VULNERABILITY TO SCHIZOPHRENIC EPISODES AND THEIR PREVENTION IN ADULTS

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Primary prevention of psychopathology refers to the elimination of the potentially noxious internal and external factors that might produce deviant behavior as well as to the fostering of the factors of normal development. In a recent paper, Kessler and Albee (1975) compared the field of primary prevention to the Great Okefenokee Swamp. While joining in the tribute they paid to all the investigators who have dared to tread this treacherous ground, we would also like to raise the question of why the early explorers have all devoted their attention to the tiny seedlings and low vegetation that grows there rather than to the full grown trees that dot the terrain. Our analogy refers to the fact that the last decade's rapid proliferation of research on vulnerability to schizophrenia has almost all been focused on children at risk. Much less research has concerned the problem of identifying and intervening in the lives of adults who are vulnerable to schizophrenic episodes.

In one sense, our concern appears to be outside of the area Caplan (1964) defines as primary prevention. Instead of contending with the problem of how to prevent episodes of schizophrenia from appearing at all, we have decided to confine ourselves to the more focused and presumably more
manageable problem of preventing further episodes in those who have already suffered one. But in another sense, we are plunging into the crux of the primary prevention problem. The issue turns on how one answers the question: the primary prevention of what? If one defines schizophrenia as a trait, which, once manifested, continues chronically until death, then all preventative measures begun after its onset must be regarded as either secondary (aimed at early treatment) or tertiary (aimed at rehabilitating the patient and minimizing long-term damage). However, if one regards a single episode as a time-limited state, which marks those who are vulnerable to transient episodes, then the task of aborting new episodes is one of primary prevention. To grasp this distinction, it is only necessary to understand our premise that the person who has suffered one episode of schizophrenia will not always be schizophrenic. We will, however, always be vulnerable to episodes of schizophrenia. From our point of view, therefore, primary prevention involves blocking only the subsequent full-fledged episodic expressions of schizophrenia -- something which may really be possible. We will put off until a much later date the task of eliminating the genetic and acquired bases of vulnerability -- something which will certainly be very difficult and may ultimately prove to be impossible except through retroactive birth control.

Thus, we propose that the first step in primary prevention research on adults is to learn how to identify the high-risk, vulnerable individual by examining those who have already succumbed once to an episode of schizophrenia. The initial state of preventive interventions should be to abort a second episode in such individuals. Once we understand the contingencies under which subsequent episodes occur, we may eventually be able to prevent the first one. After overcoming the episodes, we may at least be able to progress toward eliminating or preventing the development of the
underlying vulnerability itself.

Having reduced the imponderable problem of primary prevention to a somewhat more ponderable dimension, we face the need for certain definitions and assumptions underlying our task. We must further explore the question of what we are trying to prevent, and what the causes of the episodes may be. Of course it is possible that even though we are ignorant of cause, we may inadvertently do some good preventive work. This was the case with the miasmists who assumed that the swamp miasma was the source of mists and vapors which caused malarial (bad air). Although this theory was wrong, it spurred preventionists to build houses higher above the swamp areas. This prevented contact with mosquitoes and pollution, curing the problem by a stroke of good luck. Similarly, obstetrics has found ways of reducing eclampsia even though the origin of the disorder is still unknown (McNeil, this conference). But even the miasmists had some kind of model to guide their efforts. What descriptive and etiological models of schizophrenia are available to us today?

Let us first dispose of the model that mental disorder is a myth. There are two aspects to scientific investigation: the conceptual framework or model and testing the hypotheses emanating from it. In the conceptual framework we deal with abstractions in the form of concepts. In the testing of the hypotheses we deal with ostensive facts to determine the tenability of the hypotheses. About facts you can ask whether they are true or mythical. About concepts or methods for organizing facts -- you can only ask if they are useful. That mental disorder is still a useful concept will become apparent in this paper, if it is not already apparent to everyone.
Another problem which merits comment is that of the rigorous definition of concepts. It is a generally bruited claim that our definitions of diagnostic categories, or even our criteria for the presence of mental disorder are quite imprecise. True as this statement is, we must not forget that only in mathematics can we find iron-clad definitions. Such rigor in behavioral science could lead only to rigor mortis. Even in physical medicine, Feinstein (1967) and Palek and Moser (1975) have reminded us that criteria for the presence of physical disorders leave room for a substantial error margin. Finally, let us not forget that Julian Huxley (1940) found it impossible to give a rigorous definition of species:

"there is no single criterion of species...a combination of criteria is needed, together with some sort of flair."

Instead of striving for a rigorous definition of schizophrenia, therefore, we must seek a description that permits observers to agree about whether the disorder is present or absent. Current studies (Spitzer et al, 1975; Spitzer & Fleiss, 1974) on the reliability of psychiatric diagnoses indicate that considerable inter-rater agreement (e.g. kappa = .84 for the presence of schizophrenia) can be achieved by using explicit sets of diagnostic criteria. Thus, if schizophrenia is a myth, at least it is one which doctors can diagnose similarly.

**Descriptive Models of Schizophrenia: Chronic Condition or Time-Limited Episodes?**

The history of diagnostic schemas for schizophrenia leaves us a legacy
of two main traditions. One tradition is to rely primarily on the clinical symptom picture to diagnose schizophrenia, and to interpret the presence of classic symptoms like hallucinations or thought disorder as evidence for the presence of the disorder. Eugen Bleuler represented this tradition, and he has been followed by many others including Schneider, Langfeldt, the staff of the Biometrics Research Unit, and the staff of the WHO Pilot Study of Schizophrenia. In the other tradition are those who argue that such classic symptoms are not in themselves useful as diagnostic criteria since they are manifested by a diverse and heterogeneous group of patients. This latter tradition holds prognosis or actual outcome as the key to diagnostic certainty. Kraepelin, and more recently Feighner (1972) and Kety (1975) are spokesmen for the position that a definite diagnosis of schizophrenia can only be made if symptoms are chronically present for a long time and the patient displays either a deteriorating or an unremitting disease course over time.

