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THE EPIDEMIOLOGY OF SCHIZOPHRENIA

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We want to understand the distribution of schizophrenic disorders in human populations and the factors that contribute to high and low rates, because without such understanding we cannot plan treatment programs or appraise our efforts at prevention. Behind every action program for any disorder there lies a picture of the condition's epidemiology; sometimes this picture is firmly based on established facts, but more often it is inferred from limited data mixed with general theoretical preconceptions. The uses of epidemiological knowledge are grouped under seven conventional headings: (1) historical trends, (2) community diagnosis, (3) individual risk, (4) beyond the clinical horizon, (5) identification of syndromes, (6) working of the health services, and (7) the search for causes.

Historical Trends

Are schizophrenic conditions becoming more common, less common, or is their rate of occurrence stationary? Is it a condition, such as intestinal ulcers and lung cancer; a growing health problem; or, like tuberculosis and rubella in the United States, a problem of declining importance?

There are no data suitable for providing a firm answer to this question. In thinking about the various possibilities, several general facts should be kept in mind. Ackerknecht pointed out that there is a tendency for

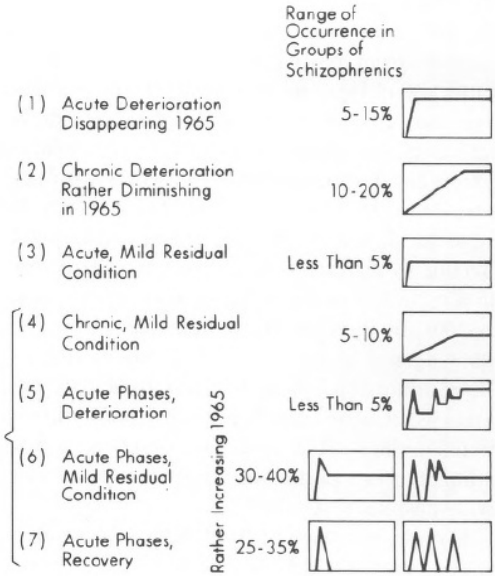
progressive thinkers to believe that social progress leads to more mental disorder. The term "schizophrenia" itself was coined by E. Bleuler during 1911 to symbolize a shift in conception from the earlier picture of dementia praecox and the universally deteriorating course that the name implied. Hence nomenclature and diagnostic criteria took a big step. Since then E. Bleuler's move away from Kraepelin's view of a uniformly deteriorating condition has gone furthest in the United States, where the concept of schizophrenic conditions in the absence of psychotic disruption of mental functioning has developed. This concept is clearly symbolized in the term "pseudoneurotic schizophrenia," which, in effect, says that there are cases of schizophrenic disorder that masquerade as neurosis. The tendency to regard psychiatric patients with no gross overt psychotic manifestations as schizophrenic has gone further in the United States than in Europe and most other parts of the world. In Denmark, attention to psychogenic psychoses removes from the schizophrenic category many cases that even other Europeans would regard as schizophrenic. Hence, appraisal by clinical records over time is bound to be difficult, since the criteria for making the diagnosis have been shifting during the past century.

But these changes have not affected such investigators as Manfred Bleuler, E. Bleuler's son, who said, "When I speak of schizophrenia, I mean real psychosis. I excluded from the study border-line cases, as for instance pseudoneurotic patients who had never been psychotic in the social sense. . .

." He followed 208 of 216 schizophrenic patients admitted to his hospital in Zurich in 1942 or 1943. He followed them until death or 1963, using a description of the various courses developed by

Bleuler. Figure 30-1 tells his story. He emphasized that the 1965 percentages are based on familiarity with his own similar cohort published twenty-five years earlier and with the still earlier studies of his father and of Kraepelin: "catastrophic schizophrenia is dying out. ... By 1941 the rate had gone down to 5-18 per cent [of an earlier cohort]. . . . This most terrible form of schizophrenia has become increasingly rare. ... In my material not a single psychosis beginning after 1942 has developed in this way."

FIGURE 30–1. **Long Courses of the Schizophrenias According to Catamneses of 316 Schizophrenics Completed in 1941.**



Reprinted from M. Blueler, "A 23-Year Longitudinal Study of 208 Schizophrenics and Impressions in Regard to the Natures of Schizophrenia" in D. A. Rosenthal and S. S. Kety, eds., *The Transmission of Schizophrenia*. (Oxford: Pergamon Press, 1968, p. 5. Reprinted with permission.)

Despite some weaknesses in these studies, they deserve special attention because of the author's high qualifications for forming judgments on the matter. His impressions and data are not out of keeping with other evidence. The most malignant forms of schizophrenic psychoses are becoming rarer and the more benign forms more common than before, at

least as percentages of cohorts newly admitted to a service. This method of appraising a change in the frequency of the most severe forms of a condition has a weakness: The proportion of newly admitted patients who pursue the malignant course in later cohorts as compared to former cohorts could drop because the number of milder cases being admitted has risen. But when M. Bleuler said that none of his cases starting after 1942 showed the most malignant course he saw earlier, the argument is strengthened.

