Conquering Hair Loss©
Nasser Razack, MD

Chapter 12 - Hair Loss and Heart Disease

“Coronary heart disease is a silent disease, and the first manifestation is frequently sudden death.”
-Herman Hellerstein

As stated in the previous chapter, recent evidence indicates that male-pattern baldness is associated with an increased risk of heart disease. Since most people reading this book suffer from androgenic alopecia, it's a good idea to review what the current medical literature has to say about the association between male pattern baldness and heart disease. In addition, what we can do to lower the risk of heart disease will also be discussed.

I would like to clarify one thing before proceeding. As an author, my primary goal in the process of writing this book was to provide the reader with as much information as possible on the subject of hair loss. As a physician, I cannot ignore the association between heart disease and androgenic alopecia. Although this chapter does not directly discuss hair loss, I strongly urge you to read it. In many ways, it just might be the most important chapter in this book.

Although this chapter is the longest chapter in this book, it only scratches the surface of what we know about heart disease and what we can do to lower its risk. You might be reading this book because you have androgenic alopecia. If this is the case, you have an increased risk for heart disease. I strongly urge you to discuss this risk with your physician and to acquire as much knowledge as possible on how to prevent heart disease. There are many books devoted to heart disease. One I would strongly recommend is Conquering Heart Disease by Harvey B. Simon, MD.

One and a half million heart attacks occur in the United States every year, establishing cardiovascular disease as this nation's number one killer. It accounts for roughly 600,000 deaths each year. Almost 25% of American adults have some type of cardiovascular disease. Two out of every five Americans will die from it and close to 20% of these people will be less than sixty-five years old.

The medical term for a heart attack is myocardial infarction. Heart attacks occur because the blood vessels that nourish the heart with oxygen, the coronary arteries, become occluded. Oxygen is essential for the heart to function properly. Once these arteries are not able to supply the heart with enough oxygen, the individual muscle cells of the heart can die (infarct). If enough of these cells die, the result is a heart attack (Figures 12-1 and 12-2). One way to prevent heart attacks is to prevent the coronary arteries from becoming occluded.

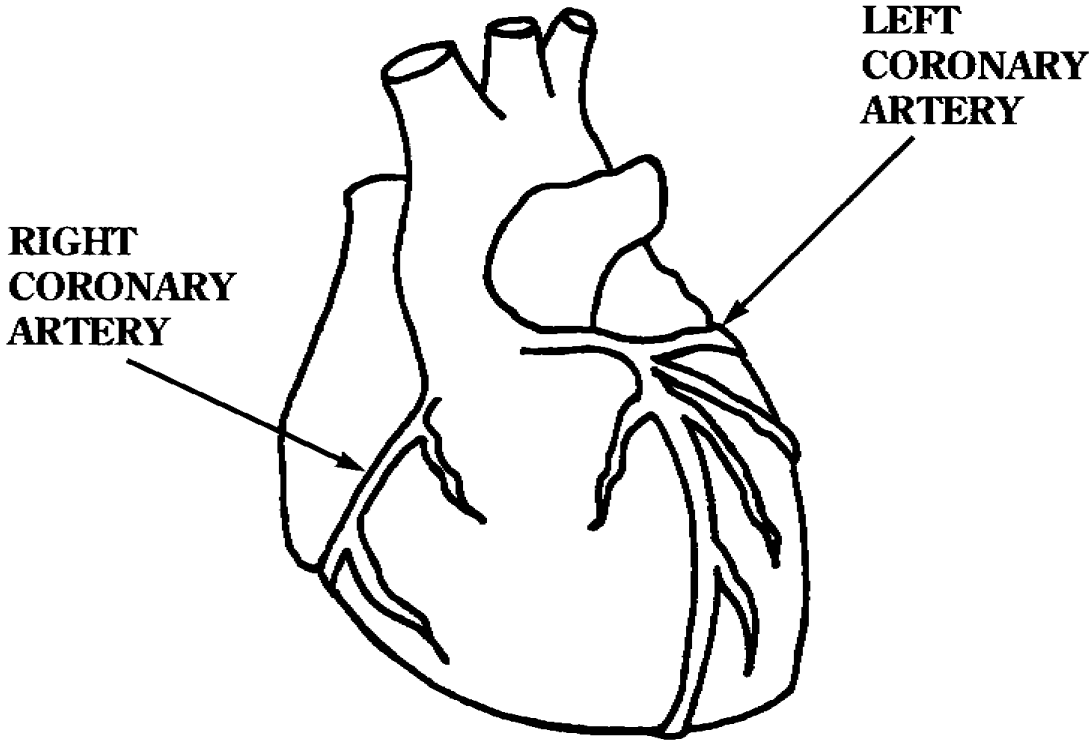

FIGURE 12-1 Diagrammatic representation of the coronary arteries. These are the two major arteries that supply the heart with oxygen.

