

# Lipids pt.2

By Radha Krishnan

# Outline

- Lipoproteins
- Apolipoproteins
- Lipoprotein transport
- Vitamins
- Eicosanoids
- Naming Lipids

# Lipoproteins

- Lipoproteins = complexes that transport **hydrophobic lipids** in blood (Hydrophilic environment)



Chylomicrons    VLDL    IDL    LDL    HDL

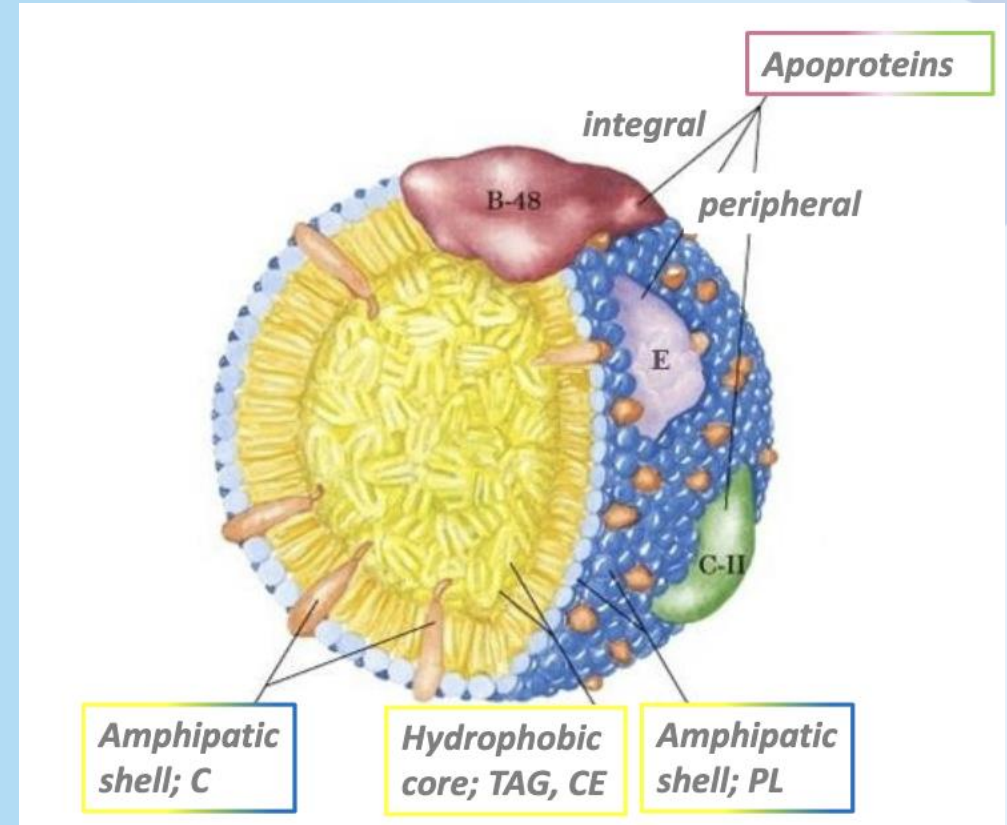


Lipoprotein	Main Content	Function
Chylomicrons	Dietary TAG	Transport TAG from intestine → tissues
VLDL (Very Low-Density Lipoprotein)	TAG (endogenous)	Liver → peripheral tissues
IDL (Intermediate Density Lipoprotein)	Intermediate	VLDL remnant
LDL (Low-Density Lipoprotein)	Cholesterol	Deliver cholesterol to cells
HDL (High Density Lipoprotein)	Protein-rich and Cholesterol rich (after peripheral tissues)	Reverse cholesterol transport

# Structure of Lipoproteins

Structure:

- **Core (hydrophobic):** triacylglycerols (TAG), cholesteryl esters (Fat soluble vitamins as well!)
- **Surface (amphipathic):** phospholipids, free cholesterol, apoproteins



# Apolipoproteins!!!

Apolipoprotein	Lipoprotein	Function
Apo B-48	CM and remnants (unique to CMs)	Structural protein for chylomicrons
Apo B-100	VLDL, IDL, LDL	Binds <b>LDL</b> receptor
Apo C-II	CM, VLDL, HDL	Activates <u>lipoprotein lipase (LPL)</u> → TG breakdown
Apo E	CM remnants, IDL, HDL (present in CM/VLDL)	Mediates uptake by liver
Apo A-I	HDL (unique to HDL)	Activates <b>LCAT</b> → cholesterol esterification

