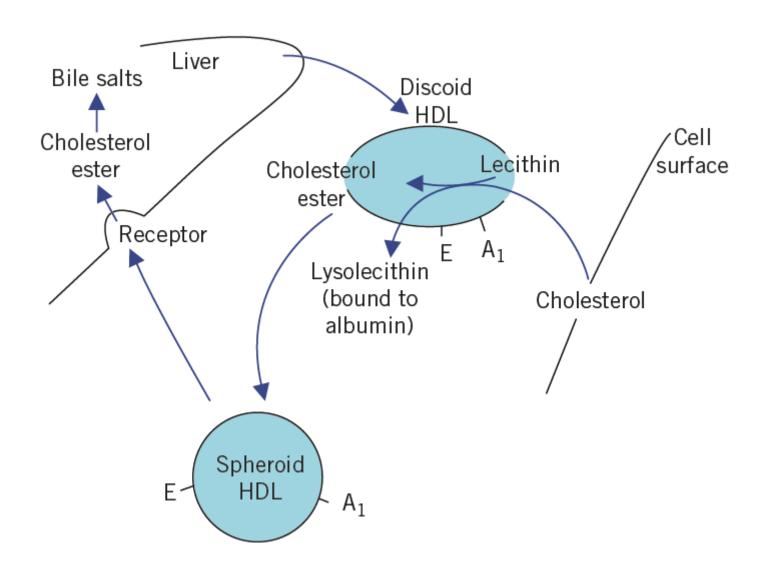
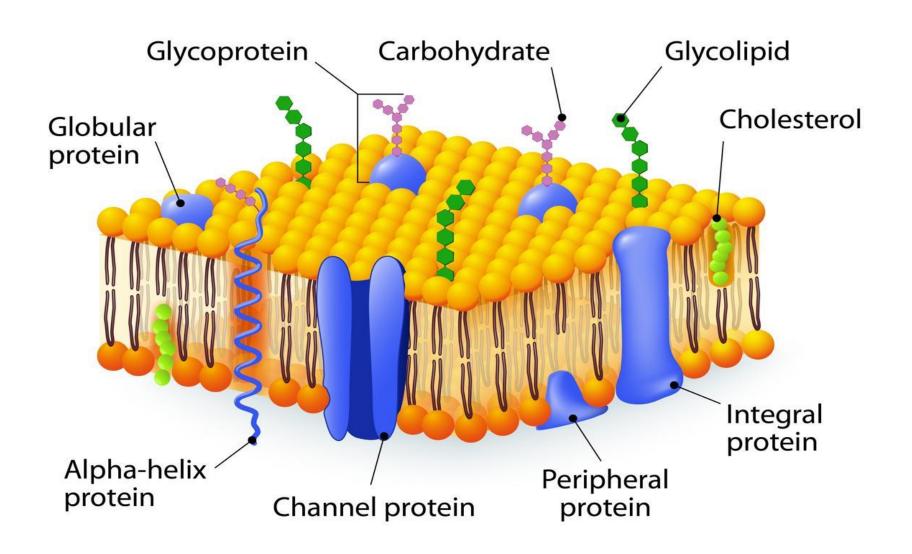
Dyslipidemia

The name has been changed from hyperlipidemia to dyslipidemia?



