Does epidemiology have a place in general practice?

SCOTT HENDERSON MD, DSc, FRACP, FRCP, FRANZCP

The uniqueness of all people can be balanced by the epidemiological perspective. Epidemiology searches for patterns occurring in the whole population, giving clues to causes, risk factors, the course of diseases, their likely outcomes and the impact of our treatments. GPs use such information every working day.

Most of general practice is about individuals. But when they are considered collectively, looking for general patterns, this is epidemiology. The most succinct definition of epidemiology is 'the mass aspects of disease'.[1] Far from being a morass of unintelligible statistics (as many doctors think) or acute outbreaks of infectious diseases (as the public often think), epidemiology sparkles with appeal and opportunity.[2-4] This is mainly because what we encounter individually in our clinical practice can only be a tiny part of the whole picture of disease, the causes, course and outcome. Epidemiology contributes to understanding the occurrence of disease in all humankind, and the laws that determine what is happening.

This article gives an account of what epidemiology does and how epidemiologists go about their work. Here are some of the best known successes in the past:

- cholera and the domestic water supply on Broad Street, London – John Snow recognising that the incidence was very different between the two sides of the street, and that these were serviced by different water supply companies, one having water contaminated by sewers
- cigarette smoking and lung cancer – Sir Richard Doll's findings on British doctors
- coronary heart disease found to be much higher in London Transport bus drivers than in the more active conductors (no wonder that J.N. Morris, who did this work, still swims most days, and is now over 90 years of age!)
- the contraceptive pill and the risk of breast cancer
- diet and cardiovascular disease or cancer.

Professor Henderson is Visiting Fellow, Centre for Mental Health Research, The Australian National University, Canberra; and Clinical Adviser in Mental Health, Commonwealth Department of Health & Aged Care, Canberra, ACT.

This image is unavailable due to copyright restrictions

PIERRE-YVES GOAVEC/IMAGEBANK

How possible risk factors are assessed

Suppose we want to know if sexual abuse in childhood makes people vulnerable to depression in adult life. For such a question, or any other putative link between a disorder and a previous environmental exposure, one strategy is to complete four entries in a 2 by 2 table.

The 2 by 2 table is a fascinating tool, but it is also deceptively simple (see the box on page 149). In a whole population (say, a large country town), we need to find:

- the number of adults who develop a depressive disorder in the course of a year and who also report having been sexually abused as a child (cell A)
- the number who are not depressed but who report having been abused (cell B)
- the number depressed but not abused (cell C)
- the number not depressed and not abused (cell D).

Please note that it is not good enough to take only the patients who reach you in your surgery. There are certain to be

many people who are equally depressed but who do not seek professional help, irrespective of having been abused or not abused in childhood. Of particular importance to the analysis is cell B: people who were abused but are not depressed. Only when the numbers for all four cells are found can we learn if childhood abuse is associated with (although not necessarily the cause of) depression in adulthood.

Paul Mullen and his colleagues have gone into this issue very carefully in Otago, New Zealand, working on a large community sample of women. They found that childhood sexual abuse is indeed related to poorer adult mental health, but also that sexual abuse is very commonly associated with many other nasty experiences.[5]

Notice, in passing, the industry that has grown among some 'therapists' who, on being consulted by a person in cell C (depressed but not abused), embark on an unrelenting effort to help the person 'recover' memories of what the therapist believes must have taken place.

How epidemiologists think

How do epidemiologists go about their work? The way in which candidate risk factors come to be selected for study is itself very interesting. After all, if it were not for some guiding principles, it would be like looking for one needle in a large haystack. There are three paths:

- the inspired hypothesis
- a coarse observation
- theory-driven enquiry.

The inspired hypothesis

A sharp-eyed clinician develops the idea that some factor is present more often than by chance in a certain disorder, and that the factor may contribute to the onset of the disorder. This is exactly what happened to the ophthalmologist Sir Norman Gregg in Sydney when he noticed that many disabled children had mothers who had had rubella during their pregnancy. The association was later confirmed and shown to be causal. So here a hypothesis arose in the course of clinical work, was taken out of the consulting room or clinic, and was tested on a larger scale by epidemiological methods. The causal link was confirmed, and finally the knowledge was applied in prevention.

A coarse observation

A second pathway starts with a coarse observation of a link between a disorder and, say, some demographic variable. In the aetiology of schizophrenia, a slight excess in winter birthdates was noticed in large series of patients. This led to work suggesting a possible excess of schizophrenia in the offspring of women who had had influenza during the middle trimester of their pregnancy. Next was work on the 1946 birth cohort in Britain that yielded the important finding that persons who later developed schizophrenia had been recorded as children to have had more speech and educational problems, more social anxiety and a preference for solitary play. An excellent review of this field is by Jablensky.[6]

Theory-driven enquiry

In epidemiological research on Alzheimer's disease, an inverse association was noticed between Alzheimer's disease and

Editor's comment

Advances in medical practice do not all come from the laboratory, nor do they all require the use of complex statistical analysis. In the past, watchful and thoughtful clinicians have made observations that have led to realisations that in turn saved countless lives.

