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Editorial

Nutrition and Cardiovascular Disease

Informed scientific opinion on the most appropriate dietary advice for prevention and management of cardiovascular disease has taken not a few twists, turns and even somersaults during the relatively short period of last fifty years. That this has happened in spite of intensive and careful epidemiological, clinical and expertimental research has taught us that we cannot be too careful in interpreting our results. Converting the conclusions into reliable advice requires even more caution.

The emphasis on the nutritional approach to cardiovascular disease has some justification but also plenty of limitations. The justification lies in the ease with which diet can be manipulated in contrast with other risk factors such as genes and gender. The limitations include first, the multifactorial atiology of cardiovascular disease; second, the limited efficacy of dietary treatment; third, poor patient complinance; and finally, that we are not sure what advice to give ! Following the dictum, once bitten twice shy, nutritionists have now started advising, instead of extreme changes in diet, a moderate diet akin to the traditional diets of countries having a low incidence of cardiovascular disease.

An important limitation of research on cardiovascular disease is the chronic nature of the atherosclerotic process: it takes a long time to develop, and if effectively managed, it takes a long time to regress. Therefore the effect of an intervention is usually assessed by using plasma cholesterol or lipoprotein profile as an indicator of the response. The last word on the best indicator is yet to be written, the latest candidate being Lipoprotein (a) or Lp (a) (1).

With this background, discussed below is the role of a few selected individual nutrients in influencing cardiovascular disease.

FAT

It is now reasonably well established that both the quantity and quality of dietary fat are among the nutritional determinants of cardiovascular disease. Within a fairly wide range, the quantity of fat is, however, less important than the type of fat consumed (2). That a very high fat intake is undesirable is widely accepted. The National Cholesterol Education Program (NCEP) of U.S.A. has fixed the upper limit of fat calories at 30% of the total energy intake (3). What is not equally realised is the potentially detrimental effect of a very low fat diet. A very low fat diet generally becomes a very high carbohydrate diet. Such a diet, on one hand, induces hypertriglyceridemia (4), and on the other

lowers plasma high density lipoproteins (HDL) (5). Both these effects are undesirable from the point of view of cardiovascular disease.

Saturated fats

It is now difficult to sustain the widely believed generalization that saturated fats affect cardiovascular health adversely. There are only three saturated fatty acids which raise serum cholesterol consistently and significantly. viz lauric acid (12:0), myristic acid (14:0) and palmitic acid (16:0). Stearic acid (18:0), the major saturated fatty acid in hydrogenated oils, cocoa butter and several animal fats may actually lower serum cholesterol (6,7). There are some saturated fats such as coconut oil and palm oil, which do not raise plasma cholesterol to the extent one might expect on the basis of their fatty acid composition (8,9). Further, there is marked individucal variation in the plasma cholesterol response to saturated fats (10) indicating a strong nutrient-gene interaction. It has also been observed that the fatty acid composition of plasma lipoproteins is influenced by that of dietary fats. A higher proportion of saturated fatty acids may render plasma LDL less susceptible to oxidative modification to form the type of chemical species which has been implicated in pathogenesis of atherosclerosis (11). Moreover, it may not be appropriate to view the health implications of saturated fats solely from the viewpoint of serum cholesterol. The type of fats incorporated in cell membranes are determined to a significant degree by the type of fats consumed in the diet. Saturated fats decrease the fluidity of cell membranes (12), thereby imparting the membranes greater physical stability. Also, saturated fats can stand oxidant stress better than unsaturated fats, thereby imparting greater chemical stability. Stability of cell membranes may be important not only for the integrity of immunological mechanisms and protection against carcinogens but also for preventing degenerative diseases in general, including cardiovascular disease.

