

is schizophrenia universal? an open question

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“ . . . insanity belongs almost exclusively to civilized races of men: it scarcely exists among savages, and is rare in barbarous countries.”

J.C. Prichard, *A Treatise on Insanity*, 1835

Is schizophrenia universal? A whole generation of psychiatrists is being trained to assume that it is. The question is not even asked anymore as it was 20 years ago. The universality of schizophrenia is now widely considered to be an established fact.

When textbooks of psychiatry deal with the question at all, it is usually in a tone of dismissal: “Sociocultural studies have demonstrated that schizophrenia occurs in both primitive and civilized societies and that incidence rates, when properly evaluated, do not vary significantly” (Knox and Tourney 1965). Schizophrenia is said “. . . to have a world-wide distribution” (Crocetti and Lemkau 1967), and to have been recognized “. . . in all cultures in which mental disorders have been subjected to thorough study” (Noyes and Kolb 1963).

In fact, however, there is no evidence upon which to base a belief in the universality of schizophrenia. The studies which have been used to support this belief are found, on careful examination, not necessarily to point in this direction at all. If anything, they may lead to the opposite conclusion: Schizophrenia may *not* be a universal disorder.

In an attempt to reopen the question of schizophrenia’s universality, this paper will review the pertinent original studies. It will make an important distinction in the concept of universality between westernized parts of developing countries and nonwesternized parts. To say, for instance, that schizophrenia occurs in Nairobi, the westernized capital of Kenya, says nothing about its occurrence (or absence) among tribesmen around Lake Rudolf, where there has been minimal influence by Western civilization. If sharp differentials exist in the occurrence of

schizophrenia among cultures with varying degrees of westernization, then it may provide us with clues to the etiology of the disease. This appears to be the situation.

The paper will not attempt to go beyond reopening the question. It will not examine the implications for various theories of schizophrenia’s etiology if the disease is not universal. “Westernization” is used loosely to indicate technology which is usually introduced by Western influence. It includes the appearance of such commodities as prepared foods, drugs, cigarettes, and basic supplies for the household, and also the introduction of advanced education and such medical practices as widespread immunization. Many of these elements could theoretically furnish clues to the etiology of schizophrenia.

Most of the studies under discussion define schizophrenia in terms that are comparable to the dementia praecox of Kraepelin. In more modern terms, these authors are referring to “process” schizophrenia, which includes individuals who become psychotic in late adolescence or early adulthood, and whose onset of illness is insidious and without major apparent precipitating factors, whose symptoms include a depressed or flat affect, and whose course is one of intermittent confinement or hospitalization. Cases of “process” schizophrenia may usually be differentiated from the “reactive” type who have a more acute onset, major precipitating factors, and a better prognosis.

Early Studies

Early interest in the question of whether schizophrenia is universal was shown by Kraepelin (1919). Shortly after the turn of the century, he visited mental hospitals in Singapore and Indonesia. He claims to have found cases of dementia praecox in two institutions and, on the basis of that observation, concluded that it was a universal disease.

A major limitation of Kraepelin’s observations is that they were confined to hospitalized cases. In developing countries of the world the only

individuals who are likely to be in a mental hospital are those who have had sufficient contact with the government to have called attention to themselves. Then, as now, mental hospitals were often used interchangeably with prisons. In Singapore or Djarkarta, both colonial outposts of long standing by the time Kraepelin visited, the people who would most likely have been hospitalized were houseboys, laborers, and civil servants—the people who would have had the most contact with incoming Western technology. This is a criticism that may be leveled at all studies in this field which rely on hospitalized cases—the fact of being hospitalized in itself indicates a strongly preselected group of people. The hospitalized population is not random, but rather is drawn from the population of the hospital's immediate surroundings (usually the capital city). This population has been exposed to many of the same aspects of Western technology as the people in more developed countries.

It is customary to dismiss the concentration of urban dwellers among hospitalized schizophrenics in developing countries. According to this line of reasoning, case finding is easier in urban areas; one assumes that rural schizophrenics have escaped hospitalization simply because they haven't been found. An alternative explanation which must be kept in mind is that there is a true difference in the prevalence of schizophrenia between the urban (technologically developed) portions and the rural (undeveloped) portions.

