SESSION I

THE ROLE OF FATS

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Coronary heart disease. The size and nature of the problem

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Summary

In the U.K., coronary heart disease has reached epidemic proportions. It is the commonest cause of death after the age of 35 years and the fastest rate of increase is in early middle age. The epidemic is due mainly to our way of life. The most important factors are dietary, with smoking, physical inactivity and stress also contributing. Twenty independent working parties from different countries have reviewed the dietary evidence and reached a strong consensus on dietary recommendations. Little action has been taken in the U.K. The Coronary Prevention Group has been formed to consider the reasons for this inaction and also the implication for research, the government, the Ministry of Agriculture, Fisheries and Food, the Department of Health and Social Security, the food and agriculture industries, caterers, nutrition education and for individuals, of the dietary recommendations.

IN THE U.K., coronary heart disease (CHD) has reached epidemic proportions. The World Health Organization (WHO) has said that CHD threatens to become the greatest epidemic mankind has ever faced—unless action is taken. There is much evidence that it is man-made and due to changes which have come about in the way we live.

CHD is now the commonest cause of death in males after the age of 35 years, with the fastest rate of increase in the age group 35-44 years. Forty per cent. of the deaths from CHD occur before retirement and about one man in 3 or 4 can now expect a heart attack or stroke before the age of 65 years, and often long before. Others have only

a brief retirement. The incidence in women is starting to catch up. Scotland now shares with Finland the highest mortality from CHD in the world.

Atherosclerosis is sometimes quite advanced in young adults, usually starts in childhood and pathological changes, considered to be pre-atheromatous, may already be present in infancy. These are formidable facts. There will be no spontaneous improvement. Further advances in acute coronary care can achieve little because most of those who die do so too quickly for medical attention, and most have advanced coronary arterial disease. The answer to the coronary problem can only be in prevention.

That the causes of this epidemic must lie largely in the way we live is shown by the steep rise in the incidence in recent years (Fig. 1); the wide variation in mortality between populations which cannot be explained by race (Figs. 2, 3); the experience of migrants from low to high risk countries; and the striking changes in mortality rates which occurred during and after recent wars.

Lessons from Japan

Japan is a modern, industrialized, stressed, well nourished and heavily-smoking society with more hypertension than any other country, but it does not have a CHD problem. The answer is most likely to lie in the fact that the concentration of fats (lipids) in the blood is low by Western standards, but 'normal' by comparison with the world's low risk populations. This can be explained by their habitual healthy diet which is low in saturated fat and cholesterol with a high polyunsaturated : saturated (P/S) ratio. It is

characterized by relatively little protein from farm animals, more sea food than meat and little dairy produce, together with a high intake of cereals, pulses and other vegetables. The Japanese consume little fat and it mainly comes from polyunsaturated sources. In Japanese who emigrated to the United States of America and changed their habits, blood cholesterol concentrations rose, atherosclerosis developed and in due course they had much the same incidence of CHD as the indigenous host country. Diet is the only major variable likely to be responsible for these findings. The lesson seems clear. If the diet is a healthy one and not associated with hyperlipidaemia, other risk factors, including genetic predisposition, heavy smoking, hypertension, stress and diabetes are of relatively little importance as regards CHD. If, however, there is a dietary change in a Western direction, the sequence of events is predictable.

Similar conclusions can be drawn from studies on other migrants, from the 3 populations of South Africa and from Mediterranean and Adriatic countries which eat well, but do not have a significant CHD problem.

There is no suggestion that in the U.K. we should follow these low risk diets precisely, but we could do so in nutritional principle, within our agricultural potential and staying within our basic eating habits.

FIG. 1. Percentage increase in death rates due to coronary heart disease of men and women in England and Wales in age group 35-44 years.

FIG. 2. Mortality due to coronary heart disease: males 45-54 years; deaths per 100,000; WHO 1974-1975.

FIG. 3. SMRs for ischaemic heart disease (ICD 410-414) in the regions of England, Wales and Scotland. Calculated using average number of deaths 1969-1973 and 1971 census populations. Males 45-64 years.

The links

The links between diet, plasma cholesterol, platelet function, atherosclerosis and CHD are immensely strong.

The evidence is based on the following:

(1) No population habitually consuming a 'healthy' diet, similar to that recommended below, has a CHD problem whereas every population habitually consuming food of the type condoned in the U.K. as wholesome has a very high incidence.

