 100	100-129	130-159	160-189	≥ 190
HDL Cholesterol				
> 40			≥ 60	
VLDL Cholesterol				
< 30	-	-	-	-
Triglycerides				
< 150	-	150-199	200-499	≥ 500

1.7 Cholesterol Ratios

Cholesterol ratios are ratios between serum levels of different types of cholesterols, the most common of which are total cholesterol to high density lipoprotein ratio (total/HDL ratio), low density lipoprotein to high density lipoprotein ratio (LDL/HDL ratio), and high density lipoprotein to low density lipoprotein ratio (HDL/LDL ratio). These are employed to help determine risk for atherosclerosis and cardiovascular disease. While the medical community is divided over the effectiveness of using these ratios for this purpose, a brief review is necessary.

Total/HDL ratio is determined by dividing the total cholesterol (TC) level by the HDL level (TC÷HDL, or TC/HDL). A ratio below 5/1 is generally considered acceptable, with 3.5/1 or below considered ideal.

LDL/HDL ratio is determined by dividing the LDL level by the HDL level (LDL÷HDL, or LDL/HDL). A ratio below 3.5/1 is generally considered acceptable, with 2.5/1 or below considered ideal.

HDL/LDL ratio is determined by dividing the HL level by the LDL level (HDL÷LDL, or HDL/LDL). A ratio above 0.3/1 is generally considered acceptable, with 0.4/1 or above considered ideal.

While these ratios may be useful in some ways, the medical community is divided on whether they are better than total cholesterol or LDL cholesterol levels in predicting CAD risk. For treatment of dyslipidemia, the absolute numbers for LDL and HDL are generally considered more relevant.

1.8 Determining Risk

While ATP III classifies levels of serum lipids from "optimal" to "very high", the optimum level for each individual varies depending on other risk factors for CHD, such as age, male gender, family history of CHD, smoking, hypertension, physical inactivity, overweight and obesity, diabetes, heavy alcohol consumption, and stress. The process used to determine CHD risk level and corresponding optimal serum lipid levels is detailed in the "ATP III Guidelines At-A-Glance Quick Desk Reference" (available n PDF download from www.nhlbi.nih.gov/guidelines/cholesterol/atglance.pdf). Below is a brief discussion of the significance of the different risk categories.

High-risk
The target LDL level for high-risk patients is <70 mg/dL. Current guidelines recommend dietary therapy for high-risk patients whose LDL levels are >100 mg/dL and drug therapy for those whose LDL levels are ≥130 mg/dL.

Intermediate Risk
The target LDL level for intermediate-risk patients is <100 mg/dL. Patients who exceed this level can begin therapeutic lifestyle changes (TLC) if their calculated 10-year risk is less than 10%. If calculated 10-year risk is between 10% and 20%, then drug treatment should be initiated. Immediate drug therapy is recommended for intermediate-risk

patients whose LDL levels are ≥160 mg/dL, even if calculated 10-year risk is less than 10%.

Low-risk
An LDL level ≤160 mg/dL is the goal for low-risk patients. Low-risk patients with LDL levels between 160 mg/dL and 189 mg/dL should begin TLC. If a 3-month trial of TLC fails to reduce LDL levels to <160 mg/dL, drug therapy can be considered. According to current guidelines, low-risk patients whose LDL levels are ≥190 mg/dL should be started on drug treatment.

1.9 Therapeutic Options:

Drug Treatment
Four major classes of medications are used to treat dyslipidemia:

- HMG-CoA reductase inhibitors (statins)
- Bile acid sequestrants
- Nicotinic acid
- Fibric acids

Statins (rosuvastatin, atorvastatin, simvastatin, pravastatin, fluvastatin, and lovastatin) can lower LDL levels 18% to 55%, and triglyceride levels 7% to 30%. They can also raise HDL levels by 5% to 15%. Major side effects include myopathy and elevation of liver enzyme levels.

Many clinical trials have shown that statins reduce incidence of major coronary events, CHD death, and stroke, and they may also reduce the need for coronary procedures, and lower total mortality. However, there is controversy.

Statins work by inhibiting the enzyme HMG-CoA reductase and are therefore also known as HMG-CoA reductase inhibitors. The process begins with acetyl-CoA. Three acetyl-CoA molecules combine to form hydroxymethyl glutaric acid (HMG). The step from HMG to mevalonate requires HMG-CoA reductase. Statin drugs work by inhibiting this enzyme, and herein lies the potential for numerous side effects because statin drugs inhibit not just the production of cholesterol, but a whole family of intermediary substances, many if not all of which have important biochemical functions.

Cholesterol is one of three end products in the mevalonate chain. The two others are ubiquinone and dilochol. Ubiquinone, also known as Co-Enzyme Q10, is a critical cellular nutrient biosynthesized in the mitochondria. It plays a role in ATP production in the cells and functions as an electron carrier to cytochrome oxidase, a major respiratory enzyme. The heart also requires high levels of Co-Q10. Ubiquinone is found in all cell membranes, where it plays a role in maintaining membrane integrity critical to nerve conduction and muscle integrity. Co-Q10 is also vital to the formation of elastin and collagen. Side effects of Co-Q10 deficiency include muscle-wasting leading to weakness

and severe back pain, heart failure (the heart is a muscle), neuropathy, and inflammation of the tendons and ligaments often leading to rupture.

