VULNERABILITY AS THE COMMON DENOMINATOR FOR ETIOLOGICAL MODELS OF
PSYCHOPATHOLOGY

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Psychopathology, the science underlying the diagnosis, prognosis, treatment and evaluation of outcome of mental disorders has been characterized by some as suffering from a plethora of theories and a paucity of facts, and by others as plagued by too many facts and too few theories. In truth we have a great plenty of both but our difficulties stem from their failure to intermesh. As Wittgenstein ( ) once said, "Our problems and methods pass one another by." One way of preventing such bypasses is to construct a scientific model based on the theory, and subject it to testing against observed facts. Such a model is composed of a parsimonious number of dimensions from which specific testable hypotheses can be drawn. It is essentially a reconstruction and simplification of nature from one point of view for the purpose of studying a given phenomenon. A poem by Edna St. Vincent
Millay I came across recently seems to probe the dilemma to its true depth:

"Upon this gifted age, in its dark hour,
Rains from the sky a meteoric shower
Of facts ... they lie unquestioned, uncombined.
Wisdom enough to leech us of all our ills
Is daily spun; but there exists no loom
To weave it into fabric..."

The loom for weaving the wisdom into fabric is in our case the scientific model. While there are many scientific models for explaining the etiology of mental disorders and I have pointed to at least six of them in various places, (Zubin, 1972) today, I would like to distil their essence into one elixir -- that of vulnerability.

Let us first begin with an assumption which has won many adherents in both the physical and mental health camps (Audy) namely, that health depends on the individual's ability to rally from insults whether chemical, physical, infectious, psychological or social. Thus, health and disease can be seen as the varying states of a person's dynamic equilibrium with his environment, both external and internal. When this equilibrium is pushed out of balance disease ensues. The ability to resist the environmental stressors we will call vulnerability. It indicates the degree to which one is capable of maintaining his health despite external and internal threats to his homeostasis and
integrity. If his resistance is insufficient, he will succumb and develop an episode of illness. Since the same stressful events do not inevitably produce disorders in all of us, we need to recognize the existence of individual differences in vulnerability to the same stressors. The highly vulnerable succumb readily to even mild stressors while the least vulnerable may not succumb even to the most severe stressors.

The relation between vulnerability and stressors is shown in Graph I

Insert Graph I

It will be noted in Graph I that as long as the stress is below the threshold of vulnerability, the individual remains well, responding to the stressor in an elastic, homeostatic way, within the bonds of normality. When the stress goes above the threshold line, the person may develop a psychopathological process, which we shall call an episode. As the stress recedes below that line the actual psychopathological process, i.e., the episode, will end, and the person begins to return to his preepisode status.

What are the facts which have led me to appeal to vulnerability to life event stressors as a suitable common denominator for the various etiological models for the mental disorders?

First is the well-documented finding that life event stressors play an important role in the development of both physical and mental disorders as shown in the works of Holmes ( ), Rahe ( ), George
Brown ( ), Dohrenwend ( ), and many others. A life event stressor refers to such events as bereavement, promotion, marriage, divorce, etc. The readjustment made necessary by their occurrence induces the stress which may by itself trigger an episode, or do so with the help of accumulated prior similar stresses.

This field of investigation is still quite new and the available techniques for defining the boundaries of life events and their taxonomy are still to be worked out. Some investigators have failed to find triggering life events in some of their patients, but it is at least a tenable assumption that with better instrumentation such triggering effects, especially if internal events are also considered, will be found in most instances.

While the underlying vulnerability would be explained differently by the various etiological models, some focusing on the external events while others on internal events or structures, the importance of these life event stressors in eliciting an episode is quite clear.

Second is the fact that the course of mental disorder is not a continuous one. The patients with affective disorders may have a varying series of episodes leaving them quite mentally clear between episodes. Even the course of schizophrenia is no longer regarded as continuous, with interepisode periods of normalcy in the majority of patients who have recurring episodes.
Third, is the fact that good premorbid personalities tend to recover from their episode, while poor premorbid personalities tend to persist in their episode. This seems to hold true of most mental disorders (Zubin, 1974). We had found this to be true in our own review of prognosis in schizophrenia (Zubin, et al.) and the literature corroborates our finding both before and since our study was completed. One of the most striking corroborations comes from the earlier paper by Eliot and Patrick Slater in their classic heuristic theory of neurosis (Slater & Slater, 1944). They point out that their theory, "...relates the problems of psychiatry to the general concept of human variability which has been developed by psychometric psychology, and which again is related to the system of applied logic which has been found suitable for the biometric sciences as a whole. It therefore places at the disposal of psychiatry the scientific techniques, particularly those of statistical analyses which have been developed for the biometric sciences by Galton, Pearson, Fisher and others."

A fourth fact that we need to take into account is the distribution of coping ability which varies considerably in the general population independently of mental disorder. Good coping ability characterizes those who are quite capable of dealing with the usual exigencies of life involved in social, marital, occupational and general adjustment.
Poor coping ability characterizes those whose adaptation is on a very low level. Armed with these three concepts — vulnerability, life stressors and coping ability, we can proceed to establish the structure of our framework for models of etiology based on the vulnerability of man to life event stressors.

