Natural Ways to Reduce High Cholesterol

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In A Nutshell

41% of Canadians have high Total Blood Cholesterol Levels (above 5.2 mmol/L or 200 mg/dL) - a cardinal risk factor for heart disease, stroke, and other life-threatening cardiovascular problems (Conversion = 0.026)

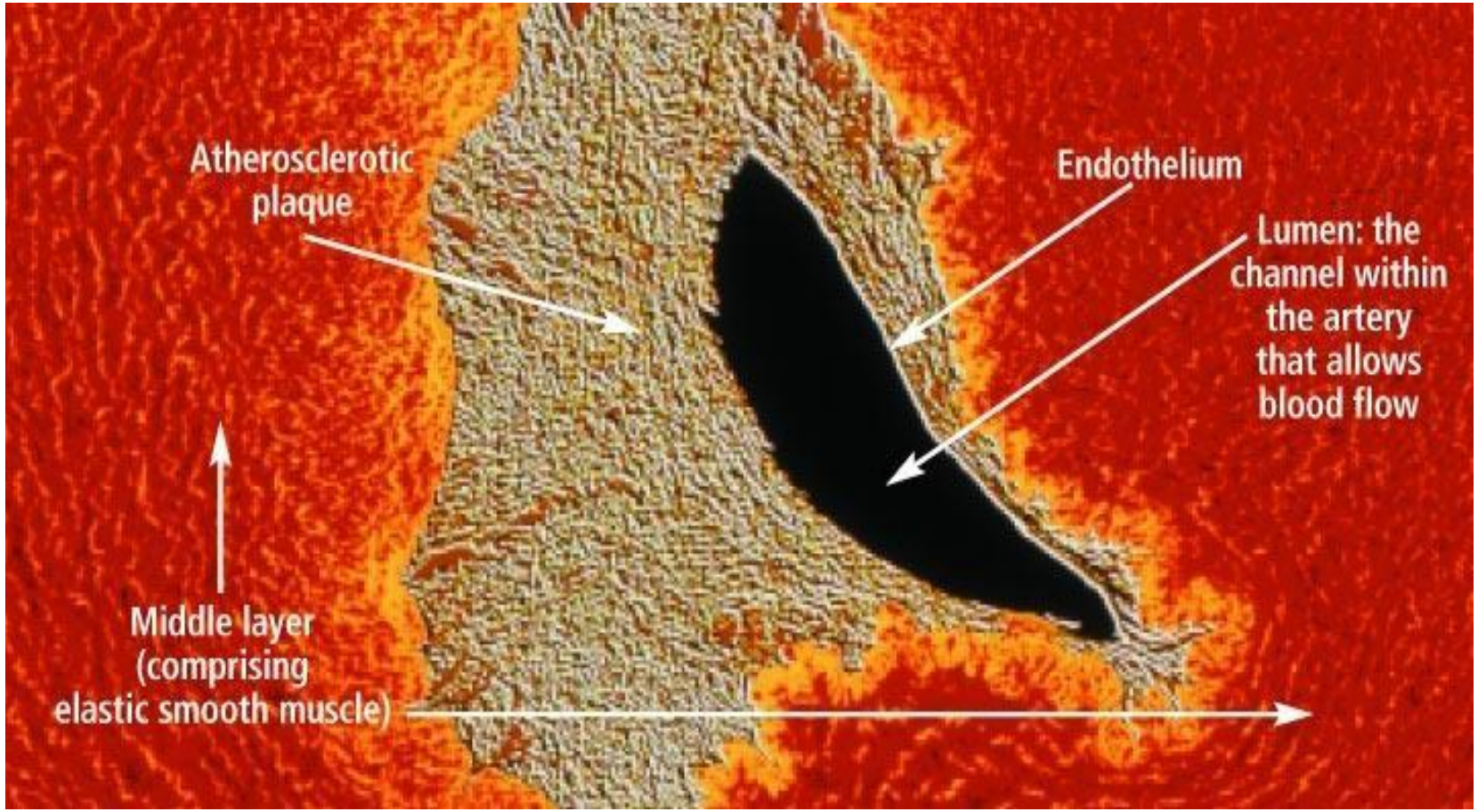
Thus 200 mg x .0.026 = 5.2 (But most ideal level = 150 mg/dL or 3.9 mmol/L)

Approximately 5% of individuals are born with a strong genetic defect (i.e., LDL-Receptor defect), which is responsible for sky-high cholesterol levels, and these individuals require drug therapy to reduce their levels into a safer range. Without drug therapy heart attacks begin in these individuals at age 35-40 and are inevitable by age 60 yr.

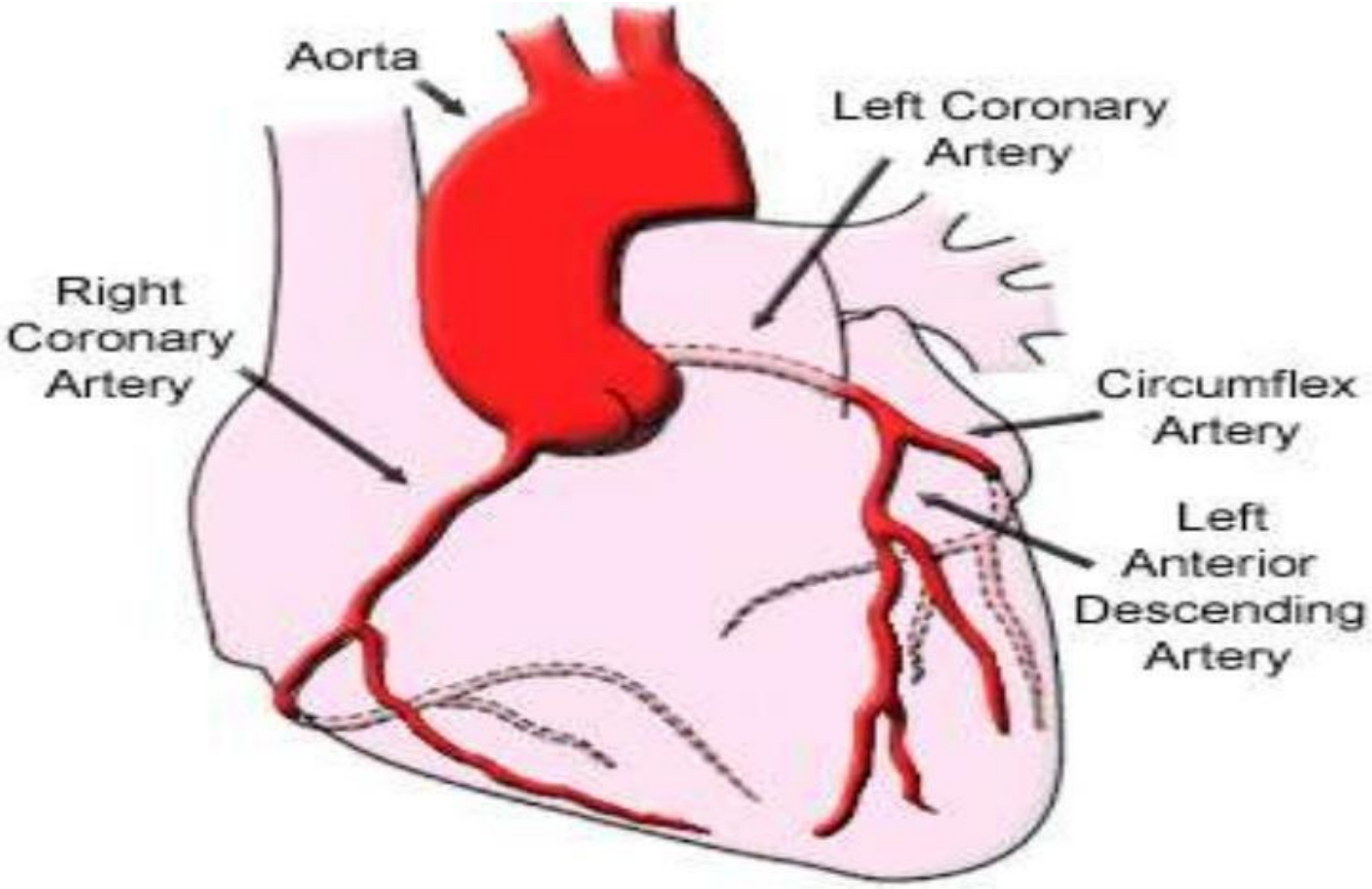
However, majority of people can get their blood cholesterol level into the desirable range through diet, nutritional and lifestyle medicine (if they really want to – but most people don’t want it badly enough, so they take drugs)

You Weren’t Born with Plaque: How Does It Develop?

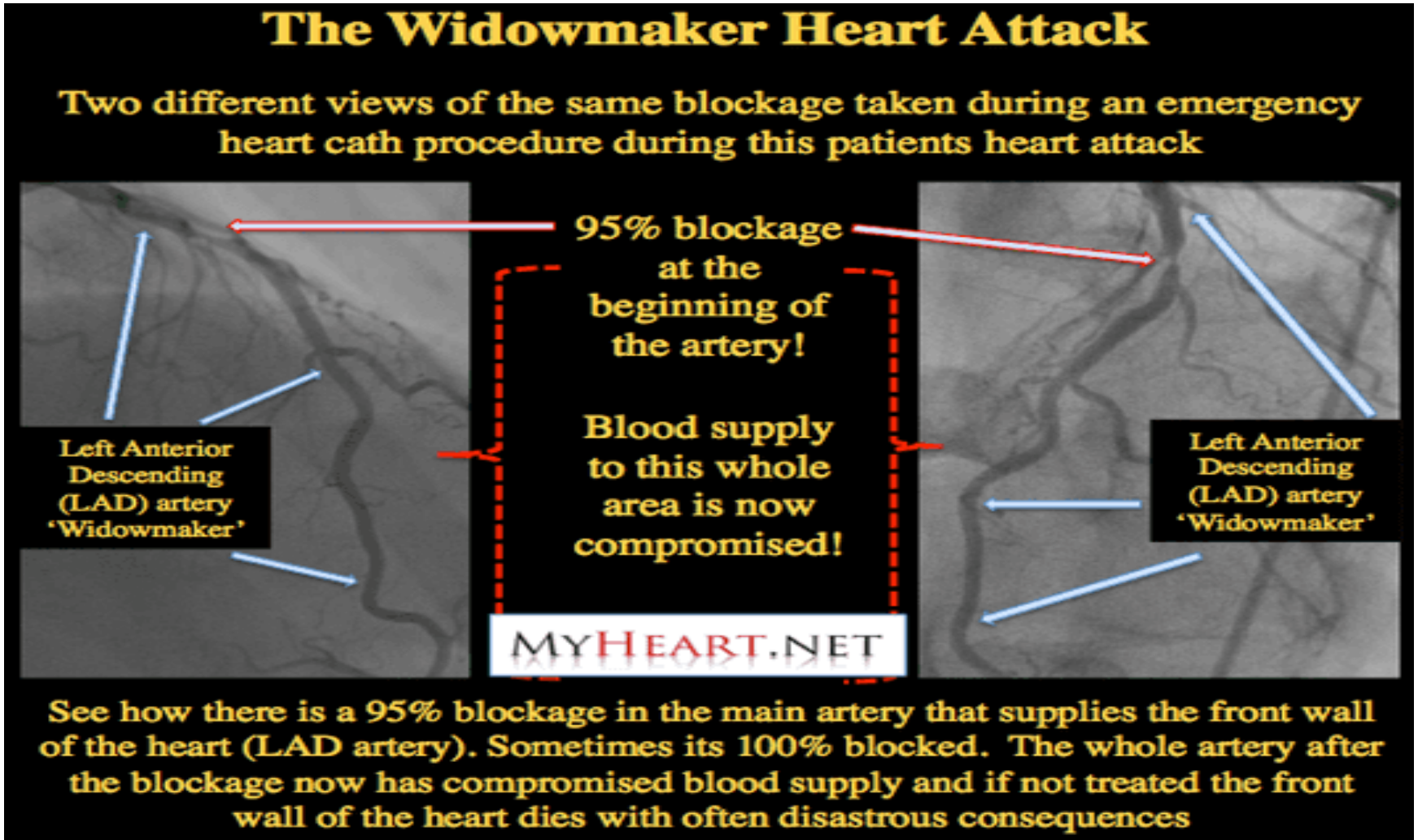
Advanced Atherosclerosis: Foam cells that comprise a main feature of atherosclerotic plaque are macrophages that have ingested unlimited amount of LDL-cholesterol.