Finally, there is controversy regarding the mechanism by which statins reduce CHD risk. While statins significantly reduce serum cholesterol levels, this may not be the cause of reduction of cardiac events. The fact that some studies have shown that statins can prevent heart disease, at least in the short term, is most likely explained not by the inhibition of cholesterol production but because they block the creation of mevalonate. Reduced amounts of mevalonate seem to make smooth muscle cells less active, and platelets less able to produce thromboxane. Atherosclerosis begins with the growth of smooth muscle cells in side artery walls, and thromboxane is necessary for blood clotting.

Bile acid sequestrants (cholestyramine, and colestipol) can reduce LDL levels 15% to 30% and raise HDL levels 3% to 5%. They have no effect on triglyceride levels. Major side effects include gastrointestinal distress, constipation, and a decrease in the absorption of other drugs. Clinical trials have shown that these agents reduce the incidence of major coronary events and CHD death.

Nicotinic acid (niacin/vitamin B3) can reduce LDL levels 5% to 25% and triglyceride levels 20% to 50%. Nicotinic acid also raises HDL levels 15% to 35%. Major side effects of nicotinic acid include flushing, hyperglycemia, hyperuricemia, gastrointestinal distress, and hepatotoxicity. Clinical trials have shown it can prevent major coronary events.

Fibric acids (fenofibrate, and gemfibrozil) can reduce LDL levels 5% to 20% and triglyceride levels 20% to 50%, as well as raise HDL levels 10% to 20%. Major side effects include dyspepsia, gallstones, myopathy, and unexplained noncardiac death. Clinical trials have shown that they lower the risk of major coronary events.

Effects of the major dyslipidemia drugs on serum lipid levels

Drug Type	**Effect on LDL**	**Effect on HDL**	**Effect on Triglycerides**
Statins	Reduce 18-55%	Raise 5-15%	Reduce 7-30%
Bile acid sequestrants	Reduce 15-30%	Raise 3-5%	No effect
Nicotinic acid	Reduce 5-25%	Raise 15-35%	Reduce 20-50%
Fibric acids	Reduce 5-20%	Raise 10-20%	Reduce 20-50%

Hormone replacement therapy. ATP III makes it clear that hormone replacement therapy (HRT) is not a substitute for lipid-lowering drugs in dyslipidemic women. Although HRT lowers LDL levels, studies have not shown that it reduces the risk of coronary events.

Therapeutic Lifestyle Changes (TLC)
TLC (therapeutic lifestyle changes) includes diet, weight, and exercise guidelines as set forth in ATP III. The guidelines are as follow:

- TLC Diet:
    - Saturated fat < 7% of daily calories, cholesterol < 200mg/day.
    - Consider increasing:
        - viscous fiber (10-25g/day; e.g., cereal grains, beans, peas, legumes, fruits, and vegetables).
        - plant stanols/sterols (2g/day). These are present naturally in small quantities in many fruits, vegetables, nuts, seeds, cereals, legumes, vegetable oils, and other plant sources, but the amounts are generally small. Some foods are now fortified with stanols, so these can be considered for incorporation in the diet as well.)
    - Weight management
    - Increased physical activity

1.9 Non-Mainstream Ideas about Cholesterol and Dyslipidemia

The above discussion has focused primarily on the mainstream views of cholesterol and dyslipidemia. However, there are divergent opinions that are worth examining.

While the mainstream medical establishment has interpreted the research to indicate elevated cholesterol is a major cause of cardiovascular disease and premature death, there are others who dispute these claims. One researcher, Dr. Uffe Ravnskov, who has conducted extensive reviews of the literature and conducted some meta-analyses notes that studies show people with the highest cholesterol live the longest, and that many studies of elderly people show that high cholesterol is not a risk factor for coronary heart disease. He notes that many studies have found that total mortality was inversely associated with either total or LDL-cholesterol, or both, that high cholesterol levels are protective against infection, and that the risk of dying for patients with chronic heart failure is strongly and inversely associated with total cholesterol, LDL-cholesterol and also triglycerides; those with high lipid values live much longer than those with low values.

Ravnskov asserts that, "Most studies of young and middle-aged men have found high cholesterol to be a risk factor for coronary heart disease, seemingly a contradiction to the idea that high cholesterol is protective. Why is high cholesterol a risk factor in young and middle-aged men? A likely explanation is that men of that age are often in the midst of their professional career. High cholesterol may therefore reflect mental stress, a well-known cause of high cholesterol and also a risk factor for heart disease. Again, high cholesterol is not necessarily the direct cause but may only be a marker. High cholesterol in young and middle-aged men could, for instance, reflect the body's need for more cholesterol because cholesterol is the building material of many stress hormones. Any possible protective effect of high cholesterol may therefore be counteracted by the negative influence of a stressful life on the vascular system."[ii]

For further reading on the controversies surrounding this topic, The International Network of Cholesterol Skeptics (http://thincs.org) has a wealth of information.

2. Dyslipidemia in Chinese Medicine

2.1 Fundamentals of Dyslipidemia in Chinese Medicine

Dyslipidemia is not a traditional disease classification in CM. Modern books on the subject of hyperlipidemia state that if falls within the scope of traditionally recognized conditions such as *phlegm turbidity, phlegm dampness, turbid obstruction, obesity*, etc, and that the main organs involved are the Spleen, Liver, Kidneys, and Heart.

