Medical Committee
The University of Jordan

SLIDE SHEET

Sheet: 21

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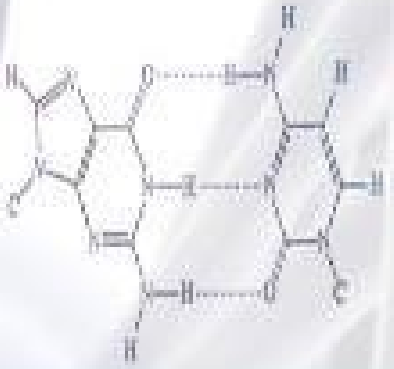
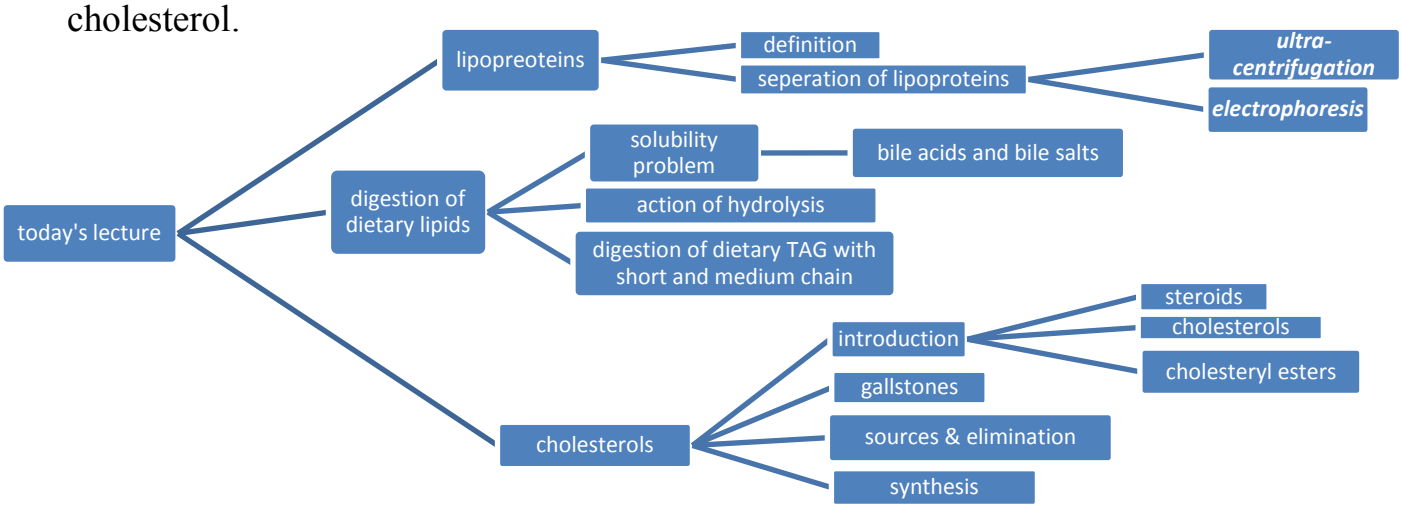
Biochemistry



Majida Al-Foqaraa'

Digestion and Transport of TAG by Plasma Lipoproteins & Cholesterol Metabolism

In today's lecture we are going to discuss two topics. Firstly, we are going to continue we have started in the previous lecture (lipoproteins), and we'll start talking about cholesterol.

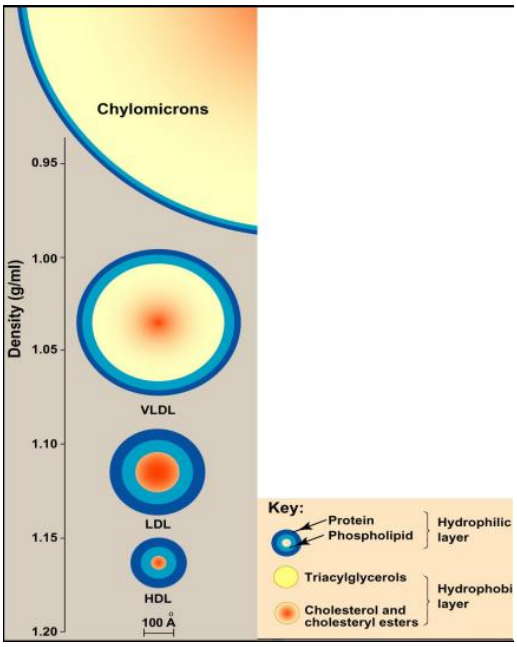


Lipoproteins

Lipoproteins are aggregates of large no. of molecules that aggregate together forming spherical particles that have *proteins* and *phospholipids* on their surface with *free cholesterol*, while the interior of the particle is filled with *non-polar (hydrophobic) lipids* like *TAG* and *cholesteryl ester*.

*** How can we separate these?!**

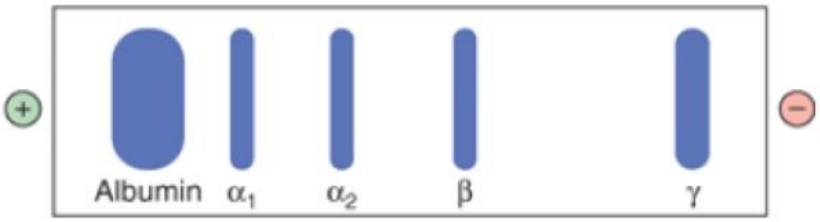
1. **Ultra-centrifugation**: They can be separated based on the density by a process known as ***ultra-centrifugation*** that separate proteins according to their density, but this process is difficult and requires sophisticated and expensive instruments. So according to their density they can be separated into Chylomicrons, HDL, LDL, VLDL, etc.



2. **Electrophoresis:** lipoproteins can be separated by ***electrophoresis*** which is a different method.
But how electrophoresis happens?!

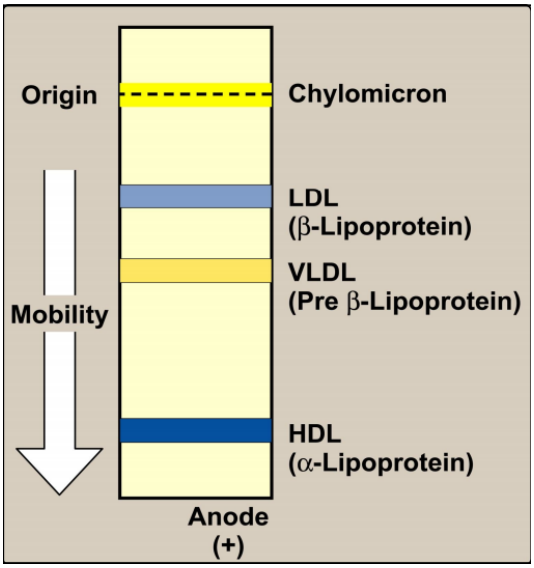
You have taken this before for the separation of plasma proteins.

