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Schizophrenia from an American Perspective

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The theme of this volume is the search for a definition of schizophrenia which would encompass its essence, i.e., arrive at the core nature of this disorder. This raises a philosophical and semantic issue, namely, what is the purpose of defining such concepts as schizophrenia, and what are the tests of its value. The possible options open to us are answers to the following questions: (1) does such a definition exist; (2) is it a true definition; (3) is it a useful definition? As to whether schizophrenia exists: what can be said with conviction? Conventional wisdom can help us here, and its answer would probably be yes, given the fact that schizophrenia runs in families and that it has a certain reliability regarding age of onset. Outcome would lead one to accept its existence in a pragmatic way. As Seymour Kety once said, "If it is a myth, it is a myth with a strong genetic component." Whether we can define it in such a way that it will be true is debatable, but the usefulness of a concept is possibly as important as its truth value. Even if its truth has not been established, it is still acceptable if its usefulness can be demonstrated for the purposes of classification, treatment, and communication. The question of whether or not we can arrive at its essence raises the problem identified by Aristotle, who claimed that every category has an essence. However, schizophrenia may have more than one essence, for, as Wittgenstein has asserted, the fact that a thing has only one name does not mean that it is only one thing. That a tree is a thing is

1Joseph Zubin died quietly on December 18, 1990 at the age of 90. His colleagues wish to acknowledge his teaching and mentoring to students of psychopathology from a variety of professional disciplines, and his enthusiastic pursuit of knowledge. The major contributions of this paper were made by Dr. Zubin.

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generally accepted, but its significance varies with the aspect of the tree that is of interest, and this can vary with whether it is being considered by a house owner, an environmentalist, a forester, a lumberman, a landscaper, or a tree surgeon (who considers whether it is vulnerable to disease). The degree of rigor required for our definition of schizophrenia is debatable. Pertinent to our problem is Huxley’s discussion about defining a species:

There is no single criterion of species. Morphological difference; failure to interbreed; infertility of offspring; ecological geographical, or genetical distinctness—all those must be taken into account, but none of them singly is decisive. Failure to interbreed or to produce fertile offspring is the nearest approach to a positive criterion. It is, however, meaningless in apogamous forms, and as a negative criterion it is not applicable, many obviously distinct species, especially of plants, yielding fertile offspring, often with free Mendelian recombination in crossing. A combination of criteria is needed, together with some sort of flair (Huxley, 1940, p. 11).

When we realize that there is no singular definition of species in biology, and yet biology flourishes, we may lower our demand for absolute conceptual rigor for a definition of schizophrenia, lest too much rigor bring on rigor mortis. Since we have not yet identified the etiology of schizophrenia, we must be satisfied with an ad hoc operational definition "subject to change" without notice, as some travel schedules warn. Despite the above considerations, we have been asked to present an American view of schizophrenia as a complement to the European views presented in this volume by Professor Manfred Bleuler.

We have the advantage of having read beforehand Manfred Bleuler’s intimate description of the development of his concept of schizophrenia, and our presentation shows its influence. He described how his father, Eugen, transmitted his views to him directly, and also how he learned these views indirectly through observing his father’s interactions with his friends Emil Kraepelin, Adolf Meyer and Sigmund Freud. He distilled this intellectual inheritance through his own experience as a life-long friend of his patients and their families, and as a "caretaker" rather than as a "therapist." That is why his view is so unique and precious.

You will now be able to compare how Manfred Bleuler developed his concept and how we, 3,000 miles away, one of us a Lithuanian immigrant to the United States, developed ours. To our great surprise we arrived at a common understanding, though both concepts
represent what as recently as a generation ago was perceived to be a maverick view when compared to the views of the establishment in both our cultures. The differences that do exist between Manfred Bleuler and us are largely due to the differences in our approaches. Bleuler uses the clinical method, whereas we use the biometric method, namely, the application of measurement to the clinically intuitive, subjective, and sometimes amorphous concepts that are still the basic conceptual underpinnings of the disorder. But what is the difference between the biometric and clinical approaches? The clinical method is too well known to need definition except to recall that it is essentially a bedside approach to the patient without any unnecessary restrictions. The biometric approach is more constrained by rules and regulations for obtaining reliable information, and is based on Lord Kelvin's dictum "when you can measure what you are speaking about, and express it in numbers, you know something about it; but when you cannot measure it, when you cannot express it in numbers, your knowledge is of a meager and unsatisfactory kind" (Merton, Sill, & Stigler, 1984).

Perhaps the most important distinction between the clinical and the biometric approach is in the ability of the biometrician to express the degree of certainty or uncertainty of his conclusions in some statistical form by attaching certain probabilities of being right or wrong in his judgment. While the clinician also feels degrees of uncertainty, he has no way of expressing them mathematically. Exceptional clinicians like Manfred Bleuler seem to overcome this problem.

Perhaps it is in the common objectives of clinical and biometric interviews that we can aim to bring convergence to the varying approaches. Essentially, reaching a diagnosis is the result of a series of mini-experiments in which hypotheses are tested, and further hypotheses derived based on what the patient reports, until a final diagnosis is reached. Although the open-ended interview initially may probe the specific aspects of a presenting problem in greater depth, it may also fail to detect other areas of significant pathology. The semi-structured interview provides a comprehensive and efficient overview of pathology, which may not be achieved in the open-ended format until several sessions have transpired. It is important to emphasize that these two approaches are not mutually exclusive; semi-structured interviews require the incorporation of clinical skill and judgment to recognize the nuances of patient speech and behavior that require probing.
A View of Schizophrenia in the First Half of the Century

The attitudes typical of American schools in the first half of the century towards understanding schizophrenia are typified in the personal experiences of the senior author.

I entered the field of psychopathology serendipitously because I received my Ph.D. during the Depression in 1932 in a combined, tailor-made program at Columbia University in both experimental and educational psychology with a strong statistical bent, which I contrived despite a frown from the authorities. This was in the day before such specialties as clinical or psychometric psychology had developed. The year 1932 was a bad year to start a career—it was in the depths of the Depression. There were no jobs offered so I took a volunteer job at the New York State Psychiatric Institute (PI, as it is usually called) in the experimental psychology laboratory of the late Professor Carney Landis. Psychoanalysis was in the saddle at PI and, since the institute was established for research rather than for service, the staff selected the patients in accordance with their research program, on the basis of their suitability for psychoanalysis. My knowledge of psychopathology was rather meager and my acquaintance with psychoanalysis even less. Nevertheless, I began to apply what I had learned and began with measuring the patients' personality through specially devised personality inventories developed following Woodworth's Personal Data Sheet. These inventories were the predecessors of the MMPI. I soon discovered that the patients, though carrying the label of dementia praecox, did not behave at all like dementia praecox patients. I began to wonder whether they really fitted the textbook description. I finally mustered enough courage to discuss the matter with the Clinical Director. He looked at me sternly when I said, "I don't believe that any of these patients are dementia praecox patients, nor that they are mentally sick." His answer was: "You better believe it, or you don't belong here. They may be at an early stage in their illness, but they have it with few exceptions." I then inquired whether they would eventually deteriorate and he answered "you better believe it." This authoritative admonition stayed with me for several decades and I had only freed myself from it in the 1950s.

The tendency to diagnose schizophrenia was so strong that a mere
suspicion of schizophrenia was enough to bring on the diagnosis, or as the institute director said, a touch of schizophrenia was like a touch of pregnancy. During the latter half of the 30's, somatic shock therapies—insulin and electro-convulsive therapy—were introduced, and for the first time changes in patient behavior could be observed directly without waiting years for psychotherapy to bring about its effects. Here was an open challenge to a biometrician. Could these changes be measured?

Psychological tests were of no avail since they were aimed mostly at measuring traits, which persisted, not states, which changed. Behavior rating scales were the answer and they sprouted up everywhere. Rating scales had to be produced practically overnight by such pioneers as Lorr, Wittenborn and Malamud. There is a story, probably apocryphal, told about the physiologist Hudson Hoagland, who found some biochemical change in Malamud's patients at the Worcester State Hospital and asked to see Malamud's data on the same patients. Malamud handed him his voluminous case histories. 'Are there no numbers here on their behavior?' Hoagland inquired. Malamud responded, 'No, but if you want numbers I'll make them up for you!' That night, with help, Malamud converted his descriptions of behavior into rating scales.

At last it became possible to see symptoms disappear or change before our very eyes. Apparently, for the first time schizophrenic behavior was seen as fleeting, not permanent or chronic, and could undergo change through intervention. Our laboratory became a watch-dog for evaluating therapies, tests, rating scales, and laboratory measures such as reaction time, flicker fusion, and psychophysiological measurement. Even the Rorschach came under scrutiny, and at that stage of its development it was found wanting as a test for identifying schizophrenia (Zubin, 1954). The conclusion was that if you treated the protocols as interview material and analyzed them for their content, the results would correlate with the content of the interviews on which the diagnoses were based. Furthermore, the determinants of movement, color, and shading were neither reliable nor valid using the scoring procedures of that period. More recently, these deficiencies have been carefully addressed by Exner and others (Exner, 1986).

The belief in the efficacy of the somatic therapies, including psychosurgery, was so strong that Stockings (1947) in England suggested that we discard our diagnostic categories entirely and classify patients according to the therapy to which they responded. Thus, those benefiting from insulin would be categorized as dysglycic, or from ECT as dysoxic. This past trend should serve as a warning against our current attempts at utilizing responses to treatment as a criterion for the diagnosis or search for subtypes. We would have to
classify disorders while the therapies still worked and before they went out of vogue!

To a large extent, the development of the concept of schizophrenia reflected the place where schizophrenics were to be found—in the state hospitals. Before 1890 and the advent of Kraepelin's era, only acute cases were to be found in the rather small hospitals of the United States, where patients were brought by their families. Many of the patients were no doubt dementia praecox cases who could no longer be kept at home. The psychiatrist was merely a caretaker and not much time was available for investigations. After 1890, with the rise of immigration, the mental hospitals became overwhelmed with the aged, brain injured and chronically ill, so that they became mere warehouses for managing the patients. World War II brought a depletion of the hospital medical staffs, who were needed for the armed forces, so that state hospitals deteriorated and not much progress was seen.

However, World War II stimulated a revision of the diagnostic nomenclature because it had been based on hospitalized patients who were chronically ill, and was therefore poorly adapted to the classification of members of the armed services. There was no generally accepted national nosology. The bringing together of psychiatrists from all over the United States for the military made it necessary to develop a common language. The Veterans Administration proposed a new classification system, which did not get wide acceptance because of its bias toward the veteran population. The pressure for revision was so great that the American Psychiatric Association appointed a task force. This started the series of DSM's which went through one revision before DSM-III came on the scene.

The failure of general psychological tests as diagnostic instruments (Zubin, Eron, & Schurmer, 1965) brought the realization that the only proper tool for diagnosis was the clinical interview. However, a review of the literature on the interview revealed that its reliability and validity were disastrously poor, so our group began to develop more systematic approaches. One of our friends, Mort Kramer, called attention to the discrepancy between the United States and the United Kingdom national statistics indicating that there was a 10:1 ratio of affective disorder to schizophrenia in the United Kingdom, and a converse ratio in the United States. We obtained a grant from the NIMH to study this discrepancy, and our Biometrics Research Unit utilized the newly minted systematic interviews, together with the Present State Examination of John Wing, to examine patients in metropolitan New York at the New York State Psychiatric Institute and in metropolitan London at the Maudsley Hospital. The case records in both hospitals were subsequently reviewed to determine the
diagnoses according to standardized criteria.

The proportion of admissions for schizophrenia in New York, according to the hospital diagnoses, was higher than the proportion based on the revised diagnoses given by judges when standardized criteria were applied to the case histories. It is clear that the initial view of a tremendous rise in schizophrenia was not warranted by the case history analysis. When the case histories were examined for the presence of actual symptomatology, the diagnoses in the two institutions were similar. Thus, the study indicated that the discrepancy was not in the patients but in the style of applying diagnoses in the two countries (Cooper et al., 1972).

Historically, the American concept of schizophrenia has been much wider than the English concept, although before World War II it was rather narrowly defined to describe primarily hospitalized patients. When psychiatric practice extended beyond the hospital walls, the concept began to broaden as the influence of Eugen Bleuler spread and as psychoanalysis entered the picture. As milder schizophrenia began to be diagnosed it became apparent that many of the symptoms were on a continuum from mild to severe; clinicians began to see a "touch" of schizophrenia in many of their more functional patients. In this way the diagnosis of schizophrenia became diluted.

When the US-UK Project, followed by the World Health Organization study (WHO, 1979), demonstrated that the US diagnoses of schizophrenia were broader than in any other country, the need for narrowing the concept to agree with the rest of the world was recognized. Furthermore, as the results of the US-UK Project became clear, it also became apparent that these results were not merely of academic interest. Since the administration of treatment, especially drugs, was often dictated by diagnosis, many patients were given inappropriate treatment when their diagnosis was inaccurately determined. This realization introduced a careful reexamination of the efficacy of drugs. The overdiagnosis of schizophrenia in the US presented a diagnostic challenge for American psychiatrists. The four A's postulated by Eugen Bleuler, which had such a great influence in the USA (associative loosening, affective blunting, ambivalence, and autism), were not a sufficiently constraining basis for making rigorous definitions. To counter this trend, Kurt Schneider's (1959) first rank symptoms were brought to bear on diagnosis in the United Kingdom. While in retrospect these first rank symptoms may be nonspecific to schizophrenia and more useful in determining severity of the illness rather than its diagnosis, they nevertheless helped to bring about greater rigor. Thus, Bleuler's accessory symptoms were brought back into primary focus, and since these psychotic symptoms were more easily discriminated from normality with their "yes or no" presence,
they added greater rigor to defining the diagnosis. In the conference organized with the help of the American Psychopathological Association, which preceded the launching of the US-UK Project (Zubin, 1961), Carl Hempel and, somewhat earlier, Ernest Stengel, went one step further and proposed an operational approach for the definition of mental disorders by requiring that the diagnosis meet certain minimum criteria. Two more developments aided in narrowing the definition of schizophrenia. The St. Louis school at Washington University presented the first narrow set of diagnostic criteria including requirements as to the duration of symptoms (at least 6 months) and the presence of severe psychotic symptoms such as delusions and hallucinations (Feighner et al., 1972). This was soon followed by the development of the Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978). Currently, we are awaiting the fourth version of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-IV) and the tenth version of the International Classification of Diseases (ICD-10).

Vulnerability Theory: A Model Integrating Disparate Etiologies

Thus far we have dealt with the phenomenology of schizophrenia as it developed in the United States with a special focus on diagnosis. Having established the reliability, if not the validity, of the diagnosis of schizophrenia, it became necessary to deal with issues of validity by examining etiology. Since there was still no known basis for etiology, a set of scientific models covering the various approaches to etiology was proposed (Zubin, 1972).

The need for scientific models to explain observed facts is well illustrated by De Sola Price in his book Science Since Babylon (1975). He points out that the ancient Babylonians were fact-mongers and provided sufficient data on planetary movements to be able to predict their future positions, but had no conceptual framework for their orbits. The ancient Greeks, on the other hand, were not so interested in number crunching, but were conceptually minded, so they provided the geometry for the orbit of the planets. It was not until Alexander the Great brought these two civilizations together that the modern scientific endeavor was born in which conceptual models could be tested by empirical observation. Until the 1950s, psychiatry was largely a conceptual field, in which the theories had little connection with statistics that enumerated the frequency and distribution of mental disorders.
After first suggesting that various models of etiology, (including biochemical, genetic, learning, and environmental sources), could all contribute to the etiology of schizophrenia (Zubin, 1972), it became clear that no single model was appropriate to explain all of the findings. Consequently, an integrative theoretical formulation, the Vulnerability model, was advanced (Zubin & Spring, 1977). This model postulates that an enduring vulnerability to schizophrenia results from interactions among the various contributing etiological sources, rather than from any single etiological source. The appearance of symptomatology results because the vulnerable individual fails to cope with changing stresses in the environment. Thus, vulnerability is manifested by the possession of a lower threshold for coping with stressful life events. Following the onset of symptoms, reduction of stress below threshold can be expected to lead to remission of symptoms. In essence, the theory provides a heuristic for understanding that the schizophrenic episode itself is not a permanent condition, but that the potential for episodes persists. Coincident with this interactional view of vulnerability in the development of schizophrenia are data indicating the importance of psychosocial and ecological factors, as well as data indicating less severe long-term outcomes for schizophrenic patients than had previously been claimed (Zubin, Magaziner, & Steinhauer, 1983).

The vulnerability hypothesis also predicts that indicators or markers can be found in patients prior to or following episodes, as well as among their unaffected relatives. Such familial markers, including, but not limited to, genetic factors, would reflect vulnerability, but not indicate schizophrenia per se. A framework for examining a variety of probable behavioral and neurophysiological indicators was developed (Zubin & Steinhauer, 1981). Additional developments of the vulnerability model have emphasized interactional (Nuechterlein & Dawson, 1984) or specific physiological (Mirskey & Duncan, 1986) aspects of the vulnerability dimension.

We have also been concerned with understanding how a relatively permanent vulnerability may be reconciled with a differential sensitivity to the same stressors at different points in an individual’s life. Recently, we modified the basic vulnerability model by introducing a separate dimension that accounts for changes in tolerance to stress (Steinhauer et al., 1991). The tolerance dimension provides for the intervention of psychotherapeutic or biological treatments as a means of raising the individual’s threshold for coping above their usual or minimal level.
Atypical Notions Regarding Schizophrenia

It is clear that we share common ground with Manfred Bleuler in that our views of schizophrenia are not the same as those of our respective cultures. We differ from the traditional views of schizophrenia with regard to the following notions:

1. A more optimistic view of outcome. A disbelief that once schizophrenic, always schizophrenic, i.e., that it is not a permanently chronic condition necessarily leading to deterioration.

2. An acceptance of vulnerability, and not chronicity, as the permanent characteristic of schizophrenia.

3. An acceptance of the belief that schizophrenia is an episodic disorder. Not only is this a consequence of the view necessitated by the vulnerability hypothesis, it is also born out in reviewing a variety of long-term follow-up studies (Zubin et al., 1983), including the work of Manfred Bleuler.

However, some of our deviations from establishment thinking may not be fully shared by Professor Bleuler.

Kraepelin was probably wrong in endowing the schizophrenic with an intact sensorium. During the past three decades quite a number of brain function indicators have been identified that discriminate between schizophrenics and general population control groups, if not between schizophrenics and other mentally disordered individuals. Examples of such indicators include: the amplitudes of the P300 component of the event-related potential; pupillary dilation response; heart rate changes related to information processing activities; smooth pursuit eye movement abnormalities; behavioral indicators of vigilance (e.g., the continuous performance test); and iconic memory (e.g., span of attention).

Perhaps the most convincing evidence that the brain of the schizophrenic is not intact comes from the comparison of the brain structures of discordant identical twin pairs. The presence of greater ventricular enlargement in the brain of the affected twin compared to that of the unaffected twin (Suddath et al., 1990) seems to imply that the brain of the schizophrenic, and presumably the sensorium, are not intact. Although it is not yet certain whether the observed changes are antecedents or consequences (including possible consequences of treatment), the results do offer a potential testimony for the environmental triggering of a genetic vulnerability. Further evidence of environmental triggering is provided by Tienari's findings that the
schizophrenic phenotype emerges primarily in those vulnerable adoptees who are subjected to a disruptive family environment, and not in the equally vulnerable adoptees whose families are not disruptive (Tienari et al., 1978).

Kraepelin may also have erred in his significant clinical contribution regarding the absolute separation of dementia praecox (schizophrenia) from manic depressive psychosis. Although it has served heuristically to stimulate investigations, this separation has not been confirmed. While the excess of schizophrenic, but not manic depressive, illness in the biological relatives of schizophrenic adoptees offers some evidence for specific genetic transmission of these functional disorders, the presence of the manic depressives still has to be explained. The difficulty for the dichotomous scheme is easily seen in the diagnostic dilemma produced by atypical or intermediate cases, a dilemma that has often been resolved through the arbitrary imposition of a single diagnosis. To resolve this issue will require developing models that will distinguish, on the one hand, a single vulnerability with variable expression, and on the other hand, two independent vulnerabilities coexisting in the same individual, perhaps due to assortative mating (as suggested by Dr. Loring Ingraham, personal communication). It may be that the two functional disorders form a continuous spectrum with many intermediate types, rather than a dichotomy, so that what we have is a psychotic manifestation that can assume either a schizophrenic or an affective expression depending on premorbid personality, local traditions, ecological niche, social network, and timing of environmental trauma.

Furthermore, Brockington (1988) argues that Kraepelin’s formulation of dementia praecox lacks the essential characteristics which Aristotle demanded of a category—a single essence for identifying the concept. Instead, he notes three essences or defining principles highlighted by Kraepelin: a convergence to a defect state, the schizm [sic] or fission within the psyche, and the autistic absorption with a private world. "These principles are all completely different, and thus the richness of the psychopathology of schizophrenia, which Kraepelin and Bleuler described so vividly and which gives the "disease" its fascination and appeal, is its greatest weakness, denying any possibility of bringing the concept to a clear focus" (Brockington, 1988, p. 6). It is possible that the three Kraepelinian essences may eventually be traceable to a unique central nervous system and/or genetic deviation.

