PRINCIPLES AND METHODS OF EPIDEMIOLOGICAL RESEARCH AND THEIR APPLICATION TO PSYCHIATRIC ILLNESS*

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MAY I begin by qualifying the title of this paper? I can only illustrate some broad principles and a few of the methods that are used in epidemiology today —there is no attempt to be comprehensive.

In general, there are two ways of studying sick people; the clinical, where the individual patient is the centre of interest, and the epidemiological, where patients are studied in relation to the populations and groups from which they have come. Laboratory methods can be used in both of these approaches. Epidemiology can be defined as "the study of occurrence and distribution of disease". Paul (1958) considers it to be "concerned with the circumstances under which diseases occur, where diseases tend to flourish and where they do not". The epidemiologist tries to find populations with differing rates of illness and then compares these populations and their environments with the hope of discovering possible causes of the disease. His aim is to show how modifications of behaviour or of the environment may prevent illness; epidemiology is, in fact, the basic science of preventive medicine.

In his recent book, Morris (1957) has defined seven "uses of epidemiology". I am only going to discuss principles and methods with reference to one of these uses—but perhaps the most important of them—the "search for causes" of disease. Some of the studies described are far removed from your specialty, but this way of looking for causes of disease began with less complex conditions than psychiatric illness. I shall begin with one or two examples of how epidemiology was used in the past; these may help you to appreciate what it is trying to do today and to see how it can be applied to problems of psychiatric illness.

I. AGENT AND ENVIRONMENT

Snow on Cholera. Epidemiology originally meant the study of epidemics and my first example is a classical investigation carried out in London by Dr. John Snow in the nineteenth century (Commonwealth Fund, 1936).

By studying the epidemics of cholera in London in the eighteen-forties, Snow, a clinician and anaesthetist, became convinced that polluted water supplies were mainly responsible for the outbreaks of disease. At this time there were many water companies supplying London and most of them drew their water from the heavily polluted Thames. There was a period from 1849 till 1853 when there was no cholera in London and during this time the Lambeth Water Company moved their waterworks upstream to Thames Ditton and so obtained

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relatively pure water. But the parts of London supplied by this company were also supplied by the Southwark and Vauxhall Company who drew their supplies from the river at Battersea—a very polluted part of the Thames. These circumstances gave Snow a chance to test his hypothesis about the spread of cholera—it was an "experiment of opportunity", a "natural experiment"—and when this occurs, epidemiologists should be quick to use it.

An epidemic of cholera occurred in the summer of 1853 and Snow was able to show that in districts supplied only by the Southwark and Vauxhall the death rate was 114 per 100,000 while it was 60 per 100,000 in districts supplied by both companies; but there were *no* deaths at all in those districts supplied exclusively by the Lambeth Company—a nice gradation. This is an *ecological* or an *indirect* study—that is to say the occurrence of disease was related to a characteristic of an area, in this case the water supply. The indirect study only gives a lead to causation, since it cannot show any direct connection between an individual drinking polluted water and contracting the disease. In addition, the districts with different supplies may also have differed in many other ways, for example those with the "good" supply may have been wealthier and less overcrowded—and in fact to a certain extent this was so. The results then were at best only suggestive of a causal relationship between water supply and cholera. However, in 1854 Snow clinched the argument. He had discovered that in the districts where both companies supplied water, the companies were intermixed in almost every street, so that in every type of neighbourhood there were some houses supplied by the Southwark and Vauxhall, some by the Lambeth Company. When an epidemic occurred in July, he obtained the addresses of everyone who died from cholera in these districts of London where both water companies were operating and personally visited their houses to discover which supply was taken. The results are shown in Table I, which is an abbreviated version of one of Snow's Tables. In it he listed all the districts supplied by both water companies, gave their populations at the 1851 Census and the number of deaths in each district in the 4 weeks ending on 5 August, 1854. He then divided these deaths according to the water supply of the household. The preponderance of cases in houses supplied by the Southwark and Vauxhall Company is obvious, but another step was necessary.