If it is correct that once an episode of true schizophrenia has appeared, it runs an unrelenting downhill course, then there appears to be little hope for primary preventive measures aimed at adults. It would seem that unless measures are taken early in childhood, vulnerability will germinate to the point at which chronic mental illness is inevitable and treatment is futile. Those foolhardy ones among us who wish to intervene in the lives of vulnerable adults stand like the fabled little Dutch boy, finger in the dike, trying to hold back the North Sea.

But before we plunge ahead in this pessimistic vein, let us consider the evidence that, in contemporary form, "true" schizophrenia is an ever-present, unremitting disease.

If we first inspect our hospital populations, we find little to support this contention. In the New York State Hospitals, the average length of hospitalization for schizophrenia has fallen from several years in the pre
1950 era to 90 days in 1955 and to 37 days in 1975. Parallel trends have occurred in countries all across the globe (e.g., Miskanen, et al., 1973; Yolles and Kramer, 1969). In the New York State Hospitals three-fourths of the current chronic cases were admitted more than two years ago, and one-half of these cases have been hospitalized for over 20 years. These statistics suggest that only a small number of current schizophrenic admissions are contributing to the pool of chronic, back-ward patients that Kraepelin spoke of. Indeed, they might make us wonder whether "true" schizophrenia, defined in terms of chronicity, is largely a disease of the past.

But perhaps these statistics do not really tell the whole story. Maybe the majority of patients are merely caught in the "revolving door" policy and released without any symptomatic improvement. Although this suggestion may often contain a grain of truth, there is also contrary evidence. For example, Glick (1975) has found that schizophrenic patients randomly assigned to short-term therapy lasting only four weeks do show a substantial suppression of symptoms and Nenz, Endicott, and Spitzer (1975) have found similar results. At the discharge point of four weeks, short-term therapy patients function reliably better than other schizophrenic patients who have been assigned to longer term therapy. This finding seems to suggest that a majority of schizophrenics have experienced some remission, and, with support, are capable of improved functioning within one month of hospitalization. However, one might argue that this improvement is only fleeting and perhaps an artifact of the desire to thank or please the therapist.

To truly evaluate the hypothesis that schizophrenic reveals itself in the chronic impairment of a majority of patients, we must examine how the discharged patient functions in the community. It has long been clear that continued phenothiazine medication plays a substantial role in maintaining
discharged patients in the community. Typical of the findings in this area are Hogarty et al.'s (1975) data demonstrating that 80% of patients receiving placebo medication compared with 43% receiving maintenance phenothiazines experience significant relapses in the two years following discharge. But still, one might argue that although the drugs help patients to remain out of the hospital, the large majority of schizophrenics never recover fully enough to be functioning and contributing citizens of any wider community.

But the evidence contradicts this premise. Unless patients are already chronically ill at the time of assessment, numerous researchers have found it exceedingly difficult to define a group of newly admitted schizophrenic patients who will experience a poor outcome. For example, although Hawk et al. (1975) applied Schneider's first-rank symptoms, Langfeldt's criteria, and Carpenter's discriminating signs to patients at intake in the hopes of distinguishing a group of nuclear schizophrenics, they were entirely unsuccessful in predicting the status of patients five years later. In fact, 40% of the schizophrenic patients were among the best outcome group when compared with other psychiatric patients on a composite index of symptom severity, duration of hospitalization, and social adjustment. Similarly, of the 208 probands Manfred Bleuler followed longitudinally, 50% made an adequate adjustment to the community. Forty per cent were hospitalized occasionally, but lived most of their lives in the community. Only 10% showed the type of disease course described as typical in Kraepelin's writings and Eugen Bleuler's early work.

Manfred Bleuler's own observation is apt in this regard:

"That which my father had to a certain extent done in cross-section, I have investigated longitudinally. He could stay with his patients only as long as they remained in his clinic. When they left the clinic, they were thereafter out of sight and lost to him, and this was the case with most psychiatrists.
of his generation. For this reason, an unfavorable picture of
the course of illness had to be inferred: the improved and the
healed patients disappeared beyond the horizon of the clinic,
and he saw above all those who were unimproved or relapsed."

(Trans. D. Rosenthal, 1974, p. 92)

To summarize, the bulk of the evidence suggests that the chronic, un-
remitting schizophrenia, especially the catastrophic variety which may have
been commonplace generations ago is becoming increasingly rare. This fact
poses a problem for the Kraepelinian model of schizophrenia. Are we to say
that only those approximately 10% of cases who show no remission are to be
categorized as "true" schizophrenics, whereas the other 90% cannot be so
diagnosed? As an alternative, let us suggest another descriptive model
which at least has the virtue of more widespread applicability.

Specifically, we suggest that schizophrenia, like other mental disorders
appears in time-limited episodes induced when life stressors surpass a thresh-
hold set by the patient's characteristic level of vulnerability. Once the
episode has ended, a patient resumes functioning either at his premorbid
competence level or pretty close to it. The hypothesis that schizophrenia
is an episodic rather than a continuous phenomenon appears to be consistent
with three things we know about these patients. First, as we have already
discussed, only a very small group of schizophrenics shows a sharply deterior-
at ing level of functioning over time. Second, the course of this mental dis-
order is not a continuous one. Just as patients with affective disorders
are quite mentally clear between episodes, it is likely that the majority of
schizophrenic patients experience periods of normalcy interspersed with
their recurring episodes.

Third, our hypothesis may help to explain the well-known fact that in
schizophrenia, as in other mental disorders, the good premorbid personalities
tend to recover from their episodes, while the episodes of poor premorbid
seem to persist endlessly (Zubin, 1974; Zubin, 1975). Why should premorbid adjustment be so important in recovery? By definition, the good premorbid patient is a competent individual who is adept at coping with everyday life circumstances. Once the stress that triggered his illness is removed and the episode subsides, he should be able to resume his former place in life unless the episode/severely reduced his coping ability. In any event, we should clearly be able to detect the onset and offset of the good premorbid patient's episode, since he plummets from a high level of functioning to a very low one during the episode, and rebounds to his high premorbid level when the episode subsides. These sharp peaks and low valleys punctuating his course of illness make it easy to tell when the good premorbid's recovery has occurred.

