The cholesterol problem and its treatment

Alan E. Baklayan, Naturopath, Munich

INTRODUCTION

Ladies and gentlemen,

By chance, a well-known journalist came to me for treatment. Amongst other things, his cholesterol level was high. However, once he had undergone a course of liver cleansing, this completely returned to normal.

When the scandal of the cholesterol-lowering agent "Lipobay" broke soon afterwards, I was asked by his editors if I would like to write a book on the subject. The result was published under the title "Cholesterin: Schock und die Alternative: Die Baklayan-Methode" [Cholesterol: shock and the alternative: the Baklayan method].

WHAT EVERYONE SHOULD KNOW ABOUT CHOLESTEROL

In recent decades, cholesterol as a product of foods of animal origin absorbed in the daily diet, has been branded a scapegoat for every conceivable cardiovascular disease. In reality, however, some of the cholesterol found in the blood is produced in the liver. The body synthesises a total of 2-3 g cholesterol per day of which around 1 g is attributable to the liver. It is required in the production of bile acids and various hormones and therefore represents an essential *(vital)* substance. The remainder is supplied via our food, mainly in animal fats. However, it is not true, as stated in numerous publications, that the cholesterol level rises simply because we eat too many foods of animal origin. Cholesterol synthesis is subject to certain regulating influences. However, these can be inhibited through cholesterol absorbed with the food. In other words, cholesterol synthesis is subject to a so-called "negative feedback mechanism", which, in simple terms, functions as follows:

- **More** cholesterol in the diet means that less is produced in the body.
- **Less** cholesterol in the diet stimulates the body to increase cholesterol production.

As a result, blood cholesterol levels cannot really be reduced solely using foodstuffs which are low in cholesterol. If we alter our diet, by consuming less meat for instance, this can only ever be a supporting measure and not a form of therapy in its own right!

CONTRADICTORY CLAIMS

I cannot claim responsibility for this discovery. The diet recommended by the "National Cholesterol Initiative" contains

- carbohydrates: 50–60 % of calories
- fats: not more than 30 % of calories, of which one third each as saturated, monounsaturated and polyunsaturated fatty acids
- cholesterol: maximum 300 mg / day
- fibre approx. 35 g / day

Although this diet is very difficult for ordinary consumers to calculate correctly and even harder to adhere to, it is recommended as highly effective. At least according to the "National Cholesterol Initiative" and you can read up on it in Deutsches Ärzteblatt 87, B 991 (1990).

It reaches the following conclusion and I quote: "Thereby, cholesterol levels can be reduced by 15–20 % on average."

However, this contradicts the scientific results of studies into the long-term effects of these diets. Thus we read in the universally respected British Medical Journal (L. E. Ramsay, Dietary reduction of serum cholesterol concentration: time to think again, Br. Med J. 303, 953 (1991)):

Colloquium staged by the International Medical Working Group *BICOM* Resonance Therapy and *BICOM* Resonanz-Therapie-Gesellschaft from 3 to 5 May 2002 in Fulda

"Despite considerable efforts as regards patient education and dietary advice and adherence to the recommended diet for several years, on average only a 2 % reduction in the cholesterol level was achieved!"

And it goes on:

"A 15-20 % reduction was only attainable with a far stricter diet than that recommended and then only in hospitals or psychiatric units!"

These claims are based on the findings of ten long-term studies in various countries. The World Health Organisation (WHO) also came to a similar conclusion.

So for the record: our bodies have a self-regulating mechanism which reacts to a lower cholesterol intake from outside by increasing production. This also applies to so-called low cholesterol foods, such as low-cholesterol eggs, which the food industry is constantly serving up.

