Reversing arterial blockage: Experimental regimen that worked for man facing amputation of his lower legs

Last year (2012) I was contacted by a gentleman who had severe blockage in his legs and had been told by his physician-surgeon that he would need to amputate his legs below the knee. Naturally this chap was horrified at the prospect of living his life out of a wheelchair and/or on crutches and was frantic to know if there is anything, however experimental, that might help him avoid the surgeon's knife. Apparently he had done a Google search and come across some of my health-related hypotheses (I have since 1986, been paid by various docs & researchers to spin hypotheses and provide novel ideas they could then pursue – or not).

I could not, of course, prescribe anything whatsoever as I am not a physician and lack the qualifications to get into such matters. However, what I did do is hand off an experimental regimen I originally developed in the late 1980s and had updated recently, along with the stringent caveat that much of my own work and that which informed it involved animal models of arterial blockage and I was giving him this with the strict understanding he would share it with his primary care MD and do what he said or advised.

I heard nothing from this chap for about 8 months and then got a call. It seems he was able to get his MD to endorse my handwork which he had followed religiously. Long story short, the circulation in his legs had improved to the point that amputation was no longer on the proverbial table. Needless to say he and his wife of some 50+ years were overjoyed by his progress.

So here is what I shared with this guy – again, with the caveat it is experimental and unproved and should not be undertaken without the express consent & supervision of a duly licensed & practicing MD or DO.

Dr. Anthony G. Payne

SUPPLEMENTS

PADMA BASIC - sources & comparison prices: http://www.best-deal.com/search/landing/query/padma+basic/s/google/koid/6234666013/gkaid/61679560/adid/2066452360/gkyid/1164695747/?query=padma+basic&gclid=COjOj63ogrACFQFeTAodHVs8jg

LAY-LEVEL ARTICLE ON PADMA:

http://www.scribd.com/doc/158030311/TIBETAN-VASOACTIVE-HERBAL-BLEND-PADMA28

<u>NOTA BENE</u>: Back in the mid 1990s I placed PADMA in graduated levels in chow fed to guinea pigs with induced blood vessel blockage and observed a reversal of plaque (High dose worked quickest).

Traditional dose: 3 tablets 1 hour before or 2 hours after meals.

Vitamin D3 + K2: http://www.iherb.com/Now-Foods-Vitamin-D-3-K-2-1-000-IU-45-mcg-120-Vcaps/10056 Suggested dose: 1 capsule with meals. Check with MD or DO to see if contraindicated.

Krill Oil: http://www.iherb.com/Now-Foods-Neptune-Krill-1000-60-Softgels/22675 1 softgel after breakfast and 1 softgel after lunch or dinner

Neem: 440-500 mg capsules. 2 capsules 3 x daily with or after meals. One low cost source http://www.iherb.com/Organix-South-TheraNeem-Organix-Neem-Leaf-90-VCaps/17641

Apple pectin: 700-1g (1000 mg) capsules. 1 to 2 capsules with or after meals. One low cost source is http://www.iherb.com/Now-Foods-Apple-Pectin-700-mg-120-Capsules/361

DRUGS

Tetracycline: Advisability of using this and, if OK, safe dose to be determined solely by a licensed physician (MD or DO)

DIET: LOW FAT ORNISH DIET OR ANYTHING EQUIVALENT TO IT

Dr. Dean Ornish's Program for Reversing Heart Disease: The Only System Scientifically Proven to Reverse Heart Disease Without Drugs or Surgery http://www.amazon.com/Ornishs-Program-Reversing-Heart-Disease/dp/0804110387/ref=sr_1_2?s=books&ie=UTF8&qid=1337032298&sr=1-2

Everyday Cooking with Dr. Dean Ornish: 150 Easy, Low-Fat, High-Flavor Recipes [Paperback] -

http://www.amazon.com/Everyday-Cooking-Dr-Dean-Ornish/dp/0060928115/ref=sr_1_3?s=books&ie=UTF8&qid=1337032298&sr=1-3#_ -

NOTE: I have no commercial or other interest in any firm linked to above or below, and get no concessions or favors of any kind from linking to them or their products.

