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High Cholesterol Levels and Other Secondary Risk Factors for Cardiovascular Disease

**Dr. Rath's Cellular Health Recommendations
for Prevention and Adjunct Therapy**

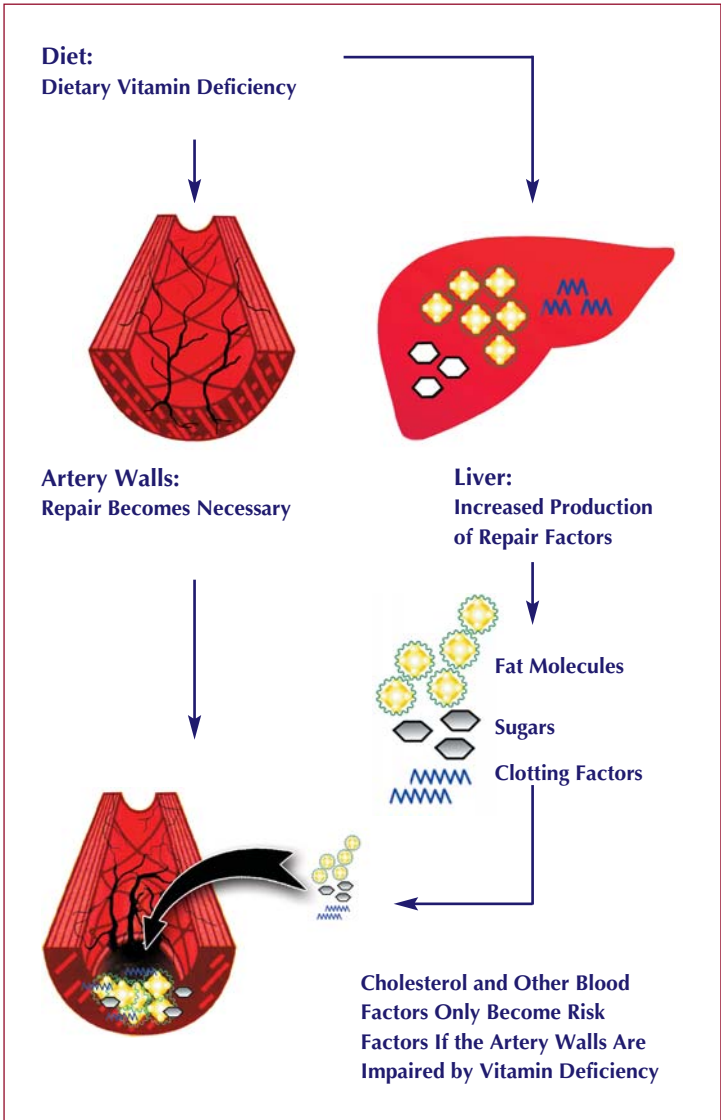
- **Cholesterol Is Only a Secondary Risk Factor**
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Cholesterol Is Only a Secondary Risk Factor

Worldwide, hundreds of millions of people have elevated blood levels of cholesterol, triglycerides, LDL (low-density lipoproteins), lipoprotein (a) and other risk factors. However, cholesterol and all other blood risk factors are considered only “secondary” risk factors because they can only cause damage if the the blood vessel wall is already weakened by vitamin deficiencies. Thus, elevated blood levels of cholesterol and other blood risk factors are not the *cause* of cardiovascular disease — they are the *consequence* of the ongoing vascular disease.

Conventional medicine, based on pharmaceutical drugs, is limited to treating the symptoms of cardiovascular disease while ignoring the root cause — blood vessel weakness. Marketing campaigns for cholesterol-lowering drugs simply proclaim cholesterol as the “scapegoat.” The latest type of these drugs (statins), which blocks the synthesis of cholesterol is being used by millions of people in the hope for treatment. However, the underlying weakness of the blood vessel wall continues untreated. According to the January 3, 1996 edition of the *Journal of the American Medical Association (JAMA)*, statins are known to cause cancer and other severe side effects, and “should be avoided whenever possible.”

