I have previously posted a summary of the "HDL3-P Activation Index" assay and a description of Reverse Cholesterol Transport (RCT) showing HDL3-P as a primary contributor to the RCT process. Additionally, HDL3-P provides anti-inflammatory and antioxidant protection to LDL most likely from paraoxonase-1. In this process HDL3 was found to be a more powerful carrier of antioxidants than HDL2 as the protector of LDL. HDL3-P is an important player in cardiovascular health since it not only protects LDL from oxidation but provides the main path for excess cholesterol removal from both peripheral cells and macrophages building plaque in the arterial intima.

What has not been known about HDL3-P is that it appears to be activated or produced after the consumption protein, with or without carbohydrates or fat. Since people are in a non-fasting state most of the day, in theory, HDL3-P should be manufactured continuously to perform its function. Most cholesterol/lipoprotein testing is performed on fasting patients so observing the production of HDL3-P has gone un-measured. The HDL3-P Activation assay measures a fasting specimen and then after the consumption of a high protein drink a second specimen is taken for comparison. By subtracting the fasting result from the postprandial result, the production of HDL3-P can be measured.

In limited testing, it appears that the HDL3-P Activation is highly correlated to cardiovascular risk with patients having existing cardiovascular disease and/or diabetes mellitus showing poor HDL3-P activation and healthy patients showing strong activation. See the examples in this post. Logically, if a person does not produce postprandial HDL3-P, they're likely to develop cardiovascular disease since RCT is impaired and LDL not well protected from oxidation. I'm currently looking for collaborators interested in doing a clinical study using this patent pending technology.