TABLE I

Mortality from Cholera in the Four Weeks Ending 5 August, 1854

Sub-Districts	Population in 1851	Deaths in 4 Weeks Ending 5.8.54	Water Supply				
			Southwark and Vauxhall	Lambeth	Pump-wells	Thames	Unknown
St. Saviour, Southwark ..	19,709	26	24	–	–	2	–
St. Olave, Southwark ..	8,015	19	15	–	–	2	2
St. John Horsleydown ..	11,360	18	17	–	–	1	–
.. .. ..	..	..	..	..	..	..	..
.. .. ..	..	..	..	..	..	..	..
Borough Road ..	15,862	21	20	1	–	–	–
.. .. ..	..	..	..	..	..	..	..
.. .. ..	..	..	..	..	..	..	..
Kennington (1st)	24,261	11	9	1	1	–	–
.. .. ..	..	..	..	..	..	..	..
.. .. ..	..	..	..	..	..	..	..
	486,936	334	286	14	4	26	4

Adapted from *Snow on Cholera*, 1936. New York: Commonwealth Fund,

He went on to find out the total number of houses supplied by each company and so was able to work out rates—Table II. The rate of cholera

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deaths per 10,000 houses was 14 times greater among those supplied by the Southwark and Vauxhall than among those supplied by the Lambeth Company. A final point from this table was that the rate in Lambeth-supplied houses (5 per 10,000) was lower than that in the rest of London (9 per 10,000) although the Lambeth houses were in the area of greatest incidence of the disease.

TABLE II

Mortality from Cholera in the Four Weeks Ending 5 August, 1854

Water Company	No. of Houses Supplied	No. of Deaths in 4 Weeks Ending 5.8.54	Rate per 10,000 Houses
Southwark and Vauxhall ..	40,046	286	71
Lambeth	26,107	14	5
All others	287,345	277	9

Adapted from *Snow on Cholera*, 1936. New York: Commonwealth Fund.

Snow, therefore, had been able to relate the occurrence of disease to *individual* or *direct* characteristics—the consumption of polluted water—not just to *ecological* or *indirect* ones, and, more importantly, to do this after reducing the effects of wealth and poverty, or in technical terms—correcting for the correlated biases. A direct study is better than an indirect one, but something more is required, other environmental factors must be accounted for. In real life situations, random allocation to two groups is impossible, but some way of getting as near as possible to this ideal is essential. Because the houses with different water supplies were so intricately mingled, Snow's natural experiment achieved the effect of almost random allocation to the two companies and this makes his results especially valuable.

Here then was a completely convincing exposition of how cholera was spread, and therefore how it might be prevented; moreover Snow indicated where the specific agent causing it was likely to be found, thirty years before Koch discovered the cholera vibrio.

Goldberger and pellagra. The next example goes further and works out in greater detail the causes and prevention of a disease—pellagra. Pellagra is coming nearer to your immediate interests, since it includes among its manifestations a well-marked psychosis. In 1914 pellagra was common in some Southern States of the U.S.A. and was still considered to be an infectious disease. In fact, however, Bacelli in Italy at the end of the 19th century had related its occurrence to the growing of certain crops and had been able to diminish its incidence by a law requiring the rural population to practise rotation of crops (Sand, 1952). Goldberger (1914, 1920), of the United States Public Health Service, carried out an epidemiological study of the disease by observing in detail the conditions under which it flourished.