It is of interest that most modern sources place dyslipidemia within the scope of traditionally recognized condition such as phlegm, turbidity, and dampness. Clearly, as discussed below, there are other mechanisms involved in the disorder, such as deficiency of yin, yang, qi, blood, and the organs, stagnation of qi and blood, etc. However, this may be due to the latter being more related to the etiology or pathomechanism of the disorder, and the former more related to the actual state of dyslipidemia. If this is true, it would imply that most modern authors consider pathogenic fluids (phlegm, turbidity, dampness) to be the main form in which elevated serum lipids present themselves. Whether it is true that all patients with dyslipidemia suffer from one or more type of pathogenic fluid, this author can't say. Is it not possible for a dyslipdemia patient to present with pure yin deficiency, or some other pattern, and no discernible pathogenic fluids? The vast majority of modern literature seems to suggest not. However, throughout the rest of this course there are ideas that seem to suggest this may be possible, and ultimately, clinicians need to keep an open mind and flexibly approach each patient without preconceived notions about pattern diagnosis. This is especially true, at least in this author's opinion, when those notions are formed around biomedically defined illnesses, such as pathogenic fluids and dyslipidemia in this case.

2.1.1 The Spleen and Dyslipidemia

Hyperlipidemia belongs to the traditional CM disease classification "phlegm turbidity", for which there are essentially two causes. The first is termed "external", and is due to irregular dietary habits (such as over-eating, eating at irregular times, or skipping meals), and excessive consumption of sweets, fatty foods, and/or alcohol, all of which can damage the Spleen and cause obstruction of the Spleen mechanism. The second is termed "internal", and is due to dysfunction of the internal organs, insufficiency of Triple-Burner qi transformation, or dysfunction of Spleen circulation/transportation leading to an inability of the body to transform fats and descend turbidity. This results in the accumulation of fats, phlegm, turbidity, and stagnant blood in the blood vessels with subsequent obstruction of the blood vessels. Regardless of the cause, internal or external, both can lead to obstruction of Spleen transportation and transformation, the formation, accumulation, and stagnation of dampness, and the development of dyslipidemia.

Obstruction of Spleen Transportation is a Key Component in Dyslipidemia

Stagnation of phlegm dampness, obstruction of the Spleen mechanism, deficiency and weakness of Spleen qi, and accumulation of fats and turbidity are all major factors in the etiology of dylipidemia.

Neijing Suwen Chapter 21, Jingmai Bielun, states, "…drink enters the Stomach and the Stomach distributes the essence qi upwards to the Spleen. The Spleen distributes essence, going upward to the Lung, which regulates the waterways and sends the fluids down to the Urinary Bladder. (The Lung) distributes the water essence to the entire body and to the vessels of the five viscera…". This clause explains the relationship between the Spleen's function of distributing essence and the metabolism and distribution of body fluids, highlighting the Spleen's important role in fluid metabolism. It can also help us understand the role of the Spleen in lipid metabolism.

Li Dong-Yuan said, "The Spleen and Stomach receive (food and drink), and then can cook, rot, and ripen the five grains. (If the clear qi does not ascend) the grain qi stagnates and flows downward, becoming dampness." In this case, the Spleen is unable to distribute the essence, qi transformation is impaired, and normal body fluids become turbid dampness. In extreme cases the sticky turbidity causes blood stagnation, and obstructs the Heart vessels.

Additionally, weakness and deficiency of the Spleen and Stomach has an impact on the metabolism of dampness and lipids that already exist. Weakness of Spleen transportation and transformation means dampness and lipids that are already present in the blood are unable to be transformed and eliminated and remain in the blood. This dampness further impairs Spleen and Stomach transportation and transformation, leading to formation of more damp turbidity and an increase of serum lipids.

In clinic it is common to see overweight and obese patients with dyslipidemia, and in fact the vast majority of them suffer from deficiency of Spleen qi. These patients are usually considered to be suffering from "deficiency-type obesity", a condition characterized by excess in the form of accumulation of pathogenic fluids, with underlying deficiency of Spleen qi. The typical patient is middle-aged or above, overweight, with symptoms such as dizziness with a heavy feeling in the head, stuffiness of the chest, abdominal fullness, nausea, fatigue, weakness, a pale tongue with a greasy white tongue-coating, and a thin, soggy pulse.

Note that while Spleen qi deficiency is characterized by poor appetite, there are many patients like this with strong appetite. This is often due to concurrent heat in the Stomach, which causes hunger. This condition is known as "strong Stomach, weak Spleen", and is characterized by hunger (Stomach heat) but inability to eat much and/or bloating and distention after eating (Spleen weakness).

The Stomach in CM is responsible for receiving food and liquids after ingestion, and for "rotting and ripening" them before passing them on to the Spleen for further digestion. Because the Stomach is in charge of receiving food and liquid, when there is heat in the Stomach hunger is usually exaggerated. However, heat in the Stomach can take two

forms. The first, Stomach yin deficiency, presents with a gnawing uncomfortable hunger, inability to eat much, and after eating there may be discomfort. The second, true heat or fire in the Stomach, presents with strong hunger and the ability to eat quite a bit, but after eating hunger returns rapidly, epigastric pain and/or burning, bad breath, bleeding gums, and hard, dry stool. In both types, thirst with a preference for cold drinks is present.

Spleen Deficiency and Dyslipidemia in Modern Research

From a biomedical perspective, the CM Spleen is closely related to the various organs and processes of the digestive system, and Spleen deficiency clearly includes weakness of digestive system function. Modern research shows that the Spleen is closely related to various receptors and enzymes in the body and that strengthening the Spleen can improve their functions, leading to acceleration of metabolic processes. Dysfunction of these receptors and enzymes is one of the main causes of dyslipidemia. Following are two representative studies.