What happens in the case of the poor premorbid patient to make him appear to remain endlessly in his episode? There are three possibilities: (1) The episode actually continues indefinitely; (2) The episode ends as in the case of the good premorbid; (3) The episode reduces coping ability. As for the first possibility, there may be a small fraction of cases in whom the episode persists endlessly. However, we would like to propose that the second and third alternatives, i.e., that the episode ends or that it reduces coping ability, describe what happens in the majority of cases. Let us assume tentatively, for the moment, that the episode usually comes to an end in the poor premorbid patient, just as it does in the good premorbid. Think of how one might determine that the poor premorbid's episode has ended, and it will immediately become apparent that this will be a very difficult task. Even when recovered, the patient's postmorbid level of adjustment is so low that he cannot cope with life's exigencies, just as he could not premorbidly. The clinical judgment of recovery should be
based on a comparison with the patient's premorbid level, but, since the premorbid level of adjustment is rarely known, this is usually not possible. Consequently, the poor premorbid patient may appear to remain still debilitated even though the episode has in fact disappeared. A similar problem is evident in detecting episode offset in patients whose coping level has actually been reduced. This loss of coping capacity might come about either directly as a result of the psychopathological process, or indirectly by the iatrogenic effects due to hospitalization or Gruenberg's Social Breakdown Syndrome. If the drop in competence has been striking, these patients may also still be mistakenly regarded as ill despite the ending of their episodes.

The unexpected and apparently spontaneous recoveries of some long-term patients may also fall within our explanatory framework. Often a patient remains in the hospital after his episode has subsided as a result of the failure to recognize that he is no longer sick. When the recognition finally comes, either on his own part or from those in his surround, he can of course be released from his bondage, and will probably be regarded as a miraculous recovery. In some cases the patient's low premorbid level of competence may even improve as a result of hospital guidance or maturation. These patients too are regarded as manifesting a miraculous and spontaneous remission of disease, when in fact the episode has long since passed and the primary change has not been in the disease process but in the further development of coping skills.

Having begun with the premise that schizophrenia is an episodic mental illness, let us move on to see if we can construct a model that will help us to investigate the causes of the episodes and to identify those who are vulnerable to them.
A Model of Vulnerability to Schizophrenic Episodes

There are of course numerous scientific models for explaining the etiology of mental disorders, and Zubin has previously discussed six of them (Zubin, 1972). The theories can be grouped into two broad categories depending on whether they exemplify a field theory or an atomic approach. In field theory models, the individual is regarded as the target of a field of impinging forces that determine his well-being or illness. These forces may take the form of presently impinging ecological stimuli (ecological model), or more remote events which have influenced developmental maturation (developmental model), or altered the acquisition of response repertoires (learning model). In the atomic approach, each individual is regarded as a discreet unit with individual characteristics which determine his mental status. The person's relevant constitutional features are reflected in his idiosyncratic genetic makeup and internal biochemical and neurophysiological characteristics. Of course, this bipartite division is something of an oversimplification, since field theories must take account of individual differences in response to impinging forces. Atomic theories must, in turn, consider the fact that the condition and expression of the internal milieu is influenced by the environmental field.

The common thread running through all these models is that each suggests a hypothesis about some ingredient in the etiology of vulnerability to mental illness. Corresponding to the two main types of etiological models, two major causes of vulnerability have been proposed. The first is a genetic component which is laid down in the genes and reflected in the internal environment and neurophysiology of the organism. The second, is an environmentally
acquired or non-genetic component of vulnerability. This is a little more difficult to describe. If we consider the evidence that early experience, early exposure to certain stresses and specific diseases, leave their mark and tend to either facilitate or inhibit the development of a disorder in later life, we can metaphorically describe this acquired vulnerability as dependent upon the "memories" of the organism for past experiences, or on the scars such experiences leave behind them. Longitudinal studies of children at risk and retrospective studies of patients have begun to suggest some remote biological, familial, and social events that may be sources of acquired vulnerability. Some contributing events may be pregnancy and birth complications, submissive relationships with siblings (Pollin and Stabenau, 1968), disturbed relationship with father, overprotective mother (Kringlen, 1968), or a lack of intimacy in adolescent peer group friendships (Kreisman, 1969).

As things stand now, we have a wealth of hypotheses about the etiological sources of vulnerability. What has been lacking, however, is a good operational definition of vulnerability that is not wedded to any single etiological hypothesis. At present our studies of vulnerable individuals are bound by narrow sampling characteristics. These sampling criteria are dictated by the investigator's favorite hypothesis about the etiology of schizophrenia. For example, we never pool together samples containing individuals at risk because they have schizophrenic parents with samples at

*Strictly speaking there are no non-genetic causes, since even the response to traumatic happenstances is at least in part dependent upon a genetic proclivity, or the trauma could have no influence. By the same token there are no non-environmental causes since genes can not work or be evoked in a vacuum.
risk because they were isolated during adolescence. Neither sample is ever compared with a group vulnerable for a different reason -- perhaps because of disturbed family communication patterns. What seems to be needed is a more generic measure of vulnerability that would enable us to initially select a more heterogeneous sample of vulnerable individuals, and then to separate out from the heterogeneous mass, homogeneous sub-groups who derived their vulnerabilities by different avenues. With such a strategy, we can study whether the genetically at risk individuals are similar to those who acquired their vulnerabilities as a result of environmental factors. Perhaps we may yet learn that vulnerabilities derived from different sources are expressed in different forms of schizophrenia.

