occurred at the level of patient, general practitioner, general physician, or cardiologist. At angiography elderly patients had more severe and extensive coronary disease and had developed greater myocardial impairment, and as these factors, the severity of symptoms, and the need for urgent surgery have all been identified as predictors of surgical mortality[6] these patients have an intrinsically higher operative risk, even before the effect of age is considered. We believe, therefore, that earlier referral and investigation of elderly patients who may be candidates for cardiac surgery—at a stage when their symptoms are limiting but not yet unstable—would yield a group for whom surgery could provide important symptomatic benefit, but at lower risk.

The implementation of such a policy would, in the context of our changing demography, place increased stress on already seriously overextended cardiac diagnostic facilities and staffing.[7] Additionally, the provision of cardiac surgery within the NHS has tended to be restricted, and most cardiac surgery units have long waiting lists, often extending to over a year for non-urgent cases. In such circumstances of limited resources there is a danger that the medical needs of elderly patients may be looked on less favourably than those of younger patients, a prejudice termed agism.[8] Although this method of discriminating between patients competing for a finite resource may be convenient, it is rarely rational, and we are not aware of any public consensus regarding prejudicial selection on the grounds of age as a means of distributing treatment within the NHS. If finite resources require some patients to receive less than optimal treatment then this should be enacted by excluding those with less potential for benefit. A 70 year old patient with limiting angina and an average life expectancy of 10-14 years may often have such potential overlooked.

The successful outcome of coronary artery surgery in elderly patients with stable symptoms who are found at angiography to have coronary disease suitable for elective surgery supports the view that it is appropriate for an elderly patient with limiting angina to be referred for coronary angiography and, when necessary, added to the cardiac surgical waiting list. Only in this way can the necessary level of funding for cardiac surgery be identified.

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Dietary reduction of serum cholesterol concentration: time to think again

L E Ramsay, W W Yeo, P R Jackson

Abstract

***Objective*—To evaluate the long term efficacy of diets in lowering serum cholesterol concentration.**

***Design*—Descriptive overview of 16 published controlled trials of six months' duration or longer.**

***Setting*—Trials had been conducted in hospital clinics (6), industry (3), mental hospitals or institutions (3), and in general populations (4).**

***Patients*—Trials had been conducted in high risk subjects (5), in unselected healthy subjects (6), or for secondary prevention in patients with coronary heart disease (5). Women were included in only four trials.**

***Interventions*—Diets equivalent to the step 1 diet were employed in eight trials, with individual intervention by dietitians (3) or occupational physicians (2) or with population advice (3). Intensive diets which were more rigorous than the step 2 diet were employed in eight trials.**

***Main outcome measures*—Net change in serum total cholesterol concentration in subjects receiving treatment with diet compared with values in control subjects after six months to 10 years.**

***Results*—In five trials with the step 1 diet as individual intervention the net reduction in serum cholesterol concentration ranged from 0% to 4·0% over six months to six years. In trials with population education reductions in cholesterol concentrations were 0·6-2·0% over five to 10 years. When population and individual dietary advice were combined changes in cholesterol concentration ranged from a fall of 2·1% to a rise of 1·0% over four to 10 years. Diets more intensive than the step 2 diet reduced serum cholesterol concentration by 13% over five years in selected high risk men in the population; by 6·5-15·1% over two to five years in hospital outpatients; and by 12·8-15·5% over one to four and a half years in patients in institutions.**

***Conclusions*—The response to a step 1 diet is too small to have any value in the clinical management of adults with serum cholesterol concentrations above 6·5 mmol/l. Current guidelines recommend screening of serum cholesterol concentration in healthy subjects, followed by treatment with a step 1 diet. The guidelines should be reviewed to provide a more realistic estimate of the effect of a step 1 diet and of the likely need for lipid lowering drugs.**

University Department of Medicine and Pharmacology, Section of Pharmacology and Therapeutics, Royal Hallamshire Hospital, Sheffield S10 2JF
L E Ramsay, FRCP, *consultant physician and professor*
W W Yeo, MRCP, *senior registrar and lecturer*
P R Jackson, MRCP, *consultant physician and senior lecturer*

Correspondence to:
Professor Ramsay.

