Perspective on Lipids and Lipid Control in Cardiometabolic Syndrome

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Visceral adiposity is thought to play a pivotal role in the pathophysiology of cardiometabolic syndrome (CMS). Visceral adiposity can activate inflammatory process in visceral adipose tissue, produce proinflammatory cytokines, and increase release of free fatty acids. In particular, free fatty acids and toll-like receptor (TLR) signaling mediates chronic inflammation in CMS and atherosclerosis.

Saturated fatty acids (SFAs) are known to activate macrophages and induce vascular inflammation. In addition, interaction between immune cells and other vascular cells (e.g. smooth muscle cells) contribute to atherogenesis. SFAs can be recognized by TLR4 complex, SFA increased the lipemia and provide lipids to be modified, SFAs modifies gut microbiota with production of endotoxin, and endotoxin causes oxidative stress and produces atherogenic lipids.

The mainstay of anti-atherosclerotic pharmacotherapy is statin-based lipid-lowering therapy. However, there is possibility of targeting other lipids for further reduction of cardiometabolic risk. A traditional pharmacologic agent, PPAR alpha agonist is known to have anti-inflammatory effect as well as controlling lipid metabolism. In addition, recent research including genetic studies indicate pathogenic role of triglycerides and triglyceride-rich lipoproteins (TRL). Pharmacologic approach with reducing triglyceride levels and cardiometabolic risk by targeting lipoprotein lipase, a key enzyme regulating TRL metabolism, is under early investigation.