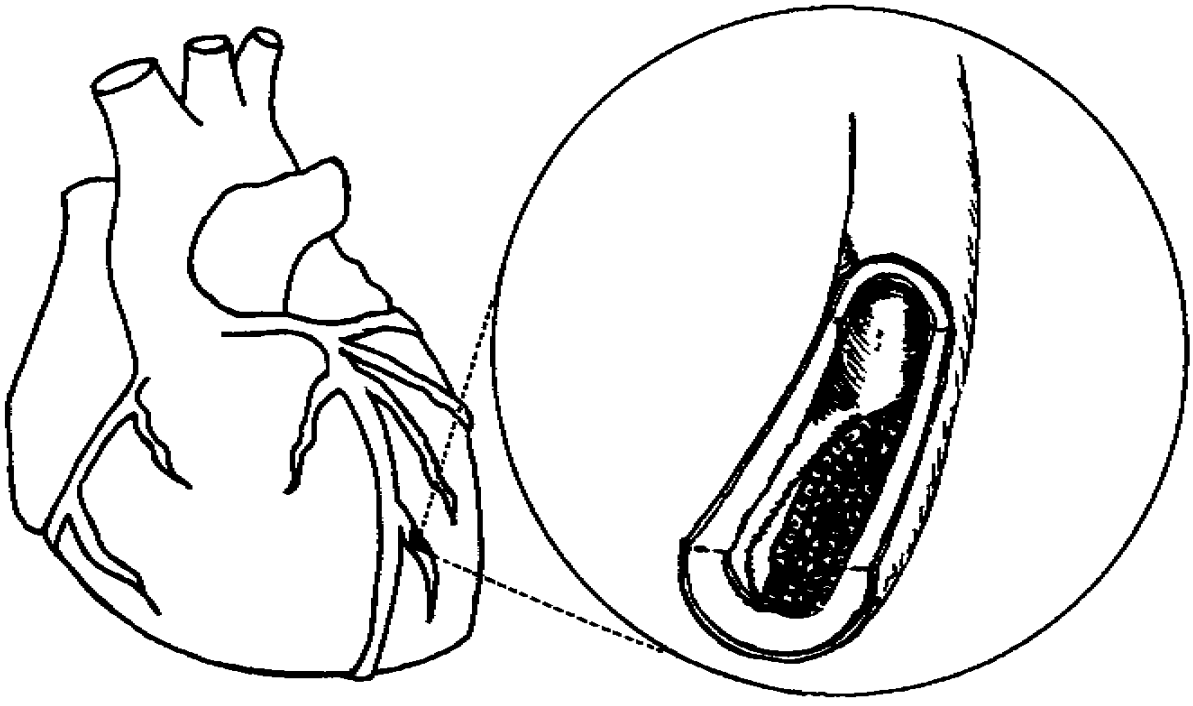

FIGURE 12-2 Diagrammatic representation of coronary artery disease. When the coronary arteries narrow or become obstructed, they cannot adequately supply the

heart with oxygen. This inadequate supply of oxygen to the heart results in a heart attack.

To prevent the coronary arteries from becoming blocked, we need to determine how they can get that way. Often, when the heart is starved of oxygen because of narrow coronary arteries, an individual experiences a distinct type of chest pain called angina pectoris. Changes in the inside lining of arteries due to excess intake of saturated fats and cholesterol is one explanation for the accumulation of obstructions within the coronary arteries. The most common cause of angina pectoris, or chest pain, is the progressive narrowing of coronary arteries by the accumulation of plaques that are atherosclerotic.