Margaret Mead (1949) was the next person to make observations of mental illness among people in a developing area. During her field work in Samoa about 1927, she described all those people who appeared "mentally diseased" among the 2,000 persons with whom she was working. She recorded two people who she said were clearly mentally deficient, one man with delusions of grandeur, and one 14-year-old boy who "relatives insisted had always been stupid but only recently became demented." Dr. Mead says the boy gave the "external picture of catatonic *dementia praecox*." She supplies no further information to allow us to differentiate this diagnosis from congenital or organic brain dis-

ease so the case must be considered to be in doubt. Furthermore, this group of Samoans had been missionized for almost a century and, according to Dr. Mead, its members were exposed to both Western medicine and goods at the dispensary and store; thus they were not without exposure to Western technology.

Between 1929 and 1937 there were three reports of the absence of schizophrenia among people not affected by Western technology. Seligman (1929) reported that in New Guinea he saw no psychosis "in the villages among natives leading their own normal life." He did describe six possible cases, however, among natives who were living along the coast in close contact with European settlers. Lopez reported similar findings in Brazil—no schizophrenia among "true primitives" of the interior of Brazil but existent cases among urbanized "natives" along the coast (Demerath 1942). And Faris (1937), who spent several years among the Bantu in the Congo, reports no cases of schizophrenia: "No records of any such cases existed, nor was there any memory on the part of those of the staff [of four large hospitals] of any such cases. In the villages attempts were made to describe the symptoms to the natives, but no comprehension of such disorders was found."

In 1936 Shelley and Watson reported the first of several studies of hospitalized Africans. They identified 30 schizophrenics out of 84 inmates at Nyasaland's only asylum. The most striking finding in their analysis of these cases was the close correlation between the diagnosis and degree of Europeanization: "The tribal incidence indicated a definite predominance of the condition in natives who had been in close contact with European civilizing influence." Eight of the 30 schizophrenics had been educated and six of these had even traveled to other countries; this is a very high percentage compared with that for the indigenous population of Nyasaland at that time.

During this same period Dhunjibhoj, a British-trained Indian psychiatrist, was making observations in a mental hospital at Ranchi in Eastern India. He described classical *dementia praecox* as being present in both sexes. "It is my experience," he wrote, "that those Indian

communities highly advanced in Western civilization and culture, etc., such as the Anglo-Indians, Parsees, the educated section of the Bengalis, are more prone to this form of psychosis" (Dhunjibhoy 1930). India had, of course, been colonized for over 100 years at this time and there was no lack of Western influence.

Similar observations have continued to be made in India. Rao showed in a 1959–1960 study that the first admission rate for schizophrenia was highest for the Brahman and Kayastha castes, the two with the highest literacy rates (Rao 1966). And Elnagar's group reported in 1971 the highest prevalence among the socially and economically most advanced groups in India.

Demerath's 1942 review article on schizophrenia among "primitives," which included Dhunjibhoy's observations, concluded that ". . . wherever schizophrenia has been reported, the society in question has been in the process of acculturation." Demerath and others (e.g., Devereux 1934) developed theories about how schizophrenia could be related etiologically to the stresses of acculturation.

Benedict and Jacks

Until the early 1940's, the universality of schizophrenia was still an open question, subject to debate. By the late 1950's, however, it had been closed. The one study most responsible for this change was a 1954 review by Benedict and Jacks, "Mental Illness in Primitive Societies." Virtually every current textbook claim of the universality of the disease cites this study as proof.

Benedict and Jacks based their conclusions on studies of five "primitive societies." The first two had been done by Beaglehole (1937 and 1950) in New Zealand from 1925 through 1935 and in Hawaii from 1930 through 1936. The fact that these were not exactly "primitive societies" appears to have escaped Benedict and Jacks; furthermore, the studies were a report only of hospitalized cases of schizophrenia and thus are of limited value. Even these two limited studies, moreover, stressed that the less developed groups (Maoris in New Zealand, full-

blooded Hawaiians in Hawaii) apparently had lower rates of schizophrenia.

The third study cited by Benedict and Jacks was Laubscher's 1938 report on the South African Bantu. Like the two studies cited above, it included only hospitalized cases. This group of Bantu was undergoing rapid acculturation at the time of the study. In addition, Laubscher has been criticized for regarding witchcraft as a phase of schizophrenia and diagnosing it as such.