As for the effect of the biometric approach on the problem of schizophrenic essence, the formulations of DSM-III and the RDC disclaim anything but sheer atheoretical empiricism. As a result, as Brockington, Kendell, and Leff (1978) remark, "the previous state of inarticulate confusion in the diagnosis of schizophrenia has been
replaced by a 'babble' of precise but differing formulations of the same concept." The consequence has been a proliferation of interviews, diagnostic criteria, and nosologies; it is now time to reconcile the data from these various approaches.

Finally, we suggest that attention be paid to the self-healing capacities of the individual. We do not yet know the reasons that some patients appear to outgrow their disorder after a decade or so, or understand why some patients suddenly cease to exhibit symptoms. Much of this may reflect the tendency of the organism to heal itself. Interventions should be aimed at helping the self-healing process to occur. Moreover, greater attention should be paid to what behaviors and attitudes the patient brings to the healing process. John Strauss has pointed out that we rarely ask the patients what they are doing to help themselves, and this is certainly a notion that should be pursued.

Why the establishment disagrees with us with regard to optimistic outcome is an interesting question. In the following quote, Manfred Bleuler explains his father's pessimism about schizophrenia:

From 1886 to 1889, E. Bleuler dedicated himself completely to his community of schizophrenics as director of the remote psychiatric clinic of Rheinau, which was then an isolated, rural sector of Switzerland. Two decades later, during and after the First World War, he went back to Rheinau to visit about once a year, usually when the weather was fine during the summer. His former schizophrenic patients always greeted him warmly, enthusiastically. Much as these greetings pleased him, he usually made the painful observation, "Most of them did seem to have deteriorated." Then, depressed, he would ask, "Is there really nothing that can stop this disease?" But E. Bleuler did not know how many improved patients were out for their Sunday walks during his visits, and certainly not how many had been released and were living at home, recovered. Had he known, and if he had not continued to meet only the most severe cases among his old problem children, his assessment of schizophrenia would have been strongly influenced. A number of generations of clinical psychiatrists had experiences similar to his (M. Bleuler, 1978, p. 413).

We try to explain the pessimism in the USA by two factors. First, there has been a persistence of the degeneration theory developed in the middle of the nineteenth century, which presumed that the first generation developed neurosis, the second generation developed mental retardation and mental disorder, and the third generation finally
disappeared for lack of propagation, only to be replaced by newcomers in subhuman development. Though science, especially genetics, has long ago disposed of this theory, it still persists in the zeitgeist and is at the bottom of the stigma that is still attached to the families of schizophrenics. It also persists in the Kraepelinian heritage of the expectation of deterioration even though Kraepelin himself had disavowed the theory in his later years.

Second, there is the experience from the daily workload of most clinicians, who deal primarily with recidivist patients who either relapse or remain chronic, and thus provides a biased view of the outcome of schizophrenia. Like Eugen Bleuler, they forget about the patients who never return because they no longer need treatment.

**Future Directions for Clinical Investigation**

The bulk of the 30 or 40 billion dollars spent on schizophrenia annually is spent on relapsing or chronic cases. If we could nick the relapse rate by only 10%, it would save billions of dollars, many times the total research budget allotted to schizophrenia. In surveying the literature, one notices that relapse appears far from being an indigenous characteristic of schizophrenia. From 23 to 50% of schizophrenics never relapse after their first episode. The WHO (1979) cross-cultural studies find 35% non-relapers in developing countries and 19% in developed countries, while Luc Ciompi in Bern, Switzerland, reports 10% in his studies (Ciompi, 1980). With our currently reliable diagnostic methods we ought to be able to contrast patients who are similar diagnostically in their first episode and then determine what factors differentiate those who later relapse from those who do not.

We will conclude with an aphorism that characterizes the essence of the difference between the optimist's and the pessimist's views of schizophrenia. The philosophical optimist suggests that this is the best of all possible worlds, to which the pessimist responds "I'm afraid so." The pessimists, who are adherents of the chronic disease model, believe that the schizophrenic, for the most part, is a continuously sick person who once in awhile relapses into health. The optimists, who are adherents of the vulnerability model, believe that the schizophrenic, for the most part, is essentially a healthy person who possesses a continuing vulnerability, and who once in awhile suffers an episode of illness. We obviously embrace the latter view.
References


