**Digestionand Absorption Of**

**Lipids**



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# Definition

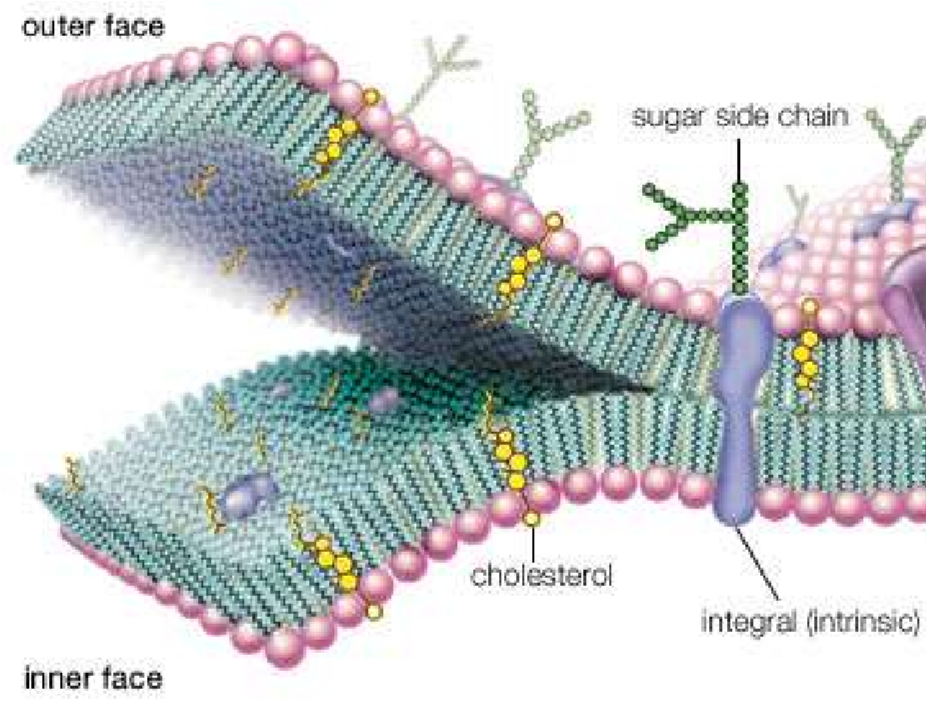
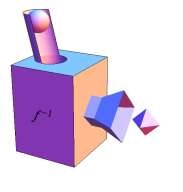
They have the common property of being

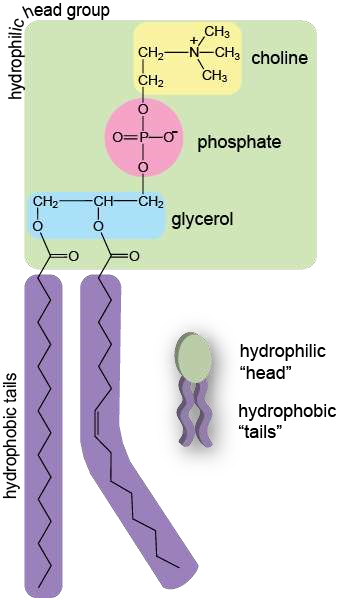


## (1)relatively **insoluble in water**

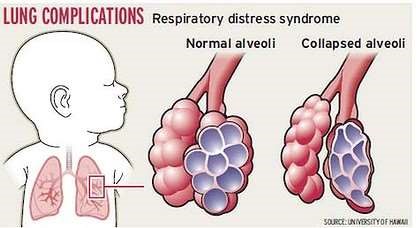
(2) **soluble in nonpolar solvents** such as alcohol, ether and chloroform.

