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# Dietary and Lifestyle Interventions for a Healthy Heart

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## Heart Facts

**T**he heart begins to beat prior to our birth and pumps life-sustaining oxygen and nutrients to the trillion of cells in our body over the course of a lifetime. The human body has more than 60,000 miles of blood vessels that serve as conduits for the passage of more than 3000 gallons of blood per day.

The heart is an amazingly simple, yet marvelous, pump. So many Americans dedicate large amounts of time and energy to gain outward signs of beauty and health; all the while the most important muscle in the body is sometimes working against all odds. Unlike other muscles, the heart doesn't get to rest between workouts.

Heart disease and stroke represent the number 1 and 3 causes of death, respectively, in the United States. These conditions are often referred to as silent killers, because the first sign of distress is often the last. The statistics are anything but silent, as they call attention to the fact that more than 43 out of every 100 Americans will die from cardiovascular disease.

The first step for most health-conscious patients who have become educated about maintaining a healthy cardiovascular system is to focus on controlling the classic risk factors (see sidebar). Although this is a good start, it does not represent the full picture when it comes to more fully addressing equally critical risk factors.

Controlling the classic cardiovascular risk factors is without a doubt a good start. However, the next step, especially for those patients with personal or family histories of heart disease, should also consider factors that lead to oxidative damage to the heart and vascular system. These risk factors can be just as important as the more classic concerns. Among these other factors are well known yet frequently overlooked considerations (see box entitled "Risk Factors for Cardiovascular Disease.").

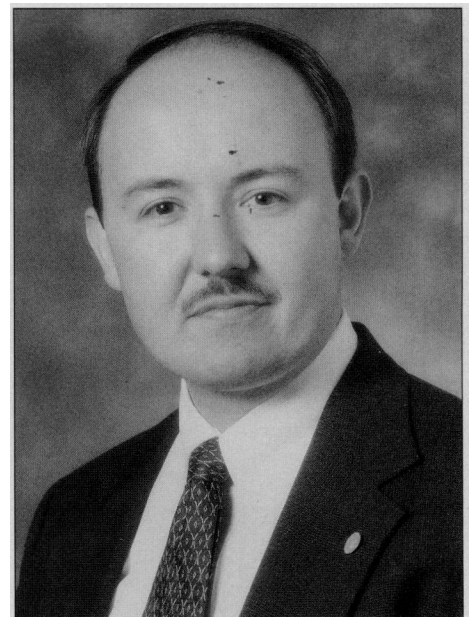
The following discussion points are some basic and fundamental approaches to decreasing this second group of risk factors associated with cardiovascular oxidative damage and to promote overall good health.

## Strategic Dietary and Lifestyle Considerations

### *Simple Dietary Insights*

*Fruits, vegetables, and more fiber.* The reasoning behind eating 5 to 7 servings of fruits and vegetables per day is not limited to the extra fiber that they offer to our diets. The panacea of fruits and vegetables includes their ability to provide the biochemical fuel to quench free radicals, as well as fiber to absorb and bind lipids to maximize excretion while enhancing overall bowel transit times. These food sources also provide the key nutrients essential to maintain the structural integrity of the cardiovascular system.

*Foods to avoid to maximize a healthy heart.* There are numerous types of foods that if avoided can actually protect the heart from unnecessary damage. Among the most common in the Standard Amer-



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ican Diet are *trans*-fatty acids (TFAs). In fact, the research on TFAs has become so substantial that the American Heart Association issued a recommendation to lessen the consumption of hydrogenated oils. Foods to avoid include fried foods, processed foods with partially hydrogenated oils, margarines, and other TFA-rich dietary sources.