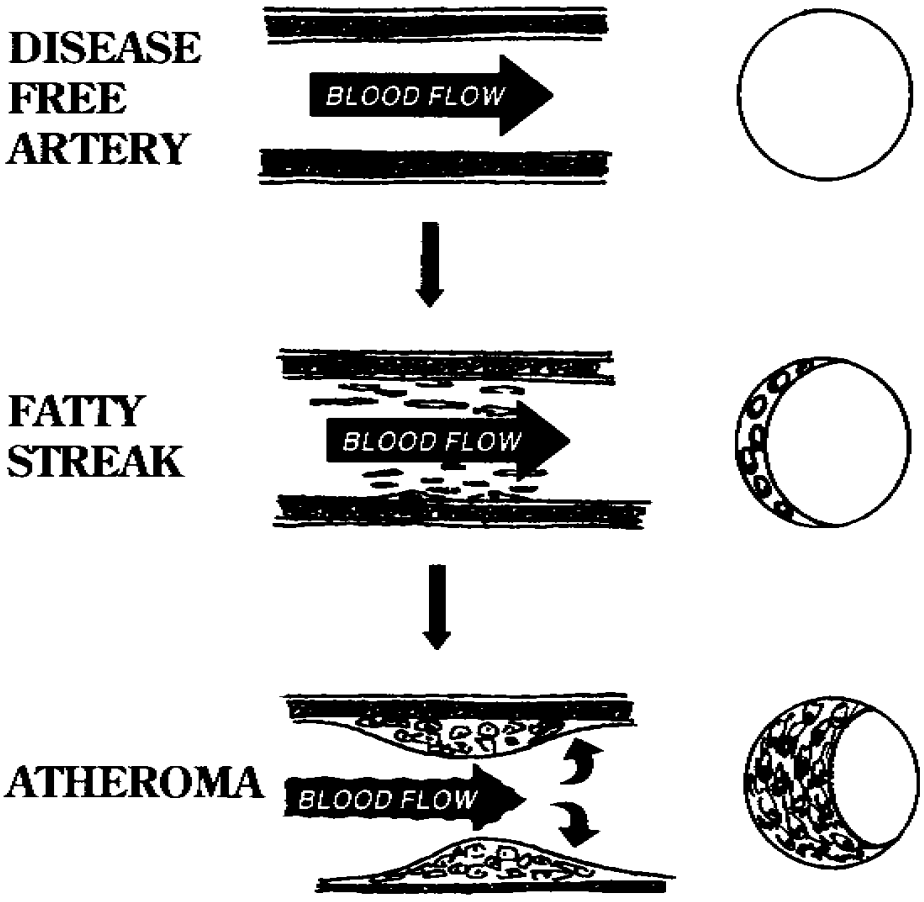

FIGURE 12-3 Progressive decrease in the cross-sectional area of an artery due to atherosclerosis.

Atherosclerotic plaques are composed of fatty substances that exist within the inside lining of an artery (Figures 12-2 and 12-3). These plaques cause narrowing of the lumen of an artery, and can block up to 85% of its cross-sectional area before any symptoms of decreased blood flow or inadequate oxygen delivery are seen. If, however, the

workload of the heart is increased and it now requires more oxygen, smaller degrees of coronary obstruction may result in inadequate blood flow.

The best way to treat heart disease is to prevent it. One way to accomplish this is by preventing the formation of atherosclerosis. This is a key concept because atherosclerosis not only affects the coronary circulation but also affects circulation throughout the body. For example, it may also affect the carotid arteries, which supply the brain with oxygen. Depriving the brain of oxygen can result in a stroke, the equivalent of a heart attack in the brain. By preventing the formation of atherosclerosis, we are not only protecting the heart, we are protecting the entire body.

The most amazing aspect about atherosclerosis is that it is preventable and it is not inevitable. In many cultures, atherosclerosis is almost unheard of. These societies have vegetable-based diets, cigarette-free environments, and routine physical activity. Prior to the industrial revolution, heart attacks were almost unheard of. Why are we now facing this epidemic? The answer is simple: our behavior as a society is the major contributor to this disease process. We avoid fiber and vitamin-rich foods, such as vegetables, and substitute them with fat-filled animal products. Individuals in our society are overweight and accustomed to an incredibly low level of physical exertion. People continue to smoke and non-smokers are constantly exposed to second-hand smoke, and, yes, this is also dangerous. In short, the disease process of atherosclerosis has been created by the way we have chosen to live our lives.

After the discussion above, a more appropriate term for heart disease would be coronary artery disease, because it is the obstruction of these arteries (that provide the heart with oxygen and other nutrients) which results in heart disease. Also, recall that these arteries are most commonly obstructed by atherosclerotic plaques. There are many conditions, or "risk factors", that can favor the development of coronary artery disease. One such factor is the serum cholesterol level: as the level of cholesterol in the bloodstream increases, so does the risk of heart disease.

In the blood, cholesterol is bound to a "carrier" protein called a lipoprotein. There are only two kinds we need discuss. One is called low-density lipoprotein (LDL). You may have heard of it being referred to as "bad" cholesterol, since cholesterol bound to LDL has been demonstrated to cause atherosclerotic plaques once it is oxidized. The key phrase is "once it is oxidized." All current research indicates that normal LDL is harmless prior to oxidation. The latter part of this chapter will discuss antioxidants, agents that help in preventing harmless LDL-cholesterol from being oxidized into the dangerous form that contributes to atherosclerosis.

Cholesterol bound to another lipoprotein, called high-density lipoprotein (HDL), is often referred to as "good" cholesterol. This is because higher levels of HDL-cholesterol are thought to prevent the formation of atherosclerotic plaques and the resulting coronary artery disease. HDL carries cholesterol away from arteries to the liver where it is broken down into bile salts and eliminated from the body. Thus, knowing the ratio of the "good" versus the "bad" cholesterol is extremely important. The risk of heart disease can be

calculated from the levels of total serum and HDL-cholesterol. To accurately determine this risk, just perform the simple calculation:

$$\frac{\text{Total cholesterol}}{\text{HDL-cholesterol}} = \text{Risk Ratio}$$

The lower the number you calculate, the lower your risk of coronary artery disease. Please refer to Table 12-1.

TABLE 12-1 Cholesterol Risk Ratios

Ratio	Risk
6	high
5	above average
4.5	average
4	below average
3	low

In addition to cholesterol, other important risk factors for coronary artery disease include smoking, high blood pressure, diabetes, and a family history that is indicative of early atherosclerotic disease. Physicians have been finding additional risk factors that may be linked to coronary artery disease, and one of these appears to be androgenic alopecia, or male-pattern hair loss. It constitutes, however, a much smaller risk factor than any of the others listed above [1].

Many researchers and physicians have raised the issue that hair loss is related to health; the first physician did so almost 200 years ago. In 1812, Napoleon marched into Russia with 500,000 men and retreated with only 40,000. His chief surgeon noted that bald men were the first to die [2]. As mentioned above, physicians are now associating hair loss with coronary artery disease. In the remainder of this chapter, this association will be explored and new avenues for the prevention of heart disease will be discussed.