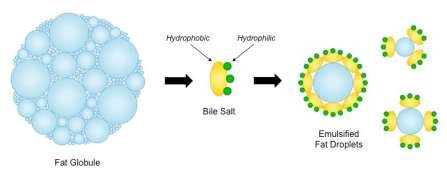
# Functions

* Storage form of energy
  + - Concentrated storage
    - 9.24kcal/g



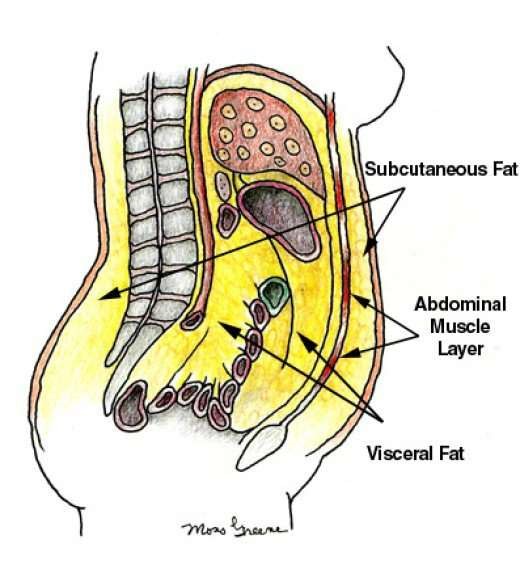
* Bio membrane
  + - Structural components
    - Phospholipids
    - Glycolipids
* Metabolic regulator



* + Steroid hormones
  + Prostaglandins
* Surfactant
  + Diphosphatidylcholine

(Lecithine)

* Detergent,emulsifier
* Electric insulator of neuron
* Insulator from external temperature
* Shape and contour to the body
* Cushioning to internal organs
* Fat soluble vitamins
* Taste and palatability



**Vs**

# Dietary fatComposition

>More than 95% : TG

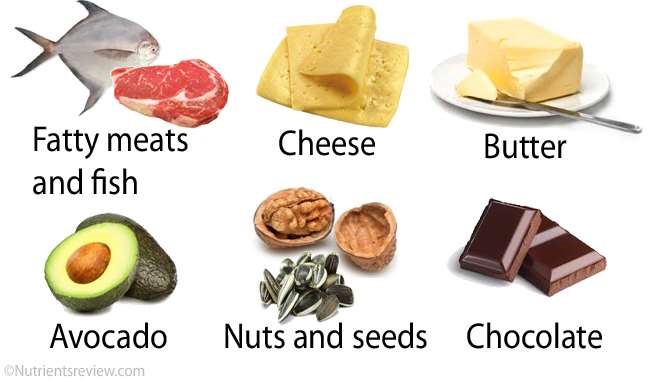
Cholesterol,

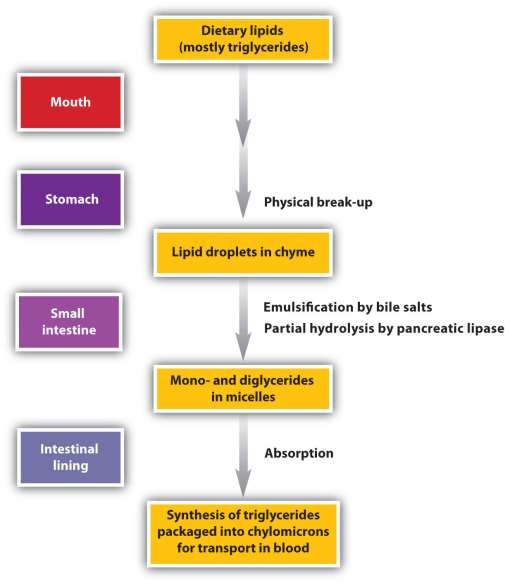
Cholesteryl esters,

Phospholipids, and

Unesterified fatty acids.

# Dietary sources ofLipids



Overview

# Digestion in Mouth

TG Lingual lipase  1,2 DAG +FFA

**Lingual lipase:**

Secreted by dorsal surface of tongue

Active at low pH (pH 2.0 – 7-5)

optimum pH 4.0-4.5

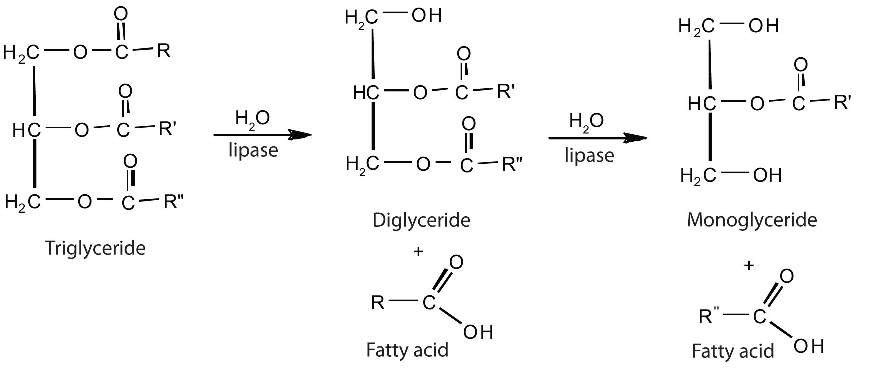
Ideal substrate-Short chain TGS

Enzymatic action continues in stomach

Short chain fatty acids: absorbed directly from the stomach wall

Enter the portal vein.

