Journal of Abnormal Psychology
1977, Vol. 86, No. 2, 103–126

Vulnerability—A New View of Schizophrenia

Joseph Zubin
Columbia University and New York State Psychiatric Institute, Biometrics Research Unit

Bonnie Spring
Harvard University and New York State Psychiatric Institute, Biometrics Research Unit

Although descriptive and etiological approaches to psychopathology have made notable advances, they seem to have reached a plateau. After reviewing the six approaches to etiology that now preempt the field—ecological, developmental, learning, genetic, internal environment, and neurophysiological models—a second-order model, vulnerability, is proposed as the common denominator, and methods for finding markers of vulnerability are suggested in the hope of revitalizing the field. It is assumed that exogenous and/or endogenous challengers elicit a crisis in all humans, but depending on the intensity of the elicited stress and the threshold for tolerating it, that is, one's vulnerability, the crisis will either be contained homeostatically or lead to an episode of disorder. Vulnerability and episode stand in a trait–state relation, and markers for each must be provided to distinguish between them.

The purpose of this article is to briefly review some of the classic approaches to understanding schizophrenia and to indicate the emerging trends. This overview suggests that after tremendous strides made in the last decade in both description and etiology, the study of schizophrenia has come to a standstill. Each of the various approaches seems now to provide only a partial answer. Perhaps for this reason, many proponents of each major theory indicate that they have gone as far as they can go with present methods and insights. A new, fresh view of the entire subject seems to be necessary if we are to move ahead. We propose that such a view emerges when we look at the common elements shared by all of the different theories.

One major stumbling block in studying schizophrenia has always been the tendency to intermesh description or phenomenology with etiology, as was done in the *Diagnostic and Statistical Manual* (*DSM II*) of the American Psychiatric Association (1968). We shall try to keep these two perspectives apart and begin with the area of description.

Descriptive Psychopathology

Written descriptions of the disordered behavior fitting our current category of schizophrenia have been available ever since the Hindu elders' observations in the *Ayur-veda* some 34 centuries ago. Moreover, such disorders were probably known to mankind long before they were recorded. We shall, however,

An abbreviated version of this paper was presented by Zubin at the occasion of the Distinguished Scientist Award given by Section III (Section for the Development of Clinical Psychology as an Experimental Behavioral Science) of Division 12 (Division of Clinical Psychology) of the American Psychological Association, Chicago, Illinois, August 30, 1975.

Portions of this article were also presented by Spring and Zubin at the First Vermont Conference on the Primary Prevention of Psychopathology, Burlington, Vermont, June 1975.

The preparation of this article has been partially supported by National Institute of Mental Health Grant MH 27342, "Vulnerability to Schizophrenic Episodes in Adults," U.S. Public Health Service. While working on this article the second author was supported for varying periods of time by a New York State Department of Mental Hygiene traineeship, an NIMH predoctoral fellowship, and a Scottish Rite Dissertation Research fellowship.

The authors are grateful for some of the material included in this article to the following members of the Biometrics Research Unit in 1975: Muriel Hammer, Anthropology; Kurt Salzinger, Behavior Analysis and Modification; Joseph L. Fleiss, Biostatistics; Barry Gurland, Diagnosis and Psychopathology; Robert L. Spitzer, Evaluation; Denise Kandel, Family and Youth Research; Ruth Bennett, Gerontology; Samuel Sutton, Psychophysiology; and David Wilder, Sociology.

Bonnie Spring is now at the Department of Psychology and Social Relations, Harvard University.

Requests for reprints should be sent to Joseph Zubin, who is now at the Veterans Administration Hospital, Highland Drive 151R, Pittsburgh, Pennsylvania 15206.

skip over the centuries and begin with the great systematizer of mental disorders—Emil Kraepelin, who combined a variety of discrete symptoms (catatonia, hebephrenia, and paranoia) into the disease category of dementia praecox. Kraepelin distinguished dementia praecox from manic depressive psychosis on the basis of ultimate deterioration in cases of the former. The Kraepelinian tradition persists today and is found in more elegant form in the concepts of "true" schizophrenia described by Feighner et al. (1972) and by Kety (Note 1). Spokesmen for this position maintain that a definite diagnosis of schizophrenia can be made only if symptoms persist chronically and if a deteriorating, unremitting disease course is followed. True schizophrenia is thus considered to be synonymous with poor prognosis schizophrenia.

Departing from the Kraepelinian view, E. Bleuler (1911/1950) eliminated poor prognosis as a criterion and specified instead a hierarchy of primary symptoms (autism, association and affective disturbances, and ambivalence) and secondary symptoms (hallucinations, delusions, negativism, and stupor). This second tradition, giving primacy to symptomatology rather than to disease course, has been further developed by Schneider (1959), Langfeldt (1939, 1956), the Biometrics Research Unit (Gurland, cited in Zubin et al., 1975; Spitzer & Endicott, cited in Zubin et al., 1975), the WHO Pilot Study of Schizophrenia (World Health Organization, 1973), and by cognate groups. These groups have specified the psychopathological characteristics of schizophrenic behavior along the well-known dimensions of perceptual dysfunction, speech disorder, delusions, hallucinations, affective and cognitive dysfunction, and so on. However, these classic symptoms do not in themselves indicate a particular diagnosis, since they tend to be manifested by a diverse and heterogeneous group of patients. To reduce this heterogeneity and yet save the symptom or syndrome approach, Feighner et al. (1972) and subsequently Spitzer and Endicott (cited in Zubin et al., 1975) have proposed operational definitions of schizophrenia and the other mental disorders for the new *Diagnostic and Statistical Manual of the American Psychiatric Association* (*DSM III*, in press). Each disorder is operationally defined by explicit criteria. These criteria take the form of sets of characteristics, a specified number of which must be present before a given diagnosis is warranted. Because the specific psychopathological features associated with each official *DSM III* diagnosis can now be explicated, the reliability of diagnostic decisions can be enhanced (Spitzer & Endicott, cited in Zubin et al., 1975) and the heterogeneity that characterized the *DSM II* diagnostic categories can be greatly reduced.

Diagnostic improvements became possible with the availability of systematic structured interviews to replace the freewheeling clinical interviews that had previously preempted the field. Structured interviews provide the great advantage of reliable and comprehensive coverage of all areas of psychopathology and behavior germaine to a set of diagnostic categories. These interviewing methods have demonstrated their value in two major studies —the US–UK Diagnostic Project (Cooper et al., 1972) and the WHO Pilot Study in Schizophrenia (World Health Organization, 1973). The US–UK project was initiated to determine why the national statistics show such disproportionate frequency of affective disorders in the United Kingdom and of schizophrenia in the United States. When the newly developed systematic structured interviews (Gurland, cited in Zubin et al., 1975) were applied to samples of patients admitted to hospitals in the two countries, the cross-national differences turned out to reflect different diagnostic practices of psychiatrists rather than differing characteristics of patients in the two countries. Whereas the US–UK project limited itself to two cultures but investigated the entire panoply of mental disorders, the WHO study limited itself to one disorder but investigated its incidence, form, and course in nine different cultures. This investigation found specific syndromes of schizophrenia ubiquitously distributed in developing and advanced cultures from Ibadan, Nigeria, to Washington, D.C.

Other advances in descriptive psychopathology have been (a) the use of computers that simulate the clinician's decision processes and

arrive at a diagnosis (Spitzer & Endicott, cited in Zubin et al., 1975; Wing, Cooper, & Sartorius, 1974); (b) the provision of mathematical methods for clustering individuals with shared psychopathological characteristics into more homogeneous subgroups (Fleiss & Zubin, 1969; Wing & Nixon, 1975); and (c) the development of behavior-analytic descriptions of patient characteristics (Kanfer & Saslow, 1969; Salzinger, cited in Zubin et al., 1975). Rather than emphasizing the presence of symptoms per se, behavior-analytic description relates deviant behavior to environmental contingencies that initiate and sustain it. Description is therefore focused on aspects of the individual and his environment that are of immediate relevance to behavior modification therapy.

A dispassionate view of where we stand in descriptive psychopathology today leads to the conclusion that we have made considerable progress and perhaps gone as far as we can go but that this is still not far enough. Through the use of systematic structured interviews and operational criteria for selecting a diagnosis, a high degree of agreement on diagnosis can be attained. We can now objectively describe the characteristics of patients we deal with so that replication of basic research and treatment findings on similar patients is possible. Moreover, we can demonstrate that individuals suffering from such syndromes of psychopathology as schizophrenia can be found in parts of the world that differ widely in cultural and ecological conditions.

Where does this leave the allegation that schizophrenia is a myth? We must bear in mind that science deals not only with ostensive facts but also with mythlike concepts or fictions that are used to organize the facts. We can inquire whether the facts are true, but we cannot inquire whether the concepts are true—we can only ask if they are useful. To the extent that trained psychologists and psychiatrists all over the world can consensually recognize the syndrome that we call schizophrenia and find this summary description of psychopathological behavior beneficial for selecting treatment strategies and interpreting research findings, schizophrenia is still a useful concept.

However, we cannot presuppose that reliable description of a syndrome implies valid understanding of its underlying cause. We need to do more than describe, since description never cured anyone, nor did it by itself reveal etiology. Although treatment can often not await a thorough knowledge of etiology, the discovery of an effective cure for any disorder is less likely to result from chance than from an understanding of factors that cause the disorder. As in the classification of organisms, in which taxonomy looks to common origins of descent, so in the classification of disorders we look for common causes producing the disorder. But how can progress be made, when with but few exceptions, we are abysmally ignorant of the causes of mental disorder and even more poignantly ignorant of the efficacy of the treatment? When faced with such ignorance, one can only contemplate possible or "as if" causes, formulate them into parsimonious scientific models, and proceed to test the hypotheses they generate.

Scientific Models of Etiology

What models have been proposed? The six models depicted in Figure 1 have been described in detail elsewhere (Zubin, 1972) with evidence on the tenability and the limitations of each. Here we will only present a short overview of all six models and briefly examine the status of two of them—the genetic and the ecological.

The etiological models can be classified into the following three types in accordance with their etiological focus: (a) field theory models focusing on forces emanating from the organism's external environment—the ecological niche it occupies—(b) behavioral psychological models focusing on forces emanating from the experience of the organism through learning and development; and (c) biological models focusing on forces emanating from the organism's internal milieu—its genes, biochemistry, and neurophysiology.

One type of field theory model is the ecological approach, which sees man's health or illness as dependent on the physical, social, cultural, educational, and economic parameters of the ecological niche he occupies. The developmental model is concerned with exog-

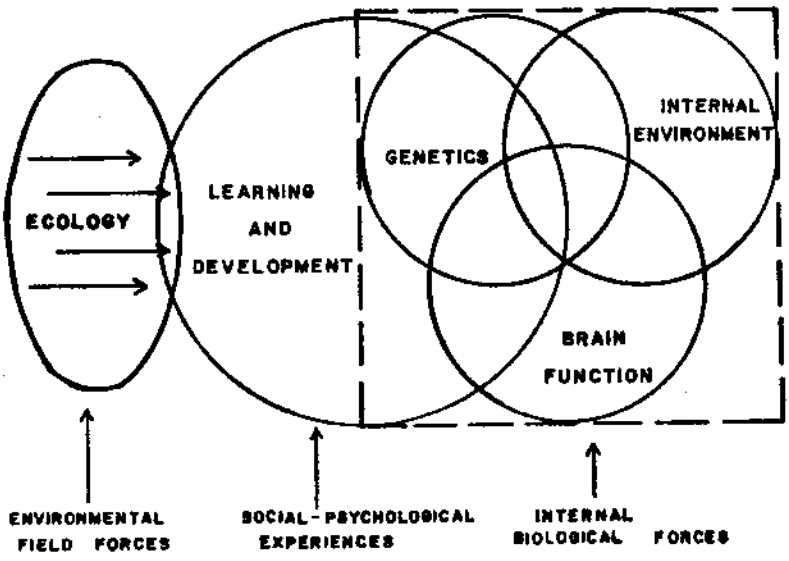

Figure 1. Scientific models of etiology.

enous and endogenous factors that affect man's progression through maturational phases and hence contribute to either buffering or fostering the development of a propensity to become mentally ill. The learning theory model has been most concerned with contingencies that elicit and sustain psychopathological behavior. This model differs from the developmental model in generally assigning more importance to current rather than historical causes of behavior. It also tends to eschew investigation of how environmental influences might act differentially on individuals with different propensities.

From a more biological perspective, the genetic model suggests that health and illness are predicated on the genetic equipment man is born with. The internal environment model stipulates that the roots of man's illnesses are to be sought in his metabolism, body fluids, and body chemistry in general. The neurophysiological model concentrates on the functioning of man's nervous system and its capacity to take in and process information.

Genetic Model

The genetic model languished for a time because some of its claims to high concordance in monozygotic twins were deflated during the 1960s. However, the separation of rearing from hereditary influence in the studies of adoptees (e.g., Kety, Rosenthal, Wender, & Schulsinger, 1968) and the monitoring of the development of high-risk children (e.g., Mednick & Schulsinger, 1968) added new dimensions to genetic research. Findings from such studies have been exciting and striking. However, they have also posed unforeseen challenges to the genetic model. Compared to the 85%-concordance rates found by Kallman (1959) in monozygotic twins, it has been somewhat disappointing that current studies find only 40%–60% [1] of monozygotic twins concordant for schizophrenia (Slater & Cowie, 1971, pp. 18–19). Moreover, even when the effects of genetic predisposition are added to those of rearing by a schizophrenic parent, as is the case for home-reared offspring of two schizophrenic parents, only 60% of their offspring are likely to become schizophrenic. The current inability to explain or predict which genetically predisposed individuals will ultimately become schizophrenic has forced us to confront our ignorance of the precise mechanism and mode of transmission of this disorder.

[1] Recent studies (Slater & Cowie, 1971, p. 19) have found only 27% concordance when both twins suffer from "strict" schizophrenia.

An additional problem has arisen from the finding that offspring and relatives of schizophrenics may show a spectrum of behavior ranging from normalcy through the schizoid spectrum and from psychopathy to core schizophrenia. The discovery of such a schizophrenic spectrum has a number of difficult implications. Taken to its extreme, it may nullify the usual procedures for comparing schizophrenic patients with normal controls on specific biochemical or other markers, since the control group may contain an unknown proportion of unexpressed genotypes for schizophrenia. Moreover, the spectrum finding raises the old question of the most appropriate means of conceptualizing the schizophrenic process. Should we adopt a dimensional approach in which the disorders in the spectrum are seen as falling along a quantitative continuum of severity? Or should we select a typological approach that delineates particular subcategories based on different etiologies?

Many prominent spokesmen for the genetic model now seem to be adopting a typological approach, which postulates that only some cases or types of schizophrenia arise from genetic proclivity:

> Several studies taken together clearly indicate that many schizophrenics have an important genetic contributant. Further studies of this sort may more clearly define the boundaries of the spectrum, but will not elucidate its mechanism. Comparing the situation to that of studies of retardation in the nineteenth century, one must expect that further progress will be made only by attempting to define homogeneous subgroups, some of which have specific biological etiologies, and exploring the biological attributes of those subgroups.
>
> These adoption studies may be compared crudely to that in which a kite was flown and an electric spark was discovered. They are provocative and, I think, exciting; but one can anticipate further progress only on the basis of careful study of electricity and not by the use of bigger and higher flying kites. (Wender, Rosenthal, Rainer, Greenhill, & Sarlin, in press)

Ecological Model

The ecological approach seeks the causes of schizophrenia in the parameters of the environmental niche that a person occupies. Cassel (1974) has pointed out that throughout history the environmental factors singled out for study have reflected both the reigning etiological theories and the existing level of technology. Thus the airs, waters, and places believed to be etiological agents in Hippocratic times have been succeeded by the microchemicals and microorganisms of today. Ironically, old etiological theories have often been permanently preserved in the names of various diseases, for example, *malaria* ("bad air"), *hysteria* ("wandering uterus"), and *melancholia* ("black bile").

Some ecological parameters that are currently proposed as causes of mental disorder include (a) low socioeconomic status; (b) disorganization in the social milieu; (c) crowding; and (d) minority status in the community. Adequate investigation of these parameters has been hampered in several ways. First, it has always been exceedingly difficult to know whether an individual's situation in an undesirable niche *causes* a disorder or *results* from a disorder. Does a high rate of illness associated with an ecological characteristic mean that the environmental factor facilitates the likelihood of a disorder? Does it mean that the social system affords especially efficient means of recognizing and selecting for patienthood disordered individuals who appear in a certain environmental context? Does it mean that schizophrenic individuals prefer and choose an environment with certain characteristics? Does it mean that schizophrenics are forced to inhabit certain types of ecology as a consequence of their illness? To date there has never been an adequate way of choosing among the competing hypotheses, although B. P. Dohrenwend and B. S. Dohrenwend (1969) have proposed some ingenious research strategies to test these issues.

Two other major stumbling blocks for the ecological hypothesis have been (a) the lack of an adequate taxonomy for parameters of the ecology that influence good and bad health and (b) the inability to do fully controlled studies on these parameters. No doubt the effect of any ecological factor on health is influenced by a host of interactions with moderator variables. For example, we know that population density takes a totally different toll depending on whether it involves overcrowding among strangers or among familiar peers. It is likely that the link between population density and high risk of disorder is

mediated by the disorganized relations that ensue between individuals. The impact of a disordered relation on mental health is in turn dependent on (a) the importance or salience of the relation that becomes disordered (e.g., mating, rearing of offspring); (b) the position of the individual experiencing the disordered relation in the status hierarchy; (c) the degree of previous experience with such disorganization; and (d) the nature and strength of available group support. But in the final analysis, progress has been prevented by our inability to measure these factors objectively or to find variables that transcend local or temporal biases.

Other Models

The developmental model, with its emphasis on rearing, has also failed to provide the answers we are seeking. Rosenthal et al. (1975) maintain that childhood rearing is less important in the production of psychopathology than is genetic loading. M. Bleuler (1974) finds that childhood neglect and deprivation of the most dire type have little or no bearing on the development of schizophrenia.

Nearly all geneticists gesture graciously toward the developmental model by acknowledging that genes do not work in a vacuum but rather require circumstances to elicit genetically encoded propensities. But neither geneticists nor developmentalists have been able to specify the circumstances that are required. Attempts have been made to delineate particular family interaction patterns that might prove schizophrenogenic (e.g., Bateson, Jackson, Haley, & Weakland, 1956). However, Jacob's (1975) careful review of this literature has shown how difficult it is to demonstrate that these patterns occur with any greater frequency in the families of disturbed compared with normal offspring. Rosenthal et al. (1975), commenting on the complexity of family interaction research, wrote the following:

> The familial behavior must be examined over appreciable stretches of time. Reliability of behavioral coding must be determined, and the selection of behaviors to be observed will have to be limited. And even with the best of circumstances, investigators will still not be able to determine from the usual high-risk study how much of the behaviors noted stemmed from familial genes or from mutual influences of parents and children. (p. 476)

Thus, here too we have reached an impasse.

The learning theory model has been primarily concerned with the contingencies that trigger a schizophrenic episode in an individual who has already developed a propensity toward schizophrenia. Beyond studying reinforcements that elicit and maintain schizophrenic behavior, this model has generally not dealt with basic causes of schizophrenic behavior. Thus it has not explained why events and reinforcements that catapult one individual into an episode of schizophrenia leave another individual unaffected.

Despite the enthusiasm recently rekindled by Kety and his group (Kety et al., 1968), the internal environment model is still only promising rather than delivering. And the neurophysiological model, with its stress on information processing, has hardly gotten off the ground.

Although space does not permit us to fully describe and analyze the contributions of these models, our general conclusion is that a formidable impasse has been reached. How can we now transcend it and start moving again? It would be foolhardy to claim that we have found the definitive answer. We have, however, decided to take a fresh view and see if a new approach is possible.