Left Anterior Descending Artery (LAD): The Widow-maker



In 25-40% of Cases – Sudden Death Heart Attack is 1st Symptom: But Risk Factor Analysis Can Signal Warning and Prompt Proactive Preventive Action

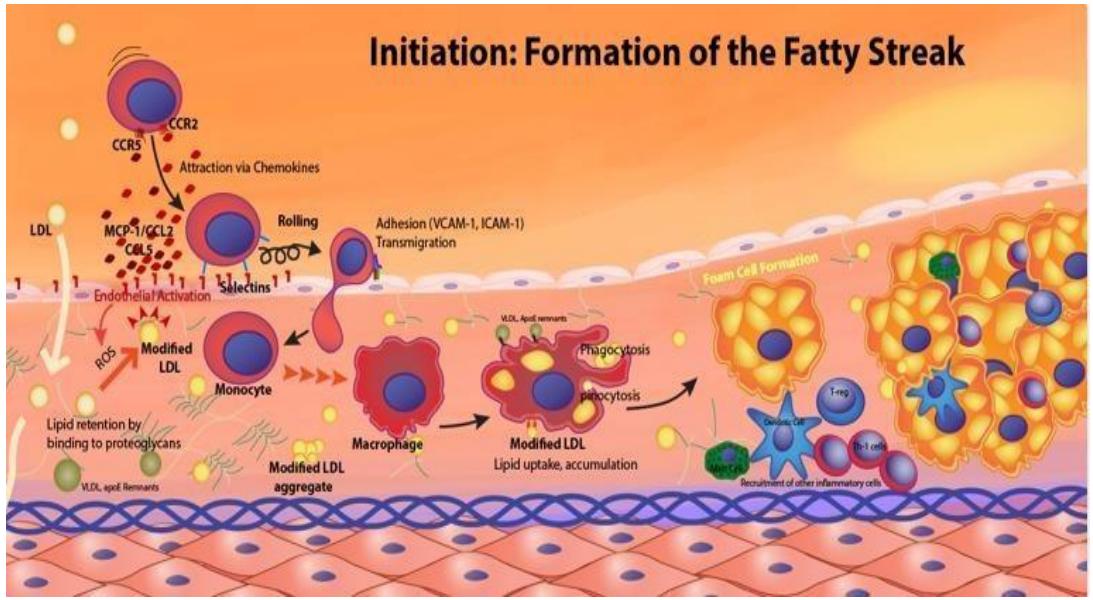


The Role of Lipids and Lipoproteins in Atherosclerosis (2019) https://www.ncbi.nlm.nih.gov/books/NBK343489/

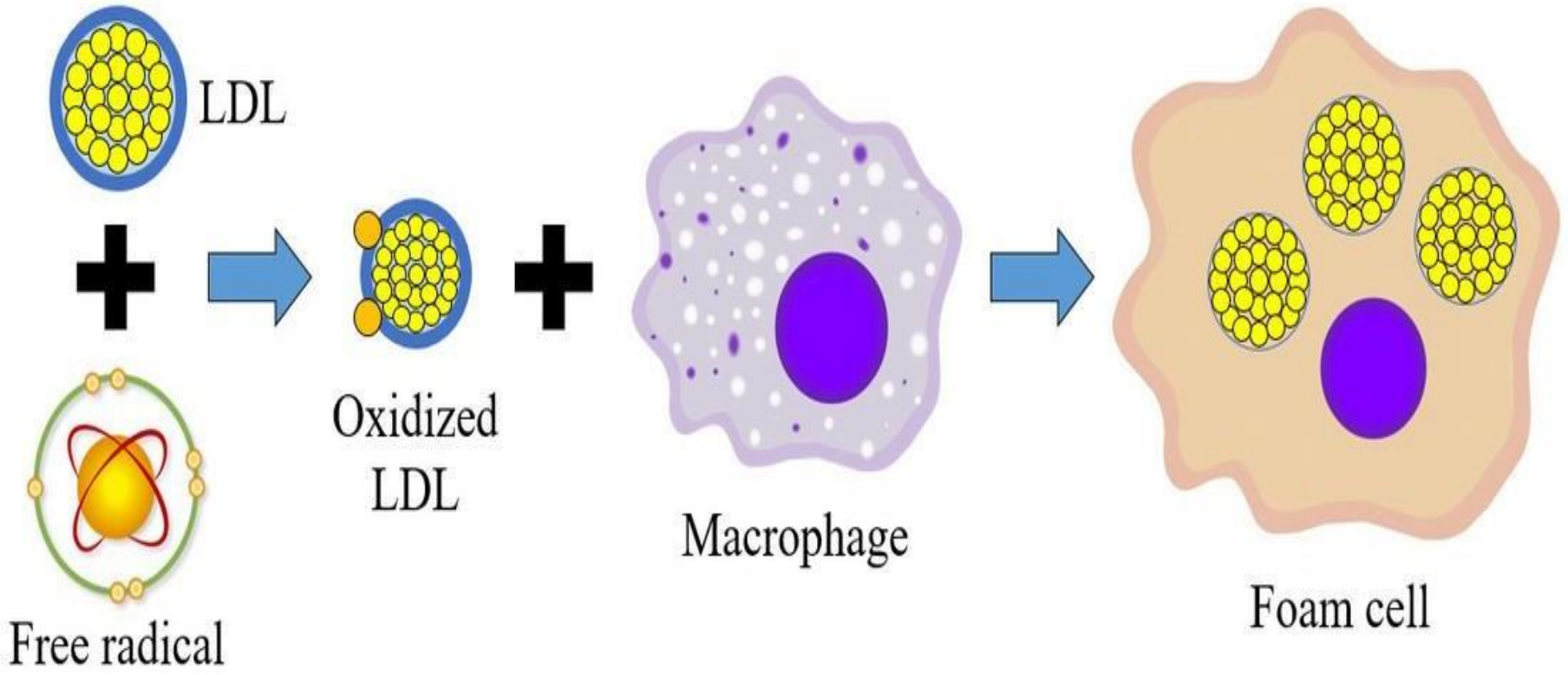
- **Arterial injury (denuding)** causes endothelial dysfunction promoting modification of apoB containing lipoproteins (LDL-cholesterol) and **infiltration of monocytes into the subendothelial space**.

- **Internalization of the apoB containing lipoproteins (LDL-cholesterol) by macrophages promotes foam cell formation**, which is the hallmark of the fatty streak phase of atherosclerosis.

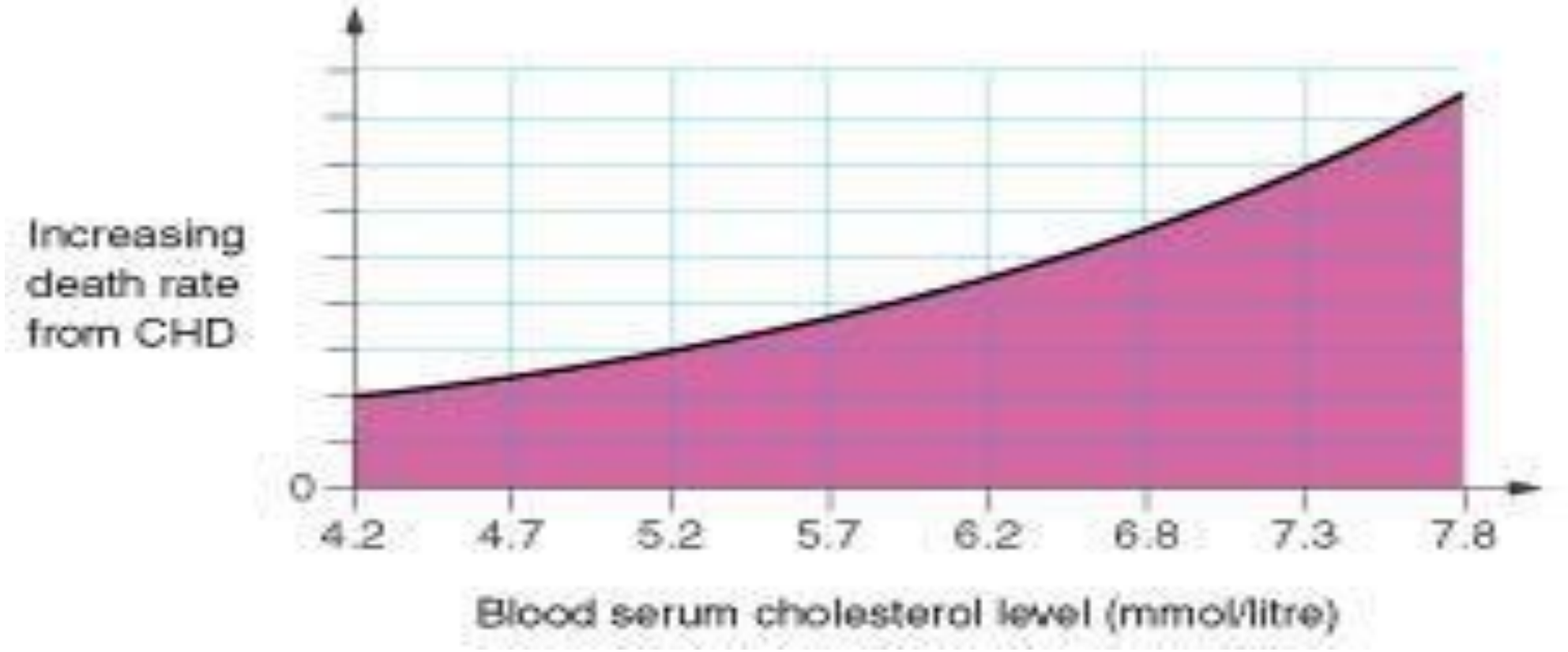
- **Macrophage inflammation** results in enhanced **oxidative stress and cytokine/chemokine secretion**, **causing more LDL/remnant oxidation, endothelial cell activation, monocyte recruitment, and foam cell formation**.



Cholesterol Contained Within Foam Cells in Atherosclerotic Plaque: Scavenger LDL Receptor on Macrophages



Blood Cholesterol is not the only risk factor, but it's a Cardinal Risk Factor
Goal: Total Cholesterol - 3.9 mmol/L or lower
Next best target is 4.7 mmol/L
LDL-Cholesterol – Goal is under 2.0 mmol/L or 1.8 mmol/L (or lower)



