PSYCHOSOCIAL FACTORS IN SCHIZOPHRENIA
IN LIGHT OF VULNERABILITY THEORY

by

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I

This paper reviews some of the essential features of vulnerability theory as it has developed over the past fifteen years. Specifically, we wish to emphasize the role played in the theory by psychosocial factors, and to contrast our approach to schizophrenia with other, similar models currently discussed in the literature. The goal is to provide a clearer perspective on the manner in which our thinking about schizophrenia departs from more established opinions in the field and, perhaps, to stimulate enthusiasm for a novel approach to the conceptualization of this condition.

Reckoning according to medical time, it seems fair to conclude that we live in a Kraepelinian Age—an era that began around 1899 with the publication of the sixth edition of Emil Kraepelin's textbook of psychopathology. In this work Kraepelin suggests, for the first time, that the fundamental core of the
condition we now call schizophrenia is a biologically founded disease process which tends to progress towards a permanent mental enfeeblement, that is, what the nineteenth century termed "dementia" (Hare 1981:94). Despite the fact that Kraepelin's original model has been modified in a number of ways, the idea of an underlying malignant disease process still serves as the basis of most clinical thinking about schizophrenia. Even in the United States, where this perspective was temporarily eclipsed by Adolph Meyer's biosocial theory and Freudian psychoanalysis, its continuing vitality and attractiveness for members of the medical profession has been highlighted by the recent emergence of a neo-Kraepelinian school of psychiatrists (Klerman 1978, Guze 1978, Blashfield 1984), the members of which have played such a key role in the development of new and controversial classificatory tools—e.g., the Research Diagnostic Criteria (RDC) and DSM-III. In what follows, I wish to propose an unabashedly non-Kraepelinian approach to schizophrenia. This approach to conceptualizing the nature of schizophrenia has come to be known as "vulnerability theory" (Zubin 1963, Zubin 1976, Zubin and Spring 1977, Zubin and Steinhauer 1981, Zubin, Magaziner and Steinhauer 1983, Zubin, Steinhauer, Day and Van Kammen 1984). Briefly put, vulnerability theory starts out by calling into doubt the existence of an underlying, malignant disease process and propounds its greatest heresy in regard to the origin of chronic deficit states. It is this body of psychiatric heterodoxy that we shall describe in some detail in the next two sections of this paper.
Despite the development of greater technical sophistication in the search for aetiological factors, schizophrenia is still a disorder of unknown origin. Ongoing research in past decades has produced at least eight kinds of aetiological models, specific variations of which have been subjected to experimental investigation. These models can be classified as biological or environmental in character: The biological consists of the (i) genetic, (ii) internal environment, (iii) neurophysiological, (iv) neuroanatomical and (v) viral theories, while the environmental include (vi) ecological, (vii) developmental and (viii) learning theory approaches.

These aetiological models have been described in detail in previous papers (see Zubin 1971, Zubin and Spring 1977, Zubin and Steinhauer 1981, and Zubin, Magaziner and Steinhauer 1983) and will only be briefly characterized here. The genetic model postulates that the aetiology of schizophrenia is to be sought in the chromosomes of the individual; unless the alleles for the disorder are inherited, no schizophrenic disorder will develop. An internal environment model suggests that the aetiology of the disorder inheres in the biochemistry of the body; work in this area focuses on neurotransmitters, metabolic and other chemical properties not directly attributable to genetic influences. A neurophysiological model assumes that the manner in which the
central nervous system processes information is related to the source of fundamental deviation producing the disorder. Neuroanatomical and viral models have experienced a recrudescence in recent years, the former postulating that schizophrenia results from structural defects in brain anatomy and the latter hypothesizing that we are actually faced with an infectious disorder.

In contrast to the preceding models, environmental theories implicate specific features of the schizophrenic's sociocultural and physical surroundings in the aetiology of the disorder. Ecological approaches emphasize the aetiological significance of factors like socioeconomic status, social isolation, immigrant or marginal status. Developmental theories seek the source of the disorder in the complex process of psychological change and physical maturation which individuals undergo as they pass from a fertilized ovum through the various succeeding stages of the life cycle. Finally, a learning theory model postulates that the reinforcement history of the individual explains the origin of the habits and behaviors that are eventually termed schizophrenic.

Neither biological nor environmentally oriented research has yet been able to isolate an aetiological factor that is necessary and/or sufficient for the emergence of schizophrenic disorders. It now seems likely that the aetiology of the disorder involves patterns of interaction between components of various models. Vulnerability theory was developed for precisely this
reason; it provides a superordinate framework for the interaction of the several aetiological models outlined above.

The vulnerability model proposes that a certain proportion of the general population is endowed with a level of vulnerability which, under suitable circumstances, will express itself in an episode of schizophrenic illness. Each aetiological model offers suggestions about the possible origins of such vulnerability. There are, however, three somewhat novel elements in our formulation. First, we acknowledge numerous contributions to an individual's degree of vulnerability, ranging from his genetic inheritance to his acquired propensities. Second, we are concerned with measuring vulnerability. That is, we are attempting to capture empirically those characteristics of individuals that might predict the probability that a schizophrenic episode will develop. Third, we distinguish between a "vulnerability" to schizophrenia, which is regarded as a relatively permanent, enduring trait, and "episodes" of schizophrenia, which are viewed as waxing and waning states. In this connection, one important area of continuing work is the search for specific "markers" associated with either the patient's underlying vulnerability to illness or the occurrence of episodes of the disorder (Zubin and Steinhauer 1981).

The vulnerability hypothesis assumes that the concept of schizophrenia does not imply a chronic disorder so much as a permanent vulnerability to develop the disorder. Episodes are
usually time limited, arising in the wake of life stress and abating when its aftereffects dissipate. When the episode terminates, the individual returns to his premorbid level of adjustment. Viewed in this way, schizophrenia becomes an episodic illness in the same sense as depression, epilepsy or allergy.