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Introduction

Every 1% reduction in serum cholesterol concentration reduces the risk of coronary events by about 1-2%.[1] Guidelines for managing patients with high cholesterol concentrations concur that diet is of prime importance in management,[2-7] and advocate as initial treatment the step 1,[6] or general lipid lowering diet (box).[8] If this proves insufficient the more intensive step 2 diet is advised (box).[6] These recommendations are based on epidemiological considerations[9] and short term experiments.[10] Estimates of cholesterol reduction by the step 1 diet range from 10% to 25%,[6 8] but there are suggestions that it may be insufficiently effective.[11-13] In Britain about 40% of adults have serum cholesterol concentrations in the moderate to high risk category and are therefore considered to need clinical care,[14-16] and a standing medical advisory committee has emphasised that cost effective management

depends on effective diet and avoiding the widespread use of lipid lowering drugs.[17] We reviewed long term controlled trials of the effect of diets on serum cholesterol concentration, focusing on the step 1 diet, which is the cornerstone of recent recommendations.[2-6]

Diets recommended for reduction of serum cholesterol[6]

Step 1 diet
- Total fat—less than 30% of total calories
- Ratio of polyunsaturated fat to saturated fat—1·0
- Cholesterol—less than 300 mg daily
- Calories—reduced to achieve desirable weight

Step 2 diet
- Total fat—less than 30% of total calories
- Ratio of polyunsaturated fat to saturated fat—1·4
- Cholesterol—less than 200 mg daily
- Calories—reduced to achieve desirable weight

Methods

Controlled trials of at least six months' duration that examined the effect of lipid lowering diets on serum cholesterol concentration were included whether diet was the only intervention or part of multiple risk factor intervention. Trials are presented as those of step 1 or equivalent diets or of more intensive diets. The number and type of subjects, setting, duration, baseline serum cholesterol concentration, and percentage fall in cholesterol concentration are tabulated. The number of subjects refers to those sampled in the intervention group at the stated time. Duration was usually until the time of final serum cholesterol measurements, but in four small trials data at two years are cited because numbers were very small beyond this.[18-21] In the World Health Organisation European trial[22 23] results from British centres after five to six years were published separately as the United Kingdom heart disease prevention project.[24 25] The five to six year results for the other centres (in Belgium, Italy, Poland, and Spain) have not been published. We have tabulated results for the United Kingdom heart disease prevention project at five to six years and for the World Health Organisation European trial excluding the United Kingdom results at four years. The intensity of intervention varied during these trials, so that the results at four years reflect maximal effort and those at five to six years less intensive effort. For the World Health Organisation European trial weighted means for the four countries excluding the United Kingdom were calculated,[22 23] as differences between centres were within the limits of sampling error.[22] Percentage change in serum cholesterol concentration was calculated as the difference in measurements in the intervention group compared with the control group expressed as a percentage of the value in the intervention group at randomisation.[22 24] The 95% confidence intervals were calculated as the difference between groups $\pm 1{\cdot}96 \times$ standard error of difference.

Results

STEP 1 DIET AS INDIVIDUAL INTERVENTION

Description of trials—Five controlled trials of step 1 or equivalent diets in individual subjects met the criteria described above (table I). The United Kingdom heart disease prevention project and the World Health Organisation European trial were aimed at reducing cholesterol concentration, cigarette smoking, body weight, and blood pressure and increasing exercise. Men were randomised according to the factory where they worked to intervention or control groups. The multiple risk factor intervention trial randomly allocated high risk men to intensive intervention to reduce smoking, blood pressure, and serum cholesterol concentration or to ordinary care.[26] The diet and reinfarction trial included a random controlled trial of dietary cholesterol reduction in men who had survived a myocardial infarction.[27] Curzio *et al* randomised hypertensive subjects with serum cholesterol concentration >6·5 mmol/l to diet or control groups.[28]

Serum cholesterol responses—Net falls in serum cholesterol concentration in these trials ranged from 0% to 4·0% over six months to six years, with the average fall being about 2%. The reduction in the multiple risk factor intervention trial (2·0%) was significant, but changes in three trials were not.[25 27 28] Only 34 women were included in these trials.

