Lipoprotein (a) - How To Reduce

By: Michael Lam, MD, MPH

Lipoprotein A, commonly called Lp(a), is a major independent risk factor for cardiovascular disease. The optimum laboratory level should be **under 20 mg/dl and preferrably under 14 mg/dl.** Currently, there are **no medications or drugs** that can effectively lower your Lp(a). A high Lp(a) is genetically linked.

Fortunately, Mother Nature has provided us a much better non-toxic alternative. It consist of large doses of vitamin C, L-lysine, and L-proline. Vitamin C, L-Lysine and L-proline are the basic building blocks of collagen. When these vitamins enter our bodies, they form collagen in large amounts. This is necessary, as collagen must be replenished in blood vessels to remain healthy and plaque free over periods of time. The reason is simple - Lp(a) is manufacturered in the liver in response to aging vascular system and "micro-fissures" in the endothelial vascular wall. The body, in its attempt to patch up these fissures, produce cholesterol and its relative Lp(a).

Unfortunately, both cholesterol and Lp(a) are sticky, making them perfect for the job. Lp(a) is many times more potent than cholesterol in its patching ability and has a tendency to attract other Lp(a) particles. The aggregation of Lp(a) forms a plaque that leads to vascular occlusion.

Lp(a) Cocktail

This mega vitamin cocktail therapy will increase blood concentrations of important substances and focuses on:

- Strengthen and heal blood vessels
- Lower LP(a) blood levels
- Inhibit the binding of LP(a) molecules in the walls of blood vessels

Only a few animals do not produce their own vitamin C, and humans are one of

them. **Vitamin C must therefore be taken from external source.** The amount varies between individuals and depends on the individual's health condition. Heart patients with serious conditions require more than normal individuals with high Lp(a). In addition to the above triple cocktail (vitamin C, L-lysine, and L-proline), other foundational nutrients are important to enhance vascular wall function. These include vitamin E and L-carnitine, a natural compound stimulating fatty acid oxidation in the mitochondria. To further enhance the effectiveness of the cocktail, it is important to use bioflavonoids and ascorbyl palmitate.

Many conventionally trained physician uses niacin to reduce Lp(a). This does work to a limited extend. Niacin reduces the production of lipoprotein A in the liver, and helps to bring down the lipoprotein A in the blood. This is what most conventional doctors use. However, this approach has its limitations because until the endothelial wall is optimized and cleared, the lipoprotein A level will not be able to reduce significantly. The effects of niacin usually hit a plateau after 6-9 months of therapy. If you are on niacin, make sure the liver enzyme levels are taken periodically to make sure the liver is able to handle the high dose of the niacin.

Vitamin C is water-soluble. A large amount is needed in order to reach adequate blood and tissue concentration. The amount of ascorbic acid can be reduced if ascorbyl palmitate, the fat-soluble form of ascorbic acid, is used at the same time. **This combination is also more effective, as it allows vitamin C can stay in the body much longer.** Improvements on LP(a) level can usually be seen within weeks to months for the majority of the people. The higher the starting value, the more significant was the reduction. Lp (a) can be completely normalized and bought to optimum level of under 14 mg/dl on nutritional therapies alone if treated properly. **Unfortunately, not all people show positive signs of reduction. Some people are particularly resistant, and may take upwards of 1 year to effect minor change.** In a small group or people, no change at all can be expected.

All people with high Lp(a) should be started on a nutritional cocktail. Even if repeat the blood level does not show any improvement, vascular integrity is enhanced. There is nothing to loose and everything to gain. Lipoprotein Lp(a) is a major and independent genetic risk factor for atherosclerosis and cardiovascular disease. It is relative of LDL or bad cholesterol The difference between Lp(a) and low density lipoproteins (LDL) is

apolipoprotein apo(a), a glycoprotein structurally similar to plasminogen, the precursor of plasmin, the fibrinolytic enzyme.

Lp(a) has the capacity to bind, fibrin and membrane proteins of endothelial cells and monocytes. It also stops plasminogen binding and plasmin generation. The inhibition of plasmin generation and the accumulation of Lp(a) on the surface of fibrin and cell membranes favor fibrin and cholesterol deposition at sites of vascular injury. Latent scurvy is characterized by a reversible atherosclerosis that closely resembles the clinical form of the disease.

Acute scurvy is characterized by microvascular complications such as widespread capillary hemorrhaging. **Vitamin C (ascorbate) is required for the synthesis of collagen,** the protein that is most critical in the maintenance of vascular integrity. **In latent scurvy, it has been suggested that large blood vessels use modified LDL, in particular lipoprotein(a) and collagen to maintain macrovascular integrity .** This is because collagen will be spared for the maintenance of capillaries, the sites of gas and nutrient exchange.

The foam-cell phenotype of atherosclerosis is identified as a mesenchymal genetic program, regulated by the availability of ascorbate. When vitamin C is limited, foam cells develop and induce oxidative modification of LDL. This stabilizing large blood vessels via the deposition of LDL. **The structural similarity between vitamin C and glucose suggests that hyperglycemia will inhibit cellular uptake of ascorbate, inducing local vitamin C deficiency.** Nicotinic acid has been made tolerable with sustained-release formulations, and is still considered an excellent choice in elevating HDL cholesterol. It is also potentially effective in reducing lipoprotein(a) [Lp(a)] levels.

Although LDL cholesterol is still the major target for therapy, it is likely that in future, other lipid/lipoprotein and nonlipid parameters will also become targets for specific therapeutic interventions. **Other significant** lipid/lipoprotein parameters that have been associated with CHD include elevated triglyceride, oxidized LDL cholesterol and Lp(a) levels, elevated C Reactive Protein, elevated homocysteine, and low HDL levels.

Normalization of Traditional Risk Factors

Optimum cardiovascular health entails controlling the well-known risk factors including elevated cholesterol levels, hypertension, and stress. Elevated cholesterol levels can be controlled through drugs or natural supplements such as fiber, niacin, and more recently, red yeast rice powder (600 mg two times a day). **Control of hypertension can be accomplished through drugs and/or natural supplementation such as Hawthorne (250 mg), magnesium (400 – 700 mg), calcium (800 – 1,000 mg), and potassium from food (400 – 600 mg). Adjunct nutritional supplementation to enhance stress reduction includes antioxidant nutrients such as**
coenzyme Q10 (CoQ10) and Vitamin E, Vitamin B complex, and Magnesium. These antioxidants counteract the increase in oxidative stress **associated with psychological stress**.

Discussion

It is obvious that the traditional risk factors such as cholesterol, hypertension, and stress, while good markers of cardiovascular disease, do not tell the whole story. The fact that many people develop cardiovascular disease in spite of normal cholesterol levels, normal blood pressure values, and relatively low stress environment imply that there are other risk factors yet to be discovered. Four such markers are explored. **Standardized laboratory references have yet to be established for these new markers and may never be established due to their multifactorial complexity of cardiovascular disease. A natural nutritional supplementation approach appears to the best available option for those who don't want to wait.**

a. **Lipoprotein(a):**

Ascorbyl Palmitate 200 – 400 mg;

L-Lysine 200 – 400 mg;

L-Proline 200 – 400 mg; and

Ascorbate 1,000 – 3,000 mg.

b. **Mitochondrial Function:**

Coenzyme Q10 30 – 120 mg;

L-Carnitine 500 – 2,000 mg; and

Lipoic Acid 150 – 300 mg.

c. **Homocysteine Axis:**

Folate 400 – 800 mcg; and

Vitamin B12 400 – 800 mcg.

d. **Oxidative Stress:**

Vitamin E 400 – 800 IU.