We take a specimen and put it in a filter paper or cellulose acetate, and under electrical field the proteins migrate towards the anode (the +ve electrode), and the fastest is Albumin then alpha 1 then alpha 2 ….



How do we visualize these proteins?!
By using a stain/ dye that specifically binds to proteins, so wherever there's a protein, we can see these proteins by our naked eye. So we can separate them into these fractions and see these fractions.

What we care about here is that WE CAN DO the same thing for the SEPARATION of LIPOPROTEINS, but here we use a stain that is specific for the lipid. (03:52-03:57)
In this case we notice that the FASTEST fraction is HDL because it has a lot of proteins (25% proteins), so HDL is the fastest > VLDL > LDL, and notice that Chylomicrons do NOT migrate (move), they remain at the origin of separation. So chylomicrons don't move in electrical field why?! Because they have very little proteins, so the charge carried by the proteins is very little, they have a large size → so they don't move in electrical field.



* In this case, HDL may be called α-lipoprotein because they migrate with α-plasma protein, LDL which migrates with β-plasma proteins → β-lipoprotein. α and β refer to plasma proteins.

* Notice that the separation in this case is not based on the density. If depending on density, HDL/ LDL/ VLDL. They're based on the presence of charges on proteins.

* The good thing about separation by electrophoresis is that it's easy to perform, simple and can be done by many labs.

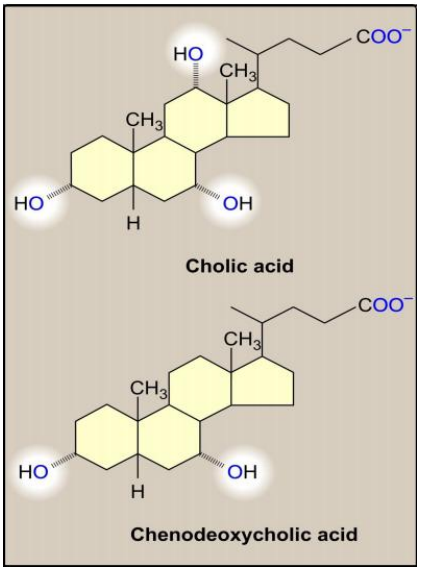
* Sometimes, we need to know the fractions (VLDL, HDL, LDL) for diagnosis of certain disease, so they're separated by electrophoresis by a simple procedure that can be done every day.

We've finished lipoproteins, now we'll move to digestion of dietary lipids.

Digestion of dietary lipids

* Digestion is important for absorption. We eat fat → before we can absorb this fat, it has to be digested.
* Digestion involves hydrolysis of 2 ester bonds to produce **2 fatty acids** and the third fatty acid will stay attached to glycerol at C#2 forming **monoacylglycerol** (MAG), so we produce 2 FA + MAG.
* Cholesteryl ester is also hydrolyzed to produce **cholesterol** and **FA**.
* This is a **hydrolysis rxn** → water is a reactant → each molecule of lipid should be attacked by water molecules → so we need proper mixing of water and TAG which can't be mixed !! → So we have *solubility problem!!* ☹ because the rxn of water with lipid requires the ability of water molecules to attack molecules of TAG.

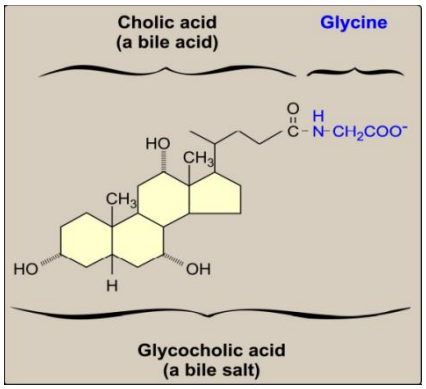
=> Now, HOW TO SOLVE THE SOLUBILITY PROBLEM?! How can we mix them together?! (Oil and water are not miscible :/
The SOLUTION is: we have **solubilizing agents,** what are these?! (Cholic acid and Chenodeoxycholic acid ,they're steroids)
* Their structure is shown here in the pic. And:
1) They remember us with cholesterol, so they're derived from cholesterol, but they're not exactly like cholesterol.
2) They're not exactly like cholesterol; they have some hydrophilic groups such as COO- (cholesterol doesn't have carboxyl group), so they're **acids** because of the presence of carboxyl group (while cholesterol isn't an acid)
3) Also, they have additional hydroxyl group(s) (hydroxyl group at C#3 is already found in cholesterol, but the OH groups at C#7 & C#12 are additional OH groups.)



So they differ little bit from the structure of cholesterol by having carboxyl group (they're acids), and the carboxyl group is more hydrophilic than the hydroxyl group.

Now, these acids that have carboxyl group are considered weak acids (their carboxyl group has a Pka around 4 or 5 as in many carboxylic acids). However, if the acid is conjugated/ added to the amino acid **glycine** (whose carboxyl group has lower pka ~2), so this will increase the acidity; so it becomes a **stronger acid**. This conjugated bile acid by amide bond to glycine is stronger acid so it (the conjugated acid) is almost totally ionized therefore it's called **BILE SALT**,

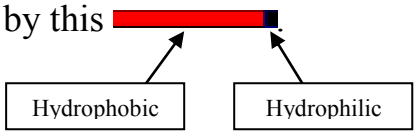
⇨ So the conjugated acid to glycine or another amino acid becomes strong acid and is called bile salt (because it's found mainly as a salt form).



Bile salt is a bile acid conjugated to amino acid glycine or another amino acid such as taurine (which is a sulfur containing a.a)

* The acid and the salt are used interchangeably, sometimes they say bile acids while another times they say bile salt, but they usually refer to the same molecules.
* The special structure of bile salts shows that the molecule has a hydrophobic nature (*the steroid nucleus*), but at one side there's a hydrophilic region → it's an **amphipathic** molecule that has a hydrophobic region and hydrophilic region,

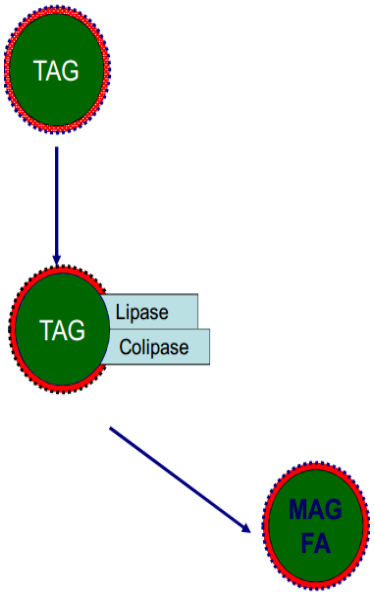
It can be represented by this



* They (bile salts) tend to form micelles, 

so they can solubilize (because they're amphipathic) and emulsify TAG.