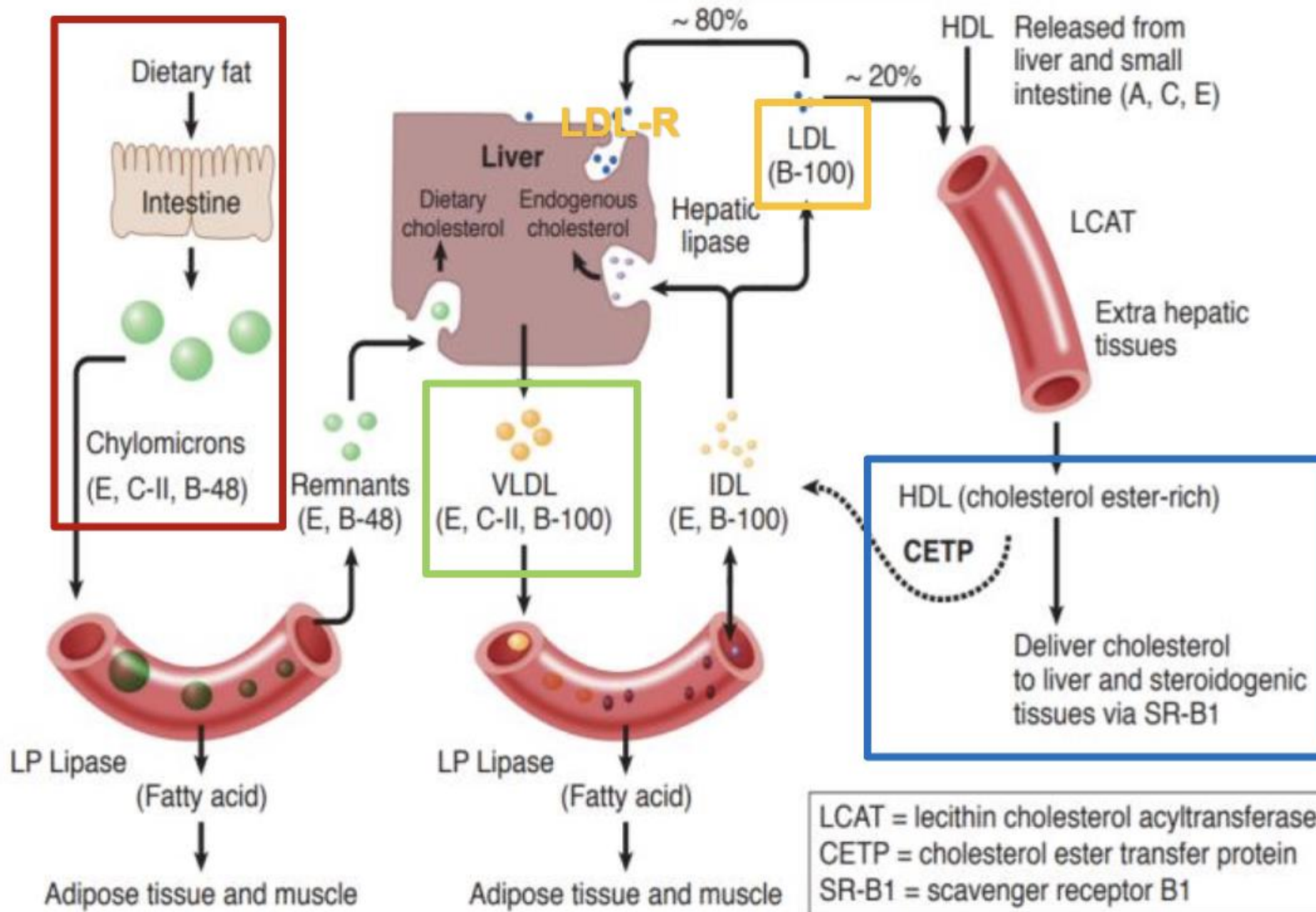


Figure I-15-5. Overview of Lipoprotein Metabolism

# Chylomicron Metabolism

Formed in intestine → carry dietary lipids

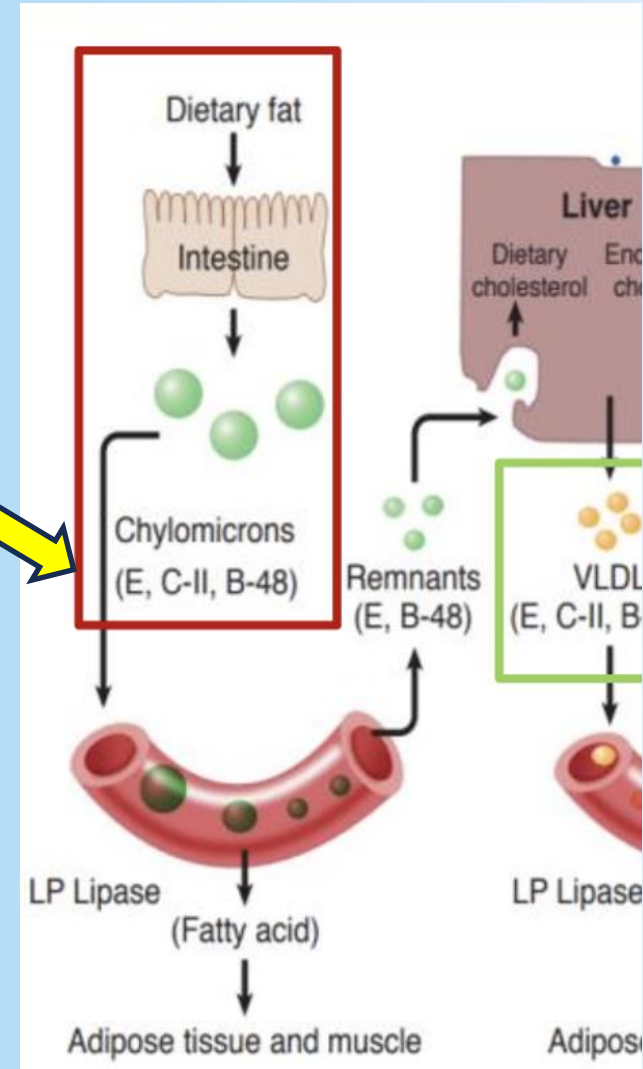
- Enter lymph → blood
- Acquire Apo C-II & Apo E from HDL

