

## Review Article

## Diet and heart disease in the 1980s

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## Summary

In a climate of increasing concern about the high incidence of coronary heart disease in South Africa, calls are increasingly being made for South Africans to alter their diet. Can this exhortation be justified, and are the right foods being singled out as culprits? If changes in the eating habits of a nation are advocated, the advice must not only be soundly based in terms of likely benefit but must also take account of potential hazards. Critical perusal of the current literature leads to the conclusion that advice promoting a fat-modified, lower-salt diet is sound, safe and likely to be effective, especially as regards the younger segment of the westernized population.

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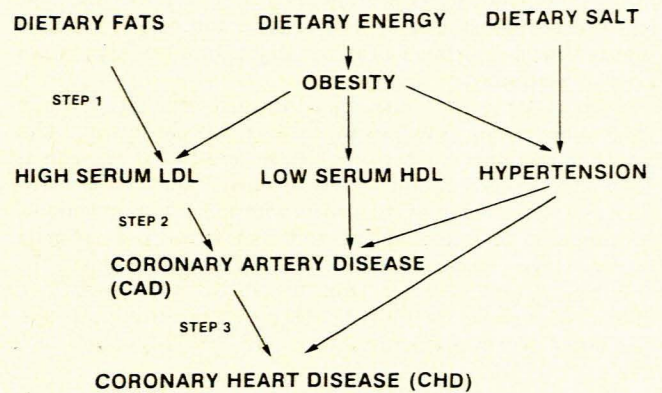


Fig. 1. A modern version of the diet-heart hypothesis<sup>2</sup> (LDL = low-density lipoprotein; HDL = high-density lipoprotein).

The idea that food influences serum cholesterol (SC) levels and, ultimately, the incidence of coronary heart disease (CHD) had its genesis in the cholesterol-fed rabbit experiments of Anitschkow and of Ignatowski in the first decade of this century. However, it received its major impetus from Keys *et al.*'s<sup>1</sup> international comparisons of fat intake, SC levels and CHD during the 1960s, and has subsequently spawned a vast research effort and an equally vast (and often contradictory) literature. Today, some three-quarters of a century later, there is somewhat more clarity about the diet-heart hypothesis (DHH), and in those areas where there is not clarity the reasons for its lack are at least known. Some unresolved challenges to the DHH remain. There are also some rather exciting newer developments.

## The hypothesis

The classic DHH can be rewritten to bring in an important, oft-neglected feature, the time-scale<sup>2</sup> (Fig. 1). While it takes only days for dietary fat excess to elevate SC levels (step 1), it takes decades for elevated SC levels to be translated into CHD (step 2). The transition of coronary artery disease (CAD) into a myocardial infarct or some other manifestation of CHD (step 3), on the other hand, occurs in a matter of seconds or minutes. This scheme of the DHH also takes cognizance of the influence of other dietary indiscretions suspected of influencing the risk of CHD, namely excessive kilojoule and salt intake. The long lag in time between induction of hypercholesterolaemia and the development of CHD, and the fact that epidemiological research has to rely on manifestations of CHD rather than the precursor CAD as an

endpoint, coupled with the confounding effects of many other environmental and genetic influences upon the DHH pathway, has made it difficult to validate the hypothesis completely.

The strongest evidence for step 1 comes from numerous metabolic unit studies carried out in the 1950s and 1960s. These studies, which were carried out under carefully controlled conditions, enabled Keys *et al.*<sup>3</sup> and Hegsted *et al.*<sup>4</sup> to elaborate formulae predicting the effect of various dietary fat modifications on SC levels. From these formulae it can be calculated that a decrease in total fat intake to 20% of energy intake from a previous 40%, and an increase in the dietary polyunsaturated/saturated fat (P:S) ratio to 1,0 from 0,4 (keeping fat at 40% of dietary energy), is equipotent in lowering SC levels by 15-18 mg/dl (0,3-0,5 mmol/l). Since a diet including 20% fat would be unacceptable to most, it is of interest to note that a compromise of a more modest fat reduction (to 30%) together with partial substitution of polyunsaturated fat for saturated fat (P:S = 1) is predicted to lower SC levels by 21 mg/dl (0,55 mmol/l). If, in addition, dietary cholesterol intake is cut back to 200 mg from a previous 500 mg, a total reduction of 29 mg/dl (0,75 mmol/l) can be obtained. In each of these cases the major part of the benefit would be obtained from the reduction in saturated fat intake.

