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Psychiatric Epidemiology

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PSYCHIATRIC EPIDEMIOLOGY

It is not the purpose of this chapter to review the current state of knowledge in psychiatric epidemiology nor to present in any details the methods of epidemiology. For both these purposes there is a moderately extensive and reasonably adequate literature available. Rather the intention is to present some of the critical issues in this field, issues that, unless resolved, severely limit the potentialities of epidemiological inquiry, and to indicate some approaches to their solution.

The contribution that epidemiology can make to furthering knowledge of any disease or disorder is directly dependent upon the adequacy with which three interrelated, but analytically separable, problems can be resolved. These three problems are:

1. The adequacy with which cases can be identified and separated from non-cases.

2. The utility of the theories invoked to explain the processes determining the condition being studied.

3. The skill with which the independent variables are selected as indicators of these determinant processes.

While these issues are central to any epidemiological inquiry, psychiatric epidemiology presents some unique problems, especially in the

designation and ascertainment of cases. This topic, including the definition, classification, and measurement of psychiatric disorder or mental health status, has been the subject of numerous conferences, debates, and studies. There is as yet, however, little consensus among experts and until comparatively recently, a lack of clarity in much of the thinking in this area. To a large extent this confusion can, in my opinion, be traced to the failure of earlier attempts to recognize that in any inquiry the classification scheme used and the phenomena to be measured will be determined in large part by the purposes of the study. That is, no single classification scheme is likely to be equally useful for all purposes.

Classification and Ascertainment of Mental Disorder

In the field of psychiatric epidemiology, one of the clearest statements of the relationship of the classificatory scheme to the purposes for which such a scheme is to be used is that made by Gruenberg. He clearly differentiated some eight or nine such purposes which, without doing violence to his ideas, could be categorized into three major groups.

1. Studies concerned with aiding the planning of health services, including a determination of priorities, facilities, personnel, necessary finances, and so on.

2. Studies concerned with the processes of recovery from mental disorders.

3. Studies concerned with the determinants of genesis of mental disorders.

It is the failure to separate out the distinctive needs, in terms of both classificatory systems and underlying theories, of studies concerned with recovery from those concerned with genesis of disease that has perhaps been responsible for much of the confusion in the past. Implicit in this lack of distinction is the notion that the causes of any illness are the same as the causes of lack of recovery from that illness. Even though as early as 1943 Halliday eloquently identified the advantages of making such a distinction, his advice has largely been overlooked in epidemiological studies, in which both the research strategy and the classificatory systems used are frequently based on the implicit assumption that these two sets of causes are identical. A few familiar examples may help make this point clear. Knowledge of abnormalities in carbohydrate and fat metabolism, including the role of insulin, has improved our ability to treat diabetics (that is, to understand something of the recovery process). This knowledge, however, is of very limited utility in explaining why diabetes occurs or why, for example, the rates in the United States have been increasing over the last half century. Further, some of those factors suspected as being important in the genesis of diabetes (dietary factors and sedentary living, for example) may also be antecedents to diseases (such as coronary heart disease) usually classified separately from diabetes. By the same token, knowledge of the relationship of

cigarette smoking to cancer of the lung has increased our knowledge of genesis but has been of no use in changing our approach to treatment. In the field of mental disorders the identification of the social breakdown syndrome by Gruenberg is a good illustration of this point. According to this concept the factors responsible for much of the disability that accompanies mental disorder in many patients are independent of the factors responsible for the initial condition. Reduction of disability can thus be accomplished by changing these relevant factors even though we may be ignorant of the causes of the underlying mental disorder.

The implications for a classificatory scheme of making a distinction between studies concerned with recovery from those concerned with onset are clear. In psychiatric epidemiological studies concerned with the recovery process (including the effectiveness and efficiency of various modalities of medical care and health services), existing clinical classifications should be the basis for case definition. Further, ascertainment can, without too much loss, be restricted to those under some form of psychiatric care. What is required is an improvement of the reliability with which such cases are labeled (that is, improvement in the standardization of criteria to be used and the ability of the investigators and clinical personnel to apply these criteria). Such attempts are currently being undertaken by a number of investigations, one of the most notable of which is that being conducted by the mental health section of the World Health Organization. The complexities of this problem of

standardization should not be underestimated. According to Wing they include:

1. Determination of the dividing line between psychotic and nonpsychotic conditions.

2. Decisions to be made when there are combinations of different forms of psychosis in the same person.

3. The influence of known or suspected etiological factors on diagnosis (for example, the effect of prior knowledge that the patient was taking amphetamines in the decision as to whether to label the condition “amphetamine psychosis” or “schizophrenia [drug induced]”).