He first studied the occurrence of pellagra in mental hospitals and showed that, although many patients were admitted suffering from pellagra and many others developed the disease after long stay in the hospital, no case had ever been recorded among the staff. In view of the intimate contacts between patients and staff it was clear that this could not be a contagious disease. He thought that dietary differences between patients and staff might be responsible and he arranged that a colleague should try the effects of an improved diet as the sole treatment of a group of women with pellagra in a mental hospital. The results showed a high cure rate (Lorenz, 1914). Goldberger

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then studied other institutions where pellagra was common. In one orphanage, for example, pellagra was only seen in the children aged 6 to 12. Those under 6 were given two mugs of fresh milk daily, while those from 6 to 12 had no milk and a diet which included fresh meat only once a week. To test his hypothesis, he carried out a simple planned experiment—this is not often possible in epidemiology. All the children were given milk every day and fresh meat four times a week, with the result that during the year of this experiment no case of pellagra occurred. In a similar experiment in a mental hospital, Goldberger found that no new case or recurrence occurred amongst a group of patients during the course of a year on special diets, while 47 per cent. of the patients in a control group had a recurrence of the disease. After further studies, including the experimental production of pellagra in a small number of convicts, Goldberger and later workers were able to give an explanation of the disease in relation to the vitamin B complex, in particular nicotinic acid, and to the action of tryptophane. Epidemiology had been as useful in uncovering the causes of pellagra as of any infectious disease.

Morris (1957) quotes in his book another interesting example of how epidemiology can be used with clinical and laboratory methods to solve a problem. A condition of epidemic dropsy was recognized in Bengal and differentiated clinically from beri-beri and hunger oedema. Epidemiological study showed that outbreaks occurred in groups, especially among middle-class people, and in particular villages and households. Diet was implicated and the disease was found to be associated with curries prepared with mustard oil. Laboratory studies were then able to show by animal experiments and with human volunteers that the disease was a poisoning caused by an oil from Mexican poppy seeds contaminating the mustard oil.

In these examples the agent causing the disease, either by its presence or its absence, was quite specific and was a necessary and apparently sufficient cause. That is to say, very many of those subjected to it in sufficient dosage contracted the disease. In these circumstances, epidemiological studies, by concentrating on the environment and ways of living of the patients, could produce strong leads to the causes and ways of preventing the disease. But this is a rare state of affairs in medicine. Usually other factors such as the susceptibility of the patient are also very important. Thus it was recognized long ago that the tubercle bacillus, although a necessary cause of tuberculosis, is not at all a sufficient one. The state of resistance of the person infected, derived from his heredity and previous experience, is very relevant. The epidemiology of tuberculosis, then, must take into account host factors as well as the agent and the environment.

II. Host, Agent and Environment

Poliomyelitis. One of the best examples of a disease with a specific causative agent but where the susceptibility of the host is all-important is poliomyelitis. Epidemiology has had a difficult task here, because the virus is widespread and susceptibles apparently few and far between. Burnet (1953) has shown how study of the age incidence—that is the number of cases occurring per 1,000 of the population at risk by defined age groups—in various epidemics has helped our understanding of the disease. For example, when paralytic poliomyelitis was first recognized here at the end of the 19th century it was a disease of babies—hence "infantile paralysis". In succeeding outbreaks the incidence among very young children has lessened while more and more cases have

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occurred in older children and young adults. However, when an epidemic occurred in Malta in 1942, incidence was highest among babies, while a similar epidemic in the isolated community of St. Helena in 1947 had a maximum incidence among young adults. It is known that young adults are the most susceptible age group when infection with a new virus is introduced into a community. (This was shown clearly in the influenza epidemic of 1918–19.) From these and other facts, a theory has been built up linking the appearance of epidemics of poliomyelitis with better sanitation—and therefore with *less* chance of infection. It is postulated that infection with the virus in this country was practically universal a hundred years ago. As sanitation improved slightly, infection became less common and infants were the first age group to lose their immunity and thus to develop the disease when infected. As sub-clinical infection became progressively less widespread, it became possible to reach adult life without developing any immunity, and thus to contract the disease when infection did occur. In Malta, sub-clinical infection had always been widespread and the epidemic developed like those here fifty years ago. In St. Helena the virus was probably introduced for the first time to a susceptible community and young adults suffered most. This theory that the better the environment, the more likely is disease to follow infection, is borne out by the Registrar General's Occupational Mortality figures for 1949–53. The mortality ratio of social classes I and II, professional and business people, is much higher than that of the other classes.