The most compelling evidence in support of steps 2 and 3 combined, that raised SC predisposes to CAD + CHD, comes from prospective epidemiological studies such as the Pooling Project, which combines data from several American studies.<sup>5</sup> This study and several others have shown that the risk of mortality from CHD starts rising after the SC level reaches about 220 mg/dl (5,7 mmol/l), and suggests that in the higher ranges of SC the risk may even become exponential.

On the basis of these and other considerations, numerous learned bodies have recommended modification of the typical Western diet in order to lower the risk of CHD, the most well known of which is the McGovern Committee's *Dietary Goals for the United States*.<sup>6</sup> Basically, all have urged weight control, a reduction in intake of fat, especially saturated fat, partial substitution of polyunsaturates for saturates, and reduction in cholesterol intake. Concomitantly, the complex carbohydrate intake is increased to compensate for the reduced fat intake. Some have also advocated reduced salt intake. Most, but not all,

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have recommended that these changes be adopted by the entire westernized population, not only those with elevated SC levels.

Since such a recommendation may affect many people — perhaps half of the population — who are not at risk, it behoves nutritional scientists to examine the quality of the evidence for causality in the DHH very carefully indeed. This has been ably done by Hulley *et al.*<sup>2</sup> The kinds of evidence they consider are: non-epidemiological (pathophysiology studies, clinical cases of rare lipid disorders, and animal studies); observational epidemiological (international comparisons, secular trends, cross-sectional and prospective studies of individuals); and experimental or interventional epidemiological (metabolic ward studies, trials of CHD prevention).

Support for step 1, that diet influences the SC level, is moderately strong from animal-feeding experiments and also from international comparisons of diet and SC levels such as Keys *et al.*'s seven-country study. Secular trends in the USA, such as a reduction in SC *pari passu* with reductions in national consumption of saturated fats and cholesterol, provide mild evidence, as do studies of individuals within a community. The strongest evidence comes from metabolic ward studies of individuals and dietary trials of CHD prevention in populations. The latter have consistently shown a 10-15% reduction in SC levels.

For step 2, that a high SC level predisposes to CAD, moderate evidence is forthcoming from postmortem and angiographic studies, rare cases of lipid disorders such as familial hypercholesterolaemia, and animal studies. For step 3 (CAD → CHD) pathological studies indicate that almost all CHD patients have pre-existing CAD, including those with rare lipid disorders. International comparisons, secular trends and prospective studies all provide mild-to-moderate evidence of causality for steps 2 and 3 combined, while intervention studies have been inconclusive.

Ability to prove causality by some of these approaches is inherently limited, but the accumulated evidence is strongly in its favour (Table I). The arguments most commonly used to refute the DHH include statements that associations in population studies cannot prove causality, that within such populations the association between diet and SC levels or CHD is frequently unimpressive, that intervention trials have generally yielded disappointing results, and that results in animal studies cannot be extrapolated to humans.