In the field theory models as well as in the atomic or biological models, the individual is a powerless pawn, at the mercy of ecological forces or in the hands of the fate dictated by heredity. This is hardly a true picture of man. In order to formulate a more acceptable definition of vulnerability, let us begin with an assumption that has won adherents in both the physical and mental health camps. This is that health prevails as long as the individual maintains a dynamic equilibrium against insults from his chemical, physical, infectious, psychological or social environment. When this equilibrium is pushed out of balance, disease ensues. The dimension of vulnerability-invulnerability describes the degree to which an individual tends to succumb or resist illness in the face of external and internal threats to his homeostasis. We recognize the existence of individual differences in vulnerability by observing the variability of responses of different individuals to the same environmental stresses. For example, if we inspect patterns of absenteeism for a grammar school classroom, it is clear that a small number of children are most vulnerable to disease and fall ill
of germs that rarely affect others in the classroom. Others of more moderate vulnerability succumb only to illnesses that also affect a substantial number of their peers. The least vulnerable rarely miss a day and remain healthy even at the height of an epidemic.

Extending this principle to the study of vulnerability to mental illness, we refer to the well-documented role of life event stressors in precipitating physical and especially psychological disorders (Rahe, 1964; Brown, 1968; Dohrenwend, 1972). A life event stressor is an incident such as bereavement, promotion, marriage or divorce which necessitates a substantial amount of readjustment. The psychological strain induced by a single event or an accumulation of similar prior stressors often precedes and appears to trigger the onset of an episode of mental illness.

The relation between vulnerability and life event stressors is shown in Fig. 2. It will be noted that as long as the stress is below the threshold of vulnerability, the individual responds to the stressor in an elastic, homeostatic way and remains well within the limits of normality. When the stress exceeds threshold, the person is likely to develop a psychopathological episode. As stress recedes below that line, often because the person is removed from the stressful context and placed in an institution with few pressures and demands, the episode will end and the person progressively returns to his pre-episode status.

Vulnerability, therefore, is defined in terms of the constitutional factors (internal) that set the threshold at which stressful life events will catapult an individual into an episode of mental illness. From this point of view, life event stressors can be regarded as immediate or short-term states of psychological strain that serve to elicit the encoded long-term engrams or templates of genetic or acquired vulnerability.
RELATION BETWEEN VULNERABILITY AND LIFE EVENT STRESSORS

MAXIMUM

LIFE EVENTS STRESSORS

MINIMUM

LOW VULNERABILITY

HIGH

WELL

THRESHOLD

ILL
Applying these definitions, we can arrive at a measure of vulnerability that is not wedded to any single etiological hypothesis, and will thus permit us to group patients according to the degree of their vulnerability without regard to its etiological source.

Very simply, we know that all individuals who have succumbed to an episode of schizophrenia are vulnerable to some degree. Further, those individuals for whom relatively minor stresses have been sufficient to trigger the episode are more vulnerable than those whose episodes have been precipitated by a major concatenation of stressful life events. While this principle enables us to assess the vulnerability of those individuals who have already succumbed to an episode, it permits us to make only minimal evaluations of the vulnerability of individuals who have never fallen ill. We can be fairly safe in assuming that people who have experienced accumulated life event stressors sufficient to elicit an episode in many other people without themselves falling ill, are relatively low in vulnerability, or, put differently, relatively invulnerable. However, we cannot make any assumptions about the vulnerability of healthy individuals who have not experienced sustained periods of serious stress. Although this is a major limitation, it may be all we can hope for at present. Moreover, understanding this limitation might in itself contribute something to our interpretation of research findings.

For example, this principle might influence how we interpret the well-known statistic that 90% of schizophrenic patients do not have a schizophrenic parent, or Kety's finding that there is a low rate of schizophrenia among the biological relatives of acute schizophrenic patients, but a high rate of mental illness among relatives of chronic or borderline cases. These data are often mustered to support the contention that genetic factors make
either no contribution or a relatively trivial one to vulnerability in most cases of schizophrenia, or at least that the genetic contribution of vulnerability to acute schizophrenia is negligible. However, this assumption is unwarranted until we know something of the life histories of the probands and their biological relatives. It is possible that the familial genes of the chronic patients set the vulnerability threshold so low that a broad spectrum of ordinary life events is sufficient to trigger an episode. The high familial concordances in these cases would therefore derive from the fact that numerous events present in almost any life history would prove to be pathogenic for such individuals. On the other hand, it may be that the parents and other relatives of the acute patients also possess a genetically based vulnerability to schizophrenia which is of more moderate degree. Thus, unless the life histories of relatives and proband have been concordant for accumulated and severe stressful life events, one would not expect to observe episodes in both despite their shared vulnerability.

We might make an analogy by comparing schizophrenia to sickle cell anemia. Sickle cell anemia is a severe, debilitating, and usually fatal disease caused by the presence of two abnormal genes which prevent the formation of normal hemoglobin. If only one abnormal gene is present, a heterozygous condition known as sickle cell trait results. Sickle cell trait is a benign, asymptomatic condition that is detectable only under special circumstances. However, extensive and sometimes fatal sickling can be evoked even in the heterozygous sickle-cell trait individual exposed to specific stressors such as high altitude, acute alcoholism, or deep anesthesia (Harris, 1963). Perhaps acute schizophrenia, like sickle cell trait, is a genetically linked disorder which only achieves full pathological expression
when very severe or specific adverse circumstances exceed the threshold of vulnerability.

Returning to our measure, it should now be possible to categorize our patients according to their degree of vulnerability by measuring the level of stress that was required to precipitate the episode of schizophrenia. Comparing patients of equivalent vulnerabilities, it should be possible to investigate whether the symptom pattern and severity, response to therapy, and disease courses are similar or different depending on whether the vulnerability is genetically or non-genetically based. Perhaps it will be possible to determine therapeutic strategies that are optimally attuned to different subtypes of vulnerables. Moreover, just as the study of individuals who have already experienced an episode of illness can permit us to empirically isolate and test putative causes of vulnerability, the study of individuals who remain healthy even in the face of catastrophic circumstances can enable us to determine some sources of invulnerability.