### *Lifestyle Factors for a Healthy Heart*

Worrying is bad for one's health, according to a study of 1759 men. Over the course of 20 years it was noted that men who worried the most had a 241 percent increase in heart disease.<sup>1</sup>

## A 49.8 percent reduction of homocysteine, was obtained with a combination therapy of three B vitamins.

### Risk Factors for Cardiovascular Disease

#### Classically Emphasized Risk Factors

Family history  
 Inactivity/sedentary lifestyle  
 Smoking  
 Diabetes mellitus  
 Hypertension  
 Elevated cholesterol/triglycerides  
 Obesity

#### Additional Controllable Risk Factors

Elevated homocysteine  
 High trans-fatty acid intake  
 Lipid peroxidation  
 Elevated serum iron

Modifying type A behavior traits can lower cholesterol levels. It was noted in a study of 86 healthy subjects that those with the highest degree of aggression demonstrated a relative elevation of cholesterol.<sup>2</sup>

#### Possible Additions to a Good Diet

##### *Homocysteine: A Balancing Act With B Vitamins*

There is a fine balance between sufficient but not excessive levels of methionine, a key amino acid in the formation of many proteins, that when degraded yields as its first intermediate homocysteine. The evidence suggests that patients with personal or family histories of heart disease should be monitored for homocysteine levels.

Homocysteine has been found to be elevated in 20–40 percent of those patients with cardiovascular disease.<sup>3</sup> It

has been suggested in the literature that the top 5 percent homocysteine producers have a threefold increase in heart disease relative to those with normal homocysteine levels. This magnitude of damage has been attributed to the ability of elevated levels of homocysteine to increase the generation of free radicals such as hydrogen peroxide. This results in oxidative damage to the endothelial cells lining the cardiovascular system and results in lipid peroxidation of low-density lipoprotein (LDL), substantially increasing its ability to adhere to the damaged endothelial lining.<sup>4</sup> Plaque formation has also been linked to the damage to endothelial collagen that can be induced by homocysteine.<sup>5</sup>

The amount of homocysteine that accumulates within the body is largely dependent on the biochemical pathways and their ability either to remethylate homocysteine to methionine or to be further broken down into the amino cysteine. The remethylation process requires sufficient levels of both vitamin B<sub>12</sub> and folic acid. To further enhance the degradation process, which is a vitamin B<sub>6</sub>-dependent pathway, supplementation with this nutrient potentiates the biochemical pathway.

In a placebo-controlled study of 100 men with hyperhomocysteinemia, it was demonstrated that these folic acids, vitamins B<sub>6</sub> and B<sub>12</sub>, work best together. The men were given either 400 µg vitamin B<sub>12</sub>, 650 µg folic acid, 10 mg vitamin B<sub>6</sub>, or a combination. The outcome was that plasma homocysteine levels were reduced by 41.7 percent with folic acid,

14.8 percent with vitamin B<sub>12</sub>, and a negligible amount with vitamin B<sub>6</sub>. However, the most significant, a 49.8 percent reduction of homocysteine, was obtained with a combination therapy of these three B vitamins.<sup>6</sup> Numerous studies have confirmed these findings.<sup>7</sup> It should be noted that as one ages, the likelihood of elevated homocysteine increases.<sup>8</sup>

#### *Nutrient Protectors Against Oxidative Damage*

Oxidative damage to the cardiovascular system is an ever-present occurrence; however, minimizing this attack is a must to sustain optimal health. Some of the greatest oxidative stressors include reactive oxygen species, oxidized dietary fats, environmental chemicals and pollutants, and peroxides. Preventing the oxidation of LDL is critical to the creation of a successful healthy heart protocol. There are many nutrients that can be used as tools in the battle against oxidative damage and for a healthy heart. The following discussion provides an overview of some of the many potential approaches that can be considered.