Fluid mosaic theory



CH₃(CH₂)_nCOO⁻ FATTY ACID

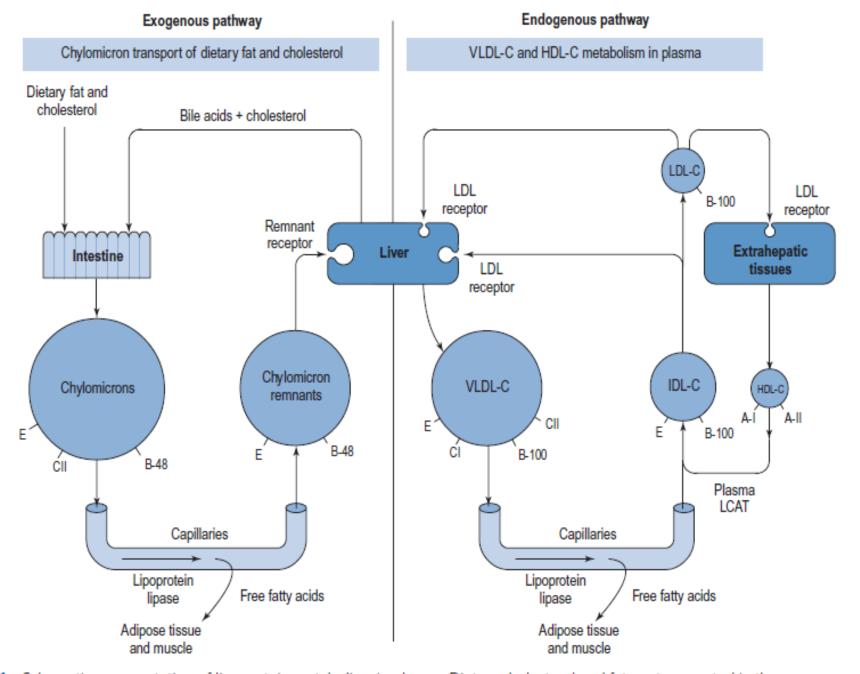
CHOLESTEROL

TRIGLYCERIDE

CHOLESTEROL ESTER

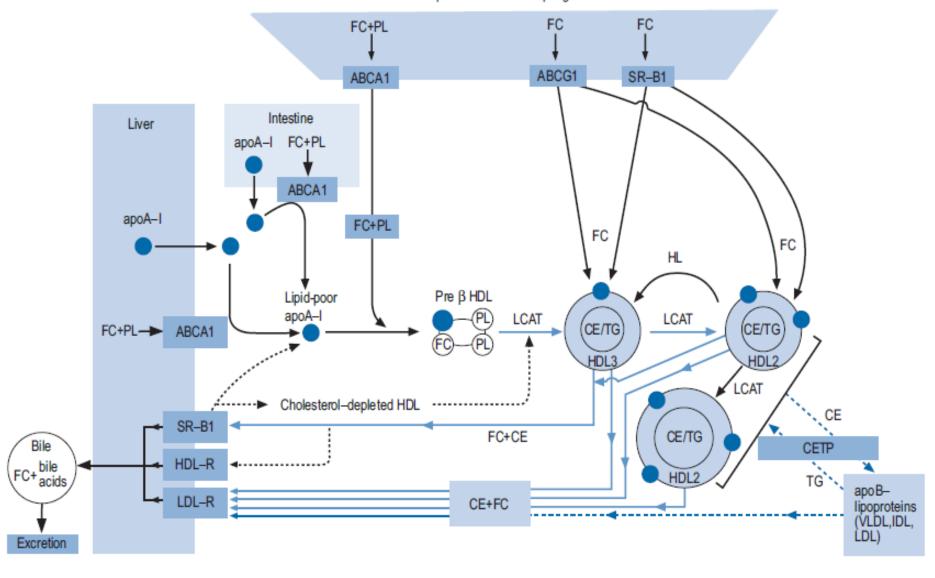
PHOSPHOLIPID

Normal physiology



. 24.1 Schematic representation of lipoprotein metabolism in plasma. Dietary cholesterol and fat are transported in the exogenous hway. Cholesterol produced in the liver is transported in the endogenous pathway.

Peripheral cells + macrophages



ABC A1 = ATP binding cassette transporter A1; ABC G1 = ATP binding casssette transporter G1; CE = cholesterol ester; CETP = cholesteryl ester transfer protein; FC = free cholesterol; HDL-R = holo HDL receptor; HL = hepatic lipase; LCAT = lecithin cholesterol acyltransferase; LPL = lipoprotein lipase; PL = phospholipids; SR-B1 = hepatic scavenger receptor B1; TG = triglycerides

Fig. 24.2 Pathways of reverse cholesterol transport in man (Chapman et al., 2010 with kind permission from Oxford University Press, Oxford)

Types of dyslipidemia

Primary and secondary

Primary DL

Familial?

Primary DL

Mainly into 3 classes

- 1. F. Hypercholesterolemia
- 2. F. Hypertriglyceridemia
- 3. F. Combined hyperlipidemia

Characteristics of major lipoproteins

Lipoprotein	Source	Composition (% mass)				Apolipoprotein	Electrophoretic mobility
		Pro	Cho	Tg	PL		
Chylomicrons	Gut	1	4	90	5	A, B, C, E	Origin
VLDL	Liver	8	25	55	12	B, C, E	Pre-β
LDL	VLDL via IDL	20	55	5	20	В	β
HDL	Gut/liver	50	20	5	25	A, C, E	α

Fredrickson's classification of hyperlipidemias

Туре	Electrophoretic	Increased lipoprotein	
1	Increased chylomicrons	Chylomicrons	
lla	Increased β-lipoproteins	LDL	
IIb	Increased β and pre- β -lipoproteins	LDL and VLDL	
Ш	Broad β-lipoproteins	IDL	
IV	Increased pre-β-lipoproteins	VLDL	
V	Increased chylomicrons and pre-β- lipoproteins	Chylomicrons and VLDL	