The following discussion is not meant to alarm you, but rather to persuade you to take action. Maybe it's time to see your doctor and to start taking the steps required to prevent heart disease. It might be time to change your lifestyle in terms of eating habits and exercise. The bottom line is simple: if your body is telling you something, you should listen. I hope that other physicians are also listening. Although the association between hair loss and heart disease is an unfortunate one, it will probably urge more physicians to consider hair loss more seriously.

A recent study performed by Lesko et al reexamined the influence of hair loss on coronary artery disease [3]. They compared 665 men who had suffered non-fatal heart attacks to 772 men who had been admitted to the same hospitals for non-cardiac reasons. They found that the risk of a heart attack increased in proportion to the degree of vertex balding. Men under the age of fifty-five with moderate hair loss on top of their heads had approximately a 30% increase in the risk of suffering from a heart attack compared to nonbalding men; those with extreme hair loss had over a 300% increased risk.

Currently, researchers theorize that heart disease may be associated with the levels of dihydrotestosterone. Since this hormone is the major contributor to androgenic alopecia, it would provide a link between heart disease and hair loss in individuals under the age of fifty-five. In contrast, the hair loss that is observed in older individuals is thought not to be caused by increased levels of dihydrotestosterone and does not appear to correlate with heart disease. Recall that dihydrotestosterone affects other organs in the body, including the prostate. It is certainly possible that it may also affect the heart. Its exact role in heart disease remains to be elucidated.

Another study adds yet more information about the association between androgenic alopecia and heart disease. Trevisan et al examined this association and also measured serum cholesterol levels [4]. They found that, in addition to androgenic alopecia being a risk factor for coronary heart disease, serum cholesterol levels were significantly higher in participants with androgenic alopecia compared to those without it. Also, the association between higher serum cholesterol levels and androgenic alopecia became weaker with increased age; that is, the older the person, the less significant this association. Again, the hair loss we experience as we age is a completely separate entity from androgenic alopecia. Both these studies clearly indicate that heart disease is associated with the hair loss that is due to androgenic alopecia and not with the hair loss that accompanies aging.

As discussed earlier in this book, dihydrotestosterone is a steroid hormone. All steroid hormones are formed from cholesterol. This means that without cholesterol, you would not be able to manufacture dihydrotestosterone. The process by which these substances affect both heart disease and hair loss still needs to be determined. However, one possibility is that individuals with high levels of serum cholesterol may have increased risk of heart disease, and at the same time, produce higher levels of dihydrotestosterone. In other words, the same cholesterol that contributes to blocking arteries is also required to make dihydrotestosterone. In individuals with androgenic alopecia this hormone leads to hair loss. Another possibility is that the same atherosclerotic process that blocks the small coronary arteries in the heart may also block the microvasculature that nourishes the hair follicles. For now, however, these are only theories.

Now that we understand the mechanisms of coronary artery disease and the risk factors that are associated with it, it's time to discuss what to do about it. It is extremely

important to stop smoking. This not only affects the heart, but just about everything else in your body. Although this vast subject cannot possibly be discussed in only one chapter, the bottom line is simple: if you smoke, please stop. Your life may depend on it. An excellent book dealing with this subject is No If's And's or Butts, The Smoker's Guide to Quitting by Harlan M. Krumholz, MD and Robert H. Phillips, MD.

Another factor that can decrease the risk of heart disease is exercise. Exercise has been clearly shown to protect against the development of heart disease. Aerobic exercise has been consistently shown to increase the levels of HDL, while decreasing those of LDL. Exercise also lowers blood pressure and, in the process, reduces arterial wall stress. This results in arteries that are healthier and less prone to the vascular injury, which is a necessary requirement for LDL-cholesterol to form fatty plaques (Figure 12-3). Exercise will also decrease the ability of blood to clot. Blood clots within narrow arteries are often the last deadly step that accounts for the majority of heart attacks. However, many studies have shown that physical exertion can cause heart attacks in individuals who are unaccustomed to it. If you're not used to exercise, please be sure to be evaluated by a physician first.

Whether or not you are concerned about heart disease ask your physician to perform a fasting lipid-profile exam. This test will allow you to know the amount of LDL-cholesterol in your bloodstream. Lowering the levels of this "bad" cholesterol reduces the risk of heart attacks. A reasonable level of total serum cholesterol consists of less than 200 milligrams/deciliter (mg/dL) with an LDL component of less than 130 mg/dL. Individuals with total serum cholesterol levels of 200 to 239 mg/dL have a borderline risk of cardiovascular disease, whereas those with levels of 240 mg/dL or greater have a high risk.

More important than the level of serum cholesterol, however, is the level of "bad" or LDL-cholesterol. Again, levels should be less than 130 mg/dL. Levels between 130 and 159 mg/dL are considered to be borderline risk factors, and those over 160 mg/dL high. Also, the higher the levels of "good" or HDL-cholesterol, the lower the risk of cardiovascular disease. The normal levels of HDL range from 30 to 75 mg/dL. Please refer to Tables 12-1 and 12-2.

