A Heuristic Vulnerability/Stress Model of Schizophrenic Episodes

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Abstract

A tentative model of schizophrenic psychotic episodes is presented, based on the evidence that certain characteristics of individuals may serve as vulnerability factors and that environmental stressors may precipitate psychotic periods in vulnerable individuals. Certain information-processing deficits, autonomic reactivity anomalies, and social competence and coping limitations are viewed as potential vulnerability factors. Stressors in the form of discrete life events as well as the prevailing level of social environmental stress are seen as factors that interact with preexisting vulnerability characteristics to produce vicious circles, which lead, in turn, to psychotic episodes. A distinction among stable vulnerability indicators, mediating vulnerability factors, and episode indicators is suggested to differentiate types of abnormalities that characterize individuals prone to or manifesting schizophrenic disorder. Some major areas of unresolved questions in relation to this view of schizophrenic psychotic episodes are discussed.

The primary purpose of this concluding article in the present series is to integrate the major conclusions of the preceding five articles into a tentative vulnerability/stress model of schizophrenic psychotic episodes that might suggest possible interacting contributions of the individual factors. Due to the relatively broad nature of the constructs that presently need to be used to bridge the diverse possible vulnerability and stress factors, the model is a framework or schema for further developments rather than a formal hypothetico-deductive model. Equally as important as the tentative model is an attempt to identify unresolved issues that now preclude a clearer theoretical integration.

Principal Conclusions of the Preceding Articles

Information Processing and Attentional Dysfunctions. Nuechterlein and Dawson's (1984) analysis of the literature indicates that several deficits on information-processing and attentional tasks show great similarity across populations at heightened risk for schizophrenic disorder, actively symptomatic schizophrenic patients, and nonpsychotic, relatively remitted schizophrenic patients. These populations have been found to exhibit similar deficits in sustaining focused attention in high-processing-load vigilance tasks (e.g., Wohlberg and Kornetsky 1973; Rutschmann, Cornblatt, and Erlenmeyer-Kimling 1977; Asarnow and MacCrimmon 1978; Walker 1981; Nuechterlein 1983; Nuechterlein, Edell, and West, in preparation), in rapid read-out from sensory storage in the presence of patterned noise stimuli (e.g., Neale 1971; Asarnow et al. 1977; Asarnow and MacCrimmon 1978; Miller, Saccuzzo, and Braff 1979; Saccuzzo and Braff 1981; Asarnow, Nuechterlein, and Marder 1983), and in short-term recall involving active processes such as rehearsal, especially in the presence of distraction (e.g., Koh 1978; Oltmanns 1978; Harvey et al. 1981; Frame and Oltmanns 1982).

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Evidence of similar deficits across risk populations and actively symptomatic patients is available for maintaining optimal readiness to respond over time (the reaction time crossover effect), selective attention with immediate response (dichotic listening), short-term recognition memory if processing load is relatively high, and word production and self-editing from semantic memory (referential communication). The presence of similar deficits across different populations suggests that these deficiencies in processing information may be enduring vulnerability-linked characteristics of individuals who are prone to schizophrenic disorders. Early evidence from short-term longitudinal studies that examined small numbers of schizophrenic patients across different clinical states also supports the hypothesis that some information processing anomalies may be enduring vulnerability indicators (Asarnow and MacCrimmon 1982; Frame and Oltmanns 1982; Laasi, Nuechterlein, and Edell 1983).

Nuechterlein and Dawson (1984) also found evidence that certain tasks detect significant deficits during the actively symptomatic schizophrenic period, particularly in poor prognosis or chronic schizophrenic patients, that have not been found in risk populations or relatively remitted schizophrenic patients. Vigilance tasks with relatively low momentary processing loads (Orzack and Kornetsky 1966) and letter-recognition tasks that use very brief exposure times without a visual mask (Braff and Saccuzzo 1981; Saccuzzo and Braff 1981) are examples.

This pattern of findings appears to be related to the momentary processing load that the tasks entail, with significant deficits being demonstrated across risk populations, actively symptomatic schizophrenic patients, and relatively remitted patients under task conditions that involve relatively high momentary processing loads. The deficits seem to be detectable across different stages of processing when task manipulations that sufficiently burden each stage are used (although manipulations that introduce difficult signal-noise discrimination might be particularly effective). Tasks that produce deficits only during the actively symptomatic schizophrenic period in poor premorbid, chronic patients seem to be characterized by relatively low momentary processing loads, often involving rapid recognition of simple stimuli and little short-term memory load.