ADDITIONAL READING

http://www.sciencedaily.com/releases/2013/02/130227151254.htm - Lipid Researcher, 98, Reports On the Dietary Causes of Heart Disease



Fred Kummerow, a 98-year-old emeritus professor of comparative biosciences at the University of Illinois, explains the primary causes of heart disease. His research contradicts commonly held notions about the role of dietary cholesterol. (Credit: L. Brian Stauffer)

COMMENT: You gotta love a guy who works in his lab sporting a bolo!



Am J Cardiovasc Dis 2013;3(1):17-26

Review Article
Interaction between sphingomyelin and oxysterols contributes to atherosclerosis and sudden death

Fred A Kummerow

Burnsides Research Laboratory, Department of Comparative Biosciences, College of Veterinary Medicine, University of Illinois, 1208 W. Pennsylvania Avenue, Urbana, IL 61801, USA

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Abstract: Despite major public health efforts, coronary heart disease continues to be the leading cause of death in the United States. Oxidized lipids contribute to heart disease both by increasing deposition of calcium on the arterial wall, a major hallmark of atherosclerosis, and by interrupting blood flow, a major contributor to heart attack and sudden death. Oxidized cholesterol (oxysterols) enhances the production of sphingomyelin, a phospholipid found in the cellular membranes of the coronary artery. This increases the sphingomyelin content in the cell membrane, which in turn enhances the interaction between the membrane and ionic calcium (Ca2+), thereby increasing the risk of arterial calcification. Patients undergoing bypass surgery had greater concentrations of oxysterols in their plasma than cardiac catheterized controls with no stenosis, and had five times more sphingomyelin in their arteries than in the artery of the placenta of a newborn. The oxysterols found in the plasma of these patients were also found in the plasma of rabbits that had been fed oxidized cholesterol and in frying fats and powdered egg yolk intended for human consumption. Together these findings suggest that oxysterols found in the diet are absorbed and contribute to arterial calcification. Oxidized low-density lipoprotein (OxLDL) further contributes to heart disease by increasing the synthesis of thromboxane in platelets, which increases blood clotting. Cigarette smoke and trans fatty acids, found in partially hydrogenated soybean oil, both inhibit the synthesis of prostacyclin, which inhibits blood clotting. By increasing the ratio of thromboxane to prostacyclin, these factors interact to interrupt blood flow, thereby contributing to heart attack and sudden death. Levels of oxysterols and OxLDL increase primarily as a result of three diet or lifestyle factors: the consumption of oxysterols from commercially fried foods such as fried chicken, fish, and french fries; oxidation of cholesterol in vivo driven by consumption of excess polyunsaturated fatty acids from vegetable oils; and cigarette smoking. Along with the consumption of trans fatty acids from partially hydrogenated vegetable oil, these diet and lifestyle factors likely underlie the persistent national burden of heart disease. (AJCD1211005).

Address correspondence to: Dr. Fred A Kummerow, Burnsides Research Laboratory, 1208 W. Pennsylvania Avenue, Urbana, IL 61801, USA. Tel: 217-344-6380; Fax: 217-333-7370; E-mail: fkummero@illinois.edu

http://www.ncbi.nlm.nih.gov/pubmed/22566693

J Biol Chem. 2012 May 7. [Epub ahead of print]

Limonoid Compounds Inhibit Sphingomyelin Biosynthesis by Preventing CERT-dependent Extraction of Ceramides from the Endoplasmic Reticulum.

Hullin-Matsuda F, Tomishige N, Sakai S, Ishitsuka R, Ishii K, Makino A, Greimel P, Abe M, Laviad EL, Lagarde M, Vidal H, Saito T, Osada H, Hanada K, Futerman AH, Kobayashi T.

Source

RIKEN, Japan;