I have mentioned poliomyelitis to illustrate two points. First, that even in a disease where the relationship between causal agent and the occurrence of clinical illness is complicated, such simple measures as age incidence can help in understanding the causes of the disease. Secondly, to show a general way of organizing an epidemiological enquiry under the headings of "time", "place" and "persons". The changing age incidence of disease in *time*, the differing age incidence in *places*, and the differing incidence amongst *persons* in different social classes all helped to clarify the natural history of poliomyelitis.

Some years ago Ryle (1942) made a plea for wider concepts of aetiology than that of a specific cause of a disease. He pointed out that the great bacteriological and clinical discoveries had led to a search for "single determining causes where none exist", and that even in such acute diseases as lobar pneumonia, many associated causes in addition to the pneumococcus play a part. A similar plea has been made by Galdston (1954). This realization that knowledge of a specific causative agent does not fully explain the causation of disease and that in many diseases there may be no such agent makes it essential to study the circumstances where "disease tends to flourish". This surely applies even more to psychiatric than to other illnesses. Much psychiatric illness, like coronary heart disease, probably has no specific causative agent, and the appearance of clinical disease depends upon the interaction of factors in the host and of factors in the environment. In this type of disease, susceptibility is no longer as simple as in poliomyelitis, but is probably a complex affair of genetic and other factors.

III. Host and Environment

Durkheim and suicide. One of the early studies of this kind of condition was Durkheim's classical work on suicide. He was investigating the social factors in the environment that appeared to be associated with suicide and I should like to consider one of his methods in detail.

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Durkheim (1951) used figures available to him in official publications —he did not carry out surveys himself. This is often an early step in epidemiological research and it may provide clues that can be followed up by special surveys.

Durkheim was interested in the relationship between religion and suicide. He found that the predominantly Catholic countries had a much lower suicide rate than the mainly Protestant ones with the mixed countries in between. However, the Catholic and Protestant countries differed in many other ways besides religion—how then was he to test whether religion was in fact the important factor? He does so quite simply by testing the relationship within countries. For example, Bavaria was the German state with the lowest proportion of suicides and the lowest proportion of Protestants. *Within* Bavaria, the relationship holds good within provinces. Similarly he analysed the figures for Prussia and the Prussian provinces. The analysis in Switzerland introduced a third factor—language, since both French and German speaking areas contained some districts that were largely Catholic and others that were mainly Protestant. He was therefore able to show that suicide was higher where there were more Protestants, holding nationality and language constant. In fact he carried out seventeen of these essentially independent comparisons between Catholic and Protestant areas: such independent comparisons are called "replications". In every case the Protestant areas had higher suicide rates. The odds against this finding being due to chance are about 100,000 to 1. Here is where statistics makes a no doubt unwelcome appearance, but there is no escaping the fact that statistics play an important part in epidemiology. Tests of significance—that is to say ways of working out the odds that, for example, the difference between the suicide rates of two groups is due to chance or not, can strictly only be applied to groups that have been made up at random. But it is impossible to assign Catholics and Protestants at random to two countries and a simple significance test between the suicide rates in Catholic and Protestant countries could tell us nothing. There are far too many unknown factors that may account for the difference. However, Durkheim's method of replications avoids many of these difficulties, and the fact that the same result is found in seventeen independent comparisons within countries and provinces gets rid of most of the correlated bias and makes it possible to work out the odds against chance very simply. This again, like Snow's first study, was an indirect or ecological one, but Durkheim was able to obtain figures of suicide rates amongst Protestants and Catholics in various countries and show that using these *individual* rates, the direct method, the same preponderance of suicide among Protestants was present.

