

after one month of the experimental diet (total fat 26% of energy; ratio of polyunsaturated to saturated fatty acids 0.75; cholesterol 314 mg/day) in both sexes. This lower concentration (means 3.72 mmol/l in men and 3.86 mmol/l in women) was constantly found throughout the following seven months of the study. We are convinced that a similar, or probably even stronger, effect on cholesterol concentrations would be seen in an identical study of patients with hypercholesterolaemia.

In conclusion, we believe that the lack of success of the eight trials to which Ramsay and colleagues refer was due not to a failure of the recommended diet to lower cholesterol concentration but to the fact that we are unable to make people eat what we want them to. This is the main problem to be solved for the health professionals concerned with primary prevention of arterial disease. How do we improve the degree to which dietary advice is followed?

The statement of Ramsay and colleagues that dietary treatment must be unpleasant to be effective was also strongly contradicted in our study. A sociological evaluation of the participants' perception of the experimental diet showed that it was well accepted and well liked.⁶ We emphasise finally that serum cholesterol concentration is only one of several risk markers of atherothrombotic disease that are influenced by diet. A step 1 type diet may also favourably affect systolic blood pressure and the components of blood coagulation and fibrinolysis.^{4,5,7}

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SIR,—The overview by L E Ramsay and colleagues showing the failure of dietary trials to reduce serum cholesterol concentration leads the authors to suggest that it is "time to think again."¹ This is salutary, but the evidence reviewed has been known for some time and repeatedly brought to the attention of cholesterol enthusiasts, without effect as they "think" in a different mode. While cholesterol screening does "expand scarce and costly resources on an intervention which has proved largely ineffective in several controlled trials," as Ramsay *et al* rightly point out, it also provides handsome profits to the cholesterol industry and it would be naive to expect that this industry would wind down simply because some critics say that the cholesterol story is unconvincing. After all, the diet that has been shown ineffective is the same as that recently recommended by a panel of experts in the World Health Organisation's dietary guidelines, with specific reference to cholesterol lowering, and is intended for the whole world.²

Ramsay *et al*, perhaps wisely, deal with only one aspect of the diet-heart hypothesis, but even this area is discussed too narrowly. They claim that the step 2 diet is "effective"—but being effective in lowering cholesterol is not synonymous with being effective in reducing mortality. For example, in a study quoted by the authors, serum cholesterol concentration was lowered by about 15% with the use of an extreme diet, but mortality from coronary heart disease and total mortality did not change; in fact they were slightly, though insignificantly, higher.³ This and other similar studies point towards the falsity of another dogma accepted by Ramsay *et al* in their first sentence as a fact: "Every 1% reduction in serum cholesterol concentration reduces the risk of coronary events by about 1-2%."¹

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SIR,—In their discussion of uncontrolled trials aimed at reducing serum cholesterol concentration, L E Ramsay and colleagues¹ make no mention of a study that I and colleagues published in 1972.² This showed the feasibility of achieving substantial reductions (mean 22%) in the plasma cholesterol concentrations of subjects with moderate hyperlipidaemia (total cholesterol concentration initially >6.5 mmol/l) by means of dietary management over a protracted period (mean follow up 18.7 months).

In our ignorance, we restricted monounsaturated as well as saturated fatty acids, but the dietary changes advised were otherwise essentially similar to those reviewed. We attributed the useful and prolonged reductions that our dietitians achieved to their knowledge and enthusiasm, regular (dietetic and medical) monitoring, the emphasis on educating the spouse who did the shopping and cooking, and a policy of telling the subjects what they could (and should) eat as well as what they should avoid. In addition, a book of recipes was provided to show that the recommended diet need not be dull. Perhaps in consequence, most subjects found that their tastes changed in favour of foodstuffs containing less saturated fat.

Though it may be impracticable to provide this type of management on a large scale, these findings indicate what can be done to help highly motivated subjects—as those who know their cholesterol concentrations to be too high tend to be, particularly if a friend or relative has suffered a coronary event.

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SIR,—It is hard to fathom how L E Ramsay and colleagues decided that the diets used in the trials cited in table II of their paper were more "effective" than the step 1 diet.¹ They defined the step 1 diet by three criteria: percentage energy derived from dietary fat, ratio of polyunsaturated to saturated fatty acids, and cholesterol content.

Comparison of intervention diets in various trials with step 1 diet

Trial	Intervention diet		
	Fat as % of total energy	Polyunsaturated: saturated fatty acids	Cholesterol (mg/day)
Oslo ²	28	1.0	289
Leren ¹	39	2.4	199
Medical Research Council ¹	46	2.0	258
Minnesota ¹	38	0.6	166
Finnish Mental Hospital ¹	31-32	1.42-1.78	186-271
Dayton <i>et al</i> ¹	39	>0.6	365
Step 1	<30	1.0	<300

Information on these criteria is available for six of the eight trials they cite (table).

In comparison with the step 1 diet only the diet in the Oslo trial can be said to be more intensive on all three criteria, and then only marginally so. When this trial is excluded the mean reduction in serum cholesterol concentration observed in the others was a worthwhile 14%, similar to the reduction observed with the prudent diet over six months in a lipid clinic.³

Flawed analyses lead to faulty conclusions, and it is unfortunate that the authors' comments on screening have already been the subject of discussion in the lay press.⁴ The notion that hyperlipidaemia will stop being a problem if it is ignored may be economically appealing but is scientifically absurd.

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AUTHORS' REPLY,—The mean reductions in serum cholesterol concentration of about 14% in the trials that Gilbert R Thompson tabulates would indeed be worth while and are not in dispute. The point at issue is the composition of the diets studied. We agree that they were not more intense than the step 1 diet by all three criteria, but five of the six were more rigorous even than the step 2 diet on at least one criterion. The other trial is difficult to evaluate because the diet of the control group was so atypical.¹

Is Thompson seriously suggesting that these trials provide evidence on the efficacy of the diets now recommended for managing high cholesterol concentrations? With the exception of the Oslo trial,² which we discussed in detail, the trials in his table either were conducted in institutionalised subjects or were of diets that proved unpalatable. The trials summarised in table I of our paper were