Vulnerability Model

Progress rarely precedes an understanding of historical pitfalls. Therefore, it is important to try to determine the general reasons why all six scientific models seem to have faltered. Perhaps the main difficulty is that each model is framed so broadly that entire schools of psychopathology can pass through its portals without even rubbing shoulders. Just as no model has been in danger of being refuted by findings that support another, neither has it been enriched by the interaction of differing views. The trend for parochialism seems unfortunate, since for too long it has permitted proponents of each model to exploit their own research without noting the progress of others. We feel that it may now be useful to examine

the various models to see whether the parts might illuminate the whole.

Our strategy is essentially pragmatic. We are trying to pool the wisdom from all of the models to analyze out a common factor that can best guide research across all the approaches. Put more picturesquely, we shall squeeze the juice out of all these models into a goblet and see what the elixir consists of. This logical factor analysis suggests that the common denominator connecting all six models is a second-order model that can be called the vulnerability model.

The vulnerability model proposes that each of us is endowed with a degree of vulnerability that under suitable circumstances will express itself in an episode of schizophrenic illness. Each etiological model offers suggestions about the possible origins of such vulnerability. Moreover, many earlier publications (Gottesman & Shields, 1972; Meehl, 1962; Millon, 1969; Rosenthal, 1970) have anticipated the vulnerability notion in discussions of the concept of a schizophrenic diathesis. 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. Finally, we distinguish between *vulnerability* to schizophrenia, which we regard as a relatively permanent, enduring trait, and *episodes* of schizophrenic disorder, which are waxing and waning states.

Further on in this article we will have more to say about the episodic nature of schizophrenia. At the moment we will be concerned simply with the concept of vulnerability. Let us preface our discussion by noting that whether such vulnerability extends to all of mankind and whether it is the same sort that predisposes an individual to disorders other than schizophrenia remains an open question. But even if only one or two percent of humanity is capable of becoming schizophrenic, this still represents 30 million of the world's population.

Corresponding to the two types of etiological models—the biological and the field theory—there are two major components of vulnerability, the inborn and the acquired. Elsewhere (Spring & Zubin, in press-b) we have described inborn vulnerability as that which is laid down in the genes and reflected in the internal environment and neurophysiology of the organism. The acquired component of vulnerability is due to the influence of traumas, specific diseases, perinatal complications, family experiences, adolescent peer interactions, and other life events that either enhance or inhibit the development of subsequent disorder.

As Audy (1971) suggested, the preservation of health requires the maintenance of a dynamic equilibrium against insults continually emanating from the chemical, physical, infectious, psychological, and social environment. When this equilibrium is disturbed beyond its capacity to reinstate its own homeostasis, a disorder ensues. An individual's vulnerability to any illness determines the ease and frequency with which suitable challenges to homeostasis will catapult him into that disorder. The highly vulnerable person is one for whom numerous contingencies encountered in daily living are sufficient to elicit an episode. Others have such a low degree of vulnerability that nothing short of a rare and probably catastrophic event would induce an episode, and even then only a very brief one.

The "challengers" that disrupt adaptation and are capable of provoking a temporary crisis or even an episode of illness may be endogenous or exogenous in origin. The endogenous events may be either biochemical or neurophysiological. Relatively little is known of what causes such internal events, but the possibilities are numerous: maturational changes within the organism, ingestion of toxic substances, inadequate nutrition, pathological responses to infection or to stress, and so on. By and large, endogenous precipitants of homeostatic disturbance are difficult to study without advanced technology, since they are generally unreported and often unperceived by the individual. Exogenous challengers are usually referred to as life events. We shall limit our discussion to these exogenous stressors, not because they are more

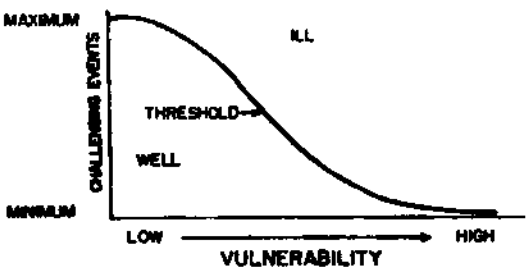

Figure 2. Relation between vulnerability and challenging events.

important than endogenous challengers, but because they are more directly measured and have been extensively studied.

There is considerable evidence that life event stressors can play a major role in eliciting both physical and mental disorders (Brown, 1968; Dohrenwend & Dohrenwend, 1972; Rahe, Meyer, Smith, Kjaer, & Holmes, 1964). A life event stressor is an incident such as bereavement, promotion, marriage, or divorce that challenges adjustment and brings in its wake a readjustment or reorganization of a person's life. It is easiest to regard life stresses as events that befall an individual regardless of his temperament or behavior. However, it is interesting to speculate on the extent to which life stressors really do occur as random happenstances. With the exception of such natural calamaties as earthquakes, it may be that a person's choice of life style contributes to his likelihood of encountering stressful events. Thus it may be possible to identify "stress-prone" patterns of living, just as we can discuss "accident-prone" individuals. However, for the purpose of conceptual clarity, we shall oversimplify and discuss life events as a dimension orthogonal to the individual's other characteristics.

Figure 2 shows the hypothesized relation between life event stressors and vulnerability. As long as the stress induced by the challenging life event stays 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 of some sort. Further, we postulate that the episode is time limited. When the stress abates and sinks below the vulnerability threshold, the episode ends and the patient returns to a state similar to his pre-episode level of adaptation.

Our model is Selyean (Selye, 1973), in which life event stressors impinging on the organism induce a state of strain. The strain in turn sets in motion adaptive capacities to overcome the stress or contain the strain. We concur with Mechanic's (1967) definition of stress as involving "a discrepancy between the demands impinging upon a person—whether these demands be external or internal, whether challenges or goals—and the way the individual perceives his potential responses to these demands" (p. 201). As Wilkins (1974) and Gross (1970) suggest, strain occurs as a result of "the failure of routine methods for managing threats" (Gross, 1970, p. 55). In brief, stressful life events tax the organism's adaptive capacities. But of what does adaptation consist?

Adaptation

Adaptation describes the extent to which an organism responds adequately and appropriately to life's exigencies. It is a biological concept with a long history and tremendous literature that cannot be summarized here. However, it may be useful to break down adaptation into its components to use the concept in our discussion of vulnerability. A simplified model of adaptation may be borrowed from Hooke's law and Young's modulus in physics. This model deals with the stress imposed on a body by a load that produces a measurable tensile strain. The classic example involves a string suspended from the ceiling to which a weight has been attached. As a result of the load, the string may (a) withstand the load without lengthening, though heat may be generated; (b) temporarily lengthen but return to its original length once the load is removed; (c) lengthen permanently; or (d) break under the load.

To extend the analogy, consider the string to represent an organism and the weight to represent a load imposed by a life event stressor. The string's tendency to stretch might be likened to the biological and

sents the motive power of an organism, competence represents its capacities. Coping efforts are analogous to the voltage of an electric current that sets a motor into operation. The motor may be intact in every respect, and its interconnection with the machine may be unchanged, but unless the voltage is maintained within proper limits the machine will not operate adequately. Competence, on the other hand, is analogous to the *capacities* of the machine, the functions for which it was equipped. If the machine is applied to a job for which its circuitry is inadequate, no work will be accomplished no matter how adequate the power supply.

In the growth of the individual, developing capacities are put to use in efforts to cope with routine daily events. Well-practiced coping strategies crystallize as effort and competence are rewarded with success in life. However, when challenging new situations arise, routine coping strategies may be inadequate and new ones may be needed. If adaptation falters in such circumstances, it will be very difficult to determine whether the fault lies in coping efforts that are sluggish or overzealous or in an inadequate repertoire of competencies.

Some encounters with stress-producing events are routinized by cultural sanctions that proscribe a wide range of institutional forms for coping. In many cultures, for example, extreme and "deviant" forms of grief are tolerated and expected; however, not all forms of stressful challenges are matched with institutionalized forms to channel coping behavior. Lazarus, Averill, and Opton (1974) have distinguished between the

> healthy, positively adaptive problem-solving efforts characteristic of low-stake situations and the primitive and maladaptive forms of coping typically associated with conditions of high stake (that is, severe threat, frustration, conflict and great challenge). (p. 305)

Most of us, if observed closely, probably display some disturbance of coping when catastrophic situations arise. In the resilient person the "mini-episode" of coping breakdown passes, and after a temporary period of distress, routine coping strategies regain the field. In a highly vulnerable person, however, a temporary breakdown in adaptation may provide an occasion for more fundamental problems to manifest themselves. The comparison may be clarified by an analogy. If a healthy person overexerts himself by running a marathon, he will be stressed, flushed, breathe harder, and be very tired. The next day, aside from some sore muscles, he will have recovered. However, a person who has recently suffered a heart attack will probably not recover so easily. The disturbances directly related to running the race may soon vanish, but difficulties due to his weakened heart may once again be manifest. So, in the person vulnerable to schizophrenia, episodes of psychiatric disorder may often follow close on the heels of periods of acute stress and coping breakdown. We need not conclude that coping breakdown causes schizophrenia, but only that it provides an opportunity for vulnerability to germinate into disorder.

This relation between deviant coping patterns and the development of a schizophrenic episode goes back historically to Adolph Meyer (Arieti, 1974, p. 16). He became convinced by his longitudinal studies that dementia praecox results from an accumulation of faulty habits of reaction to life's exigencies. Confronted with failure after failure, the patient gradually develops substitutive reactions instead of efficient adjustment to actual difficulties. These coping failures themselves do not constitute an episode of schizophrenia, but according to Meyer, the transition from maladaptation to schizophrenia is continuous and insidious. Thus, maladaptation, with its coping failures, may prepare for either the gradual transition to a schizophrenic episode or set the stage for a sudden onset of an episode following a catastrophic event. Intervention at this point, before an episode develops, may be most propitious for preventive therapeutic efforts.

French and Steward (1976) have presented a model of adaptation to a stressor that is less developmentally oriented than Murphy's (1974). They describe successful adaptation as a balance between accommodation and assimilation and suggest that a feedback loop for maintaining homeostasis might consist of three components. The first is a force F having magnitude but no direction and experienced subjectively as an "ought." Although the individual may or may not know the ob-

Table 1
Patterns of Response to Stressors in Relation to Components of Adaptation and Its Outcome

Pattern	Components of adaptation: Coping effort	Felt discrepancy	Competence	Outcome
1	+	+	+	joy
2	+	+	−	frustration-anxiety
3	+	−	+	obsessive behavior
4	+	−	−	obsessive rumination
5	−	+	+	inhibition
6	−	+	−	ineffectual complaints
7	−	−	+	unstressed
8	−	−	−	passivity (?)