Research by Xiong Wen-Sheng et al[iii] shows that regulating Spleen function and transforming phlegm can regulate lipid levels and has an effect upon enzymes involved in lipid metabolism. Lipoprotein lipase (LPL), a key enzyme in lipoprotein metabolism, catalyses the hydrolysis of the triacylglycerol component of circulating chylomicrons and very low density lipoproteins, thereby providing non-esterified fatty acids and 2-monoacylglycerol for tissue utilization. Abnormalities in LPL function have been found to be associated with atherosclerosis, chylomicronaemia, obesity, Alzheimer's disease, and dyslipidemia associated with diabetes, insulin resistance, and infection[iv]. Xiong et al found that a Chinese herbal preparation called Regulate Spleen Transform Phlegm Reduce Lipids Pill significantly increased LPL activity (more so than fenofibrate) and lowered serum triglycerides.

Regulate Spleen Transform Phlegm Reduce Lipids Pill (理脾化痰降脂片):

Fa Ban Xia 法半夏 *Rhizoma Pinelliae Tematae*
Bai Zhu 炒白术 *Rhizoma Atractylodis Macrocephalae*
Tian Ma 天麻 *Rhizoma Gastrodiae*
Ju Hong 橘红 *Pars Rubra Epicarpii Citri Erythrocarpae*
Fu Ling 茯苓 *Sclerotium Poriae Cocos*
Lai Fu Zi 莱菔子 *Semen Raphani Sativi*
Dan Shen 丹参 *Radix Salviae Miltiorrizae*
Ju Ming Zi 决明子 *Semen Cassiae*
Ze Xie 泽泻 *Rhizoma Alismatis*
Tai Zi Shen 太子参 *Radix Pseudostellariae*
Green Tea 绿茶 *Camellia Sinensis*

Research by He Ren[v] shows that the method of strengthening the Spleen, dissolving phlegm, and transforming blood stasis can reduce levels of serum total cholesterol and triglycerides, and increase levels of HDL-C. The increase in serum HDL-C concentrations also leads to an increase in LPL activity, possibly explaining the reduction in serum triglycerides.

2.1.2 Liver and Dyslipidemia

Liver Qi Stagnation as a Factor in Dyslipidemia

The Liver in CM is responsible for "free coursing" (疏泄 shu xie), which essentially means the Liver is responsible for ensuring the free and unobstructed movement of qi and the so-called qi-dynamic, or qi-mechanism, which represents the functional aspects of qi. By ensuring the free-coursing of qi, the Liver enables the physiological processes of the various systems of the body to proceed normally. When Liver function is healthy, the movement of qi and blood and the function of the organs are harmonious. When Liver qi is congested or stagnant, a wide range of disorders may result. In regards to dyslipidemia, the most important of these are the effects of Liver disharmony on Spleen and Stomach function, and blood circulation. The Liver's free-coursing function assists the Spleen and Stomach in the process of digestion and elimination. Impairment of this function leads to Spleen and Stomach dysfunction. Furthermore, as qi is the motive force behind the movement of blood, impaired qi circulation leads to impaired blood circulation and blood stagnation. These are all significant factors in both the etiology and systemic effects of dysipidemia.

Emotional imbalance and dysfunction of Liver free-coursing can result in stagnation of the qi mechanism, impairment of blood circulation, stagnation of body fluids and generation of phlegm. Ming dynasty physician Dai Yuan-Li said, "Because of qi (dysfunction) accumulation results, and accumulation of qi results in phlegm." Ming dynasty physician Dai Si-Gong said, "Emotional upset and other disturbances and abnormalities of the five emotions can cause the clear to be transformed into turbidity." These quotes help explain the role of Liver qi stagnation in the production of phlegm, dampness, and turbidity, and the development of dyslipidemia.

Modern research also shows that emotions have an effect on serum lipid levels, and that long-term depression, anxiety, and grief can lead to an increase in serum cholesterol levels[vi]. In particular, long-term stress and anxiety can cause imbalances in endocrine and CNS function, and reduced insulin secretion and lipoprotein lipase (LPL) activity[vii]. LPL increases lipid metabolism and breaks down triglycerides into glycerin and fatty acids, making them available for tissue utilization. Decreased LPL activity causes a reduction in triglyceride breakdown, leading to elevation of serum triglycerides. One study showed significant reduction in serum lipids of hyperlipidemia patients that made no dietary changes but routinely practiced relaxation techniques for 3 months[viii]. This shows the importance of stress reduction and emotional wellbeing in the prevention and treatment of dyslipidemia, and reiterates the significance of the Liver's free-coursing function in relation to dyslipidemia.

Liver Qi Invading the Spleen

Under normal physiological conditions, the Liver and Spleen mutually assist each other in their respective functions. However, under pathological conditions they adversely

impact each other. Aside from their functions of generating blood, storing blood, controlling blood, and regulating blood, the Spleen and Liver also help determine the quality of the blood that is produced. For example, when the Liver and Spleen are harmonious, the "clear" essence of the food is properly extracted by the Spleen and incorporated into the blood, the blood is circulated without impairment, and the "turbid" dregs of food are eliminated from the body. In this case, the quality of the blood is good. When Liver qi stagnates due to improper dietary habits, emotional imbalance, or other factors, the free-coursing function of the Liver is disrupted. This affects the Spleen's ability to separate the "clear" from the "turbid" and gives rise to pathological fluids such as phlegm and dampness, which clog the arteries and may lead to elevation of serum lipid levels. It also impairs the smooth circulation of blood, causing blood stagnation. In situations like these, the quality of the blood can be said to be poor.