## In tissues:

- LPL hydrolyzes TAG → FA + glycerol
- FA → uptake by muscle/adipose

## Remnants:

- Taken up by liver via Apo E receptors



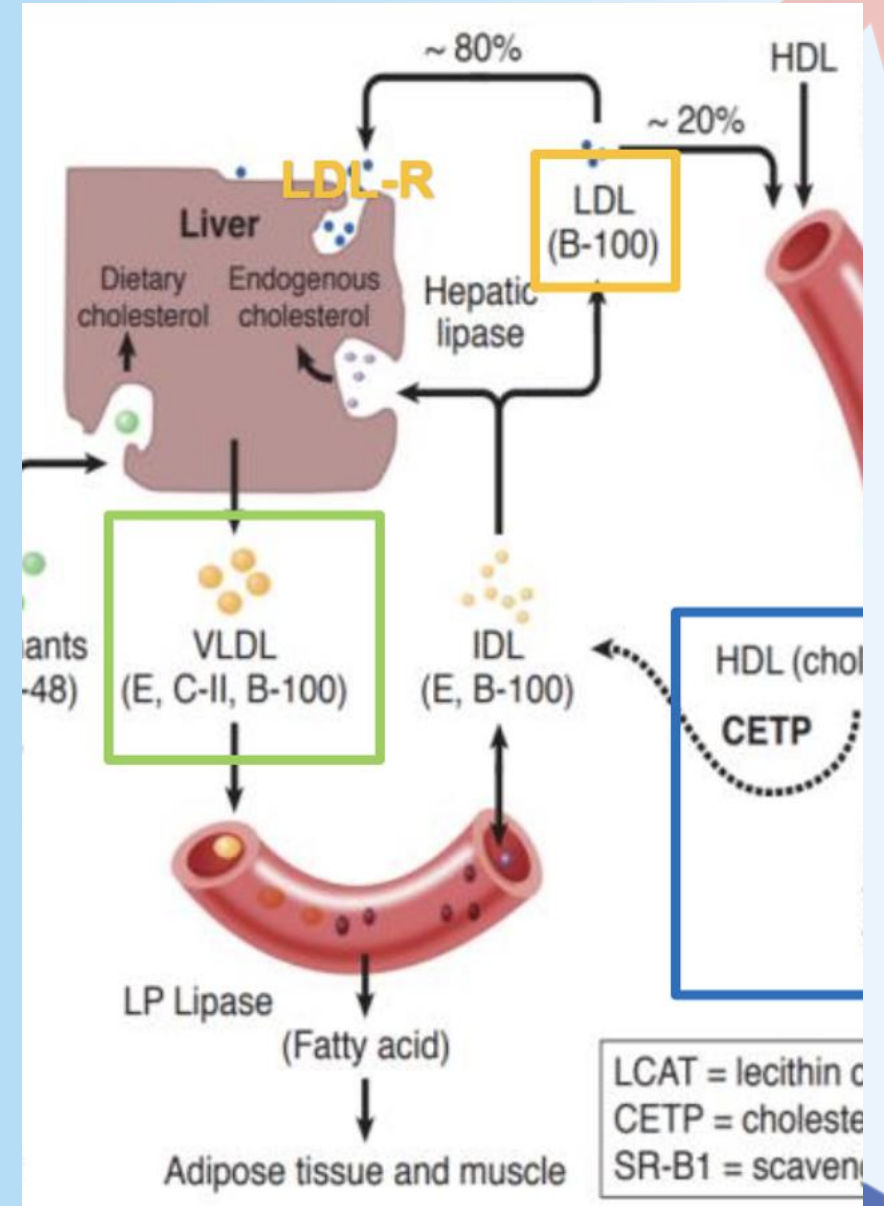
# VLDL → LDL Metabolism

Liver secretes VLDL (TAG-rich)

LPL removes TAG → VLDL → IDL → LDL