Data on schizophrenic patients in mental hospitals and in outpatient clinics suggest a modified course resulting in a falling prevalence of schizophrenia since 1955, but these data can also be interpreted in other ways. The number of schizophrenic patients in United States mental hospitals was rising steadily from the earlier part of the century until about 1955, when it leveled off at about 150 per 100,000 population (age adjusted) and began to decline steadily. The schizophrenic first admission rate to mental hospitals rose steadily from about 18 per 100,000 in 1950 to almost 20 in 1962. (The subsequent decline to about 17 per 100,000 in 1964 is due to a technical factor in the statistical reporting system. In 1962 the state mental hospital systems agreed to standardize their definition of “first admission” so as to refer to a patient’s first psychiatric inpatient experience rather than, as had often been the practice, a first admission to a particular hospital or particular state hospital system.)

A rapid decrease in the length of hospitalization episodes explains this drop in the prevalence of hospitalization for schizophrenia while the first admission rate for the condition was rising. The shortened stay of all mental hospital admissions since 1955 is a well-established phenomenon, and its impact has been felt by schizophrenics at least as much as by patients with other diagnoses. It is due to earlier releases in the face of falling death rates in mental hospitals.

Do these shortened hospital stays since 1955 reflect a shortened schizophrenic episode and thus a lowered schizophrenic syndrome prevalence in the population? Or is it simply a matter of using outpatient treatment to replace inpatient treatment for the same disorder? Or have we, as some have suggested, simply shifted the burden of chronic care from the hospitals to the community? The rapid dissemination of Thorazine and other phenothiazines and medications for treating chronic mental disorders since 1955 has been held by some to account entirely for this large shift in the locus of the chronic mental patient. If the shift is attributable to new drugs, it must be assumed that the new drugs had an effect on the types and severity of symptoms associated with the mental disorders, including schizophrenic disorders. And if the types and severity of symptoms have been changed so as to reduce the need for mental hospital care, presumably some manifestations of the schizophrenic syndrome have indeed been prevented. But as Chapter 47 makes clear, the problem is, in fact, not that simple. When the new drugs

were introduced, there was already in motion a trend toward reducing the length of hospital stay, partly in an effort to retain the patients' functional assets and community ties in order to prevent chronic deterioration. In addition, beginning in the late 1960s, there was a policy shift by state governments to reduce mental hospital stays and to reduce the mental hospital census not only as a better way of caring for patients but also as an expression of anti-state-hospital sentiment, in particular, and a taxpayer's revolt against publicly supported personal services, such as hospitals and schools. It cannot be assumed, therefore, that the patients released in short order after mental hospital admission are indeed functioning at a higher level than admissions during earlier years; they may be functioning with the same types and severity of symptoms as their predecessors but only in a different location.

This is why the data on the level of functioning of cohorts of mental hospital admissions over time are important for appraising whether a lowering of the prevalence of severe manifestations of the schizophrenic syndrome has actually occurred. To help conceptualize this issue and to work out methods for researching it, the social breakdown syndrome was developed. The evidence indicates that severe disability in personal and social functioning associated with schizophrenic disorders has become rarer as unified clinical teams moved the services to a pattern of community care. The annual incidence of new long-term episodes of chronic social breakdown

syndrome was halved with the introduction of comprehensive services rendered by unified clinical teams in a community where the services for the severely ill patient are closely integrated with the entire network of community services.

Direct evidence regarding the prevalence or incidence and average duration of schizophrenic episodes is lacking. Viewing the social breakdown syndrome as a secondary complication of schizophrenic conditions as well as of other severe mental disorders made it possible to study and report on whether that secondary complication became less common without answering the question as to whether similar changes had occurred in the schizophrenic syndrome or only in other severe mental disorders.

In discussing the possible reasons why the latest cohort of admissions to the Burghölzli Clinic in Zurich showed a benign course as compared to the earlier cohorts of admissions to the same clinic, M. Bleuler was uncertain as to whether this could in part be attributed to the fact that failure to release patients "at the right moment" does not occur any longer. If his data are looked at only in the context of what happened over many decades in a single hospital's patients, it is difficult to find the appropriate information. But if those changes are seen as part of a widespread tendency to modify the way in which hospitals are used in caring for patients with serious mental disorders, additional light can be thrown on this interesting speculation.

In the overall picture of changing patterns of care some services run ahead of the general movement and to some extent act as trail blazers, while others lag behind, remaining unchanged over the same time period. If we look at the pioneering developments inaugurated by Rees, Macmillan, and Bell (see Chapter 47, "The Social Breakdown Syndrome and Its Prevention") as having been due to the characteristics of those hospital directors rather than to any special local characteristics of the mental disorders in the communities they served, we can contrast the way mental disorder syndromes evolved in their communities to the way the same syndromes evolved in other communities during the same period to get evidence about the way in which the shift in the pattern of psychiatric care, which we refer to here simply as "community care," ameliorates the course of the schizophrenic disorders. Their experience was in the decade before the tranquilizing drugs became available. More systematic data, gathered in Duchess County since 1960, show that a similar effect was achieved beginning well after these new drugs had been incorporated into psychiatric practices.