Abstract

In order to identify novel inhibitors of sphingomyelin (SM) metabolism, a new and selective highthroughput microscopy-based screening based on the toxicity of the SM-specific toxin, lysenin, was developed. Out of a library of 2011 natural compounds, the limonoid, 3-chloro-8β-hydroxycarapin-3,8hemiacetal (CHC), rendered cells resistant to lysenin by decreasing cell surface SM. CHC treatment selectively inhibited the de novo biosynthesis of SM without affecting glycolipid and glycerophospholipid biosynthesis. Pretreatment with brefeldin A abolished the limonoid-induced inhibition of SM synthesis suggesting that the transport of ceramide (Cer) from the endoplasmic reticulum (ER) to the Golgi apparatus is affected. Unlike the Cer transporter (CERT) inhibitor HPA-12, CHC did not change the transport of a fluorescent short chain Cer analog to the Golgi apparatus or the formation of fluorescent and short chain SM from the corresponding Cer. Nevertheless CHC inhibited the conversion of de novo synthesized Cer to SM. We show that CHC specifically inhibited the CERT-mediated extraction of Cer from ER membranes in vitro. Subsequent biochemical screening of 21 limonoids revealed that some of them, such as 8β-hydroxycarapin-3,8-hemiacetal (HC) and gedunin [Neem spp. – AGP] which exhibits anti-cancer activity, inhibited SM biosynthesis and CERT-mediated extraction of Cer from membranes. Model membrane studies suggest that HC reduced the miscibility of Cer with membrane lipids and thus induced the formation of Cer-rich membrane domains. Our study shows that certain limonoids are novel inhibitors of SM biosynthesis and suggests that some biological activities of these limonoids are related to their effect on the ceramide metabolism.

PMID: 22566693

http://jn.nutrition.org/content/129/3/628.full.pdf+html -Dietary Pectin Lowers Sphingomyelin Concentration in VLDL and Raises Hepatic Sphingomyelinase Activity in Rats

http://circ.ahajournals.org/content/110/22/3465.abstract - Inhibition of Sphingomyelin Synthesis Reduces Atherogenesis in Apolipoprotein E–Knockout Mice

Pathophysiology. 2004 Oct;11(2):95-101.

Calcification in coronary artery disease can be reversed by EDTA-tetracycline long-term chemotherapy.

Maniscalco BS, Taylor KA.

4730 N. Habana Avenue, Suite 201, Tampa, FL 33614, USA.

Atherosclerosis is a complex process with multiple mechanisms and factors contributing to its initiation and progression. Detection and quantification of coronary artery calcium (CAC) scores with electron beam tomography has been shown to correlate with obstructive and nonobstructive coronary artery disease (CAD). Pathogen-triggered calcification could play a role in CAD. Recent reports suggest that infectious blood nanobacteria (NB) emerge to be such a trigger. So far, minimal or no reversal of atherosclerosis has been claimed by therapies with iv ethylenediaminetetraacetic acid disodium salt (EDTA), antibiotics, or other regimens, and therapies for atherosclerosis remain non-curative. We have now combined EDTA with antibiotic tetracycline (comET), an in vitro proven nanobacteriocidal treatment, and tested comET therapy in patients with documented CAD. Three hypotheses were probed: (1) Are NB present in patients with CAD?; (2) Does treatment with comET affect blood NB antigen and serology?; (3) Does a comET decrease CAC scores? One hundred patients with stable CAD and positive CAC scores were enrolled into a 4 month study of comET therapy. ComET therapy is composed of (1) Nutraceutical Powder (Vitamin C, Vitamin B6, Niacin, Folic Acid, Selenium, EDTA, I-Arginine, I-Lysine, I-Ornithine, Bromelain, Trypsin, CoQ10, Grapeseed Extract, Hawthorn Berry, Papain) 5cm(3) taken orally every evening; (2) Tetracycline HCl 500mg taken orally every evening; (3) EDTA 1500mg taken in a rectal suppository base every evening. CAC scoring was repeated at 4 months and serum samples were analyzed for NB antigen and serology at baseline, 2 and 4 months. Complete blood count, metabolic panel, liver function, C-reactive protein (hs-CRP) and lipids were analyzed at baseline and 4 months. Seventy-seven patients completed the study and all patients were positive for NB serology, antigen or both. Responders (n = 44; 57%) had significant decreases in total CAC scores (P = 0.001), the average decrease being 14%. Non-responders (n = 33; 44%) had no change or had increases in CAC scores. Angina was decreased or ablated in 16 of 19 patients (84%). Lipid profiles improved to non-atherogenic direction significantly (P = 0.001), a remarkable finding in a patient group where 86% were on continuous statin medication already before the trial. No adverse physiologic effects were seen in renal, hepatic, or hematopoetic systems. In conclusion, CAC scores decreased during ComET therapy trial in most CAD patients inferring regression of calcified coronary artery plaque volume. The patients tolerated the therapy well and their angina and lipid profiles improved. Further treatment trials for long term therapy with matched controls are warranted.

PMID: 15364120

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