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Cholesterol

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May 2014 **Volume 10: Issue 5**

Choked Arteries

One of the earliest works I ran across on heart disease still makes a lot of sense to me. Robert S. Ford wrote *Stale Food - vs - Fresh Food* in 1969. Ford was the brilliant President of Magnolia Laboratory in Mississippi. Ford wrote, "By feeding experiments with animals and human beings consuming nearly a quarter million dollars in labor and materials over a period of seven years I finally determined that the true cause of arteriosclerosis is simple: **STALE FOOD**." Ford was particularly concerned about "deteriorated spurious fats" including oxidized cholesterol.

If you are a typical American, you think of fat and cholesterol when you hear mention of heart disease. The first experiment linking cholesterol to heart disease in rabbits was conducted by Dr. Nikolai Anichkov in 1912. Anichkov fed rabbits cholesterol in a vegetable oil medium.

Low Thyroid

One criticism of Anichkov's work is that rabbits are naturally herbivores. Feeding them cholesterol is unnatural and raises cholesterol to very high levels. Kenneth B. Turner published a paper in 1933 showing that thyroid extract lowered very high cholesterol levels in rabbits (13.45 mmol/L) to much more normal levels (4.60 mmol/L). The thyroid extract also prevented atherosclerosis.

Subsequent research by Dr. Broda Barnes and others has shown that cholesterol levels rise when thyroid levels are low. These elevated cholesterol levels increase risk of heart disease. Administration of thyroid hormone lowers cholesterol levels and reduces risk of heart disease. Barnes wrote, "A rational approach to the prevention of heart attacks calls for the recognition of thyroid deficiency--better late than never but preferably as early as possible--and its proper treatment for the rest of life."

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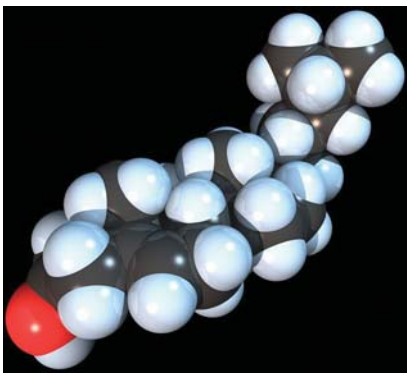

Oxidized Cholesterol

In 1956 Louis Fieser suggested that the early studies on heart disease were misleading because the cholesterol being used was oxidized. In 1976 Imai and associates separated the oxidized impurities in USP cholesterol and fed it to rabbits. The oxidized product resulted in lesions in the arteries of rabbits within seven weeks. Lesions were *not* found in animals given purified or non-oxidized cholesterol.

In 1979 Taylor and associates found that fresh USP cholesterol contained 5% oxidized products while a 5 year old jar contained 40% oxidized cholesterol. The researchers found that oxidized cholesterol is 500 times more damaging to the arteries than is pure cholesterol.

Oxidized cholesterol is found in a variety of foods. Powdered milk and eggs, Indian Ghee, cheeses aged for prolonged periods of time, pancake and custard mixes, and lard used for deep frying have all been found to contain oxidized cholesterol.

Early experiments using egg yolk to feed animals implicated eggs in causing heart disease. Powdered egg yolk was used in these experiments. Even though fresh eggs contain about 250 mg of cholesterol in the yolk (more than any other commonly consumed food), there is little risk of oxidized cholesterol if they are soft boiled.

Trans fats, like oxidized cholesterol, have also been implicated in heart disease. Trans fat comes from foods which contain partially hydrogenated oils. These fats are inexpensive and they increase shelf life and stability and improve the texture of foods. The CDC estimates that reducing trans fat in the diet could prevent as many as 20,000 heart attacks each year. It is estimated that the average American consumes 1.3 grams of trans fat each day. Trans fats are commonly found in snacks, fried items, pizzas, cake, cookies, pies, margarines and spreads, ready-to-use frosting, and coffee creamers.

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Statin Medications

Statin medications are the most commonly used treatments for reduction of cholesterol levels and the risk of heart disease. Statin medications block an enzyme known as HMG-CoA reductase which is the weak point in cholesterol synthesis in the human body. Pharmaceutical firms have created a multi-billion dollar business with products which block this important enzyme.

