بسم الله الرحمٰن الرحيم

(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)

Metabolism | FINAL 5

Lipid digestion & absorption

Written by : NST
DST

Reviewed by : Shahad Alrawi
NST member

وَلِلَّهِ الْأَسْمَاءُ الْحُسْنَى فَادْعُوهُ بِهَا

المعنى: الأول: الذي ليس قبله شيء، وكل ما سواه كائن بعد أن لم يكن، و(الآخر): الباقي، الذي لا انتهاء لوجوده، وليس بعده شيء.

الورود: ورد الاسمان مرة واحدة في القرآن الكريم.

الشاهد: ﴿هُوَ ٱلْأَوَّلُ وَٱلْآخِرُ وَٱلظَّٰهِرُ وَٱلْبَاطِنُ ۖ وَهُوَ بِكُلِّ شَىْءٍ عَلِيمٌ﴾ [الحديد:٣].

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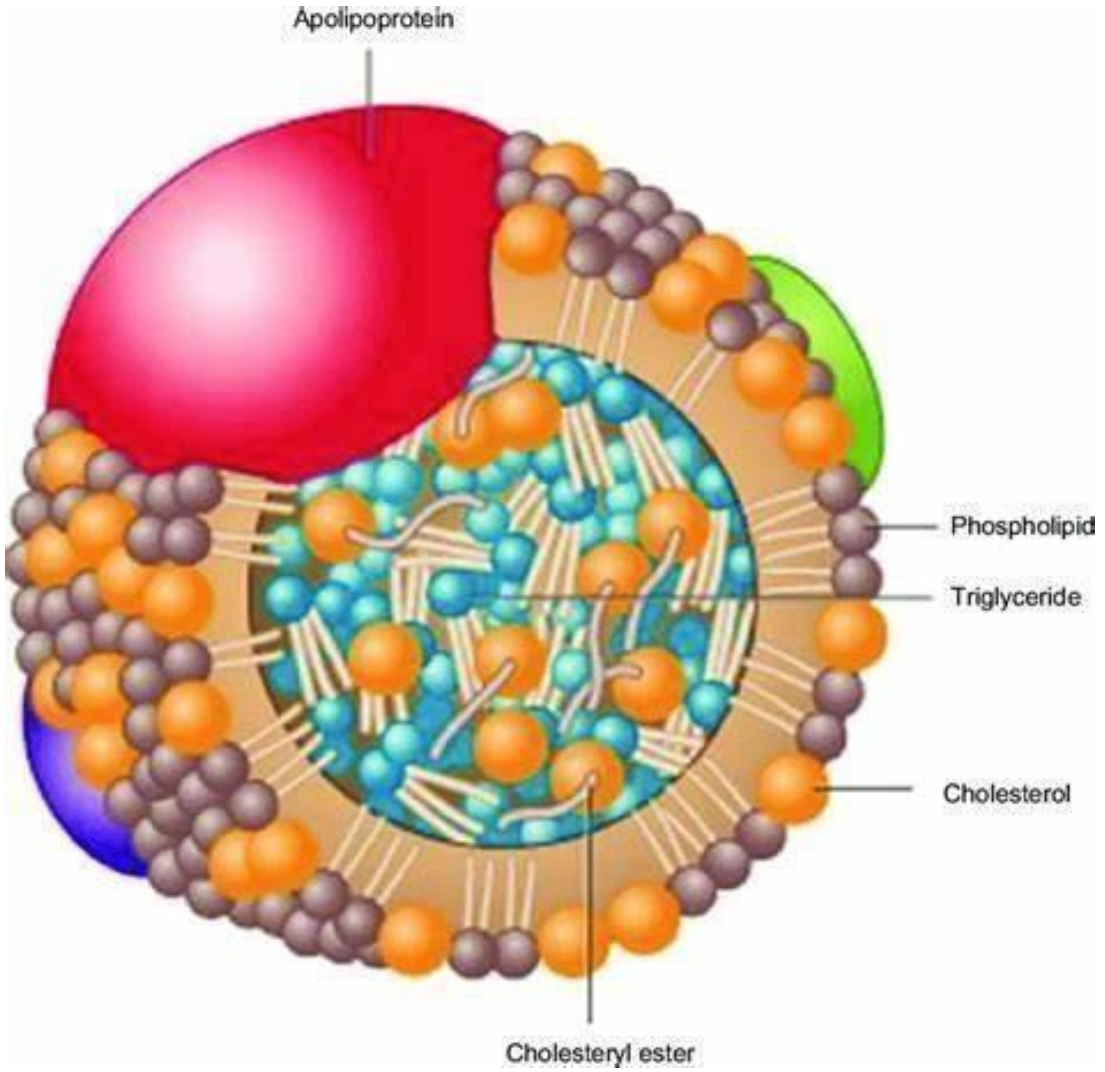
اضغط هنا لشرحٍ أكثر تفصيلًا

Metabolism of lipids:

Absorption and transport

Dr. Diala Abu-Hassan

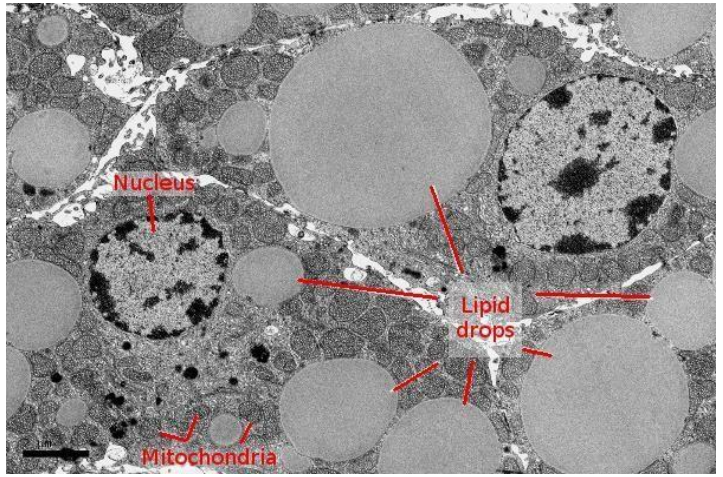


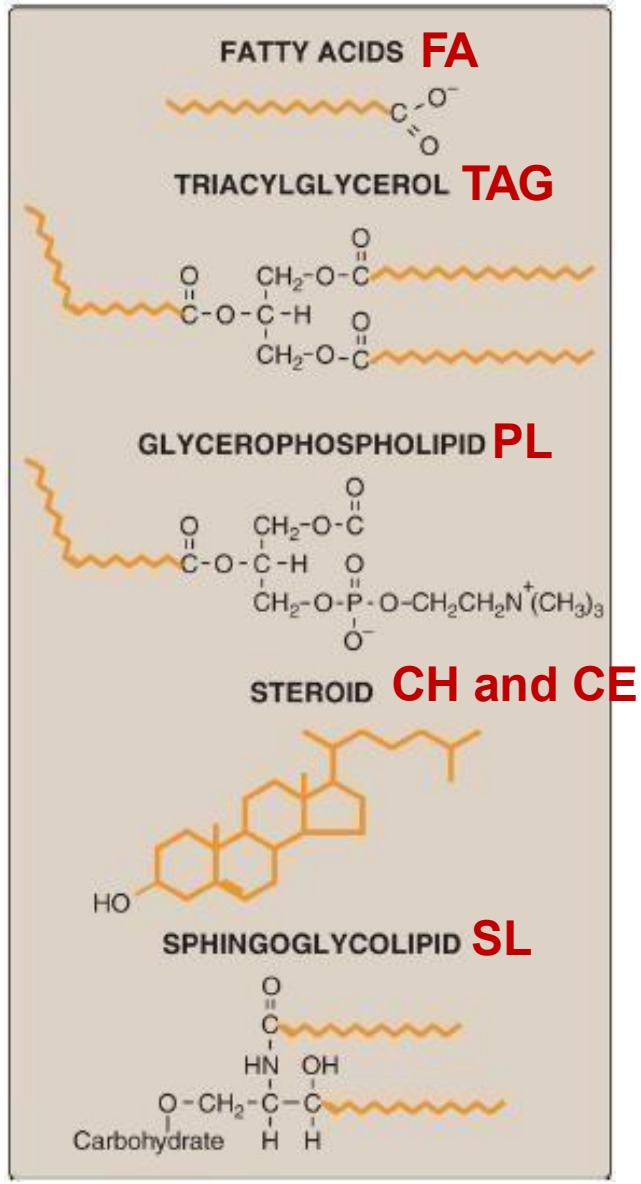
Lippincott's Biochemistry, Ch. 15

Lipids-review

- Lipids are heterogeneous, hydrophobic, they are very diverse in their structure ,they cant move on their own ,so they are compartmentalized in membranes, as droplets of triacylglycerol (TAG), or in lipoprotein (LP) particles, or protein-bound.
- Functions: Energy, structures, molecular precursors (e.g., vitamins, signaling)
- The major dietary lipids are triacylglycerol (oils and fats), cholesterol (in animal cells), and phospholipids.

Lipid droplets grow in size when ever we have more lipids present in the cells



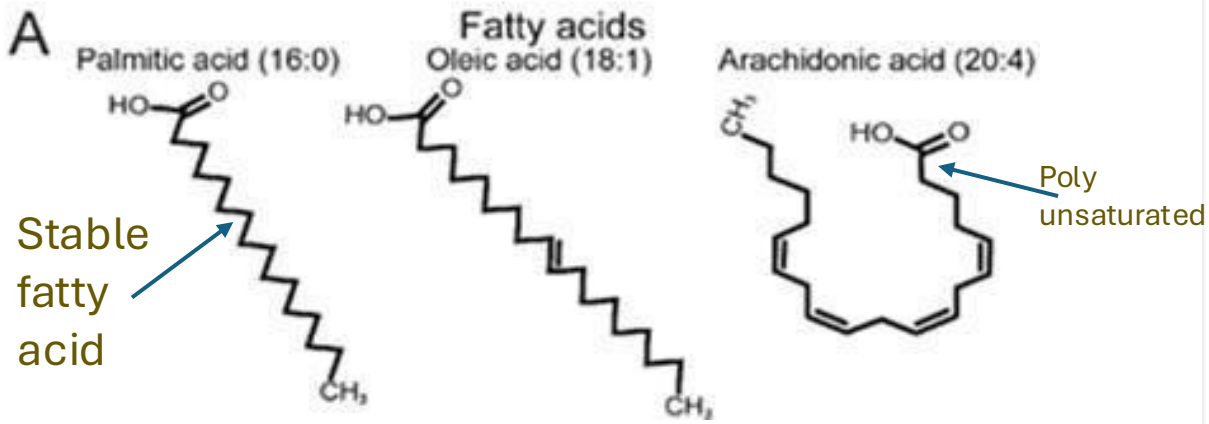


Lipids-review– further explanation

- Lipids are heterogenous group of molecules, they don't have a strict functional group that characterize them, they have different structures, what is common between these lipids is that they are all considered hydrophobic.
- As these lipids are hydrophobic, they don't like to face watery compartments, that's why they tend to cluster forming droplets with their polar groups facing the hydrophilic environment, remember that this clustering is mediated by hydrophobic interactions.
- Lipids have multiple functions, they store energy, they form structural units for membranes, they serve as a precursor for many anabolic pathways that synthesize sex hormones, vitamins, bile acids, they are also involved in signaling pathways. (remember PIP2, DAG)
- Lipids are obtained from our diet, most of them are in the form of TAGs (found in fat and oil), cholesterol is found in animal-based food. Lipids are also used as emulsifiers (e.g., lecithin; another name for phosphatidylcholine) in food industries.

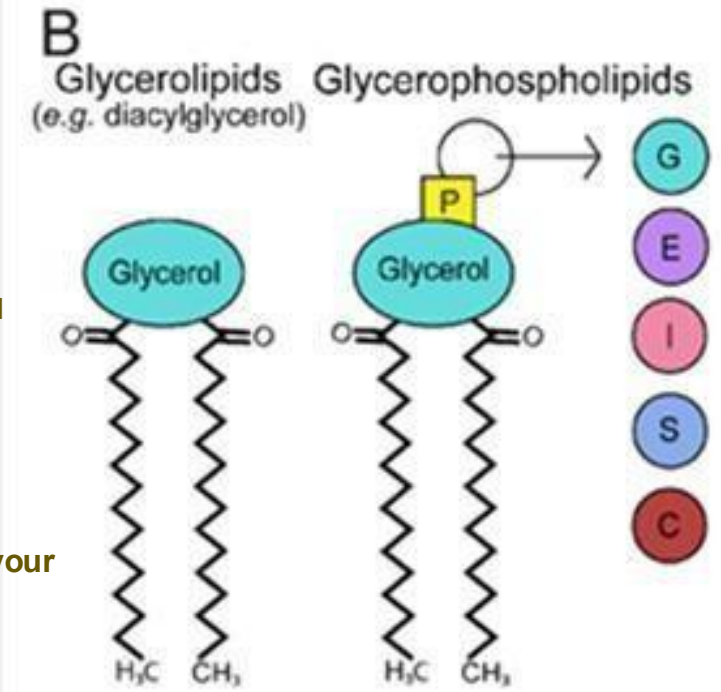
TAGs: Triacyl Glycerols

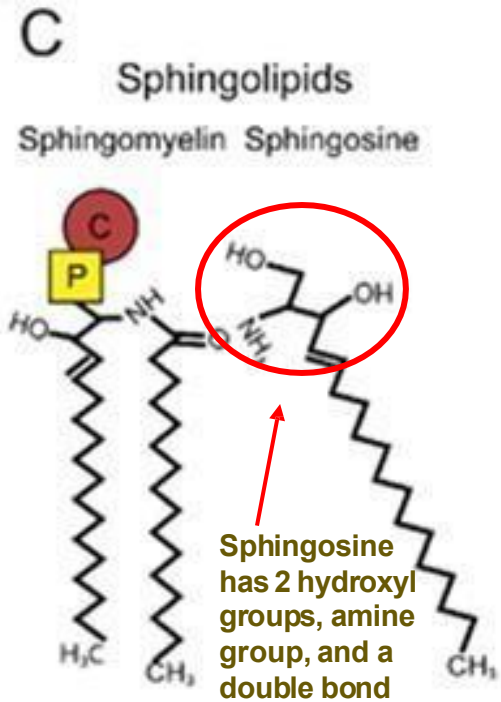
Structure and classification of lipids



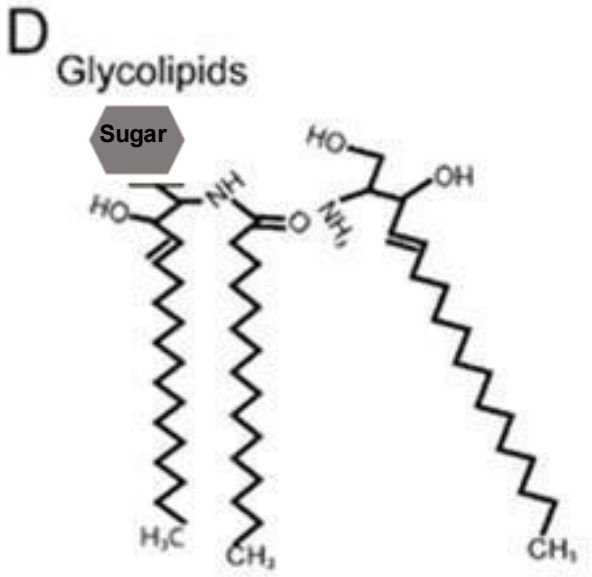
A Lipid could be structured of: ***(majority of the unsaturated fatty*** **acids in your cells are present in the cis configuration)**

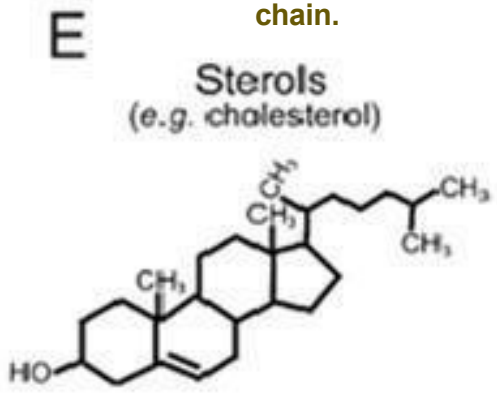
- **Fatty acid**: carboxylic acid with long hydrocarbon chain, could be saturated, monounsaturated, polyunsaturated.
- **Glycerophospholipid**: two fatty acids joined to a glycerol molecule, with a phosphate group attached to glycerol, different groups bound to the phosphate gives us different types of glycerophospholipids.
- **Sphingolipid**: a sphingosine molecule which makes on of the tails (has a long hydrocarbon chain within its structure) attached to a fatty acid and a phosphocholine giving sphingomyelin, substituting the phosphocholine with sugars gives us a glycolipid.
- **Sterol:** a steroid nucleus (three six-membered rings and one five-membered ring fused to each other) with side chains attached to these rings, cholesterol (which is a sterol), could be used to synthesize vitamins and bile acids.