# Triglyceridedegradation



## Digestion inStomach

### **Gastric Lipase**

* Optimum p H 5.4 (4-7)
* Acid stable
* Gastrin→ Chief cells
* Requires the presence of Ca++
* Short and medium chain fatty acid (30%)

## Fats ingastric emptying : Satiety

Fats → Enterogastrone

↓

inhibits gastric motility

↓

↓ rate of emptying of stomach

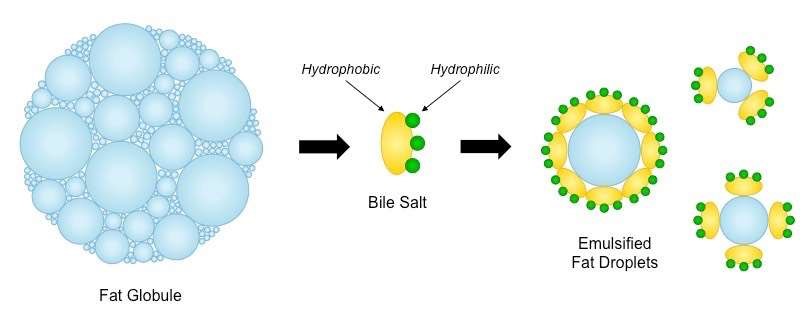
↓

high satiety value.

### Significance of Lingual & GastricLipases

* **Neonates**
* Pancreatic insufficiency
* **Cystic fibrosis**
* **other pancreatic disorders**
* short and medium chain fatty

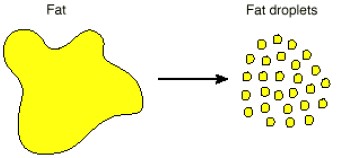
Emulsification anddigestion

* Lipids
  + hydrophobic
  + poorly soluble in the aqueous environment
  + **Lipase**, : water soluble
  + can only work at the surface of fat globules.
* Digestion is greatly aided by

**Emulsification** : breaking up of fat globules into much smaller **emulsion droplets**.

## Emulsification anddigestion

**TG digestion :**

* occurs at lipid-water interfaces
* Rate **α** surface area
* increased by
* churning peristaltic movements of the intestine
* emulsifying action of bile salts
* Emulsification takes place in the duodenum.

## Digestion in smallintestine

* Major site of fat digestion
* Effective → Pancreatic lipase and bile salts.
* Bile salts → effective emulsifying agents
* Secretion of pancreatic juice is stimulated by?
* acid gastric /Protein rich content in duodenum
* Secretin/ Cholecystokinin Pancreozymin

## GI hormones

**Secretin-** ↑ secretion of electrolytes and HCO3rich fluid components of pancreatic juice **Pancreozymin of CCK** – stimulates the secretion of the pancreatic enzymes

**Cholecystokinin of CCK-PZ-**

Contraction of the gall bladder

Dilatation of spincture of Oddi

**Hepatocrinin-**

Released by intestinal mucosa

stimulates more bile formation (poor in bile acid)

## Contents of PancreaticJuice

Pancreatic Lipase- triglycerides

Phospholipase A2- Phospholipids

Cholesterol esterase- Cholesteryl esters

## BileSalts

* Required for the proper functioning of the pancreatic lipase enzyme
* Combines **lipase** + 2 X **Colipase**.
* Enhances the lipase activity
* Emulsification of fats