The first of these arguments is valid, and reflects an inherent limitation of observational epidemiology. However, the strength of the association between SC levels and CHD, the dose-response relationship and the consistency with which it is found in many studies are in favour of causality.<sup>7</sup> With regard to the last point, if animal experiments cannot (with the necessary circumspection) be applied to humans, then much of medical science would have to be consigned to the waste-paper basket. The inability to show a relationship between diet and SC levels within a population should not surprise anyone, given the relative homogeneity of such a diet, variable individual responses, and the imprecision with which food intakes can be measured. In spite of these handicaps, studies have given positive results, the most recent of which was the Western Electric Study.<sup>8</sup> This study showed intrapopulation correlations between SC levels and dietary saturated fatty acids as well as with the dietary score of Keys *et al.* It also showed a significant association between changes in SC levels 19 years later and changes in saturated fatty acid intake, cholesterol intake and the score of Keys *et al.*

The criticism that dietary intervention trials have not lowered mortality from CHD has some validity, as indicated by inspection of data from the two best-controlled studies, the Helsinki Mental Hospital Study and the United States Veterans Administration (VA) Study.<sup>9</sup> Both studies used a high P:S ratio diet without lowering total fat intake. Cholesterol intake was modestly reduced in the VA trial (to about 365 mg/d), but the reduction in

**TABLE I. DIET AND HEART DISEASE — THE EVIDENCE**

FOR	AGAINST
1. Intergroup associations between consumption of cholesterol, total and saturated fat, and SC	1. Associations do not prove causality
2. Intergroup associations between dietary lipids and CHD	2. Within population groups the associations between diet and serum lipids are frequently unimpressive
3. Inter- and intrapopulation association of SC (and certain lipoproteins) with CHD	3. Dietary intervention trials have generally been inconclusive
4. Metabolic studies in animals and humans confirm effect of dietary lipids on SC	4. Results in animal experiments cannot be extrapolated to humans
5. In animals atherosclerosis can be induced by high-lipid diets and regressed by low-lipid diets	
6. Arterial lesions in animals and humans with certain hyperlipaemias contain excessive cholesterol	
7. Certain genetic hyperlipaemias established in early life show premature CHD	

the number of fatal infarcts was not significant. Non-fatal infarcts were significantly reduced in both, most notably so in the Helsinki study. Non-cardiovascular mortality increased in both studies, but overall mortality was similar in both the experimental and the control group. If one keeps in mind the fact that these were not primary prevention trials since many of the subjects already had arterial disease of long duration owing to their age, it is surprising that any favourable effect could be demonstrated at all. In addition, these were not trials of the 'prudent diet', since the total fat (and, in Helsinki, cholesterol) intake was not reduced. In consequence, the lowering in SC levels achieved was rather modest (12-18%).

Perhaps the most important observation to be made from these and four other dietary intervention trials is that all showed the same trend towards lower mortality and morbidity from CHD.<sup>10</sup> A primary prevention trial of sufficient statistical power to prove or disprove regression is unlikely ever to be carried out since it would require about 60 000 participants to be followed up for 10 years,<sup>11</sup> so that decisions about causality will, perforce, have to be taken on the basis of the present evidence.

### The role of certain foodstuffs — some controversies

The literature is replete with conflicting statements about the contributions of individual food components to risk of CHD, e.g. eggs, milk, margarine, sugar, fibre and salt. The controversies are partly due to the enormous variety of foodstuffs which interact and contribute towards serum lipid levels. The role of any single foodstuff is therefore likely to be modest; the cumulative effect of many different foods determines a favourable or unfavourable lipid profile. Only under strictly controlled conditions is it possible to isolate the effect of single foods.



## Eggs

Eggs are the richest source of dietary cholesterol, and before 1972 no less than 10 reports of rigorously controlled, albeit short-term, metabolic ward studies uniformly showed that egg consumption raised SC levels by a mean of 19%. Subsequently a number of egg supplementation studies in free-living and in hospitalized subjects failed to show such a relationship. These non-metabolic ward studies suffer from the drawback that they were not always possible to control — did the subjects actually eat the eggs, and if they did what other adaptations did they make in their diets? Recently, Roberts *et al.*<sup>12</sup> conducted a unique experiment in which half a cup of egg ( $\pm 400$  mg cholesterol) daily or a cholesterol-free egg substitute was administered to free-living subjects for 4 weeks in a double-blind, cross-over design such as is used for drug trials. The results were unequivocal: whole eggs raised the SC level and the egg substitute did not. This study can be regarded as definitive as regards the short-term effect of eggs on the SC level. Results of similar egg-feeding studies over a longer term have not yet been reported.