(2) The International Atherosclerosis Project, which established that epidemic CHD was invariably based on advanced atherosclerosis and had strong correlations with diet.

(3) Clinical correlations with biochemical and pathological findings.

(4) Prospective studies in populations which vary widely in their dietary habits, in plasma lipid concentrations and in CHD mortality.

(5) Laboratory correlations between plasma lipid concentrations, platelet function and diet.

(6) The sequence of events in migrants from low to high risk countries.

(7) Prospective community studies, of which that in Framingham in the United States is the best known and of longest duration.

(8) Many years of experimental work, including the use of ordinary human diets in non-human primates.

(9) Laboratory work on mechanisms at cellular level.

(10) The fact that familial hypercholesterolaemia is associated with premature atherosclerosis and early death and that cutaneous xanthomata, of similar lipid composition to that in atheromatous plaques, regress on the prudent diet recommended for coronary prevention provides yet further evidence.

Experimental studies

There are limits to direct observations in human subjects, but excellent experimental models have long been available. The most impressive studies are those carried out in the last 15 years in Chicago (Wissler *et al.*, 1971; Wissler and Vesselinovitch, 1977a, b). They have given an ordinary Western diet in similar quantities, but suitably prepared to make it acceptable, to non-human primates which closely resemble humans in their relevant anatomical, physiological and biochemical features. The foods were therefore high in saturated fat and cholesterol with a low P/S ratio. They responded by developing similar elevations of plasma cholesterol, similar changes in the coronary arteries (which cannot be distinguished under the microscope from those occurring in humans) and similar clinical complications. As in humans, a platelet reaction and thrombus formation may occur in due course. Impressively

these changes do not occur or are mild and never obstructive if instead a prudent diet is given which is similar to that now recommended for CHD prevention.

Encouragingly, if the lesions are not too advanced, considerable regression may follow on changing back to the normal diet.

Evolving man

Evolving man branched from basically vegetarian stock. Later he became a hunter and gatherer of wild animal and vegetable life, but continued to live mainly on fruit, plant food and nuts for a million years or more. He was breast fed. Energy intake was matched by expenditure. His meat was lean and occasional, he had no dairy produce and there was no food industry. Eggs can only have been available from the nests of wild birds in season. Fat consumption must have been low and largely unsaturated.

A change began when he first settled as a farmer less than 10 000 years ago, domesticating a few animals and cultivating a few crops. Changes were slow until about 200 years ago. Then, following the agricultural and industrial revolutions, there came a population explosion, movement to towns and need for transportation and preservation of food. A food industry was born. It became possible to feed animals through the winter and then fatten them for the market. Later, meat was also imported. Margarine was first produced about 100 years ago. Only recently has there been a trend from poverty, which was the lot of the majority, to relative affluence, and with it a trend from vegetable to animal protein and an increase in the consumption of meat, dairy produce, eggs and saturated fat. Now most cereals, including flour used for bread, are refined, thereby removing the germ, the bran and many nutrients, few of which are put back.

Now, half our food is processed, using saturated fat, sugar, salt and numerous additives. The composition is mostly unknown to the consumer.

Britain is not well fed. A sufficiency of what is considered by the Ministry of Agriculture, Fisheries and Food (MAFF), the Department of Health and Social Security (DHSS) and the British Nutrition Foundation (BNF) wholesome (safe) food is compatible with widespread mal- (bad, wrong) nutrition. Clinicians know that an apparently healthy, well nourished person may be dead within the hour from some complication of long-standing mal- (wrong) nutrition which is the fundamental factor responsible for atherosclerosis.

Our present diet is uniquely abnormal. It was not planned or tested for health. Malnutrition from undernutrition and deficiency disease has been replaced by that from overnutrition and imbalance of nutrients, of which the most important is fat. The