Increased intake from external sources does not necessarily lead to higher cholesterol levels as endogenous production is driven down. This is repeatedly portrayed differently by certain publications yet you can easily see for yourselves that we would not need cholesterol-lowering agents if the same effect could be achieved simply by changing diet. Then it would not be the pharmaceutical companies who were earning billions with their cholesterol-lowering drugs but possibly the tomato, garlic and artichoke growers. I believe the conflicting scientific findings are generally due to insufficient test periods. The human body requires around four weeks to adapt to a change in circumstances. In practice this means that simply changing diet for around four weeks produces a temporary drop which then rises just as quickly to virtually the same level as at the start of the changeover due to the negative bio-feedback mentioned earlier.

Probably the most important enzyme in the body's synthesis of cholesterol is HMG CoA reductase. It determines the rate of synthesis. Modern cholesterol-lowering agents can also be referred to as "cholesterol synthesis enzyme inhibitors" as they inhibit the activities of this very enzyme. This reduces the body's synthesis of cholesterol leading to a clearly measurable reduction in the cholesterol level in the blood.

We must study cholesterol theory in more detail to grasp the exact processes involved.

WHAT EXACTLY IS CHOLESTEROL?

Cholesterol – essential yet at the same time dangerous

The human body needs cholesterol for a number of vital functions.

- The bile acids required to digest fats are manufactured from cholesterol
- Cholesterol is vital for synthesising new tissue.
- Cholesterol is an essential component of every cell in the body.
- Cholesterol is essential for synthesising important hormones, particularly the sexual hormones testosterone (in the testes), oestrogen and progesterone (in the ovaries).
- A precursor to vitamin D is manufactured with the aid of cholesterol.

So it is clear that cholesterol is an absolutely essential substance without which our bodies could not function, in fact they could not even synthesise properly.

On the other side, cholesterol sits in the dock, accused of numerous damaging and even lethal effects. If the cholesterol level in the blood is too high for a prolonged period of time, the risk of arteriosclerosis (also spelt *atherosclerosis*) increases considerably. Too much cholesterol in the blood leads to deposits on the walls of the vessels which supply the heart with the blood it needs. This particularly affects points where the internal lining of the artery (known as the *endothelium*) is no longer completely intact. This narrows the vessels even more, impairing the blood flow. The more deposits form, the narrower the space through which the blood can flow. Circulatory disturbances occur, arteriosclerosis begins.

Sometimes deposits break away from the vessel walls and are carried away in the blood. This then leads to the accumulation of a blood clot and a risk of infarction. If tissue is not supplied with blood for prolonged periods, it is damaged and may ultimately become completely necrotic.

Since cholesterol-related deposits occur particularly frequently in the coronary vessels responsible for the blood supply to the heart, they are the most dangerous risk factor for so-called coronary heart disease. These often take the form of angina pectoris attacks and, in the final analysis, lead to myocardial infarction, a condition quite rightly feared by everyone. These are, in my opinion, the main points of the charge which is repeatedly laid

before us with finger raised and which serves to justify the millions of cholesterol-lowering drugs prescribed. Yet let us examine this in more detail:

Mistakes made when assessing cholesterol

For decades cholesterol was measured only as a total value in the blood. As was later discovered, this led to completely false interpretations as no distinction was made between the various lipoproteins.

For it is not actually the cholesterol, which has such a positive and also negative influence on our bodies, but the lipoprotein ratio in the substance. The **cholesterol hypothesis** tells us:

> So-called LDL receptors are located on the surface of the cells of the liver. They remove from the blood the LDL (*Low Density Lipoprotein*), one of the lipoproteins containing cholesterol which we shall explain in more detail later, circulating in the blood. If the LDL ratio is particularly high and the number of LDL receptors correspondingly low, this causes arteriosclerosis. So it is not cholesterol as a complete substance which is responsible for this disorder but the lipoproteins it forms part of and especially the ratio between the different types.

The lipoproteins and their role

The cholesterol in the blood is surrounded by a lipoprotein shell. These fat and protein compounds have an important role. The lipoprotein shell enables the cholesterol to be carried in the blood as the lipid substance is extremely insoluble.