CoEnzyme Q10

Statin medications block more than cholesterol formation. Co-enzyme Q10 is produced on this same metabolic pathway. Loss of this enzyme can lead to damage to the mitochondria where energy is produced in the body.

Statin drugs *block the synthesis of coenzyme CoQ10*, a critical antioxidant in two stages of energy production in the mitochondria (complex one and complex two). Loss of coenzyme Q10 increases damage to mitochondrial DNA. Repair of DNA damage requires glycohydrolase, a compound which can not be synthesized without dolichols *which are also inhibited by statin medications*. Thus use of statin drugs both increases damage to the DNA of the mitochondria where energy is produced in the cells and also blocks the formation of an essential substance involved in repair of this damaged DNA.

Proper muscle functioning is dependent upon adequate levels of Co-enzyme Q10. Many people develop severe muscle pain while using statin medications for this reason. Carried to its extreme, blockage of CoQ10 can lead to rhabdomyolysis. This is a nasty condition in which damaged muscle tissue breaks down. The breakdown products are released into the bloodstream and can lead to kidney failure and death.

Cholesterol Synthesis

Statin drugs block cholesterol synthesis throughout the body including the brain. In 2001 Frank W. Pfrieger discovered that cholesterol controls the building of synapses in brain cells. The synapse is the structure which permits one nerve to communicate with another. Failure of synapse function can result in memory failure.

The ability of statin drugs to block cholesterol synthesis in the brain and the importance of this substance for synapse formation explains why transient memory loss and cognitive impairment have been associated with the use of statin medications.

Duane Graveline, a surgeon employed by NASA, was placed on a statin medication on two separate occasions. Both times he suffered severe episodes of transient memory loss.

In 1999 when Graveline began Lipitor he lived on the side of a mountain in Vermont. His normal activity consisted of climbing the mountain and cutting and splitting his own firewood. In just a few years after his use of statin medication he was a doddering old man with withered limbs who needed a walker to get around. His neurologist suspected mitochondrial mutation secondary to statin use, possibly the most serious side effect of statin use.

Dolicols

As mentioned already, statin medications block the production of dolichols involved in DNA repair. Dolichols are also involved in cell communication, cell identification and functioning of the immune system. Abnormalities in dolichol functioning may play a role in emotional and behavioral abnormalities associated with use of statin medications.

Behavioral abnormalities associated with statin drug use are severe irritability, homicidal impulses, threats to others, road rage, generation of fear in family members, and damage to property. Dolichol abnormalities may be involved with these symptoms.

NF-kB

Statin medications block the synthesis of another important substance known as nuclear factor kappa B (NF-kB). NF-kB triggers inflammation when the body is under attack. Graveline writes, “The second action of statins is to inhibit NF-kB and this appears to be the secret to their remarkable effectiveness in cardiovascular risk reduction.”

NF-kB plays a key role in immune function. Supression of this substance reduces the risk of heart disease, but it may also make us more susceptible to faulty immune function.

A trial of pravastatin, a statin medication, found that the drug did decrease deaths from heart disease, but increased deaths from cancer. Goldstein wrote in *The Lancet*, “This exactly cancelled the mortality benefit of coronary heart disease death, which was 4.2% in the placebo group and 3.3% in the pravastatin group. All-cause death was unchanged, implying that pravastatin treatment changed

how a patient died by increasing cancer death and decreasing death from coronary heart disease. Pravastatin decreases natural killer cell cytotoxicity. Low cytotoxic activity of natural killer cells is associated with increased cancer risk."

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Lower Cholesterol

The main purpose for which statin medications are prescribed is to lower cholesterol. This naturally raises the question, "Does reducing the level of cholesterol necessarily reduce the risk of atherosclerosis and heart disease?"

Uffe Ravnskov, M.D., Ph.D., conducted a meta-analysis of 26 controlled cholesterol-lowering trials. The total number of fatal heart attacks in both treatment groups and control groups was 2.9%. The total number of deaths was 5.8% in the control groups and 6.1% in the treatment groups. This does not provide optimism for benefit from lowering cholesterol levels.

