Smart Life Forum

Roundtable of experts including Dr. Alan Brauer, Dr. Robert Cathcart, Steven Wm. Fowkes, Dr. Phillip Miller

Making Sense of Heart Disease Theories

Cubberly Community Center
4000 Middlefield Road, Room H1, Palo Alto, California

September 15, 2005 at 7:00 PM

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Future Speakers:

- October 20, Raymond Peat, PhD
Protecting and Restoring the Nervous System

- November 17, TBD

- December 15, TBD

- Jan 19, David Brownstein, MD
Iodine; Why You Need it, Why You Can't Live Without It

Roundtable Speakers

Our panel of experts are all members of Smart Life Forum and represent a broad range of expertise in alternative medicine and will be focusing on heart disease for this meeting . The audience is encouraged to ask questions. The enlightening article on heart disease below is written by Stan Field who could not attend and whose bio follows.

Stanford Field received his Bachelor of Science in Chemical Engineering from Penn State in 1951 and an MS in Meteorology from the U.S. Naval Postgraduate School in 1955. After working as a chemical engineer for four major corporations, he settled in at Stanford Research Institute for the next 23 years, retiring as Director of Energy Programs. Since joining the Smart Life Forum in 1996 he has been studying and writing articles in Biochemistry. His 12 articles appear on our SLF website under the “Too Late Schmart” newsletter series.

Making Sense of Heart Disease Theories

Toward Clarification: Much of this article is based on the confluence of biochemistry, cardiology and physiology where the complexity of interactions induces a great deal of uncertainty. The complications of various theories has led to the development of the “risk factor” or “cookbook” approach to manage heart disease. The main objective of this article is to go beyond the “risk factor” approach by clarifying the heart disease theories, elucidating important biochemical mechanisms and offering an orthomolecular protocol for a healthy heart.

Contents:

Cholesterol Theory

During the early 1900s, heart death rates, especially among men, increased significantly. Pathologists found that coronary arteries were blocked by cholesterol-rich plaque. This finding correctly created the assumption that cholesterol was linked to heart disease. By the 1950s, epidemiologists found that there were more cardiovascular disease (CVD) deaths in countries where blood cholesterol levels were higher. Although there was no clear biochemical explanation, this was the genesis of the cholesterol theory of CVD.

The artery blockage problem was urgent, and biochemical explanations would require a great deal of time. So, the medical community went with the epidemiology. Pharmaceutical companies responded by developing a variety of chemicals to hinder normal body biochemical reactions. The most prominent “drug” became the “statin” which successfully lowered cholesterol by blocking an enzyme that converted a 2-carbon fragment (acetate) into the 27-carbon cholesterol molecule. The statin also prevented the formation of coenzyme Q-10 which is essential for the energy system of each of the trillions of cells of the body. This inhibition is especially detrimental to the heart muscle cells which are continuously used. The heart and the brain cells never rest. **The use of epidemiology without knowledge of the biochemistry involved caused dependence on the “risk factor” approach and its attendant uncertainties.**

Important knowledge on the biochemistry of heart disease was obtained from examination of the bodies of *young men* killed in the Korean (1950s) and Viet Nam (1960-70s) wars. Autopsies revealed that the men had significant atherosclerosis (blockage of coronary arteries with cholesterol plaques). It was found that oxidized cholesterols (oxycholesterols) caused the blockages. The oxycholesterols were fed to the soldiers in the form of powdered eggs and powdered milk that had come from the oxidation induced

by the spray drying of eggs and milk that had been done to avoid the inconvenience of shipping the associated water to the distant battlefields.

By the 1990s, it was clear that the oxycholesterols and other forms of oxidative stress cause inflammation in the lining of coronary arteries and that the body uses its resources to repair the damage. A continuation of the oxidative stress and concomitant inflammation eventually causes a buildup of repair materials that block arteries. The result of this process has been named “atherosclerosis.”

Incidentally, a major cause of high cholesterol and obesity in our increasingly stressed-out society is the unprecedented consumption of sugar which is converted to fat and cholesterol if it is not immediately used for energy.

Homocysteine Theory

The homocysteine theory predicts that a dietary imbalance between too much methionine (an essential amino acid) and a deficiency of methylating nutrients (B6, B12, folic acid and methyl-containing compounds such as trimethylGlycine or trimethylAminoEthanol [choline]) is the underlying cause of death and disability from vascular disease. That imbalance causes a buildup of homocysteine which oxidizes cholesterols to the perilous oxycholesterols.

The excess methionine also causes the buildup of homocysteine thiolactone which causes the oxidation and aggregation of low density lipoprotein (LDL) cholesterol. The homocysteine thiolactone / LDL cholesterol aggregate is released from the liver and is taken up by macrophages of the arterial wall to form foam cells which decompose the aggregate into plaque composed of oxidized cholesterol and oxidized fats. Oxygen radicals damage the cells that line the arteries. The foam cells release pro-inflammatory cytokines that induce blood-clotting factors and fibrin formation at the site of the damage. The blood clot formation and fibrous tissue then become encrusted with calcium deposits to form a brittle, tough and hardened arteriosclerotic patch that cannot be cut with a scalpel. A continuation of this process completely blocks the arteries (arteriosclerosis).

Lysine forms an adduct with homocysteine thiolactone which can be eliminated through the liver's detoxication system.