**Coli**

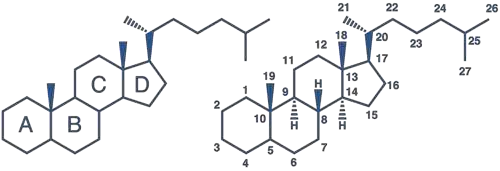
**pase**

**lipase**

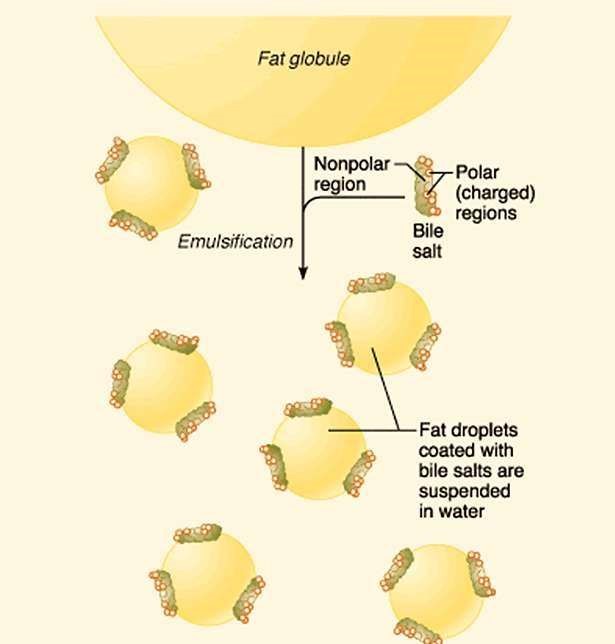
**TG**

**particle**

## BileSalts

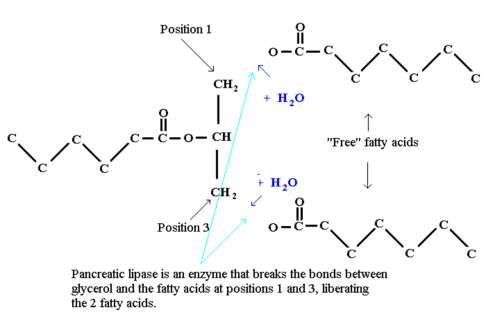
* Synthesized in the liver
* Stored in the gall bladder
* Derivatives of cholesterol
* sterol ring + side chain + glycine / Taurine
* Na & K salts of Glycocholic & Taurocholic acid
* Entero-hepatic circulation

# Emulsification by bilesalts



interact with the dietary lipid particles and the aqueous duodenal contents stabilizing the lipid particles as smaller preventing them from coalescing

TG degradation by pancreaticlipase

* specific for I0 ester linkages
* Cant act on ‘2
* Acts on FA present at position 1 and 3

↓

α,β diglyceride

↓

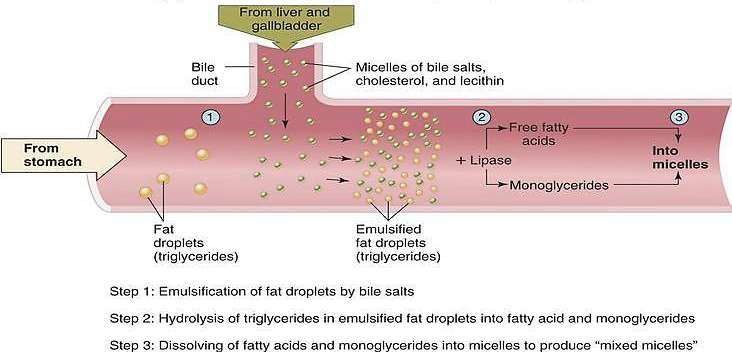
β mono glyceride

## TG degradation by pancreaticlipase

Isomerase

* β- Mono acyl glycerol ---------------→ α- Mono acyl glycerol Pancreatic lipase
* α- Mono acyl glycerol ---------------→ Glycerol + FA
* Primary product
* β- Mono acyl glycerol (78%)
* α- Mono acyl glycerol (6%)
* free fatty acids
* glycerol (14%)