Polyunsaturated fats

Polyunsaturated fats are just as heterogenous, chemically and physiologically, as saturated fats. Two groups of polyunsaturated fats are of major importance in human diets.

n-6 Polyunsaturated fats : These include linoleic acid (18:2, n-6,9) and arachidonic acid (20:4, n-6,9,12,15) which are the predominant fatty acids in corn oil, cottonseed oil and sunflower oil (13). It is true that fats containig a high proportion of n-6 polyunsaturated fatty acids (PUFA) fats consistently reduce serum cholesterol. But they also impair immune response (14), possibly by decreasing the physical and chemical stability of cell membranes. Further, the n-6 PUFA content of the diet should be viewed in relation to its n-3 PUFA content. There are several physiological functions for which n-6 PUFA prove to be a poor substitute for n-3 PUFA (Table I).

n-3 Polyunsaturated fats : These include alpha-linolenic acid (18:3, n-3,6,9), eicosapentaenoic acid (20:5, n-3,6,9,12,15) and docosahexaenoic acid (22:6, n-3,6,9,12,15,18). The major sources of n-3 PUFA in the diet are mustard oil, rapeseed oil, soyabean oil, beans, green leafy vegatables, and fish oils. n-3 PUFA have several physiological effects which may be beneficial from the point of view of cardiovascular disease. These effects include reduction in platelet aggregation, reduction in arterial blood pressure, and favourale effect on plasma lipids (15, 16).

TABLE I : Physiological effects of n-6 and n-6 PUFA compared.

	n-6 PUFA	*n-3 PUFA*
Essentiality	Essential	Essential
Effects on plasma cholesterol	Reduce total LDL and HDL cholesterol.	Reduce total and LDL cholesterol. No consistent effect on HDL cholesterol
Important metabolic products	Prostaglanidins (2-series). Thromboxane (TXA_2) Leucotrienes (4-series) n-6 DHA substitute (if n-3 PUFA deficient)	Prostaglandins (3-series) Thromboxane (TXA_3) Leucotrienes (5-series) Docosahexaenoic acid (DHA)
Effects of metabolic products	PGI_2 inhibits platelet aggregation	PGI_3 inhibits platelet aggregation.
	TXA_2 is a strong placelet aggragator Leucotriene B_4 is a strong inducer of inflammation. n-6 DHA substitute impairs retinal function	TXA_3 is a weak platelet aggregator Leucotriene B_5 is a weak inducer of inflammation. DHA is an important constituent of brain and retina.

However, excessive enthusiasm for n-3 PUFA may turn out to be as misplaced as that for n-6 PUFA. Therefore it may be advisable to focus on, first, a reasonable intake of n-3 PUFA containing dietary fats; and second, an n-6 to n-3 ratio of at the most 10 to 1 in the diet. It is inadvisable to achieve a very high n-3 PUFA intake by taking fish oil concentrates (17).

Monounsaturated fats

These include palmitoleic acid (16: 1, n-7), oleic acid (18: 1, n-9), erucic acid (22: 1, n-9 cis), and brassidic acid (22: 1, n-9, trans). High intake of monounsaturated fatty acids (MUFA) in Mediterranean countries, which have a low incidence of cardiovascular disease, has attracted widespread attention. Most studies have found MUFA neutral with respect to their effect on plasma cholesterol but some recent studies have found that they reduce plasma cholesterol (18, 19). The usual explanation is that they may reduce plasma cholestrol indirectly by replacing saturated fats. The high MUFA content of palm oil may also explain why it does not raise plasma cholestrol in spite of its high saturated fat content (9).

