Linus Pauling himself ingested 18,000 mg daily of vitamin C daily in order to approximate the tissue levels of an animal of his body weight.

When serum Lp(a) is elevated, high dose *Lp(a) binding inhibitors* can profoundly interfere with the disease process. Binding inhibitor formulas that include proline have been documented to lower Lp(a) in six to 14 months. In cases where serum Lp(a) is not reduced, binding inhibitors become even more important to neutralize Lp(a), regardless of their effect on serum Lp(a) levels.

Recently a reevaluation of the Framingham Heart study found that Lp(a), and not ordinary LDL, is highly predictive of CVD. The Oxford researchers said that elevated Lp(a) increases the risk of heart attack and stroke by 70%.

The on-going lack of scientific curiosity or interest by organized medicine in the Pauling/Rath theory and Pauling's high-dose therapy may well be recognized as the greatest lapse of 20th century medicine.

Heart disease protocol

Vitamin C as *ascorbic acid* should be taken up to bowel tolerance (**3 to 18 g** *per day in divided doses*)**.**
Lysine should be taken from **3 to 6 g** daily for the greatest therapeutic benefit (*2 to 3 g daily for prevention.)*
Supplement Coenzyme Q10 (100 - 300 mg daily) (*High vitamin C and several vitamins will help stimulate your own synthesis of CoQ10 which is vital for proper heart function*.)
Proline from 250 mg to 2000 mg daily. (*This added factor may lower elevated Lp(a) within 6 to 14 months*.)
Eliminate trans fats/hydrogenated oils. Increase Omega-3 oils. (*These protocols increase vitamin C utilization by cells*.)
Follow Paulings general heart and cardiovascular recommendations provided in his book *HOW TO LIVE LONGER AND FEEL BETTER* , e.g., **Vitamin E** - 800 to 3200 iu ,**Vitamin A** - 20,000 to 40,000 iu , and **Super B**-Complex, esp. Vitamins B6 and B3
Supplement the mineral Magnesium (300 to 1500 mg) and avoid Manganese (*No more than 2 mg of manganese. USDA researchers report that elevated manganese, more than 20 mg daily, competes with magnesium uptake in the heart causing irregular heart beats*.)
Avoid refined carbohydrates, especially sugars which crowds out the similar vitamin C molecules from entering cells.
Supplement the amino acids **Taurine, Arginine and Carnitine** (**1 to 3 g**).
Add a good mineral/multivitamin

Vitamin C and Heart Disease

By Owen Richard Fonorow, © 2005

Cardiovascular Diseases The few species which include humans, that do not make their own vitamin C suffer a condition called *atherosclerosis*, where white plaques narrow the arteries. This disease has been misnamed "heart disease" because it often leads to a heart attack. The disease is *not* prevalent in species that make their own Vitamin C.

Chronic Scurvy Heart disease is a misnomer because there is no malfunction of the heart. The underlying disease process is characterized by scab-like build-ups that adhere to the walls of blood vessels. As the arteries narrow, the blood supply to the heart and the other organs is reduced, resulting in angina ("heart cramp"), heart attack and/or stroke. The more correct terminology is ***chronic scurvy***, a sub clinical *(difficult to detect*) form of the classic vitamin C deficiency disease *scurvy*.

Lp(a) Binding Inhibitors Chronic scurvy appears in the few beings that do not make vitamin C, and its primary characteristic, *atherosclerosis,* is caused by a sticky form of cholesterol called *Lp(a).* Linus Pauling and Matthias Rath invented *Lp(a) binding inhibitors to* prevent this molecule from binding to the walls of damaged arteries. Together, vitamin C and the amino acid lysine are the primary binding inhibitors. These substances at high dosages are patented to prevent and to destroy existing atherosclerotic plaques. *Vitamin C*, will cure the chronic scurvy, reduce oxidation, increase collagen for healing and improve the health and strength of arteries, while high dose *Lysine* destroys Lp(a) plaques.

Twelve years of clinical experience with high dosages of the *Lp(a) binding inhibitors* **vitamin C** and **lysine** in humans have matched Pauling's own initial findings and case studies. The dosages of **vitamin C** (*as ascorbic acid*) that achieve therapeutic effects are between 3 g to 18 g (3000 mg to 18,000 mg ascorbic acid) daily. The dosages of **lysine** are between 3 to 6 g (3000 mg to 6000 mg). These vital nutrients, taken together from 2 to 4 times daily, are clinically effective at the recommended strength. Only high, sustained dosages of the Lp(a) binding inhibitors become the specific ***Pauling Therapy*** for rapid reversal of "heart disease."