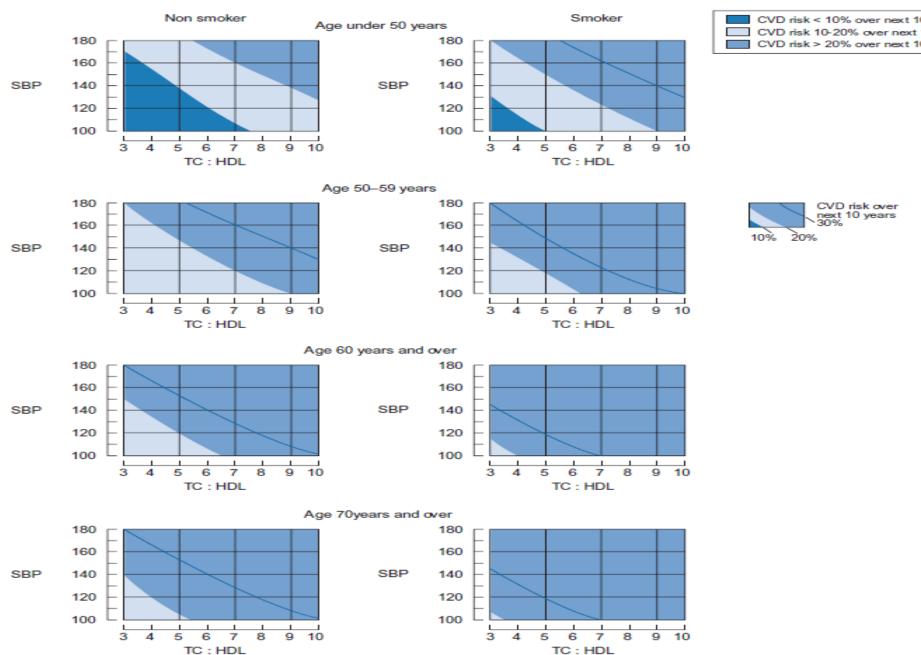
Secondary DL

- DM 1,2
- Hypothyroidism
- Chronic renal failure
- Nephrotic syndrome
- Obesity
- Alcohol
- Drugs (diuretics, B blockers, OCPs, corticosteroids, ciclosporin, enzyme inducers)

Primary prevention of stroke and MI

Risk assessment

NON-DIABETIC MEN



.3 Joint British Societies' cardiovascular disease risk prediction chart for non-diabetic men 2009 (reproduced with listy of Manchester). SBP, systolic blood pressure mmHg; TC:HDL, serum total cholesterol to HDL cholesterol ratio.

Secondary prevention (treatment)

Lipid profile
(Friedewald equation)
Life style change

Drugs

- 1. Statins
- 2. Fibrates
- 3. Cholesterol binders
- 4. Cholesterol absorption inhibiters
- 5. Nicotinic acid





A 15-year-old woman presented to the surgical unit with acute pancreatitis. Some of her laboratory results were as follows:

Plasma (fasting)

Cholesterol 33.4 mmol/L (3.5–5.0)

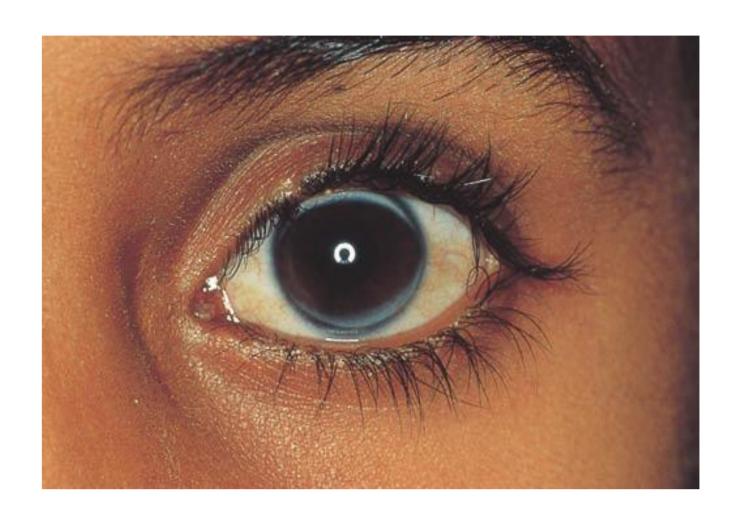
Triglyceride 69.1 mmol/L (0.3–1.5)

HDL cholesterol 0.9 mmol/L (1.0–1.8)

Amylase < 20 U/L (< 200)

On examination, she had eruptive xanthomata on her arms and thighs and fundoscopy revealed lipaemia retinalis.





A 23-year-old woman had her plasma lipids checked by her general practitioner because her father had died of a myocardial infarction aged 44 years. Her 24-year-old brother had hyperlipidaemia. Her renal, liver and thyroid function tests were normal, as was her blood glucose.

Plasma (fasting)

Cholesterol 11.4 mmol/L (3.5–5.0)

Triglyceride 1.1 mmol/L (0.3–1.5)

HDL cholesterol 1.2 mmol/L (1.0–1.8)

On examination, she had tendon xanthomata on her Achilles tendons and bilateral corneal arci.

A 43-year-old man attended the vascular surgery outpatient clinic for peripheral vascular disease. He was a non-smoker but had undergone a coronary artery bypass graft the year before. Some of his laboratory results were as follows:

Plasma (fasting)

Cholesterol 8.7 mmol/L (3.5–5.0)

Triglyceride 9.1 mmol/L (0.3–1.5)

HDL cholesterol 0.86 mmol/L (1.0–1.8)

His apolipoprotein E genotype was E2/E2

On examination, he had tuberous xanthomata and palmar striae.





Thank you