Perspectives on omega-3 fatty acids

Viewpoint

The following article examines omega-3 fatty acids derived from vegetable sources. It was compiled from information provided by Patricia Johnston, University of Illinois professor, who spoke on this topic at the North Central AOCS Section's symposium on "Dietary Aspects of Fats and Oils" on March 17 (see related article in "Inside AOCS") in Champaign, Illinois, and by J. Edward Hunter of Procter & Gamble, who wrote an article on this topic for the March 1987 issue of n-3 News.

PAT JOHNSTON:

In the past year, there has been a flood of news items in the press and on television and radio about the health benefits of omega-3 fatty acids. Almost without exception, the sources of omega-3 fatty acids described are fish, fish oils and fish oil concentrates. The emphasis, therefore, has been on the elongated, desaturated fatty acids of the omega-3 family: 5,8,11,14,17-eicosapentaenoic acid (EPA or $20:5\omega 3$) and 4,7,10,13,16,19-docosahexaenoic acid (DHA or $22:6\omega 3$). Seldom has there been mention of vegetable oil sources of the parent of the family 9,12,15-octadecatrienoic or alpha-linolenic $(18:3\omega 3)$ acid.

Some of the beneficial effects of omega-3 fatty acids, such as protection against "sticky" blood platelets and therefore risk of heart attack, seem to be mediated by EPA, which competes with arachidonic acid (20:4 ω 6) for the enzyme leading to the production of prostaglandins and thromboxanes. As a consequence of this competition, fewer of the platelet-aggregating eicosanoids, such as thromboxane A₂, are produced. There is also evidence that weakly aggregatory 3-series prostaglandins and thromboxane are produced from EPA. The overall effect is less likelihood of life-threatening thrombi formation.

While there is no doubt that the ingestion of fish and fish oil containing the preformed EPA leads to a more prompt and profound increase in body tissue EPA, the use of vegetable sources of the omega-3 parent, alpha-linolenic acid, should be given thought as well, and the possible disadvantages of fish oil concentrates in particular should be addressed.

Linseed oil, which contains as much as 60% alpha-linolenic acid and about 20% linoleic (18:2 ω 6) acid, currently is not used as an edible oil in the United States, although the U.S. Department of Agriculture Handbook No. 8 lists linseed as an edible oil. Additionally, the oil from a genetic variant of rapeseed-canola oil-has now been introduced into the U.S., and this contains about 10% alpha-linolenic acid. We and others have shown that feeding linseed oil and linseed/ soybean mixtures to laboratory animals effectively reduces the production of prostaglandins derived from arachidonic acid (2series prostaglandins), makes platelets less sticky and modulates immune cell function.

It has been claimed that dietary alpha-linolenic acid in man is poorly converted to EPA and DHA. This claim has been based on analysis of serum fatty acid profiles, which largely reflect the dietary fatty acid pattern and not the elongation/desaturation in various cells. Indeed, it has been shown that intake of an alpha-linolenic acid-rich margarine leads to a small but significant increase in platelet EPA in man with striking antiaggregatory effects. Moreover, an absolute increase in alpha-linolenic acid in human adipose tissue has been associated with a decrease in both systolic and diastolic blood pressure. While linseed oil may not become a popular edible oil in the U.S., incorporation of the flaxseed from which it is produced into baked and other food products is a possible way of increasing alphalinolenic consumption.

While the facts are certainly not all in, there is reason to believe that the use of vegetable sources of

alpha-linolenic acid may presently be preferable to the consumption of fish oil concentrates. As pointed out by the Wellness Letter of the University of California at Berkeley, the following possibilities exist. High intake of fish oil could lead to dangerous anticlotting effects in the event of accident or surgery. Fish oil concentrates may contain contaminants such as pesticides, especially if prepared from fish livers. Some fish oils, such as cod liver oil, are high in vitamin A and D. which are toxic in excess. Ingestion of fish oil over a long period of time can lead to stress on the vitamin E status. Some supplements, however, do contain vitamin E.

Other possibilities come to mind. For example, does the long-term consumption of the highly unsaturated fish oil supplements lead to increased peroxide formation in vivo?

Until further research is done, most nutritionists and public health officials appear to favor fish over fish oil concentrates as a dietary source of omega-3 fatty acids. In the meantime, flaxseed in foods and high alpha-linolenic oils may be a safe alternative.

J. EDWARD HUNTER:

The consumption of fish and fish oils has been linked to possible health benefits, such as reduced risk for coronary heart disease, attributed largely to the omega-3 fatty acids present in fish oils. Fish oils, however, are not the only source of omega-3 fatty acids.