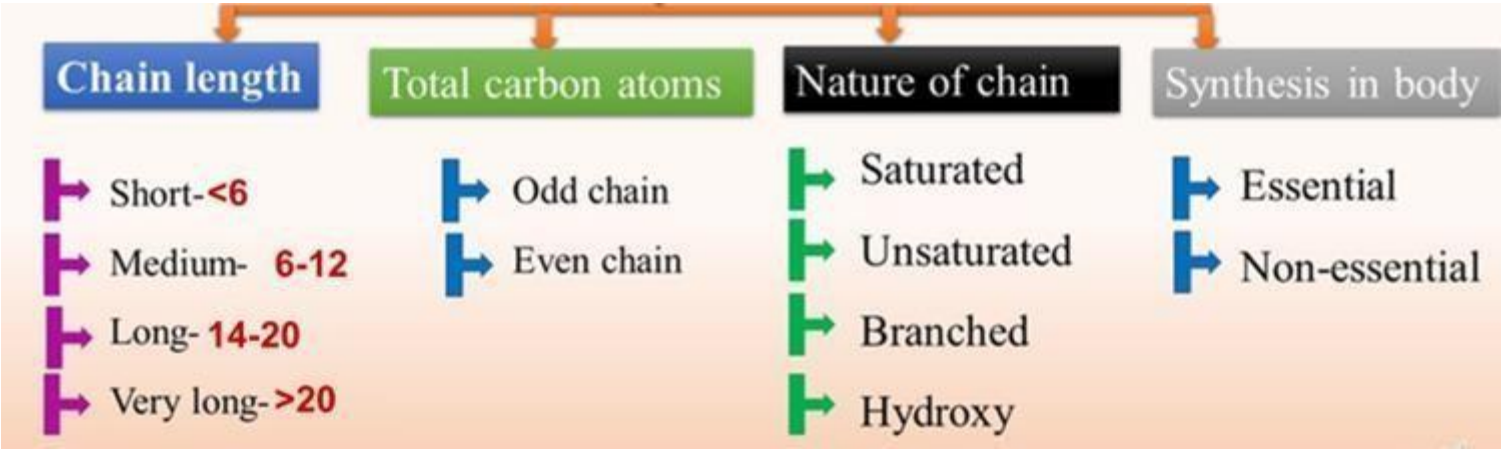
Sphingosine has 2 hydroxyl groups, amine group, and a double bond within the hydrocarbon chain.





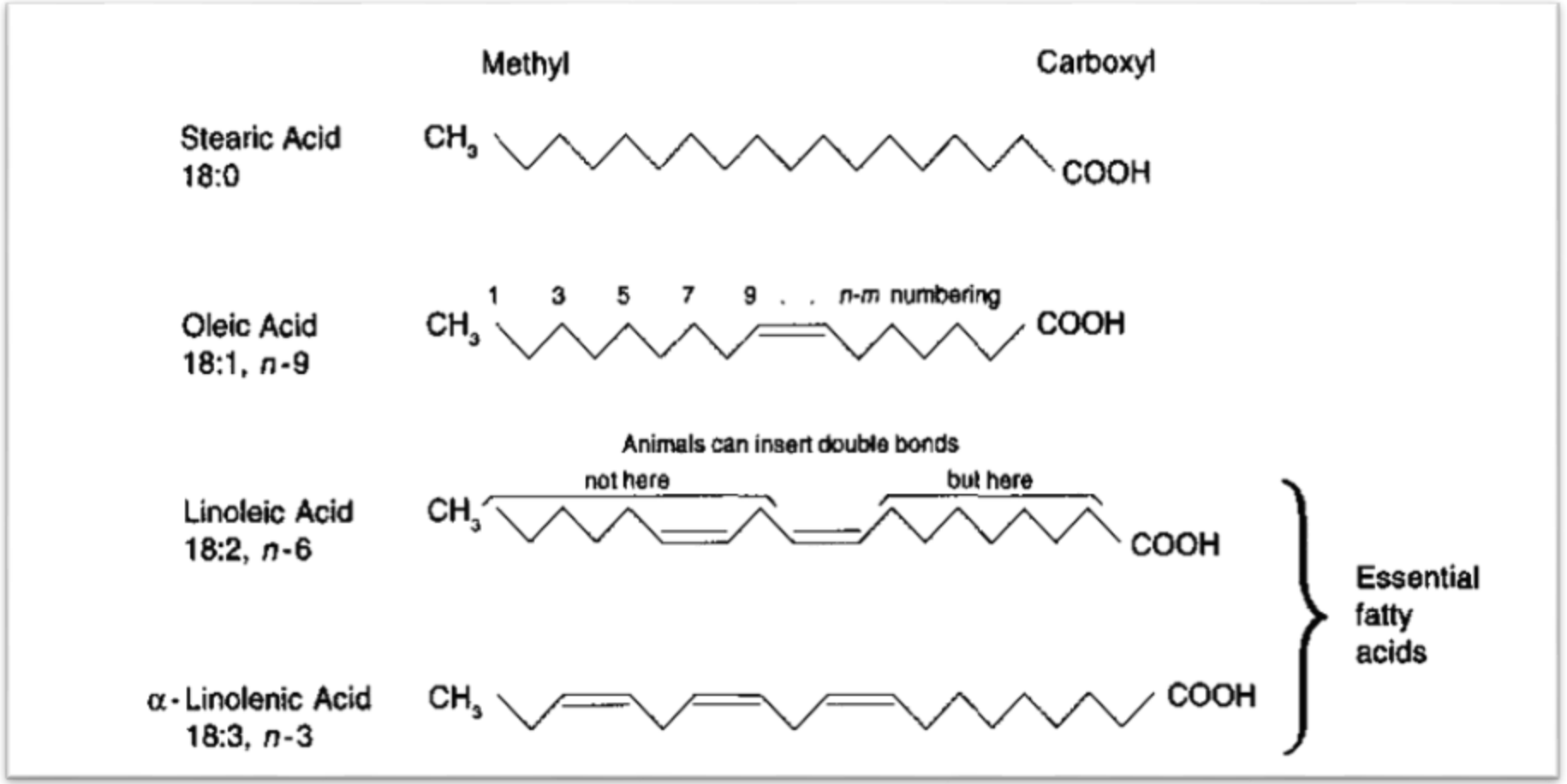
FattyAcids

- Double bonds in FA are always spaced at three-carbon intervals.
- The addition of double bonds decreases the melting temperature (Tm) of a fatty acid.
- Increasing the chain's length increases the Tm.
- Membrane lipids typically contain unsaturated long-chain fatty acids (LCFA) to maintain fluidity.
- Fatty acids with double bonds beyond the 10th carbon are essential.



Notice the different classifications of FAs, the predominant FAs in our bodies is even with 16 and 18 carbons chain.

FA: Fatty Acid



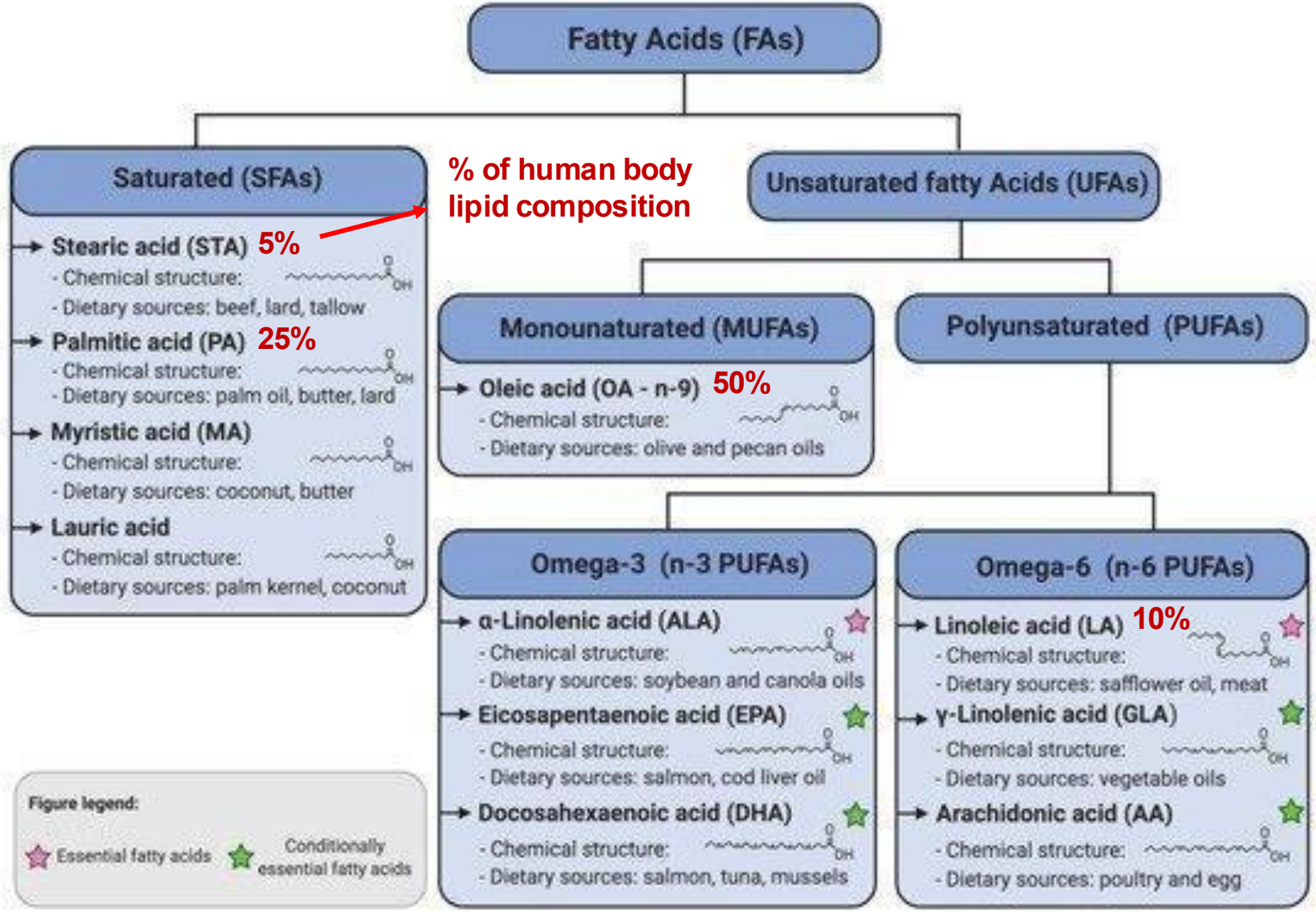
- ***One of the classifications of FAs is:***
  - Essential FAs: linoleic acid and linolenic acid, we get them from our diet as our bodies they can't synthesize FAs that have a very long chain with double bonds farther than carbon number 10 or 12; as we don't have the enzyme that can introduce these double bonds.

  - Non-essential FAs: can be synthesized in our bodies.

Not all the long unsaturated FAs are essential, only linoleic and linolenic acids are essential; as they are used in the synthesis of arachidonic acid.

FattyAcids

- Stearic (18C) and Palmitic (16C) acids (Saturated FAs) constitute 30% of total body FAs.
- Palmitic acid is present more as it is used more in the synthesis of other FAs and as lipid anchor for proteins by binding to them covalently, in a process named palmitoylation; myristylation also happens using myristic acid.
- Oleic acid, which is a monounsaturated FA, constitutes around 50% of FAs.



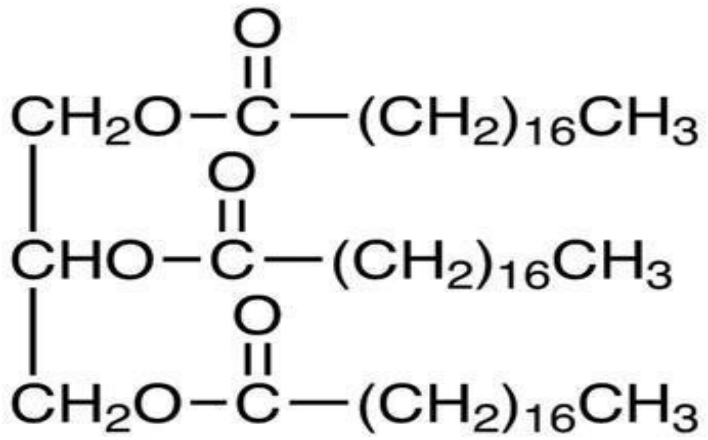
Forms of fatty acids

- **Free fatty acids** (FFA): occur in all tissues and in plasma (particularly during fasting).
  - >90% of the plasma fatty acids are in the form of fatty acid esters (primarily TAG, cholesteryl esters, and phospholipids) carried by circulating lipoprotein particles. (attached to other structures)
  - ➢ FFA is found under fasting conditions, glucagon gets released and the binding of it to lipocytes will increase the hydrolysis of the ester bonds in TAG.
  - Plasma FFA are transported on albumin from adipose tissue (where they are stored) to most tissues (where they are needed to provide energy).
  - ➢ As long chain FAs have big hydrophobic parts, they can't move by their own through the blood, binding to albumin will mediate their transport, short and medium chain FAs move by their own.
- **FFA can be oxidized** (broken up into acetyl CoA) in many tissues:
  - Liver and muscle, to provide energy
  - ➢ Under fasting conditions, the liver extracts energy by introducing the acetyl-CoA molecules, that are provided from the catabolism of FAs, into the Krebs cycle, remember that under fasting condition, the liver will also start the pathway of gluconeogenesis to provide glucose for other tissues, both processes of gluconeogenesis and Krebs cycle consumes oxaloacetate, so the liver will balance these pathways and use the excess acetyl-CoA in ketogenesis.
  - Liver to synthesize ketone body

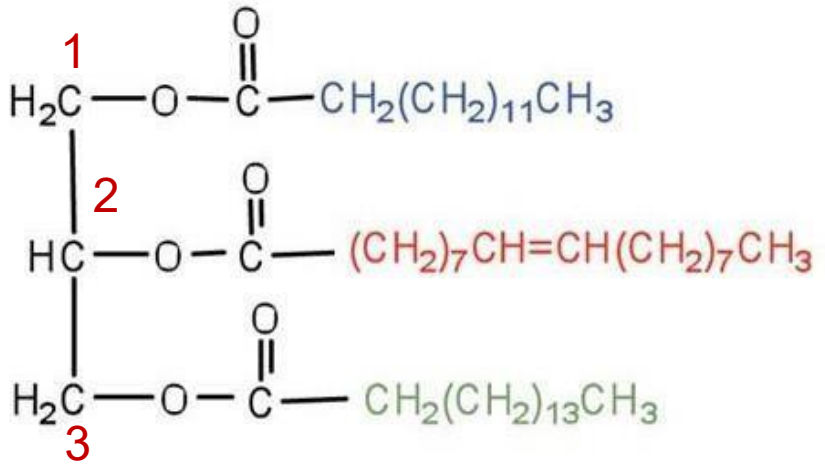
Forms of fatty acids-pt.2

- **Structural FA**: membrane lipids as phospholipids and glycolipids.
- **Protein-associated FAs** facilitate membrane attachment.
  - Such in the FAs that are used in palmitoylation and myristylation.
- **FAs are precursors** of the hormone-like prostaglandins.
  - Synthesis of arachidonic acid from fatty acid precursors (specifically linoleic acid), arachidonic acid
    - is used for the synthesis of prostaglandins, thromboxanes, leukotrienes.
- **Esterified FAs**: in the form of TAG stored in white adipose tissues as the major energy reserve of the body. Under fasting conditions
  - FAs are esterified to cholesterol (in FAs associated to lipoproteins) or esterified to TAG (the storage form of lipids in lipocytes).