Lipoproteins are classified and named according to their density. The most important here are the low density lipoproteins, particularly VLDL (*Very Low Density Lipoproteins*), LDL (*Low Density Lipoproteins*) and high density HDL (*High Density Lipoproteins*)**.**

As mentioned earlier, one of the roles of lipoproteins is to transport the lipids in the blood.

So-called "good" HDL

To put it simply, HDL transports the cholesterol away from the body's cells and back to the liver. There the cholesterol is converted into bile acids. These are then in turn delivered to the intestines. Some of the bile acids are excreted there largely by bonding to dietary fibre. Some travel back to the liver. In simple terms, therefore, HDL can be said to dispose of cholesterol. In disposing of cholesterol through the blood stream, HDL also dissolves the deposits on the vascular walls, thus contributing to **reducing the risk of arteriosclerosis** mentioned earlier. However, it is not, as is often erroneously suggested, the only substance responsible.

So-called "bad" LDL

LDL is responsible for transporting the cholesterol from the liver into the cells of the body, as is the even lower density VLDL. There cholesterol is processed further and absorbed. Certain reception points in the cells, known as LDL receptors, are responsible for absorbing cholesterol. They operate like doors and the LDL has the correct "key" to open them. Only then can a cell admit the lipid. If the number of LDL receptors drops or if they are not capable of being "opened", the LDL ratio in the blood increases. The associated risks for the person concerned also increase considerably. Lipometabolic disorders can also lead to an increased LDL ratio.

However, not all LDL is the same

If it was assumed some years ago that LDL was a substance whose action could be measured as uniform, it was recently discovered that there are lipoproteins with even lower density, so-called VLDLs, whose effect is even more dangerous.

On average the total cholesterol value consists of:

- HDL cholesterol: 25 %
- VLDL cholesterol: 15 %
- LDL cholesterol: 60 %

Consequently, a high cholesterol value is frequently, but not always, synonymous with a high LDL ratio.

Therefore, when the VLDL and LDL ratio increases as the cholesterol level rises yet the HDL ratio does not change noticeably, only then does this composition signify an increased risk. This is the current state of the cholesterol hypothesis and the charges constantly raised in relation to cholesterol are seen as directly connected and providing proof of this hypothesis. In other words: increasing the LDL/VLDL ratio increases the risk of myocardial infarction and arteriosclerosis. However

this **direct connection** is now also being called into question in numerous publications:

Further contradictions

A 60 % decline in cardiac mortality has been observed in the USA in recent decades. Yet cholesterol levels have only fallen by around 3 %. Even taking other possible factors into account, such as the drop in the number of people smoking and therapeutic measures, it is hard to explain this discrepancy.

A second point to consider: when compared, there is very little difference between the cholesterol values of patients with myocardial infarction and healthy subjects (cf Klepzig. Ztschr. Kardiologie 81, 347, 1992, in Gothenburg/Sweden).

The most important study was probably that carried out on twelve different European population groups (F. K. Gey, American Journ. Clin. Nutrition 53, 326 S (1991), in which no link could be established between cholesterol values and heart disease.

Certainly the so-called CARE study (also known as the 4S study) of high risk heart patients established that, at very high cholesterol levels (mean values: total cholesterol 260 mg %, LDL cholesterol 188 mg %), only a slight drop in fatal and non-fatal myocardial infarction was recorded and no effect was observed on patients with an LDL level below 125 mg %. When one considers that this study investigated the action of cholesterol-lowering agents using statins, then this result is remarkable at the very least.

If this is all highly consistent, one must ask oneself with some irritation: how can the problem originate at the vascular walls?

Lp(a) – a link hitherto largely ignored yet so important

There is also another protein, which is a lot less well known to the general public than the aforementioned lipoproteins. Nevertheless, it plays an important part in cholesterol levels: apoprotein.

When apoprotein, or apo-a for short, combines with LDL, this produces a lipoprotein known as Lp(a), expressed by the formula:

LDL + apo-a = Lp(a) .