Note. Table adapted with modifications from French and Steward (1976, p. 469, Table 1).
Plus = presence of component; minus = absence.

ject or survival value of the "ought," he feels a drive to do something about correcting an imbalance. The "ought" therefore corresponds to the subjective triggering event for a coping effort. The second component is a cognitive perception of the discrepancy between the observed stressed state of the organism, its prior state, and the state of adaptation toward which it strives. The felt discrepancy corresponds to the organism's ability to identify the disparity between its own present state of distress and the remembered or imagined happier circumstances. It helps define a direction for coping efforts to return the organism to its original state or to some other adaptive state. Put simply, the felt discrepancy entails the identification of "what's wrong." The third component is the biological and/or cognitive structural capacity to correct the felt discrepancy. This corresponds to our concept of competence and refers to the repertoire of skills and capacities at the organism's disposal to formulate coping strategies that will reattain adaptation.

French and Steward (1976) provide an elaborate typology for all possible patterns of presence or absence of the three independent components and the type of mini-episode and residual state that will ensue in response to a stressor. We have presented their scheme in modified form in Table 1. Thus, if all three components are present in adequate amount, only a brief mini-episode occurs in response to the stressor, successful adaptation ensues, and the organism experiences a state of joy (Pattern 1). If competence is insufficient even though the other components are present in sufficient amount, a state of frustration ensues (Pattern 2). If coping efforts are insufficient but the other two components are adequate, an inhibitory state sets in and no solution is sought (Pattern 5).

The other patterns give rise similarly to a variety of other responses, and whether a catastrophic situation ensues or a mini- or maxi-episode develops depends on the individual's assets and degree of vulnerability. But the important point is that the initial response to the stressor—the crisis—is a breakdown in coping that is either transient, or if it persists, one that provides the matrix for the development of an episode.

Relation Among Vulnerability, Life Event Stressors, and Adaptation

What is the relation between vulnerability and the various components of adaptation, and what is the association between failures in adaptation and the onset of episodes of psychiatric disorder?

First, we have already suggested that stresses of a catastrophic nature for an individual generally produce some measurable disruption in adaptation. The disruption need not be gross; it might perhaps be described as a mini-episode of failure in adaptation. We shall take up the problem of how to index a mini-episode at a later point. Here it must be remembered that the stress value of various

life events depends on the perception of threat by the individual. And although Holmes and Rahe (1967) have demonstrated group-based similarities in the stress value of various life events, there is still considerable room for individual differences.

When adaptation goes awry and a mini-episode appears, the individual's degree of vulnerability determines the severity and longevity of the consequences. Phrased positively, invulnerability describes the organism's ultimate resilience to the load imposed on it by life event stressors. Failures in adaptation may take the form of temporary disability—the pain and distress that will reduce efficiency and happiness—but stop short of becoming enduring disruptions of the organism's functioning. One form of transient failure in adaptation is the "bad day" or "rough week" familiar to everyone. Such normal fluctuations in adaptation are ephemeral and do not usually require therapeutic intervention. In a vulnerable individual, however, what begins as a mini-episode may develop into an episode of psychiatric disorder that is far more overwhelming to the person, is longer lasting, and involves a more total disabling of behavior.

In both mini-episodes and episodes of psychiatric disorder, the outcome may be either assimilation or accommodation. In assimilation, adaptation is regained primarily by altering the environment. When the stress is eliminated, either by the organism's own instrumental behavior, by the passage of time and circumstance, or by the assistance of some external agency, the organism returns toward its preepisode status. Perhaps the primary concept behind the development of asylums for the psychiatrically ill was to remove patients from the stresses of life and allow the assimilative process to take its course. In episodes of psychiatric disorder, as well as in mini-episodes of failures of adaptation, we have suggested that a full return to preepisode status can occur. However, the process may take longer in episodes of psychiatric disorder and may be complicated or even prevented by other adverse effects of institutionalization and stigmatization. The other outcome of a mini-episode or major episode is accommodation, in which the organism alters its internal state to meet the needs of the environment. Optimally, such alterations are adaptive and represent growth that may prove to be preventive of future episodes. Alternatively, they may be regressive and lead finally to an entrenchment of psychopathology and a loss of basic capabilities.

Return for a moment to the French and Steward (1976) model: In which component of adaptation may we expect to see signs of an encroaching episode of failure of adaptation? Is it a drop in coping effort, an inability to perceive the discrepancy between the stressed and optimum states, a loss in competence, or a patterned combination of all three components? At present, it would be far too complicated to deal with all three elements and their interactions. Hence we will place a hunch on one component—the coping effort.

This tentative decision is based on an assumption that competence and the cognitive ability to perceive discrepancy are stable, structural characteristics of the organism, developed over long experience with previous situations requiring adaptation. The exertion of coping efforts, on the other hand, seems to be a more labile, dynamic force that can wax and wane with time and occasion. It is functional rather than structural, serving to successfully evoke the capacities of the organism to contend with problems or to inhibit and fail to elicit adaptive strategies. Again, one might liken the cognitive and competence components to the structure of a machine, whereas the coping-effort component is the power needed to make the machine function.

Although we propose that a diminution, exacerbation, or inappropriate application of coping efforts may perhaps be the ideal markers for an episode, we should point out the difficulty of separating the three components of adaptation in actual measurement. Precisely because the structural cognitive and competence components can only be energized by coping efforts, it would be difficult to elicit and demonstrate their intactness during an episode. It is apparent that individuals of the most excellent premorbid status display extraordinary degrees of incompetence and gross lapses of cognitive insight during the height of an episode of psychiatric disorder.

Premorbid Competence and Vulnerability

If one were to question laymen, clinicians, and researchers about vulnerability to psychiatric disorder, one would probably unearth the implicit assumption that there is a greater-than-chance association between incompetence and high vulnerability or between high competence and invulnerability to psychopathology. Such an implicit assumption has become more explicit with the advent of program planning to prevent psychiatric disturbance among children at some risk for psychopathology. Quite frequently such programs are designed to incorporate competence-enhancing interventions on the grounds that these interventions should simultaneously prove to reduce vulnerability.

Let us therefore examine the relation between vulnerability and preepisode competence. It is of course clear that during an episode, a patient's competencies appear to be at a low ebb. However, our concern is not with the patient's capacity to display his skills and abilities during his illness, but rather with his pre-episode demonstrations of competence and their relation to vulnerability. In other words, is a person less vulnerable to psychiatric disorder because he is generally competent at making use of many response options and meeting life exigencies successfully? Or is competence independent of vulnerability?

There are several reasons why we shall tentatively assume that competence is orthogonal to vulnerability. First, there are but few data available, and until more adequate data are brought forth it is best to adopt the null hypothesis. Second, the evidence for interdependence stems largely from studies examining the prevalence of psychiatric disorder as a function of socioeconomic status. The general finding in this area has been the negative correlation between socioeconomic status and rates of schizophrenia. However, there are numerous difficulties in generalizing from this result to the conclusion that competence and vulnerability are interrelated. First, in smaller towns, the negative correlation does not generally hold. Second, the generalization entails the ecological fallacy, which infers from correlations between group means an interpretation that should only be inferred from the total correlations, including within-group correlations. Third, it is unclear whether the negative correlation is due to (a) a causal relation between socioeconomic status and schizophrenia or to (b) the downward drift of schizophrenics on the socioeconomic-status scale. Fourth, and very fundamental, is whether socioeconomic status can be interpreted as a valid index of competence. There are many who might question whether financial income, occupational level, or level of education accurately reflect an individual's general level of success in dealing with life exigencies. Even when we put aside this almost imponderable and value-laden question, however, can we gain any better understanding of the relation between competence and vulnerability by examining one individual component of socioeconomic status?

There is no certainty that occupational level —one of the chief elements of socioeconomic status—is negatively related to schizophrenia. Findings by Turner (in press) indicate that nearly two thirds of the premorbid schizophrenics in the sample drawn from the Monroe County Case Register (New York) were employed before entering the hospital, a figure only 20 percentage points lower than that for the general population. Furthermore, the proportion of these employed patients who were working at skilled manual levels or higher premorbidly corresponds to the similar proportion in the general population—about 70%. It is also interesting to note Serban's (1975) finding that the proportion of those schizophrenic admissions to Bellevue Hospital in New York City who were never employed was no greater than the proportion in the Bellevue catchment area (3.2% to 3.5%). Christensen (1974) found that in a follow-up of a cohort of admissions, the rehospitalized patients had a better employment record but a more severe level of psychopathological disturbance at follow-up than did their peers who were not rehospitalized. Thus, occupational competence seems to provide no protection against initial admission to a psychiatric hospital or against rehospitalization.

One of the most incisive studies of the compatibility between mental disorder and the working world is based on an investigation of union members of the Amalgamated Clothing

Workers of America (Weiner, Akabas, & Somner, 1973). A special clinic was established for the purpose of preventing mental disorder from interfering with worker productivity. Taken as a whole, the patients were able to work at a level almost as productive as that of their demographic and occupational peers. The neurotics even excelled their peers, whereas the psychotics fell somewhat below in earning power. Only 20% of the patients had not returned to their jobs by the end of the 6-month therapeutic endeavor. Thus, 92% of the patients were at work immediately prior to their entry into the clinic; from 73% to 87% worked during the period of treatment; and fully 80% were back at work at the end of treatment.

Two tentative conclusions might be drawn from all of these findings. First, occupational competence generally confers no immunity to schizophrenia. Second, the highly vulnerable individual, even when ill, may be able to demonstrate satisfactory levels of work competence under suitable conditions.