Triacylglycerol



Tristearin
a simple triglyceride

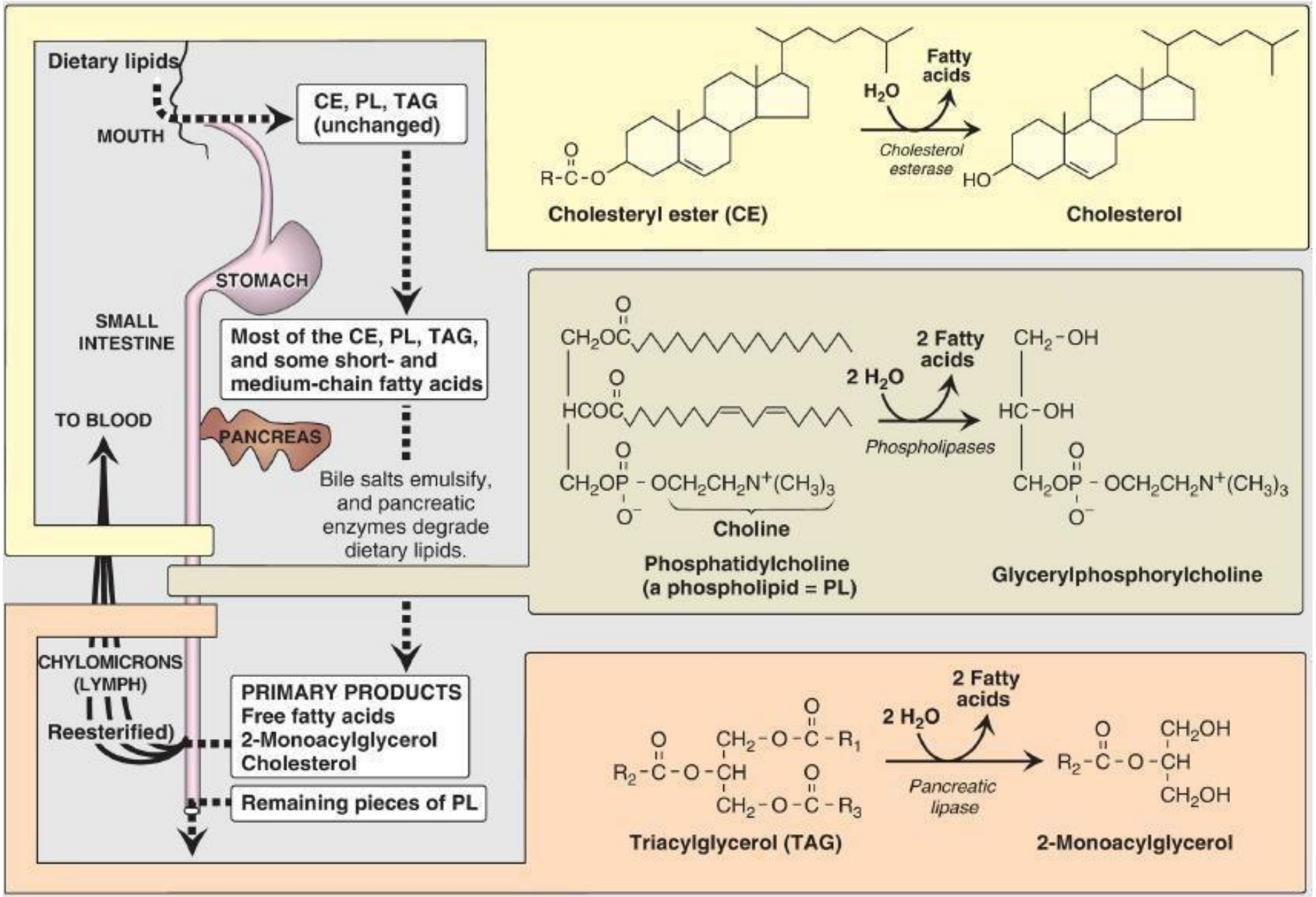


a mixed triglyceride

TAG is composed of a glycerol molecule attached to three FAs with ester bonds, the FAs could be all saturated, all unsaturated or mixed, usually the middle one is unsaturated.

Digestion oflipids

More complex than sugar digestion since there are many different types of lipids with different bonds to be digested



Steps of Lipid Digestion

1. **Oral cavity (lingual lipase):**
   - Secreted from the back of the tongue and is then mixed with the saliva.
   - Separates FAs from the backbone in TGAs.
   - A small % of FAs are produced as food stays in the mouth only for little time.
   - Only hydrolyzes Small- and Medium-chain FAs (SCFAs and MCFAs, respectively).
   - Acid stable; it can work in the stomach after it moves with the food (see next point).

2. **Stomach (gastric lipase):**
   - Secreted from the gastric mucosa.
   - Same function as lingual lipase (and only SCFAs and MCFAs as well).
   - Both gastric and lingual lipases function in the stomach (acid stable).

3. **Intestines (specifically the duodenum):**
   - Pancreatic lipase hydrolyzes long-chain FAs (LCFAs) from TAGs.
     - The presence of LCFAs in the diet inhibits lingual and gastric lipases.
     - Pancreatic lipase cleaves both FAs at C1 and C3 of the glycerol backbone of TAGs.
     - The result is a mixture of 2-monoacylglycerols and free FAs.
   - Phospholipase:
     - Cleaves both FAs of glycerophospholipids, leaving glycerylphosphorylcholine.
   - Cholesterol esterase:
     - Hydrolyses cholesterol esters, producing cholesterol and free FAs.

Digestion in the stomach

- Acid-stable lipases: lingual lipase and gastric lipase (responsible for 30% of lipid hydrolysis)
- They have an optimum pH of 2.5 – 5.
- They do not require bile acid or colipase for optimal enzymatic activity **(their substrate are polar [SCFA & MCFA]).**
- Gastric lipase will be stopped by long chain free fatty acids
- Main target: triacylglycerols with short- and medium-chain fatty acids (≤ 12 carbons)
- Significant in infants and patients with pancreatic lipase deficiency or pancreatic insufficiency (e.g., cystic fibrosis).
  - The action of lingual lipase is significant in newborn infants.
- Short- and medium-chain fatty are absorbed in the stomach.
- **Short- and medium chain FAs are the main FAs in breast milk.**

Unlike pancreatic lipase which needs emulsifiers for LCFAs

Fatty acids	*Human milk*[a] *%*
4:0	—
6:0	—
8:0	0.16
10:0	1.82
10:1 + 11:0	—
12:0	7.89
13:0	—
14:0	9.45
14:1 + 15:0 + 15:1	0.84
16:0	22.78
16:1 + 17:0 + 17:1	3.04
18:0	6.51
18:1 (*n*-9)	28.72
18:2 (*n*-6)	15.12
18:3 (*n*-6)	0.15
18:3 (*n*-3)	0.82
20:0	0.40
20:1	0.21
20:2	0.31
20:3 (*n*-6)	0.53
20:4 (*n*-6)	0.52
20:5 (*n*-3)	0.10
22:0	—
22:1	—
22:4 (*n*-6)	0.08
22:5 (*n*-6)	0.01
22:5 (*n*-3)	0.17
22:6 (*n*-3)	0.32
24:0	0.04

Extra Reading - Wetnursing(الإرضاع)

- Breast milk was found to contain microvesicles that contain mRNA and the enzyme reverse transcriptase, which can form cDNA from mRNA and implement it into the infant's genome!
- Breast milk can also influence the DNA of the infant epigenetically by methylation (inactivating some genes).

RESEARCH ARTICLE

Breastfeeding effects on DNA methylation in the offspring: A systematic literature review

Fernando Pires Hartwig[1,2]*, Christian Loret de Mola[1], Neil Martin Davies[2,3], Cesar Gomes Victora[1], Caroline L. Relton[2,3]

1 Postgraduate Programme in Epidemiology, Federal University of Pelotas, Pelotas, Brazil, **2** MRC Integrative Epidemiology Unit, School of Social & Community Medicine, University of Bristol, Bristol, United Kingdom, **3** School of Social and Community Medicine, University of Bristol, United Kingdom

* fernandophartwig@gmail.com

Ozkan *et al. Clinical Epigenetics* 2012, **4**:14
http://www.clinicalepigeneticsjournal.com/content/4/1/14

CLINICAL EPIGENETICS

HYPOTHESIS Open Access

Milk kinship hypothesis in light of epigenetic knowledge

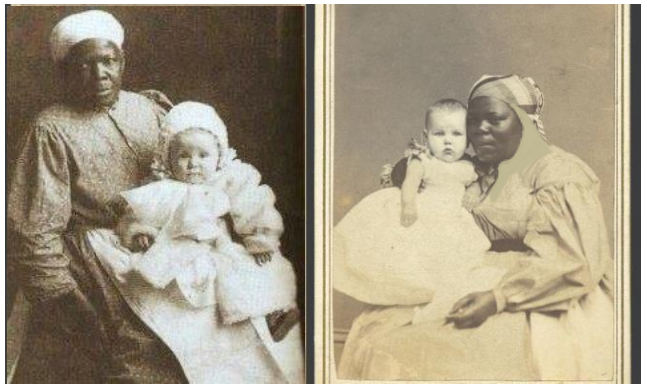
Hasan Ozkan*, Funda Tuzun, Abdullah Kumral and Nuray Duman

THEORETICAL BIOLOGY AND MEDICAL MODELLING

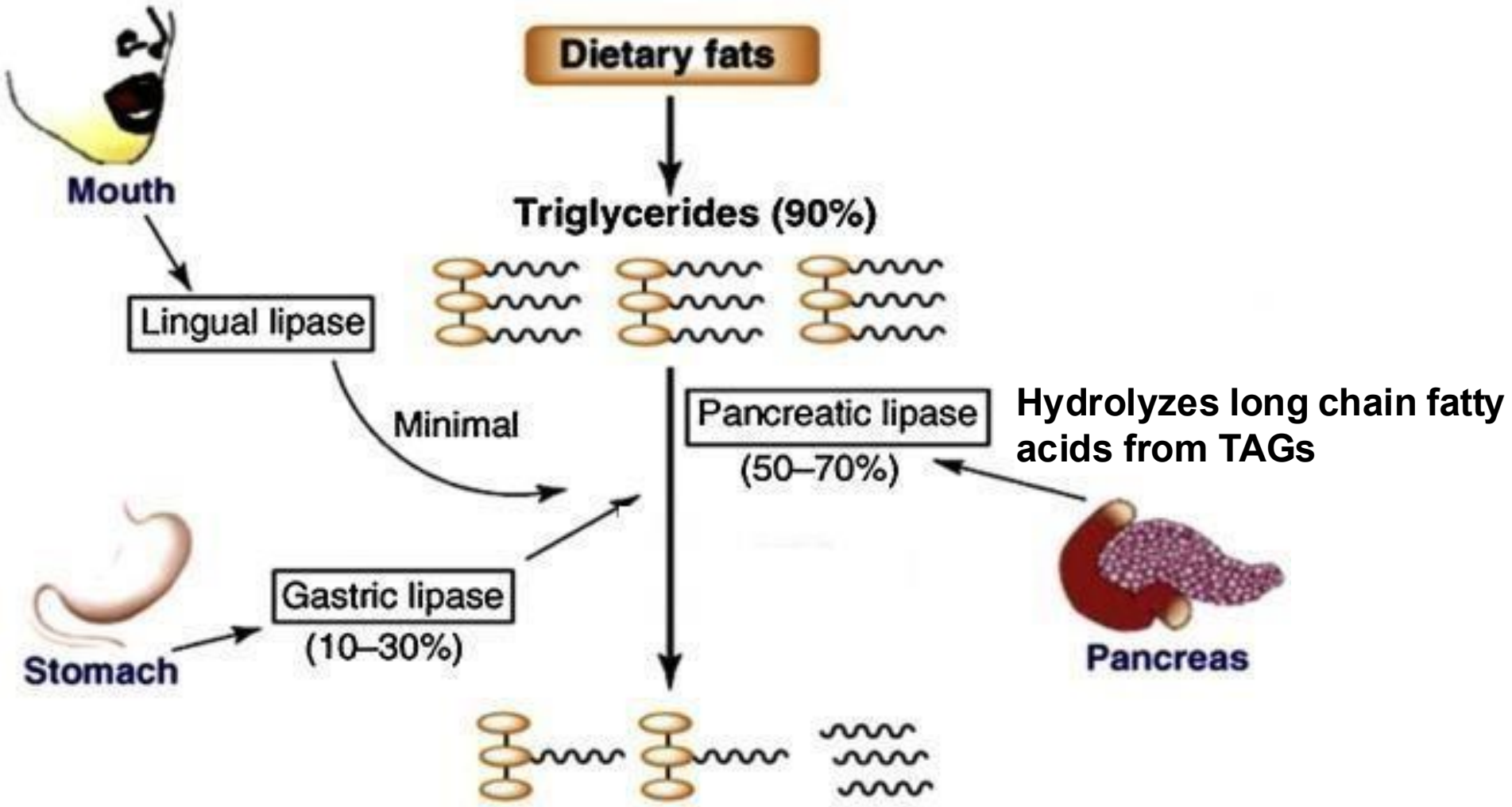
REVIEW Open Access

Integration of maternal genome into the neonate genome through breast milk mRNA transcripts and reverse transcriptase

M Kemal Irmak[1*], Yesim Oztas[2] and Emin Oztas[3]