Nuechterlein and Dawson (1984) suggested that this pattern of deficits is consistent with a reduction in the processing capacity that is available for task-relevant cognitive operations across the premorbid, actively symptomatic, and relatively remitted periods of schizophrenic disorder. This reduction in available processing capacity could have several different causes: (1) the executive decision-making regarding allocation of available capacity is not responding appropriately to task demands; (2) more processing capacity is devoted to task-irrelevant external or internal stimuli; (3) some cognitive operations that are normally carried out automatically in parallel or with relatively little demand for processing capacity are requiring more conscious, capacity-demanding processing; or (4) the total pool of processing capacity is smaller. Nuechterlein and Dawson (1984) concluded that additional experimental procedures that examine different modes of processing within similar task conditions in the premorbid, actively psychotic, and relatively remitted phases of schizophrenic disorder would be helpful in trying to decide among these possibilities.

The deficits in processing information that have been found in poor premorbid or chronic schizophrenic patients only during a period of active symptomatology, Nuechterlein and Dawson (1984) suggested, could be due to either greater reduction of the available processing capacity, a temporary disruption in automatic as well as attention-demanding cognitive processes, or a stable, more severe information-processing deficit in a subtype of schizophrenic disorder.

**Electrodermal Dysfunctions.** Dawson and Nuechterlein's (1984) review of the literature identified two different types of electrodermal anomalies in different subgroups of symptomatic schizophrenic patients. First, between 40 and 50 percent of symptomatic patients fail to exhibit skin conductance orienting responses (SCR-ORs) to innocuous, novel environmental stimuli compared to only 5–10 percent of normal subjects (e.g., Bernstein et al. 1982). Second, among the SCR-OR responder subgroup of patients, there is evidence of tonic sympathetic hyperarousal when these patients are unmedicated (e.g., Öhman 1981). The SCR-OR responder patients generally tend to be more withdrawn and conceptually disorganized, whereas the hyperaroused responder patients tend to be more behaviorally active and excited (Gruzelier 1976; Straube 1979; Bernstein et al. 1981). Electrodermal nonresponders and hyperaroused patients may also differ in performance on attentional and information-processing tasks (Straube 1979; Patterson and Venables 1980). This difference is potentially of theoretical significance because the elicitation of SCR-ORs is thought to be related to the allocation of
processing capacity to external stimuli (Öhman 1979; Dawson et al. 1982) and, as suggested previously, a reduction in available processing capacity appears to be a characteristic of schizophrenic disorders. Finally, poor short-term prognosis in acute schizophrenic patients is associated with electrodermal hyperarousal (Frith et al. 1979; Zahn, Carpenter, and McGlashan 1981), whereas good prognosis appears to be associated with normal ranges of arousal.

The evidence also suggests that these two electrodermal anomalies, along with certain other psychophysiological abnormalities, may be enduring vulnerability-linked traits, in addition to having a better relationship to temporary states. SCR-OR nonresponsiveness, for example, has been found among anhedonic individuals who are at hypothesized risk for subsequent schizophrenic episodes (Simons 1981) as well as among remitted schizophrenic patients (Iacono 1982). Thus, this specific electrodermal anomaly may be present before, during, and after schizophrenic episodes. Tonic hyperarousal, on the other hand, has been found among subgroups of remitted schizophrenic patients (Iacono 1982), but has not been observed among high-risk individuals. A related phenomenon of hyperreactivity to aversive stimuli, however, has been found among the high-risk offspring of schizophrenic patients (Mednick and Schulsinger 1968; Van Dyke, Rosenthal, and Rasmussen 1974; Prentky, Salzman, and Klein 1981).

Dawson and Nuechterlein (1984) suggested that hyperreactivity to aversive stimuli may be a longstanding trait associated with the vulnerability to schizophrenia, whereas tonic hyperarousal may emerge later in the developmental course of schizophrenic disorders. SCR-OR nonresponsiveness also may emerge later as part of a different strategy of biobehavioral coping with aversive environmental stimuli, or it may represent a vulnerability factor for a different type of schizophrenic disorder. The specificity of these anomalies to certain psychiatric disorders, the interrelationships among the different psychophysiological anomalies, and their relationships to other behavioral and clinical dysfunctions are issues that demand substantial additional investigation.