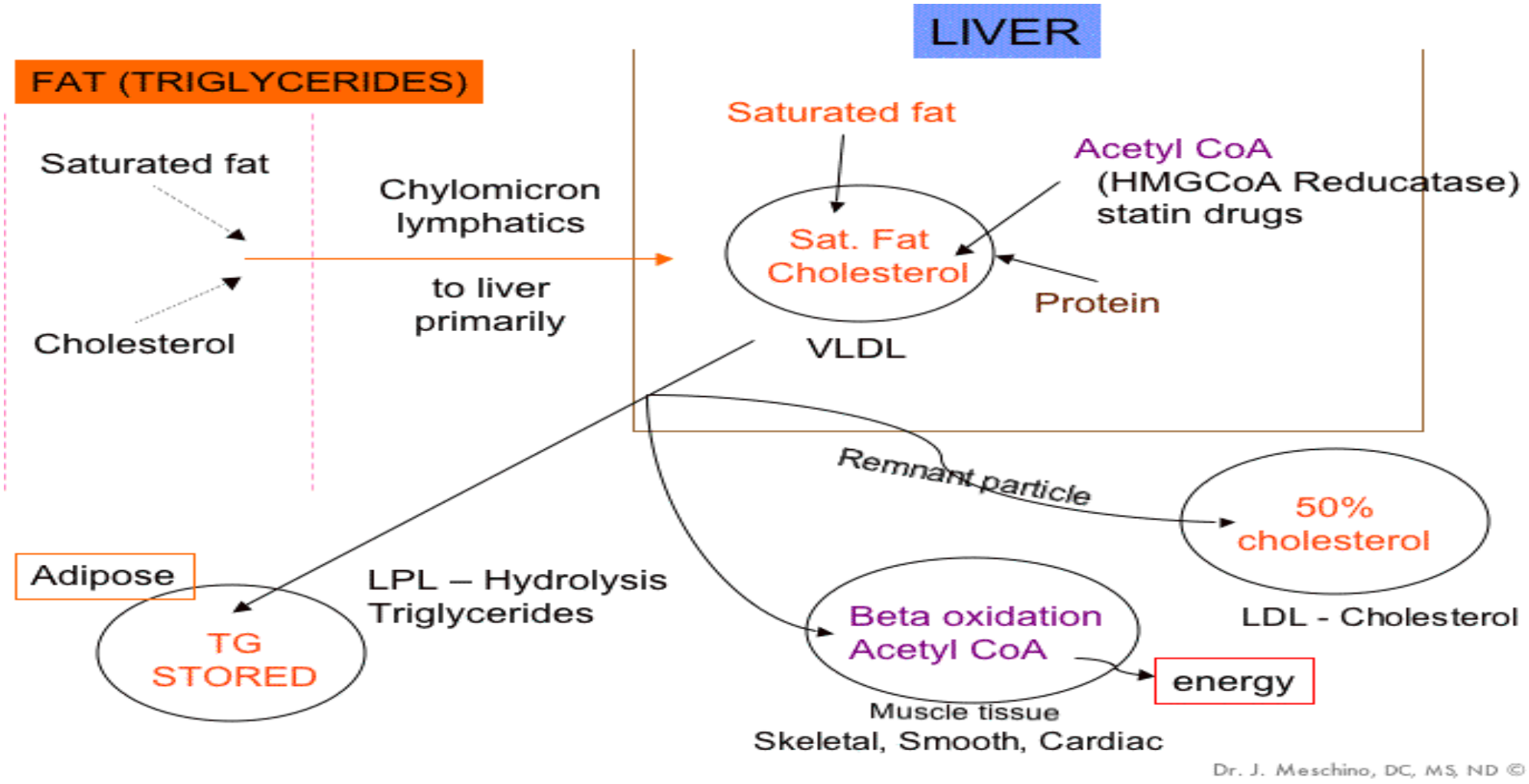
What is Total Cholesterol?

Total Cholesterol = LDL-cholesterol + HDL-cholesterol + VLDL+ Chylomicrons + IDL

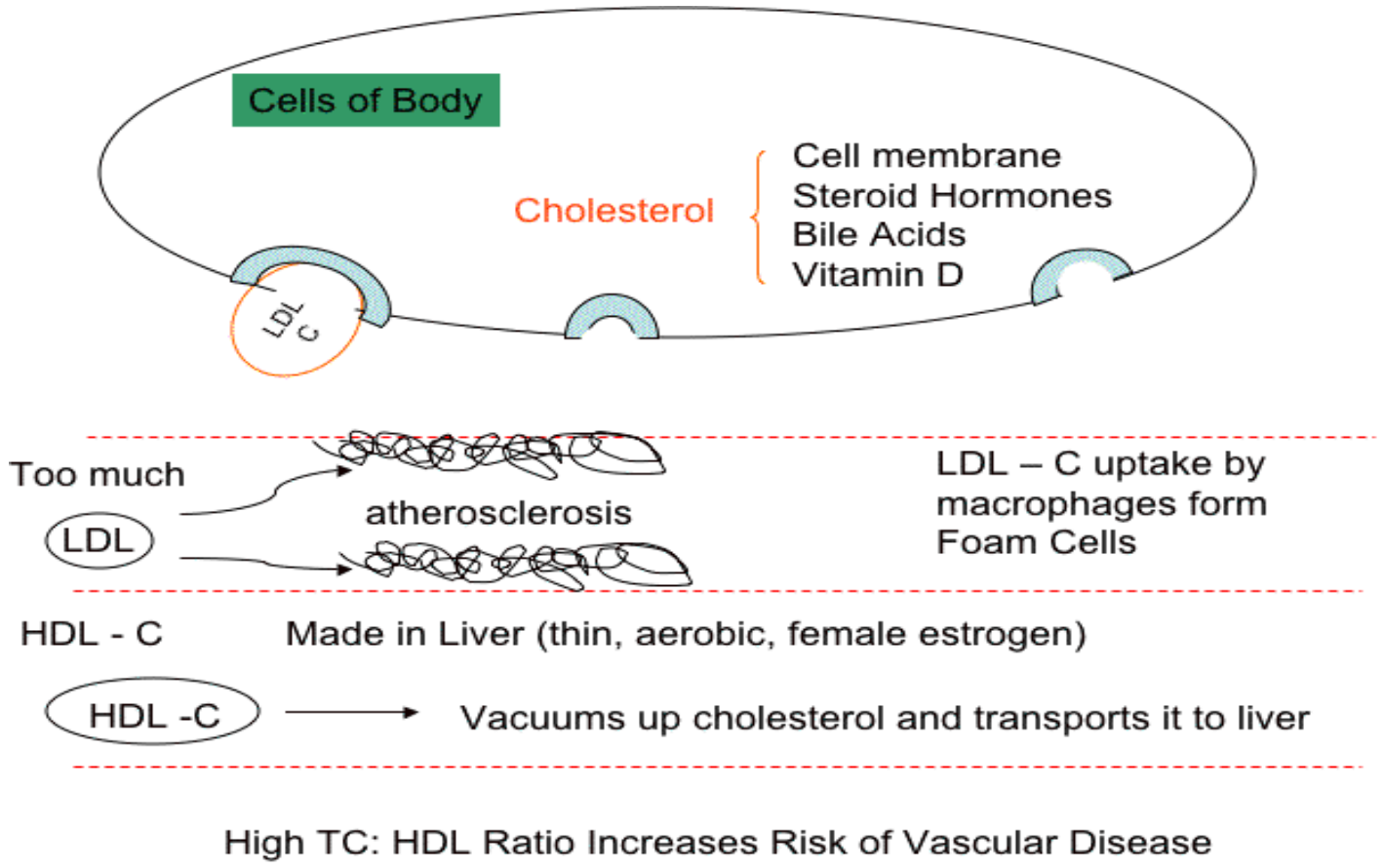
HDL-cholesterol – does reverse cholesterol transport, removing cholesterol from artery wall and bringing it back to the liver, where it can be converted into bile acids and excreted by the body via the fecal route.

Thus, a TC: HDL ratio of 3:1 or lower helps to reduce risk of CVD

Where does the Plaque Come From?
Quick Overview of Lipoprotein Physiology

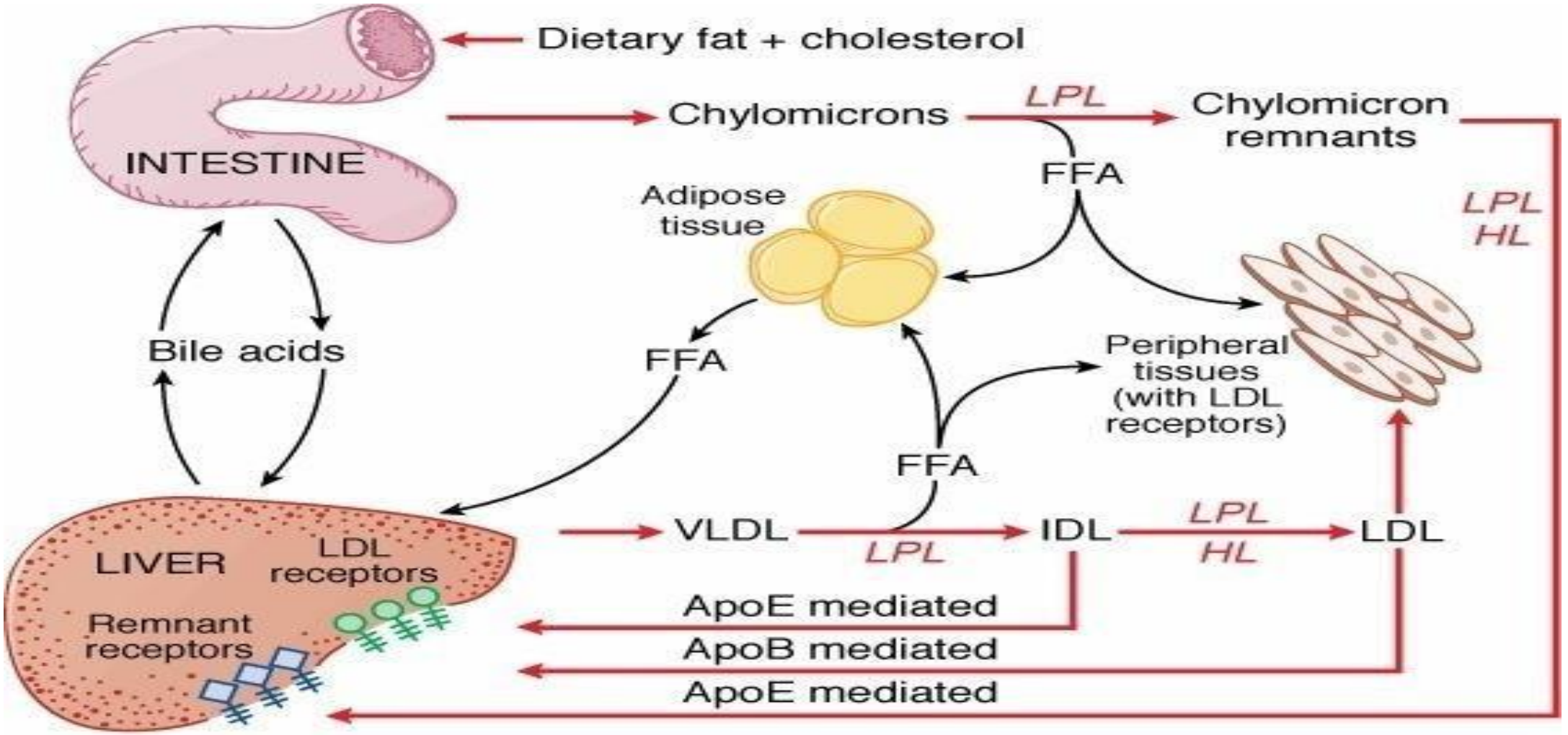


LDL-Receptor Function: Polyunsaturated and Monounsaturated Fats Increase LDL-Receptors on Liver Cells to Clear LDL from Circulation Very Effectively. Thus, lower LDL if substitute PUFA and MUFA for LCSF Foods



High TC: HDL Ratio Increases Risk of Vascular Disease

What Every Doctor Should Know About Lipoprotein Physiology:
This requires 5 minutes of intense concentration.



Summary of Apo-Proteins and Lipoprotein Physiology

- **HDL** – synthesized in the liver, donates Apo CII and Apo E to chylomicrons (chylom) and VLDL
- **Apo CII** activates LPL to clear triglycerides from chylom & VLDL by fat and muscle cells
- **Apo E** activates LDL receptors on liver cells to degrade remnant chylom and VLDL clearing them from circulation
- **VLDL**- synthesized in the liver, contains Apo-B100 and obtains Apo E and Apo CII from HDL. As triglycerides are cleared from VLDL via help from Apo CII, some VLDL becomes IDL, which can be cleared by the liver due to presence of Apo E.