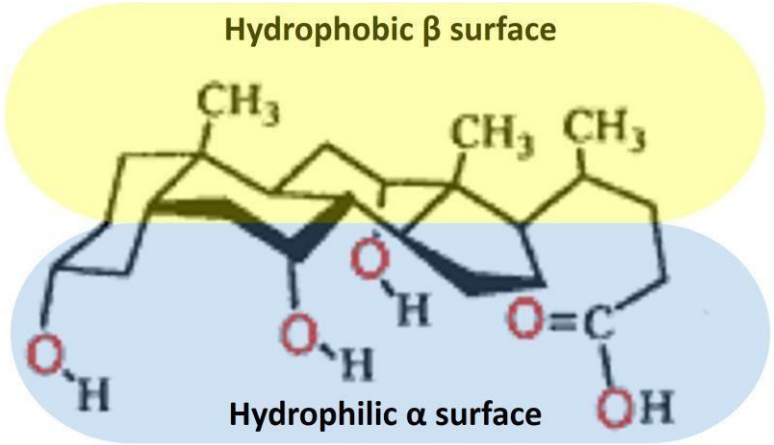


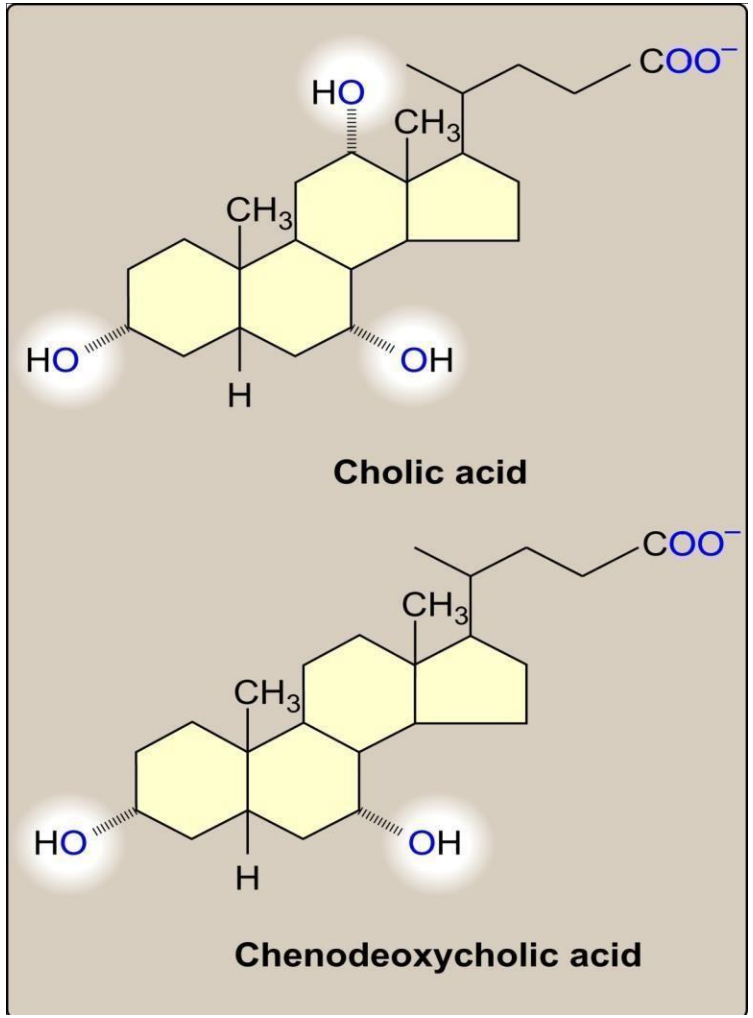
Degradation of triacylglycerol

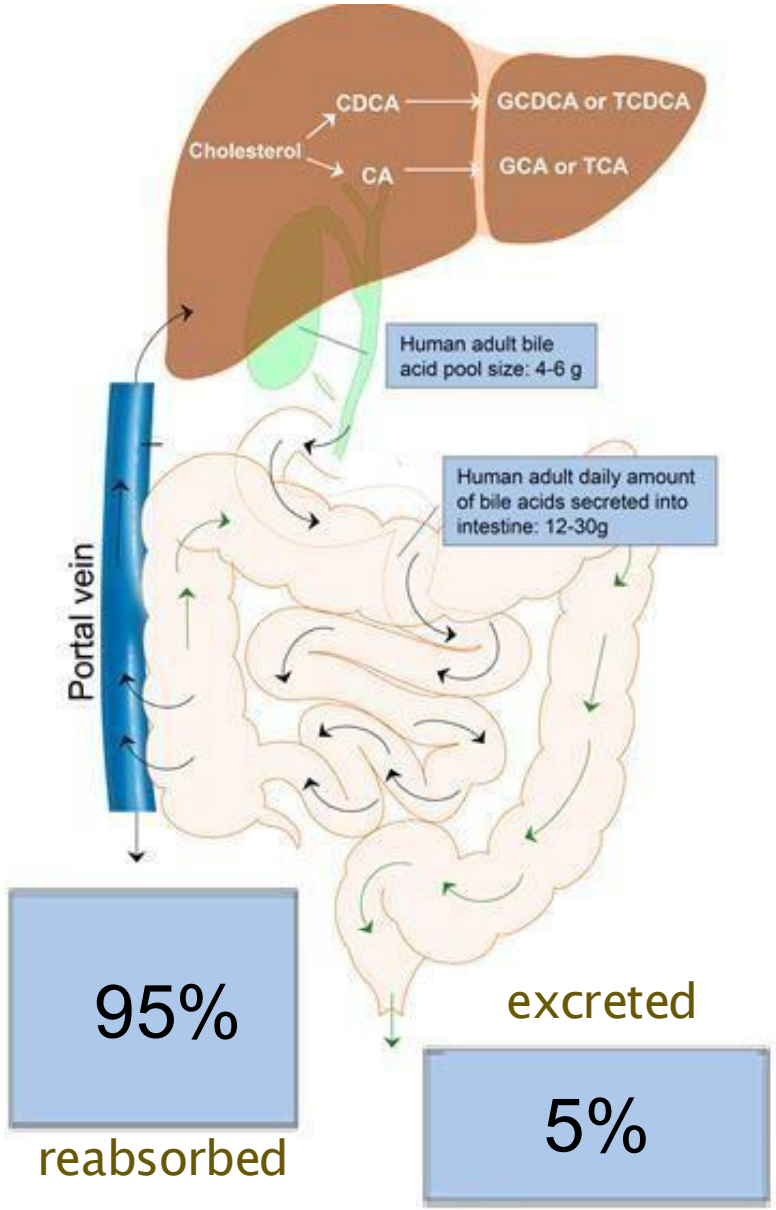


Emulsification: from drops to droplets

- Emulsification is defined as a process where one liquid is dispersed as small spherical droplets in a second immiscible (not homogeneous) liquid.
- Two mechanisms of emulsification in the duodenum:
  - Peristalsis: mechanical mixing leading to smaller droplets
  - Conjugated bile salts







Emulsifiers

Liver synthesizes and gallbladder stores.
After cholecystectomy, the patient cannot handle high-lipid meals as there is no stored bile acids.

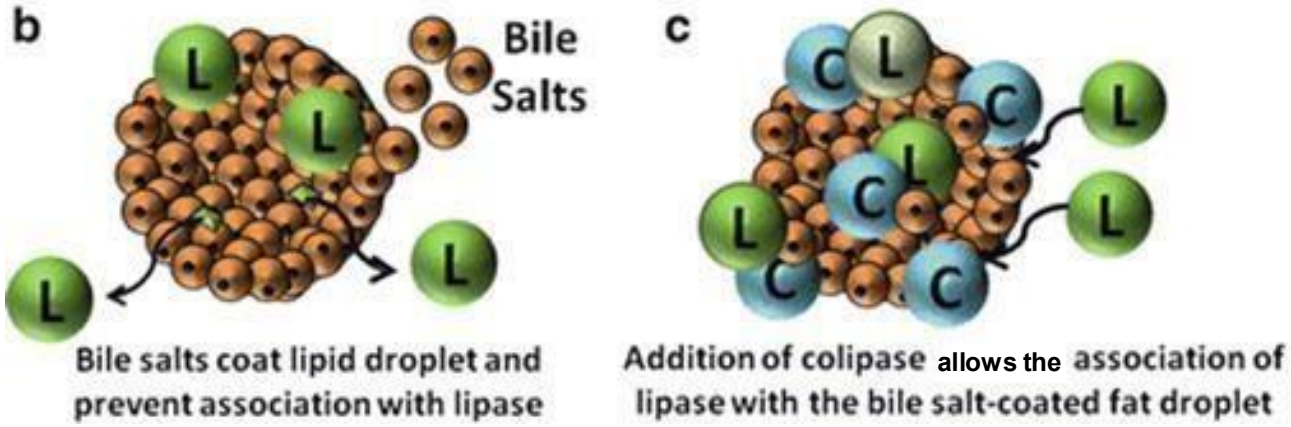
- Cholesterol-derivative bile acids (from gallbladder -> duodenum).
- Cholesterol is modified by adding more polar groups to it, making it have 2 surfaces, a polar one with all the polar groups, and a nonpolar one with the 4 hydrocarbon rings.
- See the previous slide bottom left for illustration.
- The polar surface binds the pancreatic lipase, while the nonpolar surface binds the nonpolar substrate (TAGs with LCFAs); this allows the close interaction that is needed for the catalytic activity to be done.
- Some cholesterol-lowering drugs increase the excretion of bile acids, forcing the liver to use cholesterol to synthesize new bile acids for emulsification since the reabsorbed amount is less.

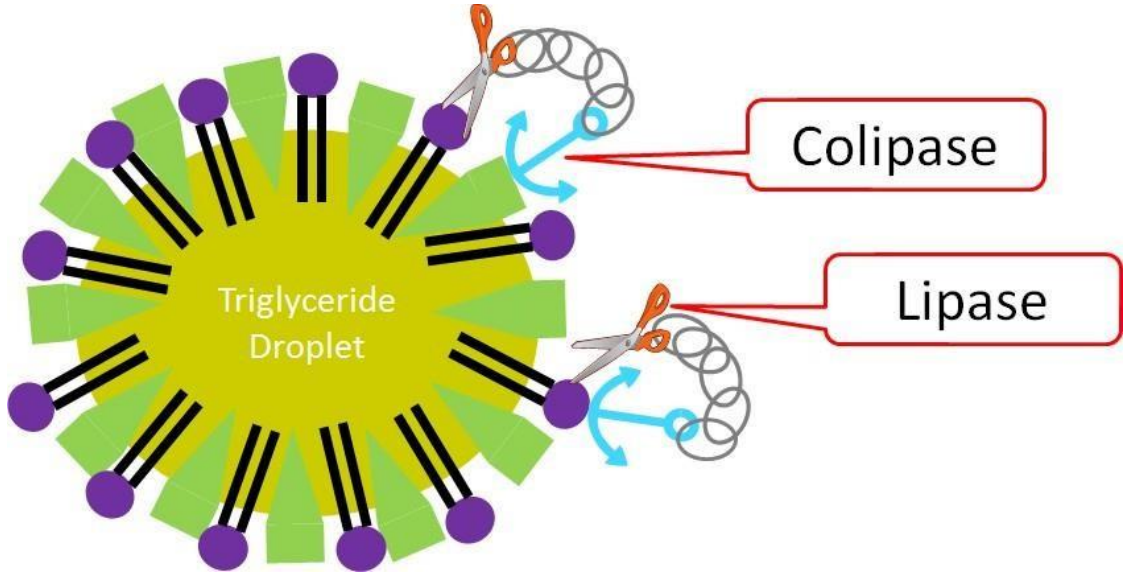
Quick Revision

- Lipid family consists of a heterogeneous compounds, which are hydrophobic (with small negligible polar part), meaning that their digestion is variable (due to heterogeneity) and complex as these compounds are hydrophobic making their adhesion to enzymes less efficient.
- The first stage in lipids digestion is on oral cavity by lingual lipase enzyme, and it hydrolyses ester bonds between short or medium-chain fatty acids and glycerol, but because of the low contact time it's not that effective.
- Secondly the previous enzyme and food is transported to the stomach where another one is introduced (gastric lipase) that works by the same mechanism alongside lingual lipase as both are stable at acidic conditions.
- At the last stage, after food particles with the enzymes and HCl from stomach to small intestines, where pancreatic enzymes are excreted to such as pancreatic lipase the one that hydrolase long-chain fatty acids, and due to the higher hydrophobicity of LCFAs, emulsifiers are needed (while in low and middle-chain fatty acids, we don't need them).

Pancreatic lipase: The significance of colipase

Pancreatic lipase is an interfacial enzyme that is most active at an oil-water interface





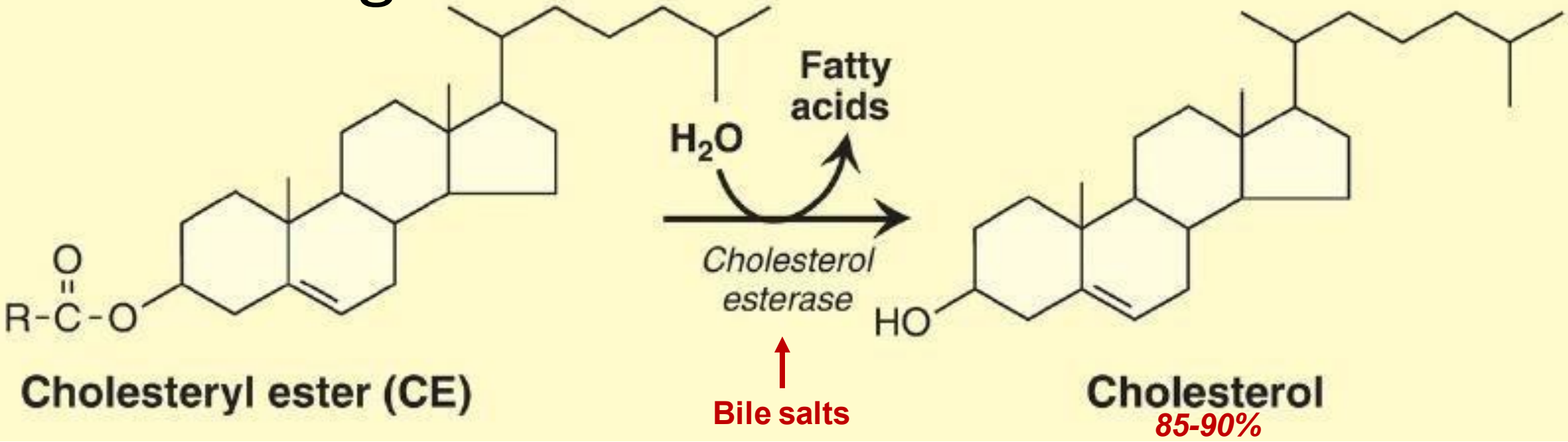
Combined pancreatic lipase-colipase deficiency is an orphan disease

- Emulsifiers (bile acids and salts) are made from cholesterol with a steroid nucleus in the center (big and rigid in structure) with the polar surface interacting with the enzyme and non- polar surface associated with fatty substrates.
- Colipase works by making a room for lipase to do hydrolysis, as it anchors lipase to a fat droplet which is covered with bile acids.

Colipase:

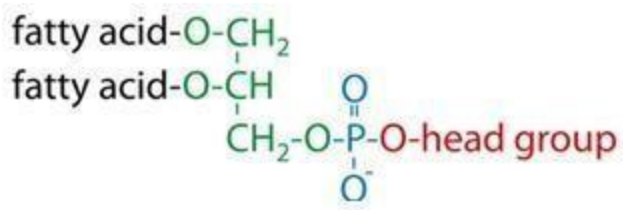
- Secreted as a zymogen (proenzyme) from the pancreas
- Activated by trypsin (By cleavage) in the small intestines
- Anchors lipase into the micelle interface at a ratio of 1:1
- Restores activity of lipase against inhibitors

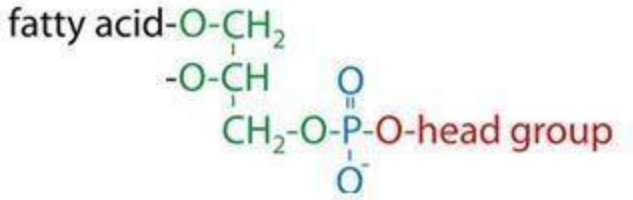
Degradation of cholesterol esters

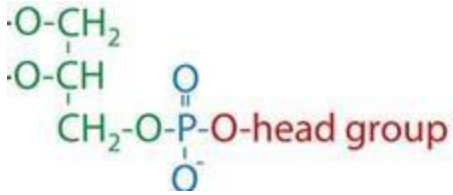


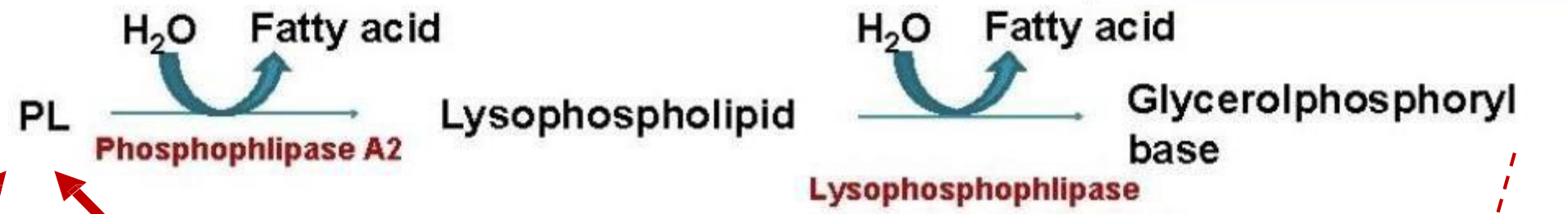
Lipids in food are mainly TAG from oils and fats, cholesterol or cholesterol esters and phospholipids. Cholesterol esters have lost the 'OH' of cholesterol and are highly hydrophobic; they thus need emulsifiers for their degradation (into cholesterol and fatty acid) by cholesterol esterase.