Other sources include low erucic acid rapeseed oil (LEAR oil, commonly known as canola oil) and soybean oil. LEAR oil, available in Canada for a number of years and approved in 1985 by the Food and Drug Administration (FDA) for U.S. food use, contains the omega-3 fatty acid alpha-linolenic acid, typically at about 10–12% of total fatty acids. Unhydrogenated soybean oil generally contains alphalinolenic acid at about 7% of total fatty acids. Fish oils usually have higher total omega-3 fatty acid content (typically about 20-25% of total fatty acids) than canola or sovbean oils.

On the other hand, fish oils have poor palatability, except perhaps in capsule form, and are significant sources of cholesterol, while canola and soybean oils are highly palatable as salad and cooking oils and contain no cholesterol.

A number of studies have confirmed that alpha-linolenic acid from LEAR oil can be converted by humans to EPA. Meanwhile, the abundance of omega-3 fatty acids in certain animal cells, particularly in the brain and retina, has stimulated the belief that omega-3 fatty acids may have an essential function in warm-blooded animals. Work by R.T. Holman and colleagues, for example, has suggested that alpha-linolenic acid or its metabolic products may have a role in neurological function in humans. As a result, linolenic acid requirements for humans have been estimated at 0.54% of calories.

M. Neuringer and coworkers have shown that young rhesus monkeys whose mothers were fed diets deficient in alpha-linolenic acid during pregnancy had significantly reduced visual acuity. This was apparently the first indication that physiological deficits may result from an omega-3 fatty acid deficiency in primates. Whether human infants also need alphalinolenic acid is not known. However, it is known that human milk contains alpha-linolenic acid, EPA and DHA.

The ability of humans to convert alpha-linolenic acid to EPA suggests that vegetable oils containing alpha-linolenic acid may have biological effects similar to those of fish oils.

In one study, S. Renaud and coworkers asked subjects to change their diet for one year by replacing butter and cream with oil and margarine rich in alpha-linolenic acid from LEAR oil. This change increased their alpha-linolenic acid intake from approximately 1.2 grams a day, or 0.37% of calories, to about 3.3 grams a day, or 1.0% of calories. The results after one year showed EPA in platelet phospholipids increased to 0.52% from 0.36%. Although moderate, these changes were associated with marked reduction in platelet aggregation.

B. Jacotot and coworkers similarly found that feeding human subjects a diet in which two-thirds of the lipids was composed of LEAR oil resulted in higher serum phospholipid EPA levels compared to feeding a sunflowerseed oil diet. These investigators, however, did not assess possible changes in platelet aggregation.

While there are no published studies that address whether alphalinolenic acid intake from vegetable (LEAR) oil diets is sufficient to benefit health, inferences can be drawn from work by D. Kromhout and colleagues published in the New England Journal of Medicine in 1985 and other studies by Renaud and colleagues published in The Lancet in 1983 and the American Journal of Clinical Nutrition in 1986. From these, one can hypothesize that an oil providing about 2.1 grams of alpha-linolenic acid per day, equal to about 1.5 tablespoons

of canola oil or 2 tablespoons of soybean oil per day, could have a biological effect similar to that of 30 grams of fish per day. If so, using a salad and cooking oil made from canola oil may be equivalent to a partial serving of fish, in terms of its ability to reduce platelet aggregation and thus lower risk of thrombosis. This hypothesis is consistent with a suggestion by W.E.M. Lands in Fish and Human Health to substitute some linolenic acid for much of the linoleic acid in the current U.S. diet to reduce the formation of arachidonate-derived prostaglandin promoters of platelet aggregation. Also, Berry and Hirsch, reporting that an absolute 1% increase in linolenic acid in human adipose tissue was associated with a 5-mm Hg decrease in the systolic, diastolic and composite mean arterial blood pressure, have suggested that increased dietary consumption of alpha-linolenic acid is associated with reduced blood pressure.

Although there is relatively little information on the biological effect of omega-3 fatty acids from vegetable oils in humans, the data that are available indicate dietary alphalinolenic acid may be associated with reduced platelet aggregation and blood pressure. These effects apparently are related to actions of prostaglandins derived from EPA, an omega-3 fatty acid in fish oils and produced by humans from alpha-linolenic acid in certain vegetable oils. Thus, individuals wishing to increase their intake of omega-3 fatty acids should consider vegetable oil, as well as fish oil, sources.

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