Are there conflicts in dietary advice for prevention of different diseases?

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The recent World Health Organization (1990) report *Diet, Nutrition and the Prevention of Chronic Disease*, which issued quantitative dietary guidelines towards reducing the incidence of many chronic diseases, addressed the question of whether the dietary advice issued for the different diseases were mutually compatible; 'In theory it is possible for a specified intake of a nutrient to increase the risk of one disease while decreasing the risk of another disease; further, this relationship could itself vary for different segments of the population'. Having addressed the question, the answer is promptly provided along predictable lines: 'Fortunately, this problem does not seem to arise, since the dietary recommendations for preventing most of the conditions prevalent in developed countries are very similar'. Table 1 presents a summary of such recommendations and in general terms the answer presented seems fair. In no instance where advice is issued does it conflict with the different diseases and conditions. Therefore, for those concerned with the nutritional aspects of public health policy and health education, there is little cause for concern that the advice being given for one disease will cause problems for another.

Nutritional science cannot, however, adopt so confident a view and indeed the further one is from the issues of public health and the closer one is to the research side of nutrition, the greater is the lack of certainty in this area. To begin with we recognize that the quality of the data on which this advice is based ranges from fair to almost non-existent. In the case of coronary heart disease (CHD) we know quite a lot about the pathology of atherosclerosis and thrombosis, about the physiology and biochemistry of associated biological risk factors and about how diet influences the directions of these risk factors. In the case of cancer the issue is much less clear cut since cancers develop in many tissues and organs and we lack any detailed information on biological risk factors. Our knowledge of how diet might be related to cancer lacks any biological intermediary and is, therefore, confined to statistical associations generated in epidemiological investigations. Because of this dearth of knowledge of the associations between diet, biological risk factors and cancer, we are obliged to accept that we do not know whether

Table 1. *Summary advice for modifications to the national diet for the prevention of chronic disease*

	Obesity	Coronary heart disease	Cancer	Diseases of the digestive tract
Energy above needs	↓	↓	↓	↓
Dietary fibre	↑	↑	↑	↑
Fat	↓	↓	↓	?
Salt	?	↓	↓	?
Alcohol	↓	?	↓	?

↓, Decrease; ↑, increase; ?, uncertain.

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dietary changes proposed to reduce CHD will influence the risk of cancer, other than from data on mortality.

DO DIETARY GUIDELINES PRODUCE A BALANCED DIET?

In examining areas of dietary advice where adverse effects might occur, it seems reasonable to begin by asking the question of whether dietary advice, when taken up by ordinary people, produces a balanced diet. The level of hard information in this area is poor. The most useful study comes from the recent work of Retzlaff *et al.* (1991) who studied 396 men at or above the 75th percentile for age-adjusted blood cholesterol. The men were subject to intense nutrition counselling and were followed over a 2-year period. A most striking observation of the study was the marked decline in total energy intake of about 1·3 MJ/d. Almost all this decline was due to a reduction in fat intake (1·3 MJ/d) with protein showing a negligible change (−50 kJ/d) and the modest decline in alcohol energy (−90 kJ/d) cancelling out the modest rise in carbohydrate intake (+200 kJ/d). Most of the dietary guidelines work on the premise that reductions in fat-energy will be compensated for by an increase in energy from complex carbohydrates. The findings of Retzlaff *et al.* (1991) would suggest that that is not the case. Whether reductions in alcohol-energy or sucrose-energy lead to long-term compensation in energy intakes from other sources remains to be seen. When micronutrient intakes were compared, there were significant reductions in the intakes of zinc, niacin, vitamin E and selenium and significant increases in the intakes of retinol, β-carotene, folate, vitamin C, magnesium, vitamin B_6, and thiamin. The decline in Zn intakes may be a cause for concern given the increased phytate content of the diet. The decline in vitamin E intakes was associated with lower fat intakes. However, the reductions in fat intakes (−9·9%) were attributable largely to the saturated fatty acids (SFA; −5·3%) and the mono-unsaturated fatty acids (−4·2%) with only a modest increase in the intake of the polyunsaturated fatty acids (PUFA; +0·4%). Since vitamin E requirements are linked to the intake of the latter, the observed decline does not appear to be of any significance. This aspect will be addressed later.