Degradation of phospholipids









Pro-PLA2

Bile salts

Trypsin

SEE NEXT SLIDE

- ***Excreted in the feces***
- ***Further degraded***
- ***Absorbed***

Regarding last slide:

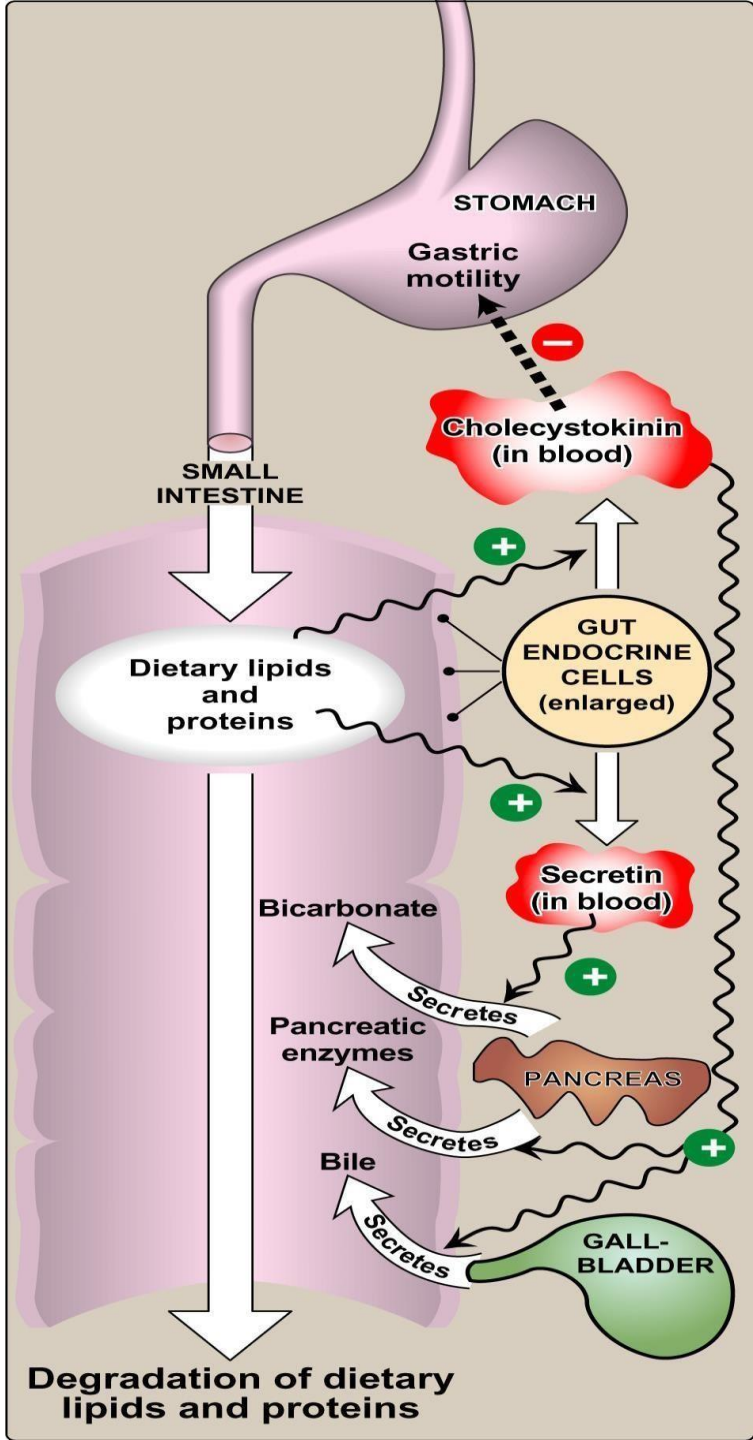
- Phospholipids are degraded by **phospholipase A2**, an enzyme that
  - Is secreted as a proenzyme and cleaved by trypsin; gets activated.
  - Needs emulsifying bile acids and salts for its function
  - acts by hydrolyzing fatty acids on the second carbon, producing a lysophospholipid that continues the process and gets hydrolyzed by **lysophospholipase**, so the second fatty acid is stripped, producing a glycerophosphoryl base (glycerol and phosphate group associated with a head group), which has different fates:
    - excretion by feces
    - Absorption
    - further degradation to benefit from its components like glycerol, phosphate and head groups.

Phosphatidyl choline (lecithin) is the major phospholipid in our food.

Hormonal control

- Entry of food (chyme) induces the release **cholecystokinin** (CCK; a peptide hormone) from the duodenum and jejunum.
  - Induces contraction of the gallbladder to release bile (bile salts, phospholipids, and free cholesterol)
  - Acts on the exocrine pancreatic cells to release digestive enzymes

    Like lipase and colipase. Activates the contraction of the gall bladder to release the bile and salts and other components into the small intestine to facilitate the emulsification
  - Decreases gastric motility to slow down the release of gastric contents So the release of fats is gradual and the process is more efficient, it's the reason why the digestion of fats takes a long time.
  - The low pH of the chyme entering the intestine induces intestinal cells to produce **secretin** (a peptide hormone).
    - Causes the pancreas to release a bicarbonate-rich solution to neutralize the pH (as the kinins has low PH) and make it optimal for the digestive pancreatic enzymes.
    - Inhibits gastric motility.



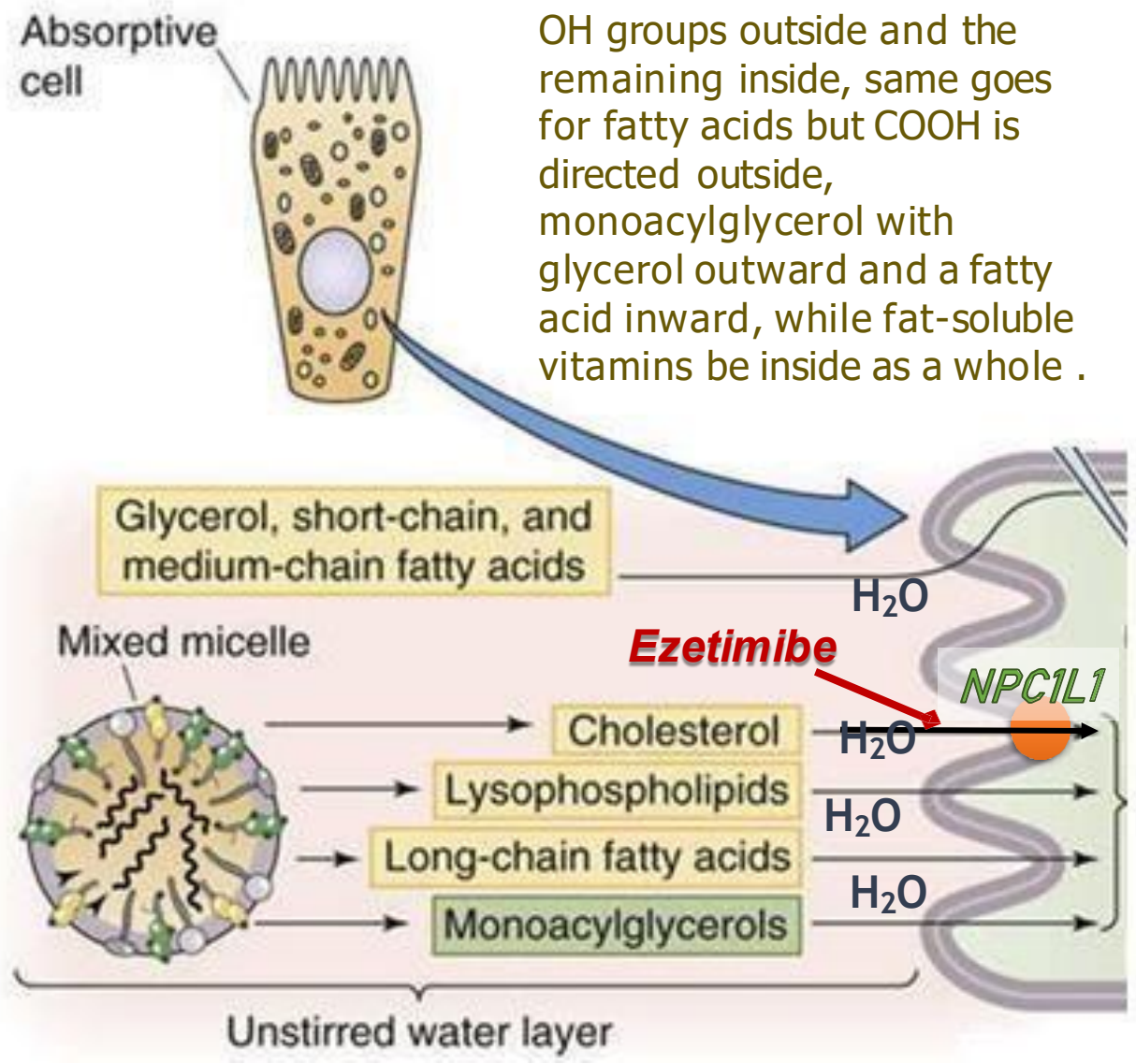
Hormonal control

Bicarbonate is secreted by pancreas after induction by secretin, because when food is brought from stomach it's acidic which is not favored by pancreatic lipase, as a result bicarbonate works by neutralizing the acidity and creating an optimal pH.

Absorption by enterocytes

- Mixed micelles are formed in the lumen from free fatty acids (FFA), monoacylglycerol, free cholesterol, bile salts, and fat-soluble vitamins.
- Cholesterol absorption is aided by an increase in dietary fat components and is hindered by high fiber content.
- The Niemann-Pick C1 like 1 protein (NPC1L1) is a sterol influx transporter (at the apical membrane) that facilitates the uptake of cholesterol via vesicular endocytosis
- Ezetimibe inhibits cholesterol absorption by suppressing the internalization of NPC1L1/cholesterol complex.
- The uptake of fatty acids across the enterocyte brush-border membrane occurs by passive diffusion and by protein–mediated mechanisms.
- Short- and medium-chain FAs are directly absorbed by passive diffusion.

Micelles are polar outside and non-polar inside, meaning that cholesterols in it direct OH groups outside and the remaining inside, same goes for fatty acids but COOH is directed outside, monoacylglycerol with glycerol outward and a fatty acid inward, while fat-soluble vitamins be inside as a whole .

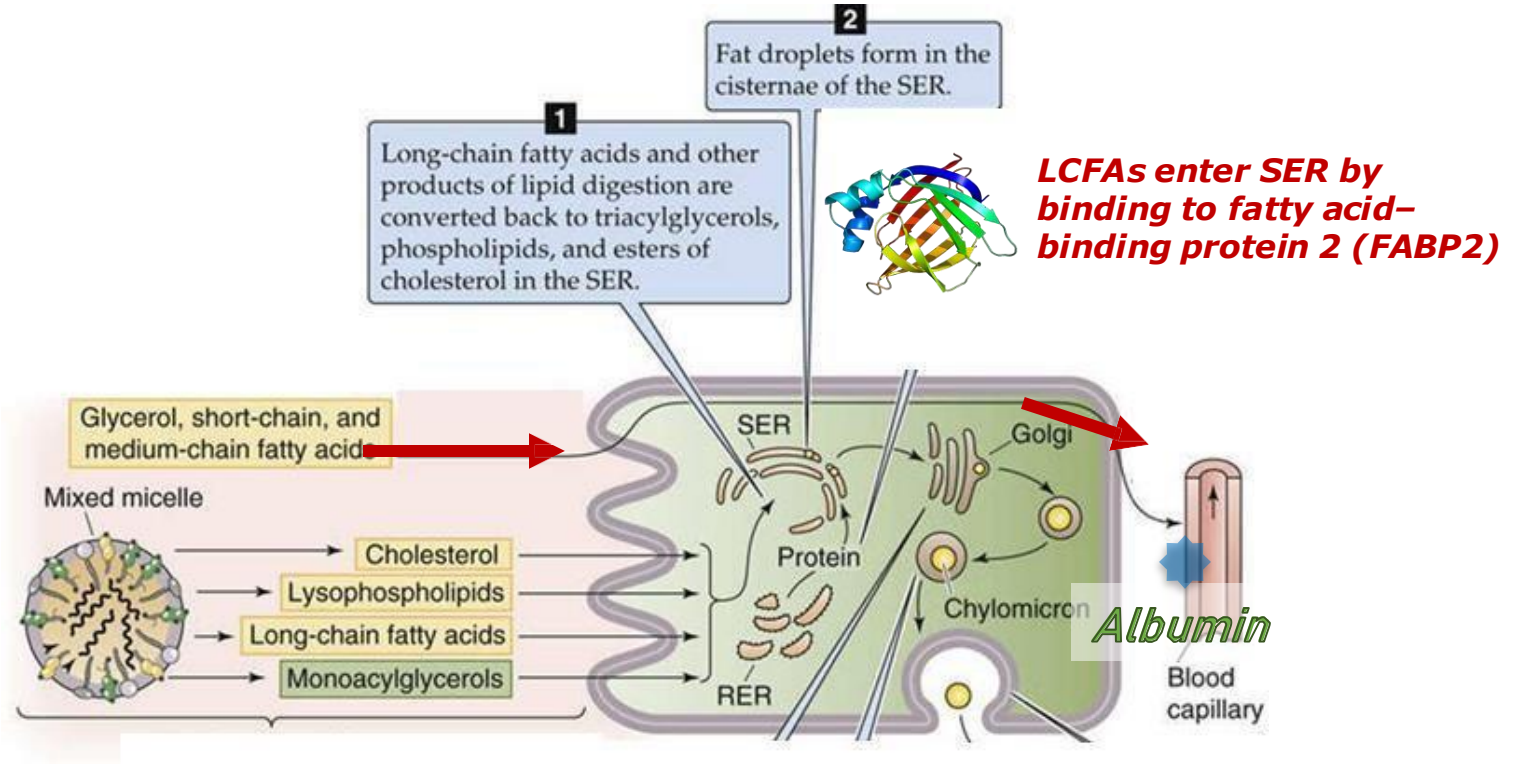


- After absorption, reverse processes are done in SER to reform complex fats (it were simplified only to permit absorption, but we need them as complex molecules to perform their biological functions in the body.