Community and Interpersonal Functioning. Wallace (1984) has reviewed the extensive literature that documents that symptomatic schizophrenic patients exhibit poor community functioning, as well as low quantity and quality of interpersonal relationships. Low rates of social interactions appear to be particularly pronounced for schizophrenic patients, more so than for nonschizophrenic psychiatric patients. However, poor interpersonal functioning does not characterize all schizophrenic patients, nor does it characterize affected patients equally at all times. The types of schizophrenic patients who exhibit interpersonal deficits most markedly include poor premorbid, thought-disordered, and chronically ill individuals. There is also evidence that the rates of social interactions vary with the severity of symptomatology, with social functioning shifting toward normal levels as the patient's symptomatology recedes.

Retrospective archival research and research with high-risk populations indicate that deficiencies in interpersonal relationships often predate the onset of schizophrenic psychotic symptoms. High-risk children of schizophrenic parents, as compared to children of normal parents, were rated as showing less social competence by their peers (Rolf 1972; Weintraub, Prinz, and Neale 1978), were rated by interviewers as higher in social isolation and depression-anxiety and obtained higher Minnesota Multiphasic Personality Inventory (MMPI) Schizophrenia scale elevations (MacCrimmon et al. 1980), and were described by teachers as having less harmonious relationships (Watt, Grubb, and Erlenmeyer-Kimling 1982). A more precise picture of the premorbid characteristics, as well as sex differences, was suggested in some studies, but findings were not sufficiently consistent to warrant a definitive conclusion. A more active social dysfunction (less cooperative, calm, pleasant, and adjusted) was reported for sons of schizophrenic patients, but not for daughters, in some studies but not others.

In any event, the findings suggest that disturbed social behaviors predate schizophrenic symptoms in at least a subgroup of schizophrenic individuals. Thus, Wallace (1984) concludes that some forms of poor interpersonal functioning are potential indicators of vulnerability to schizophrenia, and are not merely correlates or consequences of schizophrenic psychotic symptoms. Moreover, measures of social competency during childhood, adolescence, and adulthood are related to, and partially predictive of, severity of and outcome of schizophrenic disorders within a population that has already become ill.

Life Events, Familial Stress, and Coping Responses. In the article by Lukoff et al. (1984), the possible influence of several social environmental factors on the developmental course of schizophrenic episodes is suggested. The available evidence indicates that stressful life events that
are independent of the effects of the disorder may play a precipitating role in the onset of at least some schizophrenic episodes. Increased numbers of such independent life events are found to be present in the 3 to 5 weeks before episode onset (Brown and Birley 1968; Leff et al. 1973). Other stressful life events are likely to be triggered by the prodromal or residual symptoms of schizophrenic disorder, such as social withdrawal and unusual ideation, or to be associated with lifestyle factors, but might then, in turn, contribute to the movement of symptoms to a clearly psychotic level (Brown and Birley 1968; Leff et al. 1973; Jacobs and Myers 1976; Leff 1976). The premorbid vulnerability level of the patient and the prevailing level of environmental stress need to be incorporated into any model of the role of stressful life events in schizophrenic disorders, because the presence of discrete precipitating life events varies greatly across patients and accounts for a limited portion of the variance in predicting schizophrenic episodes.

One of the prominent influences on the prevailing level of environmental stress is likely to be the emotional atmosphere of the family. Studies in England and in California have consistently found that a high level of criticism, hostility, or emotional overinvolvement directed toward the schizophrenic patient by family members is related to a three- to four-fold increase in psychotic relapse rate in the first 9 months following hospital discharge (Brown et al. 1962; Brown, Birley, and Wing 1972; Vaughn and Leff 1976; Vaughn et al. 1982, in press). These “high expressed emotion” family attitudes appear not to be explained by the level of behavioral disturbance or current symptomatology shown by the patient (Vaughn and Leff 1976; Vaughn et al., in press). Their impact on relapse is meaningfully reduced in families in which the amount of time that the patient spends in direct contact with relatives is relatively low and for patients who are protected by uninterrupted maintenance antipsychotic medication. Conversation with high expressed emotion relatives has recently been found to be associated with a continued state of autonomic arousal in schizophrenic patients in a laboratory and a home setting (Tarrier et al. 1979; Sturgeon et al. 1981). Recent analyses also suggest that these parental attitudes may help to predict schizophrenia spectrum outcome within a sample of disturbed adolescents (Norton 1982). Negative affective style, which is related to high expressed emotion (Valone et al. 1983), and communication deviance have also shown a significant predictive relationship to schizophrenia spectrum disorder outcome in this same sample of disturbed adolescents (Doane et al. 1981).