Ravnskov points out that statin medications do lower risk of a heart attack. However, "the risk of having a heart attack was reduced by the same degree whether the cholesterol level was lowered by a large or small amount...this phenomenon is called 'lack of exposure-response.' Lack of exposure-response strongly indicates that the factor under investigation is not the true cause, but is secondary to the real cause....The only reasonable explanation is that the statins do more than just lower cholesterol....The proponents of the cholesterol hypothesis have simply had incredible luck in finding a substance that prevents cardiovascular disease and at the same time lowers cholesterol."

The cost of statins is very high for a very small reduction in risk from heart disease. In addition, statin medications have risks of their own. The package insert for Lipitor recommends liver function tests before use of the drug and periodically thereafter. The insert also notes that muscle tenderness or aching and muscle weakness are potential consequences of the use of the medication. Risk of hemorrhagic stroke is also increased. The drug can also cause psychiatric disorders such as insomnia, nervous system disorders like headache and peripheral neuropathy, and digestive disorders.

Failure of proper functioning of the mitochondria can result in memory impairment, neuropathy, and muscle weakness. Damaged mitochondria speed the aging process leading to physical deterioration in young people which would not normally be seen until they were much older.

The heart is a muscle which research shows is weakened by the use to statin medications. It is a great irony that many people take statin medications thinking it will reduce risk of heart disease when it has been demonstrated that the medications themselves weaken heart function. Rubinstein and associates concluded, "Statin therapy is associated with decreased myocardial function..."

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Vitamin C

If oxidized fats are a serious risk factor for heart disease, and they are, it makes sense that antioxidant intake can provide significant benefit for those at risk of developing the disease. Matthias Rath, M.D., former Director of Cardiovascular Research for the Linus Pauling Institute of Science and Medicine has written a volume which emphasizes the importance of vitamin C and other antioxidants. For example, one study found that an additional 300 mg of vitamin C a day reduced the rate of heart disease in men by half and in women by one-third. Rath wrote, "*No study with any other drug has ever shown a similar cut of heart diseases*."

Studies have shown that intake of other antioxidants such as vitamin E and flavonoids can also reduce the risk of heart disease. The Shute brothers advocated the treatment of heart disease with vitamin E in 1946. Their claims were rejected by the medical community but have since been validated. The Shute brothers did their initial work with natural vitamin E and found it much more effective than the synthetic product which was later introduced and used in scientific studies of vitamin E. The U.S. government did not acknowledge the requirement for vitamin E until 1968.

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Omega-3

Low intake or blood levels of omega-3 fatty acids are independently associated with increased risk of death from coronary heart disease. The Omega-3 Index, a measure of the quantity of higher quality omega-3 fatty acids in the blood, is inversely correlated with risk of death from heart disease. The risk of cardiac arrest in those with the highest omega-3 was only 10% of that with the lowest

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HOMOCYSTEINE

Kilmer McCully began his medical career investigating the mysterious death of an eight-year-old child. The child died of atherosclerosis.

McCully concluded that a percentage of heart disease has nothing to do with cholesterol. That young child had a genetic abnormality which resulted in accumulation of homocysteine in the blood. Homocysteine is a byproduct of metabolism of the amino acid methionine.

Homocysteine is corrosive of proteins including three main structural components of the arteries (collagen, elastin, and proteoglycans). Elevated levels of homocysteine appear to be associated not only with heart disease but also Alzheimer's, schizophrenia, and weak bones.

Homocysteine levels rise when one is deficient in nutrients known as methyl donors. This includes vitamins B6, B12, folic acid and betaine.

One can see how a diet high in meat which contains a good deal of methionine and a deficiency of B complex vitamins could set the stage for elevated levels of homocysteine and increased risk of heart disease. It would be easy to blame the saturated fat in the meat and to miss the elevated homocysteine levels associated with nutrient deficiency. Research on homocysteine suggests that heart disease may be as much about nutrient deficiency as dietary excesses.

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CONCLUSION

The studies reported here suggest that it is probably more important to avoid consumption of oxidized cholesterol and prevention of oxidation of the cholesterol circulating in the blood than it is to avoid cholesterol in the diet. Key factors in the prevention of heart disease are avoiding stale foods with oxidized fats, adequate intake of the anti-inflammatory omega-3 fatty acids, adequate methyl donors to prevent accumulation of homocysteine, and adequate intake of antioxidants like vitamins C, E and carotenoids to prevent the spontaneous oxidation of blood cholesterol. These measures can help keep an individual from becoming a heart disease statistic.

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