* This particle that is formed from bile salts emulsifies TAG.
* It's a very small particle → surface area is very large.
* This can be attacked by enzyme lipase. This lipase, which is secreted from the pancreas, is called pancreatic lipase and can attack micelles that contain TAG with bile salts TO PERFORM THE DIGESTION. With this small size, water can be mixed with TAG due to the presence of bile salts which are important in digestion "without them digestion will NOT occur because the oil/fat won't be mixed with water".



- ✓ Digestion requires:
  1) bile acids
  2) lipase

  Without them digestion is not complete and absorption is not complete!

* Bile is made in the liver and secreted from the gallbladder, whereas lipase is made in the pancreas and secreted (also) to the duodenum (small intestine), so the secretions of the bladder containing the bile reach the small intestine with the pancreatic secretions which contains the lipase and co-lipase.

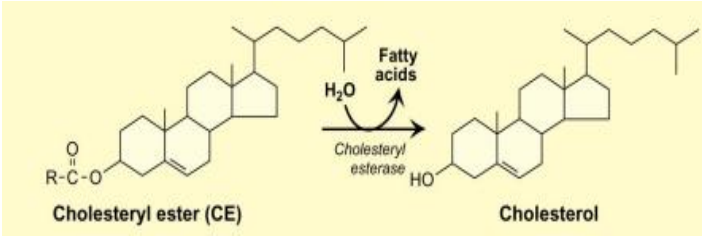
Bile salts and lipase together help in the digestion, Bile salts work for solublization followed by the action of lipase that requires another protein called co-lipase to interact with TAG in the micelles and help in solublization so TAG is converted to MAG and FA [the needed digestion happens].

* After Digestion what you have is TAG that is hydrolyzed to MAG and FA.

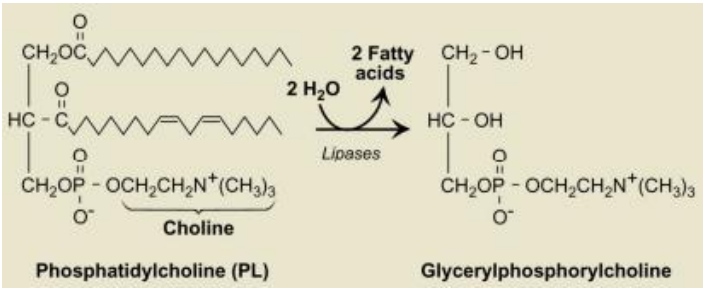
THE ACTION OF HYDROLYSIS:

Now, let's look at the **ACTION OF HYDROLYSIS** on structures:

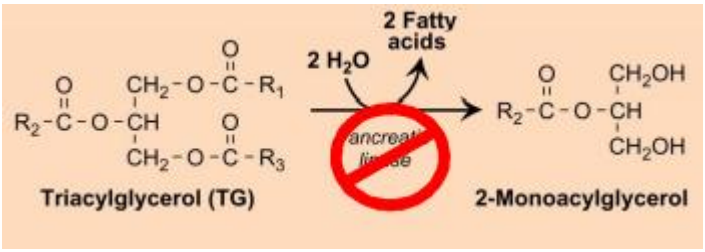
- This is **cholesteryl ester** (it's a cholesterol having FA at C#3 [esterified to C#3]), with cholesteryl esterase, **Cholesterol + FA** will be produced. This is how cholesteryl ester is hydrolyzed in the intestine.



- **Phospholipids** similarly are hydrolyzed; FA at C#1 and FA at C#2 are removed, and we end up with two **fatty acids** and glycerylphosphorylcholine. This is catalyzed by various phospholipases.



- **TAG**, (glycerol with 3 FA). The hydrolysis occurs at C#1 and C#3 leaving **2-monoacylglycerol** (i.e C#2 of glycerol is still esterified). This molecule (MAG) is an *amphipathic* molecule, it has some (water) solubility because it contains OH groups on C #1 and C #3 → it's an amphipathic molecule that → can be part of micelles.



* A student asked: "If C#2 loses its FA, can we produce glycerol?!
NO, because pancreatic lipase hydrolyzes specific bonds. Maybe a small portion is produced but these are the major products. It (C#2) may be attacked by another different lipase (because each lipase is specific for certain bond) but not by pancreatic lipase which can't attack C#2 because the shape of the molecule is different. The enzyme always recognizes the shape of the molecule and the bond to be cleaved is different in each case. So FA at C#2 is not removed by pancreatic lipase.

Digestion and absorption & inhibition of pancreatic lipase:

* Without digestion, no absorption can take place.
* Digestion is important for absorption.

* What about inhibition of pancreatic lipase?! What happens?!
Accumulation of TAG in the small intestine (in the lumen) will result.
TAG will not be digested → will not be absorbed → it will remain in the small intestine → and excreted with the stool or in the feces (it appears in the feces).

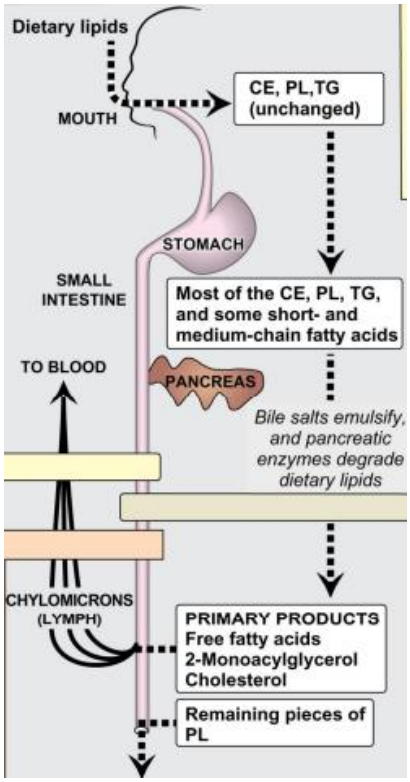
* WHY to inhibit digestion of TAG?!
To avoid obesity, so you can eat and don't worry about getting fat (obese). Of course don't do it every day!!! But if you want to eat a lot in a day, you can take a pill of Orlistat.