In contrast to this study of Retzlaff *et al.* (1991), other studies of free-living, normal healthy, normocholesterolaemic individuals, given less-intensive nutrition counselling would indicate that problems could arise. Bradley & Theobald (1988) gave practical advice to such people and observed a significant reduction in energy intake of 1·6 MJ/d over 60% of which was due to a fall in fat consumption. The intakes of thiamin, riboflavin and calcium fell considerably in women, largely because of a reduction in the intakes of dairy products. In considering possible adverse effects in the adoption of dietary guidelines by the general public, we need to consider groups with low levels of intakes of nutrients derived largely from foods which might be restricted under such circumstances. Socially disadvantaged women in Dublin were found to have lower iron intakes than the national average (8·1 *v.* 10·6 mg/d) with lower intakes of meat and poultry (96 *v.* 160 g/10 MJ per d). Clearly any uptake in advice that suggested a reduction in red meat intake among such women would pose difficulties (Gibney & Lee, 1991*a*).

DIETARY FATS AND BLOOD CHOLESTEROL

That low-fat diets reduce total and low-density-lipoprotein (LDL)-cholesterol is widely accepted. What is frequently ignored is that half the studies which have shown this effect

have also reported a decline in high-density-lipoprotein (HDL). Whilst the former would be considered desirable, the latter would not. Between 1980 and 1988, about thirteen papers were published on the effect of low-fat diets on plasma lipids. These are summarized in Table 2, for the seventeen groups of which data is available in these papers. Only two of the eighteen studies failed to show a decline in plasma total cholesterol, five failed to show a decline in LDL-cholesterol and of the eighteen studies, eight showed a decline in HDL-cholesterol. This frequency with which low-fat diets are found to lower HDL-cholesterol, may not have received much attention in the realm of public health; it has not, however, been ignored by the realm of science. Several groups have sought to explain this anomaly, the most significant of which was that of Brinton *et al.* (1990). Subjects were selected to show a wide range of HDL levels in plasma and were studied while on low-fat and high-fat diets. Both fractional catabolic rate (FCR) and transport rate (TR) of the main HDL apoprotein, apo A-I, were studied. The degree of decline in HDL-cholesterol on low-fat diets correlated significantly with changes in TR of apo A-I but not with apo A-I FCR. However, within any of the two diets, the level of HDL-cholesterol correlated significantly with apo A-I FCR but not TR. Thus, the determinant of a prevailing level of HDL-cholesterol (FCR) differs from the determinant of a change in HDL-cholesterol (TR). This might suggest that at any given level of HDL-cholesterol, a homeostasis exists which is perturbed when the level of fat is changed, presumably to re-establish homeostasis at the new level. In other words there may be an adaptive period. The values in Table 2 show that the mean duration of studies which showed a significant fall in HDL (4·1 weeks) was half that of the studies where no

Table 2. *The effects of low-fat diets on plasma cholesterol*
(Data selected from the literature for the period 1980–88 inclusive)

	Initial		Final		Change in blood cholesterol (mg/100 ml)			Duration
	Fat-energy (%)	P:S	Fat-energy (%)	P:S	Total	LDL	HDL	(weeks)
Schaefer *et al.* (1981)	37	0·3	20	0·3	–45*	–35*	–12*	2
Jackson *et al.* (1987)	40	0·4	15	0·4	–31*	–25*	– 6*	5
Bowman *et al.* (1988)	46	0·4	31	0·4	– 8*	+ 3	–10*	5
	46	0·4	31	0·4	–27*	17	– 9*	5
Brussard *et al.* (1982)	31	1·2	21	0·6	– 2	– 1	– 4	13
Wolf & Grundy (1983)	40	0·5	30	1·0	–12	–14	– 7*	4
	40	0·5	30	0·6	–16*	–18*	– 3	4
Kuusi *et al.* (1985)	38	0·2	23	0·9	–38*	–27*	–10	12
Weisweiler *et al.* (1985)	42	0·1	32	1·0	–26*	14*	+ 1	6
Kohlmeier *et al.* (1985)	43	0·2	31	2·8	–55*	–46*	+ 2	2
Grundy (1986)	40	0·3	30	1·0	–35*	–10*	+ 5	8
	40	0·3	20	1·0	–40*	–18*	0	8
Sacks *et al.* (1986)	35	0·6	27	1·2	–23*	–22*	– 3	12
Mensink & Katan (1987)	38	0·2	22	0·7	–18*	—	– 8*	5
Denke & Breslow (1988)	42	0·1	25	1·5	–37*	–20*	– 5*	3
Grundy & Vega (1988)	40	—	20	1·0	–15*	–10	– 5*	6

P:S, polyunsaturated fatty acids:saturated fatty acids; LDL, low-density-lipoprotein; HDL, high-density-lipoprotein.
*$P<0·05$.

