Nutritional Pathways to Coronary Heart Disease
An Overview

The connection between diet and coronary heart disease is not limited to serum cholesterol levels but covers all aspects of the diet

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The importance of the route connecting diet and coronary heart disease through serum cholesterol, and the debate surrounding it has at times detracted from a more global view of the relationship between the food intake pattern and coronary heart disease. It is this more global view that I wish to consider in this article.

There are several nutritional pathways to coronary heart disease, some with more supporting evidence than others. Obese people are at greater risk from coronary heart disease but, the mechanisms by which this arises are likely to be several, including increased cardiac work, altered carbohydrate metabolism, changes in serum lipids, in blood pressure and so on. Serum lipids refer not only to cholesterol, but also to its subfractions, especially HDL cholesterol, and also to triglycerides. The nutritional factors of energy balance and sodium intake and their relationship to blood pressure have been appreciated for a long time. But a wider array of nutritional factors affecting blood pressure is now appreciated.

Our own work on indices of arterial wall change, like compliance, is revealing additional risk factors for arterial wall change. The higher the plasma free fatty acid responses or levels, the less the arterial compliance. The same work has indicated that hyperinsulinaemia is also a risk factor for arterial compliance. Determinants of arterial compliance also include the classical risk factors for atherosclerotic vascular disease.

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We need to consider the putative food factors which may influence such pathways. One way of looking at food rather than nutrients in coronary heart disease includes a consideration of the extent of food variety, of the overall food pattern, and not least, of the social context of eating. When this broader approach is taken, it is possible to address ourselves not only to the potentially unfavourable, but also to the potentially favourable aspects of an affluent diet in respect of coronary heart disease. Such a diet does increase the possibilities for real biological variety in food, widens the taste options, and also increases the possibilities for eating out and associated social interaction. Variety itself allows not only essential nutrient adequacy, but also the dilution of adverse factors.

The recent, and in my view, particularly important prospective study of the social influences on mortality by Welin et al. indicates the way in which a social activity score can determine mortality over a 9-year follow-up [1]. Although the authors do not address the question, many of these activities are food-related.

Energy Balance

In one way or another, all nutritional questions and management strategies depend on energy balance. The more energy we expend, the more we can ingest, so that, in terms of striking energy balance, the preferred option must be to increase physical activity. This is valid whatever the arguments for or against a direct effect of physical activity on coronary heart disease.

In the 10-year prospective Zutphen study by Kromhout et al., men who survived ate a mean 240 calories more per day than those who died [2]. Importantly, however, this also meant a greater energy intake per kilogram of bodyweight, suggesting a greater level of physical activity, and the extra energy, in the main, came from plant food. Similar observations were made, insofar as coronary heart disease is concerned, by Morris et al. who showed that there were less cases of coronary heart disease and coronary heart disease deaths in the higher third of energy intake compared with the middle and lower thirds [3].

