Dietary Fat for the Elderly: What Are the Issues?

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Several major chronic diseases that afflict the elderly are in part related to excess dietary fat. This needs to be balanced against the nutritive value of fat and the tendency for elderly people to eat less fat by choice. Recommendations to the elderly on how much fat they should eat are therefore complex. What type of fat to eat is simpler.

Food lipids have many nutritional and biological functions as well as organoleptic properties. In the past, people have selected fat-containing foods for the flavor and texture and not the nutritive value, which fat imparts. Fats are a major source of energy, the source of the fat-soluble vitamins (A, D, E, and K) that have numerous regulatory effects, and the source of two essential fatty acids, linoleic and α-linolenic acids. The n-6 and n-3 fatty acids generate the potent eicosanoids, cellular signal transduction is influenced by inositol phosphatides, and cholesterol is the precursor of bile acids and of steroid hormones. The question is whether a significant reduction in dietary fats would jeopardize the adequate intake of these fat-dependent nutrients.

FOOD CONSUMPTION PATTERNS

Elderly people tend to eat less (1) partly in response to reduced physical activity, and partly for social and health reasons. Appetite and smell decline, while loneliness, depression, and inability to buy and prepare food reduce the incentive to eat adequately. Eating less fat is common and along with this goes the possibility of eating fewer fat-soluble vitamins. This possibility has been investigated with the general finding that in healthy elderly people there is little evidence for deficiency in vitamins A, D, and E (1). Nevertheless, the average consumption of vitamin D and of calcium is marginal and below recommended daily intakes in up to one-quarter of the older Australian population (2).

Dr. Katrine Baghurst in the Division of Human Nutrition, CSIRO, Adelaide, has unpublished data on the profile of fat consumption in older Australians. Total fat consumption is only a little below the national average for adults (33% vs. 35% of
FIG. 1. Falls in systolic blood pressure (SBP) and in diastolic blood pressure (DBP) after 6 weeks of four dietary interventions. The two cross-over periods are: (i) a low sodium intake together with either fish oil or sunflower oil; (ii) a normal sodium intake together with either fish oil or sunflower oil. (Number of subjects in parentheses.)

dietary energy). The proportions derived from saturates, monounsaturates, and polyunsaturates also resemble the national profile. Dietary cholesterol consumption is below average (230 mg for women and 290 mg for men), reflecting recent dietary changes in the direction of less animal fat (Fig. 1). The calculated intakes of vitamins A and E are adequate. This contrasts with McGandy’s findings in elderly people in Boston (3), who eat substantially reduced amounts of fat and calcium.

OVERWEIGHT

Lean body mass decreases and fat mass increases with aging. This probably reflects lower physical activity and possibly reduced resting metabolic rate (4). These factors predispose to obesity and raise questions about the role of total fat intake in the process of becoming fat and whether obesity is an important health risk in older people.

A recent prospective survey of men and women aged 55 to 74 years at entry found no additional risk for women attributable to overweight, provided there was no other clinical condition related to fatness (5). Among men who were frankly obese (>30 kg/m²), the relative risk was slightly increased over the 8.7 years of follow-up (1.1–1.2). However, low body weight (<22 kg/m²) was associated with substantial increases in mortality and morbidity that were independent of smoking. This important finding, observed in other prospective studies, raises the question whether restricting dietary fat is advisable for older people who are otherwise free of disorders that are adversely influenced by fat.
LIPID METABOLISM AND CORONARY HEART DISEASE

The major public health indication to change the nature of dietary fatty acids relates to lowering plasma cholesterol levels and consequently reducing coronary heart disease. Until recently, this was taken to be pertinent only to younger adults but this view is now being recast to include older people as well.

The first question to ask is whether cholesterol metabolism changes with aging, making a rise in the plasma cholesterol concentration inevitable. It should be noted that in some non-Western societies this rise does not occur (6). It has been postulated that the activity of the low-density lipoprotein (LDL) receptor declines with age but in the absence of a reliable assay in a readily available human tissue, we have no answer. It seems likely, though, that diminished LDL removal rather than overproduction is the cause of the age-related cholesterol rise.

The importance of this is that the plasma cholesterol, or more properly the LDL cholesterol concentration, remains an independent risk factor into old age. The Honolulu Heart Study showed this to be the case in men older than 65 years (7); this is confirmed in the most recent analysis of the Framingham cohort (8).