*β-carotene.* This fat-soluble antioxidant serves as a component of LDL cholesterol, although at a smaller fraction relative to vitamin E involvement.<sup>9</sup> Although it comprises 1/20 the amount of vitamin E, it can decrease copper-induced LDL oxidation by 40 percent.<sup>10</sup> Epidemiologic studies have shown that individuals who consume foods high in β-carotene manifest a lower risk of cardiovascular disease.<sup>11</sup>

## Flavonoids serve as potent antioxidants.

**Carnitine.** The primary fuel source for the heart is mitochondrial fatty acid oxidation, which is in large part driven by carnitine. Carnitine synthesis is dependent on sufficient levels of the following cofactors: vitamin B<sub>6</sub>, iron, vitamin C, and niacin. It is well documented in the scientific literature that during and shortly after an acute myocardial infarction (MI), carnitine levels drop. The use of L-carnitine therapeutically in acute MI patients has been shown to reduce the magnitude of tissue damage and assist in preventing ventricular arrhythmias.<sup>12</sup> In a post-MI study, use of 1 g twice daily resulted in a 90 percent decrease in mortality.<sup>13</sup>

**Coenzyme Q10.** This amazing nutrient, also commonly referred to as ubiquinone, is a ubiquitous micronutrient in the human body that serves numerous functions, including as a potent antioxidant that assists with the recycling of vitamin E. Additionally, it serves a vital role in adenosine triphosphate (ATP) production. As a result, its clinical applications frequently include use in the treatment of congestive heart failure, prevention of ischemia associated with bypass surgery, angina, and other various cardiopathies.<sup>14</sup>

**Flavonoids.** Found in various foods and botanicals, flavonoids serve as potent antioxidants. Research has demonstrated their ability to quench numerous free radicals including hydrogen peroxide, superoxide, lipid peroxide, and hydroxyl radicals.

Epidemiologic studies, including a Finnish study, demonstrate an inverse

### The Basics of Cardiovascular Supportive Nutrients

The following is a list of recommended dosages of basic cardiovascular nutrients. This abbreviated list of nutrients have been well documented in the scientific literature as being helpful in one or more clinically significant ways. However, when combining drugs and nutrients it is important to keep in mind the potential for interactions that may cause potentially harmful interactions.

Nutrient	Recommended Dosage	Frequency
Carnitine	100 mg	2–3+ times a day
Chromium	200 µg	2–3 times a day
CoQ10	100 mg	1–3 times a day
Folic acid	400 µg	1–5 times a day
Flaxseed oil	14 g	1–2 times a day
Proanthocyanidins	50 mg	2–3 times a day
Niacin (Inositol Hexaniacinate)	1000 mg	3 times a day
Pantethine	300 mg	3 times a day
Vitamin B <sub>6</sub>	50 mg	1–3 times a day
Vitamin B <sub>12</sub>	500 µg	1–3 times a day
Vitamin C	1000 mg	3–4 times a day
Vitamin E	400 IU <sup>a</sup>	1–2 times a day

Note: This is by no means an exhaustive list; rather, it is intended as a review of the most commonly used nutrients to optimize heart health. <sup>a</sup> IU, international units.

relationship with dietary flavonoids and cardiovascular mortality.

**Flaxseed oil/essential fatty acids.** Autopsy studies have demonstrated that individuals with the lowest fat tissue levels of ω-3 oils had the highest degree of coronary artery disease.<sup>16</sup> Clinical research has now demonstrated that cells that have essential fatty acids incorporated into their phospholipid bilayer are more resistant to oxidative and other forms of damage. The intake of the equivalent of 1 tablespoon of flaxseed oil has been recommended to lessen cardiovascular disease.<sup>17</sup>

**Selenium.** Glutathione peroxidase, a selenium-dependent enzyme, has been shown to help protect LDL from oxidation. The ability of selenium to work synergistically and, at times, in place of vitamin E as a nutrient in the body also suggests the importance of this trace mineral in the role of a heart protective agent.

**Taurine.** Taurine comprises 50 percent of heart free amino acids. It is believed that one of taurine's functions is as a cardiac antioxidant.<sup>18</sup> Taurine plays a key role along with glycine for cholesterol conjugation and excretion.