A wiki note: (not mentioned by the dr.)

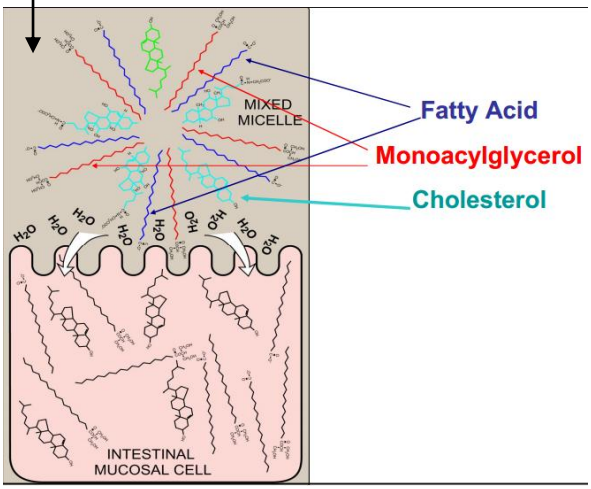
Orlistat (also known as **tetrahydrolipstatin**) is a drug designed to treat obesity. It is marketed as a prescription drug under the trade name **Xenical**. Its primary function is preventing the absorption of fats from the human diet by acting as a lipase inhibitor, thereby reducing caloric intake. It is intended for use in conjunction with a healthcare provider-supervised reduced-calorie diet.

It's a very expensive drug, one bottle containing 30 pills may cost 40 JDs.
This is an application of biochemistry.
Some people make themselves vomit!! In order not to get fat.

* This diagram is from the book, it shows you: →
The dietary lipids goes to the stomach. In the duodenum, most of cholesteryl esters, phospholipids, TAG, and some short chain FAs are digested here, and the products are FA, 2-MAG, and cholesterol, these are taken to the blood through lymph. Remaining pieces of phospholipids are passed.



* This is a micelle; showing different components of the micelle which are FA, and other products of digestion (such as MAG [which is an amphipathic molecule that remains in the micelles], and free cholesterol) that form mixed micelle.



This micelle when comes in contact with the intestinal mucosal cell, the constituents of the micelle (FA, MAG,…) are taken into cells. So digestion will not occur unless the TAG is hydrolyzed and enter the cell.

* Digestion of TAG with Short or Medium Chain Fatty Acids (4, 6, 8 carbons up to ~10 carbons):

Digestion here is a little bit different, it begins in the stomach because the tongue secrets a lipase called lingual lipase that rapidly reaches the stomach, and even the stomach secrets gastric lipase. These lipase are **ACID STABLE;** i.e they can work even though the pH in the stomach is very low (high acidity)! → they can act on TAG that is formed from short and medium chain FA.

* What is the kind of food that contains short and medium chain FA?!
Milk and dairy products, butter & cream. They all contain short and medium chain FA that can be digested in the stomach without the aid of bile salts or pancreatic lipase, so their **significance**:

- ✓ In the NEONATES, they can start digestion even before the food (milk) reaches the small intestine (duodenum).
- ✓ In the PANCREATIC INSUFFICIENCY (a problem in the pancreas, the pancreas is not working), in this case digestion of short chain FA can occur.

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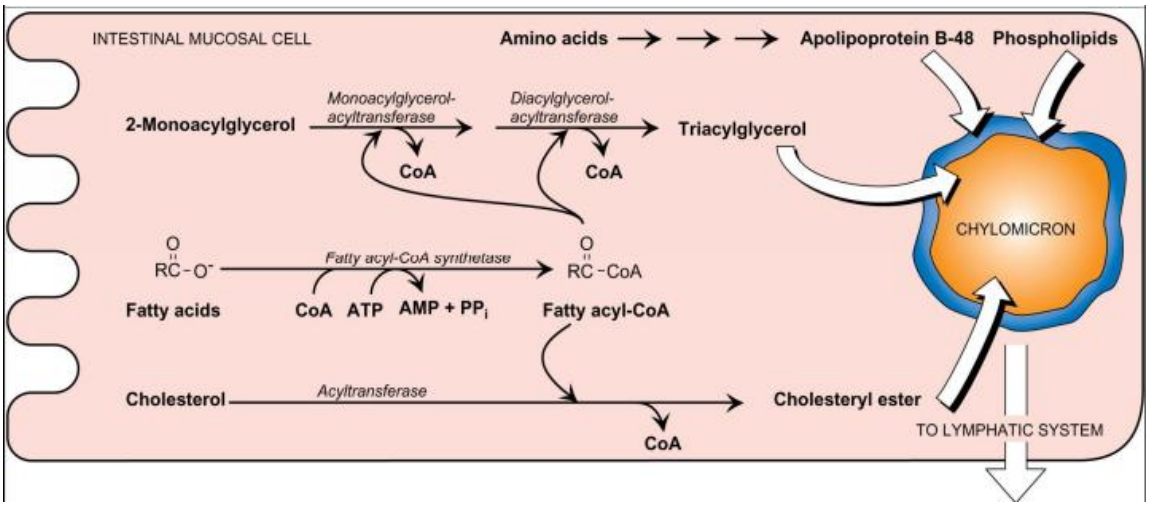
* This pic shows the intestinal mucosal cell in which MAG, FA, and cholesterol have entered. **The journey of chylomicrons**. Firstly, **HOW are CHYLOMICRONS formed?!**



The fatty acid in the cell is activated to fatty acyl CoA by fatty acyl CoA synthetase (the 1st rxn we have taken with Dr. Faisal in FA metabolism). Fatty acyl CoA synthetase adds CoA to fatty acid to make fatty acyl CoA which acts as a donor of fatty acids to MAG & DAG to convert MAG back to TAG.