**LDL:**

- Rich in cholesterol
- Delivers cholesterol to cells via LDL receptor (Apo B-100)
- ~60-80% returns to liver



# HDL & Reverse Cholesterol Transport

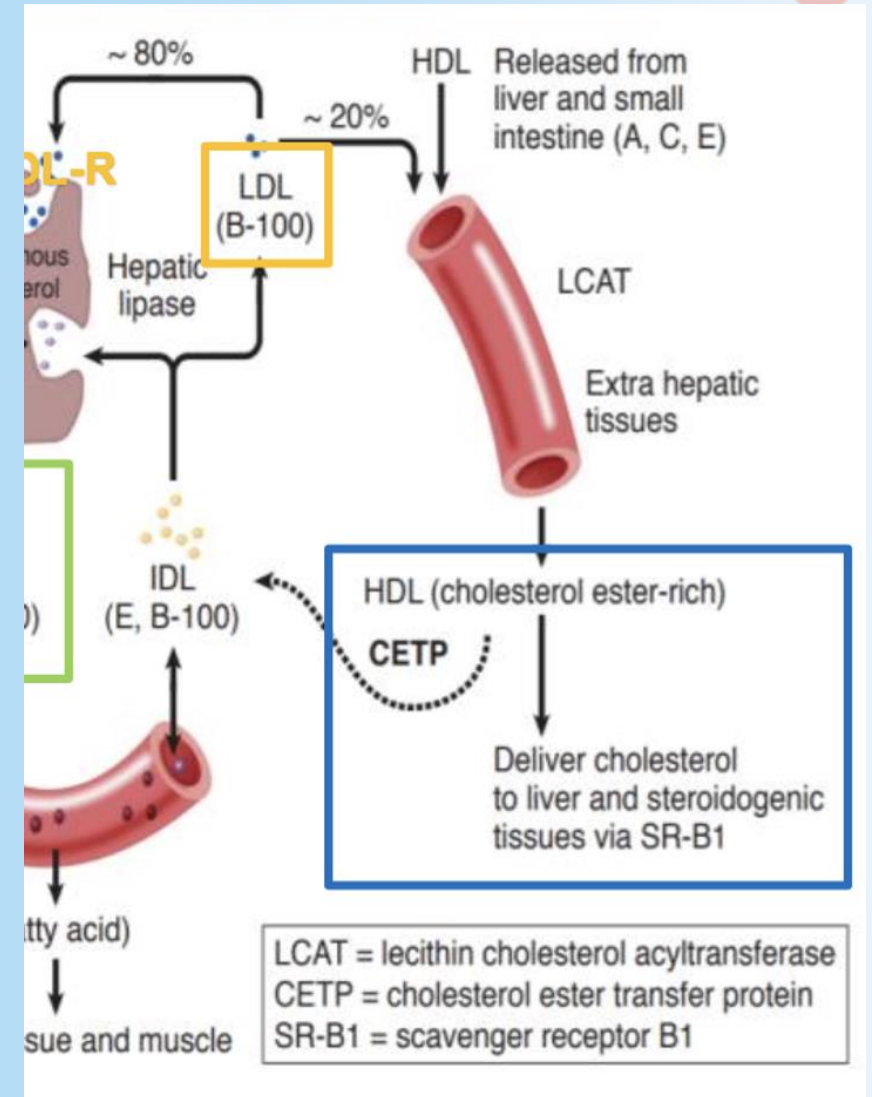
HDL collects cholesterol from peripheral tissues