A vulnerable individual is one with sufficient aetiologcal loading, regardless of its sources, to have the potential for a schizophrenic episode. The possession of this potential does not, however, make an episode inevitable. Prior estimates (see Zubin and Steinhauer 1981:79) suggest that, perhaps, as many as three of every four individuals with an aetiologcal loading for schizophrenia never experience an identified, manifest episode of the disorder. A fact, by the way, that needs to be kept in mind when undertaking the task of choosing non-schizophrenic control subjects for biological and genetic studies. The actual probability for an individual to experience an episode of illness at some point in their lifetime seems to be contingent upon at least three kinds of factors: (i) the level of their aetiologcal loading for the disorder, (ii) the overall stressfulness of the immediate environment, and (iii) the individual's personal competence and learned coping skills (i.e., premorbid personality).

With regard to the initial factor mentioned above (i.e., aetiologcal loading) we will simply append a few additional remarks to what has already been said. First, it is our suspicion that a host of very different factors (both biological and
socioenvironmental) interact together to determine an individual's overall aetiological loading. And second, this overall vulnerability to illness is not a uniform threshold, but varies widely from one individual to another. In fact, it is probably the case that an individual's overall vulnerability to illness varies substantially as he matures physically and passes through the different stages of the life cycle. We suspect, for example, that there may be a critical, but non-specific developmental change occurring in late adolescence which results in an increased vulnerability to manifest episodes of schizophrenia among individuals who already have a certain liability to illness.

The second category of factors mentioned above was "life stress." Vulnerability theory suggests that a certain critical level of life stress must be present in order to provoke the manifest appearance of schizophrenic symptoms. In this connection, there are two matters that must be noted at the outset. First, there is the fact that "stress" is a notoriously difficult concept to define. For the time being, we intend to follow Spring's (1981:26-28) approach, defining stress in terms of "environmental inputs," the significance of which are measured by "disruptions or alternations in biological, physiological, emotional or behavioral homeostatic functioning." Second, there is the problem that episodes may onset in a number of ways. Sometimes we observe a clear and precipitous change, with psychotic symptoms suddenly disrupting a state of apparent
normal functioning. More often, however, we can trace a gradual increase in the number and severity of symptoms, a process which may occur over a matter of weeks or months. In the latter kind of situation, psychotic symptoms may develop in the absence of an obvious or arresting change in the individual's personality and behavior. This continuum of onset types, ranging from the precipitous to the gradual and insidious, is difficult to explain without an equally versatile and pliant approach to the concept of life stress.

In our own research we have tried to move in the direction of life stress models that are sufficiently complex to approximate what we see in a clinical setting. As a part of this, we have come to think in terms of two broad categories of environmental inputs that affect the onset and the course of schizophrenic episodes. The first we have called (for the lack of a better term) "acute" or "immediate" stressors, a category that is essentially identical to the idea of stressful life events. Like other life events investigators (see Brown and Harris 1978:83, Dohrenwend et al. 1978:207), we shall define acute stressors in terms of changes occurring in an individual's taken-for-granted, routine patterns of everyday life. These changes must be datable (in terms of impact or first awareness) and relatively non-repetitive in nature (Day 1981:100-101). Recent reviews (see Rabkin 1980, Day 1981, Dohrenwend and Egrí 1981) of the relevant empirical studies generally conclude that acute stressors (i.e., life events), although clearly important, have only limited implications for the onset and course of
schizophrenic episodes. In other words, the totality of environmental inputs capable of provoking schizophrenic episodes in vulnerable individuals cannot be fully subsumed under the rubric of recent life events. Perhaps, the best evidence to this effect are the studies (Brown and Birley 1968, Birley and Brown 1970, Brown et al. 1973, Jacobs and Myers 1976, WHO 1984) which find that no more than half of the schizophrenic patients experiencing a precipitous onset of symptoms show any causal effect of recent stressful life events. These findings suggest that some other kind(s) of environmental inputs must be operating on the patients' liability to illness if life stress is to play the pivotal role proposed for it by vulnerability theorists.

It was this line of reasoning that led us to place a growing emphasis on the significance of potentially negative environmental inputs which have been shown to be a commonplace or repetitive feature of many schizophrenic patients' social milieu. In formulating the concept of "chronic" or "iterative" stressors, we have drawn heavily upon the research of numerous other investigators (e.g., John Wing, George Brown, Julian Leff, Evelyn Bromet, Michael Goldstein, James Jones, Jeri Doan, Eliot Rodnick, Gerard Hogarty, Margaret Singer and Lyman Wynne), as well as the data collected by the staff of our Biometrics Research Unit at the Highland Drive Veterans Administration Medical Center in Pittsburgh, Pennsylvania. To date, we have been working with the assumption that we can locate and empirically measure at least four classes of environmental settings that are
routinely stressful for individuals with a vulnerability to episodes of schizophrenia:

i. Cognitively confusing environments. The key aspect of these environments has been termed "communication deviance" (CD). Singer and Wynne (1967:148) point out that CD probably is a consequence of underlying attentional defects in the relatives of schizophrenic patients. Such individuals fail to establish and maintain shared foci of attention when they interact with others. As a result, the other individual in the transaction tends to become "lost, confused and is finally left with a sense of pointlessness that is distressing, yet hard to articulate."

ii. Emotionally critical or intrusive environments. A number of recent studies (see Kuipers 1979, Leff et al. 1982, Vaughn et al. 1983, Faloon et al. 1982) have implicated manifest expressions of emotional over-involvement, criticism and hostility by the close relatives of schizophrenic patients with relapse episodes of the disorder. These studies, carried out under the rubric of "expressed emotion" (EE) suggest that negative assessments of the patient's behavior or self, or an invasive over-concern for the patient's welfare may provoke florid episodes of illness.

iii. Overly demanding environments. It has been shown that family settings (Katz 1966, Katz et al. 1963, Hogarty 1975) in which relatives' expectations are too demanding of the schizophrenic patient may lead to a negative outcome. Similarly, a number of studies (Hogarty and Goldberg 1973, Hogarty et al. 1979) have indicated that rehabilitation programs that require schizophrenic patients to perform at too high a level may provoke relapse episodes of illness. The common denominator in both situations are demands for behavioral performance by family members or therapists that are beyond the capacities of the schizophrenic patient.

iv. A threatening or demoralizing physical environment. The characteristics of the neighborhood and the immediate personal environment are hypothesized to play a part in exacerbations of schizophrenic illnesses (see Zubin and Spring 1977). Here we may include personal safety, as well as the amenities and overall quality of life experienced by the
patient. This factor is the least studied of the above and the one that has the greatest potential importance for the group of patients lacking a family.