One modern study by Wei et al[ix] showed significant reduction in serum lipids in 160 hyperlipidemia patients by primarily using Xiao Yao San to harmonize Liver and Spleen function. This helps demonstrate that while phlegm and blood stagnation are significant factors in dyslipidemia, these are usually the result of underlying physiological imbalances, such as Liver and Spleen disharmony. Therefore, solely treating the branch (phlegm and blood stagnation in this case) is not enough- the root pathology must also be addressed.

Liver Qi Deficiency as a Factor in Dyslipidemia

Huang Di Neijing Suwen Chapter 1, Shang Gu Tian Zhen Lun, says, "When men reach 56 years of age the Liver qi is weak, the tendons cannot move, the heavenly dew is exhausted, the essence is scant, and the Kidney is weak." Su Wen Chapter 21, Jing Mai Bie Lun, says, "Food qi enters the Stomach and the fluids are dispersed to the Liver, the nutritious qi goes to the tendons; food qi enters the Stomach, the turbid qi goes to the Heart, the nutritious essence goes to the vessels." These quotes demonstrate that the Liver is dependent upon the essence derived from food for nourishment, and that as one ages the qi, blood, yin, and yang of the Liver gradually become depleted and weak. Li Dong-Yuan said, "Irregularity of happiness and anger, irregular lifestyle, and overwork and exhaustion, all damage the qi. When qi is weakened, fire flares." This further shows that deficiency of Liver qi is an important factor in dysilipidemia, particularly in the elderly.

Deficiency of Liver qi impairs the Liver's ability to carry out its normal physiological functions. Weakening of the Liver's free-coursing function can lead to formation and stagnation of phlegm dampness, stagnation of blood in the vessels, and can give rise to elevated serum lipids and pathological changes of the arteries, such as atherosclerosis. It is common to see elderly dyslipidemia patients suffering from emotional problems or depression, with insufficient hepatic production of lipoprotein-lipase (LPL), hepatic triglyceride lipase (HTGL), and lecithin cholesterol acyltransferase (LCAT). This helps demonstrate that deficiency of the Liver and stagnation of Liver qi impact the enzymes involved in lipid metabolism.

Zhao et al[x] conducted research on the effects of Zhi Gan Le Capsules in the treatment of 146 cases of qi deficiency-damp-phlegm-blood stagnation-obstruction type

hyperlipidemia, and found this preparation significantly reduced serum triglycerides, inhibited the deposit of fats in the liver, and improved blood rheology.

Zhi Gan Le Capsules
- Huang Qi 黄芪 *Radix Astragali Membranacei*
- Jue Ming Zi 决明子 *Semen Cassiae*
- Sheng Shan Zha 生山楂 *Fructus Crataegi*
- Da Huang 大黄 *Radix Et Rhizoma Rhei*
- Ze Xie 泽泻 *Rhizoma Alismatis*
- He Shou Wu 何首乌 *Radix Polygoni Multiflori*
- Zhe Bei Mu 浙贝母 *Fritillariae Thunbergii Bulbus*
- Fu Ling 茯苓 *Sclerotium Poriae Cocos*
- Chen Pi 陈皮 *Pericarpium Citri Reticulatae*
- Fa Ban Xia 法半夏 *Rhizoma Pinelliae Tematae*
- Dan Shen 丹参 *Radix Salviae Miltiorrizae*
- Ge Gen 葛根 *Radix Puerariae*
- Chai Hu 柴胡 *Radix Bupleuri*

In their analysis of the study, the researchers point out that qi deficiency is often the root-cause of phlegm-dampness stagnation and blood stagnation. Therefore, huang qi was selected for its ability to supplement the qi and support the zheng qi. Modern research shows huang qi can dilate blood vessels, improve blood circulation, reduce blood viscosity, lower serum lipids, and prevent atherosclerosis[xi]. When paired with herbs such as jue ming zi and shan zha, both of which can reduce serum lipids, and ze xie, which can improve hepatic lipid metabolism, inhibit hepatic synthesis of triglycersides and reduce fatty liver, the effects are even more pronounced.

Liver Yin Deficiency and Deficiency Heat Scorching the Fluids as a Factor in Dyslipidemia

The Liver is in charge of free-coursing, and therefore has a commanding role in the storage, distribution, and utilization of the body fluids, and in the inter-transformation of fluids, blood, and essence. After middle-age, the yin is depleted by half, the Liver and Kidneys are deficient, and essence is diminished. The resultant deficient heat begins to scorch the body fluids, and the clear fluids are transformed into turbidity. This leads to the creation of phlegm and turbidity, which in some instances may translate into elevated serum lipids. Furthermore, if deficient Kidney water fails to nourish Liver wood, Liver free-coursing is adversely affected, causing stagnation of fluids and blood, and accumulation of turbidity and blood stagnation in the vessels.

Liver and Kidney yin deficiency with heat stagnation is a common clinical presentation and should be addressed with medicinals that nourish the yin and fluids and promote circulation through the vessels. Caution must be exercised with the use of medicinals that warm and supplement the Kidney yang, for fear that they may exacerbate the fire and further scorch the fluids. Because the Liver and Kidney derive from the same source, nourishing Kidney yin in turn nourishes the Liver. When the Liver is nourished, its free-

coursing function improves, assisting Spleen transportation and transformation. The overall effect from this strategy is to transform phlegm and remove blood stasis, and promote the healthy transformation and distribution of body fluids, qi, and blood.