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change was observed (8·1 weeks). It remains possible, therefore, that adequate time may be needed before adaptation to a low-fat diet becomes complete.

During the same period fifteen studies were carried out on the effects of substituting SFA with PUFA. Almost all showed a decline in total and LDL-cholesterol with none showing a decline in HDL-cholesterol (Table 3). In contrast, many studies have shown that when monounsaturated fatty acids (MUFA) are used to replace SFA, a significant decline in HDL is observed (Table 4). If the same approach is taken with these studies as was applied to the low-fat diet data, a definite pattern emerges: where HDL is lowered, the mean PUFA:SFA (P:S) ratio for the diets was 4·7, while for those studies where the P:S ratio remained unaltered the value was 2·1. Clearly, the levels of PUFA frequently mentioned in dietary guidelines are below this apparently safe value.

On balance, therefore, it seems that the general types of advice on dietary fat issued to bring about a reduction in LDL-cholesterol do not create problems in other areas. However, these data do show that much basic work on mechanisms is needed before we can confidently predict how dietary fat influences blood cholesterol fractions.

SUGAR, ALCOHOL AND THE PERCENTAGE OF DIETARY ENERGY FROM FATS

One of the main targets of the dietary guidelines is for a reduction in the percentage of dietary energy from fat. Because of this method of expressing fat intakes, the value is subject to considerable variation, not necessarily because of a variation in absolute

Table 3. *The effects of increased intakes of polyunsaturated fatty acids (PUFA) on plasma cholesterol*

(Data selected from the literature for the period 1980–88)

	Initial		Final		Change in blood cholesterol (mg/100 ml)		
	Fat-energy (%)	P:S	Fat-energy (%)	P:S	Total	LDL	HDL
Schwandt *et al.* (1982)	37	0·3	37	1·1	–32*	28*	+ 2
Vega *et al.* (1982)	40	0·2	40	1·4	–13*	56*	– 8*
Becker *et al.* (1983)	40	0·2	40	5·0	–10*	–16*	+ 5
Harris *et al.* (1983)	40	0·7	40	3·8	–27*	–12*	+ 1
Mattson & Grundy (1985)	40	0·2	40	5·8	–37*	–30*	– 5*
Weisweiler *et al.* (1985)	42	0·2	42	1·0	–25*	–21*	+ 2
Grundy (1986)	40	0·3	40	1·7	–35*	– 8	0
Weintraub *et al.* (1988)	42	0·1	42	1·5	–32*	–20*	– 4
Turner *et al.* (1981)	40	0·2	40	8·0	–32*	–24*	– 6*
	40	0·2	40	8·0	–54*	52*	– 5*
Shepherd *et al.* (1980)	40	0·3	40	4·0	–49*	–29*	–15*
	40	0·3	40	4·0	–45*	–30*	– 9*
Schaefer *et al.* (1981)	37	0·3	40	2·0	–29	18*	– 8*
McNamara *et al.* (1987)	35	0·3	35	1·5	–25*	13*	– 3
	35	0·3	35	1·5	–24*	–12*	– 2

P:S, polyunsaturated fatty acids:saturated fatty acids; LDL, low-density-lipoprotein; HDL, high-density-lipoprotein.

*$P<0·05$.

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Table 4. *The effects of increased intakes of monounsaturated fatty acids on blood cholesterol*

	Initial		Final		Change in blood cholesterol (mg/100 ml)		
	Fat-energy (%)	P:S	Fat-energy (%)	P:S	Total	LDL	HDL
Becker *et al.* (1983)	35	—	40	1·0	–39*	–32*	– 3
Mattson & Grundy (1985)	40	0·2	40	2·0	–31*	–32*	– 1
Grundy (1986)	40	0·3	40	2·0	–34*	–32*	– 3
Mensink & Katan (1987)	38	0·2	40	0·5	–17*	—	+ 2
Bonanome & Grundy (1988)	40	0·2	40	1·6	–22*	–21*	+ 1
Baggio *et al.* (1988)	28	0·3	38	0·4	–21*	–20*	0
Grundy & Vega (1988)	40	—	40	0·9	–19*	– 3	– 1
Mensink & Katan (1989)	37	0·2	37	0·6	–29*	–23*	– 4