Two caveats need to be mentioned in connection with this list of chronic or iterative stressors. First, the four categories of stressors outlined above should not be viewed as mutually exclusive. In fact, it would not be surprising to discover that patients who are prone to experience one category of chronic stressors (e.g., high EE) are likely to also be subject to others (e.g., an over-demanding environment). The probable effect would be to produce highly "toxic" environmental situations characterized by very negative clinical outcomes. Second, we do not wish to claim that the above list is complete or comprehensive. Additional specific factors may be shown to exist in future research or it may be necessary to eventually include a general category of "ongoing difficulties" similar to the one used by Brown and Harris (1978:130-137) in their depression study.

A simple description of our proposed categories of chronic stressors tends to beg a number important questions. For example, how do the effects of chronic stressors interact with life events (i.e., acute stressors) and what influence may different patterns of (acute and chronic) stressors be expected to exert on the mode of onset of schizophrenic symptoms? We will return to some of these questions in Section III, but first it is necessary to review the role played in this model by the patient's premorbid
personality.

The role of premorbid competence and coping, the third factor mentioned above, is rather crucial to the vulnerability model; it represents the individual's capacity to actively respond to potentially stressful challenges presented by the environment and, perhaps, to fend off the impending onset of an illness episode. The vulnerability model views premorbid competence and coping as personality factors which interact with but remain independent of both environmental inputs and the individual's liability to illness. In this connection, vulnerability theorists strongly reject the notion that poor premorbid personality functioning represents early evidence for the existence of an underlying, malignant disease process, the natural evolution of which eventually leads to an active case of schizophrenia. Coping should not be confused with competence. The former term refers to the attitudinal and motivational stance of the individual faced with a task, while the latter signifies the individual's abilities, skills, and accumulated know-how in solving life's problems. It would be expected that any individual, regardless of his/her vulnerability to episodes of schizophrenia, will experience routine difficulties dealing constructively with life's challenges to the extent that deficits exist in either one or both of these essential personality functions. In other words, it is the potential consequences (i.e., an episode of illness) rather than the character of the deficits that separates an incompetent schizophrenia-prone individual from an incompetent normal.
III

How are we to conceptualize the interaction among the several key components of the vulnerability model - i.e., a personal liability to illness, acute and chronic environmental stressors, and premorbid competence and coping? It has already been noted that vulnerability theorists generally assume that a certain level of life stress is necessary to provoke the manifest appearance of schizophrenic symptoms. The simplest example is the acute "triggering" event that serves to elicit a life crisis for a vulnerable individual. Events of this sort are likely to be undesirable, novel, unexpected, unanticipated and uncontrollable; they often produce losses, and frequently require substantial readjustments in the individual's daily routine. Within the framework of this simplified example, the actual severity of the event required to set off a sustained episode of illness in a specific individual is inversely related to the individual's overall level of aetiological loading for the disorder (see Figure 1). If the acute stress of the triggering event fails to exceed the individual's threshold tolerance (i.e., the line demarcating well from ill on Figure 1), then a transient crisis may ensue but a full-blown episode is unlikely to occur.

INSERT FIGURE 1 ABOUT HERE
Fig. 1. Relation between vulnerability and challenging life events. The equation for this hypothetical curve is $VS = K$, where $V$ is the degree of vulnerability, $S$ is the degree of stress induced by the challenging event, and $K$ is a constant.
Research has demonstrated that a simple acute life stress model, similar to the one outlined above, is not adequate to the task of explaining the variety of circumstances found to be associated with the onset of schizophrenic disorders. It has become clear, for example, that the effects of acute stressors can be amplified or tempered by a number of other factors, not to mention the fact that a certain proportion of schizophrenic episodes appear to onset in the absence of any obvious external, acute stressors.

We are currently involved in research designed to test our belief that onset patterns of the kind outlined above can be explained through the action of the other key variables in the vulnerability model - i.e., chronic stressors, premorbid competence and coping. By way of illustration, certain chronic stressors like a highly critical or intrusive family environment may act to exacerbate the consequences of a life event (e.g., losing a job or separating from a spouse) for the patient's clinical state. In essence, the patient's immediate relatives may respond to any evidence of pathology or to a failure to meet their performance demands in a highly critical fashion, creating the conditions for a further deterioration in the patient's clinical state. On the other hand, more benign social situations may act in a corrective manner, reducing or, at least, not exacerbating the effect of various stressors on the patient's clinical state. One example is the kind of household environment
that relaxes performance demands or permits a temporary withdrawal from social interaction when the patient begins to feel over-stimulated and vulnerable to psychotic experiences. This example leads us to the observation that, in the greatest number of cases, the most important clinical issue is not the initial source of the patient's over-stimulation - it may even vary across different illness episodes - but his own and the environment's response to the evidence of increased tension and pathology (cf. Herz and Melville 1980).

