Smart Life Forum

Ralph Holsworth, Jr. D.O.

Nattokinase, Enzyme Treatment for Chronic Inflammation, Hypercoagulability, and Crosslinked Fibrin

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

August 18, 2005 at 7:00 PM

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Future Speakers:

- September 15, Stan Field

Making Sense of Heart Disease Theories
(A trip into the realm of cardiac confusion, where money trumps the Hippocratic oath)

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

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

Opening Bio-Med 101 Presentation

Stan Field,
“Sugar! Sweet, Satanic Seduction: The Opiate of a Stressed-Out Society”

Main Speaker

Ralph E. Holsworth, Jr. received his Doctor of Osteopathy in 1997 from The University of Health Sciences, College of Osteopathic Medicine , Kansas City, MO. He received his BS degree from the University of Texas , El Paso in 1985. In 1977-1980, Dr. Holsworth was a Nuclear Power Plant Propulsion Operator for the United States Navy.

He was the Director of Clinical and Scientific Research for Essentia Water, 1999-2001. He served as an honorarium professor at the University of Colorado at Colorado Springs , Spring 2001. He has been a research assistant involved in clinical research with oral systemic enzymes as an adjuvant in cancer treatment at The Cancer Treatment Center of Tulsa, OK in 1997. Dr. Holsworth was on the Scientific Advisory Board of Trans-formation Enzyme Company, TX from 1995-1997. Also formerly with the EPA, he was involved in assessing superfund sites and reclamation sites and was a member of the EPA's Hazardous Material Emergency Response Team in Houston, TX. He has authored several publications related to antioxidant properties of electrolyzed water and nattokinase, a fibrinolytic enzyme. He is member of the Editorial Board for the Journal of Applied Clinical Thrombosis and Homeostasis and the Japan Nattokinase Research Association. He is currently serving as a Lieutenant Commander for the U.S. Public Health Service assigned to the Jicarilla Apache Reservation in Dulce, NM.

Main Presentation

The goal of this presentation is to introduce the interplay of chronic systemic infections, chronic inflammatory states and the immune system imbalances which contribute to atherogenesis. The Theory of Protective Adaptation will be introduced to demonstrate the unifying theory of atherogenesis resulting in increased whole blood viscosity.

Therapeutic application of the medical food nattokinase, the active ingredient of the traditional Japanese folk medicine – Natto will presented. Dr. Holsworth will present laboratory and clinical findings that verify nattokinase's ability to decrease whole blood viscosity and also to prevent initiation of chronic cardiovascular diseases including essential hypertension at subcellular levels.

Objectives

1. Understand and appreciate the pathogenesis of chronic inflammatory states secondary to genetic and/or chronic infectious processes.

2. Understand and appreciate the role of increased whole blood viscosity as the common denominator of atherogenesis (i.e., The Protective Adaptation Theory).

3. Understand how a traditional Japanese medicinal food - natto prevents and treats essential hypertension and cardiovascular disease by decreasing whole blood viscosity.

4. Understand the pharmokinectics, biochemistry and biophysics related to the enzyme - nattokinase.

5. Understand which patients are candidates for the safe and effective use of nattokinase.

Highlights

Massive research efforts and numerous hypotheses have failed to identify the initiating event of atherosclerosis. Why it develops is significantly more important than how it progresses. To that end, the evolutionary approach to the origin of atherosclerosis is

presented and explained based upon the biophysical properties of the blood and their interrelationship with the blood vasculature.

Historically, cardiovascular research has focused on a biochemical approach to atherosclerosis. The results have been a very detailed and accurate histopathology of it, starting with the histological manifestations of fat-laden cells in the intima to the complex series of mechanisms. However, any theory that embraces only a biochemical, genetic, or environmental perspective leaves many questions unanswered. These questions include the following:

• Why are the arteries leading to the heart and brain so susceptible to atherosclerosis?

• Why do we not observe atherosclerotic plaques in the intramyocardial coronary arteries, the arteries of the arms or breasts, or in the veins?

• Why do people with “normal” blood pressure and “normal” cholesterol still have heart attacks?

• Why do men develop cardiovascular diseases at a younger age than do women, especially premenopausal women?

• Why is there a pattern of increased heart attacks in the morning hours (Cannon et al. 1997, Cohen et al, 1997)?

There are simply too many questions that cannot be resolved by applying only current biochemical theories. The fundamental shortcoming of current Bio-chemical theories is that they do not identify the initiating event that precedes endothelial injury.

