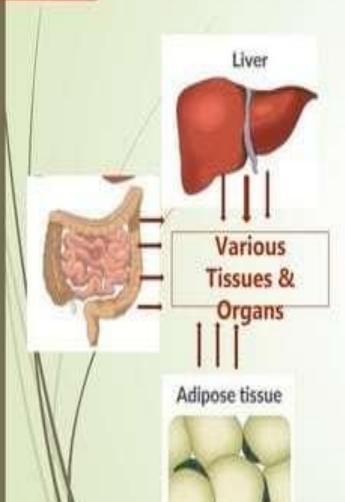
METABOLISM OF LIPOPROTEINS

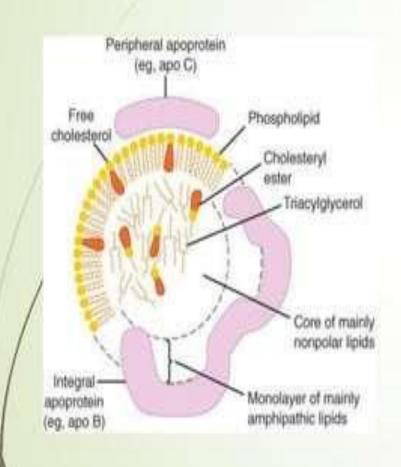
Dr. Farhana Atia Associate Professor, Biochemistry Nilphamari Medical College, Nilphamari

Lipid Transport & Storage



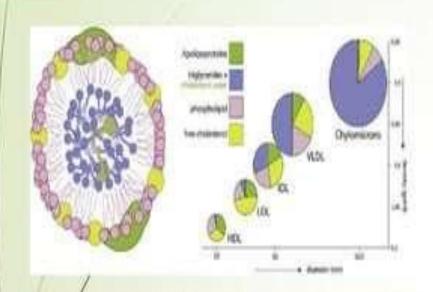
- Lipids (water insoluble)are transported in plasma as lipoproteins
- Four major lipid classes are present in lipoproteins (plasma lipids)
 - ■Triacylglycerol (16%)
 - Cholesteryl ester (36%)
 - Phospholipids (30%)
 - Cholesterol (14%)
 - ■FFA (4%)- metabolically most active

Plasma Lipoproteins



The plasma lipoproteins are spherical macromolecular complexes of lipids and specific proteins (apolipoproteins)

Four major groups of lipoproteins



Electrophoretic Pattern of Serum Lipoproteins

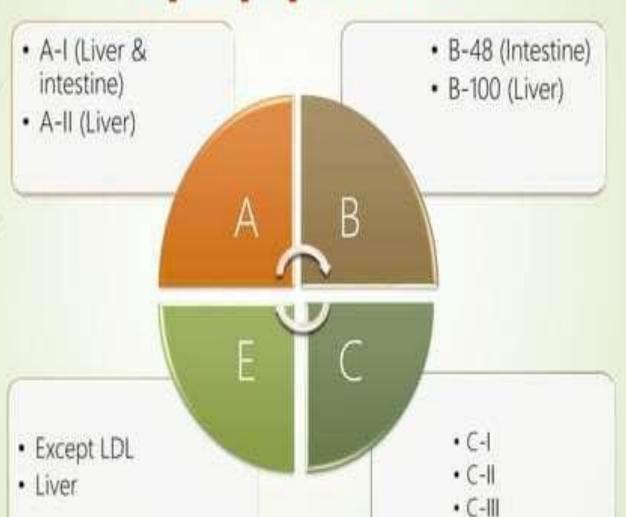


- Chylomicrons: Derived from intestinal absorption of TAG & other lipids
- VLDL: Derived from liver for export of TAG
- LDL: Represents final stage of catabolism of VLDL
- HDL: Involved in cholesterol transport, VLDL & CM

COMPOSITION

LPs	TAG	PL	Ch & ChE	Prot.	Apoprotein
СМ	90%	3%	5%	2%	B48 CII E
VLDL	60%	15%	20%	5%	B100 CII E
/ LDL	8%	22%	50%	20%	B100
HDL	5%	30%	25%	40%	A C