## Milk

The fats of whole milk are almost wholly saturated, and it would therefore be expected to raise SC levels if taken in sufficient quantity. Indeed, feeding with butterfat is one of the most efficient ways of inducing hypercholesterolaemia in animals. However, a number of workers<sup>13-15</sup> have suggested that full-cream milk (especially in the form of yoghurt) contains some substance in the aqueous phase (the 'milk factor') which actually lowers SC. Recent attempts in South Africa, Finland and Australia<sup>16-18</sup> to reproduce these results in humans have been uniformly unsuccessful. In the South African study full-cream milk and yoghurt increased SC during the first 2 weeks of a 3-week period, while only skim milk decreased it. The results were consonant with the reported changes in dietary fat and cholesterol intake induced by consuming 2 litres of each product. However, it is worth noting that the increases in SC levels in both the South African and the Australian studies were only about two-thirds of that predicted by the equation of Keys *et al.* These results were subsequently confirmed in a longer, 6-week study (J. E. Rossouw *et al.* — unpublished observations). It may therefore be concluded that South African cows do not produce the 'milk factor', or produce it in insufficient quantities!

## Margarine

In nature polyunsaturated fatty acids are folded back upon themselves at the double bond (the *cis* form), but during partial hydrogenation double bonds are broken and reformed so that some fatty acids end up in the *trans* form, with the carbon skeleton strung out on either side of the double bond. *Trans* fatty acids have no essential fatty activity, even if they are polyunsaturated.<sup>19</sup> Their effect on SC is about midway between that of mono-unsaturated and that of saturated fatty acids.<sup>20</sup> They should therefore probably be grouped with saturated fatty acids. Unfortunately, at present the South African public has no way of knowing the *trans* fatty content of margarines, which may be as high as 20% in polyunsaturated margarines and even higher in brick-form margarines. The effective P:S ratio of a polyunsaturated margarine may thus be only 1,0 as against the expected 2,0. Since a ratio of 2,0 is technologically feasible, food-labelling legislation is urgently required to correct this anomalous situation. Such legislation could also expose the hollowness of advertising claims that certain brands contain 0% cholesterol. All South African margarines are made from sunflower oil and contain no cholesterol; their effect on serum cholesterol levels therefore depends entirely on the P : S ratio. It should be noted that even at

a P : S ratio of 2,0, margarine has a neutral effect on serum cholesterol. Only in comparison with butter can it be said to have a favourable effect.

## Sugar

Countries with a high per capita consumption of sugar tend to have a high rate of CHD.<sup>21</sup> However, owing to socio-economic reasons sugar consumption is closely linked to fat intake. Multivariate analyses of cross-cultural data have generally shown no independent effect of sugar consumption on mortality from CHD.<sup>21,22</sup> Sugar does not appear to influence low-density lipoprotein (LDL) cholesterol, but may increase triglycerides<sup>23</sup> and decrease high-density lipoprotein (HDL) cholesterol levels.<sup>24</sup> In the main, the evidence linking sugar and CHD is not very persuasive.

