ATHEROSCLEROSIS Journal Article

THE FOLLOWING MEDICAL AND SCIENTIFIC MATERIAL IS FOR CONSUMER INFORMATIONAL AND EDUCATIONAL PURPOSES ONLY-- UNDER SECTION 5 OF DSHEA.

In December of 2007 an article was published in the journal ATHEROSCLEROSIS regarding a study done on our Liposomal Glutathione (LG). The study was conducted at the prestigious Lipid Research Laboratory, Technion Faculty of Medicine at the Rambam Medical Center in Haifa, Israel.

The study shows that LG has impressive antioxidant properties. This was determined by its ability to inhibit the progression of oxidation of both LDL and HDL cholesterol in an in-vitro model using cholesterol from human blood and exposed to an oxidizing metal.

Then the study looked at ApoE-deficient mice. These mice have become one of the standard animal models used to research atherosclerosis because they have very high levels of cholesterol and oxidative stress.

The study mice were fed LG for two months. During this time the levels of cholesterol decreased by about 20%.

LG ingestion in this mouse model not only resulted in the significant reduction in cholesterol levels, it slowed cholesterol biosynthesis and increased release of HDL cholesterol from macrophages, resulting in significant slowing of the atherosclerotic process.

Here is the abstract of that article:

Anti-oxidant and anti-atherogenic properties of liposomal glutathione: studies in vitro, and in the atherosclerotic apolipoprotein E-deficient mice.
Rosenblat M, Volkova N, Coleman R, Aviram M.
Atherosclerosis. 2007 Dec;195(2):e61-8.

Abstract: Liposomal glutathione, but not the control liposomes (with no glutathione), dose-dependently inhibited copper ion-induced low density lipoprotein (LDL) and HDL oxidation. As peroxidase activity was found to be present in both LDL and HDL, it has contributed to the anti-oxidative effects of liposomal glutathione. In-vitro, no significant effect of liposomal glutathione on J774 A.1 macrophage cell-line oxidative stress and on cellular cholesterol metabolism was observed. In contrast, in the atherosclerotic apolipoprotein E-deficient (E(0)) mice, consumption of liposomal glutathione (12.5 or 50mg/kg/day, for 2 months), but not control liposomes, resulted in a significant reduction in the serum susceptibility to AAPH-induced oxidation by 33%. Liposomal glutathione (50mg/kg/day) consumption also resulted in an increment (by 12%) in the mice peritoneal macrophages (MPM) glutathione content, paralleled by a significant

reduction in total cellular lipid peroxides content (by 40%), compared to placebo-treated mice MPM. MPM paraoxonase 2 activity was significantly increased by 27% and by 121%, after liposomal glutathione consumption (12.5 or 50mg/kg/day, respectively). Analyses of cellular cholesterol fluxes revealed that, liposomal glutathione (12.5mg/kg/day) consumption, decreased the extent of oxidized-LDL (Ox-LDL) uptake by 17% and the cellular cholesterol biosynthesis rate, by 34%, and stimulated HDL-induced macrophage cholesterol efflux, by 19%. Most important, a significant reduction in macrophage cholesterol mass (by 24%), and in the atherosclerotic lesion area (by 30%) was noted. **We thus conclude that liposomal glutathione possesses anti-oxidative and anti-atherogenic properties towards lipoproteins and macrophages, leading to attenuation of atherosclerosis development.** PMID: 17588583