2.1.3 Kidney and Dyslipidemia

While it is common to discuss the role of the Spleen and Liver in dyslipidemia, the role of the Kidney cannot be overlooked. One thorough review of modern Chinese medicine literature found that Kidney deficiency is considered by many clinicians and researchers to be a primary causative factor in the etiology of dyslipidemia, and in particular the relationship of Kidney qi deficiency to the production of pathological substances. Kidney qi deficiency can impede water metabolism, leading to the formation and retention of phlegm and dampness. Zhang Jing-Yue said, "The commander of water, must be the water organ (Kidney)", and, "the qi derived from essence metabolizes water and liquids." Therefore, when Kidney qi is strong, the Kidney's qi-transformation function involved in water metabolism can proceed normally. If deficient, Kidney qi transformation is weakened, and water metabolism is impaired.

Kidney deficiency as a factor in the development of dyslipidemia

While CM never developed a concept of blood lipids, very early on there was knowledge of "fat", although it appears that this fat is physiological, and may not in fact be related to the pathological fat of dyslipidemia. Ling Shu chapter 36, Wu Long Jin Ye Bie, states, "The fluids from the five grains harmoniously merge to become fat. (The fat) seeps into the bone cavities, nourishes the brain and marrow, and flows downward to the inner thighs." This explains the origin and movement of fat in the body: deriving from the fluids of food, following fluid circulation and distribution, filling the lumens of the bones, nourishing the brain and marrow, and moistening the skin and flesh. Zhang Jing-Yue said, "The fluids harmoniously merge to become fat, which fills the bone cavities, and then becomes the brain, the marrow, the essence, and the blood." Zhang here further alludes to the close relationship of fat to the Kidneys by suggesting that it in fact transforms into the substance of the brain, the marrow, and the essence, all of which are related to the Kidney. He also says fat transforms into blood, possibly alluding to the role of marrow in the production of blood, but some modern authors suggest that this may be the beginning of knowledge of the existence of blood lipids. In any case, we can see that fat is a turbid and thick portion of the body fluids, is a component (or at least pre-cursor) of blood, originates from the water and grains, and is closely related to the Kidney. Whether or not this "fat" has anything to do with the pathological lipids of dyslipidemia is debatable. But these passages do elucidate certain aspects of Kidney fluid metabolism, and may be useful for the understanding of dyslipidemia. In any case, the Kidney commands the body fluids, and plays a dominant role in the storage, distribution, and utilization of the fluids, as well as in the inter-transformation of jin, ye, essence, and blood. Deficiency of the Kidney can therefore impair the distribution of the fluids (and presumably "fat"), causing the formation of dampness, turbidity, and phlegm, which over time may flow into the vessels and lead to phlegm and blood stagnation obstructing the vessels.

Kidney Yang Deficiency, Water Congealing and Becoming Phlegm

Ming dynasty physician Wang Lun said, "The root of phlegm is water, its source is the Kidney. The action of phlegm is in dampness, and is commanded by the Spleen." This quote demonstrates that both Kidney and Spleen are related to the formation, metabolism, and movement of phlegm and dampness. The Kidney commands the source yang and the entire body's water and fluids. If Kidney yang is deficient there is not enough warmth to facilitate the steaming action of qi transformation. Therefore, fats are not transformed, turbidity congeals and becomes phlegm, and dyslipidemia may result. Symptoms of Kidney yang deficiency with concurrent phlegm dampness may include dizziness, poor memory, fatigue, cold limbs and body, poor appetite, nocturia, morning diarrhea, impotence, spermatorrhea, pale and puffy tongue with greasy or wet white coating, and deep and moderate or deep and thin pulse. In cases of Kidney yang deficiency with dampness and hyperlipidemia, herbs such as fu zi, gui zhi, huang qi, du zhong, huang jing, he shou wu, and shan zhu yu are very effective. Research by Hou Shuang-Feng et al[xii] showed that Jin Gui Shen Qi Wan was effective in preventing and treating atherosclerosis, suggesting that Kidney yang deficiency with stagnation of water and phlegm can be a significant factor in the etiology of dyslipidemia, and of atherosclerosis.

Kidney Yin Deficiency, Deficient Fire Scorching the Fluids

Ming dynasty physician Zhao Xian-Ke said, "When water does not flow it becomes phlegm. When water is boiled it becomes phlegm….When yin deficiency fire stirs, water is boiled- this action comes from the Kidney." This quote helps explain the role of Kidney yin deficiency fire in the generation of phlegm turbidity. After middle-age the Kidney yin, qi, and essence are naturally depleted. Kidney yin deficiency gives rise to deficient fire which scorches the fluids, giving rise to phlegm turbidity. The phlegm and turbidity stagnate in the body, flowing into and obstructing the vessels. Long-term stagnation of phlegm turbidity can transform into heat, further damaging the Kidney yin. Ultimately a complex syndrome of mixed deficiency and excess results. Wang Xu et al[xiii] used nourish Kidney yin, invigorate blood and transform phlegm method in the treatment of Kidney yin deficiency type hyperlipidemia. They achieved significant results in the reduction of blood sugar and serum lipids, as well as improvements in blood viscosity and blood circulation. Another study, by Ying Zhao-Hong[xiv] employed modified Liu Wei Di Huang Wan in the treatment of hyperlipidemia with good success. And yet another study by He Ren[xv] used nourish Kidney, transform phlegm, and remove blood stasis method with good results in treatment of hyperlipidemia.