TABLE 1. Recommendations of 20 committees on food and coronary heart disease

Country	General population (GP) high risk group (HR)	Fat content of total energy (%)	Increased PUFA	PUFA-SABA ratio	Daily dietary cholesterol (mg)	Reduction of sugar	Labelling of fat content of foods
Norway, Sweden and Finland, 1968	GP	25-35	Yes	—	—	Yes	Yes
United States, 1970 Inter-Society	GP	< 35	Yes	1.0	< 300	—	Yes
New Zealand, 1971 Heart Foundation	GP	35	—	—	300-600	—	Yes
New Zealand, 1971 Royal Society	HR	35	Yes	1.0	300-600	—	Yes
	GP	—	No	—	Reduce	—	—
	HR	Avoid excess saturated fat	Yes	—	Reduce	—	—
United States, 1972 American Health Foundation	GP	35	Yes	1.0	300	Yes	Yes
United States, 1972 American Medical Association	HR	Substantial decrease in saturated fat	Yes	—	Reduce	—	Yes
International Society of Cardiology, 1973	HR	< 30	Yes	> 1.0	< 300	—	Yes
United States, 1973 American Heart Association	GP	35	Yes	1.0	300	Yes	Yes
The Netherlands, 1973	GP	35	Yes	1.0	250-300	Yes	Yes
United States, 1973 White House Conference	GP	35	Yes	—	300	—	Yes
Australia, 1974 National Heart Foundation	GP	30-35	Yes	1.5	< 300	—	—
United Kingdom 1974 DHSS (COMA)	HR	Reduce total fat, especially saturated	No	—	—	Yes	—
Germany, 1975 (Federal Republic)	GP	Reduce saturated fat	Yes	—	300	—	—
Australia, 1975 Academy of Science	GP	35	Yes	1.0	< 350	Yes	Yes
United Kingdom, 1976 Royal College of Physicians and British Cardiac Society	GP	Towards 35	Yes	—	Reduce	Yes	Yes
Norway, 1975-76 Ministry of Agriculture	GP	35	Yes	0.5	Doctor's decision	Yes	Yes
Canada, 1976 Health & Welfare	HR	35	Yes	1.0	400	Yes	Yes
United States, 1977 Senate Committee	GP	30-35	Yes	—	400	Yes	—
FAO-WHO, 1978	GP	30	Yes	1.0	300	Yes	—
	GP	30-35	Yes	1.0	< 300	—	—
European Society of Cardiology	HR	30	Yes	1.0	< 300	Yes	—

DHSS (COMA)=Department of Health & Social Security (Committee on Medical Aspects).

FAO-WHO=Food & Agriculture Organization (United Nations) and World Health Organization.

new era of 'convenience' and 'fast' foods is a mixed blessing which requires detailed analysis.

The recommendations now widely agreed for a healthier diet amount, in nutritional principle, to correction of adverse changes which have come about, especially over the last 200 years. This is a tiny fraction of time on the evolutionary scale.

The consensus of opinion

In recent years 20 independent working parties from many different countries have been appointed to review the evidence on the relationship of CHD

to diet, and to make recommendations. Their reports present a strong consensus of opinion (Table 1). No comparable matter can have been subjected to more rigorous examination or vigorous debate, and it is reasonable that the advice should be accepted as the basis for action. Not to act is to condone a diet which is considered to be hypercholesterolaemic, atherogenic and thrombogenic and the fundamental cause of epidemic CHD. Other risk factors may be additive in their harmful effects, but none is essential to the development of CHD.

It is of course recognized that as new evidence

comes to light there may have to be some modifications as regards detail, but it could hardly negate what has been so firmly established. The agreement reached was the result of different groups of specialists at different times assessing the available scientific knowledge in relation to their own and other countries' experience. There has been no similar report, based on a comparable and exhaustive examination of the available data, which contradicts the conclusions reached.

Failure to take action now is tantamount to accepting a continuing high mortality and cannot be justified by the absence of unobtainable proof. It may not be widely appreciated that there can never be a trial to prove any dietary theory or a causal relationship between any food or nutrient and CHD by dietary trials. This was the conclusion reached by the Task Force appointed to review the matter in the U.S.A. in 1971. The practical difficulties of conducting such a trial include the impossibility of having a control group of adequate size for a sufficient length of time.

Nor can there be justification in waiting for significantly stronger evidence because this could not possibly be established in the foreseeable future owing to the long incubation period of atherosclerosis. Likewise there can be no justification in waiting for the detection of those at high risk who could only be given preventive advice. This is because screening whole populations is not feasible and, if it were, many if not most of those at high risk would not be detected by present standards. For practical purposes it must be accepted that all are at risk if they habitually consume our present diet. The risk is higher in all smokers and all hypertensives. The significance of other risk factors is less easy to determine.