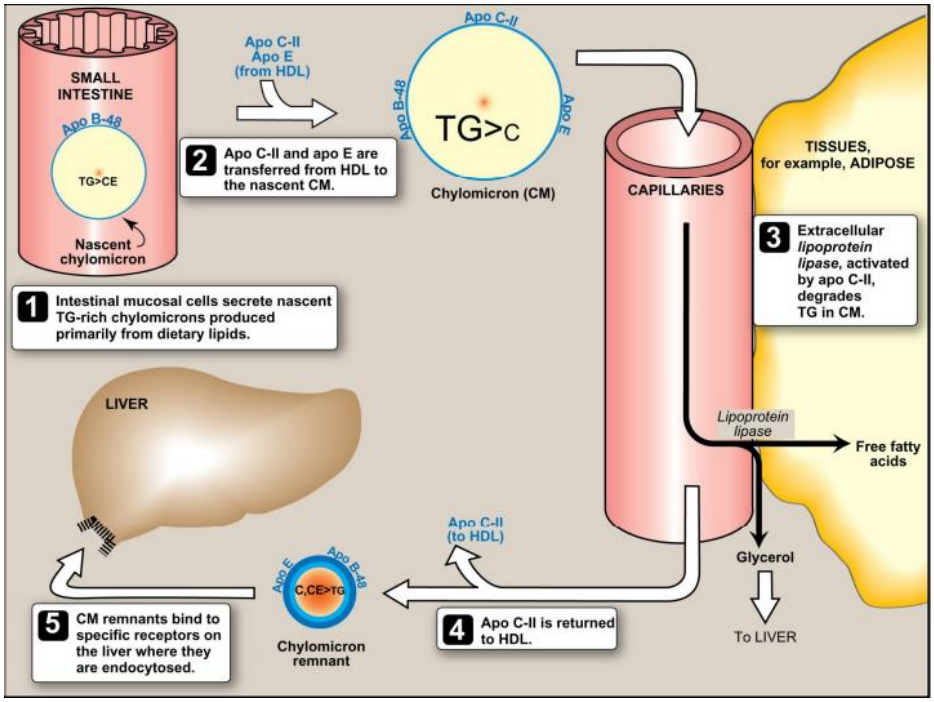
Fatty acid $\xrightarrow{\text{Fatty acyl CoA synthetase}}$ Fatty acyl CoA (donates FA to MAG & DAG)

So we've degraded the food in the lumen of the duodenum and produced MAG, fatty acids and cholesterol, but they aren't going to be transported in the blood as such, the products enter the cell but before they leave it, TAG is resynthesized again. Also, cholesteryl ester is resynthesized again.These are integrated (incorporated) together with phospholipids and Apo-lipoprotein B-48 to form these lipoprotein particle **(CHYLOMICRONS).**

* Chylomicrons are formed from products of digestion **in the small intestinal cells** to carry these TAG.

* Notice that: chylomicrons are relatively **large particles**, so they can't be secreted directly to the capillaries, they can't be transported from capillaries to blood directly.
=> They are released into the lymphatic system by exocytosis through the lymph (lymph → thoracic duct → reach the circulation through subclavian vein which is relatively large vein).
* If they were to be secreted directly in the capillary, they might block them.
Therefore, the transportatio n occurs from lymph → to large vein → to the circulation.

*** WHAT happens to chylomicrons in the CIRCULATION?!**



This is the small intestine. The chylomicrons (1.large particles that contain mainly TAG more the cholesteryl ester) are synthesized.
2. They have the protein Apo-B48.
3. They're released into the blood. In the blood, they acquire Apo-lipoprotein C2 & Apo-lipoprotein E which come from HDL ((HDL is a reservoir for Apo-C2 & Apo-E which are important for the metabolism of chylomicrons)).
4. They go through capillaries in different tissues (adipose, muscle tissue).
5. In the tissue, there are capillaries that have attached extracellular enzyme **{lipoprotein lipase}.**

*** LIPOPROTEIN LIPASE:**

1) Is made by the capillaries by the endothelial cells.

2) Its attached extracellularly to the cell (it's out the cell but still attached to its plasma membrane).

3) It requires Apolipoprotein C2 as activator.

4) It acts on TAG to hydrolyze TAG again to 3 FA and glycerol, FA enter the cell directly because FA are insoluble. Glycerol goes to the liver.

* This process continues for several hours until the chylomicrons are almost completely devoid (empty) from TAG. Very low TAG, DENSITY INCREASES ⇧.
It's now called CHYLOMICRON REMNANTS.

* **Apo-C2,** which originally comes from HDL, goes (return) back to HDL.

* **The chylomicron remnants** are taken by the liver through endocytosis, apolipoprotein E is important for binding of chylomicron remnants into the liver cells.

⇨ So this is the JOURNEY of chylomicrons **from small intestine to the liver** through the circulation.

⇨ What's the **purpose** of this journey?!
The TAG was transported from the small intestine where it's absorbed to the tissues and finally what remains is transferred to the liver (Chylomicron remnants are taken by the liver through endocytosis).

* A student asked: "do the remnants return to the intestine?!"
NO, TAG has been transferred to tissues and the liver takes the chylomicron remnants where they're metabolized.

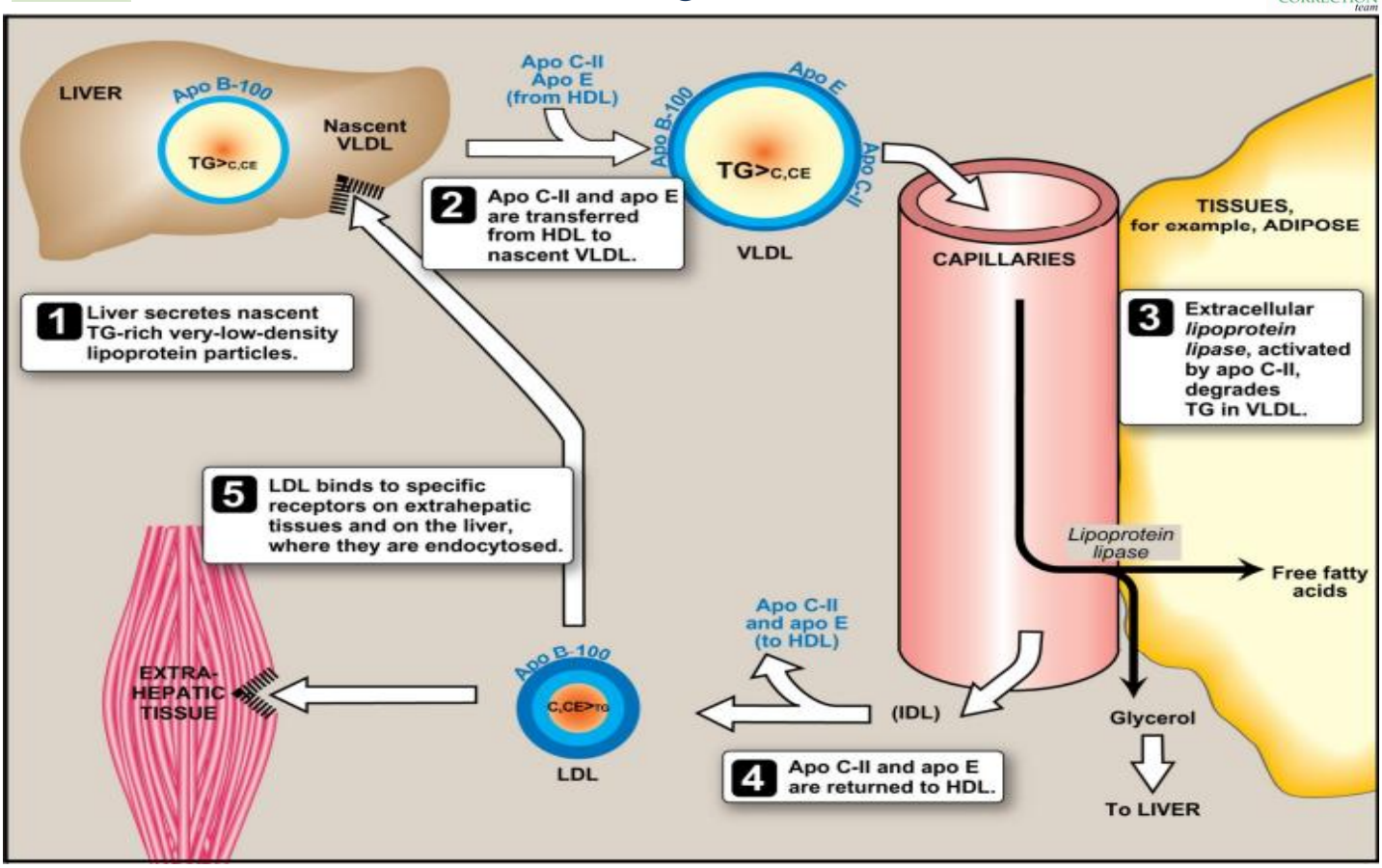
The metabolism of VLDL (very low density lipoproteins)

