Psychiatric Epidemiology and the Classification of Mental Disorder*

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It is useful to separate the causal from the clinical role of epidemiological inquiry in assessing the contribution of epidemiological research to the classification and diagnosis of mental disease. Experimental medicine, as Claude Bernard affirmed and demonstrated, consists in induced observations of the physiopathological mechanisms of disease which constitute the bedrock of taxonomic systems. In only a relatively restricted segment of their activities, however, do epidemiologists employ the experimental method. Their approach to hypothesis-testing is more closely related to ordered observation and the logic of scientific investigation and the epidemiological approach to a causal classification consists in the elimination of alternative hypotheses. It is largely dependent on, and limited by, established information pertaining to clinical diagnosis and the systematic collection of reliable morbidity and mortality statistics. Furthermore, it incorporates the concept of multiple causation, the elements of which may be predisposing, provoking precipitating or perpetuating and can operate in a chain sequence.

Recognition of the potential role of this method in the study of mental disease has long preceded the use of the word ‘epidemiology’. Sixty years ago Emil Kraepelin called it ‘comparative psychiatry’ on which he commented: ‘By comparing a large series of observed cases we can study, first how far such general characteristics as sex, age, and culture can influence the clinical picture: in the same way we can also examine how factors like occupation, climate, and the general and personal circumstances of living may colour the clinical patterns encountered. Before any such comparative study can be undertaken, however, the relevant pathological processes must first be defined and delineated.’ As Kraepelin acknowledged at the time, progress along these lines was impeded by the lack of a formal discipline of ‘comparative psychiatry’. Since then this has been underwritten by the development of epidemiology as a mode of scientific inquiry which can already claim two outstanding neuropsychiatric achievements in the elucidation of pellagra and kuru. In both instances, however, well-defined disorders were under consideration and several alternative causal theories had been advanced. The contributions of Goldberger and Gajdusek qua epidemiologists lay in the elimination of false hypotheses to establish the aetiology of the disease, paying due regard to the multiplicity of factors involved. The physiopathological mechanisms of those conditions then took their place in the causal chain.

The position is less satisfactory in the case of poorly defined, idiopathic disorders, among which most psychiatric illnesses must be included. Here the most effective use of causal epidemiological research has been carried out in the name of population genetics, for in human population genetics epidemiological methods have to be introduced as soon as one proceeds beyond the study of individual pedigrees. In itself, however, the reach of such investigations can do no more than establish a single link in the causal chain. An indication of future progress may be derived from the current status of diabetes mellitus, a disorder with which schizophrenia has often been compared. The recent work on auto-immunity, HLA systems and virology has led to a more searching mode of classification which, though still speculative, maps the varieties of the diabetic syndrome. If the promise of current biological research in psychiatry is eventually fulfilled a model of this type may point the way to a more fundamental approach to the classification of the schizophrenias, in which epidemiological studies will help to identify sub-groups based on an understanding of pathophysiology.

In turning from classification to diagnosis Kraepelin drew a clear distinction between basic pathognomic disturbances and the less constant clinical features of mental disease. None the less, as he observed: ‘Even if they are not governed by a definite pathological process but represent rather a general human reactive mechanism they must nevertheless vary to some extent in accordance with the mode of attack and the extent of the pathological process. Every illness will in this respect exhibit its own characteristics...’ For the establishment of these characteristics epidemiological research has been most valuable in two of Morris’ seven spheres of application, namely the completion of the clinical picture and the identification of syndromes.

In completing the clinical picture in breadth and in

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depth the epidemiologist comes into his own, bringing a professional expertise to the establishment of population-based rates of disease by the use of screening and survey methods, and to the assessment of observer variation. Within general medicine the need to identify the so-called 'iceberg-phenomenon' has led to a wider concept of diseases like hypertension, diabetes and arthritis. Within psychiatry, despite the problems of definition and technique associated with most mental disorders, the surveys conducted at the level of primary care and the general population have already broadened the spectrum of illness, especially in respect of the phenomena of depression and anxiety, drawing attention to the psychosocial influences on the genesis, form and outcome of morbidity in the process.

The delineation of syndromes by means of epidemiological inquiry also claims an accepted place in general medicine, as illustrated by the sub-division of chronic bronchitis into airflow limitation and bronchial catarrh and the separation of 'essential' from 'secondary' hypertension. In psychiatry the whole Kraepelinian scheme is constructed on an admixture of explicit clinical experience and implicit epidemiological thinking but, despite its achievements, the population-base proved too small and biased for valid generation, as Kraepelin himself admitted towards the end of his career. With more refined methods of clinical assessment, longer periods of follow-up and better sampling the patterns of psychotic illness are now lending themselves to reassessment. By way of example, reference may be made to the delineation of patients with recurrent affective psychotic episodes after an initial schizophrenic disorder which preceded from clinical observation to confirmation by reference to the WHO International Pilot Study of Schizophrenia. In contrast, surprisingly little work has yet been carried out with the non-psychotic disorders, which have still to attract their Kraepelin and remain in a most unsatisfactory state. Here the need for investigation of extra-mural populations becomes pressing, as work at the primary care level has demonstrated, and a rich harvest can be anticipated from the application of epidemiological techniques.

REFERENCES