Putting these two sets of observations together with M. Bleuler's observations regarding the relatively low frequency of catastrophic courses in later cohorts of schizophrenics points to the conclusion that community care of the chronic schizophrenic patient reduces the risk of chronic deterioration in that condition. To some this conclusion will appear inevitable and without need for further corroboration, while to others the conclusion will appear

repugnant and the evidence for the conclusion inadequate. I take a position that falls between these two reactions: I find the conclusion inevitable but recognize that the evidence for it is inadequate. From an abstract scientific point of view it may not appear to matter very much when we come to a conclusion on the issue. But from a practical point of view the way this evidence is evaluated makes an enormous difference. While the evidence is not absolutely conclusive that community-care patterns prevent chronic disability, it would be wrong today to say that anyone who wishes to reinstitute locked-door, isolated psychiatric services, physically and socially far removed from the world where the patients formerly lived and to which they will return if they recover sufficiently, is perfectly entitled to do so. On the contrary, the evidence is sufficiently strong to put a person who advocates such a view on the defensive; the burden of proof that this will not worsen the outcome for his patients has now become his. Those who advocate movement toward community-care patterns of delivering psychiatric services have a strong case that this leads to less chronic deterioration. The fact that some new, still unsolved problems in patient care have emerged out of community care does not weaken this case. The recurrently collapsing patient who escapes the after-care system and gets into deeper and deeper personal and social problems actually exists. Some released patients are living under indefensibly bad hygienic conditions and in situations of social isolation that cannot be regarded as beneficial. Present evidence suggests that if the

solution to these problems is sought by a return to long-term state hospital care, damage will be done. Many other patients will be kept in long-term care because of a fear that they will pursue such a course. (Our present ability for predicting this outcome is not good.) Apparently, the recurrently collapsing patient is less damaged by his failures than by long-term institutionalization. The systems of aftercare need to be strengthened, and less intensive forms of supervision than total institutions will have to be expanded (unless some new, effective treatment or preventive measure for the schizophrenic condition itself is discovered).

If the decrease in the frequency of chronic deterioration is a consequence of changed patterns of patient care (rather than historical trends in the nature of the condition or new specific treatments), there are two possible views as to what kind of change in patient care has occurred and is occurring. Some see community care as undoing a noxious form of bad care that previously produced the formerly common chronic deterioration. Others see community care as representing an innovation in social psychological insights that helps protect the patients from deteriorating tendencies. This is an interesting theoretical controversy, which will continue for a period; the arguments and evidence amassed on each side will be beneficial to psychiatry because they will force a new perspective on the distinction between iatrogenic illness and the effect of the social environment on treatment and human functioning.

Community Diagnosis

Knowledge of the size of the schizophrenic problem in a community, of which part of the population has the highest rates of occurrence, and of what part of the psychiatric load schizophrenic conditions represent, can help in planning.

One simple way to look at the frequency of schizophrenic conditions is to look at the people in psychiatric treatment on one day. Yolles and Kramer showed that people aged forty- five to sixty have the highest rate of residence in state and county mental hospitals. In this age group, about 200 out of every 100,000 residents of the United States are patients in such a mental hospital with a diagnosis of schizophrenia on any given day. They are more than 60 per cent of all mental hospital patients in that age group. This fact accounts in part for psychiatry's preoccupation with the schizophrenic syndrome and provides a measure of its importance as a form of mental disorder. At every age between sixteen and sixty-five years, more than half the mental hospital patients are schizophrenic.

But these are only half the psychiatric patients with schizophrenic disorders; there are almost as many schizophrenic patients in the other psychiatric facilities. In Maryland in 1963, about 1.3 people per 1,000 were in state and county mental hospitals (age adjusted rate). If Veterans' Administration, general and private mental hospitals, outpatient clinics, and

long-term leave from mental hospitals are added the rate is 2.3 per 1,000. In Duchess County, New York (where the age group sixteen to sixty-five numbers about 100,000), in 1970 there were 140 schizophrenic patients in mental hospitals and at least as many in other facilities on one day. Two-thirds of the latter were in the mental hospitals' after-care clinics. Schizophrenic cases do not dominate the caseloads of these other psychiatric facilities as much as they dominate those of government mental hospitals (state, county, and Veterans' Administration), where they make up half the caseload. In outpatient clinics, general hospitals, and private mental hospitals, schizophrenic cases are only about one-fourth of the adult caseload.

These summary figures give a picture of the impact of schizophrenic patients on clinical services in terms of the caseload at any one point in time. They also provide a glimpse of how large a problem schizophrenic disorders appear to be when viewed from the clinician's perspective.

This picture can be further enlarged by looking at the rate at which new schizophrenic cases come to the clinical services. In the mental hospitals, the number of first schizophrenic admissions per year was about 16 per 100,000 for all ages (adjusted). The age group with the highest first admission rate is twenty-five to thirty-four years, where it is twice the overall rate. This peak incidence rate is more than twenty-five years younger than the highest prevalence rate for schizophrenic patients in mental hospital residence. This

twenty-five- year difference between the age group with the highest first admission rate for schizophrenic patients and the age group with the highest prevalence rate of patients in residence may well account for the commonly held clinical view that schizophrenic disorders tend to be long-lived and carry a poor prognosis.

The people who reside in the center of an urban area are admitted to mental hospitals with a diagnosis of schizophrenia much more often than the people who live on the outskirts of the city. This was first shown to be so by Faris and Dunham, who used the Park and Burgess theory of urban social ecology to test a hypothesis about communication functions in different parts of the city and how defects in social communication can favor schizophrenic disorders. In the rooming house center, first admission rates for schizophrenia were more than 700 per 100,000 per year and dropped to 250 at the periphery. We can look on this finding in several different ways. The easiest is to interpret these findings as indicating that a person with a schizophrenic psychosis is much more likely to be admitted to mental hospitals if he lives in the city's center than if he lives in the periphery. Medical care facilities are well known to be used differentially by different parts of the community.