Past dietary trial evidence is often cited as an argument against introducing dietary modification because of the inconclusive nature of the data. Scientists who are familiar with the subject no longer refer to the old dietary trials as relevant to current policy. The question of the efficiency of trials had to be tested, but it can now be understood why the results, although encouraging, could not be accepted as conclusive. They were all carried out in middle-aged or elderly persons, most of whom were likely to have had at least a moderate amount of atherosclerosis, and perhaps a more serious stage of development. It is not the conventional coronary risk factors which are usually responsible for the onset of symptoms. Rather it is likely to be almost a chance event due to some complication of atherosclerosis, such as rupture of a plaque, haemorrhage into the arterial wall, or thrombus formation causing obstruction of the lumen of an artery, or microembolism causing ventricular fibrillation.

In no trial was the concentration of cholesterol in the blood reduced below that at which atherosclerosis is known often to be advanced.

It is now clear that to give conclusive evidence trials would have to be started much earlier, preferably in childhood, and last for 20–30 years at least. The ethical and practical difficulties so imposed make this course of research unrealistic.

In considering dietary modification, it is significant that there is no evidence that the recommendations of the 20 committees are likely to be detrimental to health. Contemporary western diets are in a state of continual change towards diets which are unique in composition. In nutritional principle the recommendations amount to a dietary correction—not change as such, but changing back.

Dietary principles

In nutritional principle the recommendations amount to correction of present-day excess and imbalance. In the U.K. most adults (and many children) already have atherosclerosis so that it is secondary prevention which is required, aimed at its arrest and regression and the prevention of thrombotic complications.

The advice as regards lipids amounts, on average, to halving non-essential saturated fat, halving cholesterol and doubling essential polyunsaturated fat.

The recommendations are based on the metabolic studies of Brown (1966a, b, 1971) with the reduction of plasma cholesterol (as a risk factor and as a marker) as the target. The numbers have been 'rounded off' and the cholesterol estimation is for dietary energy of adults.

	Fats (dietary energy%)				Cholesterol (mg/day)	
	Total	Sat.	Mono	Poly.	P/S	
Now	40	20	15	5	0.25	600
Target	30	10	10	10	1.0	300
(Rx)*	34	17	13	4	0.2	550

* Rx represents a 'Prescription for a better British diet' (Passmore, Hollingsworth and Robertson, 1979) but would have little or no effect on the P/S ratio or plasma cholesterol concentration.

P/S = polyunsaturated : saturated ratio.

Fatty acid composition

The precise fatty acid composition (FAC) of adipose tissue triglyceride fat is important and determined by habitual diet (with a slow turnover). Free (non-esterified) fatty acids (FFA) are released into the circulation and, if not used for fuel, are taken up by various tissues and largely determine their function. The composition of FFAs, cholesterol

esters and of phospholipids in cell membranes (e.g. arterial endothelium and platelets) is an aspect receiving increasing attention as regards atherogenesis, thrombogenesis, vascular tone and myocardial function. Lauric acid (in coconut oil) and myristic acid (in dairy fat) are powerfully atherogenic. Stearic acid (in beef fat), on the other hand, is not atherogenic but powerfully thrombogenic, and palmitic acid (in most fat) is both atherogenic and thrombogenic. The properties of the essential linoleic and linolenic series are not identical and long chain derivatives tend to be more active than their parents. These examples serve to explain the interest of some research workers. What matters to most of us is *food* and the changes we have allowed to come about unplanned and untested for health. Some understanding of mechanisms provides confidence in the giving of advice.

Aims

(1) Improvement in or maintenance of correct balance of competition between fatty acids for the enzyme systems which regulate chain length and desaturation—and hence the availability of essential fatty acids (EFA).

(2) Reduction in or maintenance of correct concentrations of plasma cholesterol and/or triglycerides.

(3) Preservation or repair of the endothelial barrier by EFA.

(4) Prevention of platelet adhesion to arterial wall and of aggregation to each other and hence the risk of thrombus formation and of platelet breakdown with release of mitogens which stimulate proliferation of smooth muscle cells in the media.

(5) Improvement in or maintenance of correct fatty acid composition of adipose tissue and hence of FFAs, cholesteryl esters and phospholipids in cell membranes.

(6) Regulation of prostaglandin activity and hence of platelet function, vascular tone, BP, blood flow and myocardial function.

Implications

Reduction in consumption of saturated fat from dairy produce, meat and meat products, and from hydrogenated fats and coconut, palm and palm kernel oils.

Increase in consumption of polyunsaturated margarines and oils from 5 to 10% of the energy. This amounts, on average, to only about 15 g/day or 15 g each of a 50% polyunsaturated margarine and oil.