- The remnant particle still remaining in the circulation becomes LDL

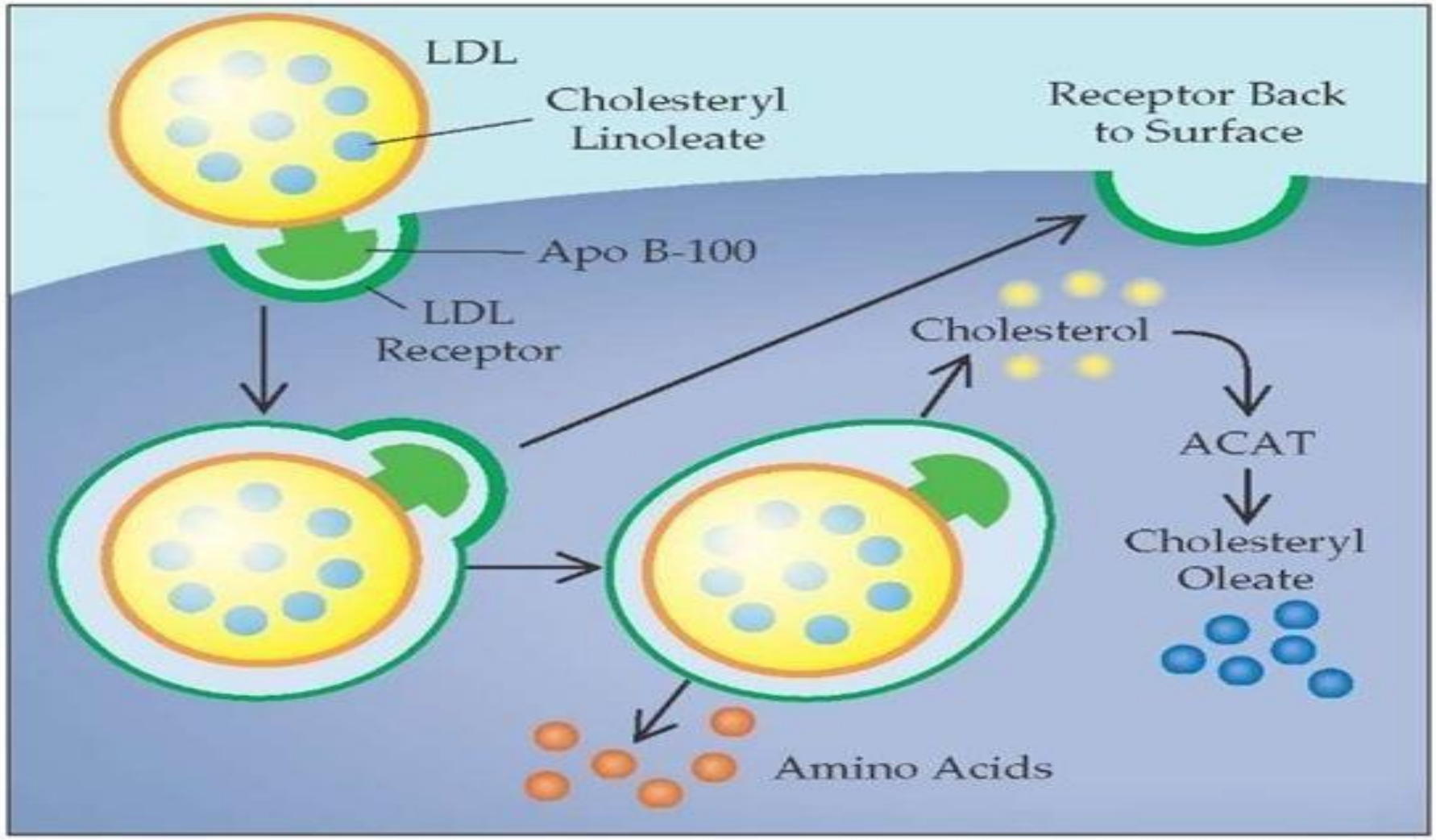
LDL - primarily due to the presence of Apo-B100 (inherited from VLDL), enables LDL to bind tightly to LDL receptors on virtually all body tissues (but can't cross blood-brain barrier) to be degraded and the cholesterol is used to make:

- Cell membrane – virtually all cells with exception of brain cells (Some HDL carries cholesterol into brain)
- Steroid hormones – adrenal, testes ovaries
- Vitamin D- in the skin upon exposure to UV light
- Bile Acids – in the liver

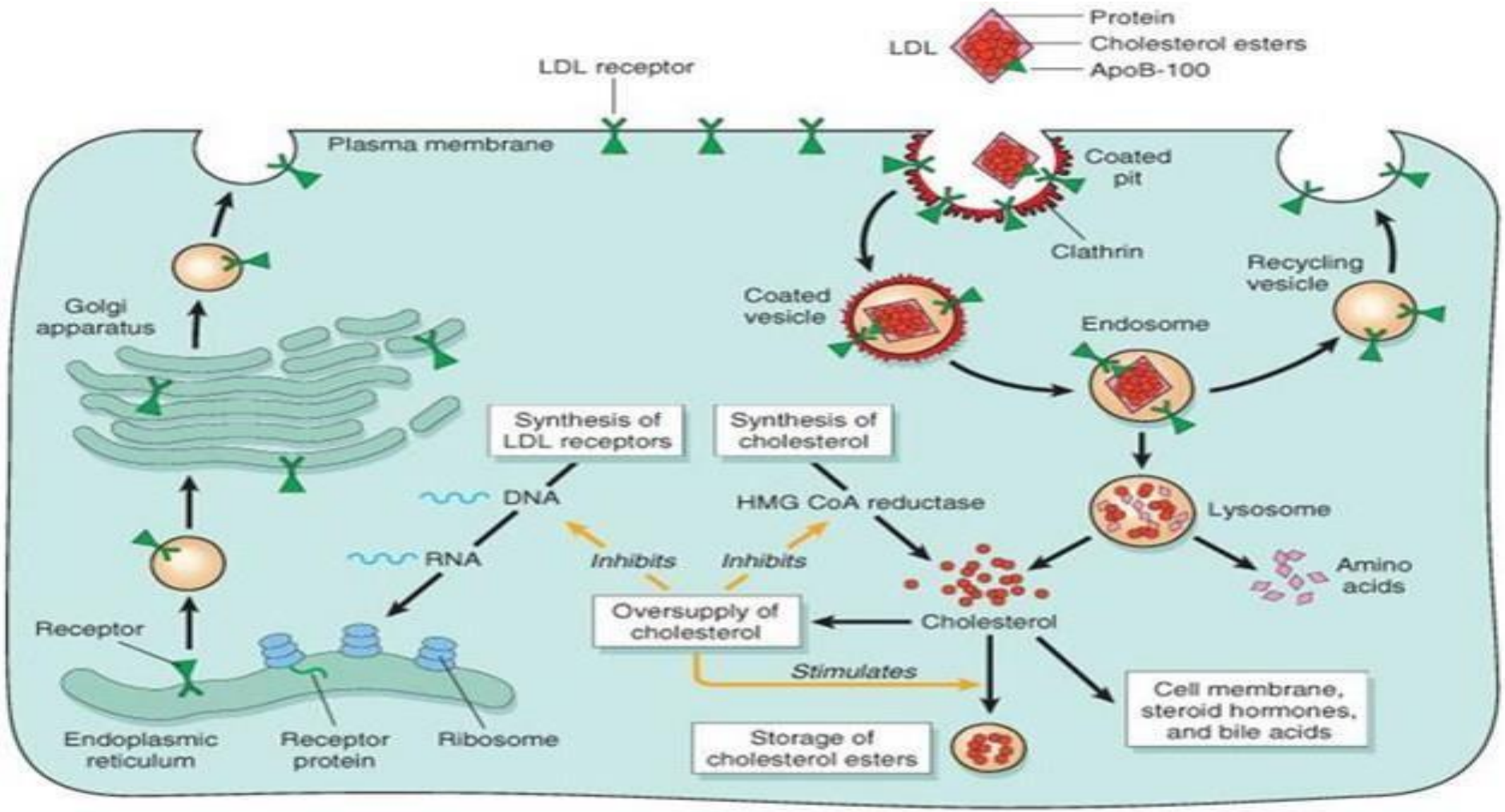
HDL – synthesized in the liver, does reverse cholesterol transport, but requires Apo A1 (ABCA1 gene). If defective get Tangiere's Disease. HDL can cross blood-brain-barrier to bring some cholesterol and phospholipids to brain, but the brain makes much of its own cholesterol from Acetyl-CoA (blocked by statin drugs, which can lead to memory loss)

Reference: Cholesterol Metabolism and the Blood Lipoproteins
Basic Medical Biochemistry: A Clinical Approach (Mark D, Marks A. Smith CM. Williams & Wilkins-publishers)

Apo B-100 Fits into LDL Receptor: Allows LDL to be Cleared from Circulation



LDL-Receptor, Coated Pit and Feedback Inhibition of LDL-Receptor Synthesis

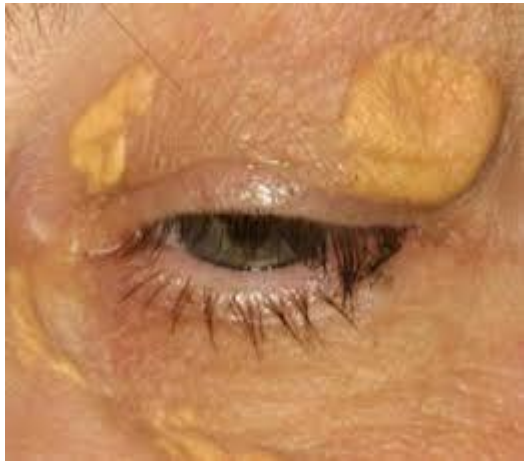


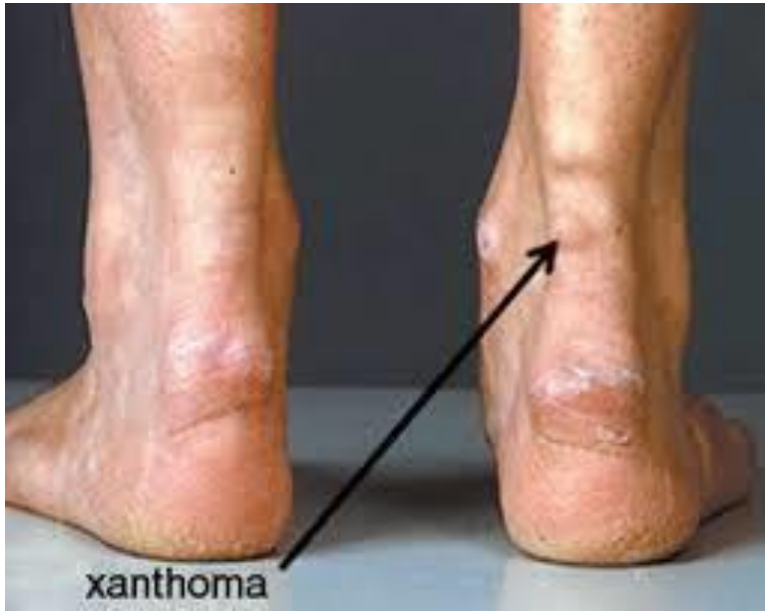
Genetic Defects Increasing Cholesterol, Triglycerides and CVD Risk

GENETIC DISORDER	PROTEIN (GENE) DEFECT	LIPOPROTEINS ELEVATED	CLINICAL FINDINGS	GENETIC TRANSMISSION	ESTIMATED INCIDENCE
Lipoprotein lipase deficiency	LPL (*LPL*)	Chylomicrons	Eruptive xanthomas, hepatosplenomegaly, pancreatitis	AR	1/1,000,000
Familial apolipoprotein C-II deficiency	ApoC-II (*APOC2*)	Chylomicrons	Eruptive xanthomas, hepatosplenomegaly, pancreatitis	AR	<1/1,000,000
ApoA-V deficiency	ApoA-V (*APOA5*)	Chylomicrons, VLDL	Eruptive xanthomas, hepatosplenomegaly, pancreatitis	AD	<1/1,000,000
GPIHBP1 deficiency	*GDIHBP1*	Chylomicrons	Eruptive xanthomas, pancreatitis	AD	<1/1,000,000
Familial hepatic lipase deficiency	Hepatic lipase (*LIPC*)	VLDL remnants	Pancreatitis, CHD	AR	<1/1,000,000
Familial dysbetalipoproteinemia	ApoE (*APOE*)	Chylomicron and VLDL remnants	Palmar and tuberoeruptive xanthomas, CHD, PVD	AR AD	1/10,000
Familial hypercholesterolemia	LDL receptor (*LDLR*)	LDL	Tendon xanthomas, CHD	AD	1/500
Familial defective apoB-100	ApoB-100 (*APOB*)	LDL	Tendon xanthomas, CHD	AD	<1/1000
Autosomal dominant hypercholesterolemia	PCSK9 (*PCSK9*)	LDL	Tendon xanthomas, CHD	AD	<1/1,000,000
Autosomal recessive hypercholesterolemia	*LDLRAP*	LDL	Tendon xanthomas, CHD	AR	<1/1,000,000
Sitosterolemia	*ABCG5 or ABCG8*	LDL	Tendon xanthomas, CHD	AR	<1/1,000,000

Abbreviations: AD, autosomal dominant; AR, autosomal recessive; ARH, autosomal recessive hypercholesterolemia; CHD, coronary heart disease; LDL, low-density lipoprotein; LPL, lipoprotein lipase; PVD, peripheral vascular disease; VLDL, very-low density lipoprotein.