We mention it because they're very similar, the difference is that: VLDLs are produced **in the liver** to transfer TAG synthesized in the liver to different tissues.

* From what the TAG is synthesized in the liver?!
From Excess carbohydrates, excess amino acids that are metabolized to Acetyl CoA → fatty acid synthesis → TAG.

* TAGs are incorporated into VLDL.

Now, this is a pic from the book.



*VLDL is 1.much smaller than chylomicrons; 2. They're released directly into the blood (opposite to chylomicrons). 3. In the blood, they acquire also Apo-C2 & Apo-E from HDL. 4. They go through the same catalyst (lipoprotein lipase) which acts on TAG, hydrolyzes them, so VLDL is nearly emptied from TAG.

* What will happen if we take TAG from VLDL?!
Lipid percentage decrease, so Density INCREASES, from VLDL to IDL (intermediate density lipoprotein) to LDL.

* The product is **IDL** (Half of it returns to the liver and the other half is converted to LDL. IDL return back the Apo-C2 & E to HDL.)
* **LDL** particles have Apo-B100, cholesterol and cholesteryl ester and much more than TAG. So they become <u>smaller particles</u> with <u>higher density</u> than VLDL.

⇨ TAG synthesized in the liver is TRASNFERRED to different tissues.
* Now, What's the difference?!
- ✓ VLDL carries **<u>endogenous</u>** TAG. "endogenous: TAG is synthesized in the body from non-lipid sources [from excess carbohydrates].
- ✓ Chylomicrons carry **<u>exogenous</u>** TAG.

Cholesterol

Chole- → related to gallbladder (such as cholecystitis, cholecystectomy and cholangitis which are related to gallbladder)
Ster- → it's a steroid (contains steroid nucleus)
-ol → alcohol (hydroxyl group at C#3)

* Cholesterol: It's named so because it's isolated from the gallbladder (its origin/source).

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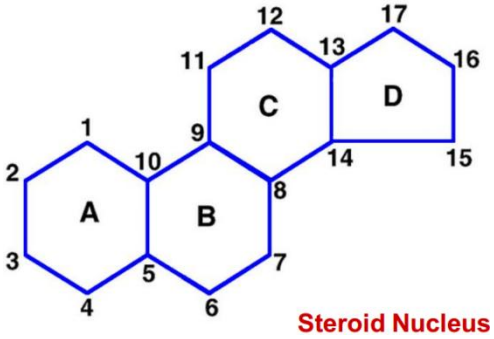
* Steroid nucleus:



Steroid Nucleus

- ✓ It's found in cholesterol
- ✓ It has 17 carbons
- ✓ Numbers are important. You are going to encounter many enzymes and compounds that refer to numbers.

  * 17-ketosteroid => it's a steroid that has ketone group at C#17 which can be used for diagnosis of diseases.

  * Another examples: 17-ketosteroid, 11-hydroxylase, vitamin D (which the active form of it is 1,25-dihydroxy cholecalciferol [C#1 and C#25 are hydroxylated]).

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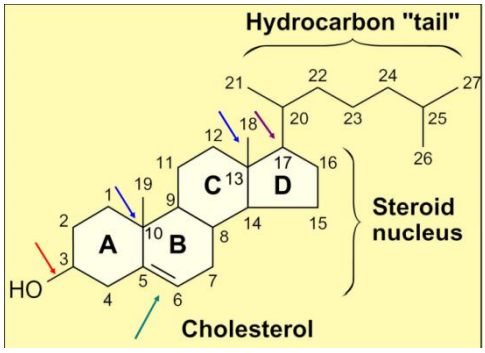
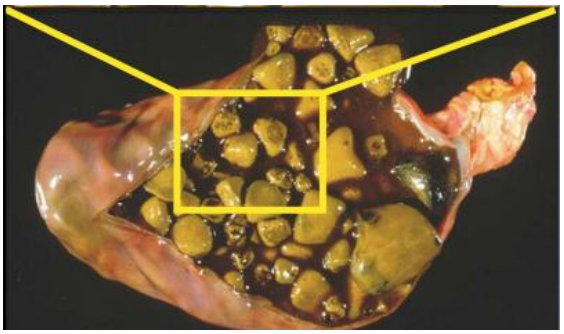
* Structure of cholesterol:

- Cholesterol is the parent compound of all steroids.
- Characterized by the presence of:
  - ✓ Hydroxyl (OH) group at C#3
  - ✓ Double bond (=) between C#5 & 6
  - ✓ Two methyl (CH3) groups, which are given no. 18 &19, at C#13 & C#10
  - ✓ Hydrocarbon chain formed from 8 carbons (given no. 20-27).
- The cholesterol is made from carbons & hydrogens with **JUST ONE oxygen atom**, so it's **INSOLUBLE in water.**



Gall bladder stones:



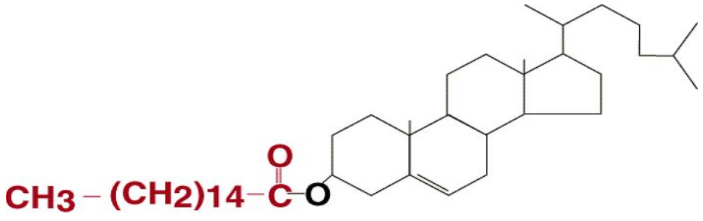
Look at this pic.
* What is this?! Stones!
This is an opened gallbladder that contains stones.
What are these stones made of?! Cholesterol!
(* remember that cholesterol is isolated from gallbladder*)

* The cholesterol in the bile is found in the solubilized (soluble) form due to the presence of bile salts and phospholipids (emulsifying agent), so it's soluble and secreted by the liver in the bile and doesn't precipitate but if there is a defect in solubilization [because of disturbance in the levels of PL and bile salts], precipitation of cholesterol will happen and result in gallSTONES!
* If somebody has these stones, can we give him a drug to solubilize them?!
NO, the drug is NOT enough to remove these stones. So we remove the gallbladder (cholecystectomy [removal of gallbladder] is the solution).
* A student asked: "can he live without gallbladder and what about the bile secretion?!" YES, he can live without it, and there's a bile secretion because gallbladder doesn't secret bile, its function is to STORE bile not to produce it.

* Cholesterol was isolated from gall bladder stones in 1774 before about 240 years ago, and since that time cholesterol has been studied by many scientists to the extent that 13 noble prizes were awarded to those scientists who worked on cholesterol.

* Cholesterol's problem why? no. 1 of death (stroke & myocardial infarction due to atherosclerosis [deposition of cholesterol in arteries]).

This is CHOLESTERYL ESTER



* This is **cholesteryl ester**; you can recognize that the hydroxyl group on C#3 is esterified to fatty acid so it's called cholesteryl ester.

* Which do you think is more soluble free cholesterol or cholesteryl ester?! Of Course, cholesteryl ester (the cholesteryl ester is more hydrophobic and less soluble in water than cholesterol; both are poorly soluble in water but soluble in bile because the presence of phospholipids & bile salts (emulsifying agents).

*** Sources & elimination of cholesterol:**

* Why do we need cholesterol if it is such so bad?!

Cholesterol is required mainly as plasma membrane component; all cells in the body have cholesterol in their plasma membrane; it modulates fluidity of the plasma membrane because it has small hydroxyl groups that could interact with water [hydrophilic] and the rest of molecule is hydrophobic.

* All (animal) cells require cholesterol as component of the plasma membrane, so if you eat anything from animal, you are eating cholesterol.

* In general, synthesis can be done by all cells, but practically liver is the 1st place in making cholesterol. Some are synthesized in small intestine and in the adrenal cortex (which synthesizes steroids and cortisone) because it converts cholesterol into steroid hormone. All cells are capable of making cholesterol; they have the enzymes required for cholesterol synthesis, but actually cholesterol is synthesized mainly in the liver. Other cells don't usually synthesize it because they don't need it at a high rate; however they are capable to, and this reflex the importance of cholesterol to all cells).

* A student asked: "can we see cholesterol in kidney stones?!"

NO, kidney stones don't contain cholesterol; cholesterol isn't secreted by the kidney.

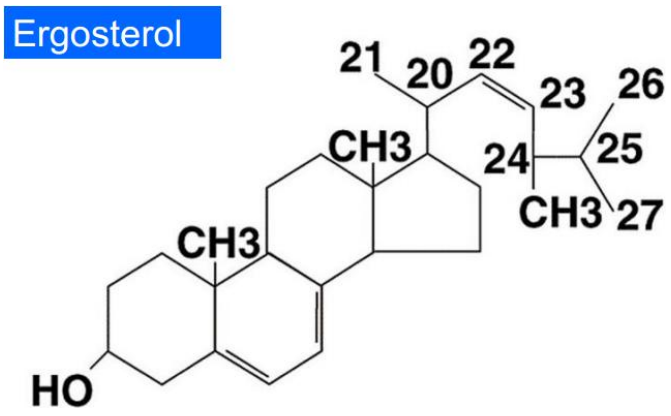
* Elimination:

* Dietary cholesterol provides about 300mg.
* If you want to be in low cholesterol diet, you should ingest less than 300mg. it's recommended that the individual shouldn't consume more than 300mg/day.
* One egg contains 250mg of cholesterol!

* Even though cholesterol can be synthesized by many cells , it can't be degraded into CO_2 and H_2O; i.e **it's not a source of energy**. It doesn't undergo degradation by β-oxidation to produce energy.

* It's **eliminated** as such or as bile salts; i.e **the liver secrets cholesterol** as cholesterol or after converting some cholesterol into bile salts.

* What is this?!
This is NOT cholesterol, but it looks like it.
It's known as **ergosterol** (Plant sterol).



* Plants don't produce (have) cholesterol, however animals do. (Whatever you eat from animals, you're eating cholesterol. But for plants this is not true).
* Plants lack cholesterol. If you have oil and it's written on it that it's FREE OF CHOLESTEROL, they're fooling you! :P because the oil has to be free of cholesterol.

* Plants steroids are POORLY absorbed by human so they're good for elimination of cholesterol, and perhaps they help to reduce the amount of cholesterol.

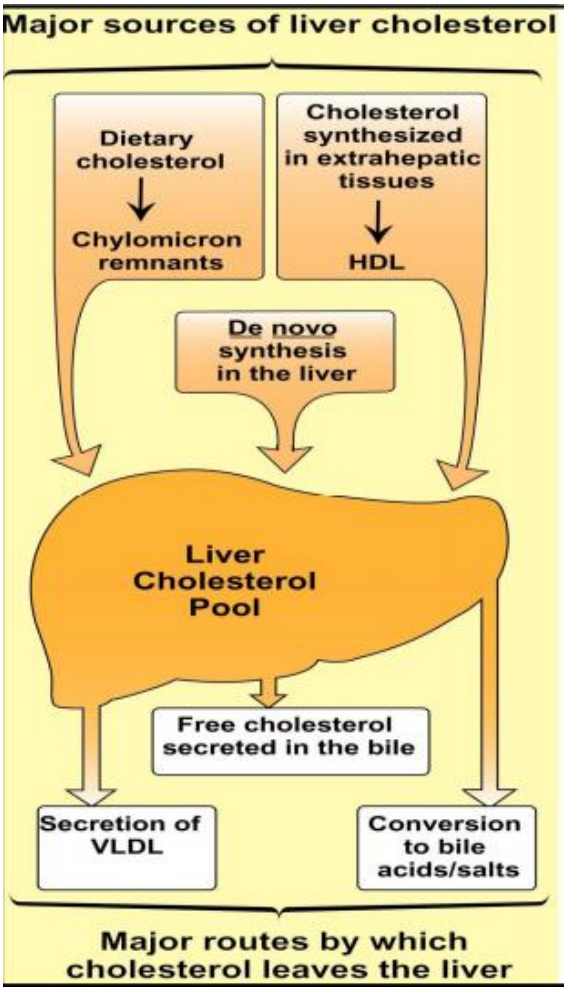
* Major SOURCES & ROUTES of cholesterol:

Sources: (how the liver receives cholesterol)

⇨ Cholesterol is synthesized in the liver as we've said. DE NOVO synthesis means that it's synthesized originally from scratch.