4. The effect of knowledge of previous diagnoses. (For example, in a patient presenting with a condition that under most conditions might not be called psychotic, the knowledge that there had been an earlier clear-cut episode of schizophrenia might lead some clinicians to re-diagnose schizophrenia.)

5. Social and cultural factors that influence diagnosis.

While it would appear that none of these problems is insuperable, requiring mainly the elaboration of a consistent set of rules (if necessary, arbitrary rules), the point to be made is that even the most satisfactory solution of all these difficulties will not provide a useful classificatory scheme for the other two major purposes, namely, administrative purposes and

studies concerned with the determination of the factors responsible for the onset of disease.

For administrative purposes, particularly for the planning of health services, it is crucial not only that cases outside as well as within the current treatment network be counted but that such cases be classified as to whether their condition is regarded as susceptible to the current technology of treatment or rehabilitation and as to whether such conditions are preventable. A beginning of such a classification is included in the guide to the control of mental disorders published by the American Public Health Association.

The greatest difficulty of all lies in the development of a suitable classificatory scheme for purposes of studying the genesis of mental disorders. Here, in addition to the problems posed by attempting to ascertain cases in the community as well as those under treatment (with all the attendant problems of variation in intensity of contact between the investigator and respondent and the problem of intra- and between-observer reliability), two rather special problems emerge.

The first of these relates to the various factors that can lead to a person becoming labeled as a psychiatric case. Gruenberg identified five such factors, which alone, or in combination, are involved in this labeling.

1. The result of an appeal for help, that is, as a result of symptoms and complaints on the part of the respondent.

2. The result of concern or fears in others.

3. The result of trouble to others.

4. The result of breaking the law.

5. The result of a failure in functioning.

The importance of such a categorization is that it makes clear a well-known but often overlooked fact as far as epidemiological studies are concerned. The labeling of an individual as a psychiatric case is usually the result of the product of two sets of potentially independent factors, the intrapersonal disorders of mood, thought, and behavior (number 1 in the above scheme) and the existing social standards and level of tolerance for deviant behavior (numbers 2 through 5 in Gruenberg's scheme). The majority of patients under psychiatric care (particularly in psychiatric hospitals) will be under such care by virtue of both sets of factors, but the relative importance of each set may vary systematically in different sorts of people. Thus it is theoretically possible that the majority of lower-class patients in a psychiatric hospital are labeled as cases by virtue of their interpersonal difficulties (that is, violation of the existing social norms) whereas upper-class individuals with the same label may be patients largely by virtue of their intrapersonal difficulties (symptoms, complaints, and so on). The situation

will be further confounded when individuals not under treatment are to be included in the study and labeled as cases. Almost by definition the majority of these will not have disturbed the social norms and expectations to the same degree as will those who are under treatment. This may mean that their intrapersonal disturbances are not so severe as in treated patients, or alternatively that the tolerance, of deviance, and the expectations for performance, by significant others are different in the case of community cases compared with treated patients. In order then to ensure that like things are being measured, particularly when comparisons between communities are to be made (where both sets of factors may be expected to vary), it would seem desirable to develop two separate classificatory schemes. (1) The first of these would be concerned with measuring disturbances in mood, thought, and behavior as reported by the respondent, independently from degree of disability or those aspects of impaired function which are dependent upon the views or expectations of others. Such an approach forms the basis for one part of the classification used by Leighton ("behavioral patterns or syndromes"); by the new instrument, the present state examination, being developed by Wing et al.; and to some extent by the approaches being used by Dohrenwend—the structural interview schedule and psychiatric status interview (although these latter two do not make this crucial distinction as clearly as might be desired). The central problem here is not only the development of reliable instruments but being able to determine the

frequency and duration of the symptoms rather than just their presence or absence at the time of examination. The organization of these symptoms into clusters and the development of syndromes are an elaboration of this approach, which also requires that this classificatory system be kept distinct from that concerned with failure of interpersonal functions. (2) Classificatory systems having to do with the failure of interpersonal functions (called level of disability by Leighton and, more broadly, level of functioning by Gruenberg) would form the basis for a separate classificatory scheme. The relationship between these two sets of indices would be an important research topic in its own right and might yield a third set of classifications.