Competition and Vulnerability

We would like to stress that the discovery of sources of invulnerability is a problem which must be approached empirically. It has long been apparent that the most common sense assumptions about factors that buffer against or facilitate the development of psychopathology often fail to meet the test of statistical significance. For example, although it seems intuitively reasonable that certain types of family interaction patterns should prove to be schizophrenogenic, Jacob’s (1975) careful review of this literature has shown how difficult it is to demonstrate that these patterns occur with greater frequency in the families of disturbed compared with normal offspring. Just as we insist that the traits contributing to vulnerability to
schizophrenia must empirically relate to the probability of developing an episode, so must we insist that traits which intuitively seem to buffer against the likelihood of mental illness meet the test of research validation.

One assumption frequently encountered in the literature is that symptomatology and social competence are inversely related. In other words, it is often taken for granted that the same skills and dispositions that promote competence, defined as success or achievement in day-to-day living, are also sources of invulnerability which reduce the likelihood of developing an episode of schizophrenia. We do not accept the assumption that competence and invulnerability are identical. Instead, we tentatively hypothesize that competence or coping ability is orthogonal to vulnerability. Thus, highly competent individuals can have all grades of vulnerability, and relatively incompetent folk can be highly vulnerable or quite immune to mental illness.

There are several reasons for this suggestion. The first is simply that until we have systematically collected more data specifically investigating the relationship between these variables, it is best to assume the null hypothesis and avoid incorporating unsubstantiated dogma into the literature. The second reason is that there is a small pool of evidence suggesting that the two may in fact be independent dimensions. The third is that measures of competence and vulnerability appear to have distinctly different uses in psychopathology research. Vulnerability indicators measure the likelihood that an individual will succumb to an episode of schizophrenia. By contrast, a sharp dip in a patient's characteristic level of competence permits us to detect the episode's onset, and a return to the premorbid baseline signals the episode's end.
It is difficult to present research findings pertinent to the relationship between competence and vulnerability because, as we will point out, few studies have unambiguously assessed either variable. Although occupational level only indexes coping ability in one of the many domains integral to competence, it is the only measure that has commonly been studied in relation to patienthood. Clark (1948, 1949) in Chicago, Hollingshead and Redlich (1958) in New Haven, Leighton (1963) in Nova Scotia, and Svalastoga (1965) in Denmark have been among the many to show that rates of schizophrenia are highest for the lowest status occupations. However, there have also been contradictory findings, particularly when research is conducted in smaller cities or when incidence rather than prevalence measures of the frequency of mental illness were used. For example, in the Hagerstown Study, Clausen and Kohn (1959) presented the following figures on the average annual rates of first hospital admission for schizophrenia per 100,000 individuals aged 15-64:

- a. professional, technical, managerial, officials, proprietors 21.3
- b. clerical and sales 23.8
- c. craftsmen, foremen, kindred workers 10.7
- d. operatives, service workers, laborers 21.7

In another investigation, Turner (1974) found that fully 67.4% of subjects ever hospitalized for schizophrenia were employed. The proportion of employed persons among general population controls who had never experienced a significant hospitalization is only 20 percentage points higher -- 57.5%. Moreover, fully 70% of premorbidly employed patients were working at a skilled-manual level or higher, compared to about 68% of the general population! Therefore, it appears that high level job skills, while integral to competence, do not render one invulnerable to schizophrenia. The mentally
ill derive from the ranks of the successfully employed as well as the un-employed, and it is far from clear that relatively low level job skills predispose not only to social maladjustment but also to episodes of schizophrenia.

One reason why we know so little about the relationship between competence and vulnerability to schizophrenia is that most of our data is derived from health care facilities, and patienthood in a health care facility is a highly inadequate measure of the presence of an episode. As the well-known epidemiological studies conducted in Midtown Manhattan and Stirling County have shown, rates of hospitalization probably underestimate the number of individuals in the community at any one time who are troubled by an episode of mental illness. One rather disturbing study in this regard was conducted by Lee (1975) on a rural sample in North Carolina. Survey results showed that although psychiatric problems were present in one-half of the homes surveyed, only 50% of the families so troubled would consider seeking help for problems of this type. Moreover, only approximately 10% would overcome the fear of being labeled mentally ill sufficiently to actually seek help at a local mental health clinic that has been in full-time operation for four years and engaged in consultative work for twelve years.

Even if we ignore the attitudinal factors that influence a patient's willingness to seek hospital attention, prevalence studies of psychiatric disorders inflate the percentage of cases from low status occupations, since such patients tend to remain in the hospital for a longer period of time than cases from high status occupations. The longer hospitalizations of patients from low status occupations may have one of two causes. Either their episodes actually persist longer, or more likely, these patients may be poor premorbid and it is difficult for staff to determine when their
episodes have ended. In addition, the poor premorbid patient may possess relatively few supports in the community. Coupled with his low competence, the poor premorbid’s lack of social supports may make it difficult for a psychiatrist in good conscience to discharge him into the cold, harsh world outside the hospital. In light of all these factors, we must conclude that the beginning and end of hospitalization tells us relatively little about the onset, duration and offset of an episode, since (a) many individuals undergoing an episode do not seek mental health care; (b) many patients continue to be hospitalized after their episodes have subsided; and (c) discharge from the hospital often bears little relationship to the offset of the episode.