Xanthomas: Related to High Cholesterol/Triglycerides/Glucose





Synthesis and Mutations of LDL-Receptors

LDL receptors are translated by ribosomes on the endoplasmic reticulum and are modified by the Golgi apparatus before travelling in vesicles to the cell surface.

Mutations

- Mutations in the gene encoding the LDL receptor are known to cause familial hypercholesterolaemia.
- **There are 5 broad classes of mutation of the LDL receptor:**

Class 1 mutations affect the **synthesis of the receptor in the endoplasmic reticulum (ER).**

Class 2 mutations **prevent proper transport to the Golgi body** needed for modifications to the receptor.

- e.g. a truncation of the receptor protein at residue number 660 leads to domains 3,4 and 5 of the EGF precursor domain being missing. This precludes the movement of the receptor from the ER to the Golgi, and leads to degradation of the receptor protein.

Class 3 mutations **stop the binding of LDL to the receptor**.

- e.g. repeat 6 of the ligand binding domain (N-terminal, extracellular fluid) is deleted.

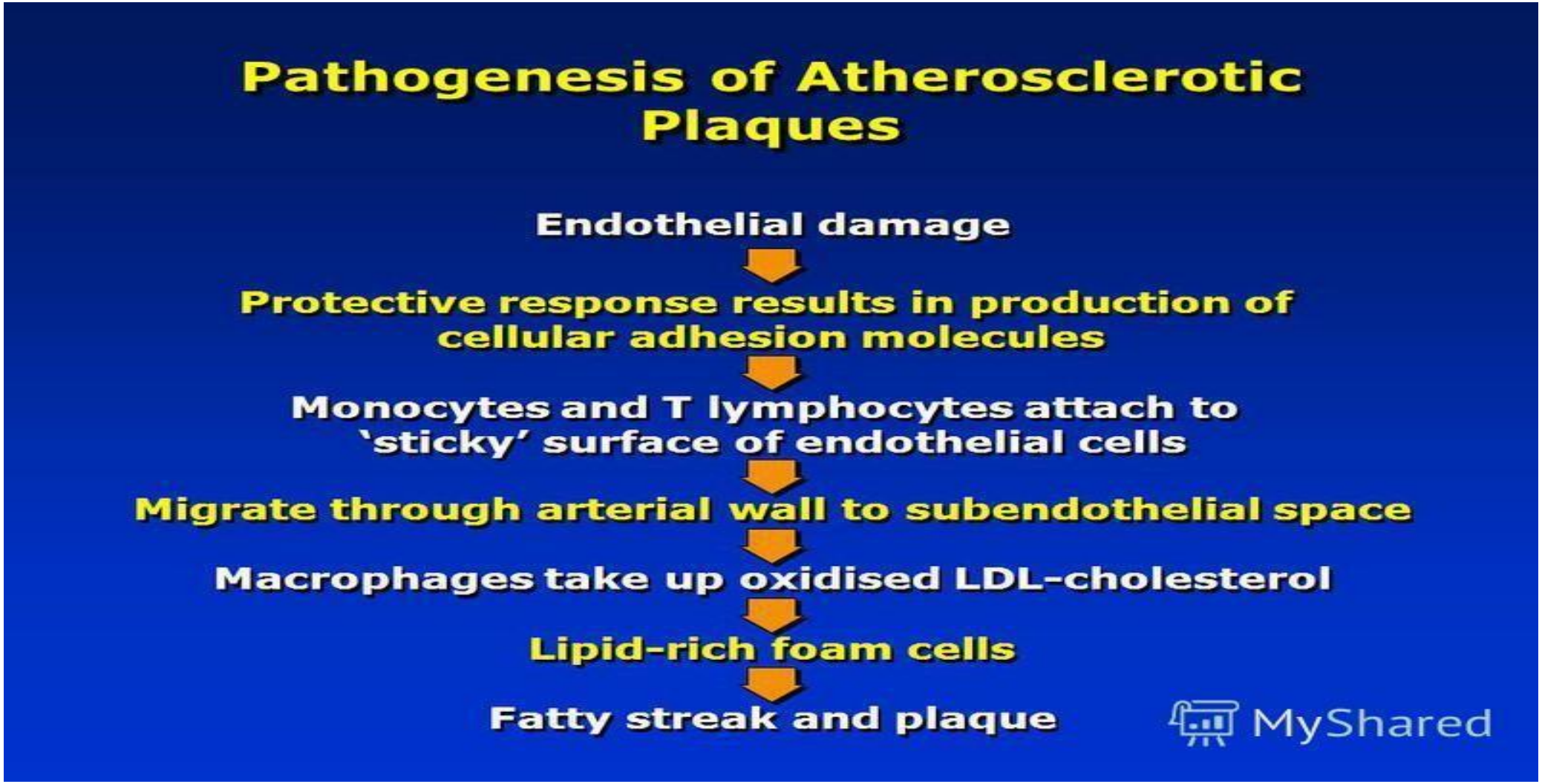
Class 4 mutations inhibit the internalisation of the receptor-ligand complex.

- e.g. "JD" mutant results from a single point mutation in the NPVY domain (C-terminal, cytosolic; Y residue converted to a C, residue number 807). This domain recruits clathrin and other proteins responsible for the endocytosis of LDL, therefore this mutation inhibits LDL internalization.

Class 5 mutations give rise **to receptors that cannot recycle properly**. This leads to a relatively mild phenotype as receptors are still present on the cell surface (but all must be newly synthesised).

People with significant LDL Receptor genetic defects NEED STATIN DRUGS TO LOWER THEIR CHOLESTEROL

The View From 30,000 Feet



Lifestyle Prevention of High Cholesterol: Lifestyle Can Keep Cholesterol in Ideal Range Without Need For Drugs In Up To 90% of Cases. How? Avoid Foods High In LC Saturated Fats/Trans-fats, Deep-fried Foods and/or Total Cholesterol

1. Beef, Pork, Lamb, Duck
2. Organ Meats (high in cholesterol)
3. Milk or Yogurt (2% or higher)
4. Cheese (4% or higher), Butter, Cream, Ice Cream, Whipping Cream etc.
5. Regular Chocolate Products (Cocoa butter)
6. Palm and **Coconut Oil**
7. High-Fat Pastries
8. Egg Yolks (250 mg of cholesterol per yolk)
9. Mayonnaise, Hollandaise Sauce, Creamy Salad Dressings etc. (use olive, canola, flaxseed oils)
10. Deep Fried Foods (French fries, potato chips, croutons, sweet potato fries, nachos, etc), as well as Breaded-Battered Meats, Fish and Poultry
11. Cream Sauces or Rose Sauces

More Specifically

- Most cakes – **other than Angel Food Cake.** The **frosting** of cake is especially loaded with saturated fat

•Donuts and many other pastries (Cruller, Danish pastry, cream puffs, chocolate eclair, pie and/or pie crust, any product with milk chocolate- chocolate bar).

- Most Muffins have more than 2 gm of saturated fat. So, check the label or the company's nutrition guide.
- Many types of biscuits
- Pancakes – depending on how they are made
- French toast
- All Beef and beef products – red meat
- All Pork and pork products
- Lamb
- Organ Meats
- Butter and Lard
- Cream and Ice cream
- Any milk of yogurt above 1% milk fat
- Any cheese above 3% milk fat
- Foods containing Palm oil
- Coconut oil and foods that contain them, including some Plant-based meat alternative products (read labels)

- Shortening
- Also, some breakfast cereals (read the package)
- Potato chips, cheeesies and the like (look for lower fat alternatives – Crispy minis, Skinny Pop Popcorn)
- Some prepackaged popcorn and popcorn you buy at the movies

•A low saturated fat diet also implies no cream sauces, no tahini sauce or creamy salad dressings, or the consumption of mayonnaise and avoid deep-fried, pan-fried and breaded or battered foods as a rule

There is a bit of fat in some low-fat protein foods, such as chicken breast, turkey breast, Cornish hen, and soy products. There is virtually no fat in egg whites, beans, peas and whey protein products, which are also great sources of protein.

Healthier fats, to use in small quantities include:

- Olive Oil and other oils high monounsaturated fat
- Avocado
- Fish and fish oil (1,000 – 3,000 mg per day)
- Flaxseed oil
- Nuts and olives (a handful of nuts per day and easy on the olives)
- Peanut butter
- Hummus- chick peas and sesame seed oil, olive oil, garlic, lemon juice and salt.

How To Lower Cholesterol: Desirable Foods

1. If no major family history of CVD, genetic predisposition or atherosclerosis risk factors then it's considered safe to consume these protein foods if you like:

- Chicken Breast (skin removed)
- Turkey Breast (skin removed)
- Cornish Hen
- Fish – no more than twice a week
- Egg Whites
- Milk, Yogurt, Kefir Milk – 0 or 1% Milk Fat (MF)
- Cheese under 4% Milk Fat (MF)
- Whey Protein or other Protein Shakes Mixes (Pea, Soy, Casein)
- Soy-based Protein Foods (TVP, Ground Round, Veggie Burgers-Dogs, Tofu, Edamame, Miso, Tempeh, etc.) – **soy products have independent cholesterol-lowering effects – blocking cholesterol and bile acid absorptio**n from gut
- Beans and Peas

2. Increase Soluble Fiber:

- Peas and Beans (also soy and soybeans) - chick peas, kidney beans etc. (legumes)
- Ground Flaxseed (1 -2 heaping tablespoons)
- Oat bran (1/3 cup)
- Many fruit and vegetables – high in pectin and fiber
- Psyllium Husk Fiber (1-3 teaspoons)

4.Artichokes – multi-modal effects on lowering cholesterol and glucose

5. Almonds (small handful)

Lipid Lowering Drugs

1. Statin Drugs – most common - inhibit liver cholesterol synthesis- blocking release of fat from the liver – fatty liver degeneration – steatohepatitis, memory loss, muscle pain
2. Bile Acid Sequestrants – bind to bile acids and cholesterol in GI tract, inhibiting absorption
3. Fibric Acid Derivatives – lower triglycerides more than cholesterol and raise HDL
4. High Dose Niacin – lowers triglycerides more than cholesterol and raises HDL to the greatest degree

All of these drugs can produce serious side effects – beyond the scope of this webinar to discuss.

Also, Cholesterol-Lowering Supplements (Gum Guggul and Artichoke Extract)

Gum Guggul

- Resin derived from the Mukul Myrrh tree
- Used extensively in Indian Medical System -1986 met government approval (India)
- Increases LDL clearance of cholesterol by the liver
- TC Reductions- 14-27% in 4-12 weeks
- Triglyceride Reductions – 22-30% in 4-12 weeks
- Improves HDL:LDL Ratio
- Non-toxic

Gum Guggul 2007 Review Paper: Cardiovascular Drug Reviews https://onlinelibrary.wiley.com/doi/full/10.1111/j.1527-3466.2007.00023.x

- In 1986, with proven efficacy and safety, guggul was approved for marketing in India as a **hypolipidemic drug.** Overall - **70-80%** of people show good response to lipid lowering effects of gum guggul - **20-30%** of people do not.

- Increases conversion of cholesterol to bile acids and subsequent excretion via fecal route - a major pathway to remove excessive cholesterol from the body

Experimental Research shows that:

- LDL-Cholesterol Catabolism – Guggulsterone has been shown to increase uptake of LDL-cholesterol by hepatic cells

- FXR Antagonist - Normally high concentrations of bile acids in the liver inhibit the conversion of cholesterol to bile acids, by inhibiting the cholesterol 7α-hydroxylase (CYP7A1) enzyme, which converts cholesterol to bile acids.