Durkheim did not find a "natural experiment" to simulate random allocation of people to the two religions but instead he used the method of replication to minimize correlated bias. His results do not demonstrate a "cause" of suicide, as Snow demonstrated a "cause" of cholera, but they do imply a strong link between suicide rates and particular religions and give clues to environmental factors favouring suicide. There is a most interesting discussion on this and other points about Durkheim's methodology in a recent issue of the *American Journal of Sociology* (Selvin, 1958).

Morris and coronary disease. A similar method was used by Morris and his colleagues (1953) in considering the relationship between physical activity of work and coronary heart disease. Very briefly, Morris found that drivers of London's double-decker buses had a higher sudden death rate from coronary

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disease than conductors. He considered three possible explanations—that drivers were constitutionally different to conductors, that there were different nervous stresses involved, or that the different physical activity of the two groups was responsible for their different mortality rates. Physical activity seemed to him the most likely explanation. (Significance tests cannot help in deciding whether or not physical activity *was* the important factor.) He went on to compare the rates in postmen and clerks in the post office and again found the physically less active group—the clerks—to have higher rates. Next, he went back to the published death rates by occupations in the Occupational Mortality volume of the Registrar General's Decennial Supplement for 1930–32. The men were divided into three groups of heavy workers, light workers and an intermediate group and again he found that the light workers had the highest rates. Such independent checks of his hypothesis make it likely that physical activity is in fact the important factor in the difference found between driver and conductors. By replication, then, Morris was able to test his hypothesis in spite of the fact that it was not possible to assign men at random to active and inactive jobs. Since then, a completely different approach —a national pathological study—has corroborated the hypothesis that physical activity at work is protective against coronary heart disease (Morris and Crawford, 1958).

Place of statistics. I should like to digress here to introduce a definition of statistics from a recent American text book (Wallis and Roberts, 1957), a definition that is at any rate more polite than many of the well-known ones. It is this—"Statistics is a body of methods for making wise decisions in the face of uncertainty". The authors point out that most scientific work consists of observation, the formulation of hypotheses, prediction of the results in certain circumstances if the hypothesis is true, and the verification of the prediction. Statistical methods are necessary in two of these processes—in the observation stage they show how best to collect data and in verification they determine whether new facts observed are consistent with the predictions and therefore with the hypothesis. It is at this stage that significance tests are used and that decisions are made on their results. The usual uncertainty is whether or not a hypothesis is "corroborated" or "falsified" by the new data and here statistical methods help towards "a wise decision".

Since epidemiology necessarily involves the use of numbers and rates and the comparison of rates between groups, a knowledge of broad statistical principles is essential. This does not mean that you must know all the tricks of the statistical trade before carrying out an epidemiological study, but simply that you must understand the basis of statistical reasoning. Both Snow and Durkheim were working before statistical theory had been developed but both were using essentially statistical reasoning and coping with "correlated bias" very successfully.

Schizophrenia. I shall conclude by discussing one or two applications of epidemiology in the study of schizophrenia—an illness where, like coronary disease, the concept of a specific agent is most unlikely. Schizophrenia has been defined as "a group of genetically determined, though environmentally precipitated, biochemical disorders" (Tait, 1957). This definition is a useful one for epidemiologists if we recognize that "biochemical" is still controversial and that "environmentally precipitated" covers a wide range of circumstances from early life to recent and current environment. In schizophrenia, then, the "host" factors probably include both the genetic constitution of the