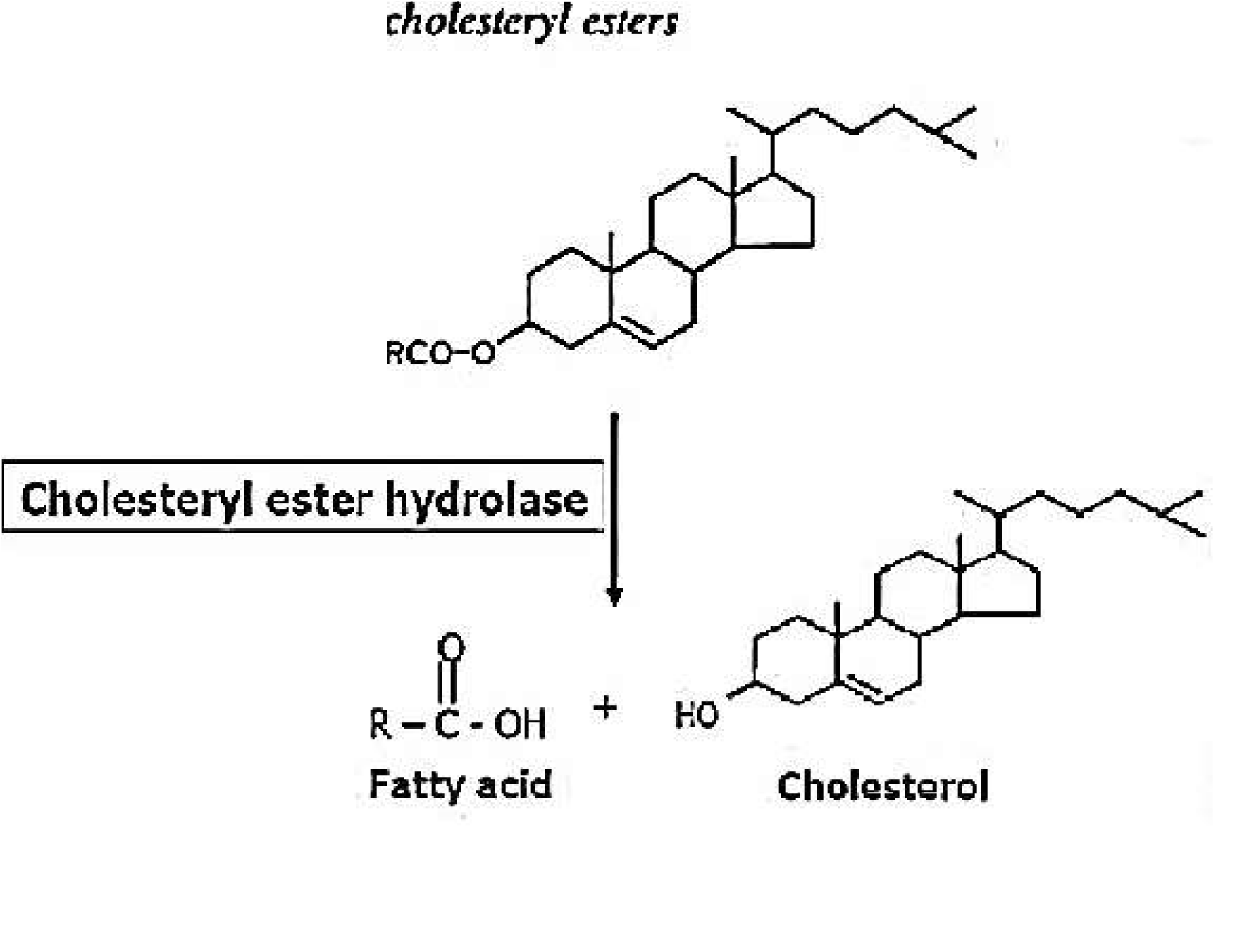
Emulsification andDigestion of TG



## Significance ofPancreatic lipase

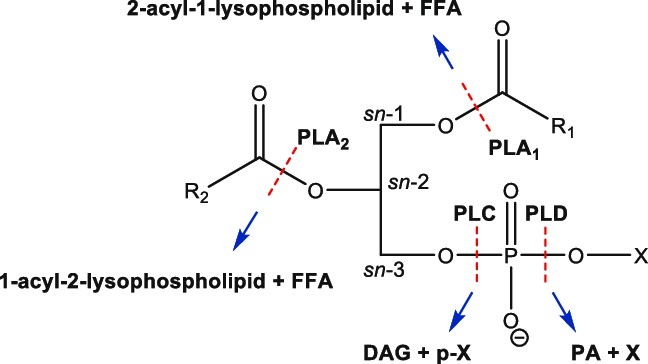
* High concentration in pancreas.
* severe **pancreatic deficiency such**
* **cystic fibrosis**
* malabsorption of fats
* **Orlistat**
* antiobesity drug
* inhibits gastric and pancreatic lipases • decreasing fat digestion and absorption • weight loss.