A second interpretation is to see the findings as indicating that living in the city's center somehow increases the probability of a schizophrenic

disorder. This, in fact, was the hypothesis advanced by Faris and Dunham. Their argument is strengthened by the fact that admissions to mental hospitals for other diagnoses do not display the same patterns as the predicted rates for schizophrenic admissions. Thus they have produced evidence that the differential first admission rates to mental hospitals by different parts of the city have a definite pattern for schizophrenic admissions, but a different pattern for organic disorders and no particular pattern for manic-depressive psychoses. Hence they can argue that the pattern observed for schizophrenic admissions is not due to a simple difference in hospital utilization rates.

A third interpretation, the drift hypothesis, states that during the preclinical phases of schizophrenic disorders people will tend to drift into the anonymous central city and thereby artificially inflate the admission rates from that part of the city. This hypothesis is difficult to refute, or even test, because it poses great methodological research problems. Dunham reviewed the studies bearing on this issue. The other side of the drift hypothesis is even more difficult to deal with. It has to do with the notion that a generation that starts life in the central city will, to the extent possible with successful job careers, migrate out of the central city to the periphery and that a family with a chronic schizophrenic member will have less successful job careers and, therefore, be less likely to succeed in moving to the more desirable city periphery. The preclinical state is associated with a failure to achieve the

upward social mobility that other people in the same neighborhood have experienced. The concept of differential migration of those at high risk of later showing a particular disorder as compared to those at low risk of exhibiting this condition was first introduced into the general field of epidemiology by investigators concerned with the distribution of mental disorders. Ødegaard's investigation of the relationship between migration from Norway to Minnesota and the probability of later mental disorders remains as the most thoroughly executed of such studies. It did show a slight association between migration and later mental hospital admission for schizophrenic psychoses. (The methods employed have since then turned out to be of great importance in the study of diseases associated with occupations; it has been found that the people in a given occupation may show a lower than expected prevalence of certain chronic disorders because those who leave the occupation are at higher risk of showing the condition later in their lives.)

These are the most important efforts to clarify variations in psychiatric treatment rates for schizophrenic disorders. More generally, in the absence of very detailed investigations, differences in treatment rates are most likely to be owing to differences in the way the psychiatric services are organized and the utilization rate of alternate forms of care. Certainly, the current statistics on changes in mental hospitals and community mental health centers are properly looked on as reflecting radical changes in policy regarding how to make psychiatric services available. At the present time it is reasonable to

look on these medical care statistics as indicating changes in the way the services relate to a pool of schizophrenic people in the population and to recognize that this pool is of undetermined size and is undoubtedly larger than the population receiving psychiatric services.

Individual Risk

By counting the frequency of new cases of schizophrenic disorder arising at each age, an accumulated risk over a span of years can be computed. Thus Yolles and Kramer estimated on the available data that about 3 percent of all U.S. males who reach their fifteenth birthday will experience an episode of schizophrenic disorder before they die. European data based on more conservative criteria for hospitalization and for schizophrenic diagnoses lead to estimates of around 1 percent.

This risk is not the same for all groups of people. It is higher in the first-degree relatives of schizophrenic patients than in the first- degree relatives of other people. To study this difference a group of people are selected as control persons for some schizophrenic cases, matching for age, sex, and whatever else the investigator thinks relevant (from each patient's town's birth register, for example). Geneticists measure the extent to which secondary cases among the relatives of the "probands" (the schizophrenic cases) are greater than the risk among relatives of the controls. This is one

way of measuring the familial aggregation of cases of a disorder; but sharing some pathological genes is not the only way by which characteristics become clustered in families. Socially inherited characteristics (such as poverty, wealth, nationality) also tend to aggregate in families. Families tend to share a physical environment so that waterborne diseases also tend to aggregate in families. Members of the same household sometimes infect each other with viruses and bacteria, and households tend to be made up of related persons.

Insurance companies frequently use information about the risk of a condition to compute insurance premiums: A company using the 3 percent risk factor would try to get enough in premiums to average out the costs of benefits. Since first-degree relatives of schizophrenic cases are known to be at a much higher risk than other people, an insurance company might wish to use such a relationship to exclude relatives from their customers.

Beyond the Clinical Horizon

In the section on community diagnosis we saw how the fact that patients in mental hospitals on any day are older than the new admissions, gives the impression that the schizophrenic condition tends to be long lasting and progressive. If patients who leave the hospital are followed over the next few years one gets a different impression. These data show a very variable outcome, some patients deteriorating steadily, some showing varying periods

of remission with intermittent relapses, and others showing a nearly complete or complete remission after a first episode of severe symptom formation with a lifetime of successful functioning and no further relapses.

The very variable course schizophrenic disorders take, described by E. Bleuler in 1911, has been repeatedly confirmed by later investigators.

E. Bleuler's main argument for reformulating the concept of dementia praecox into the group of schizophrenias was his recognition that the natural course was more variable than Morel and Kraepelin had believed. Morel's concept of *démence précoce* and Kraepelin's fondness for classifying disorders on the basis of outcome may be thought of as arising from experience when the mental hospital was mainly used as an institutional placement of last resort and most of its patients failed to adapt to life. By the end of the nineteenth century, outpatient work had become more extensive and when clinicians such as E. Bleuler saw cases in this broader different clinical context their perception of the condition's course changed.