Finally, the literature reviewed by Lukoff et al. (1984) suggests that schizophrenic patients are often ill-equipped to cope with stressful life events and family tension, because they often lack the information-processing skills to process optimal behavioral alternatives and the social skills to put these plans into action (Eisler et al. 1974; Spivack, Platt, and Shure 1976; Cohen 1978; Hersen et al. 1978; Rochester 1978). Similarly, schizophrenic patients, especially those with more severe residual symptoms, often have smaller social networks than individuals without psychiatric disorder (Pattison et al. 1975; Sokolovsky et al. 1978) and these networks often contain a higher than normal proportion of relatives as opposed to friends and neighbors (Tolsdorf 1976; Garrison 1978; Randolph and Escobar 1982).

Although causal relationships need further study, this pattern of findings is at least consistent with the notion that schizophrenic patients are likely to have fewer and less flexible personal and social resources than most persons to cope with stressful events or familial tensions when they do arise. Thus, the role of stress in schizophrenic disorder may involve not only exposure to somewhat more stressors but also limited coping resources.

The Concept of Relapse. The article by Falloon (1984) reveals the absence of a generally accepted operational definition of “relapse,” despite the importance of the concept in studies of the course and outcome of schizophrenic disorders. The most common definition of relapse has been rehospitalization, but this definition is heavily influenced by social functioning, community tenure, and nonschizophrenic symptoms. For example, Falloon notes that noisy, disruptive behavior or violent behavior is more likely to lead to rehospitalization than is withdrawn behavior, even though the underlying psychopathology may be of similar severity in each case. Another predominant definition found by Falloon (1984) refers to “clinical deterioration in mental state” or “clinical deterioration of such magnitude that hospitalization seemed imminent.” This approach also fails to state the specific symptoms being evaluated or whether symptoms that are characteristically schizophrenic reappeared or were exacerbated (e.g., depression has been included among the states that meet criteria for relapse in some studies of schizophrenia). Relatively few studies have restricted the definition of relapse to
a worsening of characteristic symptoms of schizophrenic disorder or even to a worsening of psychotic symptoms.

The lack of a clear consensus about what constitutes relapse severely limits the comparability of different studies of schizophrenic course and outcome. Beyond the consensus problem, Falloon (1984) has noted that the utility of the relapse concept is limited to the subgroup of schizophrenic patients who show full remission of psychotic symptoms. The course and outcome of a substantial subgroup of schizophrenic patients who improve following treatment but who do not attain a stable clinical remission cannot be described adequately by the concept of relapse. Falloon argues that patients with persisting psychotic symptoms cannot, strictly speaking, be said to have relapsed when their preexisting psychotic symptoms worsen. Factors related to the reappearance of a remitted symptom may, Falloon feels, be quite different from those related to the exacerbation of a preexisting symptom.

Despite these serious problems, Falloon (1984) does not recommend abandoning the concept of relapse. Instead, he recommends ways in which it may be standardized more effectively. First, the concept should be operationally defined in a way that specifies the qualitative and quantitative symptomatic characteristics that qualify as relapse. Second, social functioning, community tenure, and nonschizophrenic symptoms should be assessed independently of the concept of schizophrenic relapse. Falloon concludes that the concept of relapse in the study of schizophrenic outcome should be restricted to symptoms that are characteristic of schizophrenia, such as delusions, hallucinations, conceptual disorganization, thought insertion, thought broadcasting, and thought withdrawal. As another possible alternative, Falloon recommends a "target symptom" approach in which return of a patient's idiosyncratic pattern of symptoms, known from past episodes, is specified as the relapse criterion. Thus, Falloon's suggestions would lead to a clearer delineation of the determinants of the return of characteristic schizophrenic symptoms within schizophrenic patients as separated from the determinants of other psychiatric symptomatology and nonsymptomatic outcome dimensions such as social and occupational functioning.