In addition to being a poor indicator of the presence or absence of an episode, hospitalization may reflect and be confounded by a low level of social competence. Meyers and Dean (1968) raised this possibility when they noted that lower class psychiatric patients often have less psychopathology than upper class patients even though they are more frequently admitted. Tischler (1975) echoed this idea in a study showing that, particularly for the young, patienthood is primarily determined by factors other than symptom intensity. Individuals who are unmarried, living alone, unemployed, or of the lowest socioeconomic stratum, all of which can be construed as reflecting poor competence in significant role contexts, show a higher tendency to seek and receive mental health care than would be expected based on the prevalence of symptoms in these demographic groups in the community. Christensen (1974) has found that low competence rather than psychopathology may be the pivotal factor in hospitalization. In a five-year follow-up study of patients previously hospitalized for schizophrenia, he found that, compared to patients who were readmitted and discharged,
those not rehospitalized showed both a better employment record and a more severe level of disturbance at follow-up.*

**Competence and Life Event Stressors**

In considering the relationship between stresses induced by life event stressors and competence, we must not assume that stressful life events are more often found in those of low competence, and hence that we should expect the occurrence of episodes more frequently in the low competence group. Only if the low competence individual is also highly vulnerable will an episode be elicited, and whether high vulnerability occurs more often in those of low competence, is not established. It is possible that because the highly vulnerable tend to have more episodes, their competence may wear thin even if it was high to begin with. This downward drift of initially competent patients may contribute to the appearance of a relationship between low competence and high frequency of episodes.

**Measuring the Onset, Persistence and Offset of the Episode**

In order to test our two central premises: (A) that schizophrenia appears in time-limited episodes interspersed with periods of remission and normalization; and (B) that competence and vulnerability are independent of each other, it is clearly mandatory that we arrive at a better strategy for assessing the onset, persistence and offset of the episode. Changes in an individual's characteristic competence level would provide the ideal markers for the presence or absence of an episode. Patients can only be considered in an episode as long as psychotic symptoms interfere with com-

*In all of these studies, the accuracy and precision of the diagnosis of schizophrenia is quite variable, but the general trends probably hold up despite this source of error.*
petence to the extent of impeding the patient's usual level of performance in important domains of everyday life. Typically, patients enter a hospital at this point, or they perform other acts which effectively remove them from contexts where their behavior has become inadequate. If they do remain in situations where important role performances are still expected, the drop in their usual level of effectiveness should be apparent to their colleagues. The episode can be considered over when it can be demonstrated that the patient is capable of matching his own pre-illness level of functioning in these socially significant roles.

Although alterations in competence are optimal criteria for the onset and end of an episode, the determination of this variable is beset with all the difficulties that retrospective data suffer from in addition to a few more idiosyncratic problems of its own. Since the patient usually does not come to our attention until after he is well into a full-blown episode of mental disorder, we have a wealth of information on his lack of competence during the episode, but very little knowledge of his premorbid competence level. We might describe this as knowing his liabilities but not his assets. Second, it is difficult to know when the episode has ended and the premorbid competence capacities have been restored if the patient is still in the hospital and not exposed to the significant contexts in which his competencies are exercised. Ideally, we should be able to simulate or contrive the important contexts in order to assess competence adequately in still-hospitalized patients. Some progress has indeed been made in this area. For example, following Goldfried and D'Zurilla's behavior-analytic model for assessing competence, Goldsmith and McFall (1975) have experimented with simulated interpersonal contexts to evaluate the effectiveness of patients' social coping
strategies. But none of our behavioral assessment techniques has yet achieved the scope necessary to make a full-range evaluation of a patient's competence.

This lack of progress is no doubt partly due to several fundamental quagmires inherent in any process of operationalizing competence. The construct has generally been conceived of as a ladder to make vertical, interindividual orderings among individuals or groups of people. There are those who argue that competence is a general and uniform property of individuals, quite like the G factor for intelligence. In rebuttal, there are many who insist that the definition of competence must be specific for each particular subcultural group, or even for each important context of day-to-day life, since values and expectations vary so widely across groups of people and types of situations. Moreover, it is quite certain that any given person is likely to show different levels of competence in different situations, something like the horizontal décalage phenomenon that emerges in assessing the Piagetian stages of cognitive competence. However, while these issues are quite substantial in their own right, they should really not prove to be major stumbling blocks to devising the type of competence measure we need for research in psychopathology. First, the purpose of designing such measures for use in determining the onset and offset of episodes of schizophrenia is not to make inter-individual comparisons, but rather intra-individual ones. Thus, we are only interested in comparing a patient's present level of function with his own premorbid level, not with the performance of individuals in other demographic groups or subcultures which might hold different values and standards. The problem of one individual's variability in performance across contexts still persists. However, we regard this as a real and natural phenomenon, and not as a form of diagnos-
tic error. This "fact" of nature calls for assessment techniques that cover a full spectrum of contexts evoking significant role performances. The mapping of these significant role domains has in fact received considerable attention in recent years, and Weissman (1975) has recently reviewed fifteen scales available to assess performance in occupational, marital, extended family and community roles. However, all of these procedures have been designed to assess the social competence of previously hospitalized individuals who are now living in the community. Hopefully, it will still be possible to catalogue each area of role performance into mini-contexts and situational demands that can be simulated to test the specific behavioral competences of hospitalized psychiatric patients.

Perhaps these ideal measures of competence will be available in time to enable prospective researchers of high risk children to evaluate the premorbid level and episode-related fluctuations in competence of their subjects. However, most of us who remain completely in the dark about our patients' premorbid coping levels will meanwhile have to seek other non-competence related indicators of the onset, persistence and offset of an episode of schizophrenia. The nature of these potential indexes is still an open question. Although it would certainly be opportune to find episode markers of a biochemical or psychophysiological type, such indicators, like our competency change measure, must await future developments. Meanwhile, we must rely on the currently available indices provided by clinical observation. Even though clinical observations are not initially as valid or reliable as we might like our indicators to be, we could by continued monitoring of patients define the clinical features that reliably pinpoint the onset, persistence and offset of an illness. By moving back and forth from clinical observation to objective indicators in an iterative way (Sutton, 1973) we might ultimately use these clinical observations as criteria for
obtaining more objective systematic markers, possibly of a biochemical or psychophysiological nature.

