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Editorial

Epidemiology of Dementia: The Current State

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Sizeable expectations have been placed on epidemiology, that it may help in the growing public health problem of dementia. These expectations have been further fuelled by the emergence of scientific opportunities not commonly encountered in chronic disease epidemiology. As readers of this journal will be well aware, there has been a remarkable burgeoning of research activity by clinicians, epidemiologists and neuroscientists who, in an unprecedented manner, are now focusing their attention on dementia, and on Alzheimer's disease (AD) in particular. What has so far been learned, and what use is that knowledge? The four papers assembled here are evidence that epidemiological research has been making some welcome advances in both its methods and its findings. In this editorial, an attempt is made to look at the overall progress made, to point to areas that need strengthening and to identify some promising research questions.

There is one basic requirement in epidemiological research on dementia: the use of standardized diagnostic criteria in case-finding. This has only recently started to happen. But even standard criteria can be interpreted, or applied, in different ways. The risk of this is reduced by using a standardized instrument, deliberately designed to tap each element in the diagnostic criteria. The scores on each item of this instrument can then be assembled by an algorithm to determine if the person is a case. By the use of three things, standardized diagnostic criteria, an instrument which assesses these, and an algorithm to assemble the observations invariably, we start to be in a position to compare results across studies.

What is known from the surveys conducted to date? The integrative review of 47 prevalence studies by Jorm et al. (1987), while showing the great variability in estimates attributable to the methods used, did point to four general features in the distribution of dementia: the doubling of prevalence rates with every 5 years increase in age; the excess of women over men in the prevalence of AD and a trend towards more vascular dementia in men; the greater prevalence of vascular dementia compared with AD in Japanese and Russian studies; and the greater prevalence of AD in Western Europe. Further information is needed on the possibility of urban-rural or occupational differences, as well as broader international differences, as discussed by Jorm in this issue. It is here that the much more costly incidence studies are particularly desirable.

The evolution of epidemiological research on dementia in North America has been usefully set out by Eastwood and his colleagues. Its pace has recently quickened. The striking features are the paucity of field studies until the last decade; the development of criteria and instruments for case-finding; and the rich opportunities in the United States and Canada for epidemiological research on contrasted populations, including studies of incidence.

Case-control studies have generated most of what we know about risk factors for dementia (Henderson 1988). For AD, age is the one undisputed risk factor. Some other proposed risk factors have not yet been established, but neither have they been conclusively disproven. These include a history of dementia or of Down's syndrome in a first-degree relative, head injury, aluminium exposure, ulnar loops and analgesic abuse. In a large Australian study, Broe et al. (1990) found that a longstanding history of physical inactivity gave an odds ratio of 6.3 and 3.5 respectively for the previous 10 years and prior to 10 years. This finding clearly calls for confirmation. So far, there is no confirmed risk factor which can be modified by intervention or change in lifestyle. In this issue, Cooper has undertaken an examination of the common epidemiological features in AD, Parkinson's disease and motor neuron disease. He argues that environmental causes can be invoked as legitimate candidates for explaining the common sporadic form of AD. But there is currently no robust evidence for such an exposure. This includes the data about aluminium. For case-control studies as a whole, there is now consensus that little useful information will come from further studies of only 100–200 cases. Some risk factors may come to light from examining very large data sets, derived from pooled information. This is now being undertaken through EURODEM and the U.S. National Institute on Aging.

Some Obstacles to Progress

Epidemiological research on dementia is to some extent hampered by dichotomous thinking. The cut-point on a continuum of severity is quite arbitrarily established, but is nevertheless what defines "a case". Furthermore, the continuum is in fact a complex of two correlated dimensions: memory and intellect. The instrument should measure impairment of these in a way which is comparable (see Grayson 1987). That is, it should not be sensitive to mild impairment in, say, memory but to only severe impairment in thinking. Having determined where a person lies on the overall continuum of cognitive impair-

ment, what happens if he or she falls just short of the cut-point, or does not quite meet all the criteria for dementia? In a dichotomous model of dementia, that person is excluded. A more useful procedure is to use continuous measures in addition to counting cases. Such measures of the dependent variable will contain much more information, and are a more adequate representation of the spectrum of cognitive impairment. It is a step in the right direction that both the Draft ICD-10 (World Health Organization 1990) and DSM-III-R (American Psychiatric Association 1987) provide for grading of dementia into mild, moderate and severe states.

A second obstacle is the validity of diagnosis in epidemiological studies of dementia. In hospital settings, where most case-control studies have been conducted, it is thought that experienced clinicians are correct in their diagnosis of AD in about 80% of cases. Under field survey conditions, even with a clinician as the examiner, the rate must be considerably less (Henderson and Jorm 1987).

There is then a further question about validity. Where the diagnosis of dementia, or AD, or vascular dementia is made by any of the current criteria, it has to be asked if the validity of these criteria is itself securely established, and how this should be determined. This applies to criteria such as DSM-III-R, the NINCDS-ADRDA criteria for AD (McKhann et al. 1984), CAMDEX (Roth et al. 1986), AGECAT (Copeland et al. 1988), or the Draft ICD-10 diagnostic criteria for research.

The problem of validity is no less apparent in a third obstacle to field studies: the lack of culturally portable diagnostic criteria, and the instruments which claim to cover these criteria. The Geriatric Mental State Examination (GMS) has now been used in diverse settings, as Copeland and his associates report here. Indeed, a sizeable body of GMS data from community samples in many countries now exists, covering not only dementia and cognitive decline, but also depression and other categories. It seems likely that the GMS can be used in non-literate communities with little experience of industrialized living patterns and education. It is just such communities, as in Africa or Asia, where we would like to have comparable epidemiological data. In 1965, Kessel asked of all psychiatric epidemiology, "Are international comparisons timely?" He concluded they were not, for the very same reasons we currently face with research on dementia. What he proposed instead is also highly applicable: studies within a country, contrasting particular groups, and comparisons between fairly similar communities. An example of the latter is the US-UK Cross-national Diagnostic Project (Copeland et al. 1987) with its finding, yet to be pursued, that the prevalence of dementia was higher in New York than London.

Fourthly, there is a real need for hypotheses arising out of epidemiological findings which could be pursued in the laboratory, and for hypotheses from molecular biology or neuropathology which epidemiologists could investigate. This cross-fertilization is what happened in the kuru story. For research on dementia, the closest we have got is the link between AD and Down's syndrome, and the interest of molecular biologists in the long arm of chromosome 21; or the search by epidemiological methods to explain the presence of aluminium in Alzheimer plaques. Such interaction between disciplines needs to be deliberately sought.

Future efforts in the epidemiology of dementia carry particular promise in the following areas: very large case-control studies; exploration of the features common to AD and other degenerative disorders of the CNS; special populations carrying certain exposures or lifestyles; regional or selected international comparisons; and longitudinal studies of community samples. The authors of the four papers in this issue have provided for us a firm platform of knowledge at the heart of these issues. We now await the emergence of someone with the eye of a lynx and the right conjunction of circumstances: there is a reasonable possibility that environmental risk factors for AD will be identified by epidemiological studies. With or without this happy event, there are already many valuable advances being made in the field. The contributions brought together in this issue are testimony to these.

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