Abstracts-Triplichol

1. JANA. 2005; 1(8):21-34.

The Use of Fish Oil Supplements in Clinical Practice: A Review.

Guilliams, T.

Increasing dietary consumption of fish high in omega-3 (n-3) fatty acids is well established as a way to improve numerous health outcomes. The prevention of both primary and secondary cardiovascular events, as well as intervention for such unrelated outcomes as depression and rheumatoid arthritis are now linked with n-3 fatty acid intake. Increasing fish consumption is neither an exact science, nor without risk or consuming toxins of various kinds. The advent of highly purified fish oil supplements, now widely available, has allowed very high levels of n-3 fatty acid consumption, fish oil supplements and their fatty acids as it pertains to clinical outcomes, with an emphasis on cardiovascular health.

2. American Heart Association. http://www.americanheart.org

Fish and Omega-3 Fatty Acids. No abstract available.

3. Pantesin. http://www.pantesin.com

About pantesin. No abstract available.

4. Daiichi Fine Chemicals, Inc.

Pantethine: a review of it's CVD-related effects. No abstract available.

5. PDR health. http://www.pdrhealth.com

Pantethine. No abstract available.

6. Lonza.

Sustained-Release Niacin Tablets Dietary Supplements. No abstract available.

7. University of Minnesota, Minneapolis

Effect of Sustained Release Nicotinic Acid on Post-Prandial Lipids In Patients With Coronary Artery Disease. No abstract available.

8. Am J Clin Nutr. 1997 May;65(5 Suppl):1645S-1654S.

n-3 fatty acids and serum lipoproteins: human studies.

Harris WS.

The effects of n-3 fatty acids from fish oils (eicosapentaenoic acid and docosahexaenoic acid) and plant oils (alpha-linolenic acid) on human serum lipids and lipoproteins are reviewed. Studies were included in this review if they were placebo-controlled, crossover, or parallel design studies providing < 7 g n-3 fatty acids/d and with treatment periods of > or = 2 wk duration. Only three studies were available for evaluation of the effects of alpha-linolenic acid on serum lipid concentrations. From these studies it appeared that alpha-linolenic acid (18:3n-3) was equivalent to n-6-rich oils vis-vis lipid and lipoprotein effects. Only when very large amounts of flaxseed oil were fed did the hallmark effect of marine n-3 fatty acids-reduced triacylglycerol concentrations-appear. Thus, in terms of effects on lipoprotein metabolism, the plant-derived n-3 fatty acid is not equivalent to the marine-based acids. More studies using the marine-based acids were examined and summarized. Both crossover (n = 36) and parallel (n = 29) design studies reached the same conclusions: total cholesterol is not materially affected by n-3 fatty acid consumption, low-density-lipoprotein cholesterol concentrations tend to rise by 5-10% and high-density-lipoprotein cholesterol by 1-3%, and serum triacylglycerol concentrations decrease by 25-30%. These effects of marine n-3 fatty acids are now well-established; what remains is to determine the mechanisms behind these effects and, more importantly, their health consequences.

9. Food and Drug Administration. http://www.fda.gov

FDA Authorizes new coronary heart disease health claim for plant sterol and plant stanol esters. No Abstract Available.

10. J Clin Invest. 2004 Sep;114(6):813-22.

Disruption of cholesterol homeostasis by plant sterols.

Yang C, **Yu L**, **Li W**, **Xu F**, **Cohen JC**, **Hobbs HH**.

The ABC transporters ABCG5 and ABCG8 limit absorption and promote excretion of dietary plant sterols. It is not known why plant sterols are so assiduously excluded from the body. Here we show that accumulation of plant sterols in mice lacking ABCG5 and ABCG8 (G5G8-/- mice) profoundly perturbs cholesterol homeostasis in the adrenal gland. The adrenal glands of the G5G8-/- mice were grossly abnormal in appearance (brown, not white) due to a 91% reduction in cholesterol content. Despite the very low cholesterol levels, there was no compensatory increase in cholesterol synthesis or in lipoprotein receptor expression. Moreover, levels of ABCA1, which mediates sterol efflux, were increased 10-fold in the G5G8-/- adrenals. Adrenal cholesterol levels returned to near-normal levels in mice treated with ezetimibe, which blocks phytosterol

absorption. To determine which plant sterol(s) caused the metabolic changes, we examined the effects of individual plant sterols on cholesterol metabolism in cultured adrenal cells. Addition of stigmasterol, but not sitosterol, inhibited SREBP-2 processing and reduced cholesterol synthesis. Stigmasterol also activated the liver X receptor in a cell-based reporter assay. These data indicate that selected dietary plant sterols disrupt cholesterol homeostasis by affecting two critical regulatory pathways of lipid metabolism.

11. PDR Health. http://www.pdrhealth.com

Phytosterols. No Abstract Available.

12. Mayo Clin Proc. 2003 Aug;78(8):965-78

Efficacy and safety of plant stanols and sterols in the management of blood cholesterol levels.

Katan MB, **Grundy SM**, **Jones P**, **Law M**, **Miettinen T**, **Paoletti R**; **Stresa Workshop Participants**.

Foods with plant stanol or sterol esters lower serum cholesterol levels. We summarize the deliberations of 32 experts on the efficacy and safety of sterols and stanols. A meta-analysis of 41 trials showed that intake of 2 g/d of stanols or sterols reduced low-density lipoprotein (LDL) by 10%; higher intakes added little. Efficacy is similar for sterols and stanols, but the food form may substantially affect LDL reduction. Effects are additive with diet or drug interventions: eating foods low in saturated fat and cholesterol and high in stanols or sterols can reduce LDL by 20%; adding sterols or stanols to statin medication is more effective than doubling the statin dose. A meta-analysis of 10 to 15 trials per vitamin showed that plasma levels of vitamins A and D are not affected by stanols or sterols. Alpha carotene, lycopene, and vitamin E levels remained stable relative to their carrier molecule, LDL. Beta carotene levels declined, but adverse health outcomes were not expected. Sterol-enriched foods increased plasma sterol levels, and workshop participants discussed whether this would increase risk, in view of the marked increase of atherosclerosis in patients with homozygous phytosterolemia. This risk is believed to be largely hypothetical, and any increase due to the small increase in plasma plant sterols may be more than offset by the decrease in plasma LDL. There are insufficient data to suggest that plant stanols or sterols either prevent or promote colon carcinogenesis. Safety of sterols and stanols is being monitored by follow-up of samples from the general population; however, the power of such studies to pick up infrequent increases in common diseases, if any exist, is limited. A trial with clinical outcomes probably would not answer remaining questions about infrequent adverse effects. Trials with surrogate end points such as intima-media thickness might corroborate the expected efficacy in reducing atherosclerosis. However, present evidence is sufficient to promote use of sterols and stanols for lowering LDL cholesterol levels in persons at increased risk for coronary heart disease.