In a similar fashion, when investigators began doing follow-up studies, they exposed themselves to people with this disorder who had recovered and found that these recoveries were more common than had been thought by clinicians who only remain aware of the patients who remain in care or who return to care. Hence our picture of the schizophrenic syndrome's course and

of its distribution in the population changed as clinical services extended beyond the hospital's walls.

There is every reason to believe that as with many other conditions, such as tuberculosis, coronary heart disease, hypertension, and measles, pulling together the case records of the clinicians and clinical agencies serving a community will reveal a picture of the condition's frequency in the population, but that these clinical reports will only represent a portion of the total load of morbidity. There are several reasons for knowing this to be so. (x) Histories taken at the time of admission to clinical services reveal a period of time before seeking treatment when the new patient was clearly ill. During those months he was unknown to the clinicians and was not, in fact, anyone's patient. (2) Follow-up studies of people who have left treatment frequently reveal the existence of cases that a clinician would regard as examples of a schizophrenic condition, but at that point in time the person is no one's patient. (3) A limited number of morbidity surveys have been done, and each has added some cases previously not known to the clinicians serving the population surveyed. They have produced prevalence rates up to 900 per 100,000 in surveys in which household interviews were carried out by the investigators (that is, four times as high as reported prevalence rates for schizophrenic hospitalizations, which range up to about 200 per 100,000).

These figures give an approximate idea of the size of the schizophrenic

population lying below the clinical horizon. The reason why so little reliance can be placed in the data lies in the overall state of our knowledge regarding the schizophrenic condition. Being defined as a descriptive entity without clear margins, there is inevitable controversy regarding the identification of cases. The existence of such controversy is not a sufficient reason for regarding the condition being studied as subjective; many things are obviously objective but do not have sharp edges: storms, the earth's atmosphere, cities, mountains, the oceans, for example. Nor does our difficulty in defining the condition in words provide a good enough argument to convince one that the whole idea is without substance: We find it hard to describe odors and colors but that does not convince us that each of us is having a purely private experience when a rotten egg is opened or the sky changes color. Efforts to find cases of schizophrenic disorder in community surveys will continue as long as the syndrome remains a puzzle and a challenge to psychiatry. But we cannot expect to obtain too clear a picture with the rather elementary cameras now available.

There are two main reasons to wish for a clearer picture regarding the size of the schizophrenic problem. One is its relevance in the planning of treatment facilities. The extent to which this is a pressing need for knowledge depends on our view of the effectiveness of existing treatment techniques. If we think we have a very effective treatment that will relieve distress or prevent the progress of a progressive condition, we will want to find the early

and milder cases. The second motive for wishing to get a fuller picture of the size and distribution of the schizophrenic syndrome in human populations is the desire to discover causes. Morbidity surveys regarding schizophrenia have generally been motivated by this second need. For reasons that must be left to the social historian of psychiatry, these have mostly been efforts to measure the lifetime risk of developing the syndrome at some point in one's life as a consequence of an hereditary defect. The psychiatrists with a bias in favor of a genetic etiology have done the most to find hitherto unrecognized cases of schizophrenia in the community. Because they have tended to think of the condition as a result of an inherent characteristic of the individual, they have tended to see the variations in the clinical picture during the individual's lifetime as a methodological problem rather than as a topic for investigation. Hence, they have been content in their community surveys to ascertain people with schizophrenia either at the present or at any earlier time.

But we should recognize that household interviewing does not favor the location of people with schizophrenic syndromes if the individuals have not formerly been mental patients or had some other serious disruption of their social functioning. In fact, a rereading of Bleuler's monograph setting forth the clinical picture of the schizophrenias makes it clear that setting criteria for diagnosis in the absence of marked secondary signs and a current complaint would be extremely difficult. Studies have tended to count the number of people who have ever in their lifetimes manifested the

schizophrenic syndrome. Such studies can only answer the question, “What proportion of people born will at some time in their lives be regarded as schizophrenic?” The most carefully gathered data on this question are heavily dominated by episodes of psychiatric treatment, even when the entire population at risk was contacted personally. These figures are best thought of as an index of the frequency with which individuals are fated to exhibit a clinically recognized schizophrenic syndrome prior to their sixtieth birthday. If one thinks that the condition is due to some particular gene configuration as the key causal factor, one provides evidence that in the United States, where diagnostic criteria are broad, there is a 2 to 6 percent lifetime risk.

Because morbidity surveys yield so little information regarding the distribution of schizophrenic conditions in human communities, they are not dealt with further here. They have been competently reviewed elsewhere.

Because identification of the syndrome in the absence of treatment is not yet feasible, owing to the state of our concepts regarding its nature, some investigators have been tempted to postulate that all schizophrenic persons will ultimately turn up in the treatment statistics. While this is a convenient theory for those who have access to medical care statistics but cannot make the diagnosis in previously untreated people, it is not logically tenable as a scientific formulation of what the medical care statistics relate to.

Identification of Syndromes

The emergence of schizophrenia with a variable course from the concept of dementia praecox with a progressive dementing course was described above as a result of changing perspectives arising from an extension of clinical work and follow-up studies. The identification of a more heterogeneous, less stereotyped syndrome emerged out of this process. This is a good example of how epidemiology contributes to the identification of a syndrome.