Kidney Essence Deficiency and Stagnation of Fat

Modern medicine recognizes that hyperlipidemia, and in particular severe hyperlipidemia, often has a genetic, hereditary component. For example, deficit of LDL receptor genes can cause familial hypercholesterolemia, and deficit of lipoprotein lipase (LPL) genes can cause familial hypertriglyceridemia. From a Chinese medicine perspective, this suggests a connection to the Kidney, as the Kidney is the root of pre-heaven essence. Ling Shu chapter 8, Ben Shen Pian, states, "That which life comes from is called 'essence'. When the two essences[1] merge it is called 'shen/spirit'." This quote explains that when the essence (which is derived from the Kidney) of the parents comes together, new life is

[1] The "two essences" here is a reference to the essence from the father and mother, which come together during conception.

formed. This meaning can be extended to explain the role of the Kidney, and essence in particular, in heredity and genetics. Since a large number of hyperlipidemia patients have a family history of the disorder, heredity and genetics clearly play significant roles, implicating the significance of Kidney essence.

Kidney Tonifying Medicinals and Dyslipidemia

As previously mentioned, formulas that tonify the Kidney such as Liu Wei Di Huang Wan, Ba Wei Di Huang Wan, Zhang Jing-Yue's Jin Shui Liu Jun Tang[2], etc., have been used successfully to treat dyslipidemia in cases of Kidney deficiency leading to impaired water metabolism and generation of phlegm. Clinical and pharmacological research has also demonstrated the effectiveness of many herbs that nourish the Liver and Kidney to reduce serum lipids and prevent and treat atherosclerosis. For example:

Gou Qi Zi 枸杞子 *Fructus Lycii Chinensis*: inhibits the deposit of fats in the liver and prevent fatty liver, and promotes generation of new hepatocytes.

Sang Ji Sheng 桑寄生 *Ramulus Loranthi Seu Visci*: reduces serum cholesterol, and according to classical sources dispels wind and damp, and unblocks the blood vessels.

Jue Ming Zi 决明子 *Semen Cassiae*: when fried can supplement the Liver and Kidney. Reduces serum cholesterol.

Zhi He Shou Wu 制首乌 *Processed Radix Polygoni Multiflori*: nourishes the Liver, Kidney, blood, and essence. Can inhibit intestinal absorption of cholesterol, strengthen intestinal peristalsis, quicken intestinal transit time of fats, speed up fat metabolism, and prevent deposit of lipids on the vascular lumen.

Xian Ling Pi 仙灵脾 *Herba Epimedii*: tonifies the Kidney yang and reduces serum lipids.

Ling Zhi 灵芝 *Ganoderma Japonicum*: tonifies the qi and reduces serum lipids.

Kidney in Conclusion

Clinical experience and laboratory research has clearly shown connections between Kidney deficiency and dyslipidemia. Modern research has shown Kidney deficiency can impact blood rheology and microcirculation, and lead to accumulation of phlegm and

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[2] Jin Shui Liu Jun Tang is composed of Er Chen Tang with the addition of dang gui and shu di huang. The source text indications are for Lung and Kidney yin deficiency with water overflowing and becoming phlegm, and contains:

Ban Xia 半夏 *Rhizoma Pinelliae Tematae* 6 grams
Chen Pi 陈皮 *Pericarpium Citri Reticulatae* 4.5 grams
Fu Ling 茯苓 *Sclerotium Poriae Cocos* 6 grams
Zhi Gan Cao 炙甘草 *Radix Glycyrrhizae* 3 grams
Shu Di Huang 熟地黄 *Processed Radix Rehmanniae* 9 to 15 grams
Dang Gui 当归 *Radix Angelicae Sinensis* 6 grams
Sheng Jiang 生姜 *Uncooked Rhizoma Zingiberis* 3 to 5 slices

stagnant blood in the vessels. Deficiency of Kidney yin, yang, qi, and essence all play a role, and appropriate supplementation of the Kidney can help remove one of the sources of the phlegm and blood stagnation which are often present in dyslipidemia.

Heart

While the Heart does not play a significant role in the development of dyslipidemia, it is relevant due to the pathological impact of dyslipidemia on the Heart. The Heart governs the blood vessels, which are of course a major site of dyslipidemia-induced pathology, and circulation of blood and fluids through the vessels relies on the pumping action derived from Heart qi. Disharmony of the five viscera can give rise to phlegm and stagnant blood, which may then obstruct the yang qi of the chest, resulting in chest impediment (xiong bi 胸痹) and Heart pain (xin tong 心痛).

Phlegm turbidity, blood stasis, and cold rheum are yin pathogens and therefore easily obstruct the blood vessels, impair blood circulation, obstruct the qi mechanism, and over a long period of time they can damage and deplete the Heart yang qi. Because qi is the motive force behind the movement of blood, Heart yang qi deficiency leads to weakening of the Heart's warming push of blood through the vessels, causing further impairment of blood circulation. In addition, deficiency of Heart yang qi allows for invasion of cold pathogen, which further impairs Heart function.