Equally, elevated high-density lipoprotein (HDL) cholesterol confers protection in older as well as in younger people (9). Interestingly, octogenarians in the Framingham study were exceedingly unlikely to have reduced HDL cholesterol levels (10). A cross-sectional survey of 1280 80-year-old men in Sweden found them to have a moderately low average plasma cholesterol concentration, suggesting selective mortality (11). Nevertheless, even at such an old age raised LDL cholesterol levels predicted cardiovascular disease. The implications are that national strategies to counter coronary disease by reducing the average cholesterol level also apply to the elderly.

Raised plasma triglyceride levels are now also generally recognized as increasing cardiovascular risk, more so in women than in men (12). This is particularly true for aortofemoral atherosclerosis and when hypertriglyceridemia occurs as part of a lipoprotein phenotype which includes low HDL and/or raised LDL cholesterol (12). The rise in plasma triglyceride that accompanies aging therefore deserves remedial action.

Apart from increased fat mass, another recognized reason for the rising triglyceride concentration is reduced removal. The key control enzyme, lipoprotein lipase, falls with aging (13) and this is reflected in the prolongation of lipemia after a fat meal (14). If LDL receptor activity declines with aging, then the clearance of very low density lipoprotein (VLDL) remnants, derived from endogenous synthesis of triglyceride, will also be impaired. This in turn will aggravate the removal of chylomicron remnants, since the two products of triglyceride catabolism compete for common degradation sites.

The strategy to manage hyperlipidemia is identical to that in the general population and includes a reduction in saturated fatty acid and substitution by a mix of polyunsaturated and monounsaturated fatty acids and complex carbohydrates.
POLYUNSATURATED FATTY ACIDS AND EICOSANOIDS

It has been suggested that polyunsaturated fatty acids, mainly linoleic acid, may pose special problems, particularly in the elderly. Quite clearly, there are benefits in terms of cholesterol reduction. The n-3 polyunsaturated fatty acids (α-linolenic, eicosapentaenoic or EPA, and docosahexaenoic or DHA) also have advantages and disadvantages. The correct balance of these two classes of fatty acids is a major challenge for nutritionists and food manufacturers. These points are discussed later.

The most important metabolites of polyunsaturated fatty acids are the eicosanoids. The nature of eicosanoids is profoundly affected by the type of dietary fatty acids. Since these products influence cardiovascular functions, thrombosis, inflammation, immunity, and probably tumor promotion, the importance of optimizing dietary fatty acids is particularly relevant in the elderly. Several of the key cellular events in atherogenesis are also affected by eicosanoids.

Eicosanoids comprise prostanoids (prostaglandins and thromboxanes) and leukotrienes. Depending on the number of double bonds, these metabolites are classified as belonging to the 1-, 2-, or 3-series. The 2-series of prostanoids (PG\textsubscript{12} and TXA\textsubscript{2}) is derived from linoleic acid and is the most potent. The 3-series, derived from EPA and DHA, is much less potent and indeed reduces the activity of the 2-series. It is therefore clear that the ratio of the 2- and 3-series and hence of their precursors, the n-6 and n-3 fatty acids, will determine key biological functions and influence many of the diseases of aging. For instance, immune responses are enhanced when tissue linoleate levels fall. Most of the evidence derives from experimental animals in which high intakes of linoleic acid (and low intakes of n-3 fatty acids) suppress immunological competence and hence interfere with the control of tumor growth.

Experimental atherogenesis is also inhibited by n-3 fatty acids derived from fish oils (15). This results from suppression of several processes: attraction of lipid-laden circulating monocytes to the arterial endothelium, adhesion of those cells to endothelium, aggregation of platelets, and production of local growth factors.

The role of the 1-series of prostanoids, especially PGI\textsubscript{1}, is controversial. The initial step in the conversion of linoleic acid to arachidonic acid (the immediate precursor of the eicosanoids) is the insertion of a third double bond to produce γ-linolenic acid (still an n-6 fatty acid). This step is mediated by the enzyme Δ6-desaturase. It has been claimed that this enzyme declines with age (16) and that this relative deficiency gives rise to several chronic disorders that are common in the elderly. γ-Linolenic acid gives rise to dihomo-γ-linolenic acid, which can give rise to PGI\textsubscript{1} and/or be further desaturated to arachidonic acid. PGI\textsubscript{1} is claimed to have functions that oppose PGI\textsubscript{2}. Further linoleic acid can competitively inhibit 6-desaturase activity (17).

Recently Abraham et al. (18) have reported low adipose levels of dihomo-γ-linolenic acid in men with coronary heart disease. Based on these observations and assumptions, Horrobin and Manku (19) have recommended increased consumption of γ-linolenic-acid–rich oils (black currant, evening primrose). The controversy can only be clarified through experiments in which purified γ-linolenic or dihomo-γ-linolenic acids are eaten.
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Polyunsaturated fatty acids appear to influence the outcome of another common cause of death in the elderly: sudden cardiac arrest. Further, the extent of myocardial infarction, and the consequences of prolonged ischemia such as myocardial stunning, may also be favorably limited by fish n-3 fatty acids.

