Nutritional Influences on Illness

by Melvyn R. Werbach, MD

4751 Viviana Drive • Tarzana, California 91356 USA
Phone 818-996-0076 • Fax 818-774-1575

The Influence of Minerals on Atherosclerosis
Part One

Calcium Balance

Intracellular calcium regulates many of the physiological, biochemical, and molecular processes known to function abnormally in atherosclerosis. Moreover, increased intracellular calcium levels appear to be causally related to atherogenesis.[1]

While human studies are few, both inadequate[2] and excessive[3] calcium intake have been blamed for promoting the development of atherosclerosis. The evidence appears stronger for inadequate calcium intake since, in the elderly, low calcium and vitamin D intake, short solar exposure, decreased intestinal absorption, and falling renal function all contribute to calcium deficiency, secondary hyperparathyroidism, bone loss, and possibly calcium shift from the bone to soft tissue and from the extracellular to the intracellular compartment. Calcium deposition in the vascular wall then promotes the development of atherosclerosis and hypertension.[2]

Consistent with this hypothesis is a study that found elevated plasma parathyroid hormone levels in almost half of a group of 56 patients with coronary heart disease. Moreover, parathyroid hormone levels were highest in those patients whose disease affected three coronary vessels and in patients who had sustained a myocardial infarction.[4]

Supplementation

In an animal model of atherosclerosis, calcium supplementation was found to protect against atherogenesis. Beneficial effects included a prolongation of clotting time, decreased platelet aggregation, decreased total plasma cholesterol, and slightly decreased plasma triglycerides with a greatly increased fecal excretion of fecal lipids and saturated fatty acids.[5,6]

In a controlled human trial, calcium supplementation decreased total and LDL cholesterol in hyperlipidemic patients. While bile acid excretion was unaffected, the excretion of saturated fat more than doubled, perhaps because supplemental calcium facilitates the incorporation of free saturated fatty acids into micelles with bile acids for excretion, thus reducing the absorption of saturated fats.[7]

Chromium Deficiency

Research suggests that inadequate chromium nutriture, because of its involvement in lipid and glucose metabolism, increases the risk of atherosclerosis.[8] Aortic tissue of victims of coronary heart disease has decreased chromium levels,[9] and toenail chromium is inversely associated with the risk of a first myocardial infarction in men.[10] Even though plasma chromium levels are not in equilibrium with tissue stores, low plasma chromium levels suggest the presence of coronary atherosclerosis.[11]

Supplementation

In controlled human trials, chromium supplementation is often effective in lowering serum triglycerides,[12] and both total and LDL cholesterol,[13] while raising HDL cholesterol.[12] Both inorganic and organic (Brewer's yeast; "GTF chromium") forms of chromium were used successfully in these studies. As the efficacy of chromium supplementation in normalizing blood lipids has varied between studies, Mertz has suggested that only certain cases of abnormal lipid metabolism – possibly those specifically related to impaired glucose tolerance – are related to chromium deficiency.[14]

Copper Balance

Copper-deficient animals are hypercholesterolemic, hyperuricemic, have glucose intolerance and display EKG abnormalities. Their hearts and arteries have abnormal connective tissue, lipid deposits, and inflammatory changes.[15]

Moreover, copper-deficient female mice develop alterations in blood clotting mechanisms which could increase the risk of coronary heart disease.[16] Not only are low copper diets common in the industrialized nations, but some have ratios of zinc to copper greater than those that have produced hypercholesterolemia in animals.[15]

When healthy men were fed low-copper diets, LDL cholesterol increased and HDL cholesterol declined. These changes reversed after they received copper supplements.[17] Furthermore, atherosclerotic lesions have low copper concentrations.[9,18]

Several prospective studies have found an elevated serum copper concentration correlates directly with the risk of atherosclerosis and myocardial infarction.[19] Serum copper, however, may fail to reflect either copper intake or organ copper. In fact, animal experiments reveal that a high serum copper may actually reflect low copper nutritional status.[20]

Excessive dietary copper, like copper deficiency, *is* associated with hyperlipidemia and hypertension,[21] both important risk factors for atherosclerosis. It appears that an optimum level of copper is required to maintain antioxidant defenses; both copper excess and copper deficiency increase antioxidant stress.[22]

Iron Balance

A recent prospective study of middle-aged women found that relatively high dietary haem iron (found in red meat, seafood, and poultry) was associated with an increased risk of coronary heart disease.[23] Also, in a 17-year prospective study, men with elevated serum iron levels (>175 mg/dL) had over twice the relative risk of a fatal myocardial infarction, while women had more than five times the relative risk.[24]

These findings contrast with those of another prospective study of elderly subjects, which found consistent evidence of an increased risk of coronary artery disease at lower serum iron levels.[25] Thus, both excessive and deficient iron nutriture appear to promote atherosclerosis. In the industrialized nations, however, it appears that – for the elderly – excessive dietary iron carries a much greater risk to cardiovascular health.[26]

References

1. Phair RD. *Cell Calcium.* 1988; 9(5-6):275-84
2. Fujita T. *J Nutr Sci Vitaminol.* Tokyo, Japan. 1985; 31 Suppl S15-9
3. Seely S. *Int J Cardiol.* 1991; 33(2):191-8

◄ *continued on page 147*