Even if this problem of separate classificatory systems can be resolved, however, it still leaves unanswered the problems of what signs and symptoms (or other measures of disturbed intrapersonal functioning) are to be measured. It is here that the crucial role of the underlying theory subscribed to becomes apparent. The preoccupation on the part of many investigators with distinguishing between physical and psychological symptoms or signs, for example, is in my opinion an indication of subscription to a theoretical model that in light of current knowledge has outlived its utility. As this problem cannot be reviewed out of context of the theories concerning the nature of disease and its causes, with particular reference to the role of environmental factors in etiology, these will be now discussed in some detail.

Theories Concerning the Nature of Disease and Its Causes

Throughout history there has been a conviction in medicine that certain environmental factors are important in the etiology of disease. The specific factors deemed worthy of study, however, have varied considerably over time from the "airs, waters, places" of Hippocratic times to the microchemicals and microorganisms of today. Quite clearly the factors selected for study (from an almost infinite number of possibilities) are heavily dependent on the existing theories of the nature of disease and its causes and the existing level of technology. Comparatively recent findings tend to suggest that we need to modify some of these existing theories to allow for the possibility that one of the more important aspects of the environment for man (from a disease etiology point of view) may be the presence of other members of the same species.

Paradoxically, some of the more convincing evidence supporting this point of view comes from animal studies. To a large extent, these have been concerned with variations in the size of the group in which the animals interact and in situations that lead to confusion over territorial control. A number of investigators have shown, for example, that as the number of animals housed together increases, with all other factors such as genetic stock, diet, temperature, and sanitation kept constant, maternal and infant mortality rates rise, the incidence of arteriosclerosis increases, resistance to a

wide variety of insults, including drugs, microorganisms and X-rays, decreases, and there is an increased susceptibility to various types of neoplasia. Lack of territorial control in mice has been shown to lead to the development of marked and persistent hypertension, to increased maternal and infant mortality rates, to reduced resistance to bacterial infections, and to decreased longevity.

In addition to demonstrating the health effects of variations of the social milieu, further animal studies have provided clues as to the processes through which they may be produced. Changes in group membership and the quality of group relationships have been shown to be accompanied by neuroendocrine changes, particularly, but not exclusively, by changes in the pituitary and adrenocortical systems. The changes in some of these hormones, such as the 17-hydroxycorticosteroids and the catecholamines, especially if prolonged, can, in turn, markedly affect the homeostatic mechanisms of the body and the responses to a wide variety of stimuli. The evidence, then, from a series of studies would seem to be both sound methodologically and reasonable from a biological point of view.

Convincing as this animal work would appear to be, the relevance of these findings to human health, however, is as yet unproved, and considerable doubt exists as to the appropriate analogues in the human social system. Attempts, for example, to demonstrate that increased population

density and crowding are related to poorer health status have been unconvincing and have led to confusing and often conflicting results. A careful review of some of these studies taken in conjunction with the animal work would suggest that for future research in this area to be profitable we should abandon a search for the direct human counterpart to animal crowding or territorial confusion and concentrate instead on some more general principles, or hypotheses, that can be derived from these data. In my view, four such principles seem worth considering.

1. The social process linking high population density to enhanced susceptibility to disease is not the crowding per se but the disordered relationships that, in animals, are inevitable consequences of such crowding. These, while manifest by a wide variety of bizarre and unusual behaviors, often have in common a failure to elicit anticipated responses to what were previously appropriate cues and an increasing disregard of traditional obligations and rights. Thus, under crowded conditions, habitual acts of aggression (including ritualized aggression), subordination, or cooperation on the part of one animal fail to elicit appropriate reciprocal responses on the part of another. Characteristic obligations and responsibilities become blurred (for example, female rats cease caring for their young and male-female relationships become disturbed to a point where the equivalent of gang rapes have been reported in rats under conditions of high population density). The failure of behavior patterns to accomplish their intended results

(that is, to lead to predictable responses on the part of others) leads frequently to repetition of these behaviors with, presumably, concomitant chronic alterations in the autonomic nervous system activity and hormonal secretions associated with such activity. These, in turn, alter the homeostatic mechanisms of the organism, leading to increased susceptibility to disease.