Modern Cellular Medicine provides a new understanding about the factors causing the rise of cholesterol and other secondary risk factors, as well as their natural prevention. Cholesterol, triglycerides, low-density lipoproteins (LDL), lipoprotein (a) and other metabolic products are ideal repair factors, and their blood levels increase in response to a structural weakening of the artery walls. A chronic weakness of the blood vessel walls increases the demand and, thereby, the production rate of these repair molecules in the liver. An increased production of cholesterol and other repair factors in the liver increases the levels of these molecules in the bloodstream and, over time, renders them risk factors for cardiovascular disease. Thus, the primary measure for lowering cholesterol and other secondary



Elevated cholesterol levels are not the cause, but the consequence of cardiovascular disease.

risk factors in the bloodstream is to stabilize the artery walls and, thereby, decrease the metabolic demand for increased production of these risk factors in the liver. Therefore, it is not surprising that Dr. Rath's Cellular Health recommendations help to stabilize the artery walls and, at the same time, help to decrease blood levels of cholesterol and other risk factors naturally.

Cellular Medicine helps to expand the understanding about the different factors influencing one's personal risk factor profile. Your basic levels of cholesterol and other blood risk factors are genetically determined and cannot be changed. The two factors you can influence to lower your risk are diet and — above all — intake of specific essential nutrients that regulate cellular metabolism.

Scientific research and clinical studies have already documented the particular value of vitamin C, vitamin B3 (nicotinate), vitamin B5 (pantothenate), vitamin E and carnitine, as well as other components of Dr. Rath's Cellular Health recommendations, for lowering elevated cholesterol levels and other secondary risk factors in the blood.

Dr. Rath's Cellular Health recommendations comprise a selection of vitamins and other essential nutrients that help to normalize elevated levels of secondary risk factors. These essential nutrients lower the production rate of cholesterol and other repair molecules in the liver and, at the same time, contribute to the repair of the artery walls.

My recommendations for patients with elevated cholesterol and other secondary risk factors: lowering cholesterol without first stabilizing the artery walls is an insufficient and ill-fated cardiovascular therapy. Start as early as possible to increase the stability of your artery walls by following the recommendations in this book. As a consequence, blood levels of cholesterol and other risk factors will generally normalize. If you are on cholesterol or lipid-lowering medications, I encourage you to discontinue their use as soon as possible.

How Dr. Rath's Cellular Health Recommendations Can Help Patients With Elevated Cholesterol Levels

The following section presents letters from patients with cholesterol and other lipid disorders who have been helped by my Cellular Health recommendations. Please share this important information with friends and colleagues to enable them to lower their cholesterol levels in a natural way and to stop taking harmful cholesterol medication.

What You Should Do

- 1. Clear your mind of the belief that cholesterol causes heart disease.**
- 2. Stabilize your artery walls with Dr. Rath's Cellular Health recommendations.**
- 3. Eat more cereals, vegetables and other fiber-rich foods to "flush out" abundant cholesterol from your body naturally.**
- 4. Stop taking cholesterol-lowering medication!**

In most people who start following my recommendations, the blood levels of cholesterol and other risk factors in the blood soon decrease. We already know the reason for this effect; this essential nutrient program reduces the production rate of cholesterol and other secondary risk factors in the liver and, thereby, must lead to lower blood levels of this risk factor.

Interestingly, some patients report a transitory rise in cholesterol levels when they start taking vitamins. Because the rise in blood cholesterol levels is not the result of increased cholesterol production, it has to come from other sources — primarily atherosclerotic deposits in the artery walls. This important

mechanism was first described by Dr. Constance Spittle in the medical journal *The Lancet* in 1972. She reported that vitamin supplementation in patients with existing cardiovascular disease frequently led to a temporary increase of cholesterol levels in the blood. In contrast, the cholesterol levels of healthy test persons did not rise with vitamin supplementation.

The temporary rise in cholesterol is an additional sign of the healing process in the artery walls and the decreasing of fatty deposits. The mechanism described here is, of course, not only valid for cholesterol, but also for triglycerides, LDL, lipoprotein (a) and other secondary risk factors, which have accumulated over decades inside the artery walls and have been slowly released into the bloodstream.

My recommendations in this case: Should your cholesterol levels rise when you start following these recommendations, it can indicate the reversal of existing deposits in your artery walls. You should continue the vitamin program until, after several months, the blood level of cholesterol decreases below the initial values. A diet high in soluble fiber (e.g. oat bran, cereals and pectins) can further decrease cholesterol and other secondary risk factors in the blood.