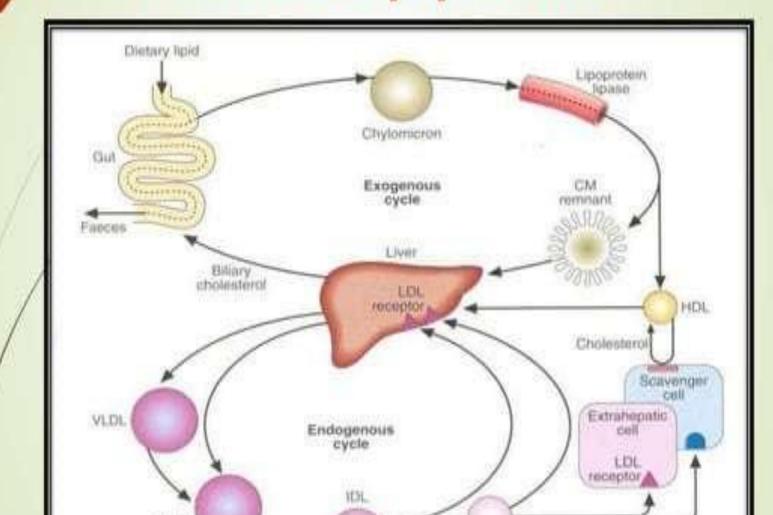
Apolipoproteins



Apolipoproteins have several roles

- Can form part of structure of lipoprotein [Apo-B]
- Act as enzyme cofactors [C-II for lipoprotein lipase, A-I for LCAT]
- Act as enzyme inhibitors [A-II, C-III for lipoprotein lipase]
- Act as ligands for interaction with lipoprotein receptor in tissue [B-100, E for LDL receptor]

Metabolism of Lipoproteins



Metabolism of Chylomicrons

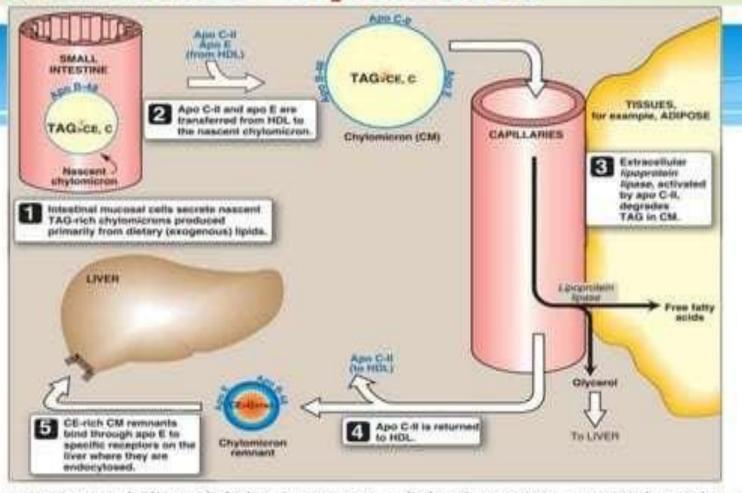


Figure 18.16 Metabolism of chylomicrons. CM = chylomicron; TAG = triacyiglycerol; C =

Metabolism of VLDL & LDL

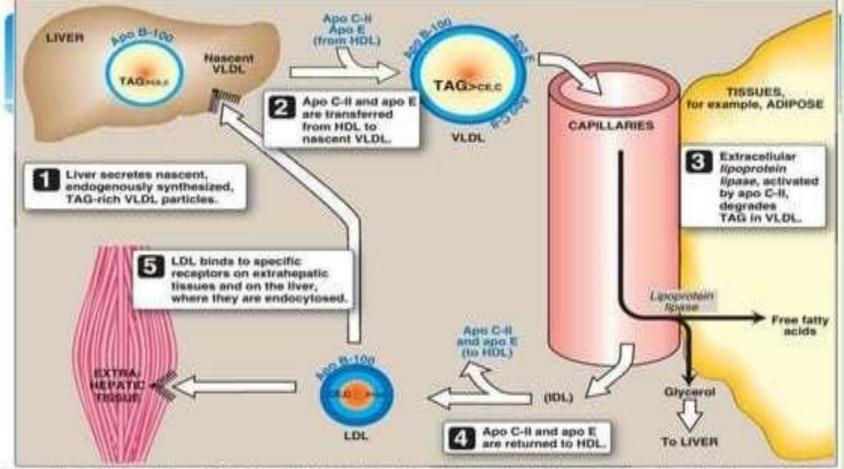
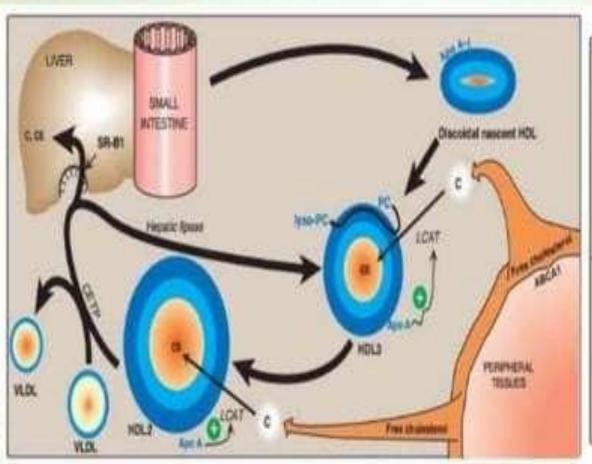
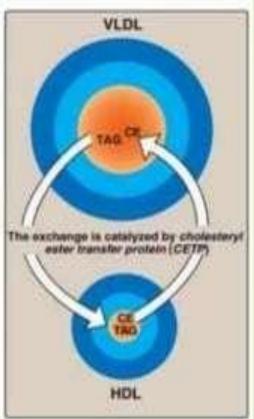