This schizophrenic condition has been further modified in the course of time as the effects of institutionalism, later dubbed institutional neurosis by Russell Barton, became visible as a characteristic of some people who spend long periods in overly structured long-term mental hospitals, whether the condition for which they were admitted was schizophrenia or some other serious mental disorder. By recognizing some manifestations of withdrawal and deteriorated functioning as a consequence of a specially structured social environment, these complications were seen as less related to the schizophrenic disorder than previously.

Still later, a syndrome that generally starts outside of the hospital and often leads to hospitalization—extreme withdrawal or extreme aggressive behavior—became identified as common to many of the mental disorders, and its course was found to be fairly independent of the diagnostic type of

mental disorder present. When it was observed that this syndrome's course was highly influenced by the ways in which the delivery of health services was organized, it took on a name, "social breakdown syndrome," and its manifestations were in a sense also removed from the concept of schizophrenia as such. This process is described in Chapter 47, "The Social Breakdown Syndrome and Its Prevention."

Working of the Health Services

Health services are of value to the extent that they lower the prevalence of disease or disability or postpone death. Knowledge regarding whether health services succeed in lowering the prevalence of schizophrenia comes only from epidemiological information. At the present time, there is no technical basis for seeking to lower the prevalence of schizophrenia by lowering its incidence; no established techniques exist that, if applied, would lead to that expectation. Even if the number of new cases of schizophrenia that start each year (annual incidence) were to remain the same, if the average duration of the episodes could be reduced, the prevalence would also be reduced. There is some reason to think that the average duration of schizophrenic episodes is getting shorter. This was discussed earlier in the section on historical trends.

But even if the annual incidence and average duration of schizophrenia

are not being changed by the health services, the amount of disability associated with schizophrenia apparently is being reduced. Much of the disability in personal and social functioning associated with schizophrenia is preventable by organizing the delivery of psychiatric services so as to give close attention to the maintenance of the patients' health functions, minimizing any possible loss of self-esteem, and by maintaining and strengthening family and community ties. (See Chapter 47, "The Social Breakdown Syndrome and Its Prevention.")

One means of accomplishing this goal is by providing more informally accessible treatment closer to the patient's home, by making inpatient care more accessible and more flexible, and by reducing the periods of hospital care, even if this means repeated hospitalizations for some people. The principle behind this approach is that patients released back to community care as early as possible in their recovery will suffer less disruption of personal and community relationships; maintenance of these relationships is important for the prevention of disability.

Experience in trying to implement policies favoring shortened hospital stays and maximum flexibility in the use of inpatient and outpatient resources suggests that greatest success is achieved when a unified clinical team has continuing responsibility for patient care at all stages of care, regardless of whether the patient is in inpatient or outpatient care at any time. This unified

clinical team must be part of an inpatient service and be able to do its own after care and its own pre-care screening as well as have access to all the other related community services, such as day hospital, adult outpatient clinic, child guidance clinics, public health nursing services, and welfare agencies. Under these conditions, the number of outpatient schizophrenic patients becomes as large as the number of inpatient schizophrenic patients on the average day. In Duchess County, New York, where such a pattern was established in 1960, this approximate equality had occurred by 1969. At that time, the median stay of all admissions to inpatient care from Duchess County (age sixteen to sixty-four) had dropped to less than twenty-one days. At that time, for New York State as a whole the median stay was fifty-five days.

The unification of inpatient and outpatient responsibility in a single clinical team appears to affect the way in which staff members deal with specific patients. This is probably due to the fact that decision regarding patient placement is made by the same staff who will be responsible for dealing with the consequences of their decision. The indications for release of patients and for their readmission depend on judgment regarding the consequences of each decision. In many concrete situations, we cannot judge with certainty what will happen after a move is made or what will happen if no move is made. A unified clinical team is in a better position to make these decisions less binding on themselves and less binding on the patients. They are also in a better position to learn from their experience.

The Search for Causes

Knowledge of the distribution of schizophrenic disorders in human populations can presumably lead to new insights regarding causal mechanisms. In medicine, in general, the clues pursued by epidemiologists often arise from the observations of clinicians who observe a connection among their cases that strikes them as different from the patterns they have seen in other disorders. At other times, clues emerge from tabulations of routinely collected information, or the careful organization of information about the occurrence of an illness. It was the ophthalmologist Gregg who first called attention to an epidemic of congenital cataracts and linked this to an epidemic of rubella after a long-time interval between epidemics when people had been free of the condition. From this emerged our present knowledge of how fetal rubella affects the development of the eye, of the heart, and especially of the central nervous system. It was the anesthesiologist Snow who, after studying the history of records regarding epidemics of cholera, concluded that it must be a waterborne disease caused by a living organism and proceeded to a series of classic epidemiological studies of epidemics in progress that demonstrated this fact long before any bacilli had been visualized and been shown to be capable of causing disease.

Epidemiological studies of schizophrenia have been dominated by the frequent clinical observation that the condition appears to cluster in families.

Another series of studies arise from a notion that the schizophrenic condition might be accountable to a specific set of distortions of interpersonal communication embedded in the structure of human communities. This series of investigations began in Chicago in the 1930s and have been pursued vigorously since. They were described briefly in the section on working of the health services. Dunham summarized the state of these inquiries so well that it is unnecessary to recapitulate these investigations here.