Key mechanisms:

- **ABCA1 transporter** → cholesterol efflux
- **LCAT** → esterifies cholesterol
- **SR-B1 receptor** → uptake by liver

Function:

- “**Good cholesterol**” → removes excess cholesterol



# Lipoprotein Lipase (LPL)

triacylglycerols  
(TAG)



free fatty acids (FAs for storage and energy) + glycerol

## Acts on lipoproteins:

- Chylomicrons (dietary TAG)
- **ONLY VLDL!** (endogenous TAG)

## Regulation:

- **Insulin ↑ LPL activity** (especially in adipose tissue)

## Key apolipoproteins:

- **Apo C-II** → activates LPL
- **Apo C-III** → inhibits LPL (clinical relevance)

## Location:

- Bound to capillary endothelium

# Hyperlipoproteinemia

Type	Defect	Lipoprotein ↑
Type I	LPL / Apo C-II deficiency	Chylomicrons
Type IIa (includes familial hypercholesterolemia)	LDL receptor defect	LDL
Type IIb	↑ VLDL + LDL	Mixed
Type III	Apo E defect	IDL remnants
Type IV	↑ VLDL	TAG ↑
Type V	↑ VLDL + chylomicrons	Severe TAG ↑

(mentioned in Mark's and the syllabus, but shouldn't be too high yield

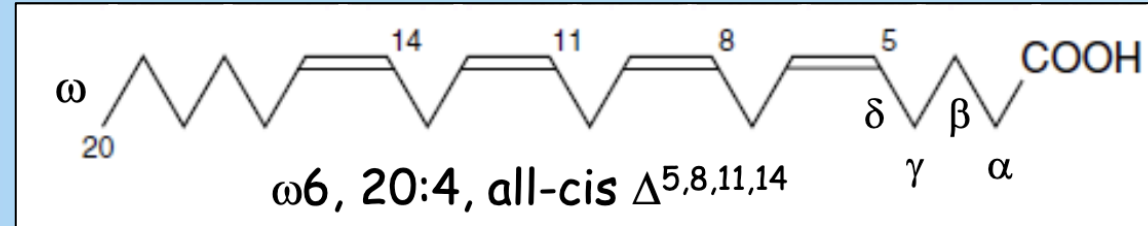


# Vitamins

Vitamin	Proper Name	Main Functions
A	<b>Retinol</b> (retinal, so retinoic acid)	Vision (rhodopsin), epithelial function, gene expression
D	<b>Calcitriol</b> (Milk (Vit D) is good for bones → bones have <u>calcium</u> → <u>calcitrol</u> )	Ca <sup>2+</sup> & PO <sub>4</sub> <sup>3-</sup> homeostasis, bone mineralization
E	<b>Tocopherol</b>	Antioxidant
K	<b>Phylloquinone, Menaquinone</b> (Qu(K)inone)	carboxylation of clotting factors (II, VII, IX, X), coagulation