All things considered, perhaps we should begin to consider the possibility that the presumed interdependence between competence and invulnerability to schizophrenia is a myth, and indeed a comforting one to many of us. One must bear in mind a number of circumstances that might give such a myth the appearance of truth. First, most of the data are based on cases of patients hospitalized in public institutions. Private hospitals or clinics where more affluent members of the population send family members have often been excluded from our statistics. Second, it is likely that highly competent schizophrenics, often with the help of supportive families, are able to find ways of maintaining themselves in the community. Autobiographical accounts (Barnes & Berke, 1971; MacDonald, 1960; Wallace, 1965) amply demonstrate the ingenuity with which some psychotic individuals can avoid unpleasant periods of hospital confinement. This more talented group of patients may therefore be particularly unlikely to be represented in our statistics for hospitalized cases. If we were to include schizophrenics at large who do not, or have not yet, reached the hospital, a much less distorted picture of the relation between competence and vulnerability would probably emerge in the direction of reducing or even eliminating patient–nonpatient differences.

Another distorting element in our data is the tendency for low-socioeconomic-status patients to remain in the hospital longer, perhaps even after their episodes have ended. Since most of our data are based on prevalence rather than incidence figures, another source of bias enters. There is also some evidence suggesting that low socioeconomic status or low competence per se may occasionally enhance the likelihood of admission to psychiatric hospitals. Thus, as Meyers and Bean (1968) have noted, lower-class patients had more frequent admissions to hospitals even though they exhibited less psychopathology than upper-class patients. The so-called marginal persons—individuals who are unmarried, living alone, unemployed, or of the lowest socioeconomic status—show higher rates of seeking and receiving mental health care than would be expected from the overall prevalence of symptoms of psychopathology in these demographic groups in the community (Tischler, Henisz, Meyers, & Boswell, 1975a, 1975b).

In concluding this section we might merely note that an individual's tendency to be hospitalized in a psychiatric institution may be influenced by many factors other than the presence of psychopathology. These factors may tend to inflate the probability that less skilled individuals suffering from schizophrenia will appear in the hospitalized population, whereas more competent individuals with schizophrenia may not. It is interesting to note that during the great depression of the early 1930s, the marginal persons described above tended to be herded into the mental hospitals (Zubin & Burdock, 1965). The decline in the resident patient population might be attributed in part to the development of welfare and social programs that make possible the subsistence of this cohort in the community.

Premorbid Coping Ability, Life Events, and Vulnerability

Coping ability is the resultant of the coping effort, or initiative, and the competence, or skill, that an organism brings to bear in for-

mulating strategies to master life situations. When an individual is described as a good coper, it is generally meant that he maintains adaptation on an even keel (a) most of the time and (b) in the face of life events that are widely recognized to be quite stressful. The poor coper, in contrast, seems to be thrown off balance (a) rather frequently and (b) more often by events that are not consensually regarded as threatening or challenging. Major or minor periods of coping breakdown or failure in adaptation will, of course, be observed in the good coper as well as in his less adept counterpart. These will be marked by such typical signs of strain as subjective discomfort or distress, feelings of helplessness or tension, withdrawal, inappropriate or ineffectual behavior, and physiological disequilibrium or vegetative disturbances, but not necessarily by episodes of mental disorder.

In general, the dimensions of coping ability and vulnerability to psychiatric disorder are probably independent of each other. The chronic ne'er-do-well, who seems to be upset by the slightest stress, is not necessarily the person most likely to develop an episode of schizophrenia. On the other hand, there may be one sense in which coping ability and vulnerability are interrelated.

It seems likely that the person in a state of coping breakdown enters a period of risk, a state of lowered psychological resistance. If the individual's vulnerability is sufficiently low, this period will pass with relatively small consequence. On the other hand, if the vulnerability is high, such sensitive points are likely opportunities for vulnerability to develop into the expression of psychopathology. The poor coper will encounter more daily events that lower his resistance and may consequently spend a greater proportion of his lifetime at risk for the manifestation of whatever vulnerability he possesses.

We might liken the occurrence of a coping breakdown to the development of a fault in the earth's surface. Ordinarily, a brief tremor will occur before a stable formation of the earth is reestablished, just as in the person's life a slight jar in the spheres of work and personal affairs will precede a new state of closure. On the other hand, if a bed of molten lava is seething below the crack in the earth's surface, the development of the fault will create an opportunity for the emergence of a volcano. In the vulnerable individual, a coping breakdown may open the way for the eruption of a psychopathological disorder.

Episodic Nature of Schizophrenia

We have postulated that the primary persistent characteristic of the schizophrenic is his vulnerability, not his disorder. This, however, flaunts the traditional view that regards schizophrenia as a permanent condition leading to chronic deterioration or unremitting impairment. A recent review (Spring & Zubin, in press-b) finds only sparse evidence for the chronic, unremitting nature of most schizophrenic illness. Is the episodic hypothesis a tenable alternative?

Hospital statistics indicate that the average duration of hospitalization has dropped from several years in the custodial period before 1956 to 37 days in 1975. This trend is a worldwide phenomenon. Most chronic patients currently in our hospitals were admitted as long as 20 years ago. We must remember that these cases are the product of the custodial-care era, and their chronicity may reflect the iatrogenic influences of long-term incarceration as much if not more than an unremitting disease course. Parallel with the reduction in chronic cases has been a considerable increase in the rate of readmission to hospitals, and although some of these readmissions may represent the unimproved patients who were released in error, or rehospitalization for lack of other living facilities, many of them may represent new episodes.

M. Bleuler's (1974) lifetime follow-up of 208 schizophrenic probands provides the most telling comment on the gradual replacement of the prototypical chronic, unremitting schizophrenia by a more episodic pattern of disorder. Bleuler found that only 10% of his patients showed the type of disease course described as typical in Kraepelin's writings and E. Bleuler's early work. Half of the patients achieved an adequate adjustment in the community, whereas 40% lived most of their lives in the community but were hospitalized occasionally.

Even the 10% of unremitted cases in M. Bleuler's (1974) sample were products of the custodial-care era, and it remains to be seen whether even this percentage will be reduced in today's more benign treatment milieu. Moreover, it will also be important to investigate whether the apparently continuous course of chronic illness may actually consist of a closely spaced series of episodes. It is possible that the chronic group of patients may include individuals of such high vulnerability that they almost continuously pass in and out of episodes with relatively slight provoking stress.

At present, the nuclear type of schizophrenic patient seems to have become increasingly difficult to find. For example, Hawk, Carpenter, and Strauss (1975) applied Schneider's (1959) first-rank symptoms; Langfeldt's (1939, 1956) criteria; and Carpenter, Bartko, Langsner, and Strauss's (1976) discriminating signs to patients at intake, but they could not find any nuclear types. Moreover, Hawk et al. were entirely unsuccessful in predicting outcome. To their surprise, 40% of their schizophrenic patients had the best outcome when compared with other psychiatric patients on a composite index of symptom severity, duration of hospitalization, and social adjustment.

With the decline in the prevalence of chronic hospitalization, a new pattern is beginning to emerge as characteristic of the schizophrenic disorder. This course seems to involve brief episodes of illness, recovery, relapse, and recovery. Recently, groups of patients randomly assigned to brief therapy lasting only 4 weeks have shown results as good or better than groups consigned to long-term therapy (Herz, Endicott, & Spitzer, 1976). That patients are responsive to therapy after so short a time lends support to the episodic nature of schizophrenia. Just as relatively rapid improvement is one side of the episodic picture, relapse is the other. The problem of relapse into new episodes has emerged as one of the most prominent issues in the maintenance treatment of patients today. Davis (1975) reviewed 24 double-blind placebo and drug maintenance studies of schizophrenics who had been released after suffering an episode of illness. He noted that 60% of the patients never relapsed during the 2-year follow-up. However, another 40% (65% in the placebo group and 30% in the drug group) did relapse and probably underwent new episodes during the 2-year follow-up. In another study of maintenance treatment, Goldberg, Schooler, Hogarty, and Roper (in press) found that 35% of schizophrenics never relapsed during the follow-up period, whereas 65% did relapse (80% in the placebo group and 48% in the drug group). At least some patients in the relapsed group recovered again. Goldberg suggests that the efficacy of maintenance medications may reflect their prophylactic value in aborting new episodes of illness rather than their effects on a continuously present disease process.

The factors influencing relapse, as well as new episodes, are clearly complex. Reports from England (Leff, 1976) indicate that the relapse rate for patients returned to hostile home environments is far greater than that for patients returned to benign home environments. It is also interesting to note that the relapse rate in developing countries is less than in developed nations, despite the fact that the proportions of deteriorating patients are comparable (Jablensky & Sartorius, 1975). It may be that a lesser degree of labeling and stigmatization in the developing countries affords the returned patient a less hostile ecological niche in the community. It may take a more "advanced" culture to stigmatize an individual into permanent deviance!

In summary, it seems that chronic unremitting schizophrenics are becoming increasingly rare. The many follow-up studies that found a pattern of disorder succeeded by cycles of improvement and relapse support the hypothesis that what is permanent about schizophrenia is the vulnerability, rather than the episodes of psychosis.

How does this hypothesis contribute to understanding the case of the schizophrenic patient who *does* appear to be chronically ill? There are at least four possible interpretations: (a) There are probably some schizophrenics who do remain unremittingly in an episode of illness, although they are in the minority. (b) As suggested earlier, others are probably patients so vulnerable that they recover only very briefly before being catapulted back into an episode. Their periods of emer-

gence from disorder are probably so brief as to go unnoticed. (c) Still others in this category may have been of such poor premorbid ability that their recovery from illness is difficult to discern. In contrast to the good coper who recovers and resumes his place in society, these patients recover most unremarkably: They remain unable to cope with life's exigencies, just as they were unable to do so premorbidly. (d) Finally, there is a group of chronic patients whose coping ability deteriorates far below its premorbid level as a consequence of the onslaught of psychopathology, isolation in the hospital or community, and disuse of social skills. Gruenberg (1967) has described a social breakdown syndrome that may characterize patients in backwards and isolated individuals. The resultant losses in coping ability may indeed be chronic, and they may be mistaken for permanent psychopathology.

Indexing the Onset, Duration, and Offset of an Episode

To test the vulnerability model and its hypothesis regarding the episodic nature of schizophrenia, it will first be necessary to develop objective indexes of the onset, duration, and end of an episode.