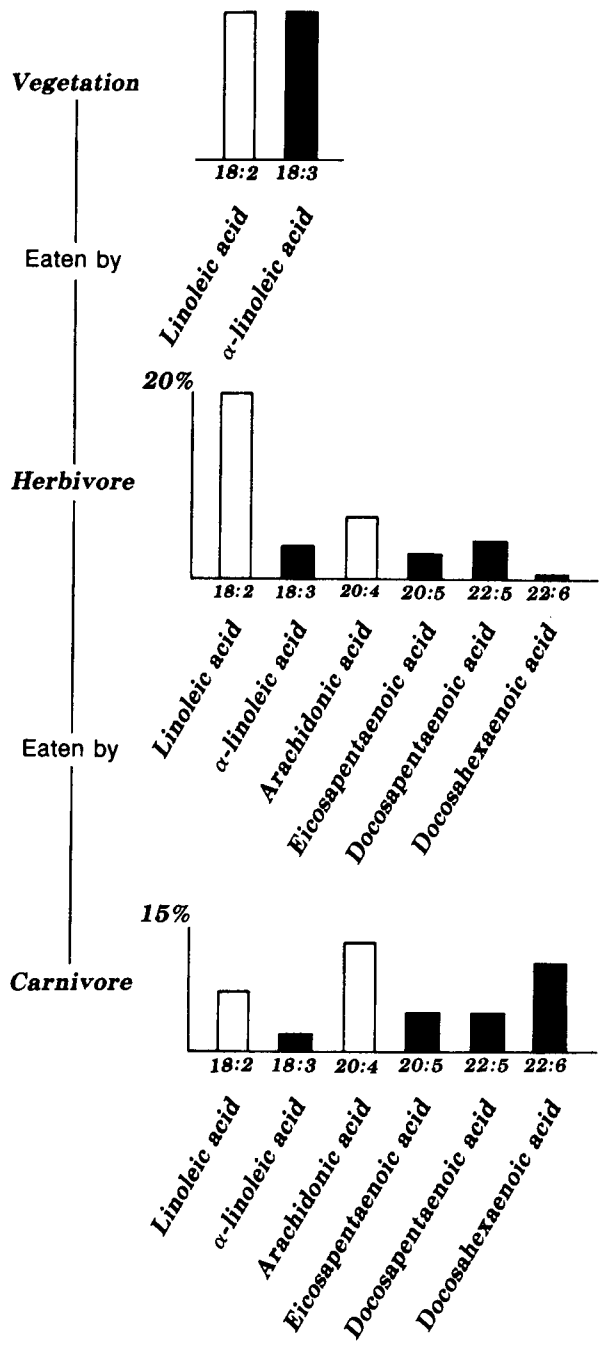

FIG. 1. *Fatty acids in the food chain, to illustrate the progression of fatty acids from the vegetation through the herbivore to the carnivore. The height of the bars represents relative amounts of the different fatty acids at each point in the chain.*

Energy Density

It is worth considering the extent to which energy density, i.e. the energy value of food measured in calories or kilojoules per unit weight or volume of that food, might be relevant to coronary risk. Individual macronutrients which one might consider of consequence in respect of coronary risk, have a cumulative effect on energy density. Remember that fat contains 9 calories per gram, alcohol 7 and protein and carbohydrate 4. Water, an important component of food, contains no calories, and dietary fibre has a small caloric value which depends on the extent to which it is converted to volatile fatty acids for absorption from the colon into the portal circulation. If we exclude alcohol from the consideration, 40% of the energy in the Australian diet comes from fat, 44% from carbohydrate and 16% from protein. Compared to other countries this is a rather energy-dense diet. Dietary guidelines advanced in various developed countries like Australia, New Zealand and the United States recommend a significant decrease in the energy density of the diet. The guidelines recommend a contribution of dietary fat to energy intake of about 30%, and of 55 to 60% for carbohydrate.

Thus, if we were to alter the energy density of the diet in a potentially favourable direction, we would decrease the intake of fat, alcohol and refined carbohydrate, and increase the intake of dietary fibre and water.

Fibre Intake

If we consider the dietary fibre question further, we know from the studies of Morris et al. that the 2 most important food variables in predicting coronary heart disease cases and deaths were energy intake and dietary fibre intake [3]. The least cases of coronary heart disease and coronary heart

disease death are found in the highest thirds of intake of dietary fibre.

In the Zutphen study [2], dietary fibre intake from a variety of sources was shown to be related to total mortality over 10 years, and also to coronary heart disease mortality.

Thus, the relevant nutritional management here is to increase plant food intake from wholegrain cereals, fruits, vegetables and nuts. The level of total dietary fibre intake from these various sources expressed in grams per day would be in excess of 35g. This is quite achievable from an Australasian dietary pattern, but contrasts with the present average dietary fibre intake of 15 to 25g.

Dietary Fat

I do not wish to dwell on the dietary fat nutritional pathway in terms of total fat intake, but it is worth saying that in Australia a reduction in total dietary fat effectively means a reduction in animal or saturated fat from ruminant meats and dairy products. To accomplish this, one needs to be attentive to cooking technique, avoiding frying, trimming all visible fat from meat, avoiding fatty spreads, choosing low-fat dairy products and avoiding biscuits and pastries.

Quality of Dietary Fat

We need to know that there are 2 families of essential fatty acids, the omega-3 and the omega-6. If we do not have these essential fatty acids, then the metabolic pathways can treat omega-9 series fatty acids, e.g. oleic acid, in their stead.

If we examine fatty acids in the food chain (fig. 1), in vegetation, i.e. plant food, we find both the omega-6 series, represented here by linoleic acid, 18 : 2, and the omega-3 series represented by α-linoleic acid, represented here by the closed bar, 18 : 3. When these fatty acids are eaten by her-

bivores, chainy longation of these fatty acids occurs for both series so that we now find arachidonic acid 20:4, omega-6 fatty acid, and the omega-3 series of longer chain polyunsaturated fatty acids, namely eicosapentaenoic acid (EPA), 20:5, docosapentaenoic acid, 22:5, and docosahexaenoic acid, 22:6. When herbivores are eaten by carnivores, there is further amplification of the longer chain polyunsaturated fatty acids representation.

Thus, in order to accomplish a change in the quality of dietary fat, one needs to use plant sources like seeds and nuts which have both mono- and polyunsaturated fatty acids. The use of avocado, which has mainly monounsaturated fatty acid, will at least decrease relatively the amount of saturated fat. Using marine sources of fat will be a way of increasing the omega-3 series of polyunsaturated fatty acids, and using lean meat will be a way of increasing both the omega-3 and the omega-6 series of fatty acids (table I).