Let us first consider the relation between coping ability and premorbid personality which is so important in the outcome of an episode of illness. Why should premorbid personality be so important in recovery? Why should the good premorbid recover from their episode and the poor premorbid persist in their episode endlessly. If we regard the episode as a response to the stress induced by life events, we can assume that once the episode ends, the former patient will return to his premorbid or preepisode adjustment and continue life where he left off before the episode overtook him. If he was able to cope premorbidly and if the episode did not reduce his coping ability, he should be able to resume his former place in life with little or no change. What happens in the case of the poor premorbid personality? Why does he appear to remain in his episode indefinitely? There are 3 possibilities: (1) the episode actually continues indefinitely; (2) the episode ends as in case of the good premorbid, (3) the episode reduces coping ability. As to the first possibility, that the episode endures in the premorbid, there may be a small fraction of cases in which this occurs.
I would like to propose that even in the case of the poor premorbid, the episode comes to an end in the vast majority of cases, even as it ends in the good premorbid. The reason why the poor premorbid is not placed in the recovery category when his episode ends is that the premorbid level of adjustment he returns to postmorbidly is so poor that he cannot cope with life exigencies, just as he could not prior to the episode. It is in fact difficult to tell whether the episode is ended since clinical judgment of recovery should be based on a comparison of the patient’s coping ability at any point with his preepisode coping ability, something which is rarely known or investigated.

As for the patient whose coping ability has been reduced by the episode, he may be regarded mistakenly as still ill despite the ending of his episode, if the drop in his coping ability is striking. This drop in coping ability may be the direct deteriorating effect of the psychopathological process or an iatrogenic effect such as the classic Social Breakdown Syndrome of Gruneberg’s.

Thus, so-called chronic patients may be in reality free of their episodic illness, but suffering from defective adjustment to society either because of their originally poor coping ability or because of the reduction in their coping ability brought on by the episode of illness.
Causes of Vulnerability and its Definition

The scientific models of etiology for which vulnerability serves as a common denominator are many and varied. In reviewing the field I found that they could be classified into two foci: (Zubin, J., 1972) those based on the field theory approach and those based on the atomic approach. In the field theory models the individual occupies a nodal point at the intersection of the field of forces that impinge on him and which determine his well-being or illness, while in the molecular approach each individual is a discreet unit with his own individual characteristics determining his status. The ecological model represents the extreme end of the spectrum for the field theories, while the genetic model represents the opposite end of the spectrum for the atomic theories. The developmental and learning theory models lie closer to the field theories but take account of individual characteristics, while the internal environment and neurophysiological models lie closer to the atomic theories but they too are not unaffected by field forces.

I do not have the time to describe these models except very briefly (slide of models)

(Give brief sketch of models)

After reviewing these etiological models one is left with the feeling that there must be a common denominator running through all of them and this is vulnerability. It should also be realized that none of the implicated etiological factors works in isolation, although our scientific models tend to treat them as such. There is always an
interaction between all of them, although the ingredients may vary from one disorder to another. Thus, it is quite possible that for schizophrenia, a higher loading will appear for the genetic component than is the case for neurosis, and that for depression, a higher loading will appear for the internal environment than would be the case for character disorders. The developmental model contains both atomic aspects (maturation) as well as field theory aspects (dependence on peers).

In defining vulnerability we might separate it into two major components corresponding to the two major types of etiological models — the atomic and the field theory models. The wired-in genetic component of vulnerability is well understood in so far as the genetic propensity for being vulnerable is laid down in the genes and reflected in the internal environment and neurophysiology of the organism.

The non-genetic or acquired vulnerability is a little more difficult to describe. If we consider the evidence that early experience, early exposure to certain stresses and specific disorders leave their mark and tend to either facilitate or inhibit the development of a disorder in later life, we can regard this acquired vulnerability to be dependent upon the memories of the organism for past experience or on the scars such experiences leave behind them.
Such vulnerability induced by past experience is well known in physical disorders. According to the World Health Organization, about 75-80% of all cancers are triggered off by early exposure to environmental agents such as industrial chemicals. No doubt exposure to viral infection and solar ultraviolet radiation are often contributing agencies. The cancer itself may not become apparent for decades yet it is often traceable to these early experiences. What role heredity plays in the development of this type of vulnerability remains to be determined.

The vulnerability of an organism may be lowered, rather than raised, as the result of earlier experiences. For example, rats develop an aversive reaction to toxic substances they have been exposed to previously, even though the poison may have been ingested in an unconscious state, and the noxious effects may not have appeared for hours or days later (32e). In general, what an immunizing exposure to a disease, or an immunizing agent does, is to lower the vulnerability of the individual.