The following letters document the rise and subsequent decrease of cholesterol in patients following Dr. Rath's Cellular Health recommendations:

Dear Dr. Rath:

*I had started taking a fiber product in February of 1994. **My cholesterol continued to climb from 280 to over 320 until May of 1994, when I began to follow your recommended vitamin program.***

***My cholesterol has dropped to 180 and my ratio of HDL to LDL is normal, as is my triglyceride level.** Most important, however, my lipoprotein (a) dropped from 15 to 1! I will continue your program forever.*

Thank you, Dr. Rath, for your work with natural therapies as a means for decreasing the risk of heart disease.

*With much gratitude,
M.R.*

Dear Dr. Rath:

*I am 45 years old, and since December of last year I have been on your program of essential nutrients. I also take a fiber formula. **Last April, my cholesterol level was 259. This April, after only 4 months on this program, my cholesterol dropped to 175!***

Dr. Rath, I truly want to thank you for helping me to be healthier and live a much fuller life.

*Sincerely,
M.W.*

Dear Dr. Rath:

*Heart disease is hereditary within my family, and my father had his first heart attack in his early 30s. I had my cholesterol checked at age 19 only to find out that **I had a cholesterol level of 392 mg/dl**. My physician did not want to place me on medication at that time, so I just watched my diet and increased my exercise. Well, as time passed, my cholesterol remained elevated, and my physician felt medication was necessary. I refused to begin medication and continued with diet and exercise.*

*At age 26, I had my cholesterol tested before I began your vitamin program, and my lab test showed a reading of 384. I immediately began following your program, with a fiber drink, and my level dropped 120 points within a 6-10 week period. **Over a four-month period, my LDL went from 308 down to 205.** This is a program that I personally follow, and continue to have positive results.*

I recommend it to my family and friends.

*Sincerely,
C.C.*

Dear Dr. Rath:

I began taking a fiber formula two years ago in September. My total cholesterol was around 177 at that time. Within 90 days, I lost 20 pounds and my total cholesterol dropped to 154.

In November last year, I started with your vitamin program. An insurance physical that was done in February of this year showed a total cholesterol (CHOL) level of 191, a triglyceride level of 244, a LDL/HDL ratio of 4.09 and a CHOL/HDL ratio of 6.8, all which were elevated. Again, note that this was in February.

*A cholesterol screening was done in March and again in June. Both showed a total cholesterol level of 134. **A lipid profile that was done in July showed a total cholesterol level of 135, a triglyceride level of 180, a LDL/HDL ratio of 1.47 and CHOL/LDL ratio down to 3.16 from 6.8.***

Your cardiovascular health program is working!

*Sincerely,
L.M.*

Clinical Studies With Dr. Rath's Cellular Health Recommendations Document Their Effectiveness in Lowering Blood Risk Factors

The effect of vitamin C on the blood levels of cholesterol and other blood fats has been documented in numerous clinical studies. More than 40 of these studies have been evaluated by Dr. Harrie Hemilä of the University of Helsinki, Finland. In patients with high initial cholesterol values (above 270 mg per deciliter), vitamin C supplementation was able to decrease cholesterol levels up to 20%. In contrast, patients with low and medium initial values of cholesterol showed only a slight cholesterol-lowering effect or the levels stayed the same.

In a study sponsored by the American Heart Association, Dr. B. Sokoloff showed that two to three grams of vitamin C per day could lower triglyceride blood levels on average by 50% - 70%. It was shown that vitamin C increased the production of enzymes (lipases) able to degrade triglycerides and lower triglyceride levels.

Dr. Jacques and his colleagues showed that people taking 300 mg of vitamin C per day also had much higher HDL blood levels than people taking less than 120 mg per day. This is particularly important since HDL (high-density lipoproteins) are fat-transporting molecules that can pick up cholesterol and other fats from the artery walls and carry them back to the liver for removal. This is yet another way vitamin C can help reduce atherosclerotic deposits and reverse cardiovascular disease. Dr. W.J. Hermann and his colleagues reported that vitamin E supplementation also increases HDL blood levels.