Reduction in consumption of animal food and hence of cholesterol. In the U.K., about 35% of dietary cholesterol comes from egg yolk, 30% from dairy fat and 25% from meat or meat fat.

Egg yolk	Cholesterol (600 mg/day)		
	Dairy fat	Meat	Miscellaneous
210	180	150	60

It can be calculated that if the goal of 300 mg/day is to be reached without unacceptably drastic restriction of meat and dairy produce, it is necessary to restrict egg yolks to not more than 3/week.

The implications for the individual are straightforward and, with co-operation from food producers and manufacturers, are likely to be widely acceptable.

The implications for the Government, agriculture, the food industry and caterers, on the other hand, are substantial but, given time to adapt and financial support meanwhile, are likely to be feasible and in the long term economical.

The Government could encourage these changes by phasing out imports of butter, meat and cereals for animal feed, by stopping subsidies on butter, and possibly taxing excessive dairy and carcass fat (above a fixed grade), and grist for white flour.

Farmers could reduce finishing (fattening) for the market and hence the fat in and around meat, fat for meat products and fat used for cooking (lard, suet, dripping).

The dairy industry could reduce the fat content of standard milk and make readily available lower fat milks and milk products (including cheeses).

The food industry could phase down hydrogenated vegetable and marine oils, and imported coconut, palm and palm kernel oils with replacement by polyunsaturated fats and oils. They could also use less egg yolk in many products.

The flour industry could make more readily available wholemeal breads at cheaper prices and actually promote them.

The Government could also encourage and support the fishing industry and market gardens, encourage local authorities to fulfil their obligation to provide allotments, and greatly improve food education. It is agreed policy that the U.K. should live more on food from its own resources. There should also be greater freedom of choice than at present.

Recommendations on what to eat

The following recommendations (slightly expanded) are based on those of the Joint Working Party of the Royal College of Physicians and British Cardiac Society (1976) and are in conformity with those implicit or explicit in most other reports.

(1) Eat less and leaner meat. Use meat more often as a flavouring agent than main constituent of a meal. Remove visible fat. Grill rather than fry. Refuse to buy fatty meat.

(2) Restrict fatty meat products and avoid animal cooking fat. Ask for lower fat sausages, beefburgers, pies and sliced meats.

(3) Instead of meat, have more non-meat meals with protein from mixed vegetable sources (cereals, pulses and potatoes) or choose more fish (fresh, frozen or tinned) or poultry.

(4) Have more cheese dishes which make a useful alternative especially if made with a low fat variety.

(5) Eat butter sparingly. In general avoid cream and top of milk. Instead of butter, choose a polyunsaturated spread. Look for the label 'High in polyunsaturates'. Always ask the manager for a polyunsaturated margarine when eating out in a restaurant.

(6) Avoid hard margarines and cooking fats sold in a packet, and also oils and fat merely labelled 'vegetable' as they may contain much saturated fat. Instead, use a polyunsaturated oil (sunflower, soya, corn or safflower).

(7) Eat more bread (preferably wholemeal). This includes sandwiches, snacks on toast and bread and cheese. Eat more cereals, including porridge, muesli and other whole-grain breakfast varieties, rice, (preferably brown), pizza, pasta (spaghetti, macaroni, ravioli, lasagne, etc.), potatoes (preferably baked or boiled), pulses (beans, peas, lentils), greens, salads, root and other vegetables, fruit and nuts.

(8) Restrict egg yolks to not more than 3/week.

(9) Restrict rich cakes, pastries, biscuits, puddings, ice-cream and confectionery.

A return to a more vegetarian way of life (on nutritional grounds) is implicit in these recommendations. Protein from mixed vegetable sources has a number of important advantages over those of animal origin, including the provision of fibre, but little saturated fat and no cholesterol. This advice is straightforward, easy to understand and to accept, and the diet usually less expensive. There can be adequate variety, as can be seen from the numerous cookery books available.

The 'fat-switch' to a polyunsaturated margarine and oil and restriction of dietary cholesterol is likely to be an essential component of an acceptable healthier diet in any country with a high incidence of atherosclerosis and CHD, if reduction of plasma cholesterol is one target.

