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

FJ Nieto

When Mervyn Susser wrote his 1989 commentary ‘Epidemiology today: A thought-tormented world’ 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.

From an entirely different perspective, however, there were those who claimed that epidemiology was already too involved with public health. 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.

And then there was Tauber’s 1995 critique of current epidemiology practice in (of all places!) the journal Science. 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, 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, and not just 50% as ‘conveniently’ quoted by epidemiologists trying to justify their occupation and research portfolios. For Beaglehole and
Magnus, the proponents of the old-fashioned ‘risk factor approach’ are only ‘distracting attention and resources’ that would be better used for implementing public health interventions and, if anything, on research on ‘upstream’ determinants.

While it is hard to disagree with the authors’ argument that translating what we currently know about CHD risk factors into effective prevention policies at the population level should be a high priority, I take issue with the assertion that there is nothing new to be learned with regard to CHD risk factors—that I found presumptuous and blatantly unscientific.

The data that Beaglehole and Magnus use to support their attributable risk estimates are largely derived from studies of adult individuals in Western societies, with CHD clinical events or CHD deaths as outcomes. But atherosclerosis is practically universal in adult individuals in our society and thus these studies can only inform us of the predictors and correlates of the late phases of the disease natural history. There is absolutely no proof that a causative model that explains 75% of the clinical events (even assuming that this is a correct estimate) would also explain the same amount of variance in the incidence (initiation) of the atherosclerotic process itself, the true target for primary prevention.

For Beaglehole and Magnus, research on ‘emerging’ risk factors (thrombotic/biochemical factors, infectious agents, early life exposures, multiple genes, oestrogen deficiency, psychosocial environment) is irrelevant because these factors fail to pass the ‘public health test’ of causality. The origins and rationale for this new causality paradigm are unclear. Moreover, even though up to five criteria are listed (including risk factor prevalence and ‘public health test’ of causality), the origins and rationale for this new causality paradigm are unclear. The effect of certain molecules may go in different pathways depending on the homeostatic balance at any given moment. Thus, a simplistic ‘toxicological-type’ analytical approach of measuring serum levels of an isolated marker and thus these chemicals in isolation might not be a meaningful approach to address an extremely complex pathophysiological question? Cytokines and other molecules interact with each other and with cellular components at the blood-endothelial interface in extremely complex ways which we are only beginning to understand. The effect of certain molecules may go in different directions depending on the homeostatic balance at any given moment. Thus, a simplistic ‘toxicological-type’ analytical approach of measuring serum levels of an isolated marker and expecting that their (dose-response) association with a certain outcome will establish or rule out the importance of a given chemical or mechanisms might be utterly irrelevant in this case.

I would argue that epidemiology should keep (or rather, recover) its broad scientific basis ranging from molecular biology to social sciences. This multidisciplinary approach to the understanding of disease causation and prevention is, to me, the essence of epidemiology. Ignoring everything outside the realm of so-called ‘epidemiologic studies’ results in a narrow understanding of the scope of the discipline; it results in bad epidemiology. Ultimately, a ‘public health test’ of causality that ignores the extraordinary complexity of disease pathogenesis...
and ignores ‘non-epidemiological’ evidence is a step backwards and will not help advance our understanding of disease aetiology and its determinants. In my opinion, this approach is no less reductionistic than a strict biomedical understanding of disease processes so severely criticized by some.5

Contrary to Beaglehole and Magnus’ belief, many, including myself, still think that some of the lines of research that they consider worthless could reveal important clues for the understanding of atherogenesis that could eventually help in developing new primary and secondary prevention strategies. These would have important implications for the development of both population-based and high-risk preventive strategies, which, as Geoffrey Rose emphasized, are to be considered complementary rather than mutually exclusive strategies.30

It is interesting to note that, in contrast to their lack of interest in the role of the six groups of emergent risk factors discussed in their commentary, Beaglehole and Magnus conclude that there is a need for ‘improving the understanding of the social and economic determinants of the major risk factors and overall population health status’.11 It seems to me that the argument against risk factor epidemiology could also be extended to research on known socioeconomic determinants of known risk factors. Don’t we also know enough about poverty being a bad thing? Being poor is clearly bad for your health (among other things). Wouldn’t this type of argument support those who question whether we really need to do more research on social and economic determinants of risk factors and disease?31 Why don’t we spend that money and time on acting against poverty?

