The Cure for Heart Disease: Condensed

By Owen R. Fonorow, Copyright 2004

'READER'S DIGEST' VERSION

Cardiovascular Diseases Those few species that fail to synthesize ascorbic acid (vitamin C) are prone to a form of ‘heart disease’ that is not prevalent in other species. The theory that Cardiovascular Disease (CVD) is related to a deficiency of vitamin C was first proposed by the Canadian physician G. C. Willis in 1953. He found that atherosclerotic plaques form over vitamin-C-starved vascular tissues in both guinea pigs and human beings. In 1989, after the discoveries of the Lp(a) cholesterol molecule (*circa* 1964) and its lysine binding sites (*circa* 1987), Linus Pauling and his associate Matthias Rath formulated a unified theory of heart disease and invented a cure. Vitamin C and lysine (and proline) in large amounts become ***Lp(a) binding inhibitors*** that restore vascular health and are patented to destroy atherosclerotic plaques.

Chronic scurvy. Heart disease is a misnomer; the underlying disease process reduces the supply of blood to the heart and other organs leading to angina ("heart cramp"), heart attack and stroke. The disease is characterized by scab-like build-ups that grow on the walls of blood vessels. The correct terminology for this disease process is ***chronic scurvy***, a slower form of the classic vitamin C deficiency disease.

The hypothesis that CVD is an ascorbic acid (vitamin C) deficiency disease was first conceived and tested in Canada. Willis devised a method of photographing plaques with X-rays and observed that atherosclerotic

plaques were not uniformly distributed throughout the vascular system; rather these "blockages" are concentrated near the heart, where arteries are constantly bent or squeezed.

Another Canadian, Paterson, had found that the tissues of heart patients were generally depleted of vitamin C, and it was well known that vitamin C is required for strong and healthy arteries. Willis reasoned that only the mechanical stress caused by the pulse could explain the typical pattern of atherosclerosis. To Willis, the body was laying down plaque precisely where it was needed in order to stabilize the vascular system.

By the late 1980s, medical researchers had made several intriguing discoveries.

First came the discovery that heart disease begins with a lesion, a crack or stress fracture, in the arterial wall. The question became, and remains, as to the cause of these lesions in human beings since they do not arise in most other animals. Then a variant of the so-called "bad" LDL cholesterol called lipoprotein(a), or Lp(a) for short, was studied and found to be *really* bad. Lp(a) is sticky because of receptors on the surface of the molecule called ***lysine binding sites***. Work that led to the 1987 Nobel prize in medicine discovered that lysine (and proline) binding sites cause the formation of atherosclerotic plaques. Then, Beisiegel *et. al.* in Germany examined plaques post mortem and found only Lp(a), not ordinary LDL cholesterol.

Lp(a) was the genetic difference between beings that suffer cardiovascular disease and those that do not. Lp(a) had evolved only in species that do not make their own vitamin C - e.g. humans and guinea pigs.

Pauling and Rath repeated the earlier Willis experiments, but this time they monitored Lp(a). They discovered that it becomes elevated in guinea pigs

deprived of vitamin C, but not in the controls. These experiments connected elevated-Lp(a) with low serum vitamin C. They realized that in most species, sufficient ascorbic acid will prevent stress fractures, but in those species that suffer chronic scurvy, Lp(a) had evolved to patch cracked blood vessels.

As chronic scurvy progresses, the liver produces more Lp(a) molecules. As the number of Lp(a) molecules increases, they tend to deposit on top of existing plaque formations. When the healing process overshoots, the arteries narrow and the flow of blood is reduced.

This problem has a solution. The Lp(a) molecule has a finite number of lysine binding sites - points of attachment to lysine. Pauling's invention - the cure for heart disease - is to increase the serum concentration of the amino acid lysine enough to make the Lp(a) unattractive. 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*.)

After all the Lp(a) molecule's binding receptors are filled with the free lysine floating in the blood, the Lp(a) molecule becomes as harmless as ordinary LDL cholesterol.

Pauling and Rath called the substances that treat chronic scurvy and destroy existing plaques ***Lp(a) binding inhibitors***. Vitamin C, to increase collagen production and to improve the health and strength of arteries, and lysine, to prevent and to dissolve Lp(a) plaques, are the primary binding inhibitors. These substances taken together are clinically effective.

Linus Pauling believed that chronic scurvy can be prevented with an orthomolecular daily intake of between 3,000 to 10,000 mg or more vitamin C. This amount approximates what the animals synthesize, and

matching animal production is the reason Pauling ingested 18,000 mg daily.

Pauling and Rath's invention for destroying existing atherosclerotic plaques is the large amount of another essential nutrient, the amino acid lysine. Pauling filmed a video lecture in which he recommended that heart patients take between 2,000 and 6,000 mg of lysine daily with their vitamin C (more if serum Lp(a) is elevated). Neither vitamin C nor lysine have any known lethal dose.

The Lp(a) binding inhibitors become the ***Pauling Therapy*** for heart disease only at high dosages, between vitamin 3 to 18 g ascorbic acid and 3 to 6 g lysine. In his video, Pauling recounts the first cases where his high vitamin C and lysine therapy quickly resolved advanced cardiovascular disease in humans. The effect is so pronounced, and the inhibitors are so nontoxic, that Pauling doubted a clinical study was even necessary.

Recently, the amino acid proline was found to be an even more effective Lp(a) binding inhibitor than lysine *in vitro*. Adding between .5 and 2 g proline may be of significant additional benefit.

When serum Lp(a) is elevated, 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 Lp(a) is not reduced, binding inhibitors become even more important to neutralize Lp(a) regardless of their effect on serum Lp(a).

Recently a reevaluation of the Framingham Heart study that Lp(a) and not ordinary LDL is highly predictive of CVD and Oxford found 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 the 20th century.

Heart disease orthomolecular protocol

NOV 2005: UPDATED PROTOCOL HERE

1. **Take Vitamin C** as ascorbic acid (*or sodium ascorbate, but this form may be less effective*) up to bowel tolerance (**3 to 18 g per day** in divided doses.)

   The half-life of vitamin C in the blood stream is 30 minutes. NIH findings indicate minimum 500 mg every 4 hours leads to highest sustained blood levels, take more before bed, trips, etc. Trouble with bloating/gas/diarrhea after your vitamin C? Try Liposomal Vitamin C
2. **Take Lysine. 2 to 3 g daily** for prevention and from **3 to 6 g daily** for the greatest therapeutic benefit**.**
3. **Supplement Coenzyme Q10 (**100 - 300 mg) (Note: Vitamin C and several vitamins will help stimulate your own synthesis of CoQ10. CoQ10 is a vital substance for energy and proper heart function. Popular drugs interfere with your body's own production of CoQ10, and they may lead to heart failure)
4. **Take Proline** from 250 mg to 2000 mg daily. (This added factor may lower elevated Lp(a) within 6 to 14 months.)
5. NEW: Eliminate man-made/processed fats, such as trans and hydrogenated fats, and supplement Omega-3 rich oils. "Research has

shown that an Omega-3 Index of 8 percent to 10 percent reduces a person's relative risk of death from coronary heart disease by 40 percent, and from sudden cardiac death by 90 percent." This benefit probably results from restored insulin-mediated glucose/vitamin C uptake into cells. [See: Protocol for Reversing Diabetes Type II by Eliminating Hydrogenated and Trans Fats and adding Omega-3 oils...]

Note: Following an Atkins-style diet will eliminate most trans fats because these "poisons" appear mostly in processed carbohydrate foods such as cookies, crackers, snacks, etc. Butter is vastly supperior to margarine. Natural saturated fats are vastly superior to any fats or oils processed for longer shelf life.

6. NEW: Eliminate ordinary sugar and refined carbohydrates. New research confirms Dr. John Ely's 30-year theory that sugar (glucose) competes with ascorbic acid (Vitamin C) for insulin-mediated uptake into cells. Taking sugar can effectively crowd out the Ascorbate. The effect of the Pauling Therapy is reportedly much more pronounced and immediate when sugar is eliminated (and good Omega-3 fatty acids are added.)
7. **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 , andSuper B-Complex, esp. Vitamins B6 and B3
8. **Supplement the mineral Magnesium** (300 to 1500 mg) and avoid Manganese (No more than 2 mg. USDA researchers report that elevated manganese, more than 20 mg daily, competes with magnesium uptake in the heart causing irregular heart beats.)

Manganese alters mitochodrial integrity in the hearts of

> swine marginally deficient in magnesium ... *These results suggest that high Mn, when fed in combination with low Mg, disrupts mitochondrial ultrastructure and is associated with the sudden deaths previously reported.*

9. **Eat salt, only unrefined salt,** Brownstein discovered literature that a low-salt diet can cause the body to change its hormonal balance as it attempts to retain sodium. This leads to a 400% chance of heart attack in those with high blood pressure and low sodium intake [*]. Refined (ordinary table salt) is poisonous, but unrefined salt has over 80 minerals and can be considered a necessary "health food."
10. A**void supplemental calcium**, and supplement vitamin K for proper calcium metabolism, especially if you have taken antibiotics or blood thinners in the past.
11. **Add a good mineral/multivitamin**
12. Supplement the amino acids **Taurine, Arginine and Carnitine (1 to 3 g).**

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