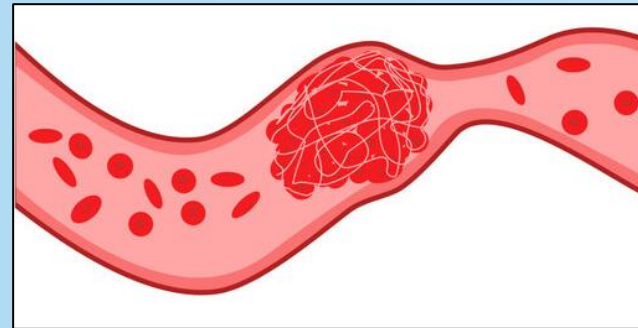
# Eicosanoids

20-carbon fatty acids (mainly arachidonic acid)

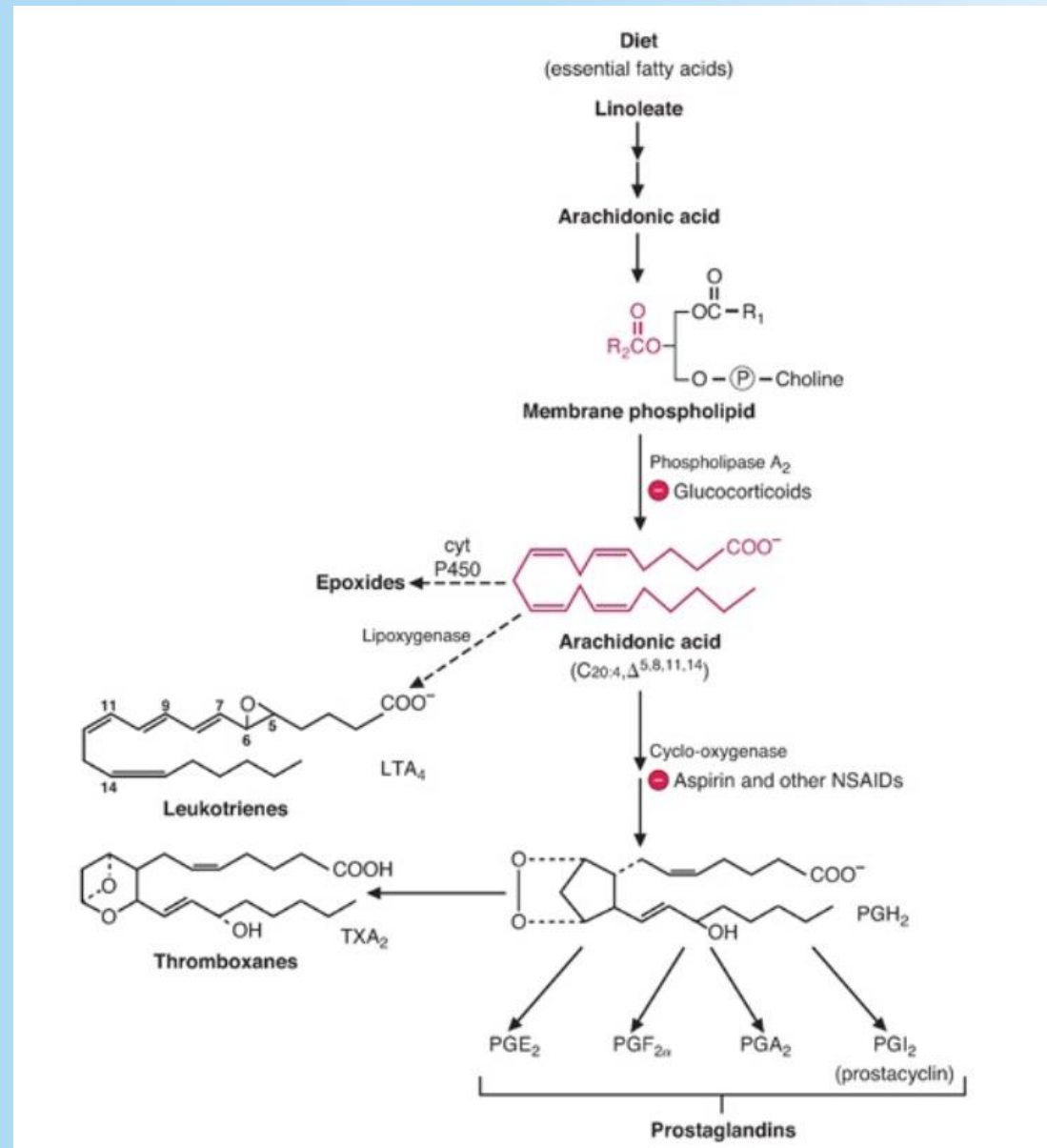


## Functions:

- Inflammation (pain, swelling, fever)
- Blood clotting (thromboxanes)
- Smooth muscle contraction and regulate BP

