Introduction

Lowering total cholesterol -- and particularly LDL cholesterol -- levels has been shown to result in a 25% to 35% reduction in cardiovascular events. This means that a large number of patients who were given the drug and lowered LDL cholesterol continued to have cardiovascular events. In another way, 80% of people who develop CAD have the same blood cholesterol values as those who do not develop CAD. Therefore, other risk factors must be present to account for the observed increased risk of vascular events. Lp(a) is one such risk factor. **(Amy 2000)**

Analysis of data from the Framingham Study has revealed that elevated serum levels of a lipid particle, lipoprotein (a) [Lp(a)], are strongly associated with atherosclerosis and are an independent risk factor for CAD.

Up to 33% of CAD patients have elevated Lp(a) levels; elevated Lp(a) levels are associated with a 3-fold increase in the risk of a primary event; and 50% of offspring of patients with CAD express the gene associated with increased Lp(a).**(Jennifer 1993)**

In the Framingham Study, elevated Lp(a) levels were comparable in risk to a total cholesterol level >240mg/dl or an HDL level <35mg/dl. In the FATS study, those with higher baseline Lp(a) levels (>90th percentile) had a significantly greater loss of minimal lumen diameter. **(Alan et al 1999).**

However, the potential relation between LP (a) and IHD has been questioned by many studies, all of which failed to identify a similar relation. Thus, there is some controversy as to the role of LP (a) as an independent risk factor for IHD or not. **(Bernard el al 1998)**

It is possible that LP (a) may contribute to IHD risk through synergistic mechanisms with LDL cholesterol as suggested by a study of familial hypercholesterolaemia, and GRIPS trial. **(Alan et al 1999)**.