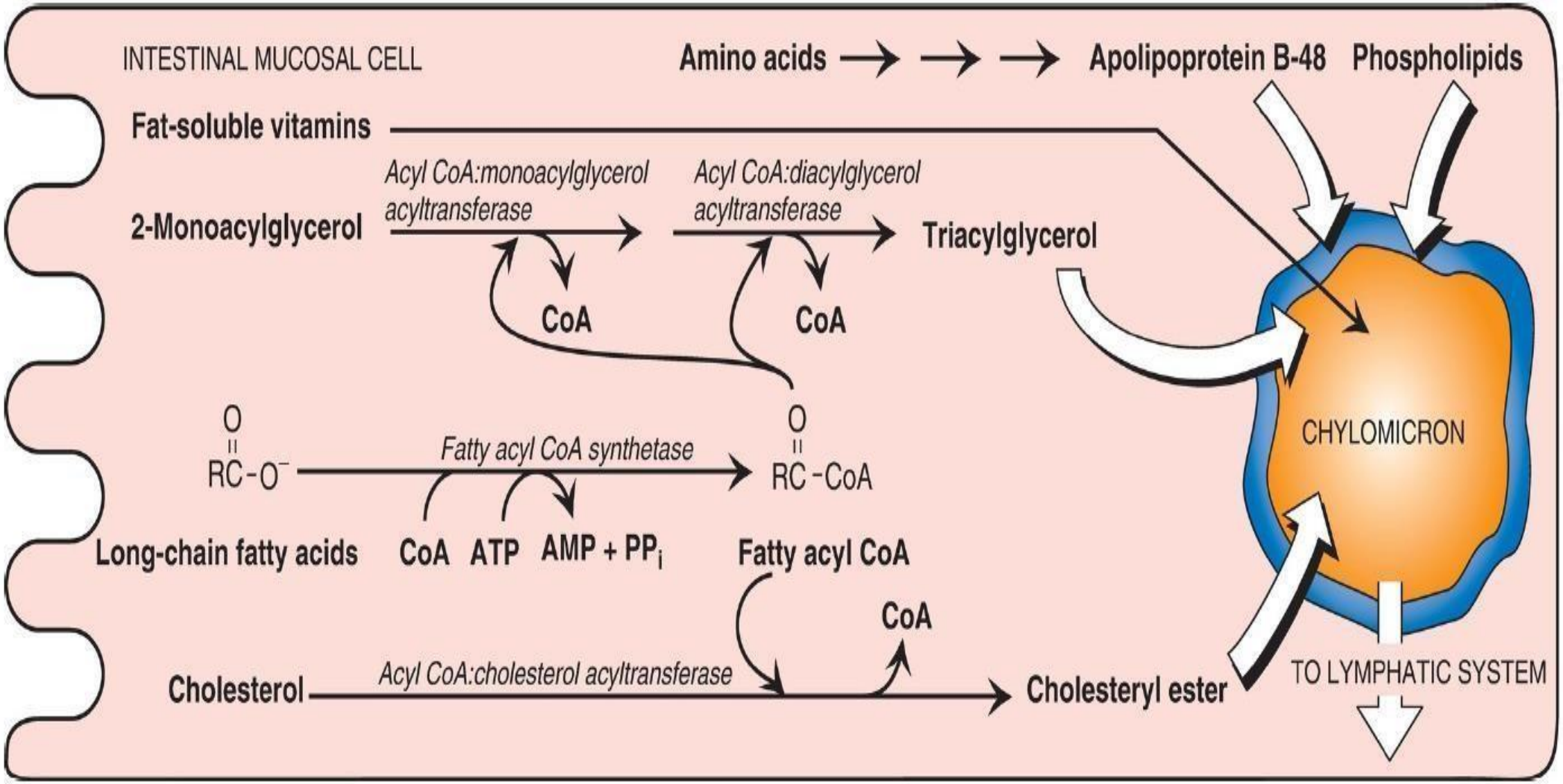
SEE next slide and focus on the synthetic pathways of cholesterol esters and TAGs.

- All of these lipids are combined to make chylomicrons, beside some proteins like apolipoprotein B48 (made at RER).
- Chylomicrons have a high percentage of TAGs and a lower one of cholesterol, and after its formation it goes to the basolateral surface going to the lymphatic system by lacteal capillary, then going to veins and entering circulation like portal circulation indirectly.

Reformation of complex lipids

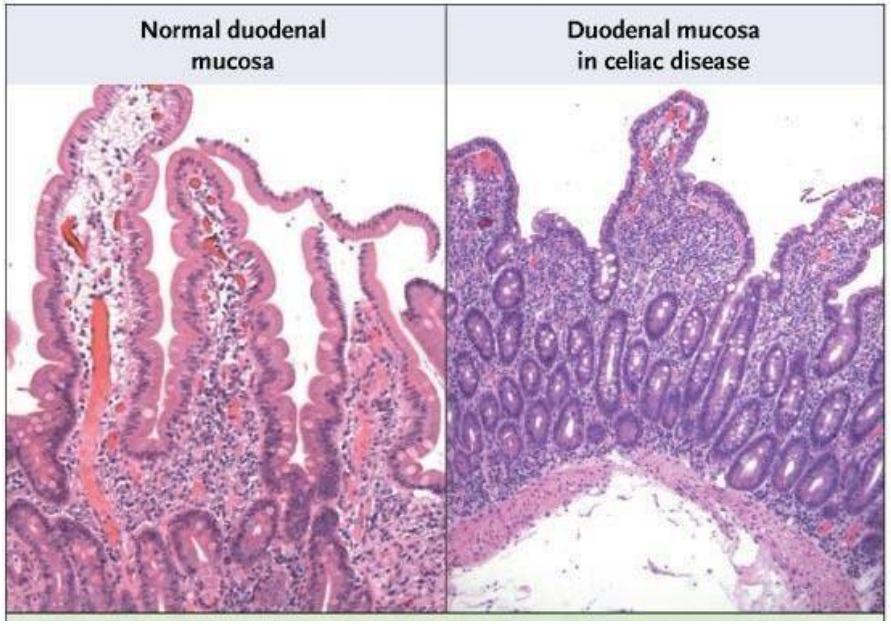


YOU HAVE TO KNOW EVERYTHING ABOUT THIS FIGURE



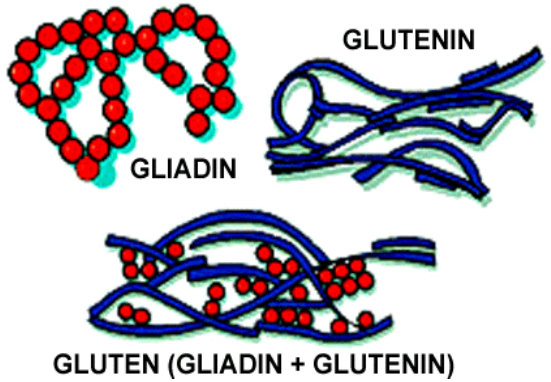
Celiac disease (CD)

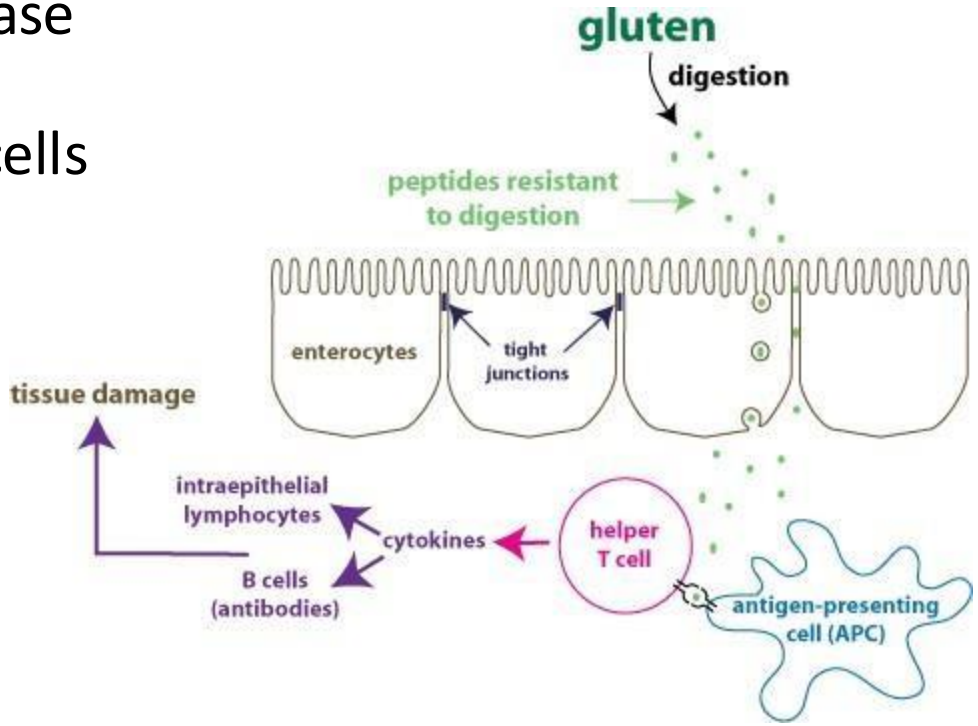
- Fat malabsorption leading to steatorrhea (excess lipids in feces) Induce an immune response that results in cellular death of intestinal cells resulting in inflammation
- It is an autoimmune response to gliadin, a peptide found in gluten (wheat, rye, and barley).
- Gliadin contains many proline (14%) and glutamine (40%) residues, making it resistant to digestion. As a consequence of high proline content (relatively)
- Lab tests: the presence of anti-tissue transglutaminase (anti-tTG) antibodies.
- Tissue biopsy: absence of villous surface epithelial cells resulting in decreased nutrient absorption.



Principal causes of steatorrhea:

1. ***Short bowel disease***
2. ***Liver or biliary tract disease***
3. ***Pancreatic exocrine insufficiency***
4. ***Cystic fibrosis***





http://courses.washington.edu/pbio376/celiac/celiacdisease-376.html

Celiac disease (CD)

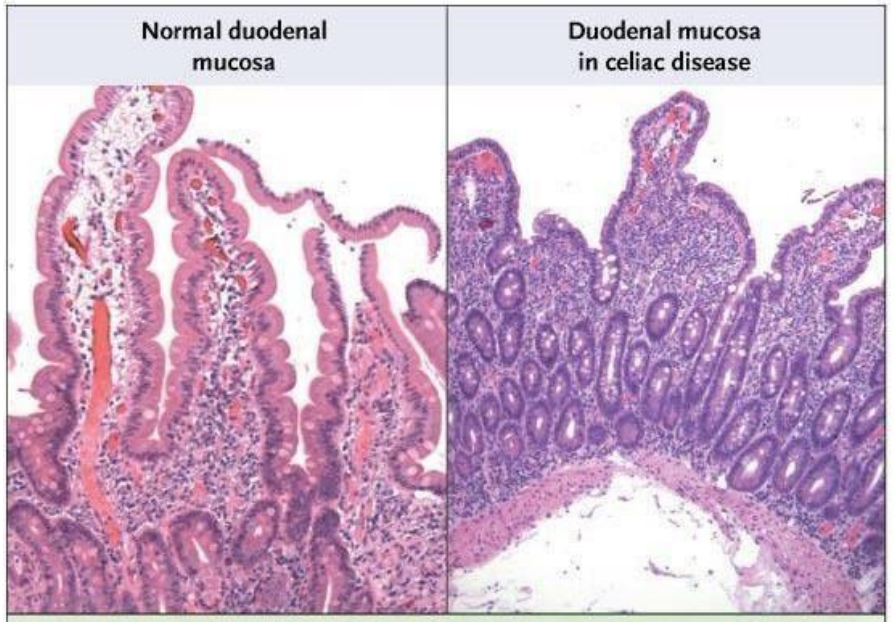
Affects all dietary components not only lipids

This leads to higher osmotic pressure in the lumen of the small intestine that results in diarrhea and gases

The only component of diet that can be seen by the patient is lipids because they are undigested and water insoluble

Further notes

- Lipids are one of the easiest ways to detect GI digestion diseases as it's hydrophobic therefore it clusters and is visible macroscopically unlike proteins or carbohydrates.
- Lipids droplets in feces is called steatorrhea, which isn't congenital but rather an autoimmune disease.
- Gluten is composed of gliadin (high proline content) that impedes digestion.
- Gluten index is a quality indicator; the lower the better.

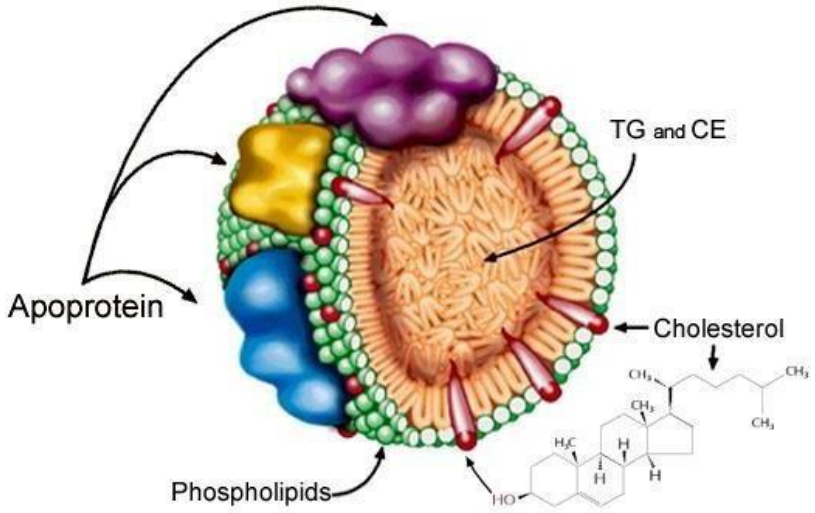


Purple color comes from inflammatory cells that invade the area, and surface area is lower due to lower cell number as a consequence of cellular damage and leads to a decrease in enzymes and transporters for absorption and digestion.

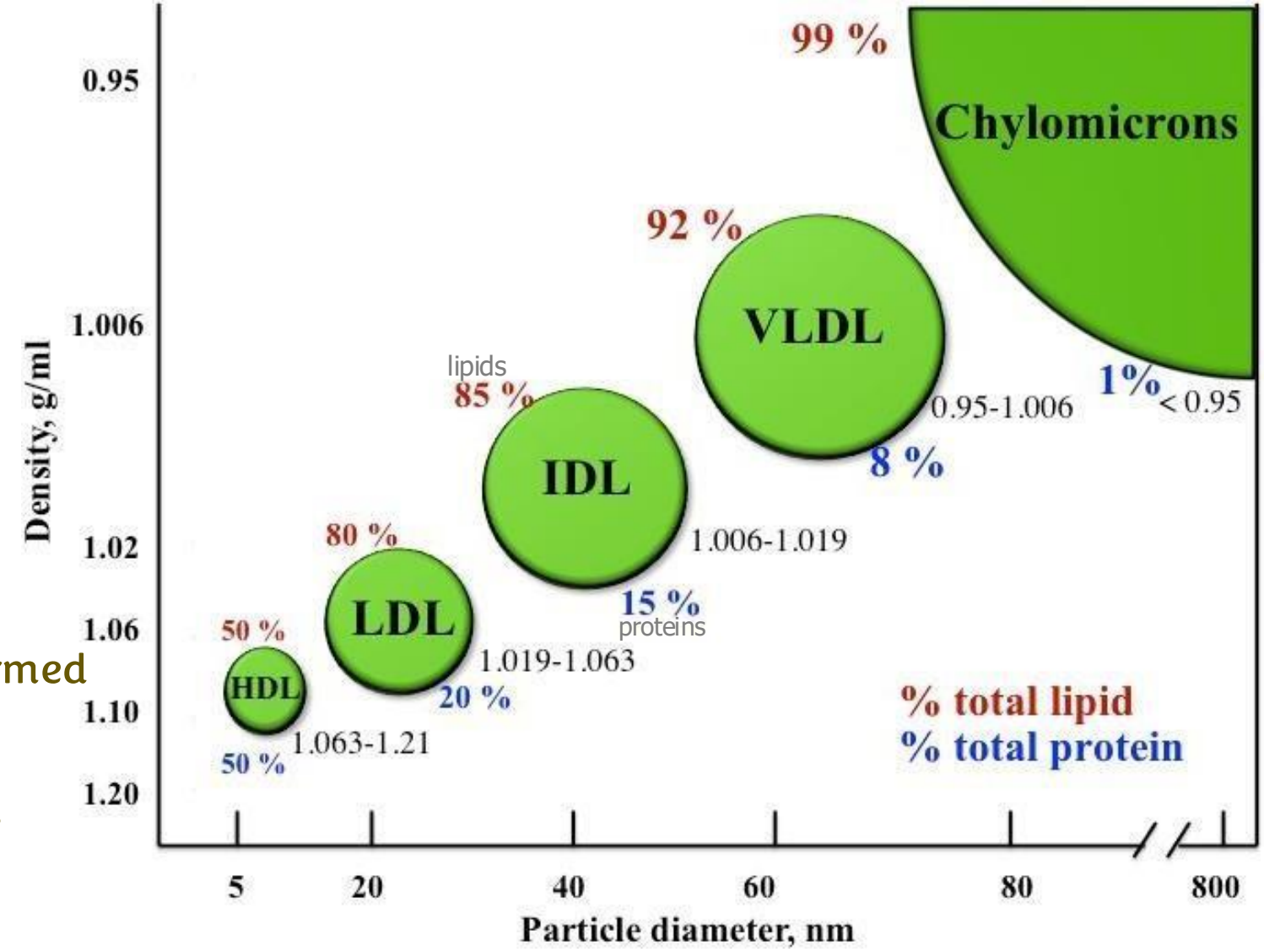
(here there is a chronic inflammation as a result of gluten allergy resulting in cellular death with time.)