## Fibre

Wheat bran, the kind of fibre obtained from eating wholewheat bread and 'high-bulk' breakfast cereals, has very little or no effect on serum lipid levels.<sup>25</sup> Oats do seem to lower LDL cholesterol levels,<sup>26</sup> as does food fibre from legumes, vegetables and fruits, i.e. gums and pectins.<sup>27</sup> It is of interest that the reduction in SC levels induced by food fibres is not usually associated with any reduction in HDL cholesterol.<sup>26</sup> The mechanism of the hypocholesterolaemic effect of fibre may involve increased faecal bile-acid loss.<sup>26</sup> In cross-cultural studies total food fibre intake is negatively correlated with fat intake, and a low fibre intake may therefore appear to be associated with CHD in univariate analyses. When the interaction with fat intake is controlled for, no significant effect of food fibre intake remains.<sup>22</sup>

## Salt

In international comparisons there is a clear relationship between salt consumption and rise in blood pressure with age. Populations consuming 5 g or less of salt per day do not show such a rise, while those consuming 10 g or more (as is the case in most Western cultures) do experience an increase in blood pressure with age. As with dietary fats and SC levels, it is likely that only those individuals with a constitutional predisposition to hypertension will develop it, given a uniformly high exposure to dietary salt. It is therefore unlikely that an association between salt intake and hypertension will be very striking within a community.<sup>28</sup>

While there is no doubt that extreme low-salt diets (such as the Kempner rice diet) lower blood pressure, these are extremely unpalatable. There has been some controversy about the effects of more moderate salt restriction, to say 5 g/d. MacGregor *et al.*<sup>29</sup> have recently provided an elegant answer. By using the double-blind placebo-controlled cross-over technique they could show that moderate salt restriction lowered blood pressure in patients with mild hypertension, and that this reduction was maintained on placebo but reversed when slow-release sodium tablets were given to return the salt intake to pre-diet levels. The dietary advice given was simply to abstain from excessively salty foods and to add no salt during the cooking of food or at the table.

## Potential hazards of lowering SC levels

Before recommendations about lowering SC levels can be made to the general public, it is essential to be sure that these measures are not only effective but also safe. Some of the putative hazards are cancer, gallstones, a lowering of HDL cholesterol levels and an increase in overall mortality. These are discussed below.



## Cancer

Carcinogenicity is an emotive issue and worthy of serious consideration, since animal experiments do show that polyunsaturated fatty acids have some co-carcinogenic potential.<sup>30</sup> The Los Angeles VA trial also suggested a higher incidence of cancer among the treated group, but pooling of the data with those from four other smaller diet trials provides some reassurance that there is no overall increased risk of cancer.<sup>31</sup> In the VA trial the cancers developed most frequently in those ingesting the smallest amount of the experimental diet. The large recently published Oslo trial also provided no evidence of increased cancer mortality.<sup>32</sup> Indeed, it would be surprising if such a relationship existed, since in humans the best epidemiological correlation with the occurrence of certain cancers (e.g. of the breast, prostate and colon) is that of total fat intake. Reduction in fat intake, especially if coupled with a more vegetarian, high-fibre eating pattern, is more likely to reduce than to increase the risk of cancer.

## Gallstones

The evidence for a greater incidence of gallstones again rests upon the VA diet trial,<sup>33</sup> and to a lesser extent the World Health Organization clofibrate trial.<sup>34</sup> In the latter it is of course impossible to separate the direct effect of clofibrate from that of a lowering of cholesterol. The same comment holds for the reported (not significant) excess of cancer in the clofibrate trial. The VA study was the only diet trial to report an increased incidence of gallstones. Human experiments show that increased flux of biliary sterols occurs upon changing from a high saturated to a high polyunsaturated fatty acid diet, but this appears to be a transient phenomenon.<sup>35</sup> Whether this effect is sufficient to precipitate gallstone formation is still an open question. Preliminary data from a primate study indicate that the 'prudent' diet is likely to be less lithogenic than the 'Western' diet (J. E. Rossouw *et al.* — unpublished observations).

## HDL cholesterol

What about HDL cholesterol? Early work using unrealistically high P:S ratios showed a decrease in presumably protective HDL levels in subjects on diets high in polyunsaturates.<sup>36</sup> Most subsequent studies have shown either no effect or a lesser reduction in HDL than in LDL cholesterol on diets closer to the 'prudent diet'.<sup>27, 32</sup> The net effect is usually an increase in the HDL:LDL cholesterol ratio.