Cholesteryl esterdegradation

* Dietary cholesterol
* mainly free (Non esterified) form
* 10-15% is in esterified form
* Hydrolase : activity ↑ by bile salts

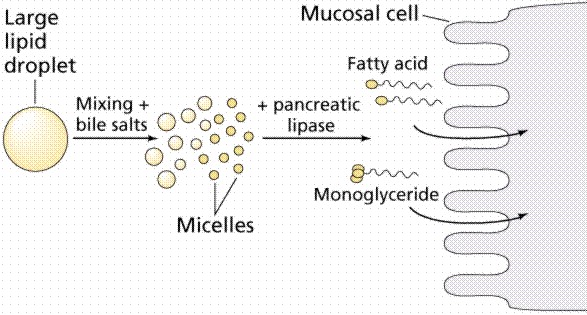
Phospholipid

degradation



## Absorption ofLipids

**Glycerol, short chain FA & medium chain FA** directly absorbed from the intestinal lumen → portal vein → liver

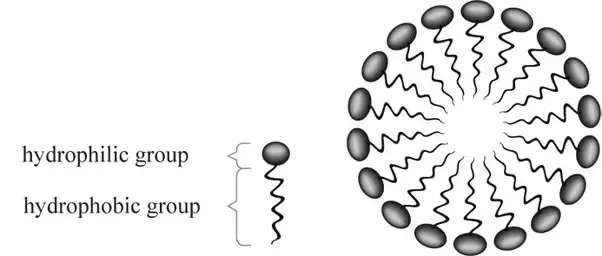


**Long chain fatty acids, free cholesterol and β- acyl glycerol**

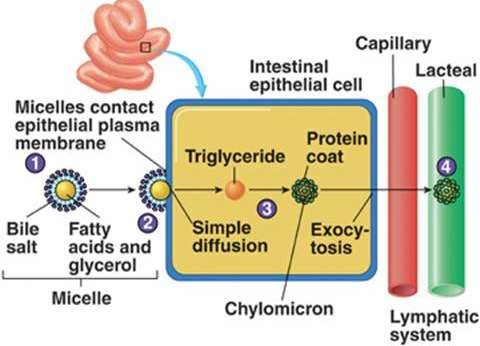
With bile salts form mixed micelles

## Micelles

* Spherical
* Clusters of amphipathiclipids
* hydrophobic groups on the inside
* hydrophilic groups on the outside of clusters
* Mixed micelles : soluble in the aqueous environment of the intestinal lumen
* Approach the brush border membrane of the enterocytes



# Micelles

* Micelles constantly break down and re-form,
* MAG & FA :
* nonpolar
* diffuse across the plasma membrane of the enterocyte.
* Some : specific transport proteins

## Clinical Significance of Cholesterol

Absorption

**Ezetimibe**

* Blocks a protein mediating cholesterol transport across enterocytes
* Effective at reducing levels of LDL cholesterol
* Clinical trials are in progress

### Resynthesis of TAG and CHE

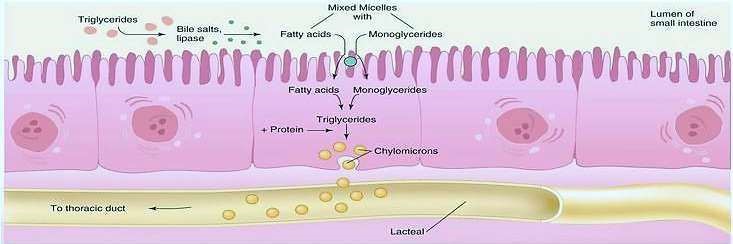
Within the intestinal epithelium

* 1- monoacyglycerols → FA + glycerol
* 2-monoacylglycerols → TAC

(**monoacylglycerolpathway)**

* Lysophospholipids → phospholipids.
* Cholesterol → Cholesteryl esters
* LCFA → TGs, PL, CHE.
* SCFA & MCFA → portal circulation
* carried by serum albumin to liver.

## Formation and Transportationof Chylomicrons



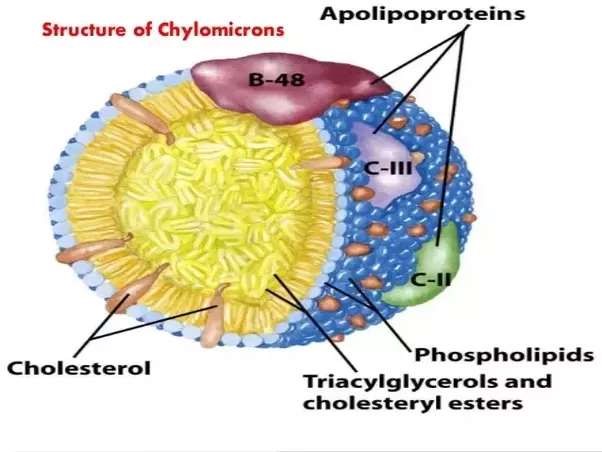
### Lipid Malabsorption (Steatorrhea)

* **lipids loss** (including fat soluble vitamins A,D E and K) in the feces.
* **Cause**
* pancreatic insufficiency
* cystic fibrosis
* chronic diseases of pancreas
* surgical removal of pancreas
* Shortened bowel, Celiac diseases, sprue or crohn’s disease
* Bile duct obstruction
* Milk and coconut oil are used therapeutically since they contain medium chain fatty acids.