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individual and his experiences earlier in life, while the clinical manifestation of the disease may possibly be precipitated by current environmental factors. It can often be difficult to decide whether a particular environmental factor is operating as a cause or whether individuals subject themselves to that factor because in some way it attracts those who are susceptible to the disease. For example, Ødegård (1936) made a careful study of the first admission rates for schizophrenia of Norwegians in Norway, and of Norwegians who had emigrated to Minnesota. He found that the admission rate was twice as high among the emigrants compared to those who stayed in Norway. On the face of it, this suggests that the difficulties facing an immigrant in a strange land are factors precipitating schizophrenia. But he went further and showed (1) that the age at onset was the same in both groups and (2) that there was no tendency for the disease to show itself during the first five years of the patient's life in America. The suggestion is, therefore, that emigrants include a higher proportion of susceptibles than the rest of the population and not merely that the emigration precipitates the disease. Similarly, is the well-known preponderance of schizophrenia among single men due to the factor of marriage "protecting" men against the disease, or is it because few susceptibles marry? Ødegård (1953) has produced some evidence which is summarized in Tables III and IV. Table III shows the very great difference between the admission rates of single and married men and women. Table IV shows that the widowed have admission rates not much in excess of those for the married but that the divorced have rates very similar to those for single people. Ødegård argues from these figures (and from clinical studies) that marriage is *not* in fact protective and that the explanation is that susceptibles rarely marry: if they do, the marriage is often unsuccessful.

TABLE III

Schizophrenia

Annual First Admission Rates per 100,000 in Norway, 1931–45 Standardized Rates, Ages 20–89 Years*

	Single	Married	Ratio of Single/Married
Men	534	124	4·3
Women	495	133	3·7

* Ødegård, Ø. (1953).

TABLE IV

Schizophrenia

Annual First Admission Rates per 100,000 in Norway, 1931–45 Standardized Rates, Ages 30–69 Years*

	Married	Widowed	Divorced	Ratio of Divorced/Married	Ratio of Widowed/Married
Men	101	189	501	5·0	1·9
Women ..	137	159	486	3·5	1·2

* Ødegård, Ø. (1953).

You will notice that the figures given in these Tables refer to first admissions to hospital. In the psychiatric field, statistics of first admissions to mental hospitals are being used in the same kind of way that mortality statistics are used in, for instance, coronary disease. That is, as a first approximation to the real *incidence* of disease, acknowledging that diagnostic difficulties and selection

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of patients will undoubtedly bias these figures in many ways. (*Incidence* is defined as the number of new cases of a disease arising in a known population within a stated interval of time. *Prevalence* is the number of cases present in the population during a stated interval.) Allowing for this bias, first admissions of men with a diagnosis of schizophrenia give the only data at present available in this country on the incidence of the disease. Incidence is a fundamental measure in the epidemiological "search for causes" since much of this search consists of comparing the number of new cases of disease occurring in various sub-groups of the population. Until better figures are available we must work with these admission statistics.

There have been many epidemiological studies of schizophrenia, especially in the United States and in Scandinavia, and these can be considered under our headings of time, place and persons.* For example, under "time", Ødegård (1954) has demonstrated how the incidence fell in Norway during the war, while there are various studies (e.g. Eaton and Weil, 1955) demonstrating the different incidence in "places" with widely differing cultures. The development of these kinds of studies may well give clues to environmental factors influencing the disease. Under "persons", there has been a good deal of interest in the *social class* of schizophrenic patients. You will all be familiar with the American studies, especially those of Faris and Dunham (1939) in Chicago before the war and Hollingshead and Redlich (1958) in New Haven very recently, that have shown how the incidence of schizophrenia is higher in slum districts of cities and among the unskilled classes generally. In this country, Brooke (1958) has shown that first admissions of men with schizophrenia are much higher amongst men in social class V—the unskilled labourers—than in the other classes. Hollingshead and Redlich also showed that the schizophrenic patients in their social class V† came from families of this class. In other words, their childhood environment had been that of the poorest section of the community. If this finding could be confirmed in this country it would be of great importance because here would be a lead to the kind of environment that might be helping to produce the disease in susceptible individuals.

