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# Reducing serum cholesterol

## Confusion remains over whom to treat

EDITOR,—Not all authors accept that lowering blood cholesterol concentration improves the chances of avoiding coronary heart disease.[1 2] Only four of the 35 clinical trials relating blood cholesterol concentration to death from coronary heart disease that George Davey Smith and colleagues analysed[3] yielded significant effects, and one of these, the World Health Organisation's study in 1978,[4 5] showed an increased rate in the intervention group.

By classifying the trials according to whether they included patients at high, medium, or low risk on the basis of the deaths from coronary heart disease per 1000 person years in the control group, Davey Smith and colleagues showed the benefit of treating the high risk group with cholesterol lowering drugs. But how does this help in the selection of patients to receive drug treatment when the clinician does not have an indicative control group? If it is argued that high risk patients should be treated then what constitutes a high risk group? If this is to be determined by the "classical" risk factors—cigarette smoking, hypertension, and high blood cholesterol concentration—the argument is circular.

If the values in table I are used to relate the mean baseline blood cholesterol concentrations quoted in the paper with deaths from coronary heart disease per 1000 person years in the control groups there is no significant relation (for all studies reporting cholesterol concentrations: $r=-0{\cdot}228$, n=33, $p>0{\cdot}05$; primary studies: $r=-0{\cdot}125$, n=7, $p>0{\cdot}05$; secondary studies: $r=-0{\cdot}194$, n=25, $p>0{\cdot}05$).

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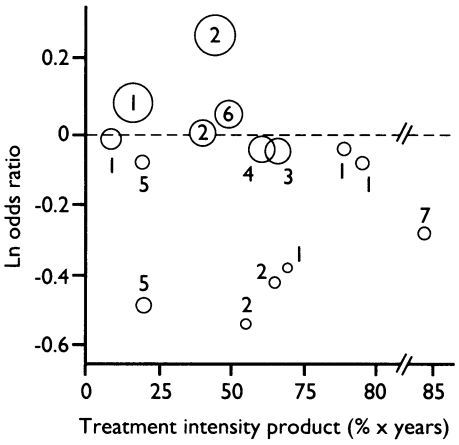
## Lower cholesterol of doubtful benefit to anyone

EDITOR,—In their meta-analysis of trials of cholesterol lowering alone as an intervention George Davey Smith and colleagues show that total mortality is unchanged or may even increase after such treatment except in a tiny minority of people at very high risk of coronary death.[1] As total mortality is the only unbiased outcome and the only one of interest to most people this finding has considerable interest, not least because the authors' prudent conclusion seems overoptimistic.

That people at high risk may profit from cholesterol lowering was concluded on the basis of the negative correlation found between coronary mortality in the control groups of the trials and odds ratios for total mortality. This correlation was mainly due to a few trials of questionable relevance. In the trial on the top of the risk roll, intervention included weight reduction and antioxidant and mineral supplementation; and numbers four and five on the list were small trials with only a few deaths and thus with highly unreliable odds ratios. Even the other high risk trials were small except the DART (diet and reinfarction) trial, but here total mortality was not affected.

The weaknesses of small trials with rare events are not balanced by use of weighted regression; if situated at the extreme end of the diagram even small trials may have a disproportionate influence on the regression equation.

The effect of cholesterol lowering is also questioned because of the lack of a dose-response relation between individual people and between trials and because long trials are no better than short ones.[2] To see if a combination of these two factors—for example, degree and duration of cholesterol lowering—might predict the outcome better I have correlated odds ratios for total mortality with a "treatment intensity product" taking into consideration both factors and using the data from the trials reviewed by Davey Smith and colleagues (figure).




*Odds ratio for total mortality against treatment intensity product (percentage net cholesterol lowering×years of treatment) in cholestrol lowering trials. The diameter of the symbols is given by $\sqrt{n/\pi}$, where n is the number of participants in the trial. To avoid bias due to the small size of trials and few events, only trials with a confidence interval narrower than 1·0 were included. The incomplete branches of the coronary drug project are not included as they did not give the final cholesterol values.*

*Interventions: 1=Diet. 2=Clofibrate. 3=Cholestyramine. 4=Nicotinic acid. 5=Colestipol. 6=Gemfibrozil. 7=Ileal bypass.*

It is unlikely that cholesterol lowering influenced the outcome of these trials because the figure shows a lack of a dose-response relation; the highly variable mortality in the four clofibrate trials is especially striking. The result is not unexpected because the list of observations in conflict with the diet-heart idea is almost endless.[3-5]

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# Measuring patients' views of their health

## SF 36 misses the mark

EDITOR,—Both of the papers on the short form 36 (SF 36) health survey questionnaire assume that the suitability of a questionnaire for assessing outcomes depends largely on its psychometric properties,[1 2] but this is far from the case.

The first issue to be considered is the actual content of the questionnaire. Consider two examples from the SF 36: "Does your health limit you in your ability to do vigorous activities, such as sports, running, lifting heavy objects?" and "Does your health limit you in your ability to walk a mile?" A vast majority of the British population do not engage in vigorous activities and are thus unable to answer the first question; if they do the response will have no meaning. Similarly, some people would not attempt to walk a mile even in the best of health for reasons of safety, climate, or indolence and others, such as those living on peripheral housing estates, may be forced to walk a mile even in the worst of health.

It is also important to ask "the outcome of what?" The prime rule of evaluation is to choose the correct tool for the task. Thus the content of the questionnaire should be appropriate for the intervention. To assume that one instrument would be suitable for assessing, say, improved antenatal care, health promotion initiatives, exercise programmes, hip replacement operations, and antihypertensive treatment is misguided.

We are told that the SF 36 measures "health perceptions." Comparisons are made with the Nottingham health profile. The Nottingham health profile, however, was developed specifically to reflect matters of concern to patients; its content was drawn from lay people and is expressed in lay terms. The SF 36 was developed by a group of American social scientists working for a health insurance survey and was designed to reflect issues of concern to policy makers. Each will thus provide a different viewpoint.

The SF 36 does not contain questions referring to sleep. This is a serious omission as changes in the pattern and quality of sleep are commonly associated with ill health.

Finally, the content of the SF 36, like that of the Nottingham health profile, was generated over 20 years ago. Questionnaires age: both the items and the language may have less relevance with time. Rather than carry out expensive renovation on tired tools, researchers should develop new and better instruments carefully targeted at requirements.

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## Reliability of SF 36 remains uncertain

EDITOR,—Two recent papers contribute to our understanding of the properties of the short form 36 (SF 36) health survey questionnaire.[1 2] Even though the data obtained from the population of Oxford[2] agree in most respects with results reported for Sheffield,[3] however, care is needed before these are adopted as norms and applied nationally to populations that may differ considerably from that of Oxford.

Andrew M Garratt and colleagues conclude that