P:S, polyunsaturated fatty acids:saturated fatty acids; LDL, low-density-lipoprotein; HDL, high-density-lipoprotein.
*$P<0·05$.

intake of fat, but because of the diluting effect of sugar- and alcohol-energy. An inverse relationship exists between the percentage energy from alcohol and that of fat (Gibney *et al.* 1989) and the same holds true for sugar and fat (Gibney, 1990). These issues are raised in the context of an analysis of the compatibility of dietary guidelines for two reasons. The first is that if reductions in sugar- and alcohol-energy are not compensated for with increases in non-fat-energy, then the percentage of energy from fat must increase. Whether that is disadvantageous or not remains to be seen. The second reason why it is worth considering this issue in this context is that it illustrates our changing knowledge of how alcohol and carbohydrate contribute to energy. Recent work by Colditz *et al.* (1991) has analysed data from two large cohort studies, the Nurses Health Study (121 700 females) and the Health Professionals Follow-up Study (51 529 males). Increasing alcohol intake did not depress energy intake from foods in men, and while it did so in women the differences in food energy intake across increasing levels of alcohol intake among drinkers only were not significant. In effect alcohol-energy increases total energy intake. However, it does not lead to increased body mass index (BMI) in men and actually suppresses BMI in women. An associated paper *The case of the missing calories* proposes a futile cycle of oxidation of alcohol to acetaldehyde with a reduction of acetaldehyde back to alcohol (Lands & Zakhari, 1991) with the loss of 6 mol ATP. Several such cycles could negate the energy derived from alcohol oxidation in carbon dioxide. If alcohol makes no net contribution to energy or less than we imagine at present, much of what is written on the relationship between fat-energy and blood cholesterol will have to be revised. In the Seven Countries Study, for example, alcohol-energy contributed as little as 3% of total energy in Finland and as much as 20% in Yugoslavia (Keys *et al.* 1986). Recent data from the Italian rural cohorts of the Seven Countries Study shows that fat-energy increases from 28 to 36% of energy when alcohol-energy (20%) is excluded (Farchi *et al.* 1989).

The analysis of existing data on BMI and alcohol-energy by Colditz *et al.* (1991) was prompted by conflicting evidence in the literature based on smaller studies. At present, there exists conflicting evidence on the relationship between energy intake from sugars

and that of total energy intake and BMI. The data of Gibney & Lee (1991*b*) suggest that energy from sugar, like that of alcohol, is added to the energy from other foods, resulting in an excess energy intake but not an increase in BMI. Other studies have not shown sugar-energy to be simply a supplement to other food energy (Department of Health, 1989). It would of course be foolish to propose that sucrose has less metabolizable energy than is currently believed. There is, however, sufficient evidence at present to suggest that excess carbohydrates may be less likely to lead to obesity than excess fat (Acheson *et al.* 1982). More recently, direct measurement of hepatic *de novo* lipogenesis in man using stable isotopes has shown that even with loads of over 200 g carbohydrate, only trivial levels of fat (<0·5 g) are synthesized (Hellerstein *et al.* 1991). Given the very low capacity of human adipose tissue to synthesize fatty acids *de novo* (Shrago *et al.* 1969), these authors raised the provocative question: 'At present, however, our results indicate that a fundamental question remains unanswered, namely, where do excess carbohydrate calories go in humans' (Hellerstein *et al.* 1991).

The consideration of the relationship between BMI, and total fat-, alcohol- and carbohydrate-energy serves to reveal the quite fragile level of our understanding about some of the issues we as nutritionists consider tried and tested facts. That has to have a bearing on how we answer the question of whether or not dietary guidelines for different diseases or conditions (e.g. CHD and obesity) are compatible.

DIETARY FIBRE

In recent years there has been an increased interest in the role of 'soluble fibre' (water-soluble non-starch polysaccharides) in reducing blood cholesterol, an interest showed both by science and commerce. In general, the hypocholesterolaemic effects of brans rich in soluble fibre, such as oat bran, are modest and these modest gains may even be rendered insignificant when dietary fat is reduced. However, increased fibre intakes from such brans will have a much smaller effect on faecal output than brans rich in water-insoluble fibre. High-fibre diets based on cereal fibre produce significantly higher faecal output (288 g/d) compared with a similar fibre content based on soluble fibre from vegetables, pulses, fruits and potatoes (108 g/d; Forsum *et al.* 1990). What is good for the cardiologist, may not be the best for the gastroenterologist!