Apart from the well known low incidence of cardiac deaths in populations with high fish consumption, there is also evidence that the EPA content of blood platelets is inversely related to clinical coronary heart disease (20). Men who had died suddenly from cardiac causes were found to have reduced DHA:arachidonic acid ratios in cardiac phospholipids (21). Finally, the single intervention study with fish and fish oils in men after myocardial infarction suggested reduction in sudden cardiac deaths (22).

N-3 fatty acids may therefore protect against tachyarrhythmias and minimize infarction. Supporting evidence of this has been obtained in experimental animal studies. Animals that were fed fish oil and subsequently subjected to myocardial ischemia have shown less damage to muscle (23), and low mortality from arrhythmias (24). Some of the protective mechanisms include diminished oxygen-related free radicals, prevention of abnormal calcium fluxes, leukocyte infiltration of myocardium, and lessened eicosanoid production (25).

Importantly, McLennan et al. (24) have shown that aging predisposes the rat heart to ventricular arrhythmia and that this is preventable by prefeeding with fish oil.

We have found that marine n-3 fatty acids lower blood pressure potently in elderly people (26). This was seen in the context of a moderate reduction of salt intake, probably because the elderly tend to be salt-sensitive. In 105 men and women aged 60 to 80 years, a combination of 4.2 g n-3 fatty acid (2.5 g EPA + 1.7 g DHA) plus an 80-mmol reduction in NaCl, lowered systolic and diastolic pressure by 7.3 and 5.3 mmHg, respectively. The study was carried out in a double-blind cross-over design, using a normal NaCl intake or sunflower oil for comparison (Fig. 1). We believe that EPA and DHA negate the hormonal and circulatory compensating mechanisms through which volume and pressure are normally maintained during salt deprivation.

It is nevertheless important that some of the potential hazards of excessive fish n-3 fatty acids are recognized, among which the increased tendency to bleed may become more easily manifest in elderly people. The suppression of inflammatory responses that may be beneficial in some disease states may be undesirable at other times.

N-3 fatty acids may adversely affect another common disorder of the elderly: glucose intolerance, reflecting resistance of tissues to the action of insulin. It may lead to frank diabetes mellitus and to hyperlipidemia [mostly hypertriglyceridemia, low HDL, and raised apolipoprotein B (15)]. This may be aggravated by the use of n-3 fatty acids because insulin secretion may be inhibited (27).

Another postulated adverse consequence of eating polyunsaturated fatty acids is the generation of oxidized lipoproteins. The concern comes from the heightened atherogenicity of oxidized LDL; whether aging predisposes to oxidation is uncertain. A preliminary report suggests that this may be so at least for elderly women taking
Lipid peroxides rose in the plasma to higher levels in the older than in the younger women, but this could not be related to lesser availability of vitamin E (a natural antioxidant).

It has frequently been suggested that linoleic acid may promote cancer. Whereas the association between total fat consumption and several human cancers is supported in epidemiological surveys, there is no clear link with fatty acid type. Although experimental tumors in animals can be promoted by high linoleate diets, there is little to suggest that this is so in humans (29). Even the often quoted finding of increased colon cancer among elderly men in the Los Angeles diet trial was subsequently overturned by the original authors (30). Careful scrutiny of similar trials showed no evidence for a dietary linoleic acid–cancer link (29). At the same time, the possibility that a high n-3 fatty acid intake may be protective must be considered. A preliminary report from the Multi Risk Factor Intervention Trial (MRFIT) has reported an inverse relationship between consumption of either plant or fish n-3 fatty acids and cancer (31).

ESSENTIAL FATTY ACIDS

The requirements for linoleic acid and for α-linolenic acid are relatively small given the high intakes of both in Western societies. There is no evidence that the need for these two essential fatty acids changes with aging. The average consumption of linoleic acid in Western diets ranges between 4% and nearly 10% of daily energy, far in excess of the 1% to 2% needed. The amount of α-linolenic acid (n-3) that gives rise to EPA and DHA is less than 0.5% of total energy and is present in abundance in plants. It is now generally accepted to be an essential fatty acid also.