TABLE 12-2 CHOLESTEROL LEVELS* AND RISK OF CARDIOVASCULAR DISEASE

	SERUM CHOLESTEROL	LDL	HDL
OPTIMAL LEVELS		less than 100	greater than 60
DESIRABLE LEVELS	less than 200	less than 130	

	SERUM CHOLESTEROL	LDL	HDL
BORDERLINE LEVELS	200-239	130-159	
HIGH RISK LEVELS	greater than 240	greater than 160	less than 35

Source: Adapted from Int Surg 76(l): 1-5, 1991.

**Note that total serum cholesterol, LDL, and HDL-cholesterol levels are expressed in mg/dL of blood.*

If your cholesterol is high, your physician will first try dietary therapy. This includes reducing the daily intake of calories, saturated fats, and cholesterol. Saturated fats are a form of harmful fat generally obtained from animal sources other than fish. In contrast, unsaturated fats appear to be beneficial in preventing heart disease. The majority of unsaturated fats are derived from vegetables as well as fish. However, some vegetable sources provide dangerous saturated fats. These include cocoa butter, coconut oil, palm oil, and palm kernel oil.

In addition to being careful to avoid these vegetable sources of saturated fats, be sure to be on the lookout for partially hydrogenated vegetable oil, which is even worse. The problem with partially hydrogenated vegetable oil is that it increases the levels of bad LDL-cholesterol and lowers the levels of good HDL-cholesterol within the body. This type of vegetable oil is man-made and does not exist in nature. Some studies indicate that partially hydrogenated vegetable oils negatively affect the LDL/HDL lipid ratio to a greater extent than saturated fats [5].

For all these reasons, it is extremely important to carefully analyze the nutrition facts printed on the label of any product you eat (Figure 12-4). Be especially careful to look at the ingredients of items marked as low or no fat. Manufacturers often use partially hydrogenated vegetable oils to substitute for fat. Also, remember that cholesterol is not a fat but a lipid. Many packages marked as fat free may contain cholesterol. The moral of the story is to read the label (Figure 12-4).

[Fig. 12-4 in "Conquering Hair Loss" is a typical "Nutrition Facts" label found on almost all packaged food products.]

These are several important things to check on Nutrition Facts labels:

1. Fat and cholesterol should be immediately checked. Beware of saturated fats and cholesterol.
2. Examine the content of fiber. More fiber is better.

3. Only 15-20% of calories need to be derived from proteins.
4. When checking ingredients, be particularly careful to avoid partially hydrogenated vegetable oils.

If your cholesterol levels are still elevated after dietary and exercise therapy, you may require one of several drugs that are currently available for reducing serum cholesterol levels. I do not discuss these drugs in this book because such information should be discussed with your physician.

It's surprisingly easy to avoid cholesterol in the diet. Our body is able to produce more than enough cholesterol by itself and there is no reason to obtain cholesterol from the diet. Almost everything that is neither an animal product nor derived from animal products is free of cholesterol. Generally speaking, the American population consumes too much meat. In particular, meats like pork and beef usually contain more fats and cholesterol than leaner meats such as poultry and fish.

In addition to providing fats and cholesterol, meat consumption leads to another problem. One of the byproducts of meat, once it has been digested, is homocysteine, which has been shown to be a major risk factor in the development of atherosclerosis throughout the body [6, 7]. Homocysteine is believed to cause injury to the inside lining of arteries, thus predisposing them to atherosclerosis [8]. For this reason, the risk of coronary artery disease increases with increasing plasma homocysteine levels, regardless of an individual's age or sex [9].

There are several causes of cardiovascular disease, but hyperhomocysteinemia (increased levels of homocysteine in the blood) can be eliminated by vitamin therapy. Chronic oxidative damage to the inside lining of arteries is another mechanism that can result in cardiovascular disease. It will be discussed shortly. Increased plasma homocysteine concentrations have also been shown to affect vessels in other parts of the body. A recent study has demonstrated that increased levels of homocysteine can contribute to the narrowing of carotid arteries. These are the blood vessels that supply oxygen and other nutrients to the brain; as a result, increased levels of homocysteine can cause the equivalent of a heart attack in the brain (a stroke).

Although the consequences of having high levels of homocysteine may sound alarming, the problem can easily be ameliorated with two simple vitamins. Folic acid and vitamin B6 have been shown consistently to lower the concentrations of homocysteine within the body. One study demonstrated that this could be effectively accomplished after fifteen weeks of vitamin therapy, consisting of five milligrams of folic acid and one hundred milligrams of vitamin B6 daily [6]. Please note that I do not recommend the use of folic acid in this quantity (5 mg daily). That dose is for therapeutic use and is not to be taken routinely. The efficacy of both these vitamins in lowering homocysteine levels has been demonstrated in other studies [10]. Please consult your physician prior to taking any supplements.

Of course, another way to solve this problem is to become a vegetarian, thereby removing the source of homocysteine. Don't forget that increased homocysteine is only one of the many deleterious effects of consuming meat, all of which cannot possibly be eliminated by vitamin therapy. For example, meat contains saturated fats and the preparation of meat (cooking) is associated with free-radical production. This means that even if you are using vitamin therapy, you should be extremely careful about what types of meat you choose to eat and how these are prepared.