Timewise documentation of the onset, course, and termination of a schizophrenic episode will not be an easy task. Ordinarily there will be a considerable time lag between the point at which a patient first enters an episode and the time when he is admitted to a hospital. Once the patient comes to our attention, he is already well into an episode and may even be beginning to emerge from his period of disorder with the help of stabilization on medication. Retrospective interviewing to establish the time, if not the behavioral correlates, of onset generally reveals considerable discrepancy between different informants. Indeed, onset has always posed one of the most baffling problems in diagnosis. Type of onset, whether insidious or sudden, is on the one hand of great prognostic importance and yet on the other hand notoriously difficult to define objectively. The difficulty of determining onset is magnified by the subtlety and complexity of behaviors that have been proposed as signposts of the beginning of a schizophrenic episode. One of the earliest suggestions was that of Berze (Berze & Gruhle, 1929), who described an early and pervasive general lowering of psychic activity. Another proposal, by Jaspers (1963), pointed to the emergence of "ununderstandable" behavior as the earmark of the onset of the disorder. Clearly these phenomena do not easily lend themselves to the formulation and quantification of objective indicators. Moreover, we can most likely not expect them to be accurately reported by patients whose disorder is often characterized by lack of insight.

Even though the patient usually comes to us too late to determine indexes of onset, perhaps we can expect greater success at observing the ending of his episode. Here again, however, several difficulties should be anticipated. Just as the date of admission to the hospital bears no necessary isomorphism with the time of episode onset, neither does time of discharge necessarily correspond to the recovery and emergence from the episode. Often patients who seem to have made adequate recoveries remain in the hospital awaiting the availability of appropriate placements in the community. Likewise, patients who still appear quite ill may be discharged to the care of their families and followed on an outpatient basis. Another problem with evaluating episode offset in hospitalized patients is that we have no opportunity to observe them in the significant contexts in which their psychopathology might be displayed. Given all of these stumbling blocks, what recourse have we?

In searching for indexes of episodes of schizophrenia, we might do well to look initially at such conditions as epilepsy and depression, in which there is some evidence of state- and trait-related markers. Thus, the beginning and end of episodes of epilepsy can be indexed by electroencephalographic characteristics before and after seizure. Similarly, in depression, there have been findings indicating that changes in sleep characteristics (Kupfer, 1976) and motility (Kupfer et al., 1974) serve to mark the beginning and end of episodes. In schizophrenia, too, a reduction in

rapid-eye-movement sleep accompanies acute episodes (Kupfer & Foster, 1975).

Another strategy may be to monitor mini-episodes or acute periods of symptom exacerbation in patients during their hospital stay. These may, in microcosm, reveal some properties of onset and offset of maxi-episodes of schizophrenic disorder. Some progress has been made in this area. Although most of it has concerned a search for indicators of the onset and offset of mini-episodes in depressed patients, it is likely that comparable indexes can be found for schizophrenic mini-episodes induced by visitors, peer stresses, and so on. In a study of the rise and fall of corticosteroid levels in reactive depression, by Sachar, MacKenzie, Binsback, and Mack (1968), it was observed that hormonal stress levels rise when the patient actively struggles with the confrontation of the loss of a love object or when his defense mechanisms falter temporarily. On the other hand, when his defenses are operating adequately and he seems clinically comfortable and adjusted, the hormonal level drops to normal. After the patient has adjusted to the hospital regime and achieved stable hormonal levels, such perturbations may therefore index the eruption of mini-episodes.

Another example is due to Schmale (1972), who suggests that giving in to feelings of helplessness and hopelessness is the earmark of a beginning episode. Luborsky and Auerbach (1969) have derived another potential mini-episode index by examining speech samples of patients in psychoanalysis taken just before instances of momentary forgetting or just prior to reports of migraine headaches or stomach pain. When these speech passages are rated for content of expression, helplessness ratings are found to be much higher than for samples taken at other times during therapy sessions. It is not entirely outside the realm of possibility that mild transient mini-episodes might also be provoked by biochemical challenges. The ethics of such experimental procedures must be carefully investigated so as not to interfere with the civil rights and freedom of the patient, but under skilled personnel, mindful of the ethical issues involved, perhaps some salutory solution can be found.

As we have suggested earlier, the beginning of an episode of psychopathology often follows close on the heels of a period of coping failure. Similarly, once the episode of disorder has subsided, one may expect the patient to once again be able to resume coping at his characteristic premorbid level and style. The concurrence of episodes of coping dysfunction and restoration with episodes of psychiatric disorder and recovery opens the way for investigation of another type of episode marker. If we were to have knowledge of the patient's premorbid level of coping ability, we could use evidence of a sharp plummeting of coping effectiveness and a return to the usual capacity for efficiency as rough boundaries to index the course of a psychopathological episode. Following Goldfried and D'Zurilla's (1969) 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. The entire spectrum of role performance should actually be sampled to see whether the capacity to cope has returned to its premorbid level. Weissman (1975) has recently reviewed 15 scales available for assessing performance in occupational, marital, extended family, and community roles. These might serve to probe for fluctuations in coping capacity that occur during hospitalization, particularly if it were possible to simulate the significant role contexts.

The first step in developing indexes of vulnerability and of the onset, duration, and offset of episodes is to turn to available techniques that objectively and reliably differentiate schizophrenic patients from normal subjects. Experimental approaches to developing such indexes have been described elsewhere (Spring & Zubin, in press-a). The next step is to begin to sort out these differences into those that characterize the patient only as long as his episode persists, those that turn out to be permanent effects of an episode of disorder, and those that characterize the vulnerable individual regardless of whether he is in an episode. Those indexes that characterize the patient only during the episode may eventually turn out to be good markers of the onset and offset of the episode. Those

that characterize the person before, during, and after an episode may turn out to be vulnerability markers. Finally, those characteristics that result from the illness may help in studying the vulnerability to relapse.

Studies of identical twins and relatives can help in discovering indexes that mark the vulnerable individual regardless of the presence of an episode. If both the proband and his nonaffected monozygotic twin or his nonaffected siblings and relatives have the same characteristic in question, that characteristic is a good candidate for becoming a vulnerability index. If the proband has the characteristic when he is in an episode but not when he has recovered, and if his monozygotic discordant twin or other blood relatives do not have the characteristic, it is a good candidate for marking the beginning and end of episodes.

Utility of the Vulnerability Hypothesis

In recent years there has been great concern with the civil rights of patients suffering from mental disorders. Many problems have been raised concerning whether civil rights are in fact abrogated when therapeutic intervention is exerted despite the patient's unwillingness or incompetence to give his informed consent to treatment. Moreover, there is growing suspicion that the consequences of being labeled and stigmatized as mentally ill may be far-reaching, dehumanizing, and injurious to civil rights. In the final analysis, attacks have often focused on the so-called medical model because it is claimed that this model adds insult to injury by considering mental disorders diseases in the same way that cancer and turberculosis are diseases.

There can be little doubt that the "disease" conception of mental disorder, regardless of its validity, influences the expectancies that the community, the hospital staff, and indeed the patients themselves hold toward an individual who has been hospitalized for psychiatric disorder. Perhaps the proposal to regard the schizophrenic as a vulnerable individual who will develop a temporary episode only under certain provocations can help to place this controversy in proper perspective. By shifting our view from regarding a person as suffering from a continuing mental disorder to regarding him as suffering from a temporary episode, and further by regarding him as being *vulnerable* rather than *diseased,* some of the controversy might become superfluous.

In a recent study by Doherty (1975), a comparison was made among patients who at first agreed (but only reluctantly) to assume the patient role and to consider themselves mentally ill but who later rejected the label (rejectors) and two other groups—label acceptors and label deniers. It is interesting to note that the staff expected the patients to accept the patient role if they were to benefit from treatment. Despite this staff expectancy, the label rejectors fared much better than the other two groups in outcome. If staff could be taught to regard patients as vulnerable rather than sick and could proceed to protect them against the stressors that elicit episodes, there would be no need to label them as mental patients or induce them to accept the label. The vulnerability label is perhaps easier to accept and live with, since it presages a time-limited episode from which the patient will recover sooner or later without being labeled schizophrenic for the rest of his life.

There are several other baffling problems that the vulnerability hypothesis may help explain. The apparently spontaneous recovery of long-standing patients falls into this category. The patient may actually have emerged from his episode much earlier, but when recognition of this fact suddenly occurs the recovery is regarded as miraculous. Placebo reactors may also be explicable on the basis of the vulnerability model. If all episodes are time limited, it is to be expected that a certain proportion of patients will in time show improvement without treatment. Placebo reactors may represent the unearned increment of spontaneous improvement.

The vulnerability hypothesis might also be useful in explaining the well-known statistic that only 10% of schizophrenics have similarly affected parents. Moreover, it might elucidate Kety et al.'s (1968) finding of low rates of schizophrenia in the biological relatives of acute schizophrenic probands, particularly in comparison with the higher rates of schizophrenia in the biological relatives of chronic schizophrenic probands. It is possible that the

familial genes of the chronic patients set the vulnerability threshold so low that ordinary life events, despite their minimal stress impact for the average person, are sufficient to trigger an episode.

On the other hand, the low rate of concordance in the blood relatives of acute schizophrenics may be due to the higher threshold of vulnerability in their families. Thus, unless the lives of blood relatives and schizophrenic probands have involved similar stressful incidents, one would not expect high concordance for schizophrenia in these groups. An analogy may perhaps be drawn between the chronic and acute forms of schizophrenia and two conditions of sickle cell anemia: sickle cell disease (the homozygous condition) and sickle cell trait (the heterozygous condition). Whereas homozygous individuals will fall ill regardless of circumstances, heterozygotes will develop the disease only if they are exposed to such specific triggering events as high altitudes, acute alcoholism, or deep anesthesia. Genetically comparable sickle cell trait carriers who avoid these situations will remain free of the disorder, and their vulnerability to sickle cell anemia may never be detected (Harris, 1963). However, the usual exigencies of living in our particular ecological surroundings are sufficient to trigger an episode in the homozygotes. Perhaps acute schizophrenia, like sickle cell anemia, will develop only in those vulnerable individuals who undergo specific adverse circumstances. Unfortunately, the specificity of these circumstances is still to be determined.