Perhaps we might begin by monitoring the behavioral, biochemical and psychophysiological fluctuations of patients already under observation in a residential ward or treatment facility. Although hospitalized patients probably become adjusted to the hospital routine, medication and treatment, there will no doubt be flare-ups of mini- as well as maxi-episodes during their stays. If these episodes could be monitored, and measures of behavioral, biochemical and psychophysiological characteristics compared with baseline measures taken during intermittent periods of tranquility, we could eventually devise indexes which characterize the onset, persistence and offset of such episodes. While episodes occurring in hospitalized patients may be of a different variety or intensity than those which initially bring the patient to the hospital, they can nevertheless serve as a starting point for investigating this important problem. Moreover, we can to some extent investigate the qualitative differences between episodes which begin in the community and those that occur in the hospital by continuously monitoring a subsample of patients from the episodes which are full-blown when they enter the hospital, through their states of normalization, and into the mini-episodes all during the course of a single hospital stay. Of course the great advantage of studying patients under continuous observation rather than only entering patients at the heights of their episodes is that we will be able to observe the onsets of the mini-episodes and compare the episode characteristics to the type of baseline premorbid data that is never available on newly admitted patients.

As a hypothetical example, let us consider a study that might be con-
ducted on patients at a residential facility. Presumably patients brought
to this facility are vulnerable individuals who have just experienced a stress-
ful event which triggered a full-blown episode. After some time, as the
acute episode subsides, the patient returns to his pre-episode level of com-
petence in coping with his environment. With the end of this episode which
brought him to the residential facility, he may be regarded as normalized at
his premorbid level, and, consequently, not in an episode. However, since
his vulnerability persists, it is possible that some stressful event occur-
ring in the institution will trigger another episode. In this case, it
would be useful if we could verify its onset by some technique other than
clinical observation, just as the courses of certain physical disorders can
be monitored by the patient's blood pressure or EEG.

Of course, the first problem facing us is to determine what kinds of
criteria might usefully be examined. Perhaps the best initial procedure
would be to study available case records to see what indicators are commonly
observed to be associated with the presence, onset, or offset of episodes.
Some of these might then be put to use in detecting onset. For example,
some possible criteria for the start of an episode might be (1) sleep dis-
turbance, (2) eating disturbance, (3) some required change in medication,
or (4) attendance at ward functions. At the point when these criteria are
met and an episode appears to be starting, a variety of other measures of
a psychological, social and physiological nature could be taken to discover
patterns in these new variables which occur at episode onset. An overall
criterion based on all these variables could be developed and tested using
canonical or discriminant function analyses to compare episode onset versus
absence of onset, onset versus offset, or onset and offset with persistence
of an episode.
It is of course an empirical question whether such indicators could be used in the community to determine the onset of episodes before they are clinically recognized. However, even research done exclusively on a hospitalized sample might have important policy implications by helping clinic staff to plan optimum discharge programs. If it were possible to truly determine the offset of the episode, perhaps earlier release could be facilitated and premature releases avoided. In addition, indicators might be discovered to predict the frequency and severity of future episodes, thereby helping to predict which patients will succeed when released and which will not.

Once the indexes are developed, it might be possible to validate them by manipulating the patient’s environment in the direction of eliciting a controlled mini-episode. There have been numerous attempts to experimentally simulate mental disorders, using drugs to evoke an episode: e.g., reserpine-induced depression, exacerbation of schizophrenic episode through Ritalin, amphetamine-induced psychosis. Certainly we would not ask patients to ingest these substances, and it may even sound ruthless to subject them to events which might be mildly and temporarily pathogenic. However, it seems well within ethical boundaries to study reversible episodes engendered by naturally-occurring, controlled events. Thus, for instance, if a parent’s visit to the hospital or the patient’s visit to his home has previously served as a triggering event for the onset of an episode, measurements on the patient might be collected before the visit is announced or expected. Corresponding measures after the visit would determine whether the indexes confirm the presence of an episode.

Summary of the Model

We can now summarize our model of vulnerability to schizophrenic episodes as shown in Tables 1 and 2.
**Table 1**

Definitions of Components of Vulnerability Model

1. **Vulnerability** - is the ability to withstand the stresses induced by life events.

2. - is an enduring trait.

3. **Episodes** - develop when life stress exceeds the threshold of vulnerability.

4. - are time-limited states of illness.

5. **Competence** - is the ability to achieve success in significant role contexts of everyday life.

6. - is orthogonal to vulnerability.
## Table 2

### Therapeutic Intervention in Episodes

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<td>- at episode onset, patient's competence drops sharply.</td>
<td>- Good premorbid return to good pre-episode status — obvious recovery.</td>
<td>- Prevent new episodes from developing by reducing vulnerability and/or stress producing nature of life events.</td>
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<td>- at end of episode, patient reattains premorbid competence.</td>
<td>- Poor premorbid return to poor pre-episode status — mistaken as still ill or chronic.</td>
<td>- Improve coping capacity of incompetent poor premorbid so that they can cope better when episode ends.</td>
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<td>- Modify environment to reduce frequency intensity of stressful life events so that there will be less opportunity for episodes to develop.</td>
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One concept that has been central in our presentation is the distinction between phenomena that are state-determined and those that are traits. We have contended that episodes of schizophrenia are psychopathological states elicited when life event stressors surpass the threshold determined by the individual's characteristic level of vulnerability. Vulnerability, by contrast, is a stable trait that characterizes the individual independently of the presence or absence of the episodes. To paraphrase, a schizophrenic is not a permanently sick individual, but a permanently vulnerable one. There are two models for the course of any disease, the infectious disease model and the chronic disease model. One can apply either to the study of schizophrenia depending on whether one's focus is the episodic states of illness or the more enduring trait of vulnerability. The infectious model describes disorders like the common cold in which the course can be pinpointed and initiated or terminated experimentally to learn more about the characteristics of the incubation period. The chronic disease model, which describes illnesses such as arthritis or diabetes, may have been appropriate to describe the chronic, nonremitting, deteriorating dementia praecox that was common many generations ago. Now it can be used most fruitfully to describe the fundamental vulnerability to schizophrenia rather than the time-limited episodes which intermittently appear.

