Commentary: Occupational therapy or the major challenge?

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At a meeting in Kuopio in Finland in the 1980s, at the welcome dinner, our host stood up to welcome the delegates. Being in the centre of the room, at any one time, he had his back turned to about half the room. Despite this, he impressed by somehow talking to both sides at the same time. This was a trick, he explained, that Finns learnt from being sandwiched between two opposing interests: Russia and Sweden. This is a useful skill explained, that Finns learnt from being sandwiched between two opposing interests: Russia and Sweden. This is a useful skill to primary prevention. Facing one way, they encounter the mainstream of clinical medicine, suspicious of prevention as an approach and doubtful of its efficacy; and biomedical research concerned with fundamental disease mechanisms. When fighting these battles my sympathies are with the risk factor brigade. On the other side, there has been a variety of positions, saying that there is much to discover about causes of CHD that would lead to better prospects for prevention than trying to cajole individuals into changing their diet, smoking, and sedentary lifestyle. Among these latter voices have been those arguing, myself included, that the causes of variation in population rates of occurrence of CHD, are social and economic. A focus on individual risk factors is misplaced.
Taking the clinical side first, it is striking what a change the statins made to the scepticism of cardiology as a whole towards plasma cholesterol. As long as the relation between plasma cholesterol and CHD was to be used as an argument for changing the fat content of the diet, there were many within cardiology who doubted the quality of the evidence. It was only epidemiological, and that could prove nothing. Come the 4S trial, followed quickly by the WOSCOPS, which showed that lowering cholesterol with statin drugs lowered CHD risk, and suddenly the cardiologists had ‘proof’. (The fact that the trial data agreed with the epidemiological data probably changed few minds about epidemiological evidence.) The cardiologists took over cholesterol and made it part of clinical medicine. Screen individual patients and treat those at high risk. Prevention became part, not of public health, but of clinical practice.

Beaglehole and Magnus continue this difficult trick of having to deal with two sides at the same time. They argue strongly that control of the CHD epidemic must not rely on ever finer approaches to assessing individual risk, and treating individuals. The underlying causes are economic, social, and cultural, so must be the remedy. This does not put them in the other camp that says we need more research on causes because our understanding is limited. More research, yes, say Beaglehole and Magnus, but on how to change population levels of the risk factors of which we already have knowledge: diet, smoking, blood pressure levels, and sedentary lifestyle.

Beaglehole and Magnus argue, compellingly, that we know a great deal about the causes of CHD. This is the first prong of a three-pronged argument. First, we know the major causes of CHD and, in high risk populations, the majority of the population is at risk. Second, they identify six strands of research on new risk factors for CHD, and are not impressed. Although the title of their paper has a ‘?’, one has the impression that their conclusion is that such research is merely ‘occupational therapy for epidemiologists!’—no ‘?’ about it. The link between the first and second parts of their argument is a dismissal of those who justify their search for new risk factors by asserting that the established risk factors account for only half the variation in CHD occurrence. Not only is this the myth of the 50%, say Beaglehole and Magnus, but the established risk factors of diet, blood pressure, smoking, and lack of physical activity account for 75%. Third, the real challenge is not to find new risk factors implicated in the causal process, but to address the gap between the knowledge we have currently and our ability to effect change in population risks of CHD.

There is much in their argument with which I agree. We do know a great deal. The risk factors listed above are likely to be causal. The underlying causes of CHD are social, economic and cultural. We should not rely on treating individuals, one at a time, to prevent the mass occurrence of CHD, especially in middle income, and poorer countries. In these, CHD has become of major importance but the prospects of controlling it with expensive drugs are available only to the privileged few. Where I take issue with Beaglehole and Magnus is that the social, cultural, and economic causes operate principally through the known risk factors that they list.

I should declare an interest here. I have spent the last 30 years doing what Beaglehole and Magnus describe as occupational therapy for epidemiologists. There is, perhaps, little surprise that I think such research has a value in addition to keeping my colleagues and me off the streets, or away from the analyst’s couch.

Let us look at the ‘myth of the 50%’ in relation to one of several problems that have kept me occupied: the inverse social gradient in the occurrence of CHD in rich countries. In the Whitehall studies of British civil servants, the lower the employment grade the higher the risk of CHD. As Beaglehole and Magnus note, we used the first Whitehall study to define a low risk group as having a cholesterol level at the low end of the distribution, a blood pressure at the low end of the distribution and to be never smokers. We calculated that if the whole population had the risk factor distribution of this low risk group, CHD mortality would be lower by two-thirds. As the risk factors predict CHD within groups defined by their socioeconomic level, it is likely that all socioeconomic groups would benefit. So far, I agree with them. But, this low risk group is only about 5% of the population. To move the average level of risk to that of the best-off 5% may be a worthy goal, but it will not be achievable in the short term. There is a continuing need for research that asks why, for a given level of risk factors, there continue to be substantial individual and group differences in CHD rates.