If we accept the vulnerability hypothesis, it would seem that therapeutic interventions can take one of two avenues. Vulnerability can be reduced or inhibited from full-blown expression through psychopharmacological intervention. On the other hand, if it is correct, as we suggested earlier, that periods of coping breakdown provide fertile ground for vulnerability to germinate into disorder, psychological intervention might be applied to restore coping ability or reduce the threatening nature of life events that produced the breakdown. Even today, the patients who continue to come to our attention are those who fail to improve and who frequently relapse, rather than the majority who make a successful recovery. It is this group of patients—the failures—who seem to merit the most concerted efforts at psychological intervention. Instead of shunting them, as we now do, through the revolving doors of our hospitals and clinics into the obscure wards of the hospital or the back alleys of the community, we should devote our best efforts and psychological skills to rehabilitating them by enhancing their competence and coping ability. We argue that the challenge of the 1970s is not to find a cure for schizophrenia, since episodes of schizophrenia are in the majority of cases self-curing! Rather, the challenge is to find ways of reducing vulnerability or improving the coping abilities and competence of the vulnerable poor premorbids so that the likelihood of future episodes can be reduced. Even if an episode does occur, the rehabilitated patient will have a better level of coping to return to when the episode passes.

Summary of the Vulnerability Model

We can now summarize our model of vulnerability to schizophrenic episodes, as shown in Table 2. The picture of the schizophrenic individual that emerges from our vulnerability model is highly speculative and controversial, but it has the virtue of being consistent with the data. In other words, it is a tenable if not yet cogent model. According to our findings, the schizophrenic is drawn from the entire range of human variation with regard to intelligence, competence, coping ability, achievement, and all other aspects of the human condition. He may be a top executive, a gifted artist, an outstanding scientist, a blue-collar worker, or an unskilled ne'er-do-well bowery bum. No stratum of mankind is spared from this blight. The one feature that all schizophrenics have in common is not the ever presence of their illness, but rather the ever presence of their vulnerability. Some of them are highly vulnerable and have repeated episodes. Others are relatively invulnerable and have but one brief episode or none at all. When episodes develop they are not lifelong. They terminate sooner or later with or without therapeutic intervention. The majority of schizophrenics today spend the major part of their lives in the community, self-supporting and indistinguishable from the rest of the

Table 2
Vulnerability Model

	Component
Vulnerability	The empirical probability that an individual will experience an episode of psychiatric disorder; an enduring trait
Coping effort	The energy exerted in situations not adequately dealt with by reflexes; orthogonal to vulnerability
Competence	The skills and abilities needed to achieve success in significant role contexts of everyday life; orthogonal to vulnerability
Coping ability	The resultant of the initiative and skill that an organism brings to bear in formulating strategies to master life situations; orthogonal to vulnerability
	Effect of stressors
Episodes of coping breakdown	Occur in all individuals when catastrophic situations arise and render routine coping strategies ineffectual; do not necessarily lead to episodes of psychiatric disorder
Episodes of psychiatric disorder	Tend to develop in vulnerable individuals who are in a state of coping breakdown; time-limited states of illness

population except in the eyes of those who have labeled them. To the best of our knowledge, half the patients recover fully, another 40% have relapses but still manage to spend most of their lives in the community, and only 10% appear to remain chronically ill. Whether their chronicity is endogenous or induced by iatrogenic or custodial factors remains to be demonstrated. At the onset of an episode, the patient's competence and ability to cope adequately with life's exigencies appear to go underground. At the end of the episode, these attributes tend to reappear at their premorbid levels, and the patient can resume his former place in society. In general, the good premorbid patient returns to his formerly good adjustment and the poor premorbid to his poor adjustment. The generally assumed negative relation between vulnerability and competence seems to be largely an artifact of our limited information regarding the full spectrum of schizophrenic illness. Our information is biased by the fact that many episodes in highly competent individuals are not recorded in our statistics and by the fact that those who fill our hospitals and clinics are largely the poor premorbid, relapsing patients.

The picture of schizophrenia presented in this article is not that which dominates our textbooks, and it may consequently be viewed with suspicion. But let us remember M. Bleuler's comments (cited in Rosenthal, 1974) on the observational biases that led his father's generation to regard schizophrenia as a chronic, unremitting, and even deteriorating condition:

> 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. (p. 92)

It is also interesting that several other workers have entertained the possibility that schizophrenia occurs in self-limiting episodes. After concluding that pre-drug-era follow-up showed as good results as drug-era follow-up, Bockoven and Solomon (1975) discuss the common philosophy that permeated the clinical and social management of patients in the two eras.

> This philosophy is based on the idea that the majority of mental illnesses, especially the most severe, are largely self-limiting in nature if the patient is not subjected to demeaning experiences or loss of rights and liberties. Therapeutic management consists first and foremost of removing these negative influences and replacing them with a positive attitude of respect for the patient's needs for human companionship and interest-holding activity. Somatic treatments are prescribed in this context to relieve

specific kinds of suffering and thereby to expedite the spontaneous healing process. (p. 796)

Apparently, Slater and Slater (1944) have proposed a concept of vulnerability that is closely similar to the concept promulgated in this paper. Even more cogently, Falconer (1965) proposed a similar mathematical model under the term "liability to diseases" that has been applied to schizophrenia by Kidd (1975).

Why then is this article necessary, if its main thrust has been anticipated by several earlier publications (Gottesman & Shields, 1972; Meehl, 1962; Millon, 1969; Rosenthal, 1970)? The answer lies in the fact that although the data on which vulnerability was based are known, their organization and application have lagged. Further, although vulnerability has been proposed several times, it has never caught on in schizophrenia. We have arrived at this formulation independently of our predecessors, driven to it not by the weight of evidence that has accumulated but by the way in which it illuminates the meager evidence. In addition, we have suggested biometric approaches for testing the tenability of the vulnerability model by indicating the need for measures of life event stressors, competence, coping, and vulnerability to schizophrenia as well as markers of the onset and offset of schizophrenic episodes (Spring & Zubin, in press).

Reference Note

1. Kety, S. *Genetic and environmental factors in the etiology of schizophrenia.* Paper presented at the McLean Hospital Symposium on the Biological Substrates of Mental Illness, Belmont, Massachusetts, February 1975.

References

American Psychiatric Association. *Diagnostic and statistical manual of mental disorders (DSM-II).* Washington, D.C.: American Psychiatric Association, 1968.

American Psychiatric Association. *Diagnostic and statistical manual of mental disorders (DSM-III).* Washington, D.C.: American Psychiatric Association, in press.

Arieti, S. *Interpretation of schizophrenia* (2nd ed.). New York: Basic Books, 1974.

Audy, J. R. Measurement and diagnosis of health. In R. Shepard & D. McKinley (Eds.), *Environmental.* Boston: Houghton Mifflin, 1971.

Ayala, F. J., & Dobzhansky, T. (Eds.). *Studies in the philosophy of biology.* Berkeley: University of California Press, 1975.

Barnes, M., & Berke, J. *Mary Barnes: Two accounts of a journey through madness.* New York: Harcourt Brace Jovanovich, 1971.

Bateson, G., Jackson, D. D., Haley, J., & Weakland, J. H. Towards a theory of schizophrenia. *Behavioral Science,* 1956, *1,* 251–264.

Berze, J., & Gruhle, H. W. *Psychologie der schizophrenie.* Berlin: Springer, 1929.

Bleuler, E. [*Dementia praecox: Or the group of schizophrenias*] (J. Zinkin, trans.). New York: International Universities Press, 1950. (Originally published, 1911.)

Bleuler, M. The offspring of schizophrenics (S. M. Clemens, trans.). *Schizophrenia Bulletin,* 1974, *8,* 93–108.

Bockoven, J. S., & Solomon, H. C. Comparison of two five-year follow-up studies: 1947 to 1952 and 1967 to 1972. *American Journal of Psychiatry,* 1975, *132,* 796–801.

Brown, G. W., & Birley, J. L. T. Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behavior,* 1968, *9,* 203–214.

Carpenter, W. T., Bartko, J. J., Langsner, C. A., & Strauss, J. S. Another view of schizophrenic subtypes: A report from the International Pilot Study of Schizophrenia. *Archives of General Psychiatry,* 1976, *33,* 508–516.

Cassel, J. C. Psychiatric epidemiology. In G. Caplan (Ed.), *American handbook of psychiatry.* (Vol. 2, 2nd ed.). New York: Basic Books, 1974.

Christensen, J. K. A 5-year follow-up study of male schizophrenics: Evaluation of factors influencing success and failure in the community. *Acta Psychiatrica Scandinavica,* 1974, *50,* 60–72.

Cooper, J. E., Kendell, R. E., Gurland, B. J., Sharpe, L., Copeland, J. R. M., & Simon, R. J. *Psychiatric diagnosis in New York and London: A comparative study of mental hospital admissions* (Maudsley Monograph #20). London: Oxford University Press, 1972.

Davis, J. M. Overview: Maintenance therapy in psychiatry of schizophrenia. *American Journal of Psychiatry,* 1975, *132,* 1237–1245.

Doherty, E. G. Labelling effect in psychiatric hospitalization. *Archives of General Psychiatry,* 1975, *32,* 562–568.

Dohrenwend, B. P., & Dohrenwend, B. S. *Social status and psychological disorder.* New York: Wiley, 1969.

Dohrenwend, B. S., & Dohrenwend, B. P. Social class and the relation of remote to recent stressors. In M. Roff, L. Robins, & M. Pollack (Eds.), *Life history research in psychopathology* (Vol. 2). Minneapolis: University of Minnesota Press, 1972.

Falconer, D. S. The inheritance of liability to certain diseases, estimated from the incidence among relatives. *Annals of Human Genetics,* 1965, *29,* 51–76.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G., & Munoz, R. Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, 1972, *26*, 57–63.

Fleiss, J. L., & Zubin, J. On the methods and theory of clustering. *Multivariate Behavioral Research*, 1969, *4*, 235–250.

French, A. P., & Steward, M. S. Adaptation and affect: Towards a synthesis of Piagetian and psychoanalytic psychologies. *Perspectives in Biology and Medicine*, 1976, *18*, 464–474.