Further clinical studies show that other components of Dr. Rath's Cellular Health recommendations work synergistically with vitamin C in lowering cholesterol and other blood fats. These components include vitamin B3 (nicotinic acid), vitamin B5 (pantothenate), vitamin E, carnitine and other

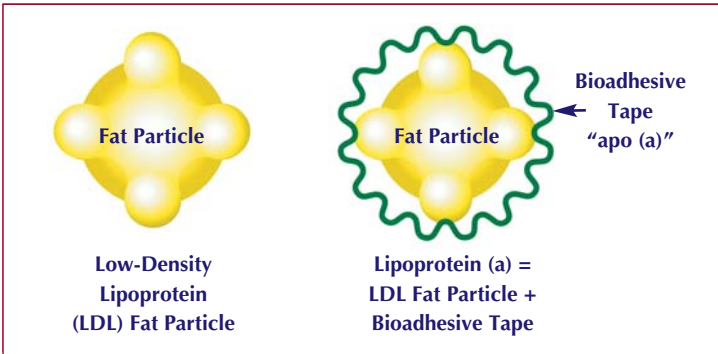
essential nutrients. This synergistic effect is an important advantage over megadose intake of individual vitamins.

Cellular Nutrients Tested	Reference
Vitamin C	Ginter, Harwood and Hemilä
Vitamin B3	Altschul, Carlson and Guraker
Vitamin B5	Avogaro, Cherchi and Gaddi
Vitamin E	Beamish and Hermann
Carnitine	Opie

Lipoprotein (a) — A Secondary Risk Factor Ten Times More Dangerous Than Cholesterol

Now I would like to introduce you to a particularly important secondary risk factor, lipoprotein (a). The genuine function of lipoprotein (a) is very useful; it fulfills a variety of repair functions, for example, during wound healing. However, if the artery wall is destabilized by a long-term vitamin deficiency, lipoprotein (a) turns into a risk factor 10 times more dangerous than cholesterol. Let's take a closer look at how lipoprotein (a) molecules differ from other fat molecules.

Cholesterol and triglycerides do not float in the blood in the way that fat floats in soup. Thousands of cholesterol molecules are packed together with other fat molecules in tiny round globules called lipoproteins. Millions of these fat-transporting vehicles circulate in our bodies at any given time. The best known among these lipoproteins are high-density lipoproteins (HDL, or "good cholesterol") and low-density lipoproteins (LDL, or "bad cholesterol").



Comparison between LDL and lipoprotein (a)

LDL cholesterol: Most of the cholesterol molecules in the blood are transported in millions of LDL particles. By carrying cholesterol and other fat molecules to our bodies' cells, LDL is a very useful transport vehicle for supplying nutrients to these cells. LDL has been named "bad cholesterol" because, until recently, researchers believed that LDL was primarily responsible for the fatty deposits in the artery walls. This understanding is now out of date.

What Are the Facts About Lipoprotein (a)?

- Lipoprotein (a), not LDL, is the most important fat particle responsible for the deposit of cholesterol and other fats in the artery walls.
- Because of its sticky properties, lipoprotein (a) is one of the most effective repair molecules in the artery wall and, with ongoing vitamin deficiency, becomes one of the most dangerous risk factors for atherosclerosis and cardiovascular disease.
- A re-evaluation of the Framingham Heart Study, the largest cardiovascular risk factor study ever conducted, showed that lipoprotein (a) is a tenfold greater risk factor for heart disease than cholesterol or LDL cholesterol.

Lipoprotein (a) is an LDL particle with an additional adhesive protein surrounding it. This biological “adhesive tape” is named apoprotein (a) or, apo (a). The letter (a) could, in fact, stand for “adhesive.” The adhesive apo (a) makes the lipoprotein (a) fat globule one of the stickiest particles in our bodies.

In a Vitamin-Deficient Body, Lipoprotein (a) Becomes the Most Important Secondary Risk Factor for:

- Coronary Heart Disease and Heart Attacks
- Cerebrovascular Disease and Strokes
- Restenosis (Clogging) After Coronary Angioplasty
- Clogging of Bypass Grafts After Coronary Bypass Surgery

Together with my colleagues at Hamburg University, I conducted the most comprehensive studies on lipoprotein (a) in the artery wall. These studies showed that the atherosclerotic lesions in human arteries are largely composed of lipoprotein (a) rather than LDL molecules. Moreover, the size of the atherosclerotic lesions paralleled the amount of lipoprotein (a) particles deposited in the arteries. In the meantime, these findings have been confirmed in a series of additional clinical studies.