Modern medicine considers there to be three main factors in the development of impaired blood circulation[xvi]. The first is coronary, wherein heart pump strength is weakened. CM explains that when Heart qi is deficient the Heart's function of pumping and circulating the blood is weakened, and stroke volume and cardiac output are decreased. The second is vascular, such as arteriosclerosis and narrowing of the vascular lumen. The third is hematological, such as changes in blood rheology. CM explains that phlegm and blood stagnation retained in the blood vessels affect the quality and nature of the blood, slow the blood circulation, and lead to obstruction and reduced flexibility of the blood vessels.

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[i] van der Steeg WA, Holme I, Boekholdt M, et al. *High-density lipoprotein cholesterol, high-density lipoprotein particle size, and apolipoprotein A-I: significance for cardiovascular risk.* J Am Coll Cardiol 2008;51:634–42.

[ii] Uffe Ravnskov, MD, PhD. *The Benefits of High Cholesterol.* Wise Traditions in Food, Farming and the Healing Arts, Spring 2004.

[iii] Xiong Wen-Sheng et al. *Research of the clinical effects of Regulate Spleen Transform Phlegm Spleen Lipid Tablets*. China Journal of Pharmacology. 2005, 5(2): 35-38.

[iv] Mead JR, Irvine SA, Ramji DP. *Lipoprotein lipase: structure, function, regulation, and role in disease*. Journal of Molecular Medicine, Volume 80, Number 12 / December, 2002: 753-769.

[v] He Ren. *Function and mechanism of Chinese herbal medicine in the treatment of hyperlipidemia*. Yunnan Journal Of Chinese Medicine, 1999,20 (2): 31-33.

[vi] Cheng Zhi-Qing. *Chinese medicine prevention and treatment of hyperlipidemia*. People's Medicine Publishing House, 2002: 34.

[vii] Ibid: 35.

[viii] Ibid: 35.
[ix] Ibid: 35.
[x] Zhao Wen-Xia et al. *Clinical and laboratory research on Zhi Gan Le Jing Capsules in the treatment of phlegm-damp-blood stagnation-obstruction type hyperlipidemia.* Zhong Guo Zhong Xi Yi Jie He Magazine, 1997, 17(8):456-458.
[xi] Cheng Zhi-Qing. *Chinese medicine prevention and treatment of hyperlipidemia*. People's Medicine Publishing House, 2002: 34.
[xii] Hou Shuang-Feng et al. *Ba Wei Di Huang Wan in the prevention and treatment of laboratory induced atherosclerosis.* First Army Medical University Report, 1994, 21(2): 51-55
[xiii] Wang Xu et al. *Boost Kidney, invigorate blood, and transform phlegm method in the treatment of geriatric diabetes with concurrent hyperlipidemia.* Jiangsu Chinese Medicine, 1998, 19(5):10-11.
[xiv] Ying Zhao-Hong. *Modified Liu Wei Di Huang Wan in the treatment of 168 cases of hyperlipidemia.* National Medical Forum, 2000, 15(1): 26.
[xv] He Ren. *Function and mechanism of Chinese herbal medicine in the treatment of hyperlipidemia*. Yunnan Journal Of Chinese Medicine, 1999,20 (2): 31-33.
[xvi] Cheng Zhi-Qing. *Chinese medicine prevention and treatment of hyperlipidemia*. People's Medicine Publishing House, 2002: 34.

Review Exercises for Personal Enrichment

Answers on the Next Page

(This is not the course quiz)

High Cholesterol & Dietetics, #1

1. Hyperlipidemia refers to high levels of…

a. chylomicrons
b. very low-density lipoproteins (VLDL)
c. intermediate-density lipoproteins (IDL)
d. low-density lipoproteins (LDL)
e. high-density lipoproteins (HDL)
f. all of the above

2. LDL cholesterol typically accounts for 60-70% of total serum cholesterol. LDL is the main atherogenic lipoprotein and is the primary target of…

a. cholesterol-lowering therapy
b. autoimmune disorders
c. both A and B

3. Hyperlipidemia belongs to the traditional Chinese medicine disease classification…

a. phlegm turbidity
b. Yin deficiency
c. Yang deficiency

4. Emotional imbalance and dysfunction of Liver free-coursing can result in stagnation of the qi mechanism, impairment of blood circulation, stagnation of body fluids and generation of…

a. Yin essence
b. phlegm
c. red blood cells

Answers to Review Exercises

1. Hyperlipidemia refers to high levels of…
a. chylomicrons
b. very low-density lipoproteins (VLDL)
c. intermediate-density lipoproteins (IDL)
d. low-density lipoproteins (LDL)
e. high-density lipoproteins (HDL)
f. all of the above
answer: f

2. LDL cholesterol typically accounts for 60-70% of total serum cholesterol. LDL is the main atherogenic lipoprotein and is the primary target of…
a. cholesterol-lowering therapy
b. autoimmune disorders
c. both A and B
answer: a

3. Hyperlipidemia belongs to the traditional Chinese medicine disease classification…
a. phlegm turbidity
b. Yin deficiency
c. Yang deficiency
answer: a

4. Emotional imbalance and dysfunction of Liver free-coursing can result in stagnation of the qi mechanism, impairment of blood circulation, stagnation of body fluids and generation of…
a. Yin essence
b. phlegm
c. red blood cells
answer: b

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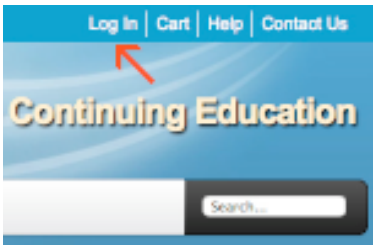

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