A Heuristic Vulnerability/Stress Model of Schizophrenic Episodes

Figure 1 summarizes a tentative interactive vulnerability/stress model of the development and course of schizophrenic psychotic episodes that incorporates the major factors that have been examined in the articles within this series. This heuristic schema is proposed as a simplified device by which the principal interrelationships among these factors in the initial development and return of schizophrenic psychotic periods can be organized. This working model has been developed in the UCLA Mental Health Clinical Research Center for the Study of Schizophrenia by the present authors in collaboration with Robert P. Liberman and Charles Wallace and has been presented in earlier forms in Liberman, Nuechterlein, and Wallace (1982) and Dawson, Nuechterlein, and Liberman (1983). The development of this framework has been influenced by the observations of Brown, Birley, and Wing (1972) and Wing (1978) regarding the impact of excessive social stimulation in schizophrenia.

Figure 1. A tentative, interactive vulnerability/stress model for the development of schizophrenic psychotic episodes
The primary components of this interactive model fall into the following four major categories: (1) enduring vulnerability characteristics, (2) external environmental stimuli, (3) transient intermediate states, and (4) outcome behaviors. According to this tentative model, certain preexisting, enduring vulnerability characteristics of the individual interact with stressful external environmental stimuli to produce transient intermediate states of processing capacity overload, autonomic hyperarousal, and impaired processing of social stimuli before the development of psychotic symptoms. These intermediate states and their behavioral concomitants tend to increase the level and frequency of environmental stressors by causing disruptions in the individual's immediate social and familial environment. The feedback loop, in turn, leads to more extreme processing capacity overload, autonomic hyperarousal, and deficient processing of social stimuli. This vicious cycle is viewed as continuing, unless successfully broken, until the transient intermediate states reach an individual's threshold point for the development of schizophrenic reality-distorting, psychotic symptoms.

The principal preexisting, enduring vulnerability characteristics that are incorporated into this schema are attentional capacity limitations, autonomic arousal anomalies, and social competence deficits, as illustrated on the left side of the top row in figure 1. These anomalies refer to response dispositions that are present years before the appearance of psychotic symptoms and that are themselves likely to be products of specific and nonspecific genetic factors interacting with early physical and social environmental influences, with genetic influences presumably being relatively strong for some vulnerability factors. These dysfunctions are assumed to be present before, during, and after schizophrenic psychotic periods. These vulnerability characteristics may be present in subtle form, as in good premorbid patients, or they may be present in quite gross form, as in poor premorbid patients.

Dysfunctions in information-processing operations that involve substantial momentary demand on processing capacity, autonomic hyperreactivity to mildly aversive stimuli, and poor interpersonal coping and competence each seem to be good candidates for vulnerability factors, but other factors may have equal or greater influence. It also seems possible that the poor social and instrumental skills and coping strategies during the premorbid period are a result of the dysfunctions in information processing and autonomic hyperreactivity rather than a separate preexisting factor. Finally, certain vulnerability factors may be relatively specific to schizophrenic disorders, whereas other vulnerability factors may be associated with increased risk for a fairly wide range of disorders.

The two major classes of environmental stimuli that are delineated in this heuristic model are social stressors and a nonsupportive social network, as shown on the right side of the top row in figure 1. Social stressors include discrete life events that are either independent of, or influenced by, the individual's behavior, as well as the ongoing familial stress level, which includes the excessive personal criticism, hostility, and emotional overinvolvement that have been shown to be prognostic factors (see Lukoff et al. 1984). We assume that these factors involve a common stressor effect and therefore have combined them conceptually for present purposes. Nonsupportive social networks include lack of social support from family, friends, coworkers, neighbors, and other sources during times of crisis. The arrows between social stressors and a nonsupportive social network in figure 1 convey the assumption that the impact of a social stressor is partially dependent upon the degree of social support that is available.

The death of a loved one, for example, may be buffered by the presence of a social support system or compounded by its absence.

