

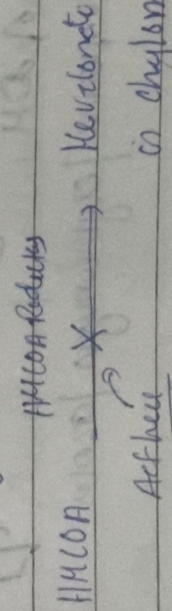
## HYPOLIPIDEMIC DRUGS

| HMG-CoA Reductase Inhibitor (Statins)                                                                                                                        | Fibrates                                                                                                                 | Stool Absorption Inhibitor                    | Post-prandial Lipid Inhibitor |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------|-------------------------------|
| <ul style="list-style-type: none"> <li>- Lovastatin</li> <li>- Simvastatin</li> <li>- Pravastatin</li> <li>- Rosuvastatin</li> <li>- Atorvastatin</li> </ul> | <ul style="list-style-type: none"> <li>Gemfibrozil</li> <li>Berberine</li> <li>Tenofibrate</li> </ul>                    | Ezetimibe                                     | Fenofibrate                   |
|                                                                                                                                                              | <u>Bile Acid sequestrants:</u> <ul style="list-style-type: none"> <li>• Colesevelam</li> <li>• Cholestyramine</li> </ul> | <u>Lipolysis &amp; triglyceride synthesis</u> | Nicotinic Acid                |

- Hypolipoproteinaemias can be secondary - Associated with diabetes, nephrotic syndrome.
- Primary due to - genetic defect of physical activity related cause.
- LDL is the primary cause of CVD
- VLDL is " " of TGs.

### HMG-CoA Reductase: (Statins)

- best tolerated hypolipidemic drugs.



in chylomicron synthesis & triglyceride

This Regulates Metabolism of LDL (LDL-C)

### Mechanism

HOA: statins completely inhibit HMG-CoA Reductase, the Rate Limiting step in cholesterol biosynthesis.



This Results in blood LDL ↓

VLDL ↓

↓ cholesterol synthesis → ↑ LDL Receptors in liver  
 ↑ LDL uptake degradation.

↓  
 Thus they're very effective in reducing plasma LDL.

Pk:  
 oral  
 M-H E-Bile

Also Reduce TG & ↑ HDL-chole level in Plasma.  
 → given once at night short half life

Use: Hypocholesterolemia  
 dyslipidemia  
 remission of CVD

↓  
 due cholesterol production ↑ so it work by Inhibit HMG CoA reductase

→ They are used for 1<sup>o</sup> & 2<sup>o</sup> hypercholesterolemia.

→ lovastatin & simvastatin are prodrugs converted in liver.

They undergo O<sup>+</sup> Phase Metabolism.

→ Beneficial effects in subject who have ↑ TC level but evidence of CAD may relate to poor CAD compliance & atherosclerotic plaque due to ↑ TC & ↓ HDL. It can reduce the change of plaque regression.

ADP: HMG CoA

- Hepatotoxicity with ↑ serum transaminase level
- Headache & sleep disturbance
- Myopathy: Muscle pain & weakness with ↑ creatinine kinase.
- Gastrointestinal: Anorexia, nausea, vomiting.
- New onset of DM.

C/I - liver disease  
 Pregnancy  
 Hypersensitivity

Drug Interaction: statins & cyclosporine / erythromycin / azoles.

## BILE ACID BINDING RESIN

cholesterol to bile Acid is ↑ → Transported to liver

↓  
 Resorption of bile Acid is ↑

↓  
 Liver

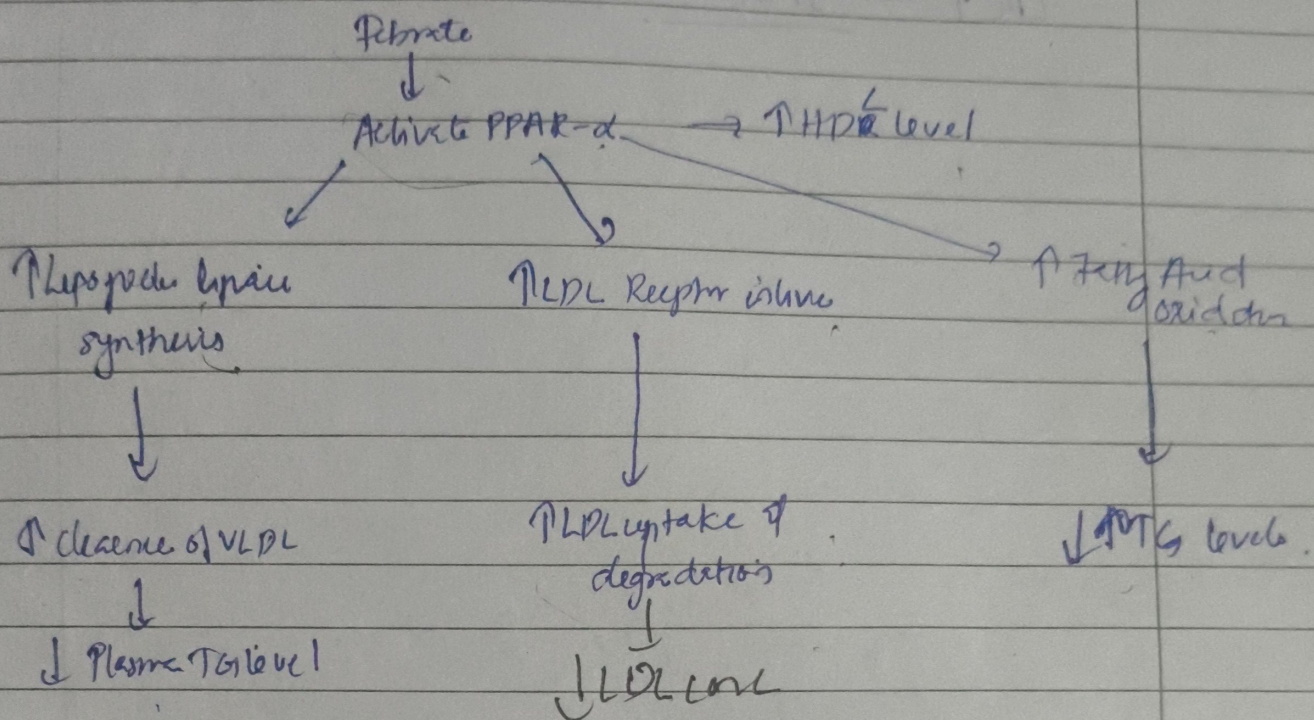
Resins bind bile Acid in the gut & interrupt their enterohepatic circulation, thus promote conversion of cholesterol to bile Acid in the liver.

⇒ ↓ LDL ↑ LDL Receptor

⇒ t/t for 1<sup>o</sup> hypercholesterolemia.

## FIBRATES: (Lipoprotein Lipase Activator) - Bezafibrate, Fenofibrate, Gemfibrozil

They Activate peroxisome proliferator - Activin Receptor  $\alpha$  (PPAR- $\alpha$ ) present in the liver, Adipose & skeletal muscle.



- Well Absorbed orally.
- effective in type II hyperlipoproteinemia.

Adverse: common effect are dyspepsia, ...

Incidence of gallstones w/ clofibrate.

## NICOTINIC ACID:

- B-complex vitamin:

Reduces TGs, VLDL, LDL, ↑ HDL levels.