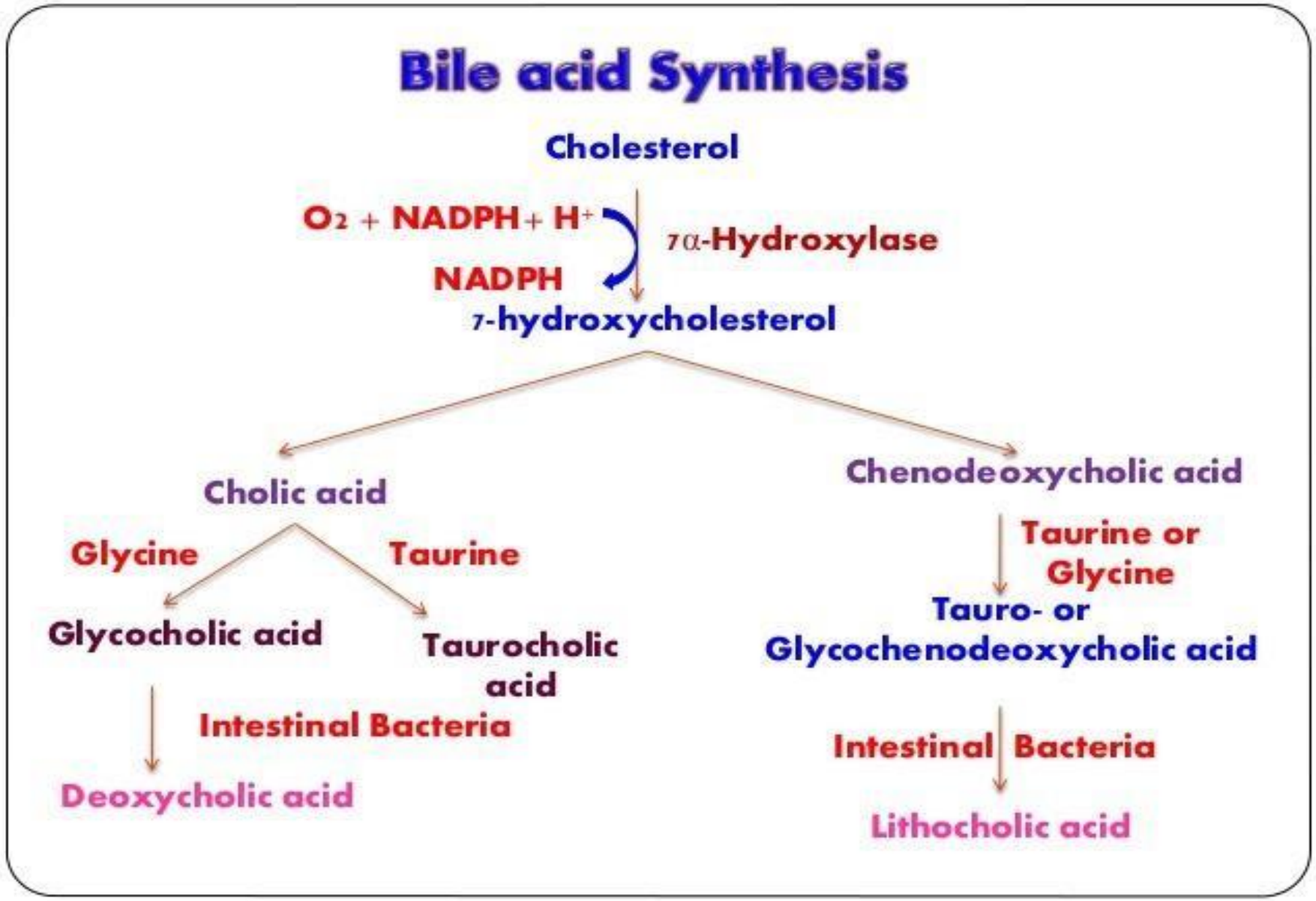
- The cholesterol 7α-hydroxylase (CYP7A1) is the rate-limiting enzyme in the classic pathway of bile acid synthesis from cholesterol in the liver

- BSEP Agonist - Guggulsterone upregulates the bile salt export pump (BSEP), an efflux transporter responsible for removal of cholesterol metabolites, bile acids from the liver. Such upregulation of BSEP expression by guggulsterone favors cholesterol metabolism into bile acids. **BSEP also increases secretion of bile acid into intestinal tract. Studies show increase (57%) in fecal excretion of bile acids**

- **Decreased LDL-cholesterol Oxidation** – Guggulsterone shown to decrease LDL-cholesterol oxidation, making LDL-cholesterol less atherogenic

- **Increases HDL-cholesterol** in approximately 60% of high cholesterol subjects

- Guggulsterone has been found to potently **inhibit the activation of nuclear factor-kappaB (NF-kappaB),** a critical regulator of inflammatory responses. Such repression of NF-kappaB activation by guggulsterone has been proposed as a mechanism of the anti-inflammatory effect of guggulsterone, which may account for its historical use in the treatment of Rheumatism.

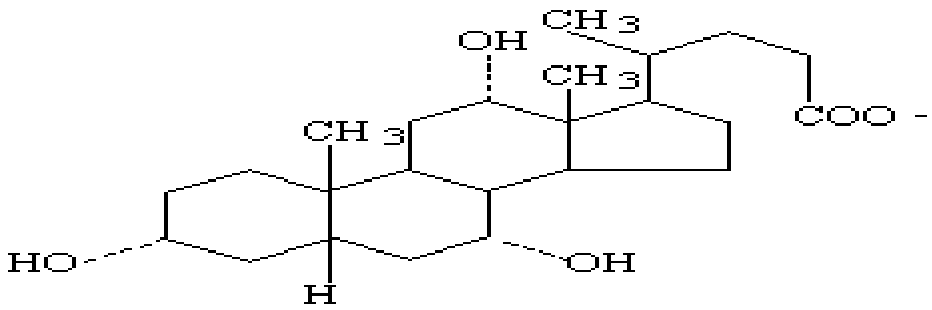

Bile acid Synthesis
Cholesterol
O2 + NADPH + H+
NADPH
7α-Hydroxylase
7-hydroxycholesterol
Cholic acid
Glycine
Taurine
Glycocholic acid
Taurocholic acid
Intestinal Bacteria
Deoxycholic acid
Chenodeoxycholic acid
Taurine or Glycine
Tauro- or Glycochenodeoxycholic acid
Intestinal Bacteria
Lithocholic acid

GUGULIPID®
ENHANCES

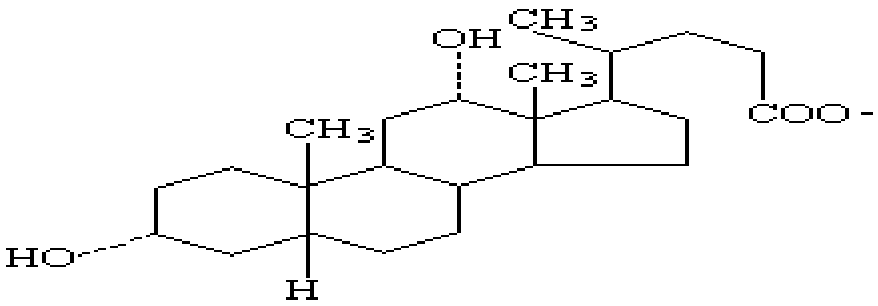
CHOLESTEROL

EXCRETION

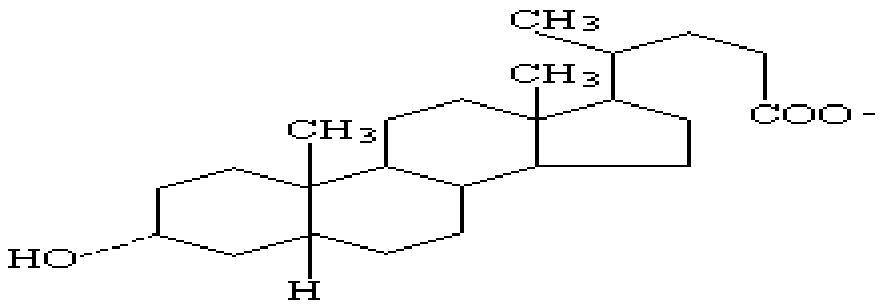
BILE SALTS (liver)



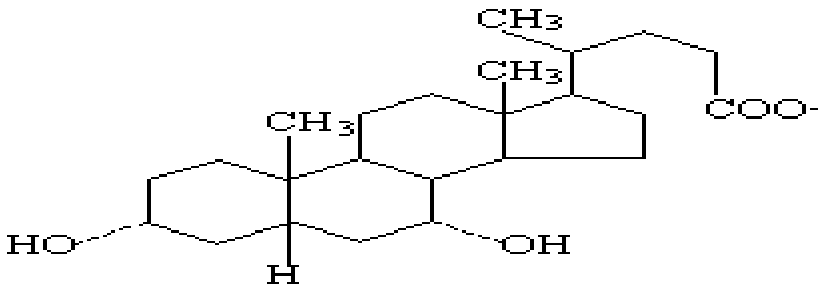
Cholate



Deoxycholate



Lithocholate



Chenodeoxycholate

Adverse Side Effects of Gum Guggul

- No significant side effects have been observed on renal and liver functions, hematological parameters, and electrolytes
- Such a safety profile is consistent with its long history of use in Ayurvedic medicine and practice.
- However, some adverse reactions have been noted in clinical trials. **Gastrointestinal discomfort** is the predominant side effect reported. Other side effects included loose stools, mild nausea, and hiccup.
- Skin rashes or hypersensitivity reactions have been reported in some clinical studies. In one study**, 9% of participants developed moderate to severe adverse cutaneous reactions within 48 hours** of the initiation of the therapy.
- In addition, a case of rhabdomyolysis was reported that might have been associated with the use of gum guggul.

Drug-Interactions and Precautions

- Co-administration of gugulipid with the β-blocker propranolol or calcium channel blocker diltiazem resulted in significant decrease in bioavailability of the two drugs. Such drug interaction is likely to be due to the activation of PXR by guggulsterone (leading to upregulation of the enzymes responsible for biotransformation of propranolol and diltiazem). **So, don't take Gum Guggul at same time as other medications.**
- Finally, safety in young children, pregnant or nursing women, or those with severe liver or kidney disease has not been established.

Reference:

Deng R.Therapeutic effects of guggul and its constituent guggulsterone: cardiovascular benefits**.** Volume 25, Issue 4 Winter 2007. Pages 375–390
http://onlinelibrary.wiley.com/doi/10.1111/j.1527-3466.2007.00023.x/full

Artichoke Extract

- In a double-blind, placebo-controlled study of 143 people with high cholesterol, artichoke leaf extract reduced total cholesterol by 18.5% as compared to 8.6% in the placebo group; LDL cholesterol dropped by 23% vs. 6%; and LDL-to-HDL ratios declined by 20% vs. 7%.

Englisch W, Beckers C, Unkauf M, et al. Efficacy of artichoke dry extract in patients with hyperlipoproteinemia. *Arzneimittelforschung*. 2000;50:260–265.

- Artichoke is known to **increase bile acid secretion by the liver**, which in turn, upregulates LDL-cholesterol receptor production in liver cell, clearing more LDL cholesterol from the blood stream. Hence artichoke enhances the excretion of excess cholesterol (in the form of bile) from the body via the fecal route

- Besides cynarin, a compound in artichoke called luteolin may play a role in reducing cholesterol.

More Artichoke Studies
(2016 – Journal of Food and Nutrition Research)

Study performed on **Diabetic Rats** showed:

Artichoke Head and Leaf ingestion administration:

1. Reduced Total Cholesterol and LDL by:
   - Converting cholesterol to bile in the liver and excreting it
   - Inhibiting cholesterol synthesis
   - Inhibiting Cholesterol absorption from gut

2. Antioxidant – which may prevent oxidation to LDL – making it less atherogenic

3. Increases liver protection against damaging agents as well

4. Diabetic rats also showed reduction in **blood sugar and triglycerides**

5 Major Protective Compounds in Artichoke:

1. Chlorogenic acid - antioxidant
2. Cynarin - antioxidant
3. Caffeoylquinic acid – cholesterol and triglyceride lowering effect
4. Luteolin – inhibits cholesterol synthesis
5. Apigenin - antioxidant

These 5 compounds appear to work synergistically to lower cholesterol and glucose.

Dietary Application

Including artichoke heads, hearts and/or leaves in your diet a 2-3 times per week may be wise, especially if wanting to:

- Lower cholesterol
- Reduce blood glucose
- Support liver health
- Boost antioxidant defense – one the richest sources of antioxidants of all foods
- Increase dietary fiber

Artichokes are low in calories, but filling and can be a healthy substitute for starchy carbs at a meal.