Ca–Fe INTERACTIONS

Ca is a nutrient which has begun to enter into consideration in the development of dietary guidelines because of its proposed link with osteoporosis, hypertension and colo-rectal cancer. Certainly, reduced Ca intakes due to reduced dairy product consumption would stir a controversy. The tendency to recommend increased or at least adequate Ca intakes in women can have a considerable effect on Fe absorption. Hallberg *et al.* (1991) examined the effect of Ca fortification of flour or bread rolls on Fe absorption and showed a significant inhibitory effect up to levels of 150 mg Ca. Either milk or cheese, providing this level of Ca, also led to a comparable inhibitory effect on Fe absorption. A scenario which saw a decreased intake of haem-Fe because of a reduction in red meat intake, coupled with higher intakes of low-fat, Ca-rich dairy products and a higher intake of phytate-rich, fibrous foods, could conceivably cause problems for some groups of females.

VITAMIN E AND PUFA

Beginning in the early 1970s and continuing on until relatively recently, much of the research into lipoprotein metabolism and atherosclerosis concentrated on LDL-receptor function. This has led to a fine understanding of the mechanisms involved in atherosclerosis associated with single gene defects in some aspects of LDL-receptor activity. Since the vast majority of the population have perfectly functioning LDL receptors, this approach did not directly assist in our understanding of why such people with normal LDL function can accumulate cholesteryl esters intracellularly. Indeed it led one expert to plead that we emulate Aladdin and let the 'Gene' out of the 'Culture Flask'! In the course of this work, it became evident that if anything was defective in the normal population, it was the LDL particle rather than the LDL receptor, such that LDL was accumulated in cells through a non-regulatory alternative (scavenger) receptor. The most obvious defect to LDL emerged as damage associated with oxidation. On that basis, interest in the role of vitamin E in cardiovascular disease intensified and a considerable literature now exists. In the experimental sphere, this ranges from reduced aortic atheromatous lesions in cholesterol-fed rabbits given vitamin E supplements (Wojcicki *et al.* 1991) to reduce cholesteryl ester (linoleate) oxidation by cultured human monocyte macrophages with supplemental vitamin E (Carpenter *et al.* 1990). This year, two very important papers were published, both showing a negative relationship between plasma vitamin E and CHD in men. Riemersma *et al.* (1991) in a study of 110 cases of angina identified by Chest Pain Questionnaire but not under clinical management, found that vitamin E remained independently and inversely related to the risk of angina after adjustment of age, smoking habit, blood pressure, lipids and relative weight. Gey *et al.* (1991) in the WHO–MONICA collaborative study showed a similar inverse relationship between vitamin E levels in plasma and the risk of CHD across Europe. It would now seem necessary to address the issue of whether our dietary advice for reducing CHD risk factors influences vitamin E status.

Retzlaff *et al.* (1991) in their study of the effects of intense dietary counselling on micronutrient status in hypercholesterolaemic men found a significant fall in vitamin E intakes. This fall was directly related to the degree of reduction in the proportion of dietary energy from total fat. In turn, most of this fall was due to a reduction in SFA with the intakes of PUFA remaining constant. On that basis it could be argued that the decline in vitamin E did not pose a problem since vitamin E requirements are related to PUFA intake. However, an inevitable consequence of this is that the vitamin E:PUFA ratio in the diet would have fallen. Calculations based on the data of Retzlaff *et al.* (1991) would suggest a trivial fall from 0·62 to 0·58 mg vitamin E/g PUFA. More detailed analysis of this data is not possible but would be welcome. In the general population of the USA, this ratio is observed to fall with increasing intakes of PUFA from 0·94 with under 5 g PUFA/d intake, to 0·44 at >25 g/d (Murphy *et al.* 1990). If, however, ratios at the 25th percentile are considered then the comparable fall in ratio over the same range is from 0·59 to 0·35 indicating that 25% of the US population with intakes of PUFA>10 g/d are at or below the accepted minimum ratio of 0·4. In the future, greater attention will have to be paid to possible detrimental effects of dietary advice on this ratio. The problem may become more pressing if evidence emerges that vitamin E intakes should be related to blood LDL-cholesterol levels as well as PUFA intake.