Collagen Theory

The earliest recorded symptoms of scurvy were made by the Egyptians around 1500 BC. Three thousand years later, scurvy emerged as a significant problem for European maritime explorers when their voyages were long enough to penetrate the Indian and Pacific oceans. In 1520, Magellan lost more than 80 percent of his crew to scurvy on the voyage around Cape Horn and across the Pacific in the first global circumnavigation. Written accounts described scurvy as follows: skin black as ink, ulcers, difficult respiration, loosening of the limbs, teeth falling out, and the rotting of gum tissue which fell out of the mouth and gave the victim's breath an abominable odor. In the 18th century, scurvy was often diagnosed as leprosy, syphilis, dysentery and madness. So what does scurvy have to do with heart disease? The Collagen Theory views heart disease as a result of chronic low-level scurvy.

In the late 1980s, medical researchers discovered that heart disease begins with a crack or stress fracture in the arterial wall in the area of high mechanical stress around the heart. The body's emergency response team releases cytokines that initiate the arachidonic acid cascade (via eicosanoids that induce inflammation). The damaged area is immediately clotted with an aggregate of platelets and a mesh of fibrin molecules that is catalyzed by calcium. Blood monocytes detect the presence of oxidized lipoproteins and enter the vascular wall to become macrophages which secrete plasminogen activators to produce plasmin which activates procollagenases to produce collagenase which dissolves the damaged connective tissue in the vascular wall in preparation for replacement. If the nutrients that build collagen are available, repair of the damage will take place. Glucocorticoids other anti-inflammatory agents and pain killers (*e.g.* , morphine, vicodin) inhibit the induction of plasmin and the repair process.

What if the repair nutrients are not available? The emergency response team causes the release of C-reactive protein which activates apoprotein(a) and other adhesion molecules to quickly mobilize a repair process. Apo (a) is attached to the outside of low density lipoprotein to form a repair complex. The aggregate of low density lipoprotein and apoprotein(a) is called “lipoprotein(a)” and is abbreviated to “Lp(a).” The Lp(a) aggregate slowly dissolves the fibrin/platelet clot before being attracted (“glued”) to collagen lysine and proline binding sites. Then Lp(a) creates binding sites for cholesterol and calcium to strengthen the patch. Thus, Lp(a) becomes the main patch for the stress fracture (the

1987 Nobel Prize in Medicine was awarded for that discovery). Thus, apo(a) is a surrogate for ascorbate and other nutrients that build collagen and elastin.

To summarize, the basis of the Collagen Theory is that the main cause of atherosclerotic plaques is the weakness of artery walls that is caused by a chronic deficiency of nutrients required to strengthen arteries. Atherosclerotic deposits develop to strengthen weakened blood vessel walls. It is well established that atherosclerotic disease does not occur in most animals because they synthesize ascorbic acid (from glucose), whereas a human cannot. Therein lies a monumentally important key to maintaining healthy arteries.

The presentation will also include a discussion of the relationship between oxidation and inflammation (what causes it, why it occurs and how it can be minimized). The World Health Organization (25-year study) of the effect of food types on heart disease mortality will be examined. A recommended protocol of food, supplemental nutrients, detoxication and emotional lifestyle will conclude the program.

Inflammation Underlies All These Theories, But What Causes Inflammation?

Inflammation is an integrated body response to factors (internal or external) which threaten the organism's integrity. The inflammatory response and its attendant pain can be initiated by any form of cell and tissue injury. It serves as an alarm system to guard against further injury, and it stimulates repair of the damaged area. Leukocytes, phagocytes and other debris-removing cells clean the area of foreign substances, necrotic debris and invading organisms. A complex chain of events is initiated to repair the damaged tissue.

The function of inflammation is to alert you to its existence (to allow you to prevent further damage) and to heal wounds. The successful inflammatory response depends on the cessation of the source of irritation or injury. Prolonged inflammation can lead to autoimmune pathology and accompanying degeneration.

Common sources of inflammation include: physical trauma, infection, lack of sleep, emotional distress and chemical toxicity (*e.g.,* pesticides, food additives, modern medicines, mercury amalgams, smoking [including second-hand smoke], excitotoxins, and estrogen mimics).

In infection, phagocytic cells produce a respiratory burst which is composed of oxidative chemicals (e.g. superoxide, hydrogen peroxide, hypochlorous acid and chloramines) that are toxic to micro-organisms. The oxidative burst creates holes in the membranes of the micro-organisms and causes the lysis of their cell contents. Some healthy body cells are unavoidable battle casualties. Chronic infections (*e.g.,* yeast) intensify immune system warfare.

In emotional distress, including lack of sleep, thoughts and moods can trigger a cascade of inflammatory reactions. Being depressed, hostile, anxious or otherwise stressed is pro-inflammatory. Chronic stress is destructive because pro-inflammatory and pro-aging forces are occurring continuously. This leads to chronic inflammatory degenerative illnesses.

Effect of Food on Heart Disease Mortality

The following is based on the World Health Organization Seven Countries Study.

Food intake patterns and 25-year mortality from coronary heart disease were studied. The final report was issued in 1999. Major countries with the lowest CHD mortality were Japan, Greece, and Italy where diets emphasized **vegetables, fish, oils and legumes (beans, nuts and seeds)** . Major countries with the highest CHD mortality were Finland, Netherlands and the United States where diets emphasized **meat, milk, sugar, potatoes and eggs.**