

Editorial

Modern psychiatric epidemiology: the challenge of risk factor research

The objective of etiologic epidemiology is to enhance our understanding of the pathogenesis of disease. Because of the emphasis on case ascertainment and standardization of diagnostic criteria, epidemiological investigations have often focused on diseases and conditions, for which there is tissue confirmation or laboratory validation. However, a large part of human morbidity cannot be pathologically confirmed. Psychiatric disorders are one example of such conditions that need to be more rigorously addressed. A causal-analytic rather than a descriptive approach is required but this is more easily said than done and examples in psychiatric epidemiology are still few. The work of Hein and Heun [1], “Risk factors of major depression in the elderly”, demonstrates how difficult this transition will be. Their baseline was a matched case control study and they now examine primarily social demographic and psychological risk indicators in a prospective follow-up design.

Long before the term “psychiatric epidemiology” was used in 1950, 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 color the clinical patterns encountered. Before any such comparative study can be undertaken, however, the relevant pathological processes must first be defined and delineated” [2]. As Kraepelin acknowledged, at the time progress along these lines was impeded by the lack of a formal discipline of “comparative psychiatry”. Consequently, his group in Munich focused on the neuropathology of psychiatric illnesses.

The history of psychiatric epidemiology has been described in terms of generations, defined mainly by means of methodology. In the first phase before World War II, which originated from German psychiatry, it was assumed that psychiatric illnesses were mainly hereditary in etiology, and research was focused on institutionalized mentally ill. Many such studies were conducted in Scandinavia or central Europe. This first generation mainly used administrative treatment statistics from hospitals to study the association of sociodemographic variables with specific mental disorders. Most of these studies had two major limitations: case ascertainment was incomplete and diagnoses were taken at face value, with little attention to their reliability or validity.

After World War II, psychiatric epidemiology was strongly influenced by sociological and social-anthropological think-

ing, especially in the United States. This second phase involved use of the census method and surveys of general population samples to measure the prevalence of distress and syndromes. The studies—of which the most famous was the Midtown Manhattan study—gave attention to the representativeness and completeness of their samples [5]. Many American social scientists and psychiatrists conducting these studies decided against using existing psychiatric nosology and substituted measures of overall mental impairment for traditional diagnostic categories. They attempted to demonstrate social factors as causal along with their unitary concept of mental illness.

During this time, there was considerable difference between the American approach and that which was adopted in continental Europe and Scandinavia. Researchers in the latter countries used traditional psychiatric diagnostic categories based on the assumption that each illness had a different underlying etiology, syndrome, course and treatment, and that biological (genetic, biochemical) factors rather than social and environmental stress would explain the cause of different syndromes, at least for the major psychoses.

During the 1970s psychiatric epidemiology entered a new phase which combined the field survey approach with a deliberate focus on specific disorders. One of the seminal studies was the US Epidemiologic Catchment Area (ECA) study [3]. The ECA study reports described the psychiatric illness picture in the United States in the early 1980s. While the ECA study was the first large epidemiological survey to apply modern diagnostic procedures, it remained primarily a descriptive study of prevalence and incidence. However, psychiatric epidemiological studies over the last decades have completed the key developments; achieved widespread acceptance of methodologies, derived descriptive data on prevalence rates and instituted a number of longitudinal studies. This provided the basis for the first causal analytical studies that focused on an evaluation of parental psychopathology, family problems, social networks and external stress. Most of these were conducted in the United States, where we find the first studies with a random sample of households using a structured diagnostic instrument, Great Britain, New Zealand, or countries with a strong affinity to this Anglo-Saxon research tradition such as Netherlands and Scandinavia. The famous Zurich Cohort Study or a more recent follow-up study of adolescents in Munich cannot conceal the fact that many other countries have not successfully adopted this approach. Arguably, there is a European divide in psychiatric epidemiologi-

cal practice. The majorities of epidemiological studies conducted in many countries are still prevalence surveys that just aim to discover rates although there is now little justification for these activities [4].

Rather, psychiatric epidemiology as an etiologic science must now turn toward questions about causes and mechanisms of psychiatric disturbances. Publications as that of Hein and Heun will have to be judged against the background whether or not the following challenges were met:

- a) The legacy of Kraepelin: Integrating measures of pathological mechanisms into psychiatric risk factor studies. It will be difficult to link psychiatric epidemiology more closely to ongoing biochemical, neurobiological, psychological or genetic research on mental disorders. Although they produce exciting knowledge, neuroscience measures of brain structure and function in general are still too expensive and impractical to use in population-based research. Furthermore, most biochemical parameters are based on peripheral blood measures that have an uncertain relation to brain metabolism. Gene–environment correlation and -interactions are quite complex in psychiatric disorders, where genotypes can act as a confounder or a modifier upon the way exposure influences disease onset. Obviously, not only biological processes are of importance. However, similar problems are encountered if researchers want to integrate detailed psychological measures of personality, peer-relations or attachment between mother and child into quantitative research. Furthermore, not only problems inherent to biological psychiatry or detailed psychological assessment have posed difficulties; in some countries, e.g. France or Italy, psychoanalytical or other concepts alien to quantitative research dominated etiological thinking for a particularly long time.
- b) The legacy of the first phase: For much of psychiatric epidemiology, and epidemiology in general, risk factor research must necessarily be population-based, in order to avoid important biases that can develop when studying only those cases who seek help or who are brought to clinical facilities by others. Less problems with data protection and privacy rulings certainly are one of the advantages countries like Scandinavia and Netherlands have when population-based studies are conducted as compared to the United States or Germany.
- c) The legacy of the second phase: The creation of diagnostic categories within which etiologic homogeneity is greater than between these categories. Classification schemas are always evolving but a major problem inherent in psychiatric epidemiology is the lack of a clear differentiation between normal and abnormal states. As long as psychiatry has to rely on phenomenological criteria (i.e. symptoms) rather than causal criteria (e.g. viruses or toxins) or biologic measurements (e.g. laboratory tests) to make diagnoses, dimensional measures and non-official classification systems must be considered in etiological studies. The dominance of the descriptive DSM system has certainly not fuelled etiological epidemiological research, but the

US, in particular in Child Psychiatry, also have a rich research tradition using continuous measures.

- d) The legacy of the third phase. For causal analytical studies high quality longitudinal data are indispensable. These will mostly be collected in form of a cohort design although the case–control like the cohort approach has a sound theoretical basis. Longitudinal data of this type are hard to deliver; a follow-up of large enough numbers of people for a long enough time without too much loss is necessary, and funding for this sort of study is particularly problematic in some countries.

In summary, causal analytical studies need to determine as clearly as possible whether a causal relation, not just an association, is likely to exist between a putative etiologic agent and an adverse health outcome. Most importantly, psychiatric epidemiology, like epidemiology of cancer, cardiovascular and rheumatologic disorders, needs a firm conceptual grounding in epidemiological principles. This is critical to the successful conduct and interpretation of epidemiological research.

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