> ***Wiki note:***
>
> ***"De novo* synthesis** refers to the synthesis of complex molecules from simple molecules such as sugars/ amino acids".

⇨ Dietary cholesterol through chylomicronn remnants reach the liver.
⇨ Cholesterol synthesized in extrahepatic tissues is also transferred to the liver.



Cholesterol is:

⇨ Transported from the liver as VLDL.
⇨ Secreted in the bile as Free cholesterol.
⇨ Converted to bile acids/slats.

* The dr. asked: "What is the most kind of food that is rich with cholesterol?!

Nuts?! !! الله يسامحك بس Plants DON'T produce cholesterol!!!
The answer is: egg ((in daily basis))
The liver is the factory for cholesterol synthesis and it's the landfill for cholesterol disposal.

- ✓ So in daily basis, the most common source of dietary cholesterol is **EGG**. One egg contains 250mg.
- ✓ The most rich food in cholesterol is **LIVER**, but we don't eat liver daily, we eat it once a year في عيد الاضحى :P

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* Cholesterol synthesis:

- Requires
  - Carbon source (Acetyl CoA) like in FA, not from CO_2.

It was found that ALL carbons (27 carbons) of cholesterol come from Acetyl CoA, nothing else! So Acetyl CoA is the only source for the synthesis of cholesterol, and they've also determined whether each carbon in the cholesterol comes from the methyl carbon (CH_3) or from carboxyl carbon (C=O) of the acetic acid BY LABELING using radioisotopes.

- Energy, to join these carbons together.
- Reducing power **(NADPH)**.

* Notice that acetyl group contains 2 oxygen atoms while cholesterol has one oxygen atom, so during synthesis oxygen is removed → which is **reduction** (loss of oxygen) → so we need reducing power, and as usual the reducing power comes from **NADPH**

- Oxygen

The oxygen that exists in cholesterol is not one of the oxygens that were found in acetyl CoA molecules that entered the rxn. All the oxygens are removed, then one oxygen is added.

* Let's look at some cholesterol synthesis intermediates, these intermediates should be memorized and it's enough to know them.
* Cholesterol synthesis may include 20 steps but we need to know the major steps/mechanism by which cholesterol is produced.
* This is isoprene (-from Isopentene: because of the presence of the double bond):

Acetyl CoA (C2)
↓
Mevalonate (C6)
↓
Isoprene Units (C5)
↓
Squalene (C30)
↓
Lanosterol (C30)
↓
Cholesterol (C27)

1) Which of the following supplies all the carbon atoms that are needed for de novo synthesis of

cholesterol?
a) Glucose
b) Acetyl CoA
c) Malonyl CoA
d) Succinyl CoA
e) Citrate
Answer: b

2) Which of the following is not needed for the de novo synthesis of fatty acids?
a) NADPH
b) Acetyl CoA
c) Oxygen
d) ATP
e) Folic acid
Answer : e

3) A 45 -year-old female presents with pain in the right hypochondrium radiating towards shoulder and back. She is diagnosed with acute cholecystitis (inflammation of the gall bladder).The gall stones can obstruct the gall bladder, leading to an inadequate concentration of bile salts in the intestine. Which of the following statements best describes the bile salts?
a) Squalene and Lanosterol are examples of primary bile salts
b) Bile salts are required for transportation of lipids from intestine to liver
c) 95% of the bile salts are excreted in the feces per day
d) They are required for digestion and absorption of lipids
e) Bile salts are synthesized from glycine and taurine
Answer: d

This question is about the previous lecture:
4) In Abetalipoproteinemia (a rare disease), lipoproteins containing apo B are not formed and lipid droplets accumulate in the intestine and liver. Which of the following lipoproteins does not contain Apo B protein?
a) LDL
b) IDL
c) HDL
d) Chylomicrons
e) Lp (a)
Answer: c

5) The modification to bile salts that increases the working pH range and amphipathic nature of bile salts is
a) 7α-Hydroxylation
b) Dehydroxylation by intestinal bacteria
c) Esterification
d) Conjugation to taurine or glycine
e) Formation of salts with sodium and Potassium.
Answer: d
6) A gall stone that blocked the upper part of the bile duct would cause increase in which of the followings ?
a) The excretion of fats in the feces

b) Formation of chylomicrons
c) Excretion of bile salts
d) Conjugation of bile acids
e) Recycling of bile salts
Answer: a

7) Which of the following apoproteins is an activator of lipoprotein lipase?
a) Apo A
b) Apo B
c) Apo C II
d) Apo D
e) Apo E
Answer: c

8) In the conversion of cholesterol to bile salts, which of the following statements best describes the process ?
a) Carbon 8 is hydroxylated
b) The side chain can be conjugated with glycine or Taurine
c) The double bond is oxidized
d) The hydroxyl group at 3 carbon remains in the beta position
e) It occurs in the bile duct.
Answer: b

9) Which of the following lipoproteins are the major carriers of Triacyl glycerol ?
a) IDL and LDL
b) VLDL and LDL
c) Chylomicrons and VLDL
d) HDL and VLDL
e) Chylomicrons and HDL.
Answer: c

10) A 40- year-old man presents with severe pain in the legs upon walking. He is diagnosed with atherosclerotic plaques in the arteries of his legs. High level of cholesterol and LDL contribute to the formation of atherosclerosis. Which of the following is digested to form LDL ?
a) IDL
b) Chylomicrons
c) HDL
d) Cholesteryl esters
e) Cholesterol.
Answer: a

"You don't have to be great to start, but you have to START to be great"
GOOD LUCK DOCTORS ☺
Your colleague: Shatha Tailakh