There is likewise a growing body of evidence to suggest that chronic stressors of the kind outlined above may play a causal role in the onset of symptomatology even in the absence of acute life event stressors (Leff and Vaughn 1980). At the present time, very little is known about how various kinds of chronic stressors may interact with one another and, perhaps, multiply the overall "toxicity" of the patient's immediate setting (see Doan et al. 1981). It would not be unexpected, however, if factors like an easily stimulated critical attitude towards the patient are found to be regularly associated with other stressors such as demanding performance expectations - e.g., the attitude on the part of close relatives or the members of a treatment team that the patient is not really as sick as he makes out and should be forced to improve upon his present level of social and occupational functioning. Even without a large number of studies in this area, there is strong reason to presume that these kinds of environmental inputs have the capacity, over extended periods of time, to erode the resilience of the patient's coping efforts.
and exhaust his/her resistance to psychotic experiences.

Another example, this time illustrating the significance of the role assigned to premorbid personality factors, concerns the higher rates of schizophrenia often observed in groups of recent immigrants. In his classic study of Norwegian migrants to the state of Minnesota, Odegard (1936) proposed the hypothesis that immigrant groups have higher rates of illness than either the host populations or their communities of origin because "psychopathic or early psychotic types" are more maladjusted and restless, leading them to emigrate at a higher rate than the members of other population groups. Our differences with Prof Odegard lie generally in matters of emphasis. Yet, they are not unimportant for thinking about the nature of schizophrenia. Odegard, for example, tends to assume that there is some kind of evolving disease process which, in its early phase, accounts for the restlessness and maladjustment of the migrant in his home community and, in its later phase, emerges as a manifest episode of schizophrenia. As we mentioned earlier, a vulnerability theorist, rather than regarding both factors as manifestations of the same disease process, would view deficits in premorbid personality functioning and the presence of an underlying vulnerability to illness as two independent variables, an approach, moreover, that rejects the sense of inevitability inherent in the idea of an evolving or progressive disease process.
From the perspective of vulnerability theory, it would be predicted that immigration to an unfamiliar cultural setting often provokes crises of competence, particularly to the extent that prior learned strategies for dealing with environmental challenges are no longer appropriate or effective. Such acute crises could be expected to be much more severe among individuals who had already displayed clear, pre-existing deficits in competence and coping prior to emigration from their home communities. In many cases, the unexpected and sudden inappropriateness of previously employed strategies only serves to further exacerbate the threatening character of the new cultural setting, effectively replacing a sense of mastery that previously cushioned the effects of stress with a growing susceptibility to experiences of frustration and despair. Given these expectations, it seems likely that individuals with a vulnerability to schizophrenia, when placed in this kind of situation, enter a period of increased risk for an episode of illness that may never have been experienced had they refrained from emigration. In other words, it is our belief that the often noted increase in the rates of illness among immigrant groups is not primarily a function of selection processes, as Odegard has suggested; instead, it our suspicion that these higher rates represent a real increase in the manifest incidence of the disorder, an increase that is due largely to an excess of episodes among vulnerable individuals whose premorbid levels of competence were sufficient to ensure at least a marginal degree of adjustment in their home communities.
Finally, the last two examples in this section briefly examine the significance of key factors in vulnerability theory for the prediction of good and poor prognosis schizophrenias. Clinical research (see Garmezy 1968) suggests that we can identify a group of poor prognosis (or "process") schizophrenics whose episodes of illness emerge from a confluence of aetiological and premorbid personality factors. Studies have found that patients of this kind often show the following sorts of characteristics: (i) an insidious onset, (ii) a long period of illness prior to identification, (iii) never married, separated or divorced, (iv) a poor and/or asocial form of premorbid adjustment, and (v) inappropriate or flat affect. As a consequence of these patients' extreme liability to illness, it would appear that environmental stressors need only be assigned a negligible role in episodes of the disorder. Defects in premorbid competence and coping are so severe, and general levels of adaptation so poor, that individuals with a substantial aetiological loading for schizophrenia are likely to become ill simply as a consequence of their routine involvement in everyday, uneventful patterns of social interaction (cf. Leff et al. 1973).

At the other pole of this continuum, we find a group of good prognosis (or "reactive") schizophrenics in whom we often observe (i) an acute onset of illness, (ii) a clear environmental precipitant, (iii) a brief duration of illness prior to identification, (iv) a definite change in premorbid personality and behavior, (v) married, (vi) a good premorbid level of social
and occupational functioning, (vii) a preservation of affect and depressive symptomatology. As a rule, vulnerability theory leads to the expectation that the greater the causal role played by acute environmental (i.e., life event) stressors, as opposed to chronic stressors, aetiological factors or premorbid personality, in the development of an individual's episode of illness, the better the chances of a favorable outcome. Dohrenwend and Egri (1981) provide the prototypical case of this principle. Drawing on the literature from wartime combat and civil disasters, these authors insist that it is possible, in the absence of an underlying aetiological loading, for extreme situations to provoke short-lived periods of psychosis that cannot be differentiated symptomatologically from more perduring episodes of schizophrenia. Schneider (1959) called psychoses of this form "psychic reactions;" for the most part, they are transient, self-limiting sorts of conditions, the acute symptoms of which rapidly disappear once the individual has been removed from the stressful situation. Within the framework of a vulnerability model, psychic reactions provoked by the collapse of a previously competent personality in the face of unexpected and extreme stress would be classified as incipient or "model" schizophrenias that remain situation specific and liable to a rapid remission precisely because the individual lacks an underlying vulnerability to illness.

IV

The two preceding sections provide a brief introduction to
vulnerability theory. We have reviewed the various components of the theory and given a number of examples illustrating the manner in which these components interact to produce episodes of schizophrenia. Within this framework, psychosocial factors play the role of aetiological agents, affecting the individual's core vulnerability to illness, as well as acting to influence the clinical characteristics, course and outcome of the disorder. In this section, vulnerability theory will be compared to a number of alternative models in the schizophrenia literature. By contrasting our views with those of other investigators, it is possible to highlight and to explore in greater detail some of the key implications of vulnerability theory for the way in which we usually think about schizophrenia.