Statistical power and patient selection—In the two smallest studies 95% confidence intervals for change in cholesterol concentration were 3·0% to −3·0%[28] and −1·4% to −5·6%,[27] and the larger studies evidently had sufficient power to exclude type 2 error as an explanation for the small responses. Selection of subjects is unlikely to have influenced the outcome in the United Kingdom heart disease prevention project, the World Health Organisation European trial, or the multiple risk factor intervention trial. In the diet and reinfarction trial patients who intended to follow an intervention diet were excluded, and there may have been some bias against intervention. In the trial of Curzio *et al* failure of 12% of patients to complete the study may have biased the outcome in favour of diet.[28]

Changes in control groups—In the multiple risk factor intervention trial study end points changed in the control group, and it was suggested that trial procedures may have influenced control subjects or that popu-

TABLE I—*Controlled trials of step 1 or equivalent diet to lower cholesterol concentrations*

Trial	Setting, subjects*	No of subjects	% Men	Duration (years)	Baseline cholesterol (mmol/l)	Change in cholesterol
	Individual intervention					
United Kingdom heart disease prevention project[24 25]	Factories, high risk	1278	100	5-6	6·6	−0·9%
World Health Organisation European trial[22 23]*	Factories, high risk	1898	100	4	6·7	−4·0%
Multiple risk factor intervention trial[26]	Employees, high risk	6428	100	6	6·2	−2·0%
Diet and reinfarction trial[27]	Hospital, after a myocardial infarction	982	100	2	6·5	−3·5%
Curzio *et al*[28]	Hospital, high risk	61	44	0·5	7·1	0·0%
	Mass intervention					
North Karelia[29]	Population	2535	49	10	7·1	−2·0%
Stanford[30]	Population (cohort)	490	47	5·5	5·5	−0·6%
	(cross section)			5·5	5·4	−1·7%
	Combined individual plus mass intervention					
United Kingdom heart disease prevention project[24 25]	Factories, all subjects	5373	100	5-6	5·6	+1·0%
World Health Organisation European trial[22 23]*	Factories, all subjects	824	100	4	5·6	−2·1%
Gothenburg[31]	Male population	1473	100	10	6·5	−0·2%

*Excluding results from United Kingdom centres.

TABLE II—*Controlled trials of diets more intensive than step 1 diet to lower cholesterol concentrations*

Trial	Setting, subjects	No of subjects	% Men	Duration (years)	Baseline cholesterol (mmol/l)	Change in cholesterol
	Free living subjects					
Oslo study[34 35]	Population, high risk	604	100	5	8·3	−13·0%
Leren[37]	Hospital, after a myocardial infarction	206	100	5	7·7	−13·9%
Medical Research Council committee[18] (soya bean oil)	Hospital, after a myocardial infarction	169	100	2	7·1	−15·1%
Research committee[19] (low fat diet)	Hospital, after a myocardial infarction	81	100	2	6·8	−8·1%
Rose *et al*[20] (corn oil)	Hospital, with ischaemic heart disease	13		2	6·8	−6·5%
	Subjects in institutions					
Minnesota[38]	Mental hospitals	4541	48	1	5·4	−13·5%
Finnish mental hospital study[39]	Mental hospitals	300	100	4·5	7·0	−15·5%
Dayton *et al*[21]	Veterans' centre	163	100	2	6·1	−12·8%

lation habits may have changed coincidentally. The authors of the diet and reinfarction trial and Curzio *et al* also advanced changes in control groups to explain, in part, the disappointing outcome. In absolute terms serum cholesterol concentration increased over five to six years in the intervention subjects in the United Kingdom heart disease prevention project and fell by 5·0% in the multiple risk factor intervention trial, 2·8% in the diet and reinfarction trial, and 4·2% in the study of Curzio *et al*. Reductions in serum cholesterol concentration were therefore modest, averaging about 3%, even when examined in this way.