### Secretion of lipidsfrom enterocytes

* Once inside the enterocyte
* monoglycerides and fatty acids → TAG.
* TAG + CH & Fat soluble vitamins → **chylomicrons**.
* Chylomicrons are **lipoproteins**, special particles that are designed for the transport of lipids in the circulation.
* Chylomicrons are released by exocytosis at the basolateral surface of the enterocytes.
* too large to enter typical capillaries.
* enter **lacteals**
* Chylomicrons then flow into the circulation via lymphatic vessels.

# Structure ofChylomicron

* *Size:* 0**.**1–1 µm
* *Average composition*

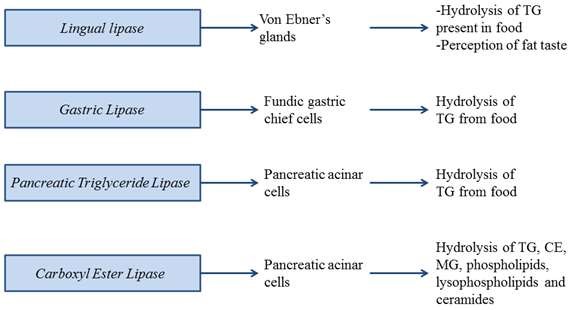
o TG (84%) o Cholesterol(2%) o Ester Cholesterol

(4%) o Phospholipid (8%) o Apo lipoproteins (2%)

## Transport and Utilizationof chylomicrons

Clinical significance ofChylomicron synthesis andutilization

* **Defective synthesis- D**eficiency of apo-B 48 protein.
* The triglyceride may accumulate in intestinal cells.
* **Chyluria-** abnormal connection between urinary tract and lymphatic drainage system of the intestines • forming Chylous fistula
* passage of Milky urine.
* **Chylothorax-** Small intestine resulting in accumulation of lymph in pleural cavity giving Milky pleural effusion

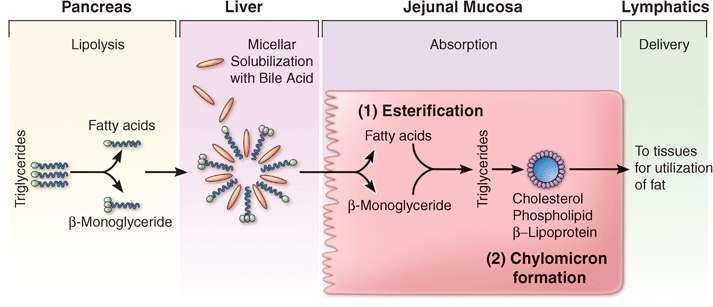


## Physiologically importantlipases

|  |  |  |  |
| --- | --- | --- | --- |
|  |  |  |  |
| Lingual /acid stablelipase | Mouth ,stomach | TAGS with medium chain FAS | FFA+DAG |
| Pancreatic lipase + co-lipase | Small intestine | TAGS with long chain FAS | FFA+2MAG |
| Intestinal lipase with bileacids | Small intestine | TAGS with mediumchain FAS | 2FFA+glycerol |
| PhospholipaseA2 + bileacids | Small intestine | PLs with unsat. FA at position2 | Unsat FFA lysolecithin |
| Lipoprotein lipase insulin(+) | Capillarywalls | TAGs in chylomicronor  VLDL | FFA+glycerol |
| Hormone  sensitivelipase | Adiposecell | TAG stored in adiposecells | FFA+glycerol |

Summary oflipid digestion and

Absorption



Chylomicrons deliver absorbed TAG to the body's cells. TAG in chylomicrons and other lipoproteins are hydrolyzed by **lipoprotein lipase**, an enzyme that is found in capillary endothelial cells. Monoglycerides and fatty acids released from digestion of TAG then diffuse into cells.