Cystic fibrosis which is a congenital disease that affects all exocrine glands and leads to steatorrhea, it's more common in western countries especially for jews. In addition, its cause is a mutation in CFTR gene which codes for a chloride channel that affect water transport leading to thickness of exocrine secretions impeding its passage in respiratory and GI tract.

Lipoproteins



Function: transport of lipids (cholesterol, cholesterol esters, phospholipids & triacylglycerols) in blood plasma.



As lipid content increases, the density decreases

Chylomicrons are the first lipoproteins formed after the absorption process.
Due to their very high lipid content, chylomicrons have a large volume and the lowest density among lipoproteins.

These types of lipoproteins differ in the type and amount of lipids and proteins they contain, as well as in their functions.

Composition of lipoproteins

	Chylomicrons	Very low-density lipoproteins VLDL	low-density lipoproteins LDL	High-density lipoproteins HDL
Density (g/ml)	< 0.94	0.94-1.006	1.006-1.063	1.063-1.210
Diameter (Å)	2000-6000	600	250	70-120
Site of synthesis	Intestine	Liver	Liver	Liver, intestine
Total lipid (wt%)	99	92	85	50
Triacylglycerols	85 Highest amount	55 Liver	10	6
Cholesterol esters	3	18	50 (bad)	40 (good)
Apolipoproteins	A, C, E, **B48** 1%only	C, **B100**, E 8%	B100 20%	A, C, E 50%
Function	Transport of <u>dietary</u> TAG to the liver	Transport of TAG from the liver to peripheral tissues	Transport of cholesterol from the liver to peripheral tissues	Transport of cholesterol from peripheral tissues back to the liver (cholesterol scavengers)

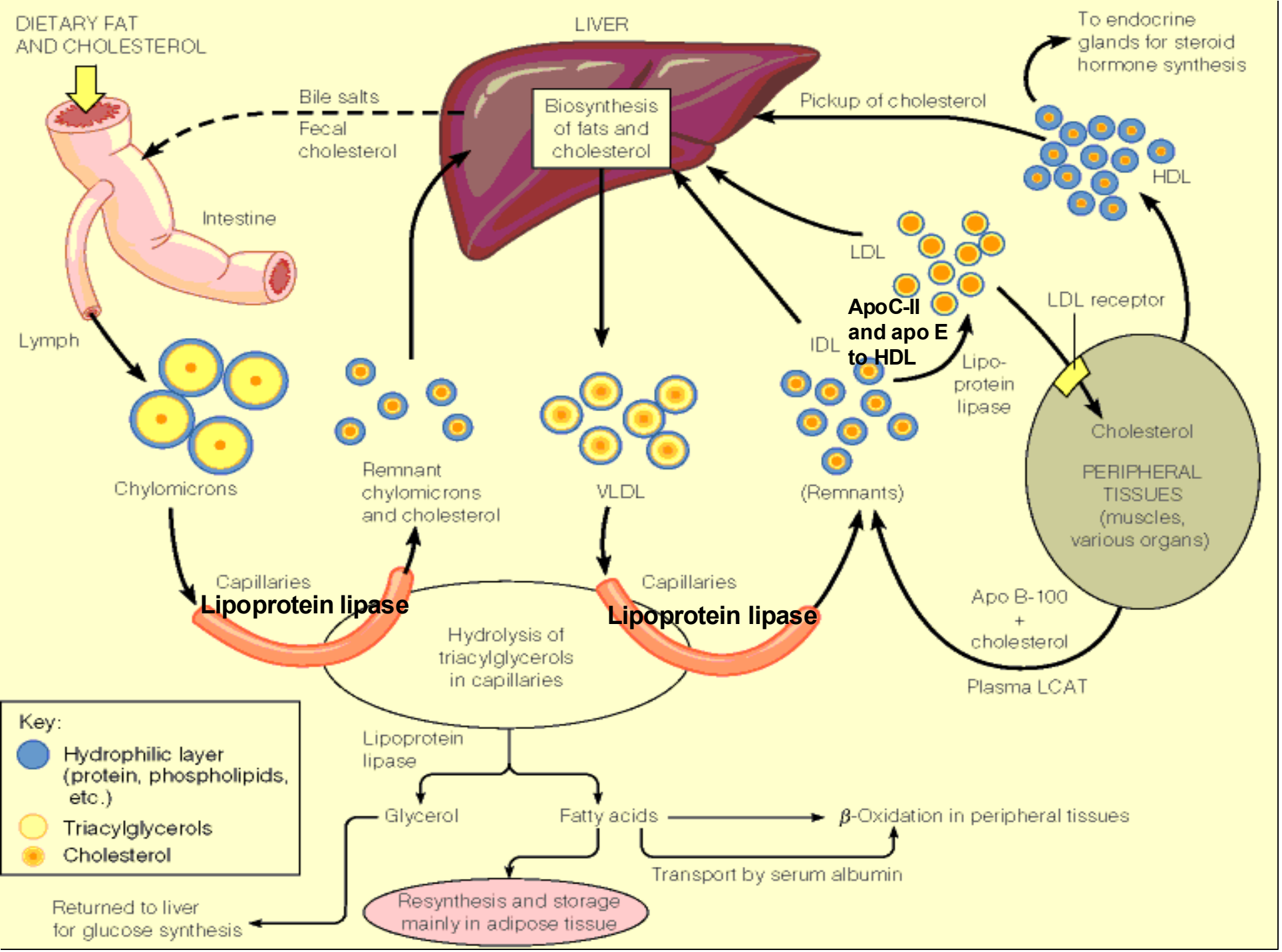
Reasons why HDL is beneficial:

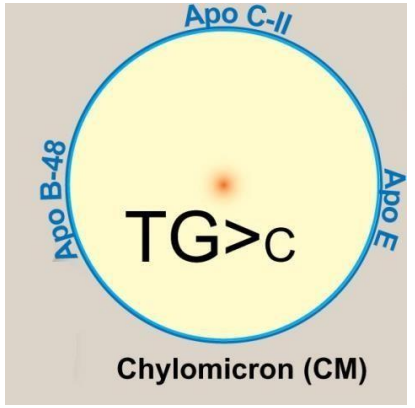
1 it contains more protein and less lipid compared to other lipoproteins.

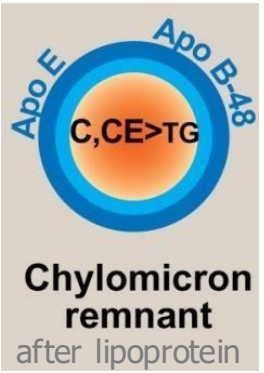
2 It facilitates the transport of lipids from peripheral tissues to the liver, where cholesterol is utilized to produce bile acids and vitamin D, reducing its levels in the body.

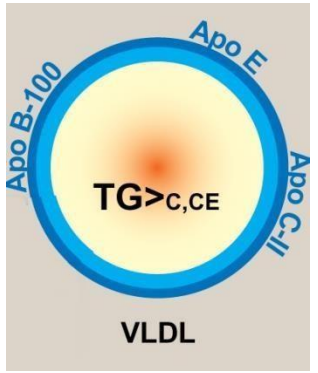
3 It transfers cholesterol to the testes and ovaries for hormone production and to the adrenal glands for cortisol synthesis.

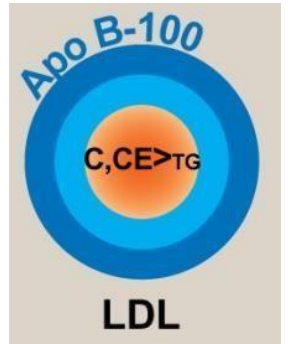
Lipid transport







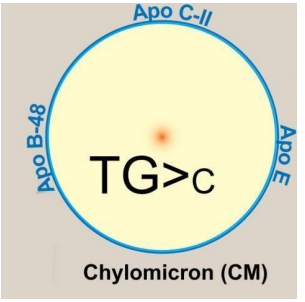




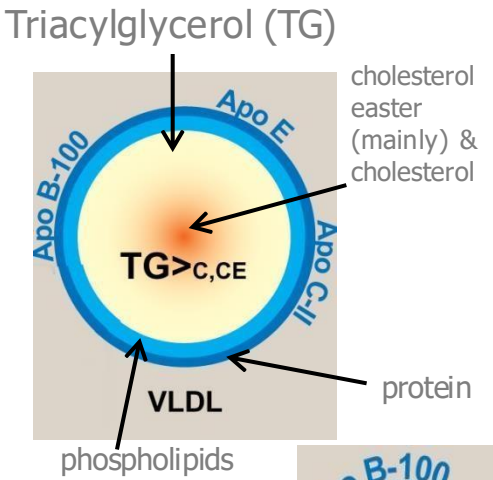
More explanation of the previous slide:

- I recommend to watch this section from the lecture min (34;35-38:36)

  click here

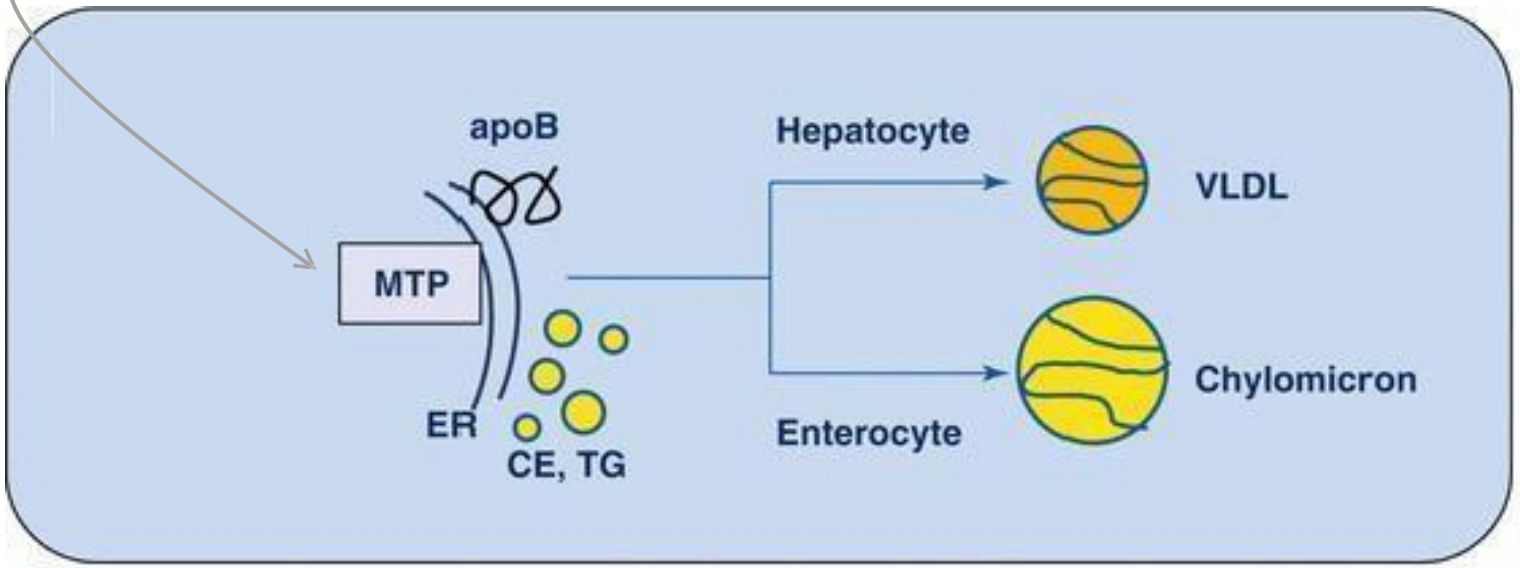


- Dietary fats enter the gastrointestinal tract (GIT) and are absorbed, leading to the formation of chylomicrons.
- During their movement through blood vessels, chylomicrons encounter **lipoprotein lipase**, a protein projected from endothelial cells into the lumen of blood vessels. Lipoprotein lipase hydrolyzes triacylglycerols, reducing their content in chylomicrons.
- The remnants chylomicrons are then taken up by the liver, which uses them to produce **(VLDL).** As **VLDL** is transported from the liver to peripheral tissues, **lipoprotein lipase** continues to hydrolyze triglycerides, reducing their content and converting **VLDL into (IDL).** The apolipoproteins (apo C and E) are removed from IDL, leaving apo B-100, which results in the formation of (LDL). These removed apolipoproteins (C and E) are transferred to (HDL).
- The **LDL** formed is distributed to multiple cells by binding to receptors on their surfaces in peripheral tissues, while some LDL remains circulating in the bloodstream and precipitate in the blood vessels and lead to atherosclerosis.

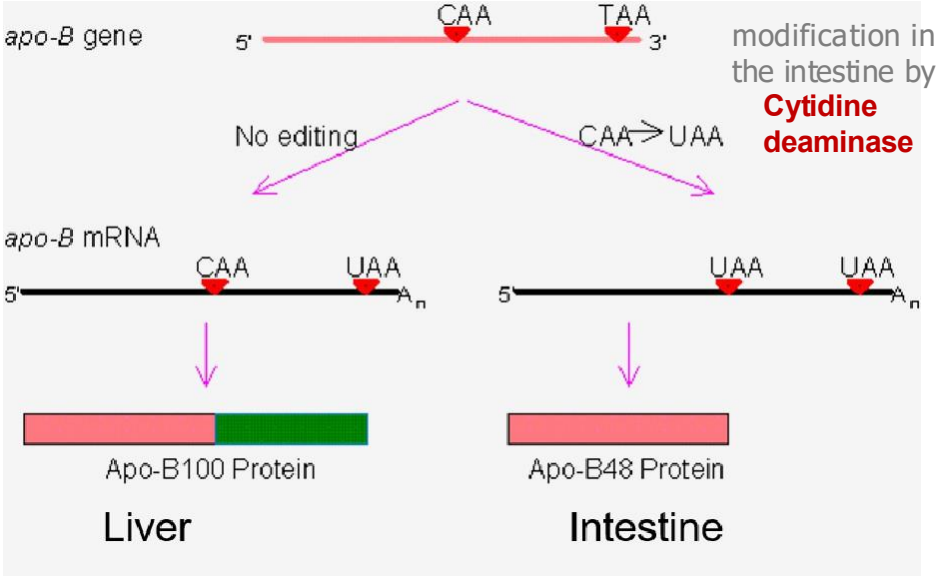


Formation and release of chylomicrons

- TAG and cholesteryl esters are packaged in chylomicrons made of phospholipids, non-esterified cholesterol, and apolipoprotein B-48.
- Microsomal triglyceride transfer protein (MTP) is essential for the assembly of all TAG-rich apoB-containing particles in the ER.