We have chosen to discuss three specific models of schizophrenia (i.e., diathesis-stress theory, Wing's social psychiatry, and social response theory) all of which have important elements in common with vulnerability theory. The first, diathesis-stress theory, is taken primarily from the work of the geneticists Gottesman and Shields (see Gottesman and Shields 1967, 1972, 1976, 1982), but draws upon the contributions of a number of other investigators such as Slater and Cowie (1971), Rosenthal (1970), Falconer (1965, 1967) and Meehl (1962, 1973).

The roots of diathesis-stress theory lie in the growing sophistication of genetic theory that followed upon the failure
to replicate early findings (e.g., Kallmann 1946) indicating very high rates of concordance for schizophrenia among MZ twin pairs. The data emerging from numerous international studies suggested to Gottesman and Shields that what was being transmitted genetically was not schizophrenia, per se, but, instead, a liability to the disorder; an approach, moreover, that was not uniformly popular among geneticists and one that inevitably led them to propose a key role for environmental factors in the transformation of the genotype for schizophrenia into a phenotypical expression of the disorder. An important aspect of Gottesman's and Shield's approach was their rejection of single gene and single major locus theories of the individual's specific genetic liability to illness. Instead, they opted for a rather complex multifactorial/polygenic mode of inheritance. From this perspective, they argued that schizophrenia is most appropriately compared to abnormalities like diabetes, arteriosclerotic heart disease and hypertension.

The most recent version of the model proposed by Gottesman and Shields (1982) still draws heavily on the original thinking of Falconer (1965, 1967). It uses the concept of a "combined liability to illness" which is derived from at least five categories of factors: (i) a specific genetic liability (i.e., alleles necessary for the disorder); (ii-iii) a general genetic and environmental liability to illness (i.e., factors that interact with and multiply the effects of a specific genetic liability); (iv-v) genetic and environmental assets (i.e., factors that serve to reduce the individual's overall level of
liability to illness). Except for the individual's specific genetic liability to illness (i.e., i. above), which is Gottesman's and Shields' primary focus of interest, very little detail is provided about the nature of the general liabilities and assets included in this model. Despite this lack of specificity, they (1982:229, 1976:377, see also Smith 1970) remain confident in their estimate that, "the genetic heritability of the liability for schizophrenia is about 70%, and the cultural-inheritance component accounts for about 20% of the combined liability." In this regard, Gottesman and Shields (1976:378) note that, even though "environmental factors may contribute only about 20 percent of the variance of the combined liability to developing schizophrenia in the whole population, they will be critical in determining whether an individual with a genetic high risk breaks down." For any particular individual, the sum of this combined liability to illness varies across time as the result of "epigenetic augmentations and reductions in liability."

By adding the dimension of time to our model... we can represent a more dynamic and realistic view of the individual's trajectory across the epigenetic landscape. The intention is to incorporate the concept of changes in effective genotype by gene regulation (the switching on and off of genes by environmental inputs), by possible critical periods (prenatal and postnatal), and by ecological inputs (assets and liabilities) into a dynamic system.

When the sum of an individual's combined liability to illness exceeds a certain threshold value, the disorder is said to become manifest. Gottesman's and Shields' model uses two
specific thresholds, a lower one for schizophrenic spectrum disorders and an upper one for schizophrenia proper. Within this context, stressful life events are a crucial feature of the diathesis-stress model. The authors suggest that when an individual's combined liability to illness approaches either one of these threshold values, an acute stressful experience may push the individual over the threshold, triggering the manifest appearance of symptomatology. Under these conditions, life events serve as a necessary but not sufficient cause of illness. At the same time, there are also numerous cases in which the individual's combined liability to illness from genetic and non-specific factors is so extreme that schizophrenic symptomatology may become manifest without the necessity of a stressful life event. For these individuals, life events are neither necessary nor sufficient components of their illness.

The diathesis-stress model discussed above is more limited than vulnerability theory in the range of issues it addresses. The former model is concerned almost exclusively with questions of aetiology and onset, whereas the latter also makes predictions about the course and outcome of the disorder. If we confine ourselves to theoretical perspectives on the aetiology of the disorder, we find that there are few significant differences between the two models. Gottesman and Shields lay particular emphasis on the piling up of specific and non-specific factors (i.e., Falconer's combined liability to illness), finally leading to a threshold value beyond which a manifest (i.e., phenotypic) expression of the disorder will be observed. In truth, there is
only a single specific factor (i.e., a polygenic complex of alleles necessary for schizophrenia) in the model and a profusion of vaguely defined non-specific factors of both a genetic and environmental character. There is also a strong inclination on the part of these theorists to argue for the probable existence of an aetiological unity underlying the variety of phenotypic patterns currently included in the category of schizophrenia (Gottesman and Shields 1982:214-218). Vulnerability theory tends to favor a similar position regarding an underlying aetiological specificity, although we recognize, like Gottesman and Shields, that this position may eventually be proven wrong. For all practical purposes, the non-specific assets and liabilities of diathesis-stress theory are subsumed in the vulnerability model under (i) the inherited and learned components of premorbid personality and (ii) the interaction between the components of various aetiological models. To summarize briefly, Gottesman's and Shields' version of diathesis-stress theory is more specific than vulnerability theory concerning the factors hypothesized to affect the aetiology of schizophrenia. At the same time, it lacks a similar breadth of concern for clinical issues bearing on the course and outcome of the disorder. Beyond terminological preferences, the two models are in substantial agreement regarding aetiological issues and, in this area, at least, demonstrate what is, perhaps, an unexpected level of agreement between biologic and psychosocially oriented investigators.

The second model to be discussed comes from the work of Wing
and his associates (Wing and Brown 1971, Wing 1978a, 1978b, 1978c) at the Institute of Psychiatry in London, UK. This model is firmly rooted in Anglo-European traditions of descriptive psychopathology and more recent British thought about the role of social factors in psychiatric disorders (e.g., Lewis 1953, 1958). In contrast to Gottesman's and Shields' focus on questions of aetiology, Wing's model is primarily concerned with socioenvironmental factors affecting the onset, course and outcome of schizophrenic disorders.