Cholesterol

Regarding cholesterol also, an intense cholesterolophobia is gradually giving way to a better reasoned moderate approach. First, about two-thirds of the population has a reasonably accurate feedback mechanism to regulate endogenous cholesterol synthesis. Therefore, within reasonable limits, the sum total of cholesterol available to the body is

constant. Second, a reduction of dietary cholesterol intake by 100 mg lowers serum cholesterol by only 0.06 mmol/L or about 2 mg/dL. (20, 21). Third, a body weight exceeding the 'normal weight' by 10 kg increases endogenous cholesterol synthesis by 200 mg per day (17). Therefore it is much more beneficial to check obesity than to reduce dietary cholesterol. Last, but not least, the only major source of cholesterol in diets prevalent in the Indian subcontinent is the egg. Since one egg contains about 250 mg cholesterol, and the NCEP step one diet allows an intake of 300 mg cholesterol per day (3), consuming one egg per day in these countries is within permissible limits. Hence spreading a fear of eggs in these countries only serves to deprive the population of a convenient and realtively inexpensive source of good quality protein and several much-needed vitamins.

DIETARY FIBER

Our views on fiber have also now reached a mature stage. While fiber is no longer considered a panacea, it has a definite place in our diet. Several experimental studies have shown that water-soluble viscous components of dietary fiber have a modest hypocholesterolemic effect (22, 23, 24). This has been attributed primarily to the bile salt-binding effect of dietary fiber (25), besides some possible effect on endogenous cholesterol synthesis (26). But, in real diets, the water-soluble dietary fiber content rarely reaches levels used in experimental diets. In practice, therefore, indirect effects of dietary fiber are probably more relevant than the direct hypocholesterolemic effect. Dietary fiber increases the bulk of food without adding to its energy content, and also reduces food intake by promoting satiation. Therefore, indirectly, dietary fiber reduces energy intake and thereby prevents obesity, or facilitates weight loss. This effect would benefit cardiovascular disease directly, and also indirectly by improving glucose tolerance and lowering blood pressure.

From the practical standpoint 'long-term' studies on high-fiber foods are more relevant than those on fiber supplements. The foods shown to have a favourable effect on lipoprotein profile include buckwheat (27), oats (28, 29), barley (30, 31), wheat germ (32) and a traditional Indian preparation containing barley, wheat and bengal gram (33). Oat and barley have been consistently found to be beneficial, possibly because of the rich beta-glucan fraction in their dietary fibre (34) and possibly also their tocotrienol content (35).

RECOMMENDATIONS

The current consensus on the subject is reflected in the NCEP recommendations (3) (Table II). Step one diet is recommended for the general population and also as initial

TABLE II: Current Dietary Recommendations*.

Nutrient	*Recommended intake*	
	Step 1	*Step 2*
Total fat calories	< 30%	< 30%
Saturated fat	< 10%	< 7%
Monounsaturated fat	10-15%	10-15%
Polyunsaturated fat	< 10%	< 10%
Cholesterol (mg/d)	< 300	< 200

*Adapted from NCEP Report, 1988 (3)

therapy for patients having ischaemic heart disease. If a three-month trial with step one diet is ineffective in patients, they are advised to switch over to step two diet. As seen, the diet is a prudent diet which avoids all extremes.

CONCLUSION

Coronary heart disease is only one of the family of diseases associated with the current lifestyle, and nutrition is only one of the components of the lifestyle. Human lifestyle has undergone changes on a staggering scale during the last few hundred years. Never before has man been so relieved of physical labour, so burdened with mental effort, and so sure of the next meal (36). Our body was not designed for persistent overeating, underexercising and mental stress. The result has been diseases such as coronary heart disease, hypertension and diabetes. We have responded, quite logically, by trying to apply compensatory corrections to our lifestyle. Since we cannot go back in time, we prefer concentrated doses of corrections while leaving the rest of the lifestyle basically unchanged. For example, we try to correct for lack of dietary fiber by taking a fiber supplement. We try to make up for physical inactivity by a short period of vigorous exercise. And, we try to meditate for a few minutes to take care of day-long mental stress. The question arises whether we can really treat the body like a mathematical proposition ? Can periodic denial compensate for perpetual excess ? And, finally, will the process of evolution one day produce a 'betterman' who can enjoy hi-tech existence without the associated blues ? These are questions to which absolute or final answers may not be possible but future research can provide some clues.

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