4.61 Lipoproteins

Lipoproteins, as the name suggests, are complexes of lipids and protein. The proteins within a lipoprotein are called apolipoproteins (aka apoproteins). There are a number of different apolipoproteins that are abbreviated apo-, then an identifying letter (i.e. Apo A) as shown in the chylomicron below.

![Chylomicron structure](image)

Figure 4.611 Chylomicron structure

The following video does a nice job of illustrating the different lipoprotein components.

**Web Link**

**Video: Lipoproteins (0:28)**

There are a number of lipoproteins in the body. They differ by the apolipoproteins they contain, size (diameter), density, and composition. Table 4.611 shows the difference in density and diameter of different lipoproteins. Notice that as diameter decreases, density increases.

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Density (g/dL)</th>
<th>Diameter (nm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chylomicrons</td>
<td>0.95</td>
<td>75-1200</td>
</tr>
<tr>
<td>VLDL (very low-density lipoproteins)</td>
<td>0.95-1.006</td>
<td>30-80</td>
</tr>
<tr>
<td>IDL (intermediate-density lipoproteins)</td>
<td>1.006-1.019</td>
<td>25-35</td>
</tr>
<tr>
<td>LDL (low-density lipoproteins)</td>
<td>1.019-1.063</td>
<td>18-25</td>
</tr>
<tr>
<td>HDL (high-density lipoproteins)</td>
<td>1.063-1.21</td>
<td>5-12</td>
</tr>
</tbody>
</table>
This inverse relationship is a result of the larger lipoproteins being composed of a higher percentage of triglyceride and a lower percentage of protein as shown below.

Figure 4.612 Composition of lipoproteins

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Protein</th>
<th>Phospholipid</th>
<th>Cholesterol</th>
<th>Tryglyceride</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chylomicron</td>
<td>100</td>
<td>90</td>
<td>80</td>
<td>10</td>
</tr>
<tr>
<td>VLDL</td>
<td>90</td>
<td>80</td>
<td>70</td>
<td>20</td>
</tr>
<tr>
<td>IDL</td>
<td>80</td>
<td>70</td>
<td>60</td>
<td>30</td>
</tr>
<tr>
<td>LDL</td>
<td>70</td>
<td>60</td>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>HDL</td>
<td>60</td>
<td>50</td>
<td>40</td>
<td>50</td>
</tr>
</tbody>
</table>

Protein is more dense than triglyceride (why muscle weighs more than fat), thus the higher protein/lower triglyceride composition, the higher the density of the lipoprotein. Many of the lipoproteins are named based on their densities (i.e. very low-density lipoproteins).

As described in the last subsection, the lipoproteins released from the small intestine are chylomicrons. The video below does a nice job of showing, describing, and illustrating how chylomicrons are constructed and function.

Web Link

**Video: Chylomicrons (0:55)**

The endothelial cells that line blood vessels, especially in the muscle and adipose tissue, contain the enzyme lipoprotein lipase (LPL). LPL cleaves the fatty acids from lipoprotein triglycerides so that the fatty acids can be taken up into tissues. The figure below illustrates how endothelial cells are in contact with the blood that flows through the lumen of blood vessels.
LPL cleaves fatty acids from the triglycerides in the chylomicron, decreasing the amount of triglyceride in the lipoprotein. This lipoprotein with less triglycerides becomes what is known as a chylomicron remnant, as shown below.

Chylomicron remnants are then endocytosed into the liver. This process of clearing chylomicrons from the blood takes 2-10 hours after a meal. This is why people must fast 12 hours before having their blood lipids (triglycerides, HDL, LDL etc.) measured. This fast allows all the chylomicrons and chylomicron remnants to be cleared before blood is taken. After the chylomicron remnant is endocytosed, it is broken down to its individual lipid components. In
the liver, VLDL are produced, similar to how chylomicrons are produced in the small intestine. The individual components are packaged into VLDL and secreted into circulation as shown below.

Figure 4.615 Chylomicron remnants are taken up by the liver. The liver secretes VLDL that contain cholesterol (C)

Like it does to chylomicrons, LPL cleaves fatty acids from triglycerides in VLDL, forming the smaller IDL (aka VLDL remnant). Further action of LPL on IDL results in the formation of LDL. The C in Figures 4.615 & 4.616 represents cholesterol, which is not increasing, rather since triglyceride is being removed, it constitutes a greater percentage of particle mass of lipoproteins. As a result, LDL is composed mostly of cholesterol, as depicted in the figure below.

Figure 4.616 Formation of IDL & LDL from VLDL
LDL contains a specific apolipoprotein (Apo B100) that binds to LDL receptors on the surface of target tissues. The LDL are then endocytosed into the target tissue and broken down to cholesterol and amino acids, as shown nicely in this video.

**Web Link**

[Video: LDL Receptor (1st 45 seconds)]

HDL are made up of mostly protein and are primarily secreted by the liver. HDL participates in reverse cholesterol transport, which is the transport of cholesterol back to the liver. HDL picks up cholesterol from tissues/blood vessels and returns it to the liver itself or transfers it to other lipoproteins returning to the liver.

![Figure 4.617 HDL is involved in reverse cholesterol transport](Image)

The animation under the transport button in the following link does a really nice job of going through the process of lipoprotein transport.

**Web Link**

[Lipoprotein Animation]

You are probably familiar with HDL and LDL being referred to as "good cholesterol" and "bad cholesterol," respectively. This is an oversimplification to help the public interpret their blood lipid values, because cholesterol is cholesterol; it's not good or bad. LDL and HDL are lipoproteins, and as a result you can't consume good or bad cholesterol, you consume cholesterol. A more appropriate descriptor for these lipoproteins would be HDL "good cholesterol transporter" and LDL "bad cholesterol transporter."
What's so bad about LDL? LDL enters the endothelium where it is oxidized. This oxidized LDL is engulfed by white blood cells (macrophages), leading to the formation of what are known as foam cells. The foam cells eventually accumulate so much LDL that they die and accumulate forming a fatty streak. From there the fatty streak, which is the beginning stages of a lesion, can continue to grow until it blocks the artery. This can result in a myocardial infarction (heart attack) or a stroke. HDL is good in that it scavenges cholesterol from other lipoproteins or cells and returns it to the liver. The figure below shows the formation of the fatty streak and how this can progress to a point where it greatly alters blood flow.

The following video does an excellent job of illustrating this process. However there are two caveats to point out. First, it incorrectly refers to cholesterol (LDL-C etc.), and second, it is clearly made by a drug company, so keep these factors in mind.

**Web Link**

**Video: Atherosclerosis (5:36)**
Despite what you learned above about HDL, a recent study questions its importance in preventing cardiovascular disease. It found that people who have genetic variations that lead to higher HDL levels were not at decreased risk of developing cardiovascular disease. You can read more about this interesting finding in the link below.

**Web Link**

*Doubt Cast on the ‘Good’ in ‘Good Cholesterol’*

The following video gives a general overview of macronutrient digestion, uptake, and absorption.

**Web Link**

*Video: Small Intestine (1:29)*

**References & Links**


**Links**


**Videos**

- Lipoproteins - http://www.youtube.com/watch?v=x-4ZQaiZry8
- Chylomicrons - http://www.youtube.com/watch?v=hRx_i9npTDU
- LDL Receptor - http://www.youtube.com/watch?v=XPGuYN7dcbE
- Small Intestine - http://www.youtube.com/watch?v=P1sDOJM658c