## Overall mortality

Very recently a number of prospective studies have shown a J-shaped relationship between SC level and overall mortality, with more cases of cancer of the colon among men and of strokes among women at the lower end of the cholesterol distribution.<sup>37</sup> The reasons for these associations are obscure. In some studies the association was only evident for the first 2 years of follow-up, suggesting that the neoplasm was already present at the time of screening.<sup>38</sup> Another possibility is that people with low SC levels are efficient excretors of dietary cholesterol, and that their high faecal sterol levels predispose them to cancer of the colon.<sup>39</sup> If this is so, then a low-fat, high-fibre diet which reduces faecal sterol concentrations<sup>27</sup> should be advantageous rather than deleterious. In any event, the association between low SC levels and overall mortality does not, on the basis of present evidence, provide any compelling reason to modify advice regarding reduction of SC, since this reduction does not appear to be associated with an increased risk of cancer. In addition, it should be borne in mind that many peoples who have enjoyed a low-fat diet all their lives as well as those who have adopted it in later life

(e.g. vegetarians) seem to have a singularly low rate of colon cancer as well as of CHD.

## Alternative diets

Where do we go from here? Are there any other fruitful avenues which can be explored in search of an alternative diet? The very low fat diet (the 'longevity' diet) advocated by Pritikin, which provides only 10-20% of dietary energy in the form of fat, makes good sense on a superficial level since populations consuming such a diet experience virtually no CHD. However, it is likely that the low saturated fat content of such a diet is the important factor, since some populations, e.g. Cretans, with a high fat intake of mainly the mono-unsaturated type have a low incidence of CHD.<sup>21</sup> The unacceptability of the 'longevity' diet to the Western palate is its main disadvantage. Of interest in the South African context is the fact that the 'longevity' diet is fairly close to the traditional diet of Blacks, and the incidence of CHD is very low in Blacks.

The Mediterranean diet with its moderately low total fat content (mainly mono-unsaturates, very low in saturates), low cholesterol and animal products and high carbohydrate content may be a more acceptable alternative. CHD mortality is low in Mediterranean countries,<sup>21</sup> and when an Italian diet was fed to North Karelian families a decrease in SC levels of 20% was induced. Interestingly, blood pressures also fell in spite of an unchanged salt intake (J. K. Huttunen — personal communication).

Oliver<sup>9</sup> has promoted the idea that a relative deficiency of essential fatty acids may be important in some circumstances, and cites the Scots as an example of a population with a high rate of CHD and a low linoleic acid content intake in cholesteryl esters, while their SC levels are not particularly high. The Scots do have a low (< 3% of dietary energy) intake of polyunsaturated fatty acids and this suggests the attractive possibility that simply supplementation with polyunsaturated fatty acids may ameliorate the CHD problem. This suggestion has not been put to the test of an intervention study, but it seems unlikely that such a mechanism is of importance except in very unusual circumstances. Populations consuming polyunsaturated fatty acids to the degree of 4-8% of dietary energy (e.g. American Whites<sup>35</sup>) have a CHD rate similar to that of the Scots.

The very low apparent incidence of CHD among Eskimos, who largely subsist on fish, seal meat and blubber, has stimulated interest in the biological effects of the marine oils. Eskimos have very low linoleic acid levels in their cholesteryl esters, but high eicosapenta-enoic acid levels.<sup>9</sup> Unsaturated fats are classed into the  $\omega 9$ ,  $\omega 6$  or  $\omega 3$  series according to where the first double bond lies from the methyl end. Oleic acid (C18:1), the main representative of the  $\omega 9$  family, is non-essential since it can be synthesized in the body. The  $\omega 6$  family is the common polyunsaturated family, with linoleic acid (C18:2) most common. It is found in vegetable oils, is essential, and is a precursor of prostaglandins.  $\omega 3$  fatty acids are found mostly in marine oils, and only to a limited extent in seeds and leaves. In marine oils eicosapenta-enoic acid (C20:5) predominates, while in seeds  $\alpha$ -linolenic acid (C18:3) predominates.