Homocysteine isn't the only substance that can lead to cardiovascular disease. In fact, of all the organs in our body, the heart is the one most susceptible to free-radical oxidative stress [11]. Free radicals are reactive, unstable molecules that occur in the body. They are able to react with any structure in the body, but what is most interesting to us is their ability to cause atherosclerosis. Free radicals have also been implicated in other diseases, such as cancer, diabetes, and cataracts [12]. The process of aging may also be related to free-radical production [13].

Free radicals are not naturally present in food but can be produced when food is cooked, especially if it is fried. Before you despair, the good news is that the heart is extremely receptive to many natural antioxidants, such as vitamins C and E. In addition to these vitamins, many other nutrients that will be discussed below have antioxidant effects. Before proceeding with this discussion, I will review in more detail how atherosclerosis occurs.

This process of atherosclerosis is initiated by high levels of LDL-cholesterol ("bad" cholesterol). LDL-cholesterol penetrates the inside wall of an artery; the degree of this penetration is directly related to the levels of LDL-cholesterol. In addition, factors such as smoking, diabetes, and high blood pressure can also contribute to increased penetration of LDL-cholesterol within the arterial wall. It is for this reason (among others) that these are major risk factors for coronary artery disease. These initial steps in the formation of atherosclerosis can be inhibited by lowering LDL-cholesterol levels in your body, while increasing those of HDL-cholesterol.

The cells that comprise the inside lining of the arterial wall make oxygen free radicals. These free radicals immediately attack the LDL-cholesterol as it penetrates the arterial wall. Once oxygen free radicals attack the LDL-cholesterol, it becomes "oxidized." This oxidized LDL-cholesterol is then made into fatty-acid free radicals. These newly formed free radicals also attack LDL-cholesterol, oxidizing it and creating more free radicals, and so on. This vicious cycle can be stopped only by quenching the free radical which causes the oxidation of LDL-cholesterol. In the time required to neutralize these free radicals, however, thousands of LDL-cholesterol molecules may be oxidized.

Oxidized LDL-cholesterol produces the arterial wall damage that eventually leads to atherosclerosis. In addition, modified LDL-cholesterol also blocks the production of natural factors that dilate arteries. Since modified LDL-cholesterol causes narrowing of the coronary arteries by these different mechanisms, it is no wonder that it is often referred to as "bad" cholesterol. To prevent this damage to the arteries from occurring,

we need to neutralize the free radicals that are responsible for causing this damage. Antioxidants are substances that neutralize free radicals. The following pages will review these amazing compounds.

VITAMIN E

Of all the known antioxidant vitamins, vitamin E was once considered to be the most potent. This position is now being challenged by another amazing nutrient called coenzyme Q10. Since vitamin E is a fat-soluble vitamin, it can be stored in various tissues throughout the body. Experiments have shown that vitamin E prevents LDL-cholesterol from being oxidized by free radicals and, thereby, serves as the first line of defense against the formation of free radicals. There have been several studies which support the use of vitamin E for preventing heart disease. Higher levels of vitamin E in the bloodstream have been associated with a lower risk of death from heart attacks and lower levels with an increased risk of coronary artery disease.

The "U.S. Nurses' Health Study" and the "Male Health Professionals Study" are two of the most elaborate studies initiated to determine the effects of vitamin E on coronary artery disease. The first study indicated that women who took a daily dose of approximately 200 international units of vitamin E for at least two years had about 40% fewer heart attacks than women taking lower doses of vitamin E. The results of the second study were similar to the first. It showed that men who took a daily dose of 100 international units of vitamin E for at least two years also had a 40% reduction in heart attacks when compared to men taking lower doses of vitamin E. Although these results may seem impressive, it appears that antioxidants probably work best when they function in concert with one another.

COENZYME Q10

Coenzyme Q10 is also called ubiquinone. This name is appropriate because coenzyme Q10 exists in every part of the body. Its major role is in the production of energy, but it is also an extremely powerful antioxidant. Studies continue to demonstrate that this substance prevents LDL-cholesterol from being oxidized and, thus, has incredible implications for the treatment of atherosclerosis [14]. It has been repeatedly demonstrated that coenzyme Q10 reduces the symptoms and the progression of heart failure [15, 16, 17]. In one study, 424 patients with six diagnostic forms of cardiovascular disease were given an average oral dose of 242 milligrams of coenzyme Q10. Statistically significant increases in heart performance were seen in all patients after an average of 17.8 months [18]. Finally, some researchers believe that coenzyme Q10 is more potent in inhibiting LDL-cholesterol oxidation than vitamin E [19].

In addition to those effects on the heart, coenzyme Q10 has many effects on the remainder of the body. Increased circulating levels of this nutrient, after two months of oral administration, correlated to an increase in perceived vigor in one study [20]. Coenzyme Q10 has been demonstrated to aid our immune system by increasing the production of both immunoglobulin G (IgG) and T4-lymphocytes [21]. Significant

increases in IgG by coenzyme Q10 have been demonstrated in a number of studies, suggesting that it may have a role in the treatment of cancer, AIDS, and other infectious diseases [22].

