The liver, influenced by insulin, synthesizes Triglycerides (TGs) and packages them into VLDL; high TGs often indicate overactive VLDL production, linked to high carbohydrate intake or insulin resistance. As VLDL travels, Lipoprotein Lipase (LPL) removes TGs, transforming VLDL into cholesterol-rich LDL for cell delivery. Clinically, prolonged LDL in the blood oxidizes. These oxidized particles are consumed by macrophages, forming Foam Cells, which are the building blocks of atherosclerosis. In contrast, HDL performs beneficial reverse cholesterol transport. Statins, prescribed for high LDL, competitively inhibit HMG-CoA Reductase, a rate-limiting enzyme in cholesterol synthesis. This action prompts the liver to increase LDL Receptors, effectively clearing "bad" cholesterol from the blood.