Figure 18.17 Metabolism of VLDL and LDL. TAG = triacylglycerol; VLDL = very-low-density lipoprotein; LDL = low-density lipoprotein; LDL = intermediate density lipoprotein; C = cholesterol; CE =

Metabolism of HDL





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Reverse Cholesterol Transport

- ☐ The selective transfer of cholesterol from peripheral cells to HDL, from HDL to liver for bile acid synthesis or disposal via the bile and to steroidogenic cells for hormone synthesis
- □It is a key component of cholesterol homeostasis

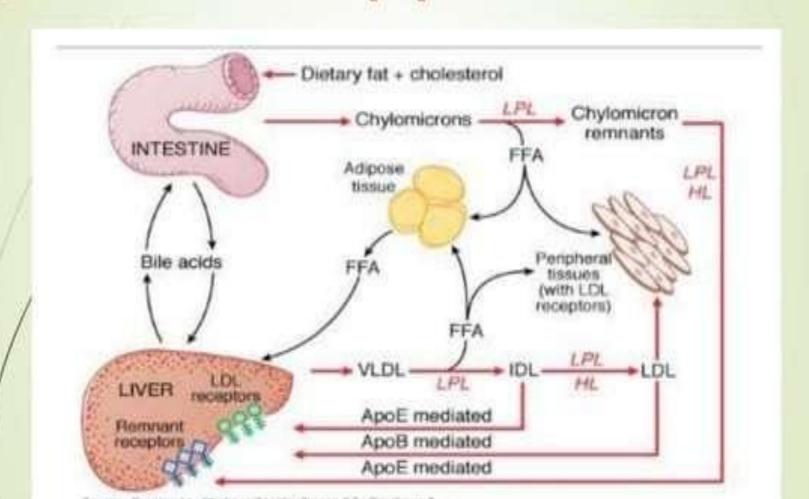
Reverse Cholesterol Transport involves

- 1. Efflux of Ch from peripheral cell to HDL
- 2. Esterification of Cholesterol by LCAT
- 3. Binding of ChE rich HDL to liver & steroidogenic cells
- Selective transfer of ChE into these cells. Then Ch is used
- 5. Release of lipid depleted HDL 3

	LP	Origin/Source	Fate
	CM	Small intestine (mucosal cell)	CM remnants are taken up by Liver through receptor mediated endocytosis
1	VLDL	Liver (Parenchymal cell)	 Can be converted to IDL (VLDL remnants) → go to liver → taken up by receptor mediated endocytosis Major fate: Converted to LDL by loosing TAG gives back apo C-II, apo E to HDL receiving some cholesterol from HDL

LP Origi		Fate	
LDL	VLDL in plasma	LDLs are taken up by extrahepatic tissues and liver by receptor mediated endocytosis About 30% of LDL are degraded by EHT and 70% in the liver	
HDL	Liver Intestine	HDL chE enter into liver- the final site of their degradation. The chE are degraded to cholesterol and utilized for the synthesis of Bile acid Lipoproteins	

Metabolism of Lipoproteins: Overview



Dyslipoproteinemias

- Primary dyslipoproteinemia
 - Inherited defect in LP metabolism
 - 1. Hypolipoproteinemia
 - 2. A Hyperlipoproteinemia
- 2. Secondary abnormal LP pattern
 - DM
 - Hypothyroidism
 - Nephrotic Syndrome

Adlanta

Frederickson's classification of hyperlipoproteinemia

Туре	Defect	Elevated LP	Elevated lipid in plasma	Risk of atherosclero sis	Name
H	Deficiency in LPL	CM	TAG	No	Familial LPL deficiency
lla	Defective LDL receptor	LDL	Cholesterol	11	Familial hyper cholesterolemia
llb	Overproduction of apo-B	LDL VLDL	TAG Cholesterol	11	
Ш	Abnormality in apo-E	CM IDL	TAG Cholesterol	11	Familial dysbetalipoprotei nemia
IV	Overproduction of	VLDL	Cholesterol	†	Familial

Thank You