The Health Variations Programme is a research programme focused on the social determinants of health inequalities. It was funded by the Economic and Social Research Council from 1996 to 2001. Its aims were to undertake multi-disciplinary social science research to advance the understanding of the social processes that underlie and mediate socio-economic inequalities in health, and to advance the methodology of health inequalities research.

Having been chairman of an MRC board, I am aware of what you might call competitive pressure for grants, and the years of battle at Board and on the Council have left me scarred. This has lead to an unreasonable caution (no—those are weasel words—a suspicion) of work where endpoints or outcome measures are difficult to define. The kind of work supported in this initiative is difficult to constrain in these terms but rather, lends itself to the discovery of things that are hard to quantify and hard to deal with in the remedial sense. They are, however, clearly important to the community. In looking at a number of reports from the initiative to see how the new biology might help to resolve some of the difficulties of research in this area, it was interesting to see a good many methodological studies designed to resolve some of the difficulties outlined above, but one study in particular is compelling since it defines, for me, another set of problems.

Under the title ‘The role of perceptions of family history in persisting inequalities in health and lifestyle’, Watt has looked at coronary heart disease (CHD). People’s ideas about their ‘family histories’ of heart disease and health-related behaviours were investigated using in-depth interviews with 61 men and women from a range of social circumstances, all in their forties and living in the West of Scotland. Interviews covered a wide range of areas, including beliefs about heart disease, discussions of whether illnesses or weaknesses ‘ran’ in the family, and discussion about inheritance.

Now, we are all aware that CHD is the leading cause of death in the UK, and that while death rates from CHD are declining, sharp class differences in mortality remain. The reasons for the slower decline in lower socio-economic groups are poorly understood (there was no discussion in the reports I have seen of the role of fetal nutrition). Differences in the lifestyle factors associated with CHD, including diet, cigarette smoking (class differences in CHD and smoking are increasing) and pattern of exercise, are all known to be important, to varying degree.

The study confirms that some people see themselves as definitely ‘having’ or ‘not having’ a family history of heart problems. Genes, or heredity, were mentioned spontaneously as a cause of heart problems by more than two-thirds of the people in this study, and almost all agreed that heredity was an important factor when asked specifically about it. However, many drew a distinction between notions of ‘family risk’ for their family as a whole and for themselves personally. They thought that they differed in crucial ways from affected family members as they did not ‘take after’ affected family members in for example, appearance, build, or health-related behaviours. This meant that they did not necessarily think they should be particularly careful about smoking, say. Some thought there was little point in taking care if they were at increased risk anyway. Two other factors were important: the perception of heart disease as a ‘good way to go’, preferable to a painful and lingering death, typically from cancer; and a conviction that the burden imposed by family history, childhood circumstances, work experiences, history of exposure to smoking and past diet left a legacy that could not be undone by making positive lifestyle changes.

The authors conclude that the lack of certainty in predicting coronary events at an individual level acts as a barrier to behaviour change, and that lay and medical views about which factors determine whether someone is at heightened risk of heart disease because of a family history overlap, but do not fully coincide.

Well, of course. At the lowest level, people simply do not take their doctor’s advice or their medicine; they will believe what they like. But more seriously, in the discussion of risk in these columns we have considered how unlikely it is to
get proper information-based risk/benefit analysis (RBA) done by unselected populations. A number of papers show (for example, reference 1) how information can be unhelpful to the non-scientific. In this context, some analyses suggest that if you want RBA done properly you have to define the non-scientific as those without a post-graduate degree in a science-based subject. Ebrahim’s important analysis shows that even those more at a relative risk than would be defined by a family history alone are resistant to advice.

But there is a much more evident problem when advising individuals—it seems to me that there is a great confusion about what is good for populations rather than individuals in determining health advice. Defined population risk factors are difficult to insert into personal risk/benefit analyses. It is not the case that a low fat diet will confer benefits on all, nor are there direct relationships for the individual between exercise and heart disease. We are all sick of hearing about how ‘Grandpa smoked 60 cigarettes a day all his life and lived to two days short of his 88th birthday’ (incidentally, as a former managing editor of a medical journal, I should make it clear to you all that these accounts used to be called ‘Case Reports’). Nevertheless, no one supposes that the fact that ‘only’ 1 in 5–6 regular smokers will get lung cancer is a good piece of news, however smokers view it.

Now, the authors believe that it is important for doctors ‘to establish the extent to which they and their patients share a common understanding of the patient’s familial risk’. And that ‘research on lay beliefs about inheritance will be increasingly relevant for health policy makers and practitioners, with developments in genetic testing for multifactorial diseases’. Up to a point, Lord Copper. What you need if you are advising an individual is data that define their risk more precisely than a population study can—a good genetic history for the family is not as good as a measurement of a biomarker for the individual. It may be that this will be a genetic screen—they are getting to be fast and cheap—or the use of a presently recordable datum (fibrinogen? arterial compliance?).

Most lay descriptions of heart disease in this work described fatal heart attacks, with graphic accounts that emphasized the suddenness and quickness of death. Very few accounts referred to the pain, disability or restrictions of living with heart disease. All in clinical practise will be aware of the large group of people affected in this way, but as a pathologist I am aware that 25% of cases of CHD present in the post-mortem room and that the perception of a rapid death in those with this disease is not an unreasonable one. These two differing viewpoints are likely to modify attitudes to risk; any system of advice must be capable of giving better information than we appear to have at present if it is to command acceptance.

One further point. The authors consider that ‘Our research has implications for health promotion experts as it highlights some specific ways in which coronary advice can be discounted or undermined’. Discounted yes, but undermined? When we have had ‘Go to work on an egg’ and ‘Drink a pint of milk a day’ within living memory, the possibility that advice may change has induced a healthy scepticism in the public mind about food-related advice. The failure of anti-oxidant studies, despite a basic science back-up, and of fibre-based dietary modifications of the adenoma/carcinoma sequence in the colon, should induce caution. To be Draconian, we need much more convincing data sets.

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References