Fish oils are particularly interesting since they are at least as effective as vegetable oils in reducing SC levels and more effective in reducing serum triglyceride levels, but do not affect HDL cholesterol.<sup>35</sup> Much recent interest has centred around the effects of polyunsaturated fatty acids on platelet and vessel wall function. Linoleic acid is converted in the platelet to a thromboxane (TXA<sub>2</sub>) which is pro-aggregatory, while in the vessel wall it is converted to an anti-aggregatory prostacyclin. Similarly, fish oils such as eicosapenta-enoic acid are converted to a thromboxane, but this thromboxane is inactive. More importantly, eicosapenta-enoic acid decreases the arachidonic acid concentra-



tion of platelets, and blocks  $\text{TXA}_2$  synthesis from it. The net effect is anti-aggregatory to such an extent that bleeding time is prolonged.<sup>35</sup> Although other polyunsaturated fatty acids may be anti-aggregatory, fish oils are unique in this respect. Perhaps this is the secret of the Eskimos' immunity to CHD; if so, it vividly illustrates that the effects of dietary fats and oils go beyond their influence on serum lipids.

### Potential benefits of the 'prudent diet'

A diet which is fat-modified, high in food fibres and low in salt may have advantages (Table II) other than lipid-lowering qualities, although these benefits need to be confirmed. They include reduced thrombogenicity of blood via the effect of polyunsaturated fatty acids on platelets, referred to above. In contrast, saturated fatty acids actually increase blood thrombogenicity.<sup>35</sup> Preliminary human studies indicate that blood pressure may be lowered by a fat-modified diet, even in the absence of salt restriction;<sup>40</sup> this effect may be mediated by prostaglandins in the kidney.<sup>41</sup> Lowering of SC levels may, in addition, reduce vascular reactivity to catecholamines.<sup>42</sup> Glucose tolerance may improve,<sup>43</sup> although the mechanism for this improvement is ill-understood. A high-fibre, high-carbohydrate diet has been shown to improve diabetic control.<sup>44</sup> It is likely that such a diet will reduce rather than increase the risk of colon and other cancers. High fibre intake may also have other beneficial effects on bowel function. It has been suggested that such a diet will result in less constipation, diverticulosis, appendicitis, haemorrhoids and gallstones.<sup>45</sup> High salt intake is epidemiologically associated with gastric cancer,<sup>46</sup> as well as with hypertension; reduced intake may conceivably have benefits beyond lowering of blood pressure.

TABLE II. POTENTIAL BENEFITS OF THE 'PRUDENT DIET'

Lowering of SC and LDL cholesterol levels
Reduced blood thrombogenicity
Reduced blood pressure
Improved glucose tolerance
Reduced risk of cancer
Improved large-bowel function

### Who should be the target population?

If we accept that the 'prudent diet' is beneficial and safe, the next question that arises concerns the groups on which intervention should be focused. Clearly certain individuals exposed to a westernized dietary milieu will be more 'at risk' of CHD than others owing to the fact that they have responded to that milieu by developing hypercholesterolaemia, hypertension, hyperglycaemia or obesity. Together with cigarette smokers, they will form the majority. The great bulk of future CHD victims will come from their ranks.<sup>47</sup> Should intervention be aimed only at them? The first and major problem with such an individualized 'medical' approach is that these high-risk persons would first have to be identified. If an appreciable proportion are to be identified, massive screening programmes would have to be undertaken in our high-risk populations, i.e. the Whites, Indians and urban Coloureds. Such programmes are clearly not feasible either from an economic or a logistic viewpoint. If intervention is to be reserved only for those who have presented themselves to the health care system, it will have a very limited impact on the total problem of CHD, not least because many members of this self-selected population are already suffering from the complications of atherosclerosis and would therefore not benefit much from secondary prevention. In addition, employment of conven-

tional cut-off points for SC and blood pressure in order to identify high-risk individuals will miss those who fall outside the upper quintile of the risk factor distribution, but who will nevertheless develop CHD owing to modest elevations coupled with other constitutional and environmental predispositions.