By virtue of its structure, apo-a can be regarded as a type of adhesive with which Lp(a) can adhere to the cell walls. This results in dangerous narrowing of the arteries since LDL is firmly attached to the insides of the arteries by apo-a. This process also has the obvious desirable effect of sealing defective areas in the cell walls. However, if too much material accumulates, the arteries become narrower and even completely blocked with the dreaded risk of infarction.

As various scientific studies have now proved, an increased Lp(a) level is a health risk ten times greater than a high total cholesterol value or a high LDL value.

No direct link between Lp(a) and the other fats in the blood has so far been unequivocally proven. In practice this means that a person can have a **completely normal cholesterol level** and **yet an increased Lp(a) value**. The risk of arteriosclerosis and infarction is considerably increased however. If the two factors are combined, increased LDL ratio and high Lp(a) values, the risk of illness is significantly increased.

ADDITIONAL VERY IMPORTANT FACTORS

According to orthomolecular medicine, the following factors play an important part in vessel damage:

1. **Antioxidants**

   According to this view, LDL does not, in itself, produce a harmful effect, only oxidised LDL. However, oxidation can be counteracted medically with so-called antioxidants. In our case it would be vitamins which produce this effect. Vitamins C, E, betacarotene, in particular, and also selenium have so far proved the best for this in practice.

   New:

   Moreover, it has been observed in experiments on animals that chronic vitamin C or E deficiency leads to arteriosclerosis in monkeys and guinea-pigs. Also, in studies of various population groups, a low blood level in vitamins C, E and betacarotene was judged a far higher risk factor for the onset of heart disease than high cholesterol.

2. **Homocysteine**

   Homocysteine is a catabolic product of the essential amino acid methionine and is a highly

toxic substance. The body needs enzymes to bring about this catabolism. These enzymes, in turn, need cofactors to operate optimally, particularly vitamin B6, B12 folic acid, B2 and vitamin C.

3. Omega-3 fatty acids

There are numerous studies on different population groups which reveal a clear link between increased intake of omega-3 fatty acids (in the form of fish) and a statistically relevant reduction in mortality from heart disease compared with the central European area. Various clinical studies have established that omega-3 fatty acids have the following effect: reduction in blood pressure, blood viscosity, blood fats (not only cholesterol and triglycerides but also lipoproteins) and reduction in dysrhythmia.

It would not be appropriate in this paper to highlight all the scientific data relating to oxidation processes, homocysteine catabolism and the effect of omega-3 fatty acids. (See bibliography.) However, it is important for all those affected to be aware of these conclusions from orthomolecular medicine in order to understand how important it is, for example, to take a vitamin preparation alongside the course of treatment I prescribe and why this preparation should continue to be taken after completion of the course of treatment (Dermavital, see footnote on page 1).

One might wonder: can the solution to the problem be that simple? Perhaps one might question whether "simply taking" a vitamin mixture can be a solution and preventive measure for such serious diseases. However, it should be remembered that, over the centuries, diseases such as scurvy and beriberi have claimed hundreds of thousand of victims. Finally, resourceful people discovered that scurvy was attributable to a simple vitamin C deficiency caused by a lack of fresh fruit and vegetables and that beriberi was due to vitamin B1 deficiency through the consumption of husked or polished rice as the staple food in Eastern Asia and Japan (thiamine = B1 is concentrated in rice husks).

Let us now return to the negative effects of Lp(a) mentioned earlier. While the lipid-lowering drugs so far available do not appear to reduce Lp(a) levels, the ability of natural substances such as vitamin C, B3 (known as *nicotinamide*) or omega-3 fatty acids to lower levels is well-known. Moreover, many scientists are now already assuming that the permanent change in our diet over the past thirty or forty years may be partly responsible for the ever-increasing problem of arteriosclerosis and risk of infarction. The proven fact that our diet has only 10 % of the vitamin C content of that 30 years ago is particularly significant.

Numerous studies reveal that evidence of Lp(a) has so far only been measured in human blood and that of primates and guinea-pigs. On more detailed examination we see that these are species which are unable to produce vitamin C themselves and must therefore absorb it in food. This further supports the theory of increased risk through a change in diet to one containing less vitamin C.