Additive or possibly interactive effects of the preexisting vulnerability factors and the external environmental stressors are hypothesized to potentiate the transient intermediate states of processing capacity overload, autonomic hyperarousal, and deficient processing of social stimuli, as illustrated in the second row of figure 1. For example, an individual who has a particularly severe limitation in available processing capacity or in the executive function that allocates available processing capacity is likely to reach a state of processing capacity overload very quickly when exposed to a stressful life situation. Such events demand accurate appraisal of the changing stimulus situation and active, flexible processing of cognitive and behavioral coping alternatives. Environmental demand to adjust one's usual, well-practiced patterns of cognition and behavior, which has sometimes been used to define a stressful life event, is likely to entail high levels of cognitive processing capacity: because it is such flexible and adaptable modes of information processing, some theorists have postulated, that require capacity-loading, controlled processing rather than automatic, highly familiar, but
relatively inflexible cognitive sequences (Shiffrin and Schneider 1977).

Similarly, an individual with unusually high autonomic reactivity to mildly aversive stimuli may become tonically hyperaroused when exposed to highly critical significant others for substantial periods of time, as even brief exposures of schizophrenic patients to high expressed emotion relatives during experimental sessions have led to continued autonomic arousal rather than normal habituation (Tarrier et al. 1979; Sturgeon et al. 1981). Furthermore, high levels of arousal are likely to exacerbate further any difficulties with processing in high processing load situations, because overly high arousal apparently leads to difficulty in fine discriminations between relevant and irrelevant stimuli (Broadbent 1971; Kahneman 1973) and reduced ability to engage in parallel or shared processing (Eysenck 1982).

Thus, early prodromal signs of a schizophrenic psychotic episode, such as difficulty concentrating and persistent tension and nervousness (Docherty et al. 1978; Herz and Melville 1980), may be concomitants of the intermediate states of processing capacity overload and autonomic hyperarousal that arise from vulnerability/stress interactions such as those just outlined. These intermediate states very likely have negative feedback effects on the social environment, creating a vicious cycle. As Dawson, Nuechterlein, and Liberman (1983) have suggested:

These intermediate states aggravate the already poor social environment by means of a feedback loop. An individual in the intermediate state, showing defective processing of social cues continue to worsen. The individual may attempt to cope with this state of affairs by withdrawing physically or psychologically from all environmental stimuli. Such a state of withdrawal has been noted in some accounts of the clinical phenomena that precede schizophrenic episodes (Docherty et al. 1978). This behavior withdrawal state might be a point during which an autonomically hyperreactive and hyperaroused individual becomes an SCR-OR nonresponder.

If the individual's attempts to cope with this increasingly aggravated internal and external condition fail, we suggest that inadequate processing of external stimuli and internal cognitive disorganization continue to increase until thinking becomes fragmented, individual features of percepts begin to be incompletely or inappropriately integrated into wholes, and the sense of self-other boundary is weakened. Schizophrenic psychotic symptoms in the form of hallucinations, delusions, and/or florid formal thought disorder begin to appear when cognitive processing reaches these reality-distorting levels, as is illustrated by the outcome in the bottom row of figure 1.

**Stable Vulnerability Indicators.**

Measures that fall into this category index stable, trait-like characteristics that are consistently deviant from normal levels, even during symptomatic remission, in schizophrenic patients, and that are independent of symptomatic changes. The top panel of figure 2 diagrammatically illustrates the defining characteristic of stable vulnerability indicators. If these variables are relatively specific to schizophrenic disorders and are also found to be deviant in risk populations, they might serve to identify schizophrenia-prone individuals and hence have long-term predictive value regarding the likelihood of future schizophrenic episodes. However, long-term predictive accuracy will be incomplete because various vulnerability factors may be present in individuals who never develop schizophrenic disorder. These variables are likely to be relatively distant from the immediate triggering processes for psychotic episodes. Thus, they do not have value for the short-term prediction of the time of episode onset.

