The Fate of Dietary Lipids

**THE TYPICAL LIPOPROTEIN PARTICLE:** is of **SPHERICAL SHAPE,** with a **COAT** and a **CORE**

**OUTSIDE:**
- **Made of** phosphatidyl choline, aka **LEcithIN**
  - This is a glycerol backbone with two fatty acids.
  - Being hydrophilic, the glycerol end of a lecithin molecule faces outwards, and the fatty acids face inwards, thus giving the lipoprotein a roughly spherical shape.
  - **THIS IS A LIPID MONOLAYER** (not bilayer like in cell membranes, because the inside of a lipoprotein is made of hydrophobic lipids)
- **ALSO:** the coat contains **PROTEINS** which give lipoproteins their name. These are responsible for targeting these proteins to specific cells.

**INSIDE:** the **TRANSPORTED LIPIIDS:**
- **Triglycerides and Cholesterol esters**

**EMBEDDED PROTEINS:**
- So-called **“Apo-lipoproteins”** because all lipid content has been leached from them.
  - **2 groups:**
    - **EXCHANGEABLE**
      - (can be swapped between two lipoprotein particles)
      - **A1, A2, C2, E**
    - **NON-EXCHANGEABLE**
      - (embedded forever in the lecithin layer)
      - **B48, B100**

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**~TYPES OF LIPOPROTEINS~**

<table>
<thead>
<tr>
<th>Type</th>
<th>Density (g/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Largest</td>
<td></td>
</tr>
<tr>
<td>Chylomicrons</td>
<td>0.95</td>
</tr>
<tr>
<td>Chylomicon remnants</td>
<td>1.0</td>
</tr>
<tr>
<td>VLDL</td>
<td>1.0</td>
</tr>
<tr>
<td>VLDL remnants</td>
<td>1.05</td>
</tr>
<tr>
<td>LDL</td>
<td>1.1</td>
</tr>
<tr>
<td>HDL</td>
<td>1.2</td>
</tr>
</tbody>
</table>

**Smallest**

*The higher the density, the greater the cholesterol content (cholesterol is the densest of the transported lipids)*

Thus, the big chylomicrons are almost completely full of triglycerides and HDL is made completely of cholesterol.

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**~LIVER~**

**HDLs:** the **GOOD cholesterol**
- ➔ are the **BACKWARD TRANSPORT** of cholesterol esters to the liver;
- **EARLY HDL:** no cholesterol; just a disk of lecithin; and ApoAI is present in the disk; docks with cells which are releasing cholesterol.
- ➔ this causes uptake of cholesterol into the HDL blob. BUT: the cholesterol is immature, i.e., needs to be esterified. This is done by the enzyme *LechithinCholesterolAcylTransferase (LCAT)* which transfers one fatty acid from lecithin into the cholesterol (thus esterifying it) and leaving a *lyso-phosphatidyl-choline remnant* (glycerol backbone with just one fatty acid);
- **THUS THE MATURE HDL IS FORGED**

**VLDLs** get synthesised and released by the LIVER: used in starvation as a source of energy

**LDLs** only one apoprotein (B100)
- ➔ bind to surfaces of cells which need cholesterol via LDL receptors
  - (statin drugs usefully promote expression of these)
  - If the apoB100 protein gets oxidised or glycosylated (e.g., in diabetes) it can no longer be recognised by the LDL receptor; needs to be taken care of by the “scavenger” receptor of macrophages; and macrophages then turn into the **FOAM CELLS** of atherosclerosis!