Benedict and Jacks acknowledge the shortcomings of these studies of hospitalized cases and put their major emphasis on two other studies—those carried out by Carothers in Kenya from 1939 through 1943 and by Tooth in the Gold Coast in 1948. Both of these, according to Benedict and Jacks, included what they term "field studies" of schizophrenia.

Carothers' study is primarily a tabulation of all first admissions to the mental hospital in Nairobi over a 5-year period (Carothers 1948 and 1951). He diagnosed 174 cases of schizophrenia out of 558 total admissions. Kenya (and especially Nairobi) had been extensively colonized by the British for many years at the time of the study.

Carothers' "field study" consisted of asking the tribal chiefs in one area of Kenya to make a census of all "insane and mentally deficient persons." The request was conveyed through the district government officer and there is no breakdown provided of types of cases. Thus there were 228 cases reported out of 616,000 persons (0.37 per 1,000) but this is a total number for all types of mental illness and mental deficiency.

Carothers' extensive writings included a monograph subsequently published by the World Health Organization in 1953; perhaps because his works were easily accessible, his findings have been widely quoted. But on examination they appear to be virtually worthless. Carothers spent most of his time trying to make his research findings support his theories about the inferiority of Africans. Specifically, he theorized that their forebrain was underdeveloped and said that they were similar in behavior to leucotomized Europeans: ". . . all the observed Afri-

can peculiarities can be explained as due to a relative idleness of his frontal lobe" (Carothers 1951). These studies, which have been widely quoted in the epidemiological literature on schizophrenia, really are more appropriate as classical works on racism.

The field study conducted by Tooth (1950) in the Gold Coast was an attempt to find cases of mental disease among the general population. The Gold Coast was divided into the north (rural, little contact with Europeans) and the south (more developed, highly missionized). Tooth found a total of nine cases of schizophrenia in the north (where he claims his case-finding methodology was much better) and 24 cases in the south. From his own report, it is not at all clear whether in fact *any* of these cases were really schizophrenia. Trypanosomiasis was endemic in the Gold Coast (especially the north) at that time and, according to Tooth, could cause a psychosis virtually indistinguishable from schizophrenia. Furthermore, he concludes:

Among the "bush" peoples, a typically schizophrenic picture is most likely to be due to organic illness, while schizophrenia itself appears as an amorphous, endogenous psychosis. But the schizophrenic psychoses occurring in the urban, literate section of the population show more nearly the same forms as are found in Europeans.

Thus, there is a question whether Tooth found any real cases of schizophrenia in the less developed part of the Gold Coast. Nevertheless, this study was cited by Benedict and Jacks as the final proof that schizophrenia is a universal disorder, and this conclusion has been quoted in psychiatric textbooks as fact.

More Recent Studies

Once an idea becomes part of a textbook, it develops a life of its own and is seldom questioned. This is what has occurred with the idea that schizophrenia is universal. More data on the incidence of schizophrenia in developing areas have become available in recent years, but there has been a tendency to interpret these findings in light of the "known" fact of universality.

Lin's (1953) field study of mental illness in three communities on Taiwan is a case in point. Among a total of 20,000 people he surveyed, he found 43 cases of schizophrenia. There was no significant difference in the prevalence of schizophrenia among the three communities; all three had prevalence figures only slightly less than prevalence figures for Europe and America. Although Knox and Tourney (1965) have cited this study as further proof that schizophrenia is found among "primitive people," the three communities studied by Lin were, in fact, a city of 120,000, a town of 20,000, and five villages which were only 7 miles outside the capital city of Taipei; the inhabitants of these communities had migrated from mainland China between 1664 and 1895, were exposed to Western technology on Formosa, and cannot in any way be considered "primitive."

More interesting for the question of universality is the subsequent study on Formosa done by Rin and Lin (1962). They surveyed 11,442 Formosan aborigines who had had varying degrees of exposure to Western technology and found a total of 10 cases of schizophrenia, yielding a prevalence rate of 0.9 per 1,000 (less than half the prevalence rate for the Chinese communities there). More importantly, most of the cases they found were characterized by acute onset, short duration of illness, and usually a complete remission; thus they may not have been "process" schizophrenia. Only two of the cases were chronic, one with progressive deterioration.

Africa has yielded little further data since Carothers and Tooth made their observations. Smartt, a British psychiatrist in Tanganyika, found 67 cases of schizophrenia among 252 people in the mental hospital there, and noted that a highly disproportionate percentage of them were from the towns and not the rural areas. This study shares not only the methodological limitation of Carothers, but his bias as well: "The African seems, in some way, to be lacking the higher moral sense which is the heritage of more advanced civilizations" (Smartt 1956).