Add them to salads or bean salads

References:

- Magied M et al. Artichoke (*Cynara scolymus* L.) Leaves and Heads Extracts as Hypoglycemic and Hypocholesterolemic in Rats. Journal of Food and Nutrition Research. Vol. 4, No. 1, 2016, pp 60-68. http://pubs.sciepub.com/jfnr/4/1/10/index.html
- http://www.oceanmist.com/artichokes/12-unexpected-artichoke-health-benefits/

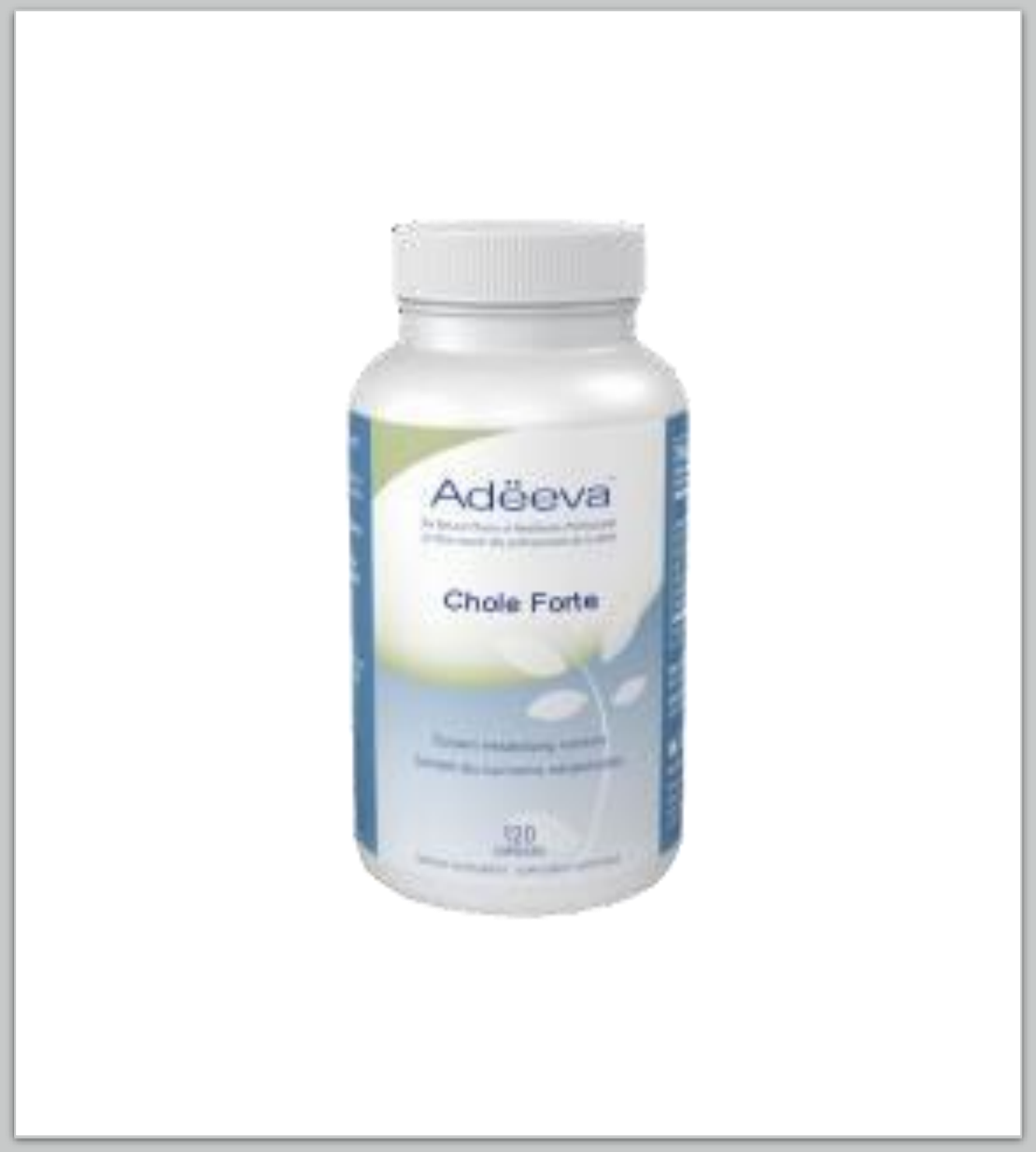
Natural Cholesterol Lowering Supplement
Example Adeeva Chole Forte

Each Capsule Contains:

1. **Gum Guggul –** 500 mg gum guggul, standardized to 2.5% guggulsterone content

2. **Artichoke** – Artichoke Leaf Extract- 200 mg, standardized to 13-18% caffeoylquinic acid content

Dosage: 3 caplets, twice daily with food.



Medscape Article: Lipids Management European Style: President Interviewed (European Atherosclerosis Study) July 14, 2016. Dr Catapano Interview

- Dr. Catapano re-emphasized that **high LDL-cholesterol is a main culprit in in risk of heart and cardiovascular disease** - a leading killer in our society and that **diet and lifestyle should be first line defense.**

- He makes the point that all randomized studies conducted to date show that the safest value for **LDL-cholesterol is under 70 mg/dl or 1.8 mmol/L (Canada).**

- The most recent 2016 findings from the Framingham Heart Study show that **men are more than twice as likely to die from Sudden Cardiac Death than women.**

- **Men ages 45- 70 roughly have a 10%** increased risk of dying from Sudden Cardiac Death, whereas for **women the risk is 3-3.5%.**

- **But those with CVD risk factors** like high LDL-cholesterol have a higher risk. And if one of the risk factors is high blood pressure then the risk becomes much higher.

References:

- Dr Catapano Medscape Interview: http://www.medscape.com/viewarticle/866069?nlid=108316_1842&src=WNL_mdplsfeat_160719_mscpedit_wir&uac=62515BJ&spon=17&impID=1159644&faf=1#vp_2\
- Sudden Cardiac Death Stats from Framingham Study Update 2016: http://www.medscape.com/viewarticle/865714

November 2016 - *British Medical Journal* (BMJ)

- A study published in the November 2016 issue of the *British Medical Journal* (BMJ) confirmed what many previous studies have shown over the years. **Saturated fat in the diet is associated with an increased risk of heart disease. Study followed over 115,000 male and female healthcare professionals**

- **The results showed there was an 18% greater risk of heart disease in the group consuming the highest amounts of saturated fats, compared with the group consuming the least.**

- **The study also showed that certain types of saturated fat were riskier than others. The three most important saturated fats with respect to heart disease risk included lauric acid, myrisitic acid and palmitic acid.**

- **Beef products are very high in palmitic acid. So are most pork products. Hot dogs, salami, bacon, sausages are also very high in this heart-disease promoting fat.**

- **High fat dairy products, like whole milk, cream, most cheeses, butter, even 2% milk, are very high in myristic acid and palmitic acid.**

- Coconut oil, which is very popular these days, and has a fat distribution that is **48% lauric acid, 16% myristic acid and 9.5% palmitic acid.** Coconut oil is almost entirely fat, and 73.5% of the fats are heart-disease promoting fats.

- When individuals replaced the fats from these foods with plant protein (like soy products, peas, beans etc.) or polyunsaturated fats, there was an 11-12% reduction in risk of developing heart disease.

- Other studies have clearly shown that lauric acid, myristic acid and palmitic acid raise cholesterol.

- **Coconut Oil studies in humans show that it raises LDL-cholesterol, when tested against other vegetable fats with lower saturated fat content (avocado, vegetable oils):** file:///H:/GIMA%20Course%20Notes%20Final/Course%20103%20Sections%207-10%20Metabolic%20Health/7.%20CVD/Additional%20pdf%20Course%20Notes%20-%20CVD/Coconut%20oil%20and%20CVD%20Risk.pdf

References:

Zong, G., Li, Y., Wanders, A.J., Alssema, M., Zock, P.L., Willett, W.C., Hu, F.B., Sun, Q. Intake of individual saturated fatty acids and risk of coronary heart disease in US men and women: two prospective longitudinal cohort studies *BMJ.* 2016;355:i5796. https://www.hsph.harvard.edu/nutritionsource/2016/12/19/saturated-fat-regardless-of-type-found-linked-with-increased-heart-disease-risk

Dr. Dean Ornish M.D. Interview: Author – Reversing Heart Disease

- **His program is for people with advanced stage heart disease** and so it is a bit more restrictive than general recommendations for non-high risk people. Program proven to **reverse atherosclerosis and shown on pre and post angiogram and other studies**.

- He precludes not only no beef, pork, high fat dairy products and vegetable oils, but also allows no chicken, turkey, and even fish**. So, its basically a vegan or plant-based diet**, **with a bit of non- fat dairy products and egg whites, and some nuts and avocado.** He **includes soy products as source of protein,** and to help prevent cancer and heart disease.

- As well, The Ornish Diet has been shown to help stabilize mild to moderate degree localized **prostate cancer in a clinical trial** with men, and it's shown to improve glucose, cholesterol and blood pressure in high-risk patients, including diabetics. And it also helps to reduce body fat in over weight patients.

- It's the best of all worlds and studies show that Dean Ornish has given us a blueprint for a very healthy diet and lifestyle program to strive for.

Reference: http://www.medscape.com/viewarticle/862903

24 Important Measurable Risk Factors For CVD Risk: Ideal Values Listed Below (see handout notes)

1. **High Total Cholesterol** (above 3.9 mmol/L;150 mg/dL)

2. **Hypertriglyceridemia** (above 1.5 mmol/L;132 mg/dL)

3**. Hyperhomocysteinemia** (above 6.3 umol/L; 0.85 mg/L)

4**. INR**_____(0.8 – 1.3)

Platelet stickiness (obese, sat fat, cigarettes, sedentary lifestyle.) – increased platelet adhesive index

5**. Hypertension** – (denuding of endothelium – endothelial dysfunction). Hypertension (BP at or above 130/80 mmHg or on antihypertensive medication increases risk)

Important Risk Factors Continued

6. **Cigarette smoking** – platelet stickiness, vasoconstriction, endothelial dysfunction and oxidation of LDL-C

7. **Low HDL cholesterol** – Men: increase risk below 1.17 mmol/L (45 mg/dL) Women: increase risk below 1.42 mmol/L (55 mg/dL)

8. **TC:HDL** ratio greater than 3.0 (upper limit of 3.9)

9. **Family history** of premature CHD (CHD in male first-degree relative <55 years; CHD in female first-degree relative <65 years)

10. **Age** (men over 45 years; women over 55 years)

11. **Physical inactivity** – poor cardiovascular fitness (low VO2 max)

12. **Waist Circumference** – related to dyslipidemia and insulin resistance (men above 36 inches and women above 33 inches) – central obesity is metabolically active

13. **Stress** (Dean Ornish Research) – cortisol, inflammation, blood pressure

Important Risk Factors Continued

14. Diabetes – increased propensity for atherosclerosis and kidney sclerosis leading to dialysis (must keep LDL below 1.8 mmol/L)

15. Fasting Glucose_____(ideal is under 90 mg/dl or 5 mmol/L – conversion factor is .055)

16. C-Reactive Protein _____(less than 0.24 mg/dL or 2.4 mg/L) – conversion factor is 10

17. Fibrinogen_____(less than 300 mg/dL or 0.88 umol/L - conversion factor is 0.0294)

18. Fructosamine -_____Normal Range: 205 – 285 umol/L

19. **Uric Acid**_____Men: 140 – 440 umol/L (2.4 – 7.4 mg/dL) Women: 80 – 350 umol/L (1.4 – 5.8 mg/dL) – endothelial dysfunction/hypertension, and platelet viscosity

Important Risk Factors Continued

20. **Metabolic Syndrome exists if patient meets three or more of the following criteria:**

- Abdominal obesity (waist circumference: men > 102 cm or 40 inches; women > 88 cm or 35 inches
- Triglycerides ≥ 1.7 mmol/L (150.5 mg/dl)
- HDL men < 1.0 mmol/L (40 mg/dl); women < 1.3 mmol/L (52 mg/dl)
- BP > 130/85 mm Hg
- Fasting glucose 5.7-6.9 mmol/L (103 -124mg/dl)