IV. Work in Progress

Two of my colleagues, E. M. Goldberg and D. H. Allcorn, began to look into this question of social class and schizophrenia by first considering the quality of the data on which the Registrar General's figures were based. It was possible that the patient's occupation as recorded in the mental hospital was inaccurate and that this was the cause of the apparent excess of men in social class V. A pilot study was carried out at Long Grove Hospital in 1956 and the occupational histories of 40 consecutive male admissions between the ages of 15 and 35 with a diagnosis of schizophrenia were investigated. It was found that the classification of their jobs on the cards sent to the General Register Office corresponded with the facts as ascertained by interview with the patient and by visits to their parents and that there was a characteristic concentration of admissions among unskilled workers.

* For a more detailed review of some of these studies, see Morrison (1958).

† In this country social class is a purely occupational classification. The five classes are made up as follows: I—professional and senior management, II—junior management, shopkeepers, III—skilled workers, both manual and non-manual, IV—partly skilled, V—unskilled workers. Hollingshead and Redlich (*loc. cit.*) use a more complicated "Index of Social Position", which includes occupation, education and address, to allocate individuals to their five classes. The two systems are therefore not directly comparable; though of course occupation is highly correlated with education and place of residence.

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The pilot series lent little support to the notion that the concentration of admissions among semi-skilled and unskilled workers reported by the Registrar General is due to their downward "drift" either before the first admission to a mental hospital, or later. Most patients in fact had stayed in the same occupational grade throughout their "careers".

In the course of visiting the families of the 40 patients in East London, Goldberg (1959) noted that nearly half of the patients' homes had been "broken" during their childhood, either through parental death or various kinds of prolonged separation. A further sizeable proportion of cases showed considerable disturbance or serious conflict in family relationships. Much gross psychiatric disorder was reported among the members of the families. These observations in conjunction with the fact that the concentration of young schizophrenics in the lowest social classes was not the result of *individual* occupational drift suggested that there might be a downward drift from one generation to another due to the operation of multiple factors, genetic and environmental.

E. M. Goldberg is now carrying out a comprehensive study of the families of young men admitted to Goodmayes Hospital in Essex. She is gathering systematic information about many aspects of the patients' families, including the occupations of grandparents, parents and siblings, the family structure and family relationships, and details of mental hospital admissions and gross mental abnormality in the family.

When we looked at the occupational distribution of the first 20 patients at Goodmayes we found the usual concentration in unskilled jobs, but the fathers of the patients had a much wider range of occupations. In fact, they seemed to represent a reasonable occupational cross-section of the population at the time of their sons' admission to a mental hospital, and there was a definite drop in social class from father to son. It was possible that the sons were too young to have reached their "final" occupational status and that the fathers were in lower social classes when they were the present age of the sons.

We decided to test the hypothesis that there is a real drop in social class from father to son, taking into account these two factors, by using data in the General Register Office from the whole of England and Wales. The hypothesis, then, was as follows: "male first admissions to mental hospitals with a diagnosis of schizophrenia show a marked excess in social class V. This excess is found among men aged 16–24 years but it is also present among men aged 25–34 years, who should have reached their 'final' occupation. The fathers of these men, however, when they were aged from 20–44 years (that is, at the time of the birth of the patients), showed no concentration in social class V but instead had the same social class composition as the population as a whole".

The General Register Office was asked, and very kindly agreed, to extract the cards* of men aged 20–34 years who were first admitted to mental hospitals in England and Wales in 1956 with a diagnosis of schizophrenia. Data on these cards included the patient's date of birth, his occupation and the address from which he was admitted. The birth certificate of each patient was then sought. When found, this gave the occupation of the father when the patient was born and the address at that time. Admission cards for each year are filed alphabetically and a pilot study was carried out in which the first 286 patients were selected. Of these 232 birth entries were found (81 per cent.). A large proportion of the 54 patients whose birth entry could not be found had foreign names.

* These are the cards sent to the General Register Office by every mental hospital for every patient admitted.

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The extraction of the admission cards and the tracing of birth entries were carried out in the General Register Office and the results were given to us only after names and addresses had been removed.