**Research has demonstrated that supplementation with a minimum of 400 IU daily substantially decreases oxidative damage to LDL.**

**Drug–Nutrient Interactions: Things to Consider**

Drug	Nutrient
ACE inhibitors <sup>a</sup>	Can increase chance of lithium toxicity
Aspirin	May lead to tissue ascorbic acid depletion
Anticoagulants	Vitamin E may potentiate their effects
β-Blockers	Act as antagonist to CoQ10
Clofibrate	Can reduce vitamin B <sub>12</sub> absorption
Furosemide	Can lead to a loss of thiamine
HMG-CoA reductase inhibitors	Increases the need for CoQ10 Use with niacin may increase chance of rhabdomyolysis and myopathy.
ProbucoI	May lead to indirect vitamin E depletion
Warfarin (Coumadin)	CoQ10 may decrease therapeutic benefit Vitamin K can antagonize the effects

Note: This chart shows examples of the many interactions that have been documented. <sup>a</sup> ACE inhibitors, angiotensin-converting enzyme inhibitors.

**Vitamin C.** Humans and only a handful of animals, including guinea pigs, other primates, and fruit bats, cannot synthesize vitamin C. Hence, to obtain sufficient levels, a person must consume it in either food or supplement form.

Ascorbic acid protects the cardiovascular system as an antioxidant and helps with the excretion of cholesterol. Vitamin C also assists in the biochemical pathway responsible for the recycling of oxidized vitamin E.<sup>19</sup>

**Vitamin E.** α-Tocopherol is essential to protect against LDL oxidation. Research has demonstrated that supplementation with a minimum of 400 IU daily substantially

decreases oxidative damage to LDL.<sup>20</sup>

Epidemiologic studies illustrate the power that vitamin E possesses to protect against this oxidative damage. A review of 39,910 male health care practitioners between 40 and 75 years of age demonstrated that those individuals who consumed more than 60 International units (IU) per day compared with those who consumed less than 7.5 IU per day had a 36 percent decreased risk of cardiovascular disease.<sup>21</sup> A similar study that included more than 87,000 nurses demonstrated similar results. When the top consumers of vitamin E were compared with those with the lowest levels, they had a 34 per-

cent decreased risk of heart attack.

Among the many other beneficial effects of consuming sufficient amounts of vitamin E is its ability to decrease total cholesterol, and help maintain an ideal balance of apolipoproteins. This ability was demonstrated in a study of 69 men. Apolipoprotein A (apo A), the principal protein in high-density lipoproteins (HDL), rose significantly, and apo B levels, the principal protein in LDL, dropped.<sup>22</sup>

At this point it is important to note that the nutrients discussed above have many other beneficial properties beyond their ability to serve as antioxidants. In addition, there are numerous other nutrients that can serve as cardiovascular antioxidants. There are also many commonly used nonherbals that also confer other means of protection and support to the heart, including lipoic acid, chromium, niacin, magnesium, calcium, potassium, sodium, and pantethine to name a few.

At the heart of natural medicine is the concept that once the cause, or in this case an imbalance, is identified then a unique and specific biochemical approach using nutrients and medication can address either an underlying predisposition or an actual disease state. To gain further insights as to which nutrients will prove most helpful for your patients' unique biochemical needs, specific laboratory testing can offer clarity and direction.

*Beyond Basic Laboratory Testing*

Testing considerations for a comprehensive panel now more frequently

## There are numerous clinically proven dietary and nutrient approaches to enhancing overall cardiovascular health.

include more than just a breakdown of total cholesterol into LDL and HDL components and triglyceride levels. Currently, for a patient with heart disease or a positive family history, it is not uncommon to request a more advanced screen including apo A, apo B, very low-density lipoprotein, fibrinogen, homocysteine, ferritin, and lipid peroxides.