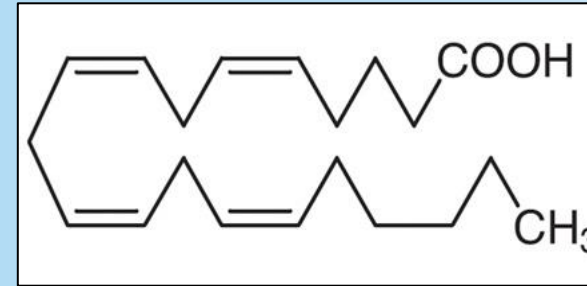


# Synthesis of eicosanoids



- **COX-1:** constitutive → normal functions (e.g. gastric protection)
- **COX-2:** inflammation, pain, fever

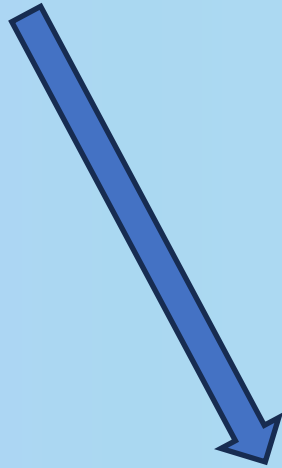
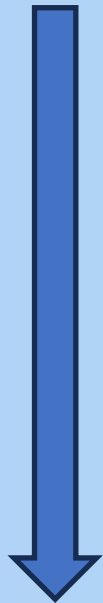
**Arachidonic acid** (released via phospholipase A<sub>2</sub>)



Steroids inhibit phospholipase A<sub>2</sub>, so no Arachidonic acid produced

**Cyclooxygenase (COX)**

NSAIDs (e.g. aspirin) inhibit COX enzymes



thromboxane (TX)

Prostaglandins (PG)

Different PGs can have different/opposing effects

**Lipoxygenase (LOX)**



Leukotrienes (LT)

# Naming fatty acids

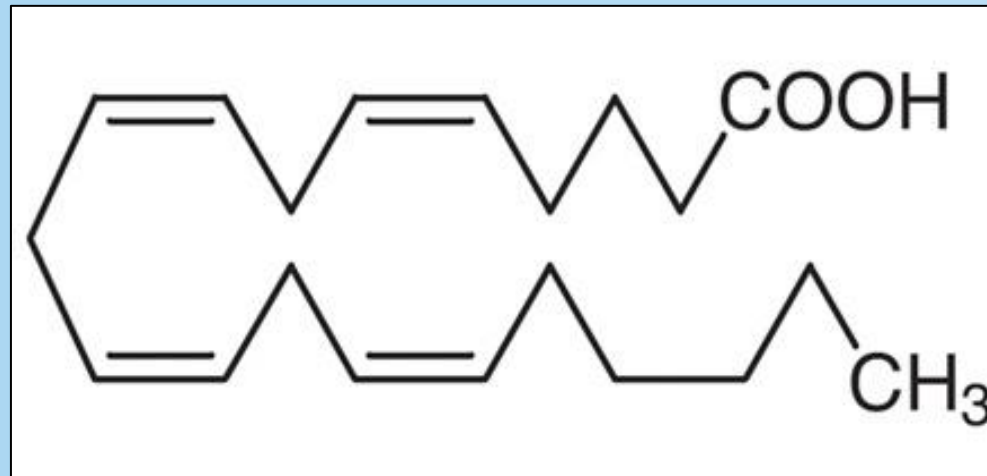
Lipid shorthand (start from CH<sub>3</sub> side!!!)

Delta  $\Delta$  Nomenclature (Start from COOH side!!!)



Number of carbons  
Number of double bonds

Location (and number) of double bonds



$\omega$  is a sleeping 3 so CH<sub>3</sub> end

