ApoC-III is an important apolipoprotein that is an independent risk factor for CHD. When present in increased amounts it is thought to interfere with binding of apoC-II to lipoprotein lipase and to prevent binding of apoB particles (LDL and VLDL) to LDL receptors. Both of these actions will delay the catabolism of TG-rich apoB containing lipoproteins, which can lead to increased blood viscosity, decreased flow-mediated dilation and elevated apolipoprotein B levels.

ApoC-III exists in three isoforms C-III₀, C-III₁ and C-III₂ contributing to 10, 55 and 35% respectively of total apoC-III levels. The isoforms are separated by their degree of sialylation (non, mono and di-sialylated), a Golgi mediated process. Sialylation is not related to lipid concentrations. ApoC-III₁ and C-III₂ are associated with the metabolic syndrome. Studies suggests that the di-sialylated CIII₂ isoform has high affinity for VLDL which makes the TG-rich particle particularly resistant to lipolysis. The increased residence time allows for CETP mediated transfer of TG to LDL and HDL and their subsequent lipolysis by hepatic lipase creating small LDL and HDL. Thus LDL particle size may be related to apoC-III sialylation and especially the apoC-III₂ isoform. This hypothesis may help explain the presence of numbers of small LDL in those with very variable plasma TG concentrations.





ApoCIII and LDL Particles



 LDL particles have delayed clearance and increased circulation time

TG-rich Lipoproteins and Inflammation









ApoC-III, Triglycerides and IR