There are numerous clinically proven dietary and nutrient approaches to enhancing overall cardiovascular health. In addition, there are numerous powerful herbal interventions that should be considered such as guggulipids, garlic, onion, hawthorn, and ginkgo among many others. Without a doubt, supporting the most magnificent yet simple pump in the universe needs just as much attention as one's biceps or wrinkle-free radiant complexion. □

### References

1. Kubzansky, L.D., et al. Is worrying bad for your heart? *Circulation* 95:818-824, 1997.
2. Muller, M.M., et al. The relationship between habitual anger coping style and serum lipid and lipoprotein concentrations. *Biol Psychol* 41:69-81, 1995.
3. Glueck, C.J., et al. Evidence that homocysteine is an independent risk factor for atherosclerosis in hyperlipidemic patients. *Am J Cardiol* 75:132-136, 1995.
4. Stamler, J., et al. Adverse vascular effects of homocysteine are modulated by endothelium derived relaxing factor and related oxides of nitrogen. *J Clin Invest* 91:308-318, 1993.
5. Stamler, J., et al. Biological chemistry of thiols in the vasculature related disease. *Nutr Rev* 91:308-318, 1993.
6. Ubbinak, J., et al. Vitamin requirements for the treatment of hyperhomocysteinemia in humans. *J Nutr* 124:1927-1933, 1994.
7. Mason, J., et al. The effects of vitamins B12, B6 and folate on blood homocysteine levels. *Ann NY Acad Sci* 669:197-203, 1992.
8. Selhub, J., et al. Association between plasma homocysteine concentrations and extracranial carotid stenosis. *N Engl J Med* 332:286-291, 1995.
9. Esterbauer, H., et al. Effect of antioxidants on oxidative modification of LDL. *Ann Med* 23:573-581, 1991.
10. Levy, Y., et al. Effect of dietary supplementation of beta carotene on human monocyte macrophage mediated oxidation of low density lipoproteins. *J Am Diet Assoc* 95:671-675, 1995.
11. Pandey, D., et al. Dietary vitamin C and beta carotene and risk of death in middle aged men. The Western Electric Study. *Am J Epidemiol* 145:1269-1278, 1995.
12. Rizzon, P., et al. High doses of L-carnitine in acute myocardial infarction: Metabolic and antiarrhythmic effects. *Eur Heart J* 10:502-508, 1989.
13. Davini, P., et al. Controlled study on L-carnitine therapeutic efficacy in post infarction. *Drugs Exp Clin Res* 18:355-365, 1992.
14. Thomas, S., et al. Cosupplementation with coenzyme Q prevents the prooxidant effect of alpha-tocopherol and increases the resistance of LDL to transition metal-dependent oxidation initiation. *Arterioscler Thromb Vasc Biol* 16:687-696, 1996.
15. Knekt, P., et al. Flavonoid intake and coronary mortality in Finland: A cohort study. *Br Med J* 312:478-481, 1996.
16. Seidelin, K.N., et al.  $\omega$ -3 fatty acids in adipose tissue and coronary artery disease are inversely correlated. *Am J Clin Nutr* 55:1117-1119, 1992.
17. Sandker, G.N., et al. Serum cholesterol ester fatty acids and their relation with serum lipids in elderly men in Crete and The Netherlands. *Eur J Clin Nutr* 47:201-208, 1993.
18. Milei, J., et al. Reduction of reperfusion injury with preoperative rapid intravenous infusion of taurine during myocardial revascularization. *Am Heart J* 123:339-345, 1992.
19. Packer, L. Interaction among antioxidants in health and disease: Vitamin E and its redox cycle. *Proc Soc Exp Biol Med* 200:271-276, 1992.
20. Jailal, I., et al. The effect of alpha-tocopherol supplementation on LDL oxidation. *Arterioscler Thromb Vasc Biol* 15:190-198, 1995.
21. Rimm, E.B., et al. Vitamin E consumption and the risk of coronary heart disease in men. *N Engl J Med* 328:1450-1456, 1993.
22. Cloarec, M.J., Alpha-tocopherol: Effect on plasma lipoproteins in hypercholesterolemic patients. *Isr J Med Sci* 23:869-872, 1987.

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