Primary, secondary and tertiary prevention

It is important to appreciate the difference between primary, secondary and tertiary prevention, although in the individual they cannot always be distinguished with certainty. This is because some children have arterial changes and in some adults myocardial infarction has been silent. Recognition of these separate forms is necessary for understanding the basis of the recommendations on diet under different circumstances and the significance of the P/S ratio in relation to total fat consumption.

Primary prevention is directed against the initial

development of atherosclerosis and the risk factors which lead to it. This is the most logical approach.

Secondary prevention is indicated in many Westernized populations, such as the U.K., because most adults and many older children already have atherosclerosis. The aim is its arrest and regression, with the prevention of complications. This requires stricter measures than for primary prevention.

Tertiary prevention refers to those who have already developed symptomatic disease in whom it can be assumed that atherosclerosis is advanced and the risk of thrombotic complications is high. Also a myocardial factor has been added. It is anomalous that many physicians now give dietary advice to those who have some clinical manifestations of CHD, but fail to appreciate that similar advice is relevant as a preventive measure to those who appear healthy.

In the very young and in populations without a CHD problem, it is primary prevention which is required. Ideally this starts at birth or even before with correct maternal nutrition during pregnancy as a preparation for breast feeding.

In future it is to be hoped that parents and school authorities will introduce primary prevention so that children are more healthily fed. It is developing tissues which are the most susceptible to dietary injury, and it is in these early years that food preferences and eating habits are often largely determined.

Coronary prevention starts in childhood

The logical approach is true primary prevention against atherosclerosis and the risk factors which lead to it from birth onwards. Atherosclerosis may be quite advanced in young adults and frequently starts in childhood whilst pre-atheromatous changes are often present in infancy. It is developing tissues which are the most susceptible to injury. At this age, directly or indirectly, only food is likely to be a major factor. Food preferences and eating habits acquired early are those most likely to be continued after leaving home. Other factors, such as smoking and physical inactivity, may also have their roots in childhood. In the U.K., according to the DHSS, the composition of the diet of children is similar to that of adults. This means beyond reasonable doubt that it is atherogenic and in need of correction.

Breast milk is best for developing arterial walls because cow's milk is of an entirely different composition and very low in EFA. New commercial formulas have been improved, but the EFA content is still considerably lower than that in breast milk. In the U.S.A. the concentration of EFA in infant formulas is much higher, and in The Netherlands often much higher still, but no adverse effects have been noted.

Protection of the arterial wall could reasonably

be added to the likely advantages of breast feeding and of maintaining a balanced diet after weaning.

Present official recommendations for nutrition in schools are a matter for concern (DES, 1975). Sound nutrition should also include the prevention of overnutrition and imbalance, and of chronic adult diseases which have their roots in childhood. These include atherosclerosis, intestinal disorders and almost certainly some forms of cancer. Why take the risk? The diet of children is uniquely abnormal on the evolutionary time-scale. The recommendations are not even in conformity with the advice of the DHSS that children should eat less saturated fat which includes butter, cream, top of milk, fatty meat and meat products, most margarines and shortenings, and hydrogenated fats and oils. Not only is there no mention of this, but the cooking fats widely used are the most saturated because they are the cheapest. Some form of animal protein is advised for every day and 'fresh meat 3 times a week'. Children have no special preference for meat which is unnecessary as a source of first class protein or any other nutrient. Fish and poultry are preferable on nutritional grounds. Animal protein has no advantage over that of mixed vegetable origin. More non-meat meals would reduce consumption of saturated fat and cholesterol, raise the P/S to a more physiological level, and provide important fibre. There is much to be said for a more vegetarian upbringing for children.

Extra school milk is unnecessary for the majority of children, and full-cream milk is always undesirable. 'Pour away the top' is a sensible slogan.

A recent personal examination has been made of more than 500 school menus from various regions in the U.K., including the constituents used by the caterers. Dr Crawford (1980) has measured the fat content and fatty acid composition. The results reveal a highly unsatisfactory situation of which many catering advisers and teachers are aware. This is one reason for welcoming a cafeteria service offering choice only but from healthier food, which can be various and attractive. Vitamin C is almost certainly often very low, but was not measured.

The reasons for inaction

The links between diet and CHD are strong and the preventive measures straightforward. Why then is there so little action in the U.K.? The explanation seems largely to lie in the nature of the medical evidence and the question of responsibility.

First, there can never be 'proof' of any dietary theory. The logically acceptable attitude to the evidence is to seek a high confidence in the theories based on the available evidence which the consensus of expert opinion provides.