For Beaglehole and Magnus, ‘research on possible risk factors and unresolved issues is at best only moderately promising’.11 The problem is that if one applies the same high standards of evidence to studies of the efficacy of cardiovascular disease prevention interventions, the results will be similarly weak if not weaker.32–34 Are we then surprised when someone claims that no more CHD prevention research should be conducted?35

The authors blame ‘the public health research community’ for their ‘reluctance to act fully on the available evidence on the causes of CHD’.11 Tobacco control and the reduction of salt content in manufactured foods are cited as examples of successful public health policies. The argument is that we need to do more stuff like this; for example, we need to work toward ‘modifying the environmental determinants of physical inactivity and the resulting obesity’. What is lacking in Beaglehole and Magnus’ commentary, however, is a discussion of concrete and specific ways for addressing the cultural and political determinants underlying these upstream conditions. If we continue their line of reasoning and arguments against ‘emergent’ risk factor epidemiology, we might also conclude, for example, that we already know enough about how to prevent sedentary lifestyles and obesity. Don’t we have enough evidence already regarding the influence on sedentarism and obesity prevalence of the public transportation infrastructure of cities, the patterns of TV-viewing in children, and accessibility to recreational facilities? The question is, then, what can we do about these problems?

Thus, perhaps the next line of the argument would be for ‘public health researchers’ to quit applying for grants from the National Heart Lung and Blood Institute and become public policy activists or lobbyists. Should then cardiovascular epidemiologists quit their academic positions and devote the rest of their careers to trying to convince legislators to ignore car manufacturers and oil companies’ lobbying efforts and raise gasoline taxes, reduce parking facilities in cities, increase public transportation services, and limit the number of hours of cartoons that a TV channel can offer per day? How do we address the high saturated fat diet in the population? Would Beaglehole and Magnus recommend nutritionists abandon their research projects on lipoprotein metabolism and become advocates for the cause of outlawing the fast-food industry?

Ultimately, the question is what is the responsibility of the public health research community relative to other sectors of society with regard to convincing the government and the public at large of the desirability (and feasibility?) of these efforts?

I am certainly not against epidemiologists becoming public health activists and advocates. Quite the contrary. Moreover, I must recognize that I agree with the premise that there is currently an imbalance in the funding of aetiological and preventive research. However, I believe that this should be discussed in a wider social and political context. Blaming research epidemiologists for doing what they are funded to do after going through a highly competitive scientific review process is simply a rhetorical and not particularly constructive exercise.

I have a hard time thinking of another scientific discipline so prone to self-criticism. No one seems to be questioning the work of entomologists or botanists studying the ecology of rare species in the rain forests, archaeologists investigating lost cultures, or astronomers studying stars billions of light-years away. Epidemiologists for some reason seem intent on questioning their own worthiness. I just wonder how much of the energy spent on self-deprecating efforts would be better used in actually working together with all the other experts involved (including biologists, social scientists and policy advocates) on improving the understanding of the distribution, determinants, and (yes, of course) trying to figure out ways to improve the health of the population. That is, actually doing our jobs.

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References

6 Charlton BG. Should epidemiologists be pragmatists, biostatisticians, or clinical scientists? Epidemiology 1996;7:552–54.
The presence of atherosclerotic lesions in the coronary arteries of young soldiers killed in the Korean War was greeted with some surprise. Certainly, the age of onset of clinically apparent disease, and the emerging findings of epidemiological studies such as Framingham pointed to middle age as the time when coronary heart disease (CHD) was determined and therefore could be prevented. In his review, Beaglehole sets out the arguments for the pre-eminence of the traditional adult risk factors for CHD—high serum cholesterol, high blood pressure, and smoking. Studies of men and women from middle age onwards show that these risk factors are associated with a large proportion of CHD, and that, in many countries, much of the population can be classified as being at high risk.