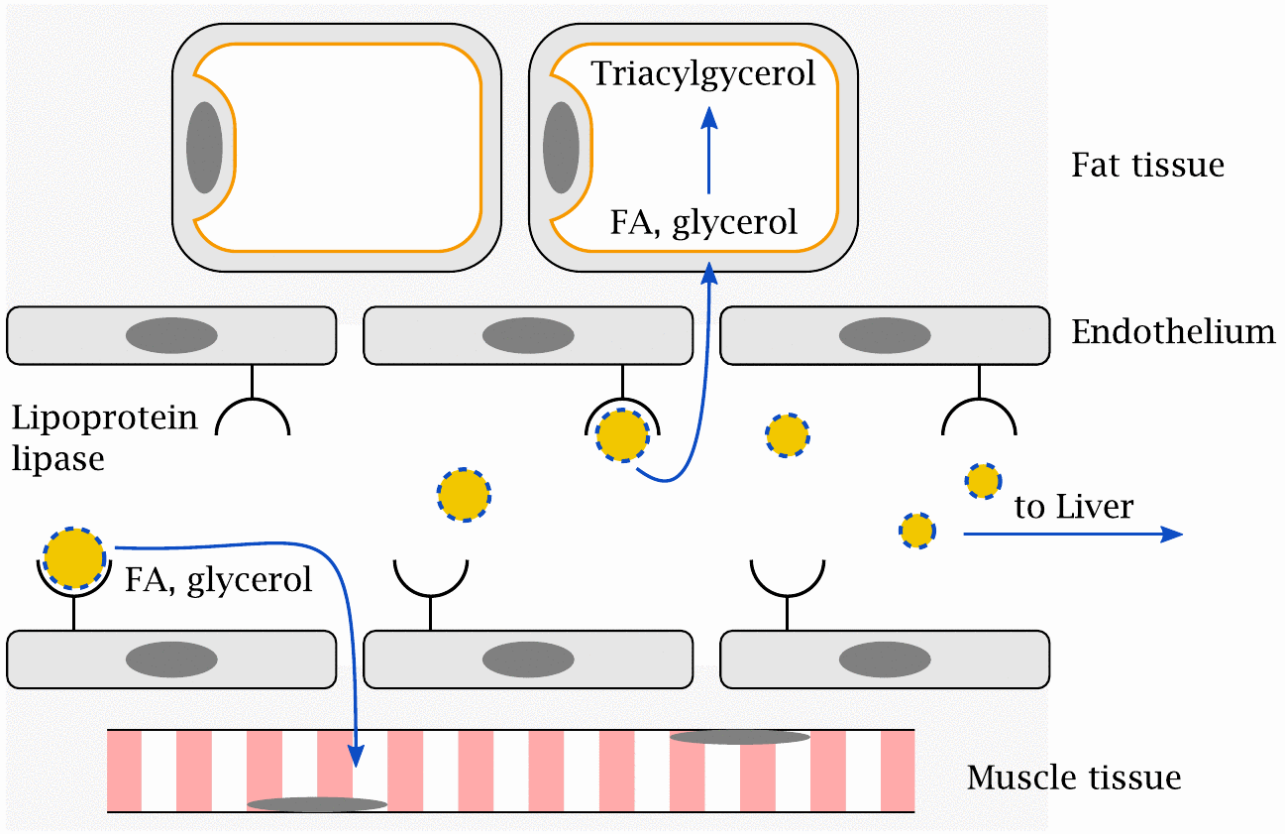


apolipoprotein B-48 (truncated form) and B-100 (long form) are encoded by the same gene



Cytidine deaminase functions in the intestine by removing the amino group from cytosine, converting it to uracil, which produces the UAA stop codon.

Fates of TAGs in chylomicrons



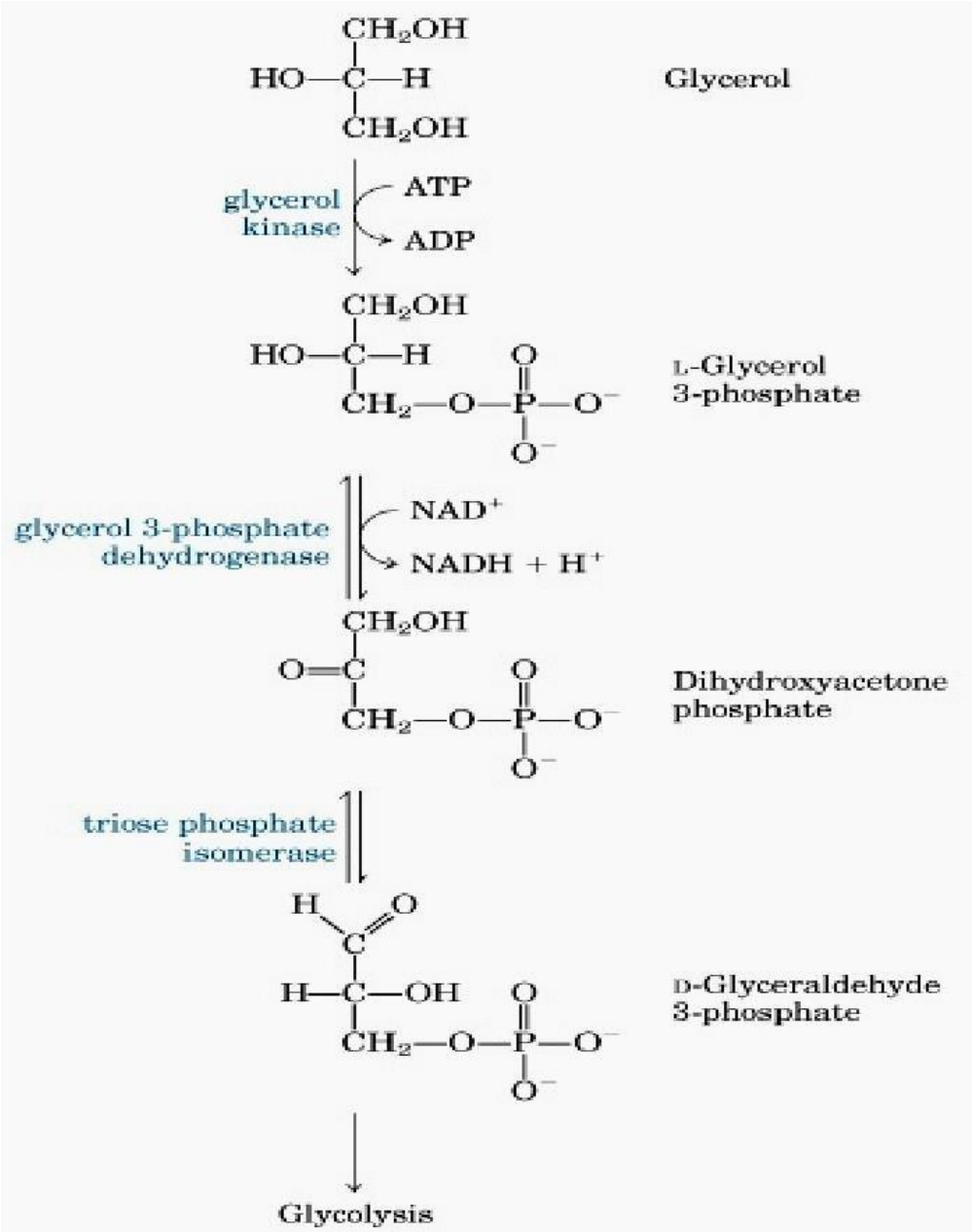
- TAGs in chylomicrons are hydrolyzed in the bloodstream by lipoprotein lipases that are anchored into the surface of endothelial cells.

- The resulting fatty acids have two possible fates:

(1) When energy is in good supply, they are converted back to TAGs for storage in adipose tissues.

(2) When cells need energy, the fatty acids are oxidized into acetyl-CoA.

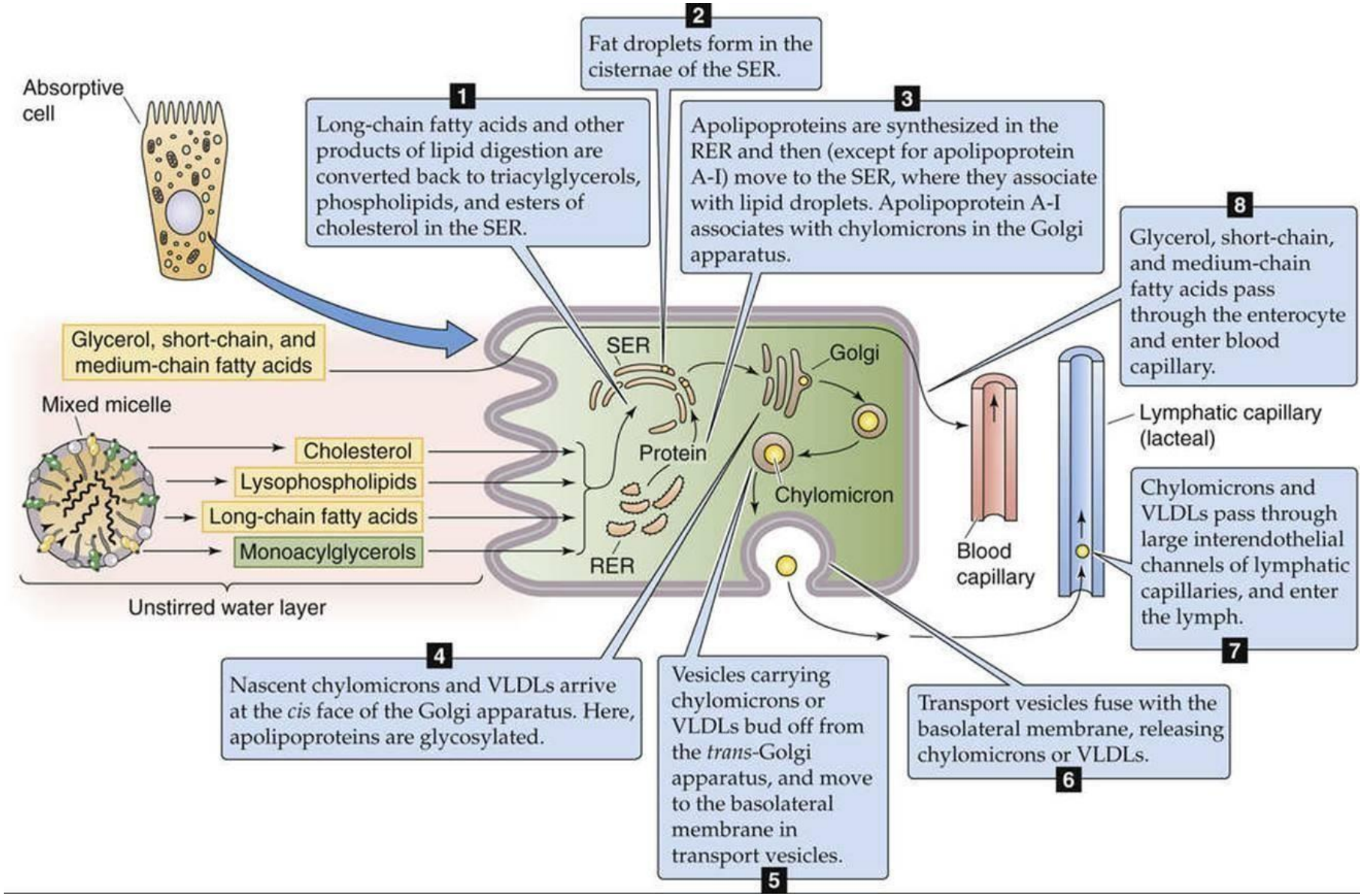
Familial chylomicronemia (type I hyperlipoproteinemia) is a rare, autosomal- recessive disorder caused by a deficiency of LPL or its coenzyme apo C-II resulting in fasting chylomicronemia and severe hypertriacylglycerolemia, which can cause pancreatitis.



Fate of glycerol

- Glycerol is carried in the bloodstream to the liver or kidneys, where it is phosphorylated and then converted to glyceraldehyde 3-phosphate and dihydroxyacetone phosphate (DHAP) for either glycolysis or gluconeogenesis or synthesis of TAG.

Summary: What happens inside intestinal cells?



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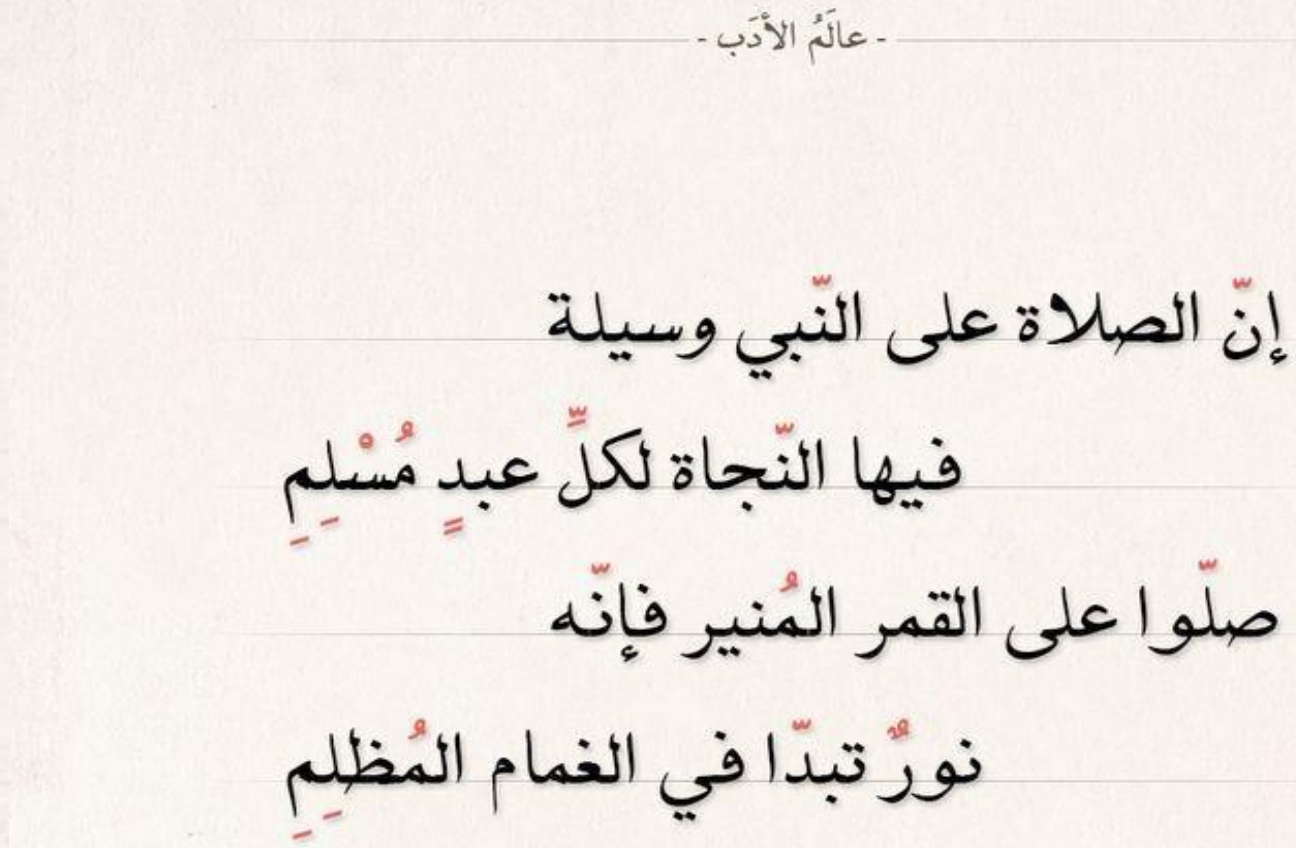

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1	Slide 16	———	The Slide is assigned as an Extra Reading
	Slide 26	———	Picture was Deleted (not mentioned in Dr's Slides)
V1 → V2			