Intervention methods—In the United Kingdom heart disease prevention project and the World Health Organisation European trial subjects were given personalised dietary advice based on diary records. The cholesterol response varied with the intensity of intervention.[25] The 4·0% reduction in cholesterol concentration at four years in the World Health Organisation trial probably reflects maximum effort, whereas the 0·9% reduction at five to six years in the United Kingdom heart disease prevention project typifies responses at other times during the trial. In the multiple risk factor intervention trial dietary advice started with weekly small group sessions, followed by individual counselling by behavioural scientists and nutritionists. In the diet and reinfarction trial advice was given by hospital dietitians who visited and telephoned regularly to reinforce their instructions. In the trial of Curzio *et al* individualised dietary advice was provided by hospital dietitians. Subjects in these trials evidently had the benefit of individual instruction at least equal to that currently available in ordinary practice.

In the multiple risk factor intervention trial the diet conformed to the step 1 diet initially, but it was intensified later to give a polyunsaturated fat to saturated fat ratio of 1·25 and cholesterol intake of 250 mg daily. The 2% reduction in cholesterol concentration at six years was therefore achieved by a diet more intensive than the step 1 diet. In the diet and reinfarction trial no mention was made of dietary cholesterol reduction, but this does not influence the response.[32] The diet employed by Curzio *et al* was equivalent to a step 1 diet for most patients, but it was more rigorous in some [J Curzio, personal communication]. The diets used were therefore more intensive than the step 1 diet in two trials, and broadly equivalent to the step 1 diet in the others. Dietary adherence was assessed only in the diet and reinfarction trial and was incomplete.[27]

Summary—These trials encompass different clinical settings, including primary prevention in high risk men[22 24 26] or hypertensive patients[28] and secondary prevention after myocardial infarction.[27] Despite this, changes in serum cholesterol concentration differed little, with a mean fall of about 2% (range 0-4%). Small reductions in cholesterol concentration cannot be attributed to lack of statistical power, changes in control groups, or subject selection. The precise contribution of inadequate intervention effort, non-adherence, and an insufficiently rigorous diet is uncertain, but diets at least equivalent to the step 1 diet clearly have a meagre effect on cholesterol concentration given the resources available and adherence expected in ordinary practice.

Mass intervention with step 1 diet—In the controlled but not random north Karelia trial population education produced net reductions in serum cholesterol concentration of 2-3% at five to 10 years.[29 33] Reductions in men (3-4%) were significant but those in women (1%) were not. In the Stanford five city project population intervention achieved non-significant mean reductions in cholesterol concentration of 0·6% by cohort sampling and 1·7% by cross sectional sampling after five years.[30]

Combined individual and mass intervention—Three studies examined population education combined with individual advice to high risk subjects. In the United Kingdom heart disease prevention project serum cholesterol concentration increased by 1·0% at five to six years, and in the World Health Organisation European trial there was a reduction of 2·1% at four years. In the Gothenburg trial the net fall in serum cholesterol concentration at 10 years was 0·2%.[31] This small response was attributed to a fall in cholesterol concentration in control subjects. At four years, however, intervention reduced cholesterol concentration by 1·2% from control values, and 0·6% from baseline values. Changes in control subjects clearly could not explain the small response at four years.

TRIALS OF MORE RIGOROUS DIET

The Oslo study[34 35] deserves special attention because it is invariably cited to support the dietary measures recommended in various guidelines. In this random controlled trial diet reduced serum cholesterol concentration by 13% over five years (table II), and, in conjunction with a reduction in cigarette smoking, reduced the incidence of myocardial infarction and sudden death by 47%. Several important points are commonly overlooked. Men were recruited by a single letter of invitation, and the 35% who did not respond probably included those least likely to comply. Subjects were then selected according to their dietary habits.[35] Those already following a fat restricted diet were excluded, but the number excluded was not stated. The men studied had severely increased serum cholesterol concentration of between 7·5 mmol/l and 9·8 mmol/l. Perhaps because of these selection procedures the subjects had a very high intake of dietary fat, averaging 44% of total energy. This is much higher than the average intake in British men (35-37%[27 36]) or the United States population (35-40%[6]). The diet used reduced total fat intake to 28% of energy intake and raised the ratio of polyunsaturated fat to saturated fat from 0·39 to 1·01, and it was more rigorous than the step 2 diet (for which total fat intake can be up to 30%.)