If we consider all the health recommendations issued to reduce the risk of infarction and arteriosclerosis, then people who practice sport, are the correct weight, eat healthily, do not smoke, have a completely normal cholesterol level and unaffected by other risk factors, should never suffer from arteriosclerosis or infarction. Yet, even amongst these people who lead an "ideal" lifestyle, there are many who are ill and who have died from infarction. A high Lp(a) value offers an explanation for these cases which could not be explained by previous theories.

Vitamin C, in particular, is an essential substance which is vital, for example, for the effective synthesis of the proteins collagen and elastin. As connective tissue these proteins are crucial for stabilising the internal walls of our blood vessels. However, vitamin C is consumed when these proteins are synthesised. Consequently, vitamin C deficiency leads to impairment of the vessel walls. **Lp(a) can thus be described as a substitute for deficient vitamin C.**

Consequently, vitamin C, which incidentally should be used in its natural form and not as ascorbic acid, is a particularly important component for supporting my therapy.

Cholesterol and parasites

I kept noticing in my tests that, very often, ascarides and ascaris larvae could be tested on the liver and gallbladder meridian when cholesterol levels were high. Moreover, infestation with lamblia intestinale, lamblia trophozoit and lamblia cysts often tested with lipometabolic disturbance. I mentioned this at the first parasite seminar back in 1999. I found an explanation in various studies which prove that ascarides and other parasites need a cholesterol-rich environment in which to develop.

Survey of vitamins required

Orthomolecular substance	Effect on heart, circulation and vessels
Vitamin B1	optimises cell function, strengthens immune system
Vitamin B2	lowers homocysteine, optimises cell function
Vitamin B6	see vitamin B2
Vitamin B12	lowers homocysteine, important for blood production
Vitamin B3 = nicotinamide	lowers lipoproteins (a), LDL cholesterol and also total cholesterol
Vitamin C, natural	lowers lipoprotein (a), lowers homocysteine, antioxidant, stabilises blood vessels, regulates lipometabolism
Folic acid	lowers homocysteine, important for blood production
Pantothenic acid	regulates lipometabolism, optimises cell function
Betacarotene	antioxidant
Selenium	antioxidant, optimises cell function

All these components are contained in the combined preparation Dermavital*. This is the first remedy manufactured by machines which have not been previously cleaned with propan-2-ol.

Omega-3 fatty acids	regulate blood clotting, reduce blood adhesion, reduce blood pressure, lower lipoprotein (a), reduce blood fats

It is even suspected that various parasites themselves excrete cholesterol.

THE NATUROPATHIC VIEW

I call it the "cool" liver approach to combating cholesterol. Yet this is nothing new, simply an old proven technique used in naturopathy which has been rediscovered.

In naturopathic circles an increase in cholesterol in the blood is quite simply the result of the liver overheating. This can be conceived as follows:

Years of eating denatured foods and excessive consumption of carbohydrates – and not consumption of fats as is often falsely claimed – leads to the liver, which is responsible for detoxifying and excreting the resulting waste products, being constantly overworked.

As the flood of waste products never stops, some are simply put on one side to be processed later, particularly in people with a physical predisposition to this. This results in the liver becoming increasingly clogged, which in turn encourages the accumulation of parasite and bacterial corpses. Like a machine which operates constantly at high revs, the liver gradually heats up, what the old naturopaths used to call the "liver overheating".

The blood is no longer detoxified adequately and remains charged with waste products, excess acids and environmental toxins. It "thickens" and irritates the vessel walls. The consequent action for every patient with this tendency is actually self-evident:

1. carry out major cleansing of the liver
2. kill all parasites through BICOM therapy and a course of parasite treatment
3. improve the flow properties of the blood (using Dermavital* and zapping*, if necessary)
4. stabilise the vessel walls with orthomolecular support (Dermavital*).