The alternative approach is to treat the entire westernized community as being 'at risk'. Community intervention would almost certainly be more rewarding than individual intervention, since the high-risk segment would be more likely to follow dietary and other lifestyle modifications if it became the community norm. The purpose of such intervention would be to shift the distribution of risk factors to the left. Even a modest shift of, for instance, total cholesterol levels could have a significant impact on CHD mortality rates, since it can be predicted that a 1 mg/dl (0,025 mmol/l) decrease in mean SC may diminish the number of deaths from CHD by 1%.<sup>2</sup> If all the elements of the fat-modified diet were to be complied with, the resultant 29 mg/dl (0,75 mmol/l) decrease in the mean SC level would then predict a similar decrease in CHD. If other dietary modifications are made, such as a reduction in energy, increase in food fibre and decrease in salt intake, the potential savings in lives could become truly remarkable. However, the objectives should be kept realistic since changes in community habits would by their very nature be slow to materialize. The time-span is also a long one, since the 'incubation period' for risk factor expression as CHD is at least 2 decades.<sup>48</sup> Maximal regression may well have a similar time-scale. Potentially the most rewarding segment of the community would be the young, since true primary prevention would be possible in them. Established fibrotic plaques in the middle-aged are less likely to benefit from a reduction in SC, as judged from primate regression experiments.

### Conclusions

Study of the facts at our disposal in 1982 leads to the conclusion that certain dietary modifications are very likely to be effective in reducing the incidence of CHD, and that they are safe. They include energy management in order to achieve and maintain near-ideal body mass, a lowering of total fat intake and especially a reduction of saturated fat intake to <30% of energy, a modest increase of polyunsaturated fatty acid (especially from fish) intake, a lowering of cholesterol intake to less than 300 mg (250 mg for women) per day, an increase in food fibre intake (especially from fruits and vegetables), and a reduction in salt intake to less than 5 g/d. Such a diet can be expected to lower the incidence of mortality from CHD, strokes and certain forms of cancer, and not to create new health hazards. It is also practical since the modifications needed are relatively modest, and makes economic sense because the increased emphasis on plant proteins, oils and fibre and on fish would ease the pressure on the householder's budget due to the high cost of producing animal proteins and fat. If the agricultural sector takes note of buying trends resulting from an active heart health campaign and makes the necessary adaptations timeously, there is no need for any particular sector to be economically disadvantaged. An ever-growing population will still have to be fed.

Rigorous scientific proof to substantiate some of these recommendations is lacking, but the weight and diversity of evidence in favour of them is such that they can be accepted as being proven beyond reasonable doubt. It is noteworthy that in countries where public education campaigns towards a healthier diet have been in progress for some years, such as Australia and the USA, the mortality rate from CHD is falling. In England, where the DHH is still regarded with some scepticism, the rate has remained constant.<sup>50</sup> Of course, diet is not the only lifestyle factor to have improved in those countries in which mortality



from CHD is declining: better control of blood pressure, a decrease in smoking and increased leisure-time activity may all have contributed, together with improvement in the management of myocardial infarctions. Nevertheless, these favourable trends do make the point that death from CHD is preventable.

Finally, the most sensible target group for intervention should be the entire westernized population, with special emphasis on the young and on the need for lifelong maintenance of a healthy lifestyle. Hopefully the hard lessons learned by westernized communities will be used to advantage to prevent those segments of the South African society not yet so compromised from making the same mistakes.

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