The same line of reasoning has led to a large number of studies that are not specific for diagnosis but are related to mental disorder in general. These investigations cannot throw light on the distribution of one disorder as contrasted to other disorders because such inquiries do not sort the population by type of disorder. Schizophrenia epidemiology cannot exploit those data. This assertion does not, however, dispose of the notion that mental disorders as a whole might have an understandable distribution in the population. If symptom formation is looked on as a matter of choice rather than as an inherent product of the condition—the clinical diagnosis—it makes sense to sum up the causes of schizophrenic disorder with those of the other functional psychoses and neuroses. Some would exclude the organic psychoses and mental retardation from this summing process, but the evidence regarding symptom choices could be used to argue that mental disorder symptoms in the presence of brain disorder are not universal and that these are just as logical to include as not. These arguments that all mental

disorders might have a common epidemiology are not refuted by the known facts about the differential distribution of schizophrenia and manic-depressive psychoses as brought out, for example, by Faris and Dunham. These could simply reflect differential symptom formations in the presence of some fundamental disorder, as yet unidentified. Thus, we see that abstract arguments do not produce definite reasons for the pursuit of one course rather than the other. This is an example of situations where choices must be judged by their products. If an approach proves fruitful in elucidating a hitherto obscure issue, it will become the approach of choice until a still more fruitful one is found. Since none of these approaches has been outstandingly fruitful in giving us practical insights into the nature of schizophrenia, they will not be given further attention here.

The studies on gene theories of schizophrenia have been equally persistent and have repeatedly led to apparently conclusive results only to fall because of technical inadequacies of the studies or because the data do not conform to any defendable gene model. These two courses of inquiry, while not actually incompatible, take their starting point from two opposing clinical impressions regarding the nature of the condition. The social origin set of studies arises because it is so clear that schizophrenics continue to carry on normal mental functions in so many instances, even while grossly psychotic. In addition even after years of dilapidated, apparently totally destroyed personality functioning, schizophrenic patients, for no obvious

reason, begin to display normal mental functioning. Hence, it is tempting to think of schizophrenia as a response of the person to a current situation and as quite different from organic psychoses, which seem to take an entirely different course in that when functions are lost they stay lost.

In contrast, if attention is paid to the apparent unmodifiability and hopelessness of some schizophrenic patients, one is tempted to think that there must have been something wrong with them even before they became manifestly mentally ill. In many case histories, there are clues that point in this direction, suggesting the presence of a handicap even before any sign of disordered functioning was observed. Every large caseload contains many patients who are long-term problems for the care-taking staff. They may have intermittent short-term episodes of social breakdown syndrome and hospitalization, but between these episodes they exhibit serious, apparently un- correctable handicaps in living and maintain only a marginal social adjustment with more or less severe symptoms of disordered mental functioning. Some of these patients appear to be mentally retarded. Some are regarded as schizophrenic. Those sticky, incurable, either excessively inactive and dependent or intermittently misbehaving individuals who are seen as schizophrenic are so obvious to the working clinicians that it is tempting to regard their condition as due to an inborn defect of the organism.

Kety and his associates conducted the most serious and important

recent attempt to isolate genetic from environmental factors in the causation of schizophrenia. The record systems and social practices of Denmark provided them with a complete list of people who had been legally adopted by courts in the City or County of Copenhagen by biologically unrelated parents (N=5,483). The country's psychiatric register and other medical, social, and police and military agency records were searched, and 507 were found to have had admissions to psychiatric facilities; of these, thirty-three were judged to be schizophrenic by a set of criteria carefully worked out among the investigators. A group of thirty- three matched controls was selected from the list of adoptees who had not been admitted to a psychiatric facility. They were matched for age at adoption, age, sex, socioeconomic status, time spent with biological parents, time spent in institutions, and time spent with foster parents prior to adoption.

The index cases (schizophrenics) had 150 biological relatives (parents, siblings and halfsiblings), while the control group had 156. These were located in the population register, and then the psychiatric register and other records were screened for the same people. The records of all forty-seven relatives who had been in psychiatric facilities were reviewed carefully in a blind fashion, and sixteen were considered in a schizophrenic spectrum.

These sixteen cases among relatives called schizophrenic by the investigators in their blind diagnoses were unequally distributed between the

two groups of biological relatives: 3 were found in the 156 relatives of the control adoptees and 13 in the 150 relatives of the index, that is, schizophrenic, adoptees. The percentage odds against this distribution occurring through sampling variation are very great, more than 99 percent probability.

Because of the way different types of schizophrenic syndromes and other mental disorders were distributed among the two sets of relatives, the authors concluded that their data were "compatible with the thesis that the schizophrenia in the probands represents some polygenic inadequacy transmitted through heredity but receiving its ultimate expression and differentiation on the basis of a complex interaction among genetic factors or between them and the environment."