Wing and Brown (1971:17) suggest that schizophrenic patients may display at least three distinct kinds of handicaps. First, "premorbid handicaps," such as low I.Q., a difficult personality, and social withdrawal, which usually exist prior to the onset of illness. The authors note that these handicaps may well be "early manifestations of a disease process before it becomes clinically manifest." Second, there are "primary disabilities" which are viewed as specific dysfunctions associated with schizophrenic disorders. Primary disabilities are of two kinds, negative symptoms (e.g., social withdrawal, flatness of affect, poverty of speech, lack of initiative, slowness, under-activity) and florid, also called positive or productive, symptoms (e.g., delusions, hallucinations, incoherence of thought, over-activity, odd behavior). These intrinsic impairments may exist together or they may appear as separate acute and clinical poverty syndromes (Wing 1978a). Finally, there are "secondary disabilities" which stem not from the illness process itself, but from the social and medical consequences of having been ill. In this category fall a
wide variety of potential handicaps like institutionalism, social stigma, the loss of self-confidence and self-esteem, induced dependence and a myriad of other deviant behaviors (Lemert 1951) which may be induced by the patient's social interaction with members of the medical profession, relatives and the lay public.

The basic tenet of this model is that schizophrenic patients are biologically vulnerable to both under and over-stimulating environments. Of course, the exact nature of this underlying core defect cannot be fully specified, but Wing seems to favor some kind of genetically transmitted defect(s) in arousal and attention. Specifically, the model suggests (Wing and Brown 1971:21-22) that,

...there are at least two fundamental processes at work. On the one hand, an under-stimulating social environment tends to increase symptoms such as social withdrawal, passivity, inertia and lack of initiative... On the other hand, there is the tendency to break down, with an effusion of florid symptoms, under conditions of social over-stimulation... These are general statements and the two processes sometimes vary together. Nevertheless, the sketch of a theory is discernable and depends upon the assumption that negative symptoms are a protective reaction against cognitive impairment. When the patient is allowed to withdraw, he does so, and the process can easily go too far. When he is not allowed to withdraw, but faced with what seem to be impossible demands, the underlying thinking disorder becomes clinically manifest in florid symptoms.

Let us recall that schizophrenia, as it is currently defined in most textbooks of psychiatry, involves a central or acute syndrome, as well as long-term states of impaired functioning (see Wing 1978a). Perhaps, the most important feature
of this model is the provision of a common sense, clinical and social framework for (i) explaining observed patterns of symptomatology in schizophrenic patients and (ii) predicting the effects of environmental factors on the course and outcome of the disorder. It serves as an alternative and useful corrective to theories which emphasize a "ruthlessly progressive" disease process amenable to little modification (Wing 1978b:125-126). In this role, Wing's model proposes a relatively concrete and researchable vision of the social conditions under which we may expect to observe maximum improvement in schizophrenic patients.

The optimum environment is presumably well structured, with clear lines of behavior laid down and a neutral type of social stimulation which does not lead to emotional over-involvement: both thought disorder and withdrawal are then minimized (Wing and Brown 1971:22).

The major points of difference between this model and vulnerability theory lie in the interpretation of premorbid handicaps and negative symptoms (i.e., the clinical poverty syndrome). Vulnerability theory assumes that schizophrenia is an episodic rather than a chronic disorder. Between episodes patients are expected to return to their premorbid levels of functioning, unless secondary factors unrelated to the individual's condition intervene to prevent this from occurring. For the vulnerability theorist, premorbid handicaps are independent (i.e., non-illness related) personality variables that interact with the patient's liability to episodes of schizophrenia, whereas Wing and his co-workers tend to view them as early subclinical manifestations of the patient's growing
sensitivity to over and under-stimulating environmental situations. The importance of this distinction should become clear once we have had the opportunity to discuss the differences in the two models with regard to negative symptoms (i.e., the clinical poverty syndrome).

The model proposed by Wing and his co-workers suggests that negative symptoms (or the clinical poverty syndrome) are an "intrinsic" or "primary disability," that is to say, a deficit in functioning which is a specific consequence of the patients' biologically grounded vulnerability to under and over-stimulating environments. Vulnerability theory, on the other hand, argues that only florid symptoms (or Wing's central syndrome) should be classified as primary handicaps. Negative symptoms and the clinical poverty syndrome are assimilated completely within Wing's category of secondary or external disabilities. In other words, vulnerability theory takes the position that negative symptoms are neither an inevitable (Kraepelin), nor an intrinsic (Wing) feature of schizophrenic disorders. It is proposed, instead, that the clinical poverty syndrome, as defined by Wing (1978b), is essentially an artifact or a social consequence of having been identified, labelled and treated as schizophrenic by medical specialists, relatives, close friends and other members of the patient's social network.

Going beyond the immediate issue of negative symptoms, the vulnerability theorist's insistence on the episodic nature of
schizophrenia raises a larger question — viz., how to explain the fact that a certain proportion of schizophrenic patients at least appear to remain chronically ill. There are four possible reasons that require mention. First, a substantial number of these patients may have been of such poor premorbid ability that their recovery from illness is difficult to discern. In contrast to affected individuals with good premorbid skills, who resume their roles in society and recover unremarkably, these "pseudo-patients" remain unable to cope adequately with their social milieu even though their episode of illness has terminated. Second, there is a group of chronic patients whose coping ability deteriorates far below its premorbid level as a consequence of factors like isolation in the hospital or community, labelling and a loss of social skills. Gruenberg (1967) has described a "social breakdown syndrome" occurring in such patients, a condition frequently mistaken for permanent psychopathology. This category also includes the vast majority of patients suffering from what has been called the clinical poverty syndrome. Third, some patients recover only briefly before falling back into an episode; their periods of emergence from the disorder are so short-lived as to often go unnoticed. A situation of this kind may be brought on by one (or a combination) of two possible situations. Either the patient lives in (or returns to) such a stressful social environment that any clinical improvements are quickly negated or the patient is so vulnerable that he may be catapulted back into an episode of illness by participation in uneventful social interaction (cf. Leff et al. 1973). Finally, there is probably a small proportion of patients — not more than
6-10% according to Bleuler (1978) — who may, in fact, remain chronically ill without a true remission.