The correct conclusion from the Oslo study is that rigorous dietary intervention in male volunteers with very high serum cholesterol concentrations and very high dietary fat intake caused a substantial fall in serum cholesterol concentration. Together with some reduction in cigarette smoking this resulted in an important decline in the incidence of coronary heart disease. Its results cannot, however, be extrapolated generally, particularly to those with less severe hyperlipidaemia; to those with more typical dietary fat intake; to women; or to the outcome with the step 1 diet. The Oslo study does not, in short, support the policies set out in recent guidelines and, conversely, recent guidelines do not recommend the form of intervention tested so successfully in the study.

In four small trials in free living subjects rigorous low fat diets reduced serum cholesterol concentration substantially by 6·5-15·1% (table II).[18-20 37] In three controlled trials in people living in institutions serum cholesterol concentration was reduced by 12·8-15·5% over one year to four and a half years (table II). The diets studied were very intensive, attaining ratios of polyunsaturated fats to saturated fats of 1·6[38] and 1·5,[39] whereas the step 2 diet aims at a ratio of only 1·4. These trials leave no doubt that modification of diet can lower serum cholesterol concentration substantially, but the diets were all more intense than those now advised and, in varying degrees, unpalatable. It seems that the dietary treatment must be unpleasant to be effective.

Discussion

Dietary change undoubtedly can lower serum cholesterol concentration, as shown by reductions averaging 12% over one to five years with rigorous diets.[18-21 34 35 37-39] However, the step 1 diet has little effect on serum cholesterol concentration in free living subjects. In trials of intervention in individual high risk subjects reductions in cholesterol concentration have averaged about 2% (range 0-4%) over six months to six years. These small responses could be due to inadequate intervention effort in some studies,[24 27] but not others,[26] or to incomplete adherence,[27 37] but above all reflect an insufficiently rigorous diet. Responses were similarly small in trials of population education[29 30 33] and when population education was combined with individual advice for subjects at higher risk,[22 25 31] with falls in cholesterol concentration averaging about 1%. Changes in control groups do not explain the small responses as falls in concentration from baseline values averaged only 3%.[25-28] Changes in control groups are in any event of doubtful relevance. Health education measures require the same rigorous evaluation as new treatments[25] and should be judged by the same yardstick. The true worth of an intervention is measured only by the net difference between intervention and control groups. Subjects treated by diet are sometimes classed as "responders" or "non-responders."[40] When the mean effect of diet is close to zero, as in these trials, responders must be balanced by a similar number of people who respond adversely. If reductions in cholesterol concentration in individuals are regarded as real and not simply due to random variation, increases in concentration must also be considered real and potentially harmful. It is wrong to count as successes the responders and disregard those whose cholesterol concentration moved in the wrong direction.

The efficacy of the step 1 diet, which is based on epidemiological considerations[9] and short term studies,[10] has been questioned surprisingly little. Ahrens, one of the few authors to express reservations about current dietary recommendations,[11 12] predicted a reduction in serum cholesterol concentration of 6% given the adherence expected in ordinary practice.[11] In the event this projection has proved overoptimistic. The best estimate of cholesterol reduction is 2%, and even the smallest trials had sufficient power to exclude reductions as large as 6%. These small responses occurred despite resources at least equal to those currently available in ordinary practice. What benefit might be expected from the reductions in serum cholesterol concentration observed in these trials of the step 1 diet? By using as a rule of thumb a 1·5% reduction in coronary events for a 1% fall in total cholesterol concentration,[1] a fall of 2% may translate to a reduction in coronary events of about 3%. A more rigorous diet is required to attain any important reduction in serum cholesterol concentration, but the feasibility, acceptability, and effectivenss of the step 2 diet have not been tested in long term controlled trials in free living subjects. The trials summarised in table II all employed diets more rigorous than the step 2 diet.