**Proposed Research** - Developing indexes of vulnerability and episodes.

If we are to accomplish the primary prevention of episodes of schizophrenia, and ultimately eliminate the underlying vulnerability, we urgently require research enabling us to distinguish between two types of indicators. The first group of indicators mark the trait of vulnerability -- i.e., they stably and reliably differentiate vulnerable individuals from those with
immunity to schizophrenia and various other gradations of vulnerability. The second type of indexes are those fluctuating, state-dependent markers that can be used to pinpoint the presence and course of the episodes, and their termination.

It is important at this point to note the difference between markers of vulnerability and underpinnings or causes of vulnerability. For example, the Wasserman test is a marker for syphilis but may be neither a cause nor an underpinning of the disorder. We do not know at this time whether the indexes to be discussed below reflect the causes of mental disorders or merely mark the vulnerable individual. We do not even know whether they are stable traits or transient states signalling the presence of the episode. This question is one we have just begun to investigate.

As we survey the pool of measures that have commonly been found to differentiate schizophrenics and normals, we face a morass of techniques which all too commonly share similar weaknesses. To summarize these sore points as briefly as possible, almost every technique shows that patients perform more poorly than normal controls. Although it is often the researcher's intent to attribute this discrepancy to central nervous system differences or disturbed information processing, other competing hypotheses loom prominently as viable alternative explanations of the data. Rarely can we rule out the arguments that patients failed to understand the instructions, were less attentive or less motivated to perform well, or applied different criteria to determine their responses. The Biometrics Research Unit staff has spent years trying to circumvent these problems by applying four maxims: (1) tasks should be as simple as possible; (2) verbal reports should be avoided wherever possible; (3) forced choice methods should be utilized to eliminate criterion problems and signal
detection analyses used when criterion cannot be eliminated by forced 
choice methods; (4) tasks on which patients perform "better" than normals 
should be preferred whenever they are available.

We have with the assumption emanating from the neurophysiological 
model that one of the etiological source of schizophrenia is to be sought 
in the way the central nervous system of schizophrenics processes incoming 
information. Our psychophysiological laboratory, under the direction of 
Dr. Samuel Sutton, has been investigating sensory and perceptual responses 
to energy stimuli for the past several decades. They have limited their 
investigation largely to the responses to the first 1000 msec following 
stimulation in the hope of reducing the influences of past experience, 
motivation etc., as much as possible. Those (Barook, Sutton and Zubin, 
1958) that have paid off have been the following: (1) crossmodality reac-
tion time; (2) pupillography; (3) sensory integrations in vision; (4) aud-
tory thresholds and masking in audition and (5) evoked potentials. We do 
not have the time to present all of these findings but will limit ourselves 
to a brief description of the sensory integration findings and the auditory 
findings.

According to the Bunsen-Roscoe Law or Block's Law intensity and dura-
tion of a stimulus can be reciprocally interchanged and yet leave the re-
response invariant, as long as the total energy of the stimulus (product of 
intensity x duration) remains invariant. This law holds for only brief 
periods following the stimulus, i.e., for the critical duration in which 
complete integration takes place. For the particular set up in our labora-
tory, this critical duration for schizophrenics lasts only from 4-6 msecs, 
while the critical duration for depressives and normals lasts much longer. 
Only those schizophrenics who have high scores on the dimension of thought
disorder show this reduced critical duration. Thus, it appears that thought disordered schizophrenics show shorter critical durations than the other groups. Moreover, on the specific test procedure that is used, their responses indicate a discrimination that is not possible for normal subjects.

The auditory technique does not differentiate between schizophrenics and normals but between affective disorders on the one hand and schizophrenics and normals on the other hand. First, the auditory threshold under forced choice conditions is higher for the affectives. Secondly, the reaction time of the affectives is enhanced (shortened) when a 25 dB click is followed 15 msecs later by a 10 dB click, while the reaction time for schizophrenics and normals is not altered.

Given techniques which are, to the greatest extent currently possible, free of the strictures of lack of motivation or attention, we can set about separating those which index the trait of vulnerability from those which measure the presence of the episode. To do so, we have adopted two simple strategies. The first is to repeatedly monitor the same patients when they are at the height of an episode and when they have entered remission and reattained their premorbid functioning. Presumably the markers for the episodes will be those techniques which show anomalous patient performance during the height of the episode but which normalize as the patient regains his premorbid status. It is likely that comprehensibility of speech as measured by the cloze technique would be a good marker of the onset, duration and offset of an episode, since the patient's disturbed speech is one of the earmarks signalling the presence of an episode. In contrast, the indicators which stably differentiate patients from normals regardless of the presence of the episode, are potential markers of the underlying vulnerability. To cross-validate the indexes of vulnerability, we are testing
relatives of probands who share some portion of the genetic and probably also some of the acquired vulnerability to schizophrenic episodes. We predict that a substantial proportion of the relatives will also show anomalous performance on our measures of vulnerability. Some may also display abnormalities on the episode indicators, and our measures of life events will enable us to detect whether these individuals also share a recent history of severe life stress in common with the proband.

What implications does our vulnerability have for prevention? According to our view, the goals of prevention are to prevent any future episodes in individuals of demonstrated vulnerability. This can be accomplished by reducing the vulnerability of the person by means of drugs or behavior modification, by controlling the environment to reduce the frequency or intensity of the life events which formerly served as triggering events for an episode, or by reducing the threatening nature of such life events. Should another episode develop in a good premorbid who has a high level of competence, no special intervention needs to be undertaken, since, once the episode is ended, he will return to his good premorbid status. In the case of the poor premorbid, efforts will have to be exerted in order to uplift his competence, so that when his episode ends, he will be able to cope with life exigencies better than he could premorbidly.

Whether a reorganization of society is necessary as Commissioner Okin has suggested before we can deal with mental disorders adequately is debatable, since there are other factors besides the ecological which seem to be sources of mental disorder. Only continued investigation of each of these models ranging from the ecological to the genetic will give us the necessary answers on how to deal with the problem. Any wholesale adoption of one model over the others before the evidence is all in, is bound to lead to false solutions and to increase rather than decrease of pain, suffering and needless expense.


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