History The theory that cardiovascular Diseases (CVD) are related to a deficiency of vitamin C was first proposed by the brilliant Canadian physician G. C. Willis, MD. By the 1940s, it was already known that vitamin C is required for strong healthy arteries. Willis and another Canadian, J. C. Paterson, both found in separate studies that the tissues of

heart patients were depleted of vitamin C. In 1953, Willis discovered that atherosclerotic plaques form over vitamin-C-starved vascular tissues in both guinea pigs and human beings. Willis published several papers on this subject that were peer-reviewed in the *Canadian Medical Journal*. His team also discovered that vitamin C can reverses the process of atherosclerosis in both guinea pigs and humans.

Willis devised the first known method of photographing plaques with X-rays. He observed that human plaques were not distributed uniformly throughout the vascular system. Instead, these "blockages" concentrate near the heart, where arteries are constantly bent or squeezed. He reasoned that only the mechanical stress, caused by the pulse, could explain the typical pattern of *atherosclerosis* in humans. The body was forming plaque precisely where it was needed in order to stabilize the vascular system.

By the late 1980s, medical researchers discovered that heart disease begins with a lesion; lesions are cracks or stress fractures in the arterial wall. The ensuing question became: *What causes lesions in human beings since they do not arise naturally in most other animals?*

Then a variant of the so-called "bad" LDL cholesterol called lipoprotein(a), or Lp(a), was studied and found to be *really* sticky. The stickiness is caused by points of attachment on the surface of the Lp(a) molecule called *lysine binding sites*. Studies that led to the 1987 Nobel prize in medicine revealed that lysine (and later proline) binding sites on the Lp(a) molecule cause *atherosclerosis*. Following that, Beisiegel *et. al.* in Germany examined human atherosclerotic plaques *post mortem* and found only Lp(a) cholesterol, not ordinary LDL cholesterol, as expected. (*Prior to Beisiegel, studies grouped Lp(a) together with LDL; both are low density lipoproteins*.)

By 1989, Linus Pauling and his associate, Matthias Rath, had formulated and published their unified theory of heart disease. Pauling/Rath theorized that the lack of vitamin C is the primary cause of heart disease in humans. Low vitamin C inevitably leads to *atherosclerosis*. They discovered that serum Lp(a) increases in response to the distress of vitamin C-starved tissues.

Pauling and Rath had repeated the earlier Willis experiments with guinea pigs, but this time they monitored Lp(a). They discovered an inverse relation between vitamin C and Lp(a). Their theory holds that in most species, vitamin C is plentiful and helps prevent arterial lesions. However, in a few species, such as humans and guinea pigs, serum Lp(a) levels increase to compensate for chronic scurvy. Thus Lp(a) is the second major genetic

difference between beings that suffer heart disease and those that do not. (*The first is the inability to make vitamin C.*) Lp(a) has evolved in humans, some primates, guinea pigs, some parrots and fruit bats to compensate for the missing vitamin C, notably to patch cracked blood vessels.

The majority of the population is not aware they have "chronic scurvy." As the condition progresses, the liver produces more Lp(a) molecules. A large number of Lp(a) molecules tend to deposit on top of existing plaque formations. When the healing process overshoots, the arteries narrow and the flow of blood is reduced. The first symptom may be a heart attack.

The Cure The Lp(a) molecules have a limited number of lysine binding sites - points of attachment to lysine. Pauling's invention - the cure for heart disease - increases serum lysine high enough to make the Lp(a) unattractive to any more lysine binding sites. As more lysine enters the blood stream, the probability increases that floating Lp(a) molecules will bind with it (*rather than with the patches of plaques growing on the arterial walls*.)

Pauling and Rath's protocols for destroying existing atherosclerotic plaques relies on more than fifty years of scientific research. Pauling filmed a video lecture in which he summarizes this research, and where he recommended that heart patients take the amino acid lysine daily in high amounts with their vitamin C. Neither vitamin C nor lysine have any known lethal dose.

In his one hour video lecture, Pauling recounts the first cases in which his high lysine therapy added to high vitamin C quickly resolved advanced cardiovascular disease in humans. Pauling doubted a clinical study was even necessary before advising heart patients of the therapy because the effect is so pronounced, and the binding inhibitors are nontoxic.

Recently, *in vitro* test tube experiments have shown that the amino acid proline may be an even more effective Lp(a) binding inhibitor than lysine. Consequently, adding between .5 and 2 g (500 mg to 2000 mg) *proline* may be of significant additional benefit.

Prevention Linus Pauling believed that *chronic scurvy* might be prevented with a daily intake of of vitamin C as low as 3000 mg. This amount approximates what some animals synthesize under normal conditions. Dr. Sydney Bush's Cardioretinometry (microscopic pictures of the retina) suggests that some people require up to 10,000 mg daily for prevention.