Blood behaves very differently in our circulatory system than water flowing in pipes. First of all, blood has a higher viscosity (thickness) than water. Increased blood viscosity and blood flow is pulsatile and the flow rate varies with time. The reason for the pulsatile flow is two-fold, a resultant of the ejection portion of the cardiac cycle and because the arterial wall is elastic. The arterial system is not a straight pipe with its many bifurcations and bends. Pulsatile blood flow imparts energy into the arterial system that is stored partially in the blood vessels.

The protective adaptation process theory organizes the arterial system's adaptative

process into two cycles, both of which originate from the mechanical stresses in the system. The first cycle is the region-specific development of arteriosclerosis, a condition in which the arteries have lost their compliance (elasticity). The second cycle is site-specific development of atherosclerosis in arteries that lost their compliance in cycle one. Although, *arterio* sclerosis is a precursor to *athero* sclerosis, the two cycles develop synergistically and reinforce each other in a vicious circle. Arterial occlusive disease results from a protective response to mechanical stress and injury, a futile effort to maintain the integrity of the vessel.

At birth, arteries are extremely compliant and stretchable, but over a lifetime these characteristics decrease as a result of the changes in wall tissue structure. The loss of compliance has been defined as medial arteriosclerosis. The changes of compliance in the arterial wall is an adaptative response to the stretching and stress of high arterial pressure, which causes extended, repeated overstretching of the arteries. No theory appropriately accounts for the initiating causal factors or factors in atherosclerosis. This is an unsettling fact on an individual and societal level. The cost to the United States in medical care and lost productivity due to cardiovascular diseases is estimated at $298 billion for 2001 (American Heart Association).

Certain types of blood flows may cause mechanical damage to the vasculature. These types of blood flows are referred as injurious pulsatile flow. In response to this mechanical injury, the vasculature develops plaques and abnormalities in the vessel wall in a predictable pattern. The presentation of these various mechanisms in a unified concept is called the protective adaptation theory. The protective adaptation theory (Kensey and Cho 1992, Kensey and Cho 1994) provides the missing link, particularly in events preceding lesion development, where current biochemical theories cannot account for the mechanisms. The “Why” of arteriosclerosis and atherosclerosis is eloquently explained by the bellwether work of The Protective Adaptation Theory. Endothelium injury is caused by a high intensity stimulus over a short period of time, for example, a coronary artery stent placement. Stress is caused by a low-intensity stimulus over a long period of time, for example, a callus is a standard adaptation of the skin to stress. A key difference between protective adaptation to stress and to injury is that protective adaptation to stress is usually reversible.

Introduction of Nattokinase

Natto, made from fermented soybeans, is a traditional Japanese food. Many people enjoy

it for its distinctive flavor, enlivened by the activity of Bacillus subitilis. Natto has a long history, and some have theorized that it may even have a prehistoric origin, possibly circa B. C. It has at least been ascertained that natto has been popular since the Edo period, 400 years ago. Originally, natto was utilized as a folk remedy for heart and vascular diseases, fatigue, and beriberi. In 1980, Dr. Hiroyuki Sumi et al. found that natto contains a potent fibrinolytic enzyme, which they named nattokinase. It was confirmed that oral administration of nattokinase (or natto) produced a mild and frequent enhancement of the fibrinolytic activity in the plasma, as indicated by the fibrinolytic parameters, and the production of tissue plasminogen activator. Nattokinase capsules were also administered orally to dogs with experimentally induced thrombosis, and lysis of the thrombi was observed by angiography (Sumi 1990). It was shown that the oral administration of natto and nattokinase enhance the fibrinolytic activity in plasma. A shortening of euglobulin lysis time (ELT) and an elevation of EFA were found for a long time (from 2 to 8 hr) after a single administration of natto ($p<0.01$) (Sumi 1989).

Conclusion

Nattokinase may prove to be a defibrinogenating enzyme that drastically decreases blood viscosity. Decreasing blood viscosity strikes at the root of arteriosclerosis and atherosclerosis as well as hypertension, peripheral vascular disease and congestive heart failure. The fibrinolytic activity of nattokinase resolves the active process of atherosclerosis and lyses thrombi. The per oral administration, prolonged half-life of 4-6 hours and extremely safe profile show favorably upon nattokinase as the key agent for restoration of vasculature health.