The medical profession is not generally trained

in nutrition and few have had the opportunity to study the evidence on CHD. Furthermore, they are not adequately trained in preventive medicine and are understandably sceptical of prevention rather than treatment. Confusion is also being caused by a vocal few who, as Truswell (1978) has pointed out, tend to get undue prominence in a democracy.

Governments have never given high priority to preventive medicine. The present Social Services Secretary recently said that health education was not a priority because 'when resources are tightly constrained and choices have to be made between treating the sick and other less immediate priorities, it is inevitable that healing must come first'. However, we need both and they are not mutually exclusive. Encouragingly, Sir George Young, Parliamentary Under-Secretary at the DHSS, at a recent conference (unpublished) said that the solution to many of today's medical problems will not be found in research laboratories, but in our Parliament, and for the prospective patient the answer may not be 'cure by incision at the operating table, but prevention by decision at the Cabinet table'. He pointed out that most of the big killer diseases of today are not caused by nature, but by our life-style. These include heart attacks, cancer, strokes and road accidents. He said that prevention does not raise medical issues, but political ones. Historically a nation would look to its doctors for better health. Now they should look to their Members of Parliament. There is growing evidence of the willingness of parliaments to legislate to protect an individual from damaging his own health. The cost of illness may not be borne by the individual, but through our NHS by taxpayers as a whole.

The DHSS (1960) takes the view that prevention is everybody's business', but seemingly not its own, and assumes that the public has been sufficiently well informed to choose wisely. This is far from being the case. The Department is under the impression that adequate information is contained in government booklets, which include: its own COMA panel report (1974), advice to the Department of Education and Science on *Nutrition in Schools* (1975), their booklet *Prevention for Health: Everybody's Business* (1960) (in which diet is virtually ignored) and, the latest, *Eating for Health* (1978). This last has caused much confusion: there is a fundamental disregard of modern research in relation to total fat, essential fatty acids, dietary cholesterol, platelet function and prostaglandin activity. The 5-food groups have little or no nutritional meaning for the clinician. Two are unnecessary at any age and the fifth is a confused mixture. Butter and margarine are considered to be equivalent. Numerous other criticisms of detail could be made. The cover illustrates a diet characteristic of

every population with epidemic CHD and contrasts starkly with the recommendations of all other working parties, including the recent authoritative FAO-WHO report on *Dietary Fats and Oils in Human Nutrition* (1977). No reasons are given except of the illogical 'no proof' variety.

The Ministry of Agriculture, Fisheries and Food has in the past been more concerned with the interests of the farming and food industries than those of the consumer. This is clear from their White Papers. In *Food from our own Resources* (Govt Command Paper, 1975) there was no reference at all to health. In *Farming and the Nation* (Govt Command Paper, 1979) there are only a few words of lip service, seemingly designed to absolve themselves (and the DHSS) from responsibility. However, the interrelationships of food, health and farming have now been raised by the Centre for Agricultural Strategy (1978), and open discussion advocated. This could be to our great economic and health advantages. The introduction by Professor Bowman should be read by all interested in this subject. The MAFF fails to recognize that a sufficiency of 'wholesome' food is compatible with widespread malnutrition.

The Government states that it cannot interfere with freedom of choice, but overlooks the extent to which it already interferes, for example by restricting the availability of fat-reduced milk, controlling carcass grading systems on the fat, subsidizing butter (and hence influencing its price), condoning inadequate and misleading labelling and allowing excessive promotion of dairy fat and other foods which doctors and nutritionists specifically advise against.

The Government also fails to encourage the provision of sufficient information to enable full choices to be made, and permits teaching kits for schools to be provided by vested interests. It lacks a food policy linked to health, a Minister of Food, and a co-ordinating inter-Ministerial committee with executive powers.

Responsibility is being placed squarely and unfairly on the individual for maintaining health in relation to diet. The DHSS argues that its role is only to provide information to the consumer to enable a wise choice about diet to be made. Not only has little information been made available, but even as regards the recommendation of the COMA panel for restriction in saturated fat, no circulars were sent to health authorities or the food industry. School and hospital meals are unchanged and caterers do not offer, for example, an alternative to butter which is a highly saturated fat and, in effect, heavily hydrogenated.

Liaison with the MAFF has been inadequate and ineffective, and the DHSS itself is inadequately informed because it has not sought advice sufficiently

widely. There is a wide nutritional gap, and leadership is conspicuously lacking.

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