The good news is that "any patient" can themselves carry out a course of treatment which includes the above four steps within a short time quite simply using appropriate measures. This simple method is successful in over 90 % of cases, even with very high cholesterol levels and no medication is necessary. In rare cases this method does not succeed with organ damage and hereditary metabolic disorders. The steps are clear and simple and can be carried out by anyone with no outside help. The course of treatment takes between 6 and 12 weeks and with your "cool" liver you will also

* Dermavital vitamins, zapper, the book "Cholesterin – Schock und die Alternative: Die Baklayan-Methode" [Cholesterol: shock and the alternative: the Baklayan method] from DermaVit KG, D-80331 Munich, phone +49-89-265635, fax +49-89-23 26 97 68.

lower your cholesterol level back to normal (see book "Cholesterin: Schock und die Alternative" [Cholesterol: shock and the alternative], p. 1).

Dietary measures are helpful to detoxify the whole body but not absolutely necessary.

On closer examination it will be seen that this view does not contradict scientific knowledge:

- A clogged liver leads to defective LDL receptors. To put it simply, they are "stuck shut". Consequently they cannot operate properly. If we now cleanse the liver carefully and maintain it in this "clean" state (this is easy, for example, with repeated cleansing and other supportive measures), the receptors are reactivated and they can resume their important role once more (see cholesterol hypothesis, page 12).
- In practice there is evidence that clogging of the liver increases even with a high HDL ratio. This is due to the cholesterol being transported from the peripheral tissue into the liver for further processing and catabolism. The waste products then settle in the liver.
- An additional vicious circle emerges as follows: bile acids required to digest fats reach the intestine where they are reabsorbed into the venous blood (*enterohepatic cycle*) mainly at the end of the small intestine (*terminal ileum*) and return to the liver to be reused. This is quite impossible if the liver is clogged as increasing backing up occurs which, in turn, leads to an increase in waste products in the blood and a reduction in the blood's flow properties.

It is significant for me that the old cleansing of the liver is directed at the numerous bile ducts in the liver which are full of these waste products.

Only if you have carried out several liver cleansings, will you have experienced how thousands of gelatinous crumbly stones are excreted in the process. At this point, at the latest, we have to acknowledge that this method cannot be questioned.

BICOM THERAPY

Important supporting measures, once the patient has been adjusted correctly (blocks, elements, elimination routes, etc.):

- liver detoxification program 430, 431
- test and treat parasites, especially ascarides, lamblia and, if necessary, various liver and intestinal flukes
- statins (eliminate Lipobay)
- metabolism programs 922, 923, 530
- if need be, test and treat food points for vitamin C and vitamin B1, selenium, etc. according to Frau Karz' method
- test and treat intestinal flora
- improve intestinal wall lymph

LITERATURE

Heart Disease. Intern. Journal Epidemiology 24, 704 (1995)

Bostom, A. G. et al: Elevated Plasma Lipoprotein(a) and Coronary Heart Disease in Men Aged 55 Years and Younger. Journal Am. Med. Assoc. 276, 544 (1996)

Genest, J. J. et al: Plasma Homocysteine Levels in Men with Premature Coronary Artery Disease. Journal American College Cardiology 16, 114 (1990)

Gey, K. F. et al: Plasma Levels of Antioxidant Vitamins in Relation to Ishemic Heart Disease and Cancer. American Journal Clin. Nutrition 45, 1368 (1987)

Riemersma, R. et al: Risk of angina pectoris and Plasma Concentrations of Vitamin A, C and Carotene. Lancet 337, 1 (1991)

Gysling, E.: Lipidsenker: Unangenehme Wahrheiten. [Lipid-lowering drugs: the unpleasant truth] Pharmakritik 9, no. 10, p. 39 (1987)

Hahmann, H. W, et al: Lipoprotein (a): Einer der wichtigsten Risikofaktoren für frühzeitige Arteriosklerose bei Männern. [Lipoprotein (a): one of the major risk factors for premature arteriosclerosis in men] Ellipse, 30.03.1992

