Chylomicrons and very-low-density lipoproteins (VLDL) are the circulating particles responsible for transport of triglycerides from intestine and liver, respectively. Cholesterol ester exists with the triglyceride as a droplet surrounded by a monolayer of phospholipid-free cholesterol and specific proteins. These apolipoprotein now number nine.

Apolipoprotein B (apo-B) is an essential component of both chylomicrons and VLDL. Other apolipoproteins are removed during triglyceride hydrolysis by lipoprotein lipase in the peripheral tissues (e.g. muscle and adipose tissue) but apo-B remains with the residual lipoprotein particles in the bloodstream. This ‘remnant lipoprotein’ may be taken up by the liver or converted by poorly understood processes to low-density lipoprotein (LDL). The latter is triglyceride-poor but cholesterol-rich. Its long half-life in plasma (3 days) results in a large plasma pool of cholesterol in humans and is strongly associated with cardiovascular mortality.

Studies in our laboratory have examined the relation between dietary changes and the turnover of apo-B in VLDL and LDL. The first series of studies contrasted the effects of a diet containing 80% carbohydrate and 20% protein to a control diet of 40% carbohydrate, 40% fat and 20% protein. In 6 subjects with mild hypertriglyceridemia (type IV), triglycerides from 150 to 350 mg/dl the VLDL was isolated and labeled with $^{125}$I (50–75 µCi) and triglycerides were labeled by intravenous injection of H-glycerol (300 µCi). The high-carbohydrate diet increased the production rate of triglyceride by 10% in VLDL but did not change the VLDL apo-B production. Thus the output of the major lipid of this lipoprotein is not directly tied to the synthesis and secretion of its major structural protein. A second finding was an increase in the removal rate of apo-B as a component of VLDL before its final conversion to LDL apo-B. Thus the apparent synthesis of LDL apo-B was reduced during a period of increased triglyceride synthesis.