Goldberg, S. C., Schooler, N. R., Hogarty, G. E., & Roper, M. Prediction of relapse in schizophrenic patients treated by drug and sociotherapy. *Archives of General Psychiatry*, in press.

Goldfried, M. R., & D'Zurilla, T. J. A behavior-analytic model for assessing competence. In C. D. Spielberger (Ed.), *Current topics in clinical and community psychology* (Vol. 1). New York: Academic Press, 1969.

Goldsmith, J. B., & McFall, R. M. Development and evaluation of an interpersonal skill-training program for psychiatric inpatients. *Journal of Abnormal Psychology*, 1975, *84*, 51–58.

Gottesman, I. I., & Shields, J. *Schizophrenia and genetics*. New York: Academic Press, 1972.

Gross, E. Work organization and stress. In S. Levine & N. Scotch (Eds.), *Social stress*. Chicago: Aldine, 1970.

Gruenberg, E. The social breakdown syndrome—some origins. *American Journal of Psychiatry*, 1967, *123*, 1481–1489.

Harris, J. W. *The red-cell: Production, metabolism, destruction: Normal and abnormal*. Cambridge, Mass.: Harvard University Press, 1963.

Hawk, A. B., Carpenter, W. T., & Strauss, J. S. Diagnostic criteria and five-year outcome: A report from the International Pilot Study of Schizophrenia. *Archives of General Psychiatry*, 1975, *32*, 343–347.

Holmes, T. H., & Rahe, R. H. The social readjustment rating scale. *Journal of Psychosomatic Research*, 1967, *11*, 213–218.

Herz, M. I., Endicott, J., & Spitzer, R. L. Brief hospitalization: A two-year follow-up. *American Journal of Psychiatry*, in press.

Jablensky, A., & Sartorius, N. Editorial: Culture and schizophrenia. *Psychological Medicine*, 1975, *5*, 113–124.

Jacob, T. Family interaction in disturbed and normal families: A methodological and substantive view. *Psychological Bulletin*, 1975, *82*, 33–65.

Jaspers, K. *General psychopathology*. Chicago: University of Chicago Press, 1963.

Kallmann, F. J. The genetics of mental illness. In S. Arieti (Ed.), *American handbook of psychiatry* (Vol. 1). New York: Basic Books, 1959.

Kanfer, F. H., & Saslow, G. Behavioral diagnosis. In C. M. Franks (Ed.), *Behavior therapy: Appraisal and status*. New York: McGraw-Hill, 1969.

Kety, S. S., Rosenthal, D., Wender, P. H., & Schulsinger, F. The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. In D. Rosenthal & S. S. Kety (Eds.), *The transmission of schizophrenia*. Elmsford, N.Y.: Pergamon Press, 1968.

Kidd, K. K. On the possible magnitude of selective forces maintaining schizophrenia in the population. In R. R. Fieve, D. Rosenthal, & H. Brill (Eds.), *Genetic research in psychiatry*. Baltimore, Md.: Johns Hopkins University Press, 1975.

Kupfer, D. J. REM latency: A psychobiologic marker for primary depressive disease. *Biological Psychiatry*, 1976, *11*, 159–174.

Kupfer, D. J., & Foster, F. G. The sleep of psychotic patients: Does it all look alike? In D. X. Freedman (Ed.), *Biology of the major psychoses: A comparative analysis*. (Association for Research in Nervous and Mental Diseases, Research Publications, Vol. 54.) New York: Raven Press, 1975.

Kupfer, D. J., Weiss, B. L., Foster, F. G., Detre, T. R., Delgado, J., & McPartland, R. Psychomotor activity in affective states. *Archives of General Psychiatry*, 1974, *30*, 765–768.

Langfeldt, G. *The schizophreniform states*. Copenhagen: Munksgaard, 1939.

Langfeldt, G. *The prognosis of schizophrenia*. Copenhagen: Munksgaard, 1956.

Lazarus, R. S., Averill, J. R., & Opton, E. M. The psychology of coping: Issues of research and assessment. In G. V. Coelho, D. A. Hamburg, & J. E. Adams (Eds.), *Coping and adaptation*. New York: Basic Books, 1974.

Leff, J. P. Assessment of drugs in schizophrenia. *British Journal of Clinical Pharmacology*, 1976, Supplement, 75–78.

Luborsky, L., & Auerbach, A. The symptom–context method: Quantitative studies of symptom formation in psychotherapy. *Journal of the American Psychoanalytic Association*, 1969, *17*, 68–99.

MacDonald, N. Living with schizophrenia. *Canadian Medical Association Journal*, 1960, *32*, 218–221, 678–681.

Mechanic, D. Invited commentary on self, social environment and stress. In M. H. Appley & R. Trumbull (Eds.), *Psychological stress*. New York: Appleton-Century-Crofts, 1967.

Mednick, S. A., & Schulsinger, F. Some premorbid characteristics related to breakdown in children with schizophrenic mothers. In D. Rosenthal & S. S. Kety (Eds.), *The transmission of schizophrenia*. Elmsford, N.Y.: Pergamon Press, 1968.

Meehl, P. E. Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, 1962, *17*, 827–838.

Meyers, J. K., & Bean, L. L. *A decade later: A follow-up of social class and mental illness*. New York: Wiley, 1968.

Millon, T. *Modern psychopathology*. Philadelphia, Pa.: Saunders, 1969.

Murphy, L. B. Coping, vulnerability and resilience in childhood. In C. V. Coelho, D. A. Hamburg, & J. E. Adams (Eds.), *Coping and adaptation*. New York: Basic Books, 1974.

Rahe, R. H., Meyer, M., Smith, M., Kjaer, G., & Holmes, T. H. Social stress and illness onset. *Journal of Psychosomatic Research*, 1964, *8*, 35–44.

Rosenthal, D. *Genetic theory and abnormal behavior*. New York: McGraw-Hill, 1970.

Rosenthal, D. Introduction to Manfred Bleuler's "The offspring of schizophrenics." *Schizophrenia Bulletin*, Spring, 1974, *8*, 91–93.

Rosenthal, D., Wender, P. H., Kety, S. S., Schulsinger, F., Welner, J., & Reiber, R. D. Parent–child relationships and psychopathological disorders in the child. *Archives of General Psychiatry*, 1975, *32*, 466–476.

Sachar, E. J., MacKenzie, J. M., Binsback, W. A., & Mack, J. E. Corticosteroid responses to the psychotherapy of reactive depressions: II. Further clinical and physiological implications. *Psychosomatic Medicine*, 1968, *30*, 23–44.

Schmale, A. H. Giving up as a final common pathway to changes in health. *Advances in Psychosomatic Medicine*, 1972, *8*, 20–40.

Schneider, K. [*Clinical psychopathology*] (M. W. Hamilton, trans.). New York: Grune & Stratton, 1959.

Selye, H. The evolution of the stress concept. *American Scientist*, 1973, *61*, 692–699.

Serban, G. Stress in schizophrenics and normals. *British Journal of Psychiatry*, 1975, *126*, 397–407.

Slater, E., & Cowie, V. *The genetics of mental disorders*. London: Oxford University Press, 1971.

Slater, E., & Slater, P. A heuristic theory of neurosis. *Journal of Neurology, Neurosurgery and Psychiatry*, 1944, *7*, 49–55.

Spring, B., & Zubin, J. Attention and information processing as indicators of vulnerability to schizophrenic episodes. In L. Wynne, R. Cromwell, & S. Matthysse (Eds.), *Nature of schizophrenia: New findings and future strategies*. New York: Wiley, in press. (a)

Spring, B., & Zubin, J. Vulnerability to schizophrenic episodes and their prevention in adults. In E. Albee (Ed.), *Proceedings of the First Vermont Conference on the Primary Prevention of Psychopathology*, in press. (b)

Tischler, G. L., Henisz, J. E., Meyers, J. K., & Boswell, P. C. Utilization of mental health services. II. Mediators of service allocation. *Archives of General Psychiatry*, 1975, *32*, 416–418. (a)

Tischler, G. L., Henisz, J. E., Meyers, J. K., & Boswell, P. C. Utilization of mental health services. I. Patienthood and the prevalence of symptomatology in the community. *Archives of General Psychiatry*, 1975, *32*, 411–415. (b)

Turner, R. J. Unemployment and psychological disorder: An assessment of the degree and nature of their association. *Social Policy*, in press.

Wallace, C. M. *Portrait of a schizophrenic nurse*. London: Hammond, Hammond, 1965.

Weiner, H. J., Akabas, S. H., & Somner, J. J. *Mental health care in the world of work*. New York: Association Press, 1973.

Weissman, M. The assessment of social adjustment: A review of technique. *Archives of General Psychiatry*, 1975, *32*, 357–365.

Wender, P. H., Rosenthal, D., Rainer, J., Greenhill, L., & Sarlin, B. *Schizophrenics' adopting parents: Psychiatric status. Archives of General Psychiatry*, in press.

Wilkins, W. L. Social stress and illness in industrial society. In E. K. Gundersen & R. H. Rahe (Eds.), *Life stress and illness*. Springfield, Ill.: Charles C Thomas, 1974.

Wing, J. K., Cooper, J. E., & Sartorius, N. *The measurement and classification of psychiatric symptoms*. Cambridge, England: Cambridge University Press, 1974.

Wing, J., & Nixon, J. Discriminating symptoms in schizophrenia. *Archives of General Psychiatry*, 1975, *32*, 853–859.

World Health Organization. *Report of the International Pilot Study of Schizophrenia* (Vol. 1). Geneva: WHO, 1973.

Zubin, J. Scientific models for psychopathology in the '70s. *Seminars in Psychiatry*, 1972, *4*, 283–296.

Zubin, J., & Burdock, E. I. The revolution in psychopathology and its implications for public health. *Acta Psychiatrica Scandinavica*, 1965, *41*, 348–359.

Zubin, J., Salzinger, S., Fleiss, J. L., Gurland, B., Spitzer, R. L., Endicott, J., & Sutton, S. Biometric approach to psychopathology: Abnormal and clinical psychology—statistical, epidemiological, and diagnostic approaches. *Annual Review of Psychology*, 1975, *26*, 621–671.

Received September 30, 1975
Revision received November 12, 1976 ■