"If you have more than 20mg/dl of Lp(a) in your blood it begins to deposit plaques, causing atherosclerosis." — Linus Pauling

Chapter 4

Lp(a)

If you are human and reading these words, then you are alive but you probably cannot make vitamin C in your body. Therefore, you owe your continued existence to a form of cholesterol in the blood that was discovered in 1962 by Blumberg, et al. This peculiar form of *low density lipoprotein (LDL)* is called *lipoprotein(a)*, "small a," or simply *Lp(a)*. Lp(a) is not to be confused with *lipoprotein(A),* "large A," another lipoprotein which often appears on lab reports. They bear no relation to each other.

Lp(a) is an ordinary cholesterol particle with a sticky *apoprotein* particle called *apo(a)* attached to its surface. Lp(a) is formed in the liver. Lp(a) does not come in a standardized size or mass; the apo(a) may attach to a variety of low density lipoproteins during its formation. All human beings have the capacity to make Lp(a) and it is found in almost all human blood. However, there can be a thousand-fold range in its plasma concentrations. High levels of Lp(a) are associated with high incidence of cardiovascular disease.

Forty years after the Willis experiments it was discovered that only this one form of cholesterol, Lp(a), begins the process of forming atherosclerotic plaques. By 1990, after this discovery was experimentally verified in Germany, Linus Pauling and Matthias Rath proposed their new theory which singled out Lp(a) as the most significant and potentially the most dangerous variant of LDL ("bad") cholesterol. According to Dr. Rath: