# Heart Disease Prevention - A Complete Nutritional Approach

**Michael Lam, MD, MPH**
**www.DrLam.com**

(READING TIPS: For fast reading, scan through the topic headings in **BOLD BLACK**, important conclusions in **BOLD BLUE,** and " Must Know " in **BOLD RED**. To jump to specific sections in this article, click on the respective **LINKS**

**Before You Begin**

Information presented here is for general educational purposes only. Each one of us is biochemically and metabolically different. If you have a specific health concern and wish my personalized nutritional recommendation, write to me by **clicking here**.

## Contents



## Introduction

At least 14 million Americans have heart disease and more than 2,600 die every day from heart attacks in the United States alone. Cardiovascular disease afflicts around 15% of adults in their late 30s to 40s, about 50% of 55 to 64 year-olds, and 65% will be afflicted in the next decade of their lives.

After 20 years of aggressive drug therapy and promotion of low-fat diets, the tide on cardiovascular disease has not reversed. Obviously, this elusive condition is far more complicated than we ever imagined. It is clear that there are other factors that have not been addressed.

## Cause of Cardiovascular Disease

For decades, the public at large has been taught that the key culprit of heart disease is high cholesterol in our blood that comes from a diet that is high in cholesterol. This notion must be downgraded.

Consider the following:

Polar bears, for example, maintain total blood cholesterol of over 400 mg/dl and they seldom develop heart attacks.

Eskimos are relatively free of heart disease. They eat animal fats from fish and marine animals liberally.

The Okinawans are the longest living population group in the world. The average life span for Okinawan women is 84 years. Their diet consists of an intake of fish 2-3 times a week and a high intake of vegetables. Their cholesterol intake on the whole is more than most.

People in North India consume 17 times more animal fat but have 7 times fewer incidences of heart disease compared to people in southern India.

In the Framingham study, men and women consumed an average cholesterol intake of 700 mg and 500 mg per day respectively (one egg provides 200 mg). The average serum concentration of cholesterol for men and women with higher than average cholesterol intake was found to be 237 and 245 mg/dl respectively. Subjects with lower than average intakes had an average serum concentration of 237mg/dl for men and 241 mg/dl for women. The actual number difference insignificant. Statistically, studies have shown that **people who consume 4 eggs per week actually have average serum cholesterol (193 mg/dl), which is the same as those who reported consuming only 1 egg per week (197 mg/dl). Clearly dietary cholesterol in and of itself is not the critical link to heart disease risks as we once thought.**

Today, few experts deny that the **low-fat message of the past three decades is radically oversimplified.** If nothing else, it effectively ignores the fact that mono-unsaturated fats like olive oil is full of omega-3 fatty acid, is good for health and must be consumed in large amounts. Bad fats such as overcooked saturated fats from meat or trans-fat from processed food should be avoided.

While a diet high in unhealthy fat can promote heart disease, it is only one of many factors that increase cardiovascular risk. Science is telling us that in fact, it is only a minor reason. Other than the familiar hypercholesteremia, **the main reason for high blood cholesterol is excessive metabolism of oxygen and sugar in our blood stream** due to the polluted environment, and a diet high in refined carbohydrate, trans-fat, and a stressful lifestyle. This leads to free radical generation that in turn damages the endothelial wall of the blood vessel. The body has an intrinsic repair mechanism to overcome the damage, but it needs the proper nutrients to get the job done. Some nutrients are made internally, while others need to be supplied externally. In the case of blood vessel repair, the key is ascorbic acid. It cannot be made endogenously and has to be taken in externally from food sources.

Sad to say, but the food we eat today is far different from that of our grandparents. Today's diet simply cannot provide all the nutrients needed by the body to repair the damaged endothelium. Our soils are depleted of nutrients, the amount of chemicals and preservatives are at an unprecedented high level, and the high heat we use to cook our food with nothing short of extreme. The wholesome meal that our grandparents ate is now replaced by frozen and processed food when we are not able to go to a fast food restaurant. Even the 65 mg of Vitamin C in one orange gets only fractionally delivered to our body by the time it makes the journey from the orchard to our kitchen. Our body was never designed to take in large quantity of glucose from the breakdown of pastas, breads, French fries, cookies, and soda over several years. It simply does not have the ability to process those foods properly without residual damaging effects.

Lacking the specific nutrients in order to carry out the repair process properly, the body puts its emergency repair team into action. It instructs the liver to produce cholesterol (a sticky and waxy substance) as a surrogate in its attempt to repair damaged arteries by covering the damaged areas. The cholesterol that is produced travels from the liver to the damaged areas as LDL (bad) cholesterol. It is further converted into oxidized LDL cholesterol and sets off a cascading inflammatory reaction. This eventually leads to a thrombus formation, the reduction of nitric oxide synthesis, a high blood pressure, and ultimately the blockage of blood vessels which can result in heart attacks or strokes.

A high cholesterol blood level can therefore be viewed as a sign of underlying vascular wall dysfunction at the

endothelium and defect in our insulin's activity against glucose. Unfortunately, this has gone unrecognized. Instead, the cholesterol myth has lead researchers to focus on stopping the production of cholesterol from the liver by the use of drugs.

## Advanced Markers of Cardiovascular Disease

**The all out assault on lowering cholesterol has failed to reduce the incidence of heart disease because the root cause of heart disease does not lie in cholesterol alone.** To use total cholesterol and LDL as a surrogate end point in measurement of cardiovascular disease risk is rudimentary at best given the amount of scientific research available.

**Are there any alternative markers that help us formulate a more complete picture of heart health from multiple angles? Indeed there are and they have been known for years.** However, most of these markers have been ignored because there are no drugs available to "normalize" them. No doubt as drugs are developed, these markers will take on significant commercial value, and that is when their importance will be publicized. For the mean time, as most medical students are taught: never order a test when you know ahead of time you are not going to know what to do with the results.

**It is far better to incorporate the following sensitive and easily obtainable indicators when assessing cardiovascular risk. They are listed in decreasing order of importance (the most important and sensitive listed first), as follows:**

1. **Lipoprotein (a)** – indicator of endothelial wall integrity
2. **Homocysteine** – indicator of free radical activity
3. **Fibrinogen** – indicator of thrombus formation and blood viscosity
4. **Arterial Stiffness** –indicator of wall flexibility and blood pressure health
5. **Cellular Energy Generation** – indicator of mitochondrial function
6. **C Reactive Protein** – indicator of inflammatory response
7. **Triglyceride** – leading cause of metabolic syndrome
8. **Total cholesterol / HDL cholesterol ratio** –key indicator in lipid metabolism
9. **LDL cholesterol** – indicator of the level of "bad" cholesterol
10. **Total Cholesterol** – overall indicator of total cholesterol in blood.

**As seen above, cholesterol is near the bottom in terms of sensitivity and predictability of cardiac accidents when compared to other indicators.**

This paper examines each of these markers in more detail and suggests conventional and nutritional therapeutics that can normalize each of these indicators.

**While none of these key indicators are by themselves and an absolute prognosticator of impending heart attack or stroke, there is little doubt that as a group, the overall predictive value is overwhelming significant and has a strong predictive value.** They offer the best that science can offer today, short of scans and invasive procedures.

### 1.The Lp(a)

Autopsy studies of heart attack victims have shown that many have clean vessels and normal cholesterol levels. It is obvious that there are other causes for heart disease. Indeed, researchers with the Framingham Heart Study (the decades-long study that brought us the term "risk factor") identified a relative of LDL-cholesterol called **lipoprotein (a) [Lp(a)], which is now recognized as a major independent risk factor for heart disease.** While LDL cholesterol maybe known as the "bad" cholesterol, Lp(a) is even worse. Lp(a) is a plasma lipoprotein that structurally resembles LDL, but with additional adhesive properties. Some of the natural cholesterol produced by the liver in response to free radical damage is converted into LDL cholesterol and its relative Lp(a). Lp(a) fosters cholesterol deposition by enhancing oxidation of LDL-cholesterol. It is the oxidized form of cholesterol that penetrates the endothelium, leading to both the buildup of plaque and

vascular disease. **It should be noted that artery blockage (plaque) is composed mainly of Lp(a) and not of ordinary cholesterol**.

Oxidized cholesterol is a free radical generator. Research has shown that rabbits that consumed a small amount of oxidized cholesterol for merely 12 weeks had atherosclerosis plaques that were two times as big as the control population. Studies reveal that heart attack risk falls 2% for every 1% drop in LDL cholesterol level.

Studies have also shown that Lp(a) holds fast to damaged blood vessels, attracting other Lp(a) molecules, and finally constituting the atherosclerotic plaques. In fact, a high Lp (a) level (more than 30 mg/dl) has been revealed to carry a 10 times greater risks for heart disease than LDL cholesterol level.

Linus Pauling, two-time Nobel Laureate, postulated that Lp(a) may be the surrogate for ascorbate in humans. Low dietary intake of ascorbate leads to weaken blood vessels because ascorbate is required for the synthesis of collagen and elastin, which strengthen the blood vessel wall. In the absence of ascorbate, Lp(a) is mobilized to repair these structural defects in arterial walls by being deposited to strengthen the tissue. However, if the plasma concentration of Lp(a) is too high, the process goes too far. Too much Lp(a) gets deposited in the arterial wall, and plaque formation is initiated.

Chronic depletion of these essential nutrients such as vitamin C, lysine, and proline in the endothelial and vascular smooth muscle cells impairs their ability to function properly. Guinea pigs fed a diet low in ascorbate rapidly developed atherosclerotic plaques, similar to those found in humans. When large amounts of supplementary ascorbate were given to these guinea pigs, there was a regression in plaque formation.

Because humans, other primates, and guinea pigs do not produce ascorbate endogenously, they have to be supplemented from external sources. Dr. Pauling concluded that the optimum intake of Vitamin C is perhaps 100 times more than the RDA (85 mg). During the last 25 years of his life (he died at age 93 from cancer), Dr Pauling increased his own intake of Vitamin C many times, taking 3,000 mg to 18,000 mg per day. This amount is consistent with the amount of ascorbate in animals that are capable of producing their own on a daily basis. Dr. Pauling believed that cardiovascular disease is the general result of ascorbate deficiency.

**Lp(a) is a simple blood laboratory test to perform. The optimum laboratory level should be under 20 mg/dl and preferably under 14 mg/dl. Currently, there is no medicine or drugs that effectively lowers Lp(a) to this level.**

**A high Lp(a) is genetically linked. The most effective and natural way to normalize it is a nutritional cocktail consisting of high dose Vitamin C ( 4-6 grams), L-lysine (2-4 grams), and L-proline ( 1-2 grams). Other synergistic amino acids such as glutamine, ornithine, and pine bark extract should also be included. Because high dose vitamin C can lead to diarrhea, it is very important to incorporate the fat-soluble form called ascobyl palmitate.** Being fat soluble, this form of vitamin C stays in the body much longer than regular vitamin C and extends the efficacy of vitamin C in the body. At the same time it reduces the amount of vitamin C needed.

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

· Strengthening and healing damaged blood vessels
· Lowering LP(a) blood levels
· Inhibiting the binding of LP(a) molecules on the walls of blood vessels

This concept of endothelial repair advanced by Dr Pauling to lower Lp(a) is simple and logical. **Once the endothelium is healed, the body will not send a signal to the liver to produce cholesterol and its related products such as LDL and Lp(a). The key is to focus on the endothelium and not focus on the liver.**

**Many conventionally trained physicians uses niacin or statin drugs to reduce Lp(a). This works to a**

**limited extent.** Statin drugs have some Lp(a) lowering effects by suppressing its production in the liver, but this is a band-aid approach and comes with side effects. Niacin also reduces the production of Lp(a) in the liver and helps to reduce its blood level . However, this approach has its limitations because until the endothelial wall is optimized and cleared, the Lp(a) level will not be reduced significantly. **The effects of niacin or statin drug therapy usually hit a plateau after 9-12 months of therapy.** The Lp(a) level seldom goes below 30mg/dl because until the endothelium is healed, the body will always instruct the liver to make cholesterol.

On the other hand, with the proper nutritional cocktail focusing on endothelial repair, drastic improvements on Lp(a) level can usually be seen within the same time frame for the majority of people. The higher the starting value, the more significant the reduction.

It is not unusual for the Lp(a) level to be slightly elevated from its baseline level in the early months of therapy ( as it is cleared from the arterial wall into the lumen) before normalizing. This is normal and is not a cause for alarm. A follow up Lp(a) test should be done 9-12 months after starting the nutritional program. While a majority of people respond favorably, some are particularly resistant, and may take up to 1 year to see a minor change. In a very small group or people, no change at all can be expected after an extended period. The good news is that there are no negative side effects. All people with high Lp(a) should be started on a nutritional cocktail program. Even if repeated blood tests do not show any improvement, vascular integrity is enhanced. There is nothing to lose and everything to gain.

## 2. Homocysteine

Homocysteine is an amino acid by-product of food metabolism. It contributes to atherosclerosis, reduces the flexibility of blood vessels, and increases clotting by making platelets stickier and slowing blood flow. Studies show a direct positive correlation between high serum homocysteine levels and the risk of heart attack and stroke.

A high homocysteine level is also associated with Alzheimer's disease, as well as depression, multiple sclerosis, menopausal symptoms, and rheumatoid arthritis.

Homocysteine is formed naturally when protein is broken down. **Too much of it causes oxidative damage to the endothelium.** Oxidative damage is caused by free radicals (byproducts of the body's normal processes that can damage body tissues). In fact, the risk for heart disease triples when the homocysteine blood level exceeds 15.8 umol/L - a reading still considered by many to be within the "normal" range (The optimum target should be under 8 umol/L). Worse yet, the odds of heart disease are directly proportional to the homocysteine concentration. The higher the blood homocysteine level, the higher the risk of cardiac disease.

This direct correlation has been well researched, including a study conducted at the University of Bergen of 2127 men and 2639 women aged 65 to 67 years between 1992 and 1993. By February 1997, 162 men and 97 women had died; 121 from cardiovascular causes (including stroke), 103 from cancer, and 33 from other causes. Using a baseline homocysteine level of 9.0 umol/L the researchers found that for every 5.0 umol/L increment increase in homocysteine levels, all-cause mortality increased by 49%, cardiovascular mortality by 50%, cancer mortality by 26%, and deaths from other causes (respiratory, gastrointestinal and central nervous system diseases) by 104%.

Looking at it another way, **dropping the homocysteine level by 5 points can reduce heart disease risk by 50%.** These percentages refer to values obtained after adjusting for a variety of lifestyle factors including cholesterol level, blood pressure, smoking, body mass index, physical activity, and baseline cardiovascular disease risk, as well as a couple of non-changing factors such as age and gender. About 78% of this study group had homocysteine levels at or above 9.0 umol/L and 12% had levels exceeding 15 umol/L. It is interesting to note that smoking and drinking coffee were associated with higher homocysteine levels while taking vitamins and exercising were associated with lower levels. The result is clear – for optimum heart health, lower the homocysteine level.

In another study published in the Journal of the American College of Cardiology (June 1, 2001;37:1858-1863), researchers found that heart disease patients who took 5 milligrams (mg) of folic acid daily ( not microgram or mcg) for 12 weeks had slightly better functioning of their arterial inner lining, or endothelium, and a greater ability to widen their arteries appropriately, compared to those who took an inactive placebo.

**It is sad to say, but only 11 percent of all Americans get enough folic acid from its main sources -** liver, kidney, broccoli, beef, kale, turnip greens, and beats. Cooking destroys as much as 90 percent of a food's folic acid content. The average American over 50 years old only takes in 130 mcg of folic acid per day. The RDA (Recommended Daily Allowance) is 400 mcg a day. Its level is also depleted by chronic alcohol consumption and medications such as anticonvulsants. In fact, studies have shown that eating 400 mcg of folic acid from food alone does not raise the serum folic acid concentration anywhere close to that obtained by simple folic acid supplementation. You need more than what food can provide.

Drugs easily deplete folic acid as well. The NSAID anti-inflammatory drugs, including aspirin and ibuprofen, deplete folic acid. The popular class of anti-ulcer drugs known as the H-2 receptor antagonists [Zantac, Tagamet, Pepcid, etc.] also depletes folic acid.

Instead of encouraging simple folic acid supplementation, the US Food and Drug Administration implemented a policy of mandating that certain food be "enriched" with folic acid in 1998. Since that time, folic acid has been added to certain grain products including cereals, breads, pastas and flour. This has resulted in higher folic acid levels in adult Americans. Unfortunately, the amount of enrichment, while enough to protect a pregnant women and their fetus from neural tube defect, is hardly enough for optimum health. Only 636 mcg is present per pound of such "enriched food". While some of these foods are good, the majorities fall in to the category of "junk food" because of its high grain and refined sugar content. Clearly, eating such "junk food" as a method to supplement folic acid is not the best way to optimize health.

**There are no medications.**

How much folic acid do you need?

RDA: 400 mcg a day
For heart heath**: 400 mcg 800 mcg a day**
To lower serum homocysteine level: 3-20 mg a day

## 3. Fibrinogen

Fibrinogen is a key indicator in heart disease risk. In one study of 116 men, it was found that people who have high LDL (bad) cholesterol but low fibrinogen levels had only 1/6th the heart attack risk of men with high LDL level and high fibrinogen levels. High fibrinogen levels promote the spontaneous formation of fibrin clots and increase the risk of heart disease. Reducing the level of fibrinogen is therefore an important part of a heart disease prevention program.

A clot is also known as a thrombus. It is formed when platelets and red blood cells come together. It is formed at the sight of the clot from soluble circulating protein called fibrinogen. This protein binds the clots together and is naturally formed in the blood after injury or trauma. The injury could be severe like when a blood vessel breaks. The injury could also be very minor from shear forces and stress of the blood flowing in the blood vessel to free radical attack on the endothelial wall caused by pollutants and sugar. During the aging process, when the collagen structure of the blood vessel wall is weakened, clots may also form. Fibrin also impairs blood flow and increases blood viscosity and pressure. Complete blockage results in heart attack or strokes.

**Laboratory testing of fibrinogen is simple and easy. However, its use has not gained widespread acceptance because there are no direct drug based treatments for elevated levels available.** The normal range is 180-340 mg/dl for males and 190-420 mg/dl for females.

**Plasmin**

While there are over 3000 enzymes in the body and there are more than 20 enzymes involved in the coagulation cascade that creates blood clots, there is only one enzyme that Mother Nature has provided to the human body that can dissolve the fibrin and break up blood clots. This enzyme is called plasmin. Unfortunately, the body's production of this declines with age. In addition to its decreased production with age, fibrinogen levels also rises 25mg/dl per decade in healthy people. In other words, as we age, our plasmin level reduces while our fibrinogen level rises. The resulting risk of cardiac incidents goes up.

Plasmin is called a thrombolytic (clot-dissolving) enzyme and is made from plasminogens through the action of the enzyme called Tissue Plasminogen Activator (TPA). Acting on the same principal, a class of drug has been developed that mimic this activity. For example, Urokinase is a drug that belongs to a class of medication called Tissue Plasminogen Activities. It is administered intravenously within a few hours after admission into a hospital after an acute onset of thrombus formation. It is also very expensive.

Are there natural compounds that have similar thrombolytic activities? Yes. Let us take a closer look.

**Natto**

In 1980, after studying physiological chemistry at the University of Chicago Medical School, Japanese researcher Dr. Hiroyuki Humi discovered that a traditional Japanese food called **natto derived from fermented soy** had the ability to dissolve clots. Specifically, he was able to identify and purify the specific enzyme in the fermented soy cheese that he called nattokinase. **Natto has been widely consumed in Japan as a condiment for over 1000 years.**

Extensive studies have been conducted worldwide on this compound. In one study, 12 volunteers (6 men and 6 women), were fed 200g (7oz.) of natto and had their thrombinolytic activities measured. Researchers found that the time needed to completely dissolve a clot was cut in half in those taking natto as compared to those who did not take it. In 1995, researchers did a study wherein the corona arteries of rats were injured to induce thrombus formation. The arteries were then completely blocked and blood flow to the brain was stopped. Three enzymes (elastase, plasmin, and nattokinase) were then tested on different rats and the researchers found that **nattokinase was successful in restoring circulation by 62%**, while plasmin was only able to restore it by 16% and elastase produced no reopening. Since natto is a natural compound, its potency has to be standardized in order to have relevancy in the studies. In Dr. Sumi's original nattokinase research paper, it was reported that natto has an average of 40 fibrinolytic units (FU).

In human research, 50-200 gram is the typical daily food dose used to supply nattokinase. This is equivalent to 2,000-8,000FUs. The nattokinase currently available in dietary supplementation supplies about 20,000 FU/g. This can be compared with serrpeptase, an enzyme from the silk worm that has fribrinolytic properties with an equivalent of 60,000 FU/g.

**Natto is a fermented cheese-like food and it use as a folk remedy for heart and cardio vascular disease has been well established.** It is produced using a fermentation process by adding a beneficial bacterium known as bacillus-natto to boiled soybeans. The resulting nattokinase enzyme is then produced when the bacillus natto acts on the soybean.

**While soy foods do contain a variety of enzymes, it is only in the natto preparation that contains the specific nattokinase enzyme. Unfermented soy products such as tofu or soymilk do not contain nattokinase.**

Nattokinase produces a prolonged action in two ways: it prevents the coagulation of blood, and it dissolves existing thrombus. Both the efficacy and the prolonged action of nattokinase can be determined by measuring the levels of EFA (euglobulin fibrinolytic activity) and FDP(fibrin degradation product) which become elevated as fibrin is dissolved. It has been shown that by measuring EFA and FDP levels that nattokinase activity can last from 8-12 hours.

Nattokinase has been subjected to 17 studies including 2 small human trials. Nattokinase has also been used to lower blood pressure in Japan. In 1995, researchers from Miyazaki Medical College and Kurashiki University of Science and Arts in Japan studied the effects of nattokinase on blood pressure in both human and animal subjects. With a single administration of 400-450g of nattokinase infused into the peritoneal, there was a 12.7% drop in systolic blood pressure within 2 hours of administration. When the same natto extract was tested on human volunteers, it was shown that when 30g of lyophilized extract, equivalent to 200g of natto food, was given, 4 out of 5 volunteers had their systolic blood pressure reduced by 10.9% and their diastolic blood pressure also reduced by 7%.

To guard against thrombus formation and to dissolve existing clots, **take 25 mg to 100 mg of nattokinase in the form of nutritional supplements if you do not like to consume natto bean. Make sure the FU value is more than 20,000 Fu/g .**

## 4. Arterial Stiffness

One of the hallmarks of aging is the loss of collagen supporting structure throughout the body. Collagen reduction is visible and presents itself in the form of wrinkles on our face and skin surfaces during the aging process. Our blood vessels are also structurally supported by collagen. **As this collagen structure deteriorates, stiffening of the arteries occurs.** Indeed, the fact that arteries stiffen with age, and that such changes are associated with an increased incidence of major cardiovascular events and increases in blood pressure, is now established beyond doubt.

Measuring the stiffness of arteries would logically provide a better insight into blood vessel health, in addition to the traditional blood pressure measurement. Scientists have machines, with a reproducible parameter termed 'stiffness index' by measuring the time delay between direct and reflected waves in the digital volume pulse. There are several apparatuses commercially available to physicians. Unfortunately, its use is not widespread because there is no drug based treatment program to reduce the stiffness once discovered.

**As collagen is lost and elasticity reduced, stiffening of the arterial wall leads to an increase in systolic and diastolic pressure.** In particular, the systolic pressure will be disproportionately higher, registering a reading of 140-160mmHg or higher. There is often a wide systolic to diastolic gap often up to 60-70mmHg (normal is 40 mmHg), with a typical blood pressure reading of 160/100mmHg without medication, and 140/90mmHg at best with medication.

Postural hypotension is also common. With reduced elasticity to normalize blood pressure, it can drop quickly as a one goes from sitting to standing. Anyone over age 45 can practically assume that arterial stiffening is already in a progressive state. Unless active steps are taken, the stiffening will continue. Those who have elevated blood pressure should be especially concerned as it may indicate arterial stiffening. Unfortunately, there are no medications that can reduce the arterial stiffness at this time.

### Nitric Oxide (NO)

In 1998, a trio of scientist's was awarded the Nobel Prize for discovering the enormous role that Nitric Oxide (NO) plays in our body. NO is the first gas discovered to have signaling properties. It is produced by one cell and is able to penetrate through the membrane and regulate the function of another cell. The discovery of this pathway opens up an entirely new principle of signaling and communications in the biological system.

Mention nitric oxide and most will think of the toxic gas produced and given off by car engines. It is a poison that up until now is thought to exist outside the body and does nothing more than cause trouble. NO was not expected to be important in higher animals such as humans. This has been proven wrong. In fact, NO is produced inside most if not all tissues by the body and plays a very important role in the cardio vascular, immune, and nervous systems.

* Nitrous oxide is known as the laughing gas, the anesthetic that is commonly used by dentists. This should

not to be confused with Nitric Oxide. *

**NO and the Cardiovascular System**

NO is produced by the inner most layer of the arteries called the endothelium. Once produced, it rapidly spreads through the cell membrane to the underlying muscle cells, causing them to relax from their default-constricted state. This results in the dilation and widening of the artery lumen. Blood pressure drops as a result. Because NO is short lived, a constant supply of it is generated by the endothelial cells in response to the sheer stress of the blood flow on the artery walls. In arthrosclerosis, the endothelium has been damaged by free radical attacks as well as plaque formation and inflammatory response. **The capacity to produce NO is reduced, and the vascular musculature constricts and blood pressure can be elevated.**

**It is now known that the normal cardio vascular contraction state is biased in one direction towards vessel constriction.** This is the body's way of maintaining the blood pressure at a slightly constricted state in order to channel adequate blood supply and oxygen delivery to the brain continuously. With the constant NO production by the endothelium, vessel dilation is sustained, and blood pressure is maintained at a normal systolic rate of around 120mmHg and a diastolic rate of around 80mmHg. **Too much NO can lead to excessive vasodilatation and a fall in the blood pressure, while too little NO can lead to a rise in blood pressure.**

The vasodilatation effect of NO applies to all blood vessels. It can initiate erection of the penis by dilating the blood vessels to the erectile bodies. This knowledge has already led to the development of new drugs to treat impotency such as Viagra.

Any interruption the production of NO interferes with the tone of the arterial muscles and the blood vessels will return to its constricted state. From this point of view, a rise in blood pressure may be due to the constriction caused by other factors such as the hormone epinephrine produced by the adrenal glands.

**In the case of heart disease the tension is focused on NO deficiency.** Healthy blood vessels are pliable and elastic by nature. They can alter their diameter instantly in response to a greater or lesser out flow of blood from the heart. This continuous change happens during exercise as well as when we are excited. This spontaneous regulation of blood pressure goes on uninterrupted 24 hours a day. As we age, the elasticity of our blood vessels declines due to collagen loss, free radical damage, and plaque accumulation. Poor diet, lack of exercise, cigarette smoking, and genetic predisposition all contribute to a breakdown of collagen fibers that support the blood vessels. This results in the lack of elasticity. Blood vessels then become passive and stiff pipe-like structures which raise blood pressure, forcing the heart to work harder.

In addition to helping the blood vessels relax, NO also helps to prevent the clogging of arteries in several ways. First, it prevents the white blood cells from sticking to the arterial wall. It also helps to prevent damage to the arterial wall by reducing the production of free radicals. In other words, it acts like an antioxidant. NO also helps to prevent the thickening of the middle (muscular) wall of the artery that can narrow the opening where the blood flows.

**Other Functions of NO**

NO gas, when inhaled by patients with pulmonary hypertension has been shown to relieve lung congestion. In a treatment for newborn babies, breathing problems can be helped by inhaling NO that relaxes constricted blood vessels and dilates the lung's blood vessels. NO is also produced in the brain in neuronal form that acts as a chemical messenger at the synapses. This has opened up a new approach to the studies of Alzheimer's disease, Parkinson's disease, and other neurological disorders. NO also inhibits the loss of bone, and the release of growth hormones may augment bone density.

**Exercise and NO**

**Exercise alone has also been shown to increase the production of NO in the body.** This may explain why exercises can reduce blood pressure.

The effect of adding the amino acid arginine and vitamins C and E to an exercise program have been shown to synergistically increase NO production. In a study conducted at UCLA, researcher Louis Ignarro studied 6 groups of 8-week-old receptor deficient male mice with high cholesterol over an 18 week period. The mice were randomly divided into 3 dietary groups called: fat with high cholesterol diet alone, fat with high cholesterol diet with antioxidant vitamin E and C, and a fat with high cholesterol with the antioxidants arginine. It was shown that the mice from all 3 groups were able to lose weight and had lower cholesterol when they exercised. The atherosclerotic legions were significantly reduced in the mice group that had arginine.

The explanation is that exercise will increase both the amount of endothelial nitric oxide synthetase (NOS) and the enzymes that will then convert the arginine into NO, which in turn lowers abnormally elevated blood pressure, prevent unwanted blood clots and early inflammation associated with coronary artery disease. Nitric oxide production is stabilized when vitamins C and E are added as these remove destructive oxidants from the blood stream.

Even without exercise, these supplements will work on their own to increase NO. Studies have shown that mice that were sedentary and fed supplements alone showed a 40% reduction in atherosclerotic legions compared to mice that were on a regular, high cholesterol diet but did not exercise or take supplements.

Exercise alone without supplementation also showed a 35% reduction in legions. Therefore, it can be concluded that amino acid supplementation has an atherosclerotic reduction effect similar to exercise. Doing both exercise and supplementing with antioxidants concurrently will produce the best results.

**Formation of NO**

NO is formed in various places in the body. In the endothelium, NO is formed by the enzymatic action of nitric oxide synthetase (NOS) on the amino acid arginine and citrulline. This process is enhanced when antioxidants are present, especially vitamin C. NO also forms in nerve cells, where it spreads rapidly in all directions and affects all cells in the vicinity. NO is also produced in white blood cells such as macrophages and NO is toxic to invading bacteria and parasites.

There are 3 forms of NOS enzymes. There is one in the endothelium, one in the immune system, and one in the brain. Genes responsible for encoding the NOS are located in chromosomes 12, 7, and 17 respectively. The discovery of NOS opens up another new class of drugs based on n-monomethyl-arginine (l-nmma), an inhibitor of the NOS enzyme. Drugs are being used to explore the possibility of blocking NO production in order to raise blood pressure. Experiments have been performed where volunteers were injected with l-nmma. Blood flow was then compared from one arm to the other arm. As l-nmma was infused, blood flow is observed to gradually decrease to half compared to that in the control arm. This has important ramifications, and drugs are being developed to raise blood pressure. Clinical application of this pathway is particularly useful for those who have acute low blood pressure as frequently experienced when in shock or trauma.

**L-Arginine and NO**

**L-arginine is an essential amino acid that is present in many foods and it is also a precursor of NO production.**

**Studies have shown that arginine, when taken in proper amounts, can stimulate NO production.** In a 1999 study, 30 impotent men were given 1500 mg of arginine each per day. It was shown that it worked no better than the placebo in terms of vasodilatation and sexual performance. However, when 21 men with mild to moderate impotence were given 3,000mg of arginine a day, significant improvement in erections as well as sexual satisfaction were reported. This study was published in the December 1998 issue of Hawaii Medical Journal. It is obvious that the use of arginine as a nitric oxide precursor is dose dependant, and a low dose regiment will not be effective.

L-arginine supplementation has also been shown to significantly reduce systolic and diastolic blood pressure. Reductions were evident in subjects when they were rested as well as when they were not stressed. The

reduction in blood pressure was associated with increased cardiac output. These findings were reported in the in the American Heart Association meeting in November 2003 where 16 hypercholestrolemic men with normal blood pressure were given 12 grams of oral arginine a day over a period of 3 weeks.

L-arginine has long been used in the enhancement of sports performance and cardiac function. A double blind placebo controlled study of 22 subjects with stable angina and supplementation with l-arginine at 1 gram twice a day has been shown to significantly improve their exercise capacity. Arginine supplementation has also been reported to result in a 70% reduction in angina attacks in another study.

L-arginine works by stimulating the production and release of NO. However, L-arginine may have separate anti-atherogenic independent in of its role in the enzymatic formation of NO. For example, l-arginine itself may have antioxidant activity. It has been shown to inhibit the oxidation of unoxidized low density lipoprotein (LDL) to oxidized LDL (ox-LDL). The oxidation of LDL to ox-LDL is believed to be a critical early step in the formation of arthrosclerosis.

L-arginine may also independently have a scavenger effect in sweeping up super oxide anions and hydrogen peroxide as well as reducing the peroxidation of lipid. Furthermore, it has been shown to have immunomodulatory activities. Supplementation of this amino acid in breast cancer has been shown to increase the quantity and cytotoxicity of natural killers (NK) and Lymphokine-activated-killer (LAK) cells. The exact mechanism is not clear but it has been shown that l-arginine is a precursor in the synthesis of tetrapeptide tuftsiin, which itself appears to have immunomodulartory activities.

Arginine is an excellent helper when it comes to wound repair. This may be due to its precursor role in the formation of l-ornthine, and ultimately l-proline. L-proline in conjunction with l-lysine and vitamin C are the key elements in collagen biosynthesis. Collagen is the main ingredient in tissue healing and scar tissue formation. Arginine participates in the maintenance of muscle and lean tissue in the body.

Arginine, in high dose, promotes an increase in the body's production of insulin like growth factor (a measure of human growth hormone). Its use, together with lysine, ornithine and glutamine, is one way to stimulate the body's release of growth hormones.

Interestingly, l-arginine has also been shown to increase sperm counts. In one early study, 178 men with oligospermia, of which 93 were diagnosed with severe oligospermia, were all given 4g of l-arginine daily. A 100% increase in sperm count was found in 42 cases resulting in 15 pregnancies. Studies have also shown that l-arginine is beneficial for people with kidney diseases as well as interstitial cystitis. It also improves kidney function in diabetic animal models and it helps promote renal vasodilatation.

In summary, arginine is a very versatile amino acid. Many of its functions are just starting to be explored. **NO produced in the body from the intake of arginine can play a major role in anti-atherogenic activity.** NO inhibits mononuclear cell adhesion, platelet aggregation, proliferation of vascular smooth muscle, and production of some reactive oxygen species, such as super oxide anions. It is a promoter of endothelium dependant dilation and is able to normalize high blood pressure. In other words, it relaxes the blood vessel and reduces the arterial stiffness. It also increases sperm count, boosts immune function, enhances male sexual disorders, restores protein balance, and speeds the healing of wounds.

**Arginine Dosage**

**Arginine is a non-toxic compound. Dosage of up to 15 grams a day has been well tolerated.** The most common adverse reaction to high doses (15-30 grams a day) are nausea, abdominal cramps, and diarrhea. Scaling back the dosage will eliminate the problem. Because high dose and long term use of arginine can lead to an increase in growth hormones, therefore, pregnant and nursing mothers should refrain from high doses of arginine supplementation. The use of arginine in the cardio vascular and erectile dysfunction settings has been very promising. While no supplementation can work 100% of the time, most people do experience some improvement when dosed properly. **For cardio vascular health doses, 2-15 grams a day should be used in divided doses.** To help sperm count, doses of 10-20 grams a day have been used. For erectile dysfunction, daily doses of 5 grams a day have been used. For interstitial cystitis, 1 to 4 grams a day is

commonly used.

**To avoid arginine's risk of promoting free radical oxidation, supplementation should always be accompanied by antioxidants including vitamin C, ascobyl palmitate, lysine, proline, a small amount of co-enzyme Q10, lipoic acid, and other antioxidants.** This is especially important for those with inflammatory problems such as arthritis as excess NO can stimulate an inflammatory response. If the immune enhancing properties of arginine are desired, always add proline and lysine. Because some infectious pathogens may actually use arginine as a fuel, lysine should be added to help neutralize any virus attacks. Children under 18 should not take arginine for any extended period of time.

Anyone concerned with cardiovascular health, and especially with normalization of blood pressure, should consider nutritional supplementation of arginine in conjunction with other synergistic and pre-cautionary co-factors mentioned above. Arginine dosage ranges from 2-5 grams a day. **Those who have a history of low blood pressure should be careful as NO may further lower the pressure.**

## 5. Cellular Energy Metbaolism

Mitochondria are the energy factories of the cell. The energy currency they produce is ATP. Generation of ATP is therefore vital to cellular processes. Coenzyme Q10, or ubiquinone, is a vital component in the ATP-generating process. It acts as an electron acceptor/proton donor; hence its presence in the body is fundamental to the support of cellular life. It is omnipresent in body tissues.

With advance technology, the cellular metabolism rate can now be measured. Unfortunately, its commercial use is not wide spread because laboratory tests are very expensive. Fortunately, there is already enough scientific research that recommends us to do all we can to enhance cellular energy generation regardless. When more energy can be generated by the heart with the same fuel, the heart does not have to work as hard. In laymen's term, you don't need to be a mechanin to know that regular use of premium grade gasoline can help to have a cleaner and more efficient engine.

The following are proven nutrients that promote cellular metabolism and should be taken by everyone concerned with heart health.

**Coenzyme Q10 (Ubiquinone)**

The body's production of CoQ10 begins to decline after age 20 to just 50% of levels by age 70. Because the function of the heart is so dependent on the energy produced with the help of CoQ10, CoQ10 is extremely important for heart health. It is also a powerful antioxidant and membrane stabilizer. The range of heart conditions for which research has found CoQ10 beneficial include (1) congestive heart failure, (2) cardiomyopathies, (3) arrhythmias, (4) angina, a lack of oxygen, and (5) muscular dystrophy.

Individuals with cardiac disorders have been identified as having abnormally low levels of CoQ10. Numerous long-term studies have been conducted to ascertain the efficacy of CoQ10. These studies indicate that there is a statistically significant improvement in the condition of those patients with myocardial dysfunctions such as ischemic cardio-myopathy or congestive heart failure when they take CoQ10. In an 8-year study of 424 patients with cardiac dysfunction, 58% improved by one functional class, 28% by two classes, and 1.2% by three classes. Further, overall medication requirements dropped, with 43% of the patients discontinuing between one and three drugs. Only 6% were required to add one drug. In another study on 40 patients undergoing elective coronary artery bypass surgery and pretreatment with CoQ10 at 150mg/day for seven days served as a protection against oxidative compounds.

CoQ10 also plays a vital role as an antioxidant in cellular membranes and plasma lipoproteins. It is present in all plasma membranes and in LDL-cholesterols. Studies illustrate CoQ10's protective action against the oxidative modification that makes LDL-cholesterol atherogenic. In its reduced form, ubiquinol, CoQ10 also functions as a chain-breaking antioxidant and is believed to regenerate Vitamin E.

You can get CoQ10 from your diet, although the amount from food intake is insubstantial. For example, one pound of sardines or 2.5 pounds of peanuts only provide 30 mg of CoQ10.

Working synergistically with CoQ10 are two endogenous antioxidants that enhance mitochondrial function and reduce free radical damage - L-Carnitine and Lipoic Acid.

**L-Carnitine and Lipoic Acid**

The ability of the cell to utilize fatty acids as a source of fuel is essential for optimizing the production of ATP by mitochondria in cardiac cells to keep the heart functioning properly. L-carnitine assists in this transportation process by bringing fatty acids from the extra-cellular space into the mitochondria. In one double blind trial, 500mg per day of a modified form of carnitine called propionyl-L-carnitine lead to a 26% increase in exercise capacity after six months.

Lipoic Acid is both a water-soluble and fat-soluble antioxidant. It neutralizes free radicals in both the fatty and watery regions of cells. This is in contrast to Vitamin C, which is water soluble, and Vitamin E, which is fat soluble. Lipoic acid is therefore called the "universal antioxidant". It has the ability to recycle both Vitamin C and E in our body. It helps break down sugars so that energy can be produced from them through cellular respiration. In addition to serving as the bulb of the body's antioxidant network, lipoic acid is the only antioxidant that can boost the level of intracellular glutathione, a cellular antioxidant of tremendous importance. Glutathione is a water-soluble antioxidant and is essential for the optimum functioning of the immune system.

**Nutritional Supplement Consideration:**
**Coenzyme Q10: 30-300 mg (less is needed if synergistic agents are added such as peperine extract that can enhance CoQ10 activities by up to 25%)**
**L-Carnitine: 300-2,000mg**
**Lipoic Acid: 75-300mg**

## 6. CRP

C-reactive protein (CRP) is a protein released into the bloodstream any time there is active inflammation in the body, such as infections and arthritis. CRP is conventionally regarded as the first-line of defense of the immune system against invading pathogens by eliminating them through the inflammatory response.