Klepzig, H. and Kaltenbach M.: Cholesterinsenkung und Lebenserwartung: Eine kritische Stellungnahme. [Cholesterol lowering and life expectancy: a critical appraisal] Zeitschrift für Kardiologie, 81, 347 (1992)

Klepzig, H.: Cholesterin: Zeit zum Umdenken? [Cholesterol: time to think again?] Fortschritte der Medizin 10, vol. 34, p. 12 (1992)

Kovacsics, M.: Margarine kriegt ihr Fett ab – viele pflanzliche Aufstriche treiben den Cholesterinspiegel hoch. [Margarine gets rid of your fat – many vegetable spreads push up cholesterol levels] Süddeutsche Zeitung, 28.03.1991

Oliver, M.: Reducing Cholesterol Does not Reduce Mortality. Journal Americ. College Cardiology 12, 8 (1988)

Pinieux, G. et al: Lipid lowering drugs and mitochondrial function. Effect of HMG-CoA reductase inhibitors on serum-ubiquinone and blood lactate/pyravate ratio. Brit. Journal Clin. Pharmacol. 42, 333 (1996)

Ramsay, L. E. et al: Dietary Reduction of Serum Cholesterol Concentration: Time to think again. British Medical Journal, 303, 953 (1991)

Ravnskow, U.: Cholesterol Lowering Trials in Coronary Heart Disease: Frequency of Citation. British Medical Journal, 305, 15 (1992)

Rimm, E. B. et al: Vitamin E Consumption and the Risk of Coronary Heart Disease in Men. New England Journal Medicine 328, 1450 (1993)

Pauling, L., Rath, M.: An Orthomolecular Theory of Human Health and Disease. Journal Orthomolecular Medicine 6, 135 (1991)

Rath, M.: Lipoprotein (a)-Reduction by Ascorbate. Journal Orthomolecular Medicine 7, 81 (1992)

Pauling, L.: Vitamin C and Cardiovascular Disease. Medical Science Research 19, 398 (1991)

Haffner, S. M.: Lipoprotein (a) and Diabetes. Diabetes Care 16, 835 (1993)

Blesalski, H. K.: Antioxidative Vitamine in der Arteriosklerose-Prävention. [Antioxidative vitamins in arteriosclerosis prevention] Therapiewoche 42, 2168 (1992)

Diplock, A. T.: Optimale Aufnahme von antioxidativen Vitaminen und Carotinoiden. [Optimal intake of antioxidative vitamins and carotinoids] VitaMinSpur 8, 11 (1993)

Esterbauer, H. et al: Antioxidative Vitamine und degenerative Erkrankungen. [Antioxidative vitamins and degenerative disease] Deutsches Ärzteblatt 87, vol. 47 (1990)

Armstrong, V.: Lipoprotein (a), ein weiterer Risikofaktor für Atherosklerose. [Lipoprotein (a), a further risk factor for atherosclerosis] Fortschritte der Diagnostik 2, 33 (1991)

Carlson, L. A. et al: Pronounced lowering of Serum levels of Lipoprotein Lp(a) in hyperlipidemic subject: treated with nicotinic acid. Journal Internal Medicine 226, 271 (1989)

Pauling, L.: Vitamin C Deficiency is Associated with High Risk of Myocardial Infarction in Eastern Finnish Men. Europ. Heart Journal 15, 168 (1994)

CARE Study (Sacks, F. M. et al)The effect of Pravastatin or Coronary Events after Myocardial infarction in patients with average cholesterol levels. New England Journal Medicine 335, 1001 (1996)

"JAMA" (Journal of the American Medical Association), general archive, issues 285, 2001, 2486 specifically

Koronare Herzerkrankungen und Vitamin-Spiegel sind stark korreliert. [There is a strong correlation between coronary heart disease and vitamin levels] Ärzte Zeitung, 29.10.1992

DeaM naturopathy Internet database,
www.deam.de
incl. list of practising naturopaths