Apparently the long term memories of prior experience either decrease or increase vulnerability. From this point of view, life events can be regarded as immediate or short term memories which serve to elicit the encoded long-term memories of genetic or acquired vulnerability. Thus one might differentiate between vulnerability and stress-producing life events by hypothesizing that vulnerability consists of long-term memories lodged either in the DNA molecules genetically or in the proteins that constitute memory traces in the nervous system, while life events produce immediate or short-term memories serving as triggers for an episode.
It is quite possible that the disorder triggered by life events in the genetically based vulnerabilities is no different from those triggered in the non-genetically based, since the number of ways in which behavior can be altered by stress producing stressors is not infinite. Whether the therapeutic strategies required for improvement are the same for both types of vulnerabilities remains to be determined.

Indices of Vulnerability

It is obvious that at this point we have no way of detecting directly the presence of vulnerability, or of measuring it. We can, however, by making certain assumptions based on our scientific models, select populations which presumably vary in degree of vulnerability. Thus, if we accept the ecological model, we would expect the occupants of noxious ecological niches to be more vulnerable than those of more favorable niches. If we adopt the developmental model, we would expect that children whose mothers underwent severe toxemias of pregnancy or birth trauma, adolescents who are deprived of satisfactory peer groups, and students who are frustrated by lack of opportunities would be highly vulnerable.

Similarly, the learning theory model would lead us to expect that children growing up in disorganized families, or individuals who have developed low self-regard because of continued failure, would be highly vulnerable to life event stresses.
The internal environment would lead us to believe that individuals who possess certain biochemical anomalies would be vulnerable, while the neurophysiological model would lead us to expect that individuals who show certain psychophysical and neurophysiological anomalies would be prone to schizophrenia.

One obvious approach has been pioneered by Haston and Denney (18) and by Mednick and Schulsinger (27) in the selection of high-risk populations genetically. Under the assumption that the genetic component is important in the development of schizophrenia, it follows that the offspring of schizophrenic parents should have a higher risk of developing schizophrenia than the offspring of parents free of schizophrenia. This indeed is the case, with the offspring of one schizophrenic parent having a risk of 15 percent of developing schizophrenia (10 to 15 times as high as in the offspring of normals) while the risk for offspring of parents both of whom are schizophrenic rises to 40 percent. This approach presents us with populations of graded risk which could yield significant information regarding the interaction of the genetic with the other etiological models. Keeping the genetic component constant we can investigate the status of individuals possessing the same risk (e.g., monozygotic twins, children of two schizophrenic parents) but who are discordant with regard to schizophrenia, to determine how the discordance arose, i.e., in what respect they differed with regard to the other etiological models.
Unfortunately, the other models have not yet provided us with measures of vulnerability as good as those which genetics is equipped with in measures of consanguinity. In this respect, the social scientists, developmentalists, behavior theorists, biochemists and neurophysiologists have failed psychopathology.

There is some indication that the ecological model which has thus far looked to measures as indicators of vulnerability may instead provide measures of vulnerability by studying the early life-events that occur in the niche the person occupies. The scars that earlier experience leave in their wake may give a measure of vulnerability.
Summary

We can now summarize our proposition that vulnerability can serve as the common denominator for the scientific models of etiology as follows:
Chart A

Vulnerability as the Common Denominator

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

2. Episodes — develop when the stress exceeds the threshold of vulnerability.

3. Episodes — are time limited.

4. Outcome of Episodes — at end of episode returns to pre-episode status.
Chart A
(continued)

Vulnerability as the Common Denominator

5. Good pre-morbids return to good pre-episode status — recover.

6. Poor pre-morbids return to poor pre-episode status — mistaken as still ill — chronic.

7. Some good pre-morbids have a reduced coping ability after the episode and are mistakenly regarded as still ill — chronic.
From the point of view of vulnerability, therapeutic intervention takes on a new direction. The immediate goal of intervention is no longer to "cure" the patient, since no "cures" are available for any of the major psychopathologies. The goal is at the very least to permit the patient to return to his preepisode coping level. The long-term goal is to reduce vulnerability to future stressors. Most of our therapeutic efforts today concentrate on the good premorbid — those who can resume their former place in society once the episode is over. These, however, will get well any how for the most part, even without therapy. We should instead concentrate on the poor premorbid who even when the episode ends can still not cope with life. Perhaps an investigation of the good premorbid who fail and the poor premorbid who succeed may give us deeper insight in how to give a new impetus to our therapeutic efforts. for the prevention of future episodes

The long term goal of reduction of vulnerability may be achieved in two ways: (1) direct reduction by chemotherapy and (2) indirect reduction by desensitizing the patient to the life events that appear threatening to him. The latter can be achieved either by psychotherapy or by behavior modification with or without the help of chemotherapy. In general, the adoption of the vulnerability hypothesis should alter our therapeutic perspective profoundly.

The crisis now facing the Department of Mental Hygiene in our state is in part a function of the failure of our delivery of service system to recognize the high degree of vulnerability that characterizes
our mental patient. Only the provision of facilities in which this be vulnerability will/reduced would eliminate the revolving door casualties and our failure to recognize the essentially vulnerable nature of our former patients lies at the basis of our current crisis.