VITAMIN C

There is a tremendous amount of literature regarding vitamin C intake and its effects on people. Vitamin C is known to be essential for the proper function of folic acid, amino acids, and hormones. Recently, research has focused on its role as an antioxidant. Oxidative stress due to the exposure of the body to free radicals is considered to be a potential cause for aging, cardiovascular disease, cataract formation, and cancer. Epidemiological studies have suggested that the consumption of vitamin C-rich foods may reduce the risk of death in middle-aged men [23]. Decreased levels of vitamin C in the blood have been associated with an increased risk of heart attacks in both men and women. Its consumption also appears to be directly related to a reduced incidence of both cardiovascular and cancer-related deaths.

The role of vitamin C as an antioxidant has been scrutinized by the scientific community. Studies reveal that vitamin C has a potential role in reducing the development of atherosclerosis [24]. In an animal model developed to assess the effects of this vitamin, the concentration of vitamin C within the heart was directly related to its dietary intake [25]. In addition, the amount of vitamin E and antioxidant enzymes in the heart also increased with increased dietary intake of vitamin C. Another study demonstrated that higher levels of plasma vitamin C are associated with increased levels of "good" HDL-cholesterol [26]. All these observations support the idea that dietary vitamin C supplements increase the antioxidant protection in the heart and thereby reduce the risk of atherosclerotic disease.

One reason for the observed increase in vitamin E levels in the heart as a result of dietary increases in vitamin C is that it appears to regenerate vitamin E [27]. Vitamin C also appears to cooperate with vitamin E; it functions as an antioxidant by attacking free radicals both independently and synergistically with vitamin E [28]. In fact, a great deal of scientific literature suggests that the protective role of many antioxidants on the heart can increase if these agents are used in combination with each other, rather than alone. However, the dose required for these agents to function as antioxidants is much greater than the current recommended daily allowance.

BIOFLAVONOIDS

Bioflavonoids are now also considered to be antioxidants. In addition, they increase the absorption of vitamin C by the body. Evidence that these substances also affect heart disease is accumulating. One study has demonstrated that the intake of bioflavonoids is inversely related to death caused by coronary heart disease [29]. Other studies have found a correlation between lower bioflavanoid intake and higher risk of coronary disease [30].

BETA-CAROTENE

Beta-carotene is also an antioxidant and, as such, prevents LDL-cholesterol oxidation. Although beta-carotene is not as potent an antioxidant as either vitamin E or coenzyme Q10, it is still very effective. When used in combination with these or other agents, it may influence the rate of progression of coronary atherosclerosis by interfering with LDL-cholesterol oxidation. Although it has not yet been confirmed, some preliminary data suggest that beta-carotene may raise the levels of HDL-cholesterol in the bloodstream. If this proves to be the case, beta-carotene would join vitamin C as a substance capable of lowering the risk of atherosclerotic disease.

NIACIN

Niacin has been shown to reduce LDL-cholesterol levels and to increase those of HDL. It has also been demonstrated to protect against cardiovascular disease. However, it needs to be administered in doses that are considerably higher than its recommended daily allowance. Although this is common for many antioxidants, niacin has one problem that the others do not have: niacin has substantial side effects including flushing, itching, and headaches. In addition to these more common and less dangerous side effects, niacin can also cause liver dysfunction, diabetes, and ulcers. For this reason, I strongly recommend that if you are interested in using niacin to lower your cholesterol levels, you should do so under the supervision of a board-certified physician, preferably a cardiologist.

GARLIC

Garlic has been utilized in herbal medicine for centuries. It has recently been receiving a great deal of attention because of its many beneficial effects in reducing several cardiovascular risk factors. For example, garlic reduces the levels of serum lipids, plasma viscosity, and blood pressure. It inhibits platelet aggregation and causes vasodilatation. In short, garlic appears to have many antiatherosclerotic properties.

The major active ingredient found in garlic appears to be allicin. Garlic tablets marketed by Kwai, which release a 0.6% concentration of allicin, are currently available in the United States. A study of forty-two individuals with hypercholesterolemia (elevated serum levels of cholesterol) who received 300 milligrams of garlic powder tablets from this company (Kwai) three times daily, was recently completed [31]. This study revealed that, in addition to lowering the different levels of serum lipids, garlic, more importantly, appears to prevent the oxidation of LDL-cholesterol. Recall that LDL-cholesterol is considered to be a major contributor to atherosclerosis, and by decreasing it, garlic lowers the risk of cardiovascular disease [32]. Finally, garlic has been shown to significantly increase capillary skin perfusion (by 55%) in healthy subjects, when compared to controls not receiving garlic [33]. This basically means that garlic increases the amount of nutritive blood supplied to the skin by small blood vessels. This would also affect the scalp and the hair follicles. Note that all these studies were conducted using 900 milligrams or less of Kwai garlic tablets.

ASPIRIN

Aspirin has long been known to help people who already have vascular disease. It particularly helps those who have had or may have had a heart attack. Aspirin blocks one of the enzymes that causes platelets to aggregate. In this way, it can substantially lower the risk of vascular events (such as clot formation) from occurring in patients with established forms of vascular disease.