The problem is to distinguish fact from fantasy, and here some epidemiological knowledge can be decisive. In this article, Professor Henderson explains why this is so, and gives examples of how ignoring these principles can lead to much wasted effort and – worse – to the proliferation of nonsense.

Dr John Ellard
Editor

Example of a 2 by 2 table

This 2 by 2 table could be used on a whole population sample (e.g. a large country town) to look for a possible link between sexual abuse in childhood and depression in later life.

Sexual abuse in childhood?	Depressed as an adult? Yes	Depressed as an adult? No
Yes	A	B
No	C	D

A: Depressed as an adult and sexually abused in childhood.
B: Not depressed, but abused.
C: Depressed, but not abused.
D: Not depressed and not abused.

rheumatoid arthritis. This subsequently led to studies of people who'd had long term use of anti-inflammatory drugs. The conclusion is that these drugs and oestrogen probably reduce the chances of developing Alzheimer's disease by slowing the deposition of beta amyloid in the brain.

The new opportunities from genetics

Many diseases that are of major relevance to the whole population are said to be genetically complex. This means they are not in the OGOD (one gene, one disorder) category, such as Huntington's disease, but are influenced by many genes, each of small effect. These genes are collectively called quantitative trait loci (QTLs), and it is these that partly determine attributes that are distributed normally in the population, such as height, intelligence or blood pressure. But quantitative trait loci can also confer vulnerability to a range of common diseases.

Epidemiology complements our personal experience in clinical practice.

With the imminent cascade of information from the human genome project, it is likely that many quantitative trait loci will be identified in the next decade. This will mean that an estimate can be made from a person's genome of what the risk will be for a particular disorder under specific environmental conditions. The quantitative trait loci are not genes for this or that disease. Instead, when acting in concert and under certain environmental conditions, they increase or decrease the chances of a disorder developing. Epidemiology will then truly be able to study nature and nurture, and the interaction of these, for all manner of pathologies.

A contentious issue

When it comes to applying epidemiological knowledge to prevention, a dispute has emerged. The traditional medical approach is to focus on those individuals who have conspicuous risk factors for, say, cardiovascular disease or suicide. This has intuitive appeal, but unfortunately it does little to improve health across the whole population, simply because of the arithmetic: most cases of cardiovascular disease do not occur in high risk individuals, and most suicides do not occur in those most at risk.

So what is much more effective is to alter the frequency of the risk factors in the whole population. This is the strategy advocated by Geoffrey Rose,[7] who compared focusing on high risk individuals to 'an attempt to control icebergs by sending warships to shoot off their visible portions'.

Take injury from motor vehicle accidents. This has been reduced by introducing seatbelts, which everyone wears, whether they are risky drivers or not. Similarly, the drop in cardiovascular diseases in Australia is probably due to general improvement in diet and lifestyle in our whole community, and not to action focused on only high risk people.

Some of us are now seeking equivalent changes to prevent suicide, depression and other common mental disorders. A strategy for preventing suicide that focuses on only high risk groups is bound to fail.[8]

Bringing epidemiology into general practice

Epidemiology has rather neglected general practice as a setting for its work. In many areas of medicine, the enquiries have been done in teaching hospitals or, going to the other extreme, in the general population. However, there is an abundance of very useful work that could be done in general practice on aetiology, factors influencing the course of disorders, and the effect of our treatments.[9] Many general practitioners would like to have some involvement in such research, especially if they could do so without financial disadvantage. A recent Commonwealth initiative, supported by the NHMRC, is aimed at just this. Some Divisions are now very likely to be able to make a real contribution to general practice-based research directed at some fundamental questions, particularly on the course and outcome of disorders. Such research is already taking place on the outcome of depression and anxiety disorders as these present in general practice.

Conclusion

Epidemiology has brought a great deal to medicine. It is a natural complement to the personal experience of each of us in our day-to-day clinical practice. There are some rich opportunities for it to be used much more in general practice. MT

References

1. Greenwood FRS. Epidemiology: historical and experimental. Baltimore: Johns Hopkins Press, 1931.
2. Morris JN. Uses of epidemiology. Baltimore: Williams & Wilkins, 1964.
3. MacMahon B, Pugh TF. Epidemiology: principles and methods. Boston: Little, 1970.
4. Susser M. Causal thinking in the health sciences. New York: Oxford University Press, 1973.
5. Mullen PE, Martin JL, Anderson JC, et al. Childhood sexual abuse and mental health in adult life. Br J Psychiatry 1993; 163: 721-732.
6. Jablensky A. Schizophrenia: recent epidemiological issues. Epidemiol Rev 1995; 17: 10-20.
7. Rose G. The strategy of preventive medicine. Oxford: Oxford University Press, 1992.
8. Rosenman SJ. Preventing suicide: what will work and what will not. Med J Aust 1998; 169: 100-102.
9. Goldberg D. Epidemiology of mental disorders in primary care settings. Epidemiol Rev 1995; 17: 182-190.