The results for the 232 patients born in England and Wales were analysed in two age groups, 20–24 years on first admission and 25–34 years. There were only 60 patients aged 20–24 years and the numbers are not sufficient to "corroborate" or "falsify" the hypothesis. The results for patients aged 25–34 years are shown in Table V.

TABLE V

Social Class Distribution of Schizophrenic Patients and Their Fathers
Male First Admissions Aged 25–34 Years
England and Wales, 1956

Social Class	Patients		Fathers	
	Observed	Expected*	Observed	Expected†
I	3	5	6	4
II	11	20	16	20
III	74	93	90	88
IV	28	25	31	31
V	45	18	27	27
Total	161	161	170	170
Not stated	11	—	2	—

* From 1951 Census distribution of men aged 25–34.

† From 1931 Census distribution of men aged 20–44.

This table shows the expected excess of patients in social class V (45 observed, 18 expected on the basis of the social class distribution of men aged 25–34 years in the 1951 Census) and it shows that the fathers of these patients conform very closely to the social class distribution of men aged 20–44 in the 1931 Census. This small sample study thus corroborates the hypothesis. Table VI illustrates the movements in social class between father and son. The numbers in italics are those patients who are in the same social classes as their fathers.

TABLE VI

Social Class Distribution of Schizophrenic Patients and Their Fathers
Male First Admissions Aged 25–34 Years
England and Wales, 1956

		Social Class of Fathers					
		I	II	III	IV	V	Total
Social Class of Patients	I	*1*	2	—	—	—	3
	II	2	*2*	6	1	—	11
	III	2	5	*46*	10	11	74
	IV	–	3	12	*10*	1	26
	V	–	2	21	9	*13*	45
Total		5	14	85	30	25	159

Numbers in italics are of patients who are in the same social class as their fathers.

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These results are very different from those of Hollingshead and Redlich (*loc. cit.*), who found that 91 per cent. of their schizophrenic patients were in the same class as their parents and that the parents, therefore, showed the same preponderance in class V as the patients.

We hope to analyse a further sample of about the same size in order to have sufficient numbers of men aged 20–24 years to test our hypothesis, and to provide another test of the results for men aged 25–34 years. In addition, we are in process of analysing the residential data for men of 25–34. First results indicate that the expected excess of patients from large towns is present but there is also an excess *born* in the large towns. In other words, the concentration of patients from the cities may not be explained by immigration from rural areas.

We are planning to check the results of the birth certificate enquiry and to obtain more detailed information about the socio-economic background of young schizophrenic patients by asking every mental hospital in England and Wales to complete a questionnaire on six consecutive male first admissions under the age of 35. We do appreciate how difficult it is to fit in this kind of extra work in hospital and we are cutting the questionnaire to the barest possible minimum. However, there is no other way of getting this information about a national sample of patients and we feel that it is important to clear up this question of whether or not a particular social environment during childhood is related to the later development of schizophrenia. We hope that this Association will give us their blessing before we approach medical superintendents.

We are thus using three complementary methods of investigation: (1) intensive clinical case studies of an unselected, consecutive series of patients and their families; (2) an extensive survey of a national sample of patients; and (3) national "vital statistical" type information. The clinical study suggested various hypotheses, and one of these has been tested by using information already available in the General Register Office. The hypothesis, now modified, is being further tested by an extensive survey.

Clinical study has therefore enabled sociological hypotheses to be formulated, but epidemiological methods are necessary to test them, since the intensive case study can only be carried out on small numbers of patients who cannot be representative of patients in the country as a whole.

V. Conclusions

I hope that these examples of how epidemiology has been used in the past and one illustration of "work in progress" today will help to enlarge your interest in epidemiological principles and methods. I am sure that the other papers to be given today will illustrate methods that have not been mentioned in this paper. Perhaps by the end of the day you will agree that epidemiology can be of use to you in your search for the causes of psychiatric illness.

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