A key question is whether or not healthy individuals should use aspirin to prevent certain forms of vascular disease, such as heart attacks. Heart attacks are considered to be a form of vascular disease because the coronary arteries that supply the heart with oxygen and other nutrients often become narrowed or blocked. Currently, a study is testing the effectiveness of aspirin in preventing vascular disease in 40,000 healthy subjects. The results should be interesting.

One study has already shown that aspirin is less effective in people with high levels of cholesterol [34]. This makes a great deal of intuitive sense because we know that once cholesterol (LDL- cholesterol) is oxidized, it contributes to the production of an atherosclerotic plaque, which can lead to the narrowing of a blood vessel. The narrower a blood vessel, the easier it is for a clot to occur, and the less effective aspirin would be in preventing clot formation.

FIBER

Fiber has been demonstrated to decrease the total serum cholesterol levels in the bloodstream. One source of fiber proven to do this is psyllium [35]. There are many commercially available products that contain psyllium. Psyllium is able to bind both cholesterol and bile acids in the digestive system and "pull" them out of the body in the stool. It is probably due to its ability to lower cholesterol that fiber intake is inversely related to coronary heart disease [36].

In summary, individuals suffering androgenic alopecia have an increased risk of heart disease. Although this risk is not as significant as that of other factors contributing to heart disease, it does exist. For this reason, it is important to try to prevent heart disease from occurring. Several different ways to accomplish this have been discussed in this chapter. It may seem unfortunate that heart disease is associated with hair loss, but it would be even more unfortunate not to take the necessary steps to prevent it. The recommended doses of all the nutrients discussed in this chapter and in the chapter before it ("Nutrition and Hair") are listed in the last chapter of this book ("Review and Recommendations").

Further reading material
DEFINITIONS

androgenic alopecia: the medical term for male-pattern hair loss. It appears to be caused by a genetic predisposition of individual hair follicles to normal circulating levels of androgens (male sex hormones). Other terms for androgenic alopecia include male-pattern baldness, female baldness, common baldness, diffuse alopecia, hereditary alopecia, and, of course, baldness.

cardiovascular disease: a disease that affects the heart and the blood vessels.

myocardial infarction: medical term for a heart attack.

coronary arteries: the arteries that supply the heart with oxygen and other nutrients. When they become narrow or occluded, the heart may not receive enough oxygen, resulting in a heart attack.

infarct: an area of sudden cell death resulting from the sudden deprivation of arterial oxygen.

angina pectoris: a common type of chest pain, often described when the heart is deprived of oxygen.

atherosclerosis: the abnormal distribution of lipid deposits within arteries. Severe atherosclerosis can lead to a reduction in cross-sectional area of arteries, making blood flow and oxygen delivery to the heart very difficult.

carotid arteries: the arteries that supply the brain with oxygen. When they are occluded by atherosclerosis, they may not be able to supply the brain with the oxygen it needs. This can result in a stroke.

stroke: the lay term for the sudden and maintained change in brain function, which is usually related to its blood supply.

coronary artery disease: caused by the progressive narrowing of the coronary arteries, the arteries that supply blood to the heart. This results in arteries which are unable to supply the heart with the oxygen it requires.

lipoprotein: a substance that carries the cholesterol in the blood.

low-density lipoprotein (LDL): often referred to as "bad" cholesterol because, once oxidized, it can contribute to atherosclerosis. Its levels in the blood are directly related to the risk of coronary artery disease.

antioxidants: agents that trap free radicals and, thus, prevent the oxidation of LDL-cholesterol.

high-density lipoprotein (HDL): often referred to as "good" cholesterol because higher HDL-cholesterol levels in the blood are thought to prevent the formation of

atherosclerotic plaques and the coronary artery disease that results. HDL carries cholesterol away from the arteries to the liver, where it is broken down into bile salts and eliminated from the body via the stool.

vertex: the top or highest point of the head.

dihydrotestosterone: a more active and potent form of testosterone, usually produced at the hormone's site of action. The enzyme 5 alpha-reductase is absolutely necessary for its production.

saturated fats: the fats that contribute to heart disease.

unsaturated fats: fats that appear to be beneficial in preventing heart disease.

partially hydrogenated vegetable oil: a man-made modification of unsaturated vegetable oil, which is probably more dangerous to our health than saturated fats.

homocysteine: an amino acid found in the body. Its levels increase with meat consumption and directly correlate with the risk of heart disease. The risk of coronary artery disease increases with increasing plasma homocysteine levels, regardless of an individual's age or sex.

free-radical oxidative stress: the type of stress encountered when free radicals react with and oxidize components within the body. For example, free radicals can oxidize LDL-cholesterol.

free radicals: reactive, unstable molecules that occur in the body.

ubiquinone: another name for coenzyme Q10.

immunoglobulin G (IgG): is an antibody produced by our immune system.

T4-lymphocytes: a specific subset of white blood cells produced by our immune system.

AIDS: Acquired immunodeficiency syndrome. A chronic retroviral infection with human immunodeficiency virus (HIV) which produces severe, life-threatening defects in the immune system.

platelets: these are components within the blood that are essential in forming blood clots. By decreasing their ability to aggregate, their ability to cause a blood clot is also reduced. This is important because these clots often occlude blood vessels.

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