The third model for discussion takes a position similar to vulnerability theory with regard to the matter of chronicity in schizophrenia. It was proposed by Waxler (1974, 1977, 1979) and draws upon a number of cross-cultural studies (Lambo 1960, Rin and Lin 1962, Fortes and Meyer 1969, Raman and Murphy 1971, WHO 1979, Waxler 1979, see also Day 1982) which indicate that schizophrenic psychoses with a good prognosis are more frequent among tribal peoples and populations living in developing countries than among the members of Western industrialized societies. For the sake of convenience, this may be called a "social response model."

Waxler's social response model is firmly grounded in Lemert's (1951, 1962, 1967) sociological theory of deviance. In brief, Lemert's theory argues that it is necessary to distinguish between primary and secondary forms of deviance. Primary deviance may arise from any source; it may characterize a large portion of the individuals in the population at any point in time, and it usually disappears of its own accord if ignored by an individual's culture-mates. Secondary deviance represents an amplification of the individual's primary deviance. Theoretically, secondary deviance is conceptualized as the individual's reaction to the response of his culture-mates to the individual's primary deviance. This process of deviance
amplification occurs through complex negotiations between the "deviant" individual and the various members of his social milieu.

Within this framework, Waxler (1974, 1977) argues that the acute phase of most schizophrenic disorders is a form of primary deviance that normally disappears in a short time, unless there is an adverse reaction to the patient's initial symptoms from the social environment. Residual defects and chronic illness states are viewed as a kind of secondary deviance that constitutes a socially induced amplification and prolongation of the patient's primary deviance.

The theory predicts that it is the social response to the person with psychiatric symptoms that is the prime determinant of whether he will remain sick... The career of the sick person is influenced, not by biological factors that cause severe or mild symptoms, curable or non-curable illnesses, but by the way his family, his neighbors, and, most significantly, his doctors respond to him once any sort of symptoms occur (Waxler 1974:383).

In a subsequent article, Waxler (1977) took up the question of why the beliefs and expectations found in non-Western, non-industrialized societies should be of the sort that infrequently promote chronic states of secondary deviance. Waxler theorizes that the culturally sanctioned responses to primary forms of deviance in small-scale societies have the functional purpose of reintegrating social groups (e.g., families, kin groups, villages) and, in so doing, indicate to the individual that he is suffering from a short-term remedial condition. Modern societies, on the other hand, are said to develop specialized institutions
(e.g., courts, prisons, mental hospitals, cf. Cooper and Sartorius 1977) that encourage (i) the separation of the individual from his family and community of origin, (ii) the bureaucratic stigmatization of the social self, and (iii) the imposition of professional expectations concerning recidivism and chronicity - all of which contribute to a deviance amplifying state of social alienation.

Waxler's (1974:394) model has a number of implications for the design of services and intervention strategies. In the developed countries, the primary goal should be to assist the patient in resisting the deviance amplifying effects of labeling, while not interfering with proven beneficial components of modern medical treatment. The author indicates that this plan could be carried out by broadening the base of current programs and implementing measures such as the following: increase community participation in the operation of clinics and hospitals; breakdown monolithic treatment systems into small, unrelated units; reduce the number of records kept on patients; and introduce alternative theories of mental illness. With regard to the developing countries, Waxler emphasizes the need to avoid recreating the same kinds of large, impersonal, medical-bureaucratic treatment systems that are currently found in many parts of the developed world.

Compared to the other models reviewed in this section, Waxler's social response theory pays little attention to the
matter of aetiology. It is not even clear whether Waxler believes that the primary deviance leading to the diagnosis of schizophrenia has some specific biological foundation or is simply a phase of disturbed behavior emanating from various non-specific causes.

Labeling theory is not interested in explaining the primary cause of the initial psychiatric illness, or "primary deviation." Whether psychiatric illness results from genetic factors, biochemical deficits, certain family dynamics or anomia is not important... It is assumed that many such symptoms appear in many people at one time or another; yet relatively few of these people find their way into formal treatment systems and into the sick role (Waxler 1974:382).

Vulnerability theory makes some very definite assumptions about the aetiology of the "primary deviance" involved in schizophrenia and the nature of the core defect underlying behavioral disturbances observed during manifest episodes of the disorder. This is clearly the most important distinction between the two models.

With regard to the issue of chronicity, social response theory is a bit more limited in the range of explanations it provides for persistent negative and residual symptoms - e.g., Waxler simply does not consider how poor premorbid coping may subsequently be mistaken for a chronic disease state, although she would probably agree strongly with position taken by vulnerability theorists. Still, within the limited domain where the two theories do overlap there is complete agreement. For the vulnerability theorist, Waxler's work serves as a useful
conceptual appendix to the theory of negative symptoms (i.e., clinical poverty syndrome) outlined above. It also brings to bear the findings from an additional literature on the issue of chronic schizophrenia, in effect, substantiating and expanding upon the views and evidence presented by vulnerability theorists (see Zubin and Spring 1977:117-119, Zubin and Steinhauer 1981:483-484, Zubin et al. 1983:558).