These results contrast sharply with assertions in many guidelines and reviews that serum cholesterol concentration will fall by 10-25% in response to a step 1 diet.[4-8 14 41] Why are perceptions of efficacy so unrealistic? Among the reasons are overreliance on short term experiments, controlled studies of rigorous diets in "captive" populations, and uncontrolled observations. The Oslo study has understandably been given considerable weight, but with no recognition that the subjects were highly selected and the diet much more rigorous than the step 1 diet. Evidence from the other controlled trials reviewed here has been ignored, with uncontrolled studies purporting to show efficacy being cited instead. For example, one study[42] cited by a standing medical advisory committee[17] is held to show efficacy of dietary intervention over six years. In this uncontrolled trial serum cholesterol concentration fell from 6·03 mmol/l to 6·01 mmol/l over one year—a change of 0%.[40] The responses at six years [42] were attained by "losing" non-responders, a manoeuvre which is inappropriate, as discussed above. A perception of efficacy is reinforced in everyday practice by regression to the mean, which may produce a fall in cholesterol concentration of 5% between two visits without any intervention.[26 34] This is the likely explanation of larger responses to diet in some uncontrolled trials.[43] The inflated perception of efficacy affects appraisal of cost effectiveness. Kristiansen *et al* concluded that a strategy of screening cholesterol concentration followed by dietary advice alone might cost £12 400 per life year gained in middle aged men and about £62 000 in women.[44] However, these calculations assumed a reduction in cholesterol concentration by diet of 10%. The cost per life year gained with a 2% cholesterol reduction by the step 1 diet would be about £62 000 for men and £310 000 for women. Use of lipid lowering agents would apparently increase costs about 10-fold.[44]

Recent guidelines suggest that most people with abnormal cholesterol concentrations—as many as 90%[41]—will be managed by diet, with few subjects needing lipid lowering drugs.[4-8 45] This seems totally unrealistic. In Britain general practitioners who screen can expect cholesterol concentrations higher than 6·5 mmol/l in about 40% of adults,[15 16 46] who are considered to be at moderate to high risk and to need clinical care.[4 5 14] The target serum concentration recommended is 5·2 mmol/l,[4 5 8 14 45 47] and the fall in concentration required is therefore at least 20%. Given a response of 2%, use of the step 1 diet cannot possibly attain or even approach the target serum cholesterol concentration. Doctors and their patients are being boxed into an impossible corner by current guidelines. When the step 1 diet fails the more intensive step 2 diet is advised, although its feasibility and acceptability have not been examined and the resources for such intensive intervention are not available to most general practitioners.[16]

The consequences are not difficult to predict. Doctors will have two options—to leave the raised cholesterol concentration uncorrected or prescribe lipid lowering drugs. As has been pointed out,[48] the "cholesterol numbers" dominate medical consultations, and widespread use of lipid lowering drugs is the likely outcome. Considering the high prevalence of people with moderate to high risk cholesterol concentrations in the British population this is an unattractive proposition.[47] Doctors need to formulate their policy for screening, recognising that the diet recommended has little impact on serum cholesterol concentration and that screening followed by an ineffective diet may force the use of lipid lowering drugs. Knowledge of the serum cholesterol concentration is not essential to identify high risk subjects,[49] and a decision not to screen need not be a recipe for inaction. Much can be gained from tackling other risk factors such as cigarette smoking, hypertension, and obesity, and for those at very high risk treatment with aspirin is an option.[50]

Brett has discussed the ethical aspects of dietary intervention when unequivocal proof of benefit is lacking.[51] He considered such dietary advice ethical provided that there was hope of benefit and the diet was harmless. Considering these criteria, little benefit can be expected from a step 1 diet used for intervention in individuals—perhaps a 3% reduction in coronary events. Concern has been expressed about possible risks of lowering cholesterol concentration,[52 53] but a step 1 diet may be considered harmful in a broader sense. It expends scarce and costly resources on an intervention which has proved largely ineffective in several controlled trials. Furthermore, belief that diet is effective may foster wide use of lipid lowering drugs and transform healthy subjects into patients, consequences certainly not intended by those who issued guidelines or advised governments on policy. The ethics of seeking out healthy individuals, measuring cholesterol concentrations, and offering intervention of such limited efficacy needs to be reconsidered. Guidelines for detection and management of raised cholesterol concentration should be revised to incorporate a more realistic estimate of the response to diet. The above strictures do not necessarily apply to dietary advice as a population intervention. It is of concern, however, that this has proved effective in lowering cholesterol concentration in only one non-random trial, and in that trial only in men.[30 31]

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