Omega-6 Polyunsaturated Fatty Acids

There is a large body of literature suggests that higher intakes of omega 6 polyunsaturated fatty acids (PUFAs) reduce risk for coronary heart disease (CHD). The American Heart Association just published a science advisory in Circulation on January 27,2009 titled “Omega-6 Fatty Acids and Risk for Cardiovascular Disease”. There are some noted physicians and medical groups that have recommended substantial reductions in the intake of PUFAs due to this possible association.

Linoleic Acid (LA) is the primary dietary O-6 PUFA and cannot be synthesized by human beings. Therefore it has to come from food sources. While there are no firm requirements for the amount of daily intake, it is felt that .5-2% of the total energy is sufficient. The LA can be converted into something called linolenic acid. This can be converted into the metabolically active O-6 PUFA called arachidonic acid (AA). This is very important as I will explain a little later. LA comes primarily from vegetable oils like corn, sunflower, safflower, and soy. According to the National Health and Nutrition Survey data indicates that usual intake of LA in adults greater than or equal to 19 years of age is 6.7% of energy.

The arguments for the reductions in the intake are based on the assumption that because there is an inflammatory component to CAD and AA is the substrate for the production of a variety of proinflammatory mediators, reduction in the intake of LA should reduce the inflammatory potential and therefore lower the risk for CAD. Despite this fact, O-6 PUFAs also have anti-inflammatory properties. In one study, when healthy volunteers were given 7 times the usual intake of AA in a 7-week controlled feeding study, no negative inflammatory response was observed. Other studies also support this finding. At present, there is little direct evidence of a net proinflammatory, proatherogenic effect of LA in humans.

The cholesterol lowering effect of LA is well established. Epidemiologically, the replacement of 10% of calories from saturated fatty acids with O-6 PUFAs is associated with an 18 mg/dl decrease in LDL cholesterol which is greater than that observed with similar replacement with carbohydrate. These findings confirm a beneficial effect on cholesterol lowering of O-6 PUFAsbeyond that produced by the removal of saturated fatty acids. Favorable effects of higher LA intake on cholesterol levels are thus well documented and would predict significant reductions in CAD risk. Nevertheless, not all studies support a beneficial effect of LA of coronary heart disease risk markers. For example, an angiographic study published in 2004 reported a direct association between PUFA intakes and artery narrowing in women with CAD. Like many studies, this particular study did not evaluate outcomes so indeed it is possible that this association did not result in increased morbidity and mortality.

In summary, AHA advisory was undertaken to summarize the current evidence on the consumption of PUFAs and CAD risk. Data from randomized trials, case-control and cohort studies, and long term animal feeding experiments indicate that eating at least 5-10% of energy from O-6 PUFAs reduces the risk in relation to lower intakes. The data also suggest that higher intake seems to be safe and even more beneficial. To reduce PUFA levels from their current levels would be more likely to increase than to decrease risk for CAD.