In this section, we have attempted to locate vulnerability theory in relation to models drawn from genetics, social psychiatry and anthropology. All of these models have something to say concerning the reactivity of the schizophrenic patients to their social environments, either in the development of the disorder (Gottesman and Shields), its onset (Gottesman and Shields, Wing), or its course and outcome (Wing, Waxler). Clearly, vulnerability theory is more comprehensive in the range of its concerns than any of the three alternative models discussed here. As a rule, the theory integrates different (conceptual) levels of pathological phenomena by giving increasing prominence to psychosocial factors as we move from the level of aetiology to that of onset, course and outcome of the disorder. It should also be clear by now that the single greatest divergence of vulnerability theory from more established medical models of psychopathology is its position on the matter of chronicity and, particularly, its insistence that social factors are the sole significant cause of negative symptoms. On this issue, a vulnerability model has far more in common with sociological theories of deviant behavior (e.g., Lemert, Waxler)
than with current psychiatric perspectives (e.g., Wing).

IV

In the introduction to a recent paper, the distinguished Swiss psychiatrist Luc Ciompi (1984) observes that,

It is perhaps surprising that someone who, in many years of research and clinical practice within a classical framework, has occupied himself with long-term studies and rehabilitation problems concerning chronic schizophrenia should finally ask the provocative question, whether the object of his entire research could be nothing but an "artifact." Such a question is provocative insofar as, in more than three-quarters of a century of scientific research, the chronic course of this serious and most important mental illness has been considered by several generations of investigators to constitute the very essence of schizophrenia.

The vulnerability model has its roots in a similar sense of skepticism and dissatisfaction with the medical model based on the tradition of Kraepelin's thought. What is really at issue is the assumed existence of a biologically grounded disease process that inevitably leads in the direction of intellectual and emotional deterioration. Although agreement with such a position is no longer obligatory for psychiatrists, neither is it a straw man that simply caricatures an outmoded point of view. Many clinicians and research workers continue to insist that there is no such thing as "good outcome" schizophrenia. In Section III of this paper, we also gave careful attention to a modified version of the traditional medical model. By stressing the reactivity of primary handicaps to environmental over and under-
stimulation, Wing has attempted to minimize the "ruthlessly progressive" notion of earlier theories, while maintaining the potential for chronic deterioration as a biologically grounded component of the schizophrenia. Yet, even a modified medical model that makes chronicity contingent on factors external to the disease process may not be sufficient to quiet growing doubts and criticism. Again, quoting Luc Ciompi (1984):

It would be entirely thinkable that the acute psychosis, in accordance with the suspicions of many researchers, represents, in analogy to an intoxication from amphetamines or hallucinogens, a predominately biochemical and somatic illness on a genetic base, while chronic forms could be much less an illness in the medical sense than a psychological and social consequence of specific situations following single or multiple occurrences of an acute psychotic episode.

Ciompi's comments serve to emphasize the extent to which vulnerability theory calls for a basic reorientation in our accepted modes of thinking about schizophrenic patients. Rather than looking upon them as sick people who once in a while experience periods of health, vulnerability theory suggests that schizophrenic patients are essentially healthy individuals who suffer one or more episodes of illness. Specifically, vulnerability theory assumes that the only permanent feature of schizophrenia is a liability to episodes of the disorder. Episodes are normally time limited; they are provoked by life stress and eventually abate under the impact of treatment and/or a modified social milieu. Following episodes of the disorder, patients are expected to return to their premorbid levels of functioning. Chronic illness states, when observed, are largely a
function of psychosocial factors. For example, the patient may find himself shuffled between a series of equally "toxic" social settings (e.g., family, hospital, community setting), never escaping for long the kinds of stressful circumstances that initially precipitated the disorder. Alternatively, it may be that what is observed is actually a form of "pseudo-chronicity" that arises when the patient's routine level of personality functioning is too low to permit a simple determination of the end of an episode.

It is also worthwhile to ask why the dynamics described by vulnerability theory remained unrecognized for so long? At least three reasons require mention. First, chronic patients accumulate over time even though proportionately their numbers are not very large. Oftentimes, these patients suffer from the effects of "toxic" environmental influences (e.g., iatrogenic, nosocomial, chronic stressors), a situation which keeps them in the hospital (or in treatment) and makes them available for ongoing medical evaluation. Second, longitudinal studies of schizophrenic patients have followed probands for only relatively brief periods of time (usually no more than a decade). The effect of this has been to focus attention on the phase of the overall course of the disorder during which patients' normally have maximum difficulties with psychopathology. More recently, however, the findings from a number of long-term (e.g., lifetime) follow-up studies have become available (Bleuler 1978, Ciompi 1980, Huber et al. 1980). Contrary to established expectations, the data from these studies indicate that the long-term trend in schizophrenia
is towards remission and improved outcome, rather than in the direction of deterioration or episodic recovery. Even patients who spend a number of years in chronic illness states seem to eventually demonstrate progress in the direction of a more benign outcome. A final factor that deserves consideration is the possibility that the outcome of schizophrenia has become increasingly benign since Kraepelin's time (see Zubin et al. 1983, Hare 1981, Odegard 1967). Here it may be suggested that the lack of effective treatment for acute episodes of the disorder and the generally poor social conditions found in the late nineteenth and early twentieth centuries contributed substantially to the uniformly disastrous sorts of outcomes associated with dementia praecox. The negative influence of these factors may have been further exacerbated by self-fulfilling expectations of the clinicians charged with the care of the severely mentally ill. We would speculate that improvements in the quality of life experienced by patients over the past thirty-five years and recent changes in the outlook of care providers have not only led to a greater frequency of benign outcomes, but made it possible to begin to distinguish between the real and artifactual components of schizophrenic disorders. Parenthetically, we may also remind ourselves that this is precisely the kind of evolution in the perception of the disorder that would have been predicted on the basis of vulnerability theory.

From a historical perspective, it therefore appears that
vulnerability theory is an idea whose time has come. In light of currently available evidence, even the most orthodox of our psychiatric colleagues would probably have to agree with Ciompi's (1984) conclusion that such a perspective, "cannot be pushed aside, in spite of the revolution its acceptance would entail for psychiatry."

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