Most of the PUFA in the diet is of the *n*-6 series derived from plant oils. There is, at least in the nutrition literature, a rapidly increasing interest in the marine oil-derived *n*-3

PUFA because of the capacity to reduce the thrombotic tendency of blood. Given that the *n*-3 PUFA have a far greater number of double bonds than the *n*-6 PUFA, their capacity for oxidation is much greater. Schafer & Overvad (1990) have shown that adipose tissue levels of vitamin E are positively correlated with vitamin E intake and negatively correlated with adipose tissue *n*-3 PUFA, such that together these two factors account for 82% of the variability of adipose tissue vitamin E. This of course supports the concept that vitamin E functions to scavenge PUFA peroxyl radicals. Encapsulated supplements of *n*-3 PUFA have also become popular. Meydani *et al.* (1991) have shown that such supplements, notwithstanding their fortification with vitamin E, lead to significant increases in circulating malondialdehyde (MDA), a crude but useful marker of lipid peroxidation. This increase was particularly marked in older subjects increasing at the rate of 1·25 mmol MDA/ml plasma per month of supplementation.

Given the relative novelty of the role of vitamin E in cardiovascular disease and the relative dearth of literature on the role of diet in determining vitamin E levels in plasma, it may seem somewhat unfair to ask those who issue dietary guidelines to give guarantees that their advice will not be detrimental to vitamin E status. It is, however, one area which merits immediate attention by nutritional science.

EPIDEMIOLOGY

It is not unreasonable to propose that any of the following could mediate as biological risk factors in the relationship between diet and chronic disease: cytokines, monocytes, neutrophils, macrophages, LDL, HDL_2, HDL_3, oxysterols, growth factors, fibrinogen, platelet-activating factor, lipoprotein (a), nitric oxide, vitamin E, vitamin C, β-carotene, the renin–angiotension loop, post-prandial lipaemia, hormones, colonic fermentation, lysolecithin, xenobiotic metabolism, Ca, eicasanoids, copper:Zn ratios and so on. Indeed many of these have been reviewed for their role in chronic disease and some have merited entire symposia, if not several symposia. When we address the question of whether dietary guidelines for a reduction in the various chronic diseases in which these may play a role, are mutually compatible, we reach an impasse. For many of these, we know nothing of the direction in which they would move with dietary intervention and how the myriad biological effects of dietary intervention would pan out in disease statistics. The only alternative available is to see how disease and mortality statistics emerge on implementing dietary advice. If there is a nutritional panacea for all chronic disease and if our advice is in the direction of this panacea, then we should see a decline in all diseases when dietary advice is implemented.

It is, however, difficult using time-related statistics within one country or using international comparisons, to dissociate the impact of dietary advice from other factors such as smoking, screening programmes, pharmacological management of hypertension and hypercholesterolaemia, stress management, exercise, cardiovascular surgical procedures and acute medical care. One approach is, therefore, to look at intervention studies which have introduced a single factor, whether diet or drugs, to lower blood cholesterol. Earlier this year, Oliver (1991) undertook such an analysis in a paper with the provocative title: *Might treatment of hypercholesterolaemia increase non-cardiac mortality*. Oliver (1991) draws our attention to the fact that non-cardiac deaths increased significantly in many of the major single risk factor (hypercholesterolaemia) intervention trials such as WHO Clofibrate Trial, The Lipid Research Clinics Trial, The Helsinki

Heart Study, The Los Angeles Veterans Administration Trial and the Finnish Mental Hospitals Study. He further cites meta-analysis of existing data by Muldoon *et al.* (1990) using six trials with data on 24 849 males, by Rossouw *et al.* (1990) using eight trials with 7837 participants in secondary prevention studies and Peto *et al.* (1991) with data on 42 000 subjects in twenty-two primary and secondary prevention trials. All three meta-analyses show a significant increase in non-cardiac mortality in groups treated for lowering blood cholesterol. (Of course, all these studies did show a lowering of CHD.) Oliver (1991) points out in this review: 'At present, available data indicate that total mortality is unchanged when hypercholesterolaemia is lowered; the fall in cardiac mortality is offset by an apparent increase in non-cardiac deaths. These findings can no longer be dismissed as a statistical quirk which will hopefully disappear when new trials are reported. The problem was first raised in 1978 and has been observed consistently since then.'

Which brings us back to the original question. Are there conflicts in dietary advice for prevention of different diseases? For those involved in public health policy and involved in nutrition education in the clinical or community setting, the fair answer is 'no'. For nutritional science, however, on which such groups depend for guidance, the answer has to be: 'We really do not know enough to be able to answer that question'.

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