Cholesterol Intake

It is worth just saying that the serum cholesterol level is influenced more by saturated fat than by polyunsaturated fat and more by these fats than by dietary cholesterol. This, however, does not mean that the change in the form of cholesterol

TABLE I. *Ways to accomplish change in the quality of dietary fat*

Recommendation	***Main fat type***
Use plant sources, e.g. seeds, nuts	Mono- or polyunsaturated
avocado	Monounsaturated
Use marine sources	Polyunsaturated - omega-3
Use lean meat for structural fat	Polyunsaturated - omega-3 and -6

after a meal, in the form of chylomicron remnants, could not be important in terms of atherogenesis. There is an emerging understanding of the levels of total serum cholesterol at which we should be concerned. The National Heart Foundation of Australia recommends that we have total cholesterol levels of less than 6.5 mmol/L. From the work of Goldbourt et al. [4], total cholesterol levels of less than 5.6 mmol/L might be fairly safe. Other assessments of threshold would suggest perhaps 5.2 mmol/L. Thus, we could say that in terms of HDL cholesterol, this becomes an important issue once one's total cholesterol is above 5.2 or 5.6 mmol/L.

The nutritional management of cholesterol intake will be to reduce egg yolk consumption (I would particularly recommend the use of cholesterol-free egg mix), to have organ meats only occasionally and to prefer plant foods which, of course, contain no cholesterol at all.

Alcohol Intake

Not only is alcohol intake a determinant of energy balance, but also of serum lipids, and quite clearly now, of blood pressure, both systolic and diastolic, in a fairly linear fashion. Thus, as far as the nutritional pathway to coronary heart disease via hypertension is concerned, alcohol is an important consideration.

Elemental Intake

Sodium

For people whose blood pressure is sensitive to sodium, and this may be increasingly important with advancing years, one needs to be aware of the sources of sodium in food. Only about one-third of the sodium intake in the Australian diet is discretionary, either at the table or in cooking. Most sodium comes from bread and cereal. Thus, we must seek out low-sodium bread and low-sodium cereals. Cheese, soups, and sources of monosodium glutamate like soya sauce are also important. We also need to be increasingly aware of the sodium/potassium ratio.

Secondary Nutritional Management

There are 2 important trials to help us consider this issue. The first is the Helsinki trial, a 7-year prospective angiographic study on 28 patients with coronary heart disease and 20 controls, using diet and drugs [5]. Importantly, there was no progression of coronary lesions if the LDL cholesterol to HDL cholesterol ratio was in the region of 3.9, but considerable progression if this ratio was in the vicinity of 5.

Next, there is the Leiden trial [6]. This was a 2-year trial involving 39 patients on a vegetarian diet where the polyunsaturated to saturated fat ratio was greater than 2, and where the cholesterol intake was less than 100mg per day. The coronary lesion growth was proportional to the total of HDL cholesterol ratio. There was no lesion growth if this ratio was less than 6.9. The data showed a clear relationship between the change in vessel diameter and the total to HDL cholesterol ratio.

Dietary fat modification may have a place in the management of angina

Dietary Fat and Angina

It is also worth noting those studies which indicate that dietary fat modification may have a place in the management of angina. A study from Denmark by Thuesen et al. reduced the dietary fat contribution to energy from 40 to 10%, increased the carbohydrate intake from 45 to 75% and reduced the energy intake from 2788 kcal to 1342 kcal [7]. The trial lasted 3 months, the mean pacing time to angina increased significantly from 139 to 291 seconds, and the lactate production decreased significantly from 31 to 19 μmol/minute.

In a Boston study by Ribeiro et al. a comparison was made of exercise alone *versus* diet and exercise, where the diet consisted of 15% of food energy from fat and 70% from carbohydrate [8]. The study was conducted over 10 to 16 weeks.

With exercise alone, 15% had no angina. Where diet was also included, this was increased to 69%.

Thus, it is clear that the dietary management of people with coronary heart disease is also worthwhile, but not perhaps for the reasons that people have previously thought. That is to say that irrespective of what might or might not happen to the serum cholesterol level, dietary management remains an important strategy.

Last, but not least, the nutritional management of coronary risk also provides optimal nutrition, a particularly important consideration for older people.

References

1. Welin L, et al. Prospective study of social influences on mortality. Lancet I: 915, 1985
2. Kromhout D, et al. Dietary fibre and 10-year mortality from coronary heart disease, cancer and all causes. Lancet 2: 518, 1982
3. Morris JN, et al. Diet and heart: a postscript. British Medical Journal 2: 1307, 1977
4. Goldbourt U, et al. Total and high density lipoprotein cholesterol in the serum and risk of mortality. British Medical Journal 290: 1239, 1985
5. Nikkilä EA, et al. Prevention of progression of coronary atherosclerosis by treatment of hyperlipidaemia. British Medical Journal 289: 220, 1984
6. Arntzenius AC, et al. Diet, lipoproteins and the progression of coronary atherosclerosis. New England Journal of Medicine 312: 805, 1985
7. Thuesen L, et al. Beneficial effects of a low-fat low-calorie diet on myocardial energy. Lancet 2: 59, 1984
8. Ribeiro JP, et al. The effectiveness of a low lipid diet and exercise in the management of coronary artery disease. American Heart Journal 108: 1183, 1984