Lipoprotein (a) blood levels vary greatly between one individual and another. What do we know about the factors influencing the lipoprotein (a) levels in the blood? Lipoprotein (a) levels are primarily determined by inheritance. Special diets do not influence lipoprotein (a) blood levels. Moreover, none of the presently available lipid-lowering prescription drugs lower lipoprotein (a) blood levels.

The only substances that have, thus far, been shown to lower lipoprotein (a) levels are vitamins. Professor Carlson showed that two to four grams of vitamin B3 (nicotinic acid) a day could lower lipoprotein (a) levels up to 36%. Because high levels of nicotinic acid can cause skin rashes, you are advised to increase the daily intake of nicotinic acid gradually. Our own

research has shown that vitamin C alone or in combination with lower dosages of nicotinic acid may also have a lowering effect on the production of lipoproteins, and thereby, lower lipoprotein blood levels. Together with the “Teflon” agents lysine and proline, these two vitamins can considerably decrease the cardiovascular risk associated with lipoprotein (a) levels.

Therapeutic Approaches to Reduce the Risk From Lipoprotein (a)

1. Lowering of Lipoprotein (a) Blood Levels
 - Vitamin B3 (Nicotinate)
 - Vitamin C
2. Decreasing Stickiness of Lipoprotein (a)
 - Lysine
 - Proline

Lipoprotein (a) is a particularly interesting molecule because of its inverse relationship to vitamin C. The following discovery triggered my interest in vitamin research: lipoprotein (a) molecules are primarily found in humans and in a few animal species unable to produce vitamin C. In contrast, animals able to produce optimum amounts of vitamin C do not need lipoprotein (a) in any significant amount. Lipoprotein (a) molecules apparently compensate for many properties of vitamin C, such as wound healing and blood vessel repair. In 1990, I published the details of this important discovery in the *Proceedings of the National Academy of Sciences* and cited Dr. Linus Pauling as co-author of this publication.

The Cholesterol – Heart Disease Fallacy

While reading this section, you may have asked yourself the questions: “But what about cholesterol? Are those reports about cholesterol only media hype?” Unfortunately, this is the case. Here are some of the sobering facts:

The leading medical speculation about the origin of cardiovascular disease is as follows: high levels of cholesterol and risk factors in the blood damage the blood vessel walls and lead to atherosclerotic deposits. According to this hypothesis, lowering cholesterol is the primary measure to prevent cardiovascular disease. Tens of millions of people worldwide are currently taking cholesterol-lowering drugs with the expectation that they will help fight cardiovascular disease. The marketing propaganda behind these cholesterol-lowering drugs is worthy of a closer look.

In the 70s, the World Health Organization (WHO) conducted an international study to determine whether cholesterol-lowering drugs could decrease the risk for heart attacks. Thousands of study participants received the cholesterol-lowering drug “Clofibrate.” This study could not be completed because those people who took the cholesterol-lowering drug experienced too many side effects. Thus, in the interest of the health and lives of the study participants, this cholesterol-lowering drug study had to be called off.

In the early 80s, a large-scale study in more than 3,800 American men made headline news. This study tested whether the cholesterol-lowering drug “Cholestyramine” could lower the risk for heart attacks. One study group took up to 24 grams (24,000 mg) of Cholestyramine every day over several years. The control group of this study took the same amount of a placebo (ineffective control substance). The results of this study were that in the cholesterol-lowering drug group, the same number of people died as in the control group. Particularly frequent among those patients taking this cholesterol-lowering

drug were accidents and suicides. Irrespective of these facts, those interested in marketing the drug decided to promote this study as a success. The fact that in the drug group there were slightly fewer incidences of heart attacks was marketed as a confirmation of the cholesterol-heart attack hypothesis. Few people bothered with the actual death figures of this study.