This hypothesis would suggest that in human populations the circumstances in which increased susceptibility to disease would occur would be those in which, for a variety of reasons, individuals are not receiving any evidence (feedback) that their actions are leading to desirable and/or anticipated consequences. In particular this would be true when these actions are designed to modify the individual's relationships to the important social groups with whom he interacts. Such circumstances might occur in a variety of situations.

It is highly probable that when individuals are unfamiliar with the cues and the expectations of the society (as in the case of migrants to a new situation or of individuals involved in a rapid change of the social scene, such as the elderly in an ethnic enclave caught up in the process of urban renewal), their actions would be unlikely to lead to the consequences they anticipate and thus, owing to the chain of events suggested above, they should be more susceptible to disease than are those for whom the situation is familiar. Some circumstantial evidence supporting this point of view exists. Scotch and his

collaborators found that blood pressure levels among Zulu who had recently migrated to a large urban center were higher than both those who had remained in their rural tribal surroundings and those who had lived for more than ten years in the urban setting. In two independent studies, Syme demonstrated that occupationally and residentially mobile people have a higher prevalence of coronary heart disease than have stable populations and that those individuals displaying the greatest discontinuity between childhood and adult situations, as measured by occupation and place of residence, have higher rates than those in which less discontinuity could be determined. Tyroler and Cassel designed a study in which death rates from coronary heart disease, and from all heart disease, could be measured in groups who were themselves stable but around whom the social situation was changing in varying degree. For this purpose they selected forty-five- to fifty-four-year-old white male rural residents in the various counties of North Carolina and classified these counties by their degrees of urbanization. Death rates for coronary heart disease and all heart disease showed a stepped gradient with each increase in the index of urbanization of the county. In a further study Cassel and Tyroler studied groups of rural mountaineers, one of which was composed of individuals who were the first of their family to engage in industrial work while the second comprised workers in the same factory drawn from the same mountain coves and doing the same work for the same pay as the first group, but who were the children of previous

workers in this factory. The underlying hypothesis was that the second group, by virtue of their previous experience, would be better prepared for the expectations and demands of industrial living than the first and would thus exhibit fewer signs of ill health. Health status was measured by responses to the Cornell Medical Index and various indices of sick absenteeism. As predicted, the first group had higher Cornell Medical Index scores for both physical and emotional symptoms and higher rates of sick absenteeism, after the initial few years of service at each age, than had the second.

A second set of circumstances in which the individual would not be receiving any feedback that his actions were effectively modifying the situation might occur where there is some evidence of social disorganization. This, while still being far from a precise term that can be accurately measured, has proved to be a useful concept in a number of studies. In the hands of several investigators, for example, various indicators of social or familial disorganization have been related to increased rates of tuberculosis, mental disorders, deaths from stroke, and prevalence of hypertension.

Clearly, though none of these studies prove that this postulated psychosocial process is an important determinant of the higher disease rates, they are all at least consistent with such a notion.

2. Not all members of a population are equally susceptible to the effects

of these social processes. Systematic and regular differences have been observed with the more dominant animals showing the least effects and the subordinate ones having the most extreme responses. These differences are manifest both in the magnitude of the endocrine changes as well as in increased morbidity and mortality rates. Conceivably these findings may, in part, explain the high levels of blood pressure found in American blacks, who not only usually occupy a subordinate position in society but whose lives are frequently characterized by considerable evidence of social and familial disorganization. At the very least, such findings would suggest that studies aimed at identifying the health consequences of migration should distinguish those migrants who occupy subordinate positions in their host countries from those who occupy positions of power or prestige.

3. The third principle is concerned with the available protective factors, those devices that buffer or cushion the individual from the physiological or psychological consequences of social disorganization. These would seem to be of two general categories, biological and social. Under biological factors would be included the adaptive capacities of all living organisms, the capacity, given time, to adjust physiologically and psychologically to a wide variety of environmental circumstances. In animals, this is illustrated by the higher responses of laboratory-naive animals to given stimuli than of veteran animals and to the much lower rate of pathology in animals born and reared in crowded conditions than in animals transferred to these conditions some

time after birth. In humans, the finding that death rates from lung cancer in the United States, when controlled for cigarette smoking, are considerably higher in the farm born who migrated to cities than in lifetime urban dwellers (despite the longer exposure of the latter to atmospheric pollution) would seem to be evidence of the same phenomenon.