RECOMMENDATIONS

1. The elderly can participate in the general population strategy for a modest reduction in fat consumption. The objective is to reduce the risk of coronary heart disease by lowering plasma cholesterol, which remains a risk in older people. Cancer risk may also become less.
2. However, elderly people should not reduce fat intake markedly. They tend to eat less by choice so that further restriction of fat may lead to nutritional deficiency.
3. Weight reduction through fat restriction is not justified in the absence of obesity-related risk factors. Abnormally low body weight carries greater mortality in the elderly than overweight.
4. The nature of dietary fatty acids should resemble that advised for the population as a whole: less saturated fat and more unsaturated fat. However, there is a strong case for reducing the n-6:n-3 ratio, that is, increasing the proportions of n-3 fatty acids from plants and fish. Excess linoleic acid may interfere with immunological processes and aggravate inflammatory responses, atherogenicity, thrombosis, and possibly tumor formation, disorders to which the elderly are prone.
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5. Lifestyle measures such as physical activity should be encouraged to stimulate normal eating and reduce the risk of nutrient inadequacy. However, it is most unlikely that the elderly are at risk from essential fatty acid deficiency.

REFERENCES


**DISCUSSION**

*Dr. Kritchevsky:* Could you summarize Dolachek’s findings on n-3 fatty acids in the MRFIT study?

*Dr. Nestel:* The findings relate to 18,000 people in the MRFIT study who took part in nutrition surveys and intervention studies (1). There was a twofold difference in mortality from coronary heart disease (CHD) and cancer in favor of n-3 fatty acids between the lowest and the highest n-3 intake quintiles. When other risks such as high blood pressure, unfavorable plasma lipids, and smoking were taken into account, a greater benefit from n-3 fatty acids was shown.

*Dr. Meredith:* Canola oil is becoming very popular. Do you have any reservations about older people increasing their intake of this type of oil?

*Dr. Nestel:* If you asked me what was the optimal fatty acid mix in the diet, I should say it closely resembles canola oil, which has 9% saturated fatty acids, 50% oleic acid, and a good ratio of linoleic to linolenic acids. I do not believe there is a case for restricting fat in the elderly unless there is a specific reason to do so. Of all the oils, I think canola oil may be the most desirable.

*Dr. Rush:* Would you comment on the potential entry into the food supply of sucrose polyester and what it might do to vitamin requirements?

*Dr. Nestel:* For the population as a whole, I don’t have any major reservations, but in elderly people taking a restricted energy intake, and hence a restricted fat intake, a further reduction in available fat solubility for fat-associated nutrients would be compromised by sucrose polyester.

*Dr. Stern:* I feel that elderly people should be advised to lower their fat intake, but the reason should be to limit empty calories and thus help maintain their intake of essential nutrients, rather than, for example, to lower their total blood cholesterol. What are your views on this?

*Dr. Nestel:* I am not so concerned about the quantity of fat but rather about the fatty acid mix within the dietary fat. I have looked at this very narrowly from the point of view of healthy individuals with no relevant risk factors. Nevertheless, I think one should be considering what a high fat diet might be doing to glucose intolerance, which is obviously a common condition in the elderly, or what its role might be in cancer of the colon. These matters are still under review. My feeling at present is that there is no urgency for the elderly to lower their fat intake but that we should be thinking about altering the nature of the fatty acids.
Dr. Kritchevsky: Could you comment on triglycerides as a possible risk factor?

Dr. Nestel: Triglycerides are making a bit of a comeback. One reason for this may be the recognition that hypertriglyceridemia alone is not really a very common cause of CHD but that the hypertriglyceridemia that causes coronary heart disease is usually the high triglyceride, low HDL syndrome, or combined hyperlipoproteinemia in which LDLs are raised also. One problem in the past was a failure to recognize the large biological and diurnal fluctuations in triglyceride levels, so that when triglycerides were measured on one occasion only, they couldn't possibly be as good an index as the HDL cholesterol, which varies relatively little from day to day.

Dr. Berry: I'm interested in your statement that the strategy for hyperlipidemia in the elderly should be the same as for younger age groups. I find it hard to believe that someone with hypercholesterolemia at the age of 70 has the same risk as someone at the age of 30. Elderly people with hypercholesterolemia probably have as yet unknown protective factors. If you feel an intervention is essential, then surely it would be best to try to raise HDL by exercise and to try to modify the habitual diet by manipulating the polyunsaturated/saturated (PS) ratio, which is anyway rather high in your population.

Dr. Nestel: Aortic atherosclerosis and carotid atherosclerosis are disorders that occur a decade later than coronary heart disease. Aortic atherosclerosis in particular is strongly correlated with hypertriglyceridemia. I believe the nutritional strategies I have outlined are not harmful and may be beneficial.

REFERENCE