We turn then to the question of how much of the social gradient can be explained by the current level of risk factors that we find in Britain. Our answer from Whitehall and Whitehall II has been less than one-third. Commentators have wondered if this answer is dependent on our multivariate models or measurement imprecision. We can answer the question simply.

In Whitehall II, there is no social gradient in plasma total cholesterol. This can not be part of the explanation for the social gradient in CHD.

Similarly, there is a very weak gradient in blood pressure.

The only major risk factor that contributes substantially to the gradient is smoking. In Whitehall II smoking prevalence was less than 20%. We find the same social gradient in CHD in non-smokers that we do in smokers.

Our calculation from Whitehall II is that smoking and blood pressure account for 26% of the gradient in CHD incidence in men and 22% in women. Adding in physical activity increases our explanatory power.

Without consideration of some of the newer risk factors dismissed by Beaglehole and Magnus, we get nowhere near the figure of 75%. Social and economic factors are important but not only because of their effects on conventional risk factors. We, particularly, have been interested in psychosocial factors. Others have been interested in factors operating earlier in the life course. The fact that ‘causality is still unproven’ is, in my view, not a reason to stop research on these newer risk factors, but to continue it. If the amount of research devoted to the ‘established’ risk factors had been devoted to some of the newer approaches, we would be in a better position to ‘estimate their overall contribution to population CHD levels’.

What does it mean to say that 75% of occurrence of new cases is ‘explained’ by the major risk factors? Infection with the tubercle bacillus ‘explains’ 100% of the occurrence of new cases of tuberculosis (TB), in that you cannot, by definition have TB without such infection. It does not explain why some infected people get clinical disease and others not, nor particularly, why TB has been a disease of the least well off in society. With infectious disease, the 100% figure is useful if, for example,
a vaccine is available to prevent infection. In its absence, the importance of the other factors determining disease rises.

To return to the CHD example, if it were possible to move the levels of diet, activity, smoking, and blood pressure of the 95% of the population designated as ‘high risk’ down to the level of the lowest risk 5%, there would be great benefit in terms of reduction of CHD. For Beaglehole and Magnus, the political challenge of how to achieve that should exercise the time and talents of all of us. Wearing one hat, epidemiologists may well rise to that challenge. Wearing another, there is the continued challenge of asking why, among people more or less equally exposed, there remains such marked differences in the rate of occurrence of CHD. I would not characterize the first as the problem to be solved and the rest as occupational therapy.

References

Commentary: The epidemiology of self-deprecation
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When Mervyn Susser wrote his 1989 commentary ‘Epidemiology today: A thought-tormented world’\(^1\) he probably did not realize how prophetic his words would be. During the ensuing decade, epidemiology journals were inundated by self-critiques and soul-searching commentaries. For Susser, epidemiology had abandoned its substantive-oriented nature to become a technique-oriented discipline, more concerned with its analytical methods than with its primary goals of guiding disease prevention and public health. Further elaborations of these criticisms urged a renewed emphasis on the population and societal perspectives of epidemiology.\(^2–5\)

From an entirely different perspective, however, there were those who claimed that epidemiology was already too involved with public health.\(^6–9\) According to these critics, epidemiologists had become data torturers with an agenda of ‘social agitation’ and constantly made exaggerated recommendations aimed at promoting costly and invasive public policy interventions. For these authors, epidemiology is not a real science but a mere collection of inductive tools useful to the astute biologist (the ‘real’ scientist) to make predictions about population health.\(^7\)

And then there was Tauber’s 1995 critique of current epidemiologic practice in (of all places!) the journal Science.\(^10\) This journalistic article was largely based on quotes from leaders in the field criticizing the ‘sin’ of overinterpreting small effects found in observational studies, particularly when this information made it to the mass media and the general public. Commentaries and debates then flooded epidemiology journals and seminar series in academic institutions: Who are we?, where are we coming from?, where are we going?, … are we real scientists?

Now, it appears, it is the turn of the subspecialties. According to Beaglehole and Magnus’ provocative commentary in this issue of the *International Journal of Epidemiology*,\(^11\) the claim that more research on emerging coronary heart disease (CHD) risk factors is needed, is just the epidemiologists’ own ‘occupational therapy’. For these authors, we already know all that there is to know about the determinants of CHD: high serum cholesterol, high blood pressure, cigarette smoking, and physical inactivity explain 75% or more of the CHD incidence,\(^12\) and not just 50% as ‘conveniently’ quoted by epidemiologists trying to justify their occupation and research portfolios. For Beaglehole and