However, recent studies have shown that CRP is much more than that. In a study published in the New England Journal of Medicine, researchers analyzed over 20,000 blood samples taken from women enrolled in the Women's Health Study, a long-term study that enrolled and followed apparently healthy women for a number of years. It was found that an elevated blood level of CRP is strongly predictive of future cardiovascular events such as heart attack and stroke. In other words, CRP is an independent marker of cardiovascular risk, and may be a partial explanation for why some patients develop significant coronary artery disease despite normal cholesterol levels. In this study, women with low CRP and low cholesterol have the lowest risk, while those with high CRP and high cholesterol had very high risk. Women with either high CRP or high cholesterol also had elevated risk. Interestingly, those with high CRP but normal cholesterol apparently had a higher risk than those with normal CRP and high LDL cholesterol. CRP is a predictor of future atherosclerotic events.

CRP binds to LDL in the artery wall, creating an "oxidized LDL" that is thought to be the cause of inflammation. The inflammation process attracts macrophages. These macrophages then become "foam cells", initiating a cascade of events leading to the generation of atherosclerotic plaques.

CRP therefore is tied into cardiovascular risk by at least two distinctive pathways. The importance of CRP as an advanced-screening tool of cardiovascular risk cannot be ignored. In fact, it may be just as important as elevated LDL cholesterol levels. Without measuring CRP level, many high-risk patients would be missed.

Fortunately, **CRP is an easy and inexpensive blood test to perform. The normal value is under 1mg/dl. There are no drugs or medications that can definitively reduce CRP levels.** There is suggestive evidence that both aspirin and statin drugs can reduce CRP levels to a certain degree. However, there are side effects accompanying the use of these drugs. Certain lifestyle changes can also lead to a reduction in CRP levels such as quitting smoking, limiting sugar intake (which would lessen the likelihood of metabolic syndrome), and practicing good dental hygiene to help prevent periodontal disease (gum disease).

**Fortunately, taking nutrients with anti-inflammatory properties such as molecularly distilled fish oil high in omega-3 will help, in combination with compounds such as bromalin, curcumin, cat's claw, olive leaf, and fibrin dissolving nutrients such as natto.**

## A. Omega-3 Fatty Acid

Omega-3 fatty acids provide a range of benefits and protection for the heart and body. In addition to reducing the risk of heart disease, they also help prevent blood clotting, heart attacks, and irregular heartbeats which all could lead to sudden cardiac death. They are anti-inflammatory, and inflammation is a key initiator of the atherosclerotic cascade leading to plaque formation and sudden death. Omega-3 also has anti-cancer functions, as we shall see.

Omega-3 fatty acids can be divided into 3 main categories -- Eicosapentaenoic Acids (EPA), Docosahexaenoic Acids (DHA) and Alpha-Linolenic Acids; out of which EPA and DHA have the most beneficial effects. EPA and DHA are found mainly in fish oils while Alpha-Linolenic Acids are usually derived from plant sources such as soybeans, canola, walnut and flaxseed.

Of all the fatty acids in the blood including saturated, monounsaturated, and polyunsaturated, only the percentage of long chain omega-3 predicted fewer sudden deaths. In a study of 11,323 recent survivors of heart attack, 1 gram of omega-3 or 300mg of Vitamin E or both was given. The usual pharmacological regiment and lifestyle recommendations were made. It was shown that omega-3 and not Vitamin E improved survival. After 3 months of remaining on regiment of omega-3, patterns showed a 41% decrease in mortality, a 53% reduction in sudden death after 4 months, and a 30% decrease in cardiovascular mortality after 12 months. There was also a 5% decrease in triglyceride but not total cholesterol, HDL, or LDL cholesterol.

Increasing the intake of EPA and DHA will lead to an increase of omega-3 fatty acids in tissue or cellular lipids and circulatory lipids. At the same time, it will reduce the omega-6 fatty acids such as LA and Arachidonic Acid (AA), which is not beneficial to our bodies.

These fatty acid shifts are particularly pronounced in the cell membrane-bound phospholipid components. Cell membranes and their functioning, for example, improved with reduced inflammatory response. There is also reduced platelet aggregation and enhanced blood flow. The vasodilatory effect will increase the lumen size of vascular system. Studies have shown that fish oil concentrates that provide EPA and DHA at intakes of up to 2-4grams a day, when taken over a few weeks, can lower various risk factors for heart disease. These effects include an anti-thrombotic effect, lipid (triglyceride) lowering, reduced blood and plasma viscosity, and improvements in endothelial dysfunction.

Omega-3 fatty acids accumulate to a considerable extent in various sites including circulating blood platelets and the heart and serum phospholipids. The accumulation of EPA and DHA in platelets leads to a decrease in platelet adhesiveness, aggregation, and an overall reduction in thrombogenicity. Antiatherogenic effects of omega-3 fatty acids have also been shown in animal studies with similar results. Eicosanoid formations are also influenced positively. The eicosanoids formed via oxygenase enzymes acting on AA and EPA includes prostaglandins, leukotrienes and thromboxanes. Both eicosanoid-dependent and eicosanoid-independent processes mediate the benefits of omega-3 fatty acids on cardiovascular disease. For example, the reduced blood platelet reactivity (antithrombotic effect) with increased EPA and DHA intakes involve the reduced formation of the proaggregatory eicosanoid known as thromboxane A2 (TxA2).

## B. Curcumin

Curcumin comes from turmeric root and is an ancient spice within the ginger family that is widely used in cooking. Its use dates back to the time of Egyptian pharaohs more than 6,000 years ago. A tall, stemless, perennial plant cultivated throughout the tropics, turmeric is what gives curry its unique color and flavor.

In addition to its kitchen uses, curcumin has been used by traditional medicine for wide variety of ailments including liver disease indigestion, urinary tract diseases, inflamed joints, insect bites, and dermatological disorders. Although the chemical structure of curcumin was discovered in 1910, it was only during the mid 1970s that the potential uses of curcuminoids in medicine began to be extensively studied. It has been shown that curcumin has both strong anti-oxidant and anti-inflammatory properties. Its anti-inflammatory property helped bring curcumin into the forefront of heart disease prevention supplements.

Inflammation results from a complex cascade of chemical reactions in a series of actions triggered by the body's response to tissue damage. This damage may be caused by physical traumas including various diseases and surgeries. It can also come from chronic minute free radical damage to endothelial walls over time. Curcuminoids prevent the synthesis of several inflammatory prostaglandins and leukotrienes. Curcuminoids inhibit several enzymes that participate in the production of inflammatory metabolites in the body. **The natural anti-inflammatory activity of curcuminoids is comparable in strength to steroidal drugs as well as nonsteroidal anti-inflammatory drugs as indomethacin and phenylbutazone, which have dangerous side effects.**

In a double blind controlled study, the patients were split up into three groups. The first group received curcumin (400 mg);the second group received the anti-inflammatory prescription drug phenylbutazone (100 mg); the third group was placed on a placebo. Each group took their given doses three times daily for five consecutive days after surgery for either a hernia condition or an accumulation of fluid in the scrotum. The results showed that curcumin was just as effective as phenylbutazone in reducing post-operative inflammation.

Inflammation is known to be associated with increased levels of lipid peroxides and free radicals, which are generated by the liver as well as by inflamed tissues in the body. Animals fed curcumin showed decreased levels of lipid peroxides and subsequent reduction in the processes of inflammation. In one study, curcumin was shown to be eight times more powerful than vitamin E in preventing lipid peroxidation. With decreasing oxidation of the endothelium, more nitric oxide is produced and the arterial stiffness is lessened.

Curcumin has a similar anti-inflammatory action to aspirin. However, unlike aspirin curcumin inhibits the production of inflammatory prostaglandins. It does not affect the synthesis of prostacyclin, an important factor in preventing vascular thrombosis. Compared to drugs, curcumin may therefore be preferable for patients who are prone to vascular thrombosis and require anti-inflammatory and/or anti-arthritic therapy.

**Dosage: 50-200 mg.** Since curcumin also lowers cholesterol levels by increasing the flow of bile out of the liver, those with biliary tract obstruction should not use curcumin. **Always take curcumin with food.**

## C. Bromelain

Discovered in 1957, bromelain is the name of a group of protein-digesting, or proteolytic enzymes that are found in the pineapple plant. Depending on the source it is usually distinguished as either the fruit bromelain or stem bromelain. All commercially available bromelain comes from the stem. Bromelain is a natural blood thinner and an anti-inflammatory agent. It works by breaking down fibrin, a blood clotting protein that can prevent healthy circulation and tissues from draining properly. Bromelain also blocks the production of compounds that cause pain and swelling.