In this view, schizophrenia is not a simple Mendelian gene disorder, such as phenylketonuria or hemophilia. A single gene defect lays the ground for the whole schizophrenic spectrum syndrome. The available data force us to think of a dominant gene with a very low penetrance, that is, most people who have the defective gene never exhibit a schizophrenic syndrome. Furthermore, because of the relatively low fertility of known schizophrenics as compared to the rest of the population, this gene would tend to disappear over several generations, which the available evidence contradicts. There are two ways of maintaining a single gene defect in the presence of these facts:

(1) one can assume a fairly high mutation rate, leading to new, not inherited, cases; or (2) one can assume that the many carriers of the gene who do not exhibit the schizophrenic syndrome are more fertile than the rest of the population. If one is attracted to the alternative explanations available to account for the data, hypotheses become more complex and inherently more difficult to test. The difficulty in testing them and their complexity is not an overwhelming argument against them; the only strong argument is the razor discovered by William of Occam, the fourteenth-century thinker to whom is attributed the law of parsimony: Do not multiply hypotheses unnecessarily.

Another variety of causal theory associated with an inborn condition looks toward an injury during the period of fetal development.

The argument for considering this type of hypothesis is weightier than is generally recognized in psychiatric circles because the advances in knowledge regarding prenatal injury are not widely known among psychiatrists interested in schizophrenic research; they are more widely known and more attended to by people concerned with the causes of mental retardation and the causes of congenital anomalies, such as anencephaly, harelip, and clubfoot. The types of insults seem to be less important than the stage of embryological development when they act. Infections, injuries, toxins, and malnutritions have all been implicated in congenital anomalies. In general, the risk of these events rises with undesirable physical and social

environments, and this is presumably what the investigators using the Danish population controlled for when they selected their controls as coming from similar socioeconomic backgrounds. But if this possibility is considered, it may explain the higher frequency of deaths among the biological families of the index (schizophrenic) adoptees as compared to its frequency among the controls' biological families. The percentage odds against the possibility that the higher death rate among the biological mothers of the schizophrenic adoptees is owing to sampling variation are 94 percent. A larger proportion of the fathers died as did a larger proportion of the half-siblings. It is enough to make one uneasy about rejecting the possibility that the schizophrenia, in fact, being investigated was something emerging at least in part from unfavorable environments leading to central nervous system damage during fetal development. The fact that some of the excess deaths were suicides can be used to weaken this argument.

Neither the sociogenic theories nor the gene theories can be refuted by today's evidence. Nor can one reject the theory of a fixed defect arising after conception but early in life. On the contrary, each can be defended with some evidence. On review of the evidence, it seems fair to say that further progress will not be made by opposing these theories but will more likely emerge from a new kind of approach to the etiology of schizophrenia that either straddles these three notions or looks in an entirely new direction.

Since each of the three approaches assumes a long-standing difference in the organism, the obvious way of seeking to straddle these approaches is to seek a direct way to identify this difference through an examination of the organism at a level somewhere between the gene composition and psychopathological. The identification of such a difference between schizophrenic individuals and other people would immediately open a new door to further investigations with the hope of untangling the knot. If one could show that schizophrenic individuals have a difference (or defect) that is otherwise relatively rare in the population, one could then investigate the epidemiology of that difference. One would also be able to search for explanations as to why those people who have the difference and do not show schizophrenia are different from others with the difference. One could begin to search for cases of schizophrenic disorder that do not exhibit the difference and see if they have characteristics to distinguish them from other types of schizophrenic cases.

With so much promise, it is no wonder that biochemists have invested so much (as yet unsuccessfully) in trying to locate a metabolic difference common in schizophrenics but rare in other people. So have some psychologists. One promising line of approach is suggested by Belmont, Birch, and Belmont: A defect in the cross-sensory recognition of sensory stimuli, in the presence of intact intra-sensory mechanisms and of motor pathways and of intelligence, produces a set of defenses that can lead to schizophrenic

syndromes. This possibility has been investigated among childhood schizophrenics with reported positive results. But, of course, we do not know whether childhood schizophrenia is the same disorder as adult schizophrenia. Nor does there exist at present an adequate test for intersensory integration of stimuli among adults.

It is almost certain, at any rate, that the next step forward in unraveling the causes of the schizophrenic syndromes will be through either some such intermediate mechanism that can be ascertained or in an entirely new direction.

Conclusion

This review of current knowledge regarding schizophrenia's epidemiology deliberately started out with no attempt to define the condition being studied. In a stock-taking of our present state of knowledge it is a good approach, because it leaves us open to shifting views of its nature as we take account of the different evidence on the matter. In the conduct of a single piece of research this procedure would not be defendable. If others are to be able to interpret research data, the investigator needs to present the criteria used in that particular study as clearly as possible. Serious attempts to incorporate into our understanding the results of investigations conducted by others require that we master the investigator's methods and research

approaches to the maximum extent possible.

The nature of the group of schizophrenias cannot be settled by argument, but will in time become settled through careful, thoughtful investigations. On today's evidence, it is a good bet that this group of disorders will become broken into several subjects as our knowledge advances and that each subject will come to be seen as a manifestation of a condition that has other manifestations and that some cases of the condition will show no clinical pathology. Clinical investigations and laboratory investigations will play a part in this process. Epidemiological investigations may provide the key that unlocks the mystery (as they did with respect to pellagra, lung cancer, and fetal rubella) or laboratory investigations may provide the key (as they did in syphilis), or clinical investigations (as occurred in epidemic pleurodynia—Iceland disease).

Future epidemiological studies of schizophrenia will benefit from recognizing the fact that we now know that the epidemiology of inpatient care for this condition and the epidemiology of social breakdown syndrome associated with the condition are each distinct from the epidemiology of schizophrenia.

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