21. LDL cholesterol (above 2.0mmol/L , some argue - 2.5mmol/L)

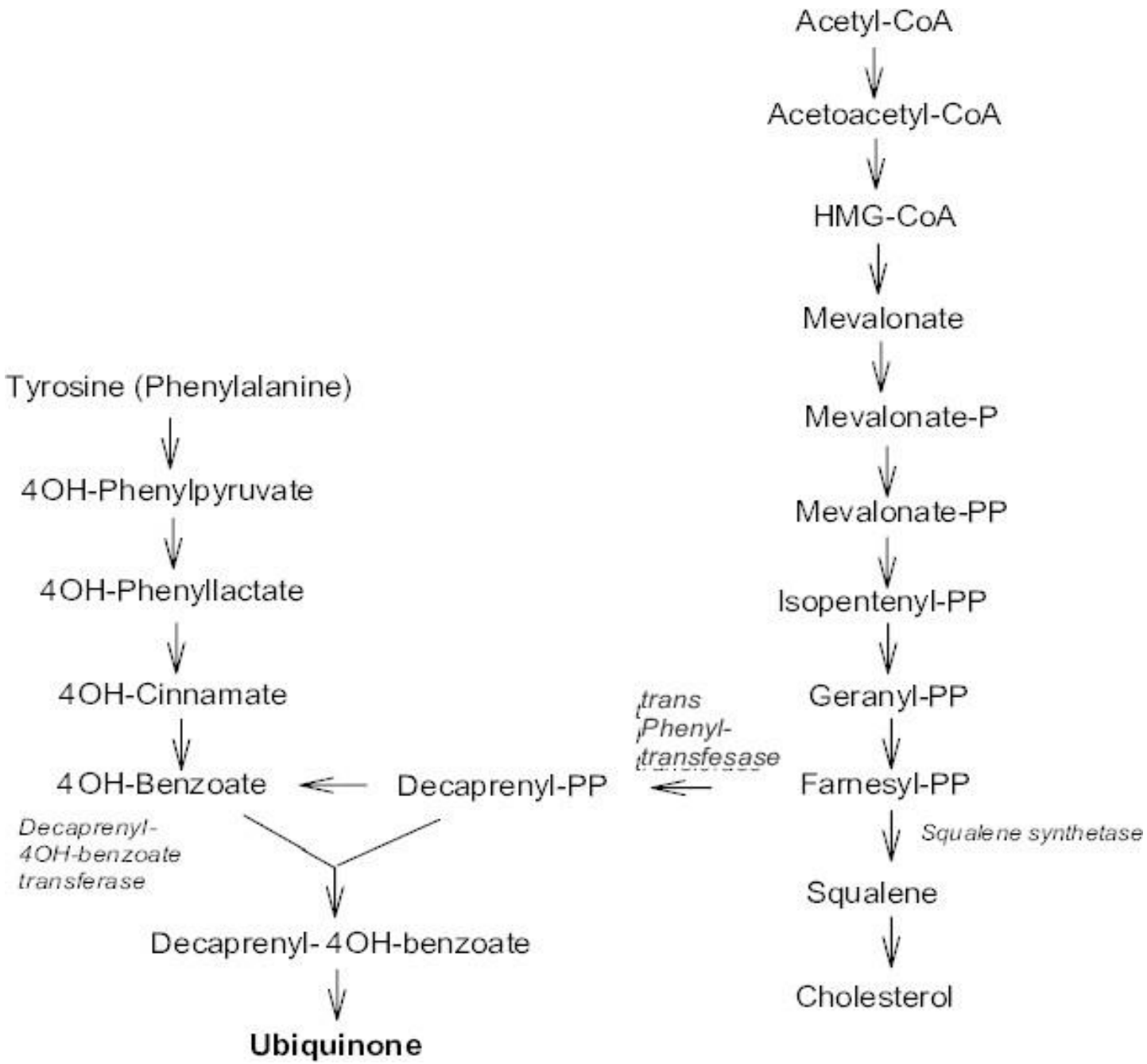
Other Considerations: - Kidney Disease

22.Blood Creatinine _____Normal is 0.6 to 1.2 mg/dl 60-110 µmol/L) in adult males and 0.5 to 1.1 mg/dl (45-90 µmol/L) in adult females

23. GFR (eGFR < 60 mL/min is a concern) _____

24.Positive CVD History - Heart Disease, Stroke, Deep Vein Thrombosis, Chronic Renal Disease or any vascular problems or procedures (angioplasty, by-pass surgery, coronary stent)

Statin Drugs are HMG CoA Reductase Inhibitors, but also Reduce CoQ10 Synthesis (Ubiquinone)



Reducing Statin Drug-Induced Muscle Pain

- Muscle aches and pain, occur in about 11% of patients using statin drugs (some reports suggest as high as 25%), with 4% of patients reporting very severe muscle pain.

- To make things worse, muscle pain related to the use of statin drugs is often made much worse or triggered by exercise. This is quite paradoxical in that exercise is also an important way to reduce risk of heart disease and stroke

- In recent years it has been discovered that taking three vitamin supplements, along with statin drug therapy, shows promise in reducing the drug's muscle pain side effects. **The three vitamins are Coenzyme Q10, Vitamin E and Vitamin D.**

- The reason these vitamins may help is because statin drugs not only block the synthesis of cholesterol in the body, but also block the synthesis of coenzyme Q10.

- **A decrease in coenzyme Q10 inhibits energy production in muscle cells, produces higher levels of free radicals that damage muscle cells, creates mitochondrial dysfunction and allows the muscle membrane to leak products out of the cell into the surrounding environment.**

- All these factors are associated with increased muscle damage and pain and are made worse when people exercise, as **exercise puts an even greater demand on the of the muscle's energy system and creates even more free radicals.**

- **Secondly, the increased free radicals that occur in the muscle from statin drug use causes an efflux or exiting of the amino acid glutamate out of the muscle.**

- **Glutamate stimulates pain nerve endings around the muscle (NMDA receptors), which also causes pain to occur.**

- Studies show that taking **vitamin E supplements** can **stop the muscle from shuttling glutamate out of the cell,** which prevents pain from occurring via this mechanism.

- Vitamin E supplementation gives muscle cells more antioxidant protection to quench or neutralize free radicals generated from statin drug use. **As such, muscles cells don't feel the need to pump glutamate out of the cell in the process of trying to synthesize glutathione** – an antioxidant cells make to help quench free radicals.

- Also, studies show that individuals with low or **sub-optimal vitamin D** status are more inclined to have **statin drug-induced muscle pain.**

- When these individuals get their **vitamin D blood level** up into the more ideal range **(over 75 nmol; 30 ng/ml)** through supplementation, they often can resume statin therapy without feeling muscle aches and pains.

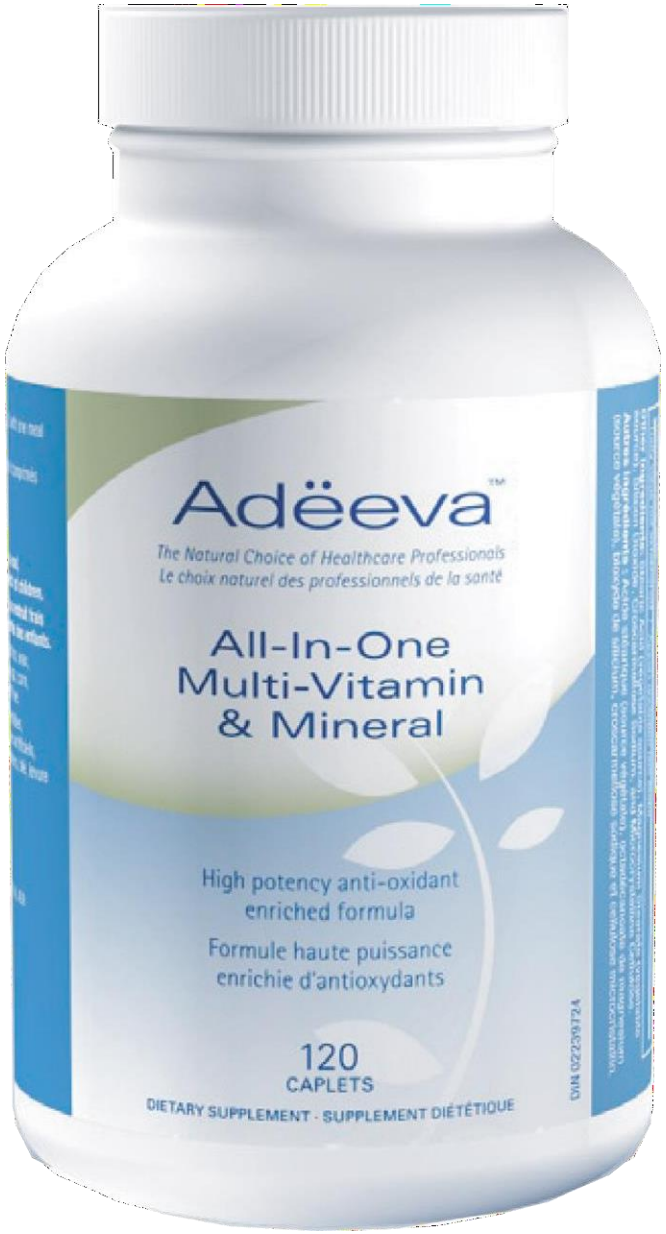
- Vitamin D has many positive effects on muscle function and integrity and so it stands to reason that adequate vitamin D can help maintain muscle cell integrity under the challenge of statin drug therapy.

- So, in patients prescribed a statin drug, it may be wise to take 90-200 mg per day of coenzyme Q10, 200-400 IU per day of vitamin E and to get blood vitamin D level checked.

- If blood vitamin D level is below 75 nmol/L (30 ng/ml) then it may be wise to take an extra 1000-2000 IU of vitamin D per day until blood vitamin D level is in the more desirable range.

Reference

Parker BA, et al. Effect of statin on skeletal muscle: Exercise, myopathy, and muscle outcomes. Exercise Sports Science Review Journal. 2012. 40(4):188-194 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3463373/



Adeeva Multiple Vitamin

Cardiovascular Features

1. Vitamin E – 400 IU
2. Vitamin D – 1,000 IU
3. B-50 complex (to lower homocysteine)
4. 500 mg calcium/200 mg magnesium (blood pressure control)
5. Selenium – 200 mcg (heart failure studies – along with CoQ10)

Adeeva High Potency Multiple Vitamin and Mineral

All-In-One Multi Vitamin and Mineral		
Vitamin A		2,500 I.U.
Beta Carotene		10,000 I.U.
Vitamin C		1,000 mg
Vitamin D		1000 I.U.
Vitamin E succinate		400 I.U. (natural)
Thiamin		50 mg
Riboflavin		50 mg
Niacin		50 mg
Vitamin B-6		50 mg
Folic Acid		400 mcg
Vitamin B-12		50 mcg
Biotin		300 mcg
Pantothenic Acid		50 mg

Adeeva High Potency Multiple Vitamin and Mineral Continued

All-In-One Multi Vitamin and Mineral Continued		
Calcium		500 mg
Iron		6 mg
Magnesium		200 mg
Zinc		15 mg
Selenium		200 mcg
Copper		2 mg
Manganese		5 mg
Chromium		50 mcg
Molybdenum		50 mcg
Bioflavonoids		50 mg
Lutein		6 mg
Lycopene		6 mg

Adeeva Cardio Essentials

Each Capsule Contains:

1. CoQ10 – 30 mg
2. Hawthorn – 37.5 mg (std grade of proanthocyanidins – i.e., vitexin)
3. Quercetin – 50 mg

To Keep the Heart Strong As You Age:

- By Age 45 – 1 capsule per day
- By Age 60 – 2-3 capsules per day
- **Statin Drugs – 3 capsules per day**
- High Blood Pressure – 2-3 capsules, twice daily

Natural Cholesterol Lowering Supplement
Example Adeeva Chole Forte

Each Capsule Contains:

1. **Gum Guggul –** 500 mg gum guggul, standardized to 2.5% guggulsterone content

2. **Artichoke** – Artichoke Leaf Extract- 200 mg, standardized to 13-18% caffeoylquinic acid content

Dosage: 3 caplets, twice daily with food.

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