Bromelain, when taken orally, is absorbed through the gastro-intestinal tract, with up to 40% absorption. Because it comes from a natural source, a variety of destinations have been used to indicate the potency and activity of this compound. Research studies vary in destinations utilized. The most common unit includes

RORER units (RU), gelatin dissolving units (GDU), and milk clotting units (MCU). One gram of bromelain standardized to 2000MCU is the equivalent of 1g of 200GDU of activity or 8g of 100,000RU of activity. Bromelain's cardio benefit properties were first discovered in 1972. It was found that it has the ability to prevent aggregation of blood platelets. In a study, bromelain was administered to 20 volunteers with a history of heart attack or stroke and who had high platelet aggregation values. Bromelain was shown to decrease blood aggregation in 17 of the subjects and normalize values in 8 of the 9 subjects whom previously had high aggregation values.

Bromelain is an effective fibrinolytic agent. In high doses, there is a corresponding reduction in the serum fibrinogen level shown in rats, with both prothrombin time (PT) and activated partial thromboplastin time (APTT) markedly prolonged. With the presence of bromelain, the conversion of plasminogens to plasma is enhanced. The spread of the coagulation process is limited due to fibrin degradation. In addition to the platelet pathway, bromelain also has direct as well as indirect actions involving other enzyme systems and exerts its anti-inflammatory effects. Experimental studies using bromelain have shown its ability to suppress inflammation is similar to that of prednisone. This is due to its ability to selectively modulate the biosynthesis of thromboxanes and prostacyclin. These 2 groups of prostaglandins with opposing actions ultimately influence the activation of cyclic-3,5-adenosine (cAMP), an important cell growth modulating compound.

**Dosage: 1,000 to 6,000 mg with potency of 3,000 GDU/gram.**

## 7. Triglycerides

Of the four commonly measured lipid markers (total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides)**, triglycerides are the most underappreciated and perhaps the most important.** The reason – we don't know enough about triglyceride metabolism within the body.

Triglycerides are etherified fatty oils that form the core of chylomicrons and VLDL cholesterol. Triglycerides and cholesterol both measure the total amount of lipoproteins in the serum. The associated cardiovascular disease risk prediction offered by triglycerides and cholesterol by themselves is 44%. When coupled with low Vitamin A and E, with the ratio of (cholesterol + triglycerides)/ (Vitamin A and Vitamin E), the risk predictive power goes up to 85% accuracy.

Diets high in saturated fats, such as red meats as well as diets high in simple carbohydrates and starchy foods (such as sugar, rice, and wheat) raise serum triglyceride drastically. Only 20% of the ingested sugar load can be burned or stored as glycogen at any one meal. The remaining 80% will be stored as fat deposits or converted to triglycerides that can contribute to the buildup of acidity.

Elevated blood levels of triglycerides, but not cholesterol, have been associated with an impaired fibrinolytic system, leading to atherothrombotic stroke and transient ischemic attacks. It is a powerful predictor of myocardial infarction (tissue death).

The role of triglycerides is now only recently being studied in depth. It is clear that triglycerides are the key link that connects carbohydrates to obesity, and not dietary fats or dietary cholesterol. The dominant cause of high triglycerides is high carbohydrates and not fats. In other words**, a high triglyceride level is almost synonymous to a high carbohydrate diet and not a high fat diet.**

Because triglyceride elevation is almost universally related to dietary intake of sugar (including grains), high triglycerides are one of the most easy and straightforward problems to correct with proper diet alone. The decline is dramatic and in a matter of weeks if the proper low glycemic, low grain anti-aging diet is followed.
While a normal triglyceride level can be up to 160mg/dl, the appropriate goal for anyone serious about optimum health should target the triglyceride to be no higher than 100 mg/dl. A triglyceride count of 100 or more increases the relative risk of a new cardiovascular event by 50% and

reduces the chances of surviving a subsequent heart attack. **Medications are available to lower triglyceride levels, but they are seldom necessary as long as a strict no grain diet is adhered to.**

**Start with eliminating all grain products from the evening meal**. This includes wheat, rye, barley, potato, bread, and rice. It is usually difficult in the beginning and carbohydrate cravings may be experienced. This is quite common because the body is already addicted after years of taking in grains. If this happens, cut back by only 30% for 60 days and allow your body to have a transition. If you feel hungry 1-2 hours after a meal, eat a handful of raw nuts such as almonds or walnuts that have been presoaked in room temperature water for at least 6 hours.

As the body slowly gets used to the reduced grain intake at dinner, also reduce grain intake at lunch. Substitute with more above the ground vegetables,unroasted nuts, and eggs (raw are best, but if you need to cook them, try not to cook the yolk too ). Oils are acceptable as long they have not been exposed to high heat. Use extra virgin olive oil for salads and light stir frying, butter for high heat frying, and coconut oil for deep-frying (which should be kept to a minimum). As usual, no desserts after dinner, and reduce snacks before bedtime. All refined carbohydrates such as cookies, ice cream, and chips should be avoided. Follow the above, and the triglyceride level will come down drastically in a matter of weeks. **As the triglycerides normalize, the total cholesterol will reduce automatically, and the total cholesterol to HDL cholesterol ratio will automatically improve.**

For those unable to follow a no grain diet, taking a natural compound called panthethine at 600-1200 mg a day will effectively lower triglycerides without any side effects. Other nutritional supplementation that can help lowering triglyceride includes L-carnitine(500-3,000mg), chromium polynicotinate(400 to 1,200 mcg), venadyl sulfate(15-30mg), EPA/DHA(500 –5,000 mg)

## 8. Total cholesterol / HDL Cholesterol ratio.

Cholesterol is a key macronutrient the body cannot do without. It is a precursor to all the steroid hormones in our body including pregnenolone, DHEA, estrogen, progesterone, testosterone, and cortisol. A too low total cholesterol level (under 150mg/dl) has been associated with cancer and brain function impairment. The ideal total cholesterol level should be around 200 mg/dl.

HDL is the “good” cholesterol. It carries“bad” LDL and oxidized-LDL cholesterol from the blood stream back to the liver. The higher the HDL level the better. It is best to have an HDL level over 45 mg/dl, and anything under 30 mg/dl is considered a risk factor. Taking nutrients such as fish oil can increase HDL. Exercise has shown to increase HDL as well. It is not unusual for those in good health to have an HDL level of close to 100 mg/dl.
Total cholesterol alone is a rudimentary tool in cardiovascular health predictive value and HDL levels themselves are also a reasonable marker. **Taken together as a ratio their predictive value increases significantly.** Accompanied with high HDL cholesterol and resulting in a low total cholesterol to HDL cholesterol ratio of less than 3.5, a total cholesterol of over 200 mg/dl requires no therapeutic intervention at all.

**The ideal Total cholesterol / HDL cholesterol ratio should be 3.5 or less, and preferably under 2.5.**

## 9.LDL cholesterol

Low-density lipoprotein (LDL) is the major cholesterol carrier in the blood. If too much LDL cholesterol circulates in the blood, it can get oxidized. It is the oxidized form of this that triggers a series of inflammatory reaction in the blood stream, providing a trigger for heart attack and stroke. It is therefore also called the “bad” cholesterol for a good reason. Oxidized LDL slowly builds up in the walls of the arteries feeding the heart and brain. This with other substances can form plaque.

**A high level of LDL cholesterol (160 mg/dl and above) reflects an increased risk of heart disease. If**

**you have heart disease, your LDL cholesterol should be less than 100 mg/dl.**

One would think that its measurement in the blood should be highly complex. In reality, LDL is not even measured in the traditional lipid panel blood test. Out of the five traditional markers reported in the lipid panel, **LDL is the only marker that is a calculated number and not a measured number.**

Here is the formula:

**LDL cholesterol = total cholesterol – HDL cholesterol – (Triglyceride /5).**

You can accurately calculate the LDL cholesterol level as long as the total cholesterol, HDL cholesterol and triglyceride level are available. However, **if the triglyceride level exceeds 350 mg/dl, the total LDL level will not be accurate** based on the calculation and therefore cannot be relied upon. In this case, the actual measured LDL level should be obtained from the laboratory.

