development, education and employment agencies. It is time to invoke the mantra of an earlier hero of public health (and politics)—Rudolf Virchow—who realised that ‘mass diseases require mass solutions’. A global solidarity between public health scientists and practitioners would help us all to remain focused on the main question—what must we do to improve the population’s health?

References
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Commentary: Reflections on sick individuals and sick populations

James McCormick

Since Geoffrey Rose published his paper there has been virtually no progress in reducing the incidence of disease by population change, with the exception of reduced cigarette smoking. This is not surprising.

In order for a population strategy to be effective two requirements need to be satisfied. The first is good evidence of a causal relationship between factors prevalent in populations and disease. The second, a strategy to alter the prevalence of such factors.

Good evidence of causal relationships must come from the study of individuals. Associations within populations are subject to the ecological fallacy, that is the error of imputing causality to such observations. For example, an association between saturated fat intake and the prevalence of breast cancer does not justify dietary advice to reduce the risk of this disease. In the examples cited by Geoffrey Rose, an association between diet and coronary heart disease and an association between salt intake and hypertension, there is no good evidence of a causal relationship based upon the study of individuals. Dietary manipulation has very small effects upon cholesterol levels and no randomized trial has shown unequivocal evidence that alteration in diet can reduce mortality from coronary heart disease. Similarly even stringent control of salt intake has very limited effects on blood pressure and no trial has demonstrated that such restriction reduces mortality from stroke.

Lack of awareness of the ecological fallacy has led to a plethora of unjustified advice and, it could be argued, to the growth of national hypochondriasis and the numbers of the worried well.

Cholesterol is a good risk marker for coronary heart disease. However, it may be that cholesterol is nothing more than a risk marker and not a cause of myocardial ischaemia. Individual cholesterol levels are largely independent of diet. In addition there is the surprising finding in the 4S Scandinavian Study that the use of statins had the same good effects in those whose cholesterol was 5 mmol/l as those whose cholesterol was 8 mmol/l.1

Geoffrey Rose’s optimism was based upon observations which assumed knowledge which we did not, and do not, have. It is at least arguable that public health strategies, as distinct from advice in the consultation, require something approaching certainty and that the ‘abominable no-men’ are important guardians of the public weal.

The best population strategies for reducing disease are those which involve no input from individuals, for instance, provision of clean water or fluoridation. When the cause of misfortune is manifest, car accidents or such like, the enforcement of law governing speed or the wearing of seat belts may become a justifiable interference with individual liberty.
It is too early to tell what the effect of our knowledge of the human genome will have. There can be no doubt that the environment will still be a major determinant of disease incidence and prevalence but it may be possible to identify differences in personal risk which may benefit individuals. Perhaps the study of those who do not get diseases will become as important as the study of the afflicted.

‘Sick individuals and sick populations’ is still an important paper and Geoffrey Rose's comments upon the failure of case-control and cohort studies to detect necessary agents which are homogenous within a population remain entirely valid. However, altering population risk will, for the most part, remain an unreal aspiration.

References

Commentary: Causes of incidence and causes of cases—a Durkheimian perspective on Rose

S Schwartz and R Diez-Roux

Geoffrey Rose’s seminal 1985 article ‘Sick Individuals and Sick Populations’ and his 1992 book ‘The Strategy of Preventive Medicine’, have made a huge impact on the fields of epidemiology and public health. A casual Social Sciences Citation Index search yielded over 700 citations of this work. The central lesson that has been integrated into the field is that ‘a large number of people at a small risk may give rise to more cases of disease than the small number who are at high risk’.1(p.37) This insight, which has profound implications for intervention and prevention strategies, has been incorporated into research contexts through an understanding of the difference between measures of absolute and relative risk. But there is another aspect to Rose’s work that has had a more difficult hearing and that runs counter to mainstream epidemiological approaches solidified under the risk factor paradigm. This is Rose’s contention that the causes of cases of disease and the causes of disease incidence may be different and require different types of research strategies. In particular he argues that ‘to find the determinants of prevalence and incidence rates, we need to study characteristics of populations, not characteristics of individuals’.1(p.34) This issue has become a central theme in the ‘epidemiology wars’2 with factions sympathetic to Rose’s position arguing that epidemiology has lost its public health relevance because of a myopic concentration on individual-level risk factors.3

Rose’s contention that the key to understanding incidence and prevalence lies in ‘characteristics of populations and not individuals’ is, as Charlton4 notes, ‘a startling claim’. (p.607) After all, disease ultimately resides in the individual body and is defined at the individual level. Individual bodies get diseased and become cases. Population incidence itself is merely the averaging of these individual cases across the population. How is it possible, then, that an understanding of the causes of incidence could be different from an understanding of the causes of cases and, more generally, how can the characteristics of a population enlighten us about disease aetiology?

In order to understand Rose’s claim it is essential to examine two key underlying concepts in Rose’s writings: the concept of ‘cause’ and the relationship between wholes and parts. In what follows we will discuss these concepts and then, based on this foundation, indicate five situations where the causes of cases and incidence may deserve distinct treatment.

Two central concepts—cause and the relationship between wholes and parts

Rose’s notion of cause

The distinction between Rose’s view of causation and that of his critics lies not in the types of factors that can be defined as ‘causes’, but rather in the criteria used to create a hierarchy among the participants in the causal process. For Rose’s critics such as Charlton5,6 the most important causes, the ones afforded primacy, are those that define the pathophysiology of a disease. These are the causes that come closest to meeting the standard of Koch’s postulates in that they are specific to the disease at hand and found universally, or nearly so, among those with the disease of interest. The priority given to these types of causes is due to the greater scientific certainty and universality with which causal attributions can be made. These causes can be more easily examined with clinical data, manipulated in a laboratory context and are more easily identified in within-population comparisons than more distal, population or social causes. These types of causes, therefore, are given higher