Lambo (1955 and 1965) is also often quoted as having proven that schizophrenia exists among "primitive" Africans. His studies have

never attempted to answer whether schizophrenia occurs among these people, but rather have concentrated on the symptomatology of schizophrenia among patients who have had greater exposure to Western culture and those who have had less exposure. The Yoruba area in Nigeria from which his patients are drawn has had continuous exposure to Western technology since at least 1900. Lambo also notes, as Tooth had previously, that “. . . when a patient with schizophrenia from the non-literate group shows a cluster of symptoms which, in Europeans or in the Westernized African, would be described as typically schizophrenic, trypanosomiasis or other organic disease should be looked for” (Lambo 1965). In other words, classical schizophrenia in non-literate Yoruba may not actually be schizophrenia at all, according to Lambo.

Finally, within the past few years some preliminary data on schizophrenia in New Guinea have become available. Burton-Bradley, a psychiatrist who has been there for a decade and a half, reported 343 cases of schizophrenia among the first 1,000 cases of mental disease which he examined. Virtually every one of the cases, however, occurred among individuals who had been living in the larger towns (“the person of limited cultural contact, the so-called bush individual, very rarely presents with the symptoms of schizophrenia [Burton-Bradley 1969]”) or who had just migrated from rural areas to the towns (“Not uncommon is the acute schizophrenia of sudden onset coming on usually within three months of the patient’s leaving the village and working for the first time in a large town. Such patients readily recover and are returned to their village, at which level they can function without disturbance [Burton-Bradley 1963]”). This last group is probably “reactive” and not “process” schizophrenia.

Discussion

What conclusions can be drawn from the survey of studies of schizophrenia in less developed areas? The following points should be considered:

- There are three early anthropological observations that schizophrenia did not exist

among people with little or no contact with Western technology; two of these did report cases among people who had such contact (Demerath 1942, Faris 1937, and Seligman 1929).

- Reports of schizophrenic patients in the mental hospitals of developing countries are common; it is acknowledged, however, that these hospitals draw their patients from the most highly developed areas of the country (Burton-Bradley 1969, Carothers 1948, Dhunjibhoy 1930, Kraepelin 1919, Shelley and Watson 1936, and Smartt 1956).

- The single field study in a minimally developed area (northern Gold Coast) found nine cases of schizophrenia which, according to the author, could also have been diagnosed as trypanosomiasis (Tooth 1950). A field study in an area with varying degrees of development on Formosa found a prevalence approximately half that for the surrounding area (Rin and Lin 1962).

- The prevalence of schizophrenia in developing areas appears to be roughly correlated with the degree of Western acculturation and exposure to Western technology (Burton-Bradley 1969, Dhunjibhoy 1930, Shelley and Watson 1936, and Tooth 1950).

- If mentally ill individuals who live “in the bush” present as cases of classic schizophrenia, some psychiatrists say that they may not have schizophrenia at all but rather organic brain disease (Lambo 1965 and Tooth 1950).

- The studies which report cases of schizophrenia among true “bush” individuals usually add that these cases have an acute onset, short duration, and complete remission (Burton-Bradley 1963, Murphy and Raman 1971, and Rin and Lin 1962). Thus, these are cases of “reactive” and not “process” schizophrenia. The “reactive” variety is seen commonly among soldiers under fire, prisoners, immigrants, and Peace Corps volunteers. It may not be the same disease entity as the “process” variety (Garmezy 1968). In any case, none of the studies reported “process” schizophrenia among individuals who had not had significant exposure to Western technology.

In light of the above observations, it seems unwarranted to conclude that schizophrenia is universal. It may be, or it may not be—it is still

an open question. From the studies done to date, it can be concluded only that "process" schizophrenia apparently is found in all cultures which have been exposed to Western technology.

Clearly we need more data on this question. We need well-planned field surveys of the prevalence among groups who are in varying stages of exposure to Western technology. But the world is shrinking and television is invading the remotest regions on earth: Such studies must be done soon or it will be too late to do them at all. But until the universal prevalence of schizophrenia becomes an open question this task is unlikely to be undertaken. If we fail to do such studies, we run the risk of bypassing valuable clues to etiology.

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