In the late 80s, a new group of cholesterol-lowering drugs was introduced, which was shown to decrease the production of cholesterol in the body. Soon thereafter, it was determined that these drugs not only lowered the production of cholesterol in the body, but also lowered the manufacture of other essential substances, for example, ubiquinone (coenzyme Q-10). Karl Folkers, MD, of the University of Texas at Austin, rang the alarm bells in the *Proceedings of the National Academy of Sciences*. Dr. Folkers reported that patients with existing heart failure who took these new cholesterol-lowering drugs could experience life-threatening deterioration of their heart function.

A giant blow for the cholesterol-lowering drug industry came on January 6, 1996. On this day, the *Journal of the American Medical Association* published an article entitled “Carcinogenicity of Cholesterol-Lowering Drugs.” Dr. Thomas Newman and Dr. Stephen Hulley, of the University of California, San Francisco Medical School, showed that most of the cholesterol-lowering drugs on the market were known to cause cancer in test animals at levels currently prescribed to hundreds of thousands of people. The results from this article were so alarming that the authors raised the legitimate question: “How could it be that the regulatory agency, the U.S. Food and Drug Administration (FDA), allowed these drugs to be sold to millions of people?” The answer given by the authors of this study: “The pharmaceutical companies manufacturing these drugs downplayed the importance of these side effects and, thereby, removed any obstacles for their approval.”

The publication of the first edition of this book in 1993 explained for the first time to a broad audience that animals don't get heart attacks because they produce enough vitamin C, not because they have low cholesterol levels. Heart attacks are the primary result of vitamin deficiencies — not elevated cholesterol. It was immediately clear that cholesterol-lowering drugs, beta-blockers, calcium antagonists and many other pharmaceuticals would eventually be replaced by essential nutrients in eliminating cardiovascular disease.

The time needed to reach this goal would be dependent on one single factor only: how fast the knowledge about the connection between scurvy and cardiovascular disease could be spread. The manufacturers of cardiovascular drugs knew that they would lose a drug market worth trillions of dollars over time. This multi-trillion dollar global market of symptom-oriented drugs will inevitably collapse once millions of people learn that vitamins and other essential nutrients are the answer to the cardiovascular disease epidemic.

This is the background of why the pharmaceutical industry is spending hundreds of millions of dollars fighting the natural Cellular Medicine alternative and advertising drugs that do not cure, but cause new diseases such as cancer.

Why Bears Are Not Extinct

If anyone among my readers still thinks that cholesterol may cause heart attacks, I would like to share the following facts: Bears, and millions of other hibernating animals, have average cholesterol levels of over 400 mg per deciliter. If cholesterol were indeed the culprit causing heart attacks and strokes, bears and other hibernating animals would have long ago become extinct as a result of heart attacks. The reason why bears are still among us is simple — they produce high amounts of vitamin C in their bodies, which stabilize their artery walls so they are unaffected by cholesterol.

The fact that bears are not extinct proves:

1. Elevated cholesterol blood levels are not the primary cause of atherosclerosis, heart attacks and strokes.
2. Achieving and maintaining stability of the artery walls through an optimum vitamin supply is more important than lowering cholesterol and other risk factors in the bloodstream.
3. Cholesterol and other repair factors in the bloodstream can only become risk factors if the artery walls are weakened by chronic vitamin deficiency.



Cellular Health Recommendations for Patients With High Cholesterol and Other Metabolic Disorders

In addition, to my Basic Cellular Health recommendations (page 25), I recommend that patients with elevated cholesterol levels and other metabolic disorders take the following cellular bioenergy factors in higher dosages:

- **Vitamin C:** for the protection and natural healing of the artery walls, lowering increased production of cholesterol and other secondary risk factors in the liver and reducing elevated blood levels of these secondary risk factors
- **Vitamin E:** for antioxidant protection of blood fats and millions of cells
- **Vitamin B1:** for optimizing cellular metabolism and, particularly, for the delivery of bioenergy
- **Vitamin B2:** for optimizing cellular metabolism and, particularly, for the delivery of bioenergy
- **Vitamin B3:** for lowering the elevated production of cholesterol and lipoproteins in the liver
- **Vitamin B5:** for the structural component of the central metabolic molecule of cells (coenzyme A) and optimal metabolic burning of fat molecules
- **Vitamin B6, Biotin and Folic Acid:** for counteracting increased levels of the risk factor *homocysteine* and optimizing the metabolism of cells
- **Carnitine:** for optimizing cellular metabolism of fats and lowering triglyceride levels

Notes