In addition to these biological adaptive processes, various social processes have also been shown to be protective. Chief among these are the nature and strength of the group supports provided to the individual. In rats, for example, the efficacy with which an unanticipated series of electric shocks (given to animals previously conditioned to avoid them) can produce peptic ulcers is determined, to a large extent, by whether the animals are shocked in isolation (high ulcer rates) or in the presence of litter mates (low ulcer rates). The territorial conflict that led to elevated blood pressures was produced by placing mice in intercommunicating boxes. Hypertension only occurred, however, when the mice were “strangers.” Populating the system with litter mates produced none of these effects. In humans, small group studies have shown that the degree of autonomic arousal that can be produced by requiring solutions to what in reality are insoluble tasks is more extreme if the group is made up of strangers than when it is made up of friends. Modern studies on the epidemiology of tuberculosis in the United States and Britain have shown that the disease occurs more frequently in marginal people, that is, in those people who for a variety of reasons are deprived of meaningful

social contacts.

If these three hypotheses are correct, it would imply that the health consequences of disordered social relationships will not be universal, affecting all people in the same manner. A more adequate formulation would hold that such consequences will depend on (1) the importance or salience of the relationships that become disordered; (2) the position of the individuals experiencing such disordered relationships in the status hierarchy; (3) the degree to which the population under study has been unprepared by previous experience for this particular situation (that is, has had insufficient time to adapt); and (4) the nature and strength of the available group supports.

4. The final general principle that can be derived from the animal experiments relates to the manifestations of ill health that might be anticipated under conditions of social change and disorganization. The model of disease causation provided by the germ theory has accustomed us to think in mono-etiological specific terms. Accordingly, much of the work concerned with social or psychological antecedents to disease has attempted to identify a particular situational set (usually labeled “stress” or “a stressor”) that would have a specific causal relationship to some clinical entity, analogous, say, to the relationship between the typhoid bacillus and typhoid fever. Such a formulation would appear to be clearly at variance with the animal data, a striking feature of which is the wide variety of pathological conditions that

emerge following changes in the social milieu. A conclusion more in accordance with the known evidence would be that such variations in group relationships, rather than having a specific etiological role, would enhance susceptibility to disease in general. The specific manifestations of disease would be a function of the genetic predisposition of the individuals, their metabolic states, and the nature of the physicochemical or microbiological insults they encounter. This concept of generalized susceptibility would be consistent with the situation in the United States, where it has recently been demonstrated that those regions of the country having the highest death rates from cardiovascular disease (age, race, sex specific) also have higher than expected death rates from all causes, including cancer and infectious diseases. This illustration, of course, does not necessarily document that social processes are responsible for such an increased susceptibility, but does lend credence to the view that variations in generalized susceptibility may be a useful concept. Somewhat more direct evidence is provided by industrial studies in the United States, which have shown that managers in a company who, by virtue of their family background and educational experience, were least well prepared for the demands and expectations of executive industrial life had the highest rates of all diseases, physical illness as well as mental, major as well as minor, long-term as well as short-term.

From this formulation it would appear that an initial aim in epidemiological studies (including psychiatric epidemiological studies) would

be to identify these individuals at highest risk to disease or disorder in general. The manifestations of such increased susceptibility could be quite widespread and include many clinical entities that for therapeutic purposes are currently regarded as separate diseases. A second level question would then address itself to the determinants of the manifestations in the susceptible populations. Why are there disturbances in mood or thought on the part of some individuals and hypertension or tuberculosis in others? Here I suspect the answers are most likely to come from genetic studies, from studies of the combined effect of genetic and experiential factors on metabolic patterns, and from studies of physicochemical and microbiological insults.

The decision, then, as to the appropriate classificatory scheme to be used in studies concerned in the genesis of disease and, from such a decision, the instruments and techniques to be used to gather such information is, I submit, inextricably bound to the theories espoused by the investigator.

This chapter should not necessarily be read as a plea to accept this particular theoretical formulation, but rather that, in epidemiological studies in general and psychiatric epidemiological studies in particular, the onus is on the investigator to make his theoretical formulation explicit and the information to be obtained both for the classification of cases and characterization of the independent variables congruent with it.

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