The single focus on LDL lowering has been a pharmaceutical industry darling for the past 20 years, and for good reasons. Worldwide sales of these drugs continue to climb at a record pace. **There is little doubt that drugs can reduce LDL cholesterol aggressively.** These drugs are the synthetically derived HMG-CoA reductase inhibitors such as Iovastatin, pravastatin, and simvastatin. They are collectively called "statin" drugs. By inhibiting the production of HMG-CoA reductase, cholesterol production in the liver is reduced. **Based on the latest "scientific" recommendation to bring down the blood LDL cholesterol level to 70 mg/dl, 40 million Americans will qualify to enter this drug based cholesterol lowering program. In America alone, over 40 million prescriptions are written yearly for cholesterol lowering medications. It is estimated that in the coming years, 50% of American adults will be on these serious drugs.**

**While statin drugs are effective in lowering LDL cholesterol, they have serious side effects.** In fact, in August 2001 German Pharmaceutical giant Bayer AG withdrew the cholesterol-lowering statin drug Baycol from the market because it was linked to 31 deaths. Moreover, these deaths occurred at the manufacturer's recommended initial dose (0.4 mg/day) and at the highest dose (0.8 mg/day). The majority of deaths occurred in elderly patients and more often in women. Statin drugs can cause severe muscle weakness and pain even at low doses. Using the proper dosage is clearly an important if not critical part any drug based lipid-lowering program.

**Recent studies have also shown that high dose (80 mg) of a popular statin drug called Zocor does no better than a lower dose (40mg) in the prevention of heart attack in high risk patients.**

There are other statin drugs on the market, such as Lipitor. Like Baycol, these drugs are linked to the same rare muscle weakness, known as myositis, which occurs in about 1 in 1,000 statin users. Myositis occasionally progresses to rhabdomyosis -- a complete breakdown of muscle cells that can lead to kidney failure and death. Statin drugs also cause cognitive impairment and memory loss. It has been well known that **these drugs routinely cause cancer in laboratory animals**. Some experts believe that pravastatin (Pravachol) and fluvastatin (Lescol) may have less potential for these deadly drug interactions. The data at this time is not sufficient to declare one statin drug safer or more dangerous than the others. It will be years before we know the full side effects of statin drugs.

**Statin drugs also inhibit the intrinsic biosynthesis of Coenzyme Q10 (CoQ10)**, a central compound in the mitochondrial respiratory chain. CoQ10 is indispensable for optimum cardiac function. Reduction of CoQ10 constitutes new risk of cardiac disease, especially for those whose cardiac function is already compromised, such as those with congestive heart failure or cardiomyopathy.

While cholesterol-lowering drugs may lead to fewer heart attacks, the mechanism of action may not be related to a lowered blood cholesterol level only. **Statin drugs have been shown to reduce inflammatory response in the endothelium. It may well be that reduction accounts for the cardiac benefit effect**. The suppression of cholesterol manufacturing in the liver leading to cholesterol lowering levels may be a less important and secondary benefit. There is also a desirable effect of raising nitric oxide levels. **It is interesting**

**to note that there are natural compounds that have anti-inflammatory and nitric oxide level raising properties without side effects.**

**The optimum level of LDL is under 100 mg/dl, and over 160 mg/dl is considered high. While LDL does have predictive value in terms of cardiovascular disease risk, it should be viewed as part of an overall picture and not a stand-alone key indicator. This has not been the case, sad to say.**

**It is important to note that as the endothelium heals, the LDL level will naturally normalized without the use of drugs.** Because endothelium healing takes some time, **an immediate drop in LDL level will not and should not be expected.** For those requiring immediate normalization without drugs, the following should be considered: : panthethine(300-900 mg), panthothenic acid(600-1,500 mg), chromium polynicotinate(300 to 600 mcg), ascorbic acid(1000 to 3000 mg), guccolipid(50-200 mg) and polycosinol (5-20 mg).

## 10.Total cholesterol

Thanks to mass-market commercialization, total cholesterol testing is now easily and widely available. A simple pinprick and a drop of blood on a test strip can offer results in a matter of minutes.

It is important to note that the total cholesterol is reported based on the following formula in the laboratory:

**Total cholesterol = HDL cholesterol + LDL Cholesterol + (triglyceride/5).**

Looking at the formula, **one can easily see that if LDL, HDL or triglyceride is high, then the total cholesterol level has to be high.**

**If the total cholesterol is high and is due to high HDL cholesterol, there is no cause for alarm.** Any attempt to lower total cholesterol in such case is unwarranted. **HDL cholesterol should be as high as possible.**

**If the total cholesterol level is high primarily due to a high LDL or triglyceride level, then a cholesterol-lowering program should be considered. However, the therapeutic pathway to lowering LDL (with statin drugs or nutritional supplementation) is different from that of triglyceride lowering (by diet, drugs, and nutritional supplements). It is imperative that a critical distinction be made to determine if the root cause of the high cholesterol is due to high LDL or high triglycerides prior to initiation of therapeutic measures.**

**Specifically, if the high total cholesterol is due to a high triglyceride level, then a no grain dietary approach is best. Using drugs to normalize triglycerides without dietary change is a band-aid approach. A no grain diet will universally lower the triglyceride level unless it is a familial condition. Many well intentioned but misguided physicians embark on a program of cholesterol reduction only to find failure at the end of the tunnel.** Patients are subjected to ever-higher doses of statin drugs unnecessarily when all that is needed are simple dietary changes if the main cause of high cholesterol is due to triglyceride overload.

**Traditionally, a total cholesterol value of less than 200mg/dl is considered desirable, while the value of over240 mg/dl is considered high.** By now it should be obvious that **simply looking at the total cholesterol alone without considering HDL, LDL, or triglycerides will not give a true picture and is obviously incomplete. In this respect, it can be seen that the total cholesterol number on its own is of little significant clinical value.**

## Summary

Modern science has ushered in a series of advanced markers of cardiovascular health that was not available just 20 years ago. The traditional dependency on cholesterol as the key marker needs to be downgraded. **Far**

**more sensitive markers including Lp(a), homocysteine, C reactive protein, arterial stiffness, and fibrinogen levels are easily obtained, and reference ranges have been well established. Currently, there are no effective drug base programs to normalize these markers, and the use of these markers is therefore not widespread.**

**The use of natural nutritional supplementation to normalize these markers has been well studied and their effectiveness is not in doubt. They should represent the first line defense for those who are at risk or have damaged cardiovascular system. O**ptimization with a complete nutritional program focused on the heart will not only reduce risk, but in many cases can reverse existing damage without the side effects often seen with medications.

**For optimum heart disease prevention, the following basic comprehensive nutritional cocktail should be considered and taken on a daily basis.** No one nutrient is more important than others because each has a part to play and is important is its own right. Do not simply pick and choose.

**The advantage of having a blended nutritional cocktail is that a much lower dose of each nutrient is required due to their combined synergistic effect without sacrificing therapeutic efficacy.** At the same time, all the key cardiovascular pathway markers are covered. Because endothelium healing takes time, patience is required. **While some people notice a significant improvement in heart health in as little as a few weeks, expect 3 to 6 months for cellular nutrition to do its work is best. The key is to take the entire cocktail blend in proper dosage for a long enough time to allow the body to heal itself.** Because each person is different and the degree of existing damage varies, be prepared to allow up to 6 –12 months in selected cases. The key isto apply the right dose, and consulting a health care professional experienced in this area is highly recommended.

**Alpha Lipoic Acid –75 mg**
**Coenzyme Q10 –10 mg ( as long as enhancing agents such as peperine are included)**
**Curcumin – 20 mg**
**Folic Acid – 150 mcg**
**Fish Oil – 500 mg**
**Bromelain - 1000 mg (3,000 GDU /gram)**
**Citrus Bioflavonoids - 30 –100 mg**
**Nattokinase – 25 mg (20,000 FU/gram)**
**Magnesium - 90 mg**
**L-arginine - 600 mg**
**L- carnitine - 100 mg**
**L-lysine - 300 mg**
**L- proline -15 mg**
**Vitamin B5 (calcium pantothenate) - 70 mg**
**Vitamin C including ascobyl palmitate- 500 mg**
**Vitamin E - 75 I.U.**

Other nutrients that can be helpful include hawthorne, n-acetyl cysteine, pine bark extract, ornithine, glutamine, malic acid, citrus bioflavonoids, peperine extract.

**If there are significant cardiac health challenges such as high blood pressure, calcium plaques, or arrhythmias present, the dosage should be increased substantially up to 5 to 20 times of each nutrient, depending on the situation.**

**Source:** http://drlam.com/articles/heart-disease-prevention.asp?page=5