**Mediating Vulnerability Factors.**

These measures are deviant from normal levels during psychotic and asymptomatic states, but also covary with level of symptomatology, as shown in the middle panel of figure 2. Thus, they share some characteristics of both vulnerability indicators and episode indicators. The transient intermediate states
Figure 2. Characteristic patterns across clinical states for stable vulnerability indicators, mediating vulnerability factors, and episode or symptom indicators

shown previously in figure 1 are hypothesized to be associated with changes in mediating vulnerability indicators. Some of these variables may play a significant role in the causal chain of events immediately preceding the onset of schizophrenic psychotic symptoms. Detailed analyses might reveal that some mediating vulnerability factors are linked to and might even precede certain types of schizophrenic psychotic symptoms (e.g., formal thought disorder that involves incoherence) rather than all psychotic symptoms. Due to their involvement in processes leading to schizophrenic psychotic symptoms, some mediating vulnerability factors may be useful predictors of an impending schizophrenic episode as well as predictors of the longer term risk of an episode.

Episode or Symptom Indicators. These measures index internal processes associated with certain features of schizophrenic symptomatology. Although these variables deviate from normal levels during symptomatic episodes, they return fully to normal levels during asymptomatic periods. The bottom panel of figure 2 illustrates the defining characteristics of such episode or symptom indicators. These variables will have little short-term or long-term predictive value for subsequent schizophrenic episodes, as they are part of the episode itself rather than vulnerability factors. Such variables may be of use, however, in predicting the duration of the current symptomatic episode. Furthermore, episode indicators will be valuable in the objective evaluation of clinical improvement and treatment efficacy because they index symptomatic states.

To evaluate clearly whether a specific variable falls into one of the three classes suggested here, examination of the same individuals during a schizophrenic psychotic state as well as during a nonpsychotic, asymptomatic state is necessary. Cross-sectional comparisons of schizophrenic patients with normal subjects that use different groups of psychotic and relatively remitted schizophrenic patients will provide suggestive evidence, but cannot fully rule out the impact of cohort differences. Increased emphasis on research designs that employ repeated measures in relatively remitted states as well as in psychotic states will also help to evaluate the possible contribution of various nonsymptomatic abnormalities in schizophrenic patients to the formation of characteristic schizophrenic psychotic symptomatology.

Some Major Remaining Issues

Positive vs. Negative Symptoms of Schizophrenia. The working model that is described in this article emphasizes some predictors and possible contributors to the onset of schizophrenic hallucinations, delusions, and other aspects of the florid psychotic episode that have been described as the positive symptoms of schizophrenia. The negative symptoms of schizophrenic disorder, such as flattened affect, apathy, and poverty of content of speech, which have recently received substantial attention (Andreasen 1982a, 1982b), are typically more chronic in nature and may have different predictors and determinants. The negative symptoms have been postulated by Wing (1978) to be exacerbated by social understimulation rather than overstimulation, and Crow (1980) even suggests that negative symptoms are part of a separate disorder with a more chronic course and a structural brain abnormality etiology. Thus, the extent to which the potential vulnerability indicators that we have identified from studies of risk populations, actively symptomatic patients, and relatively remitted patients are predictive of negative symptoms as opposed to episodes of positive symptoms needs to be evaluated. Negative symptoms also complicate attempts to study relatively remitted patients, as the continued presence of a nonsymptomatic anomaly may be linked to continuous prominent negative symptoms even after the psychotic symptoms resolve. The influence of this factor on findings from postpsy-
chotic schizophrenic patients needs more systematic evaluation.

**Vulnerability Indicators and Subtypes of Schizophrenic Disorders.** The suggestion that negative and positive symptom syndromes might be characteristics of separate underlying disease processes (Crow 1980) raises the larger question of whether different potential vulnerability indicators might be related to different subtypes of schizophrenic disorder. For example, in actively symptomatic schizophrenic patient groups, skin conductance nonresponders have been found to be characterized by emotional withdrawal, part of the negative symptom syndrome. One possibility is that SCR-OR nonresponders represent a subgroup of schizophrenic patients with negative symptoms who were characterized by this electrodermal anomaly from birth, and that the findings of skin conductance hyperreactivity to aversive stimuli among offspring of schizophrenic parents reveal a vulnerability indicator for another form of schizophrenic disorder. Alternatively, electrodermal hyperresponsivity to aversive stimuli might be the primary initial vulnerability factor and skin conductance nonresponsiveness may emerge as part of a biobehavioral coping pattern of withdrawal from stimulation during the late premorbid period or at the onset of psychosis for a subgroup of schizophrenic patients. Thus, currently available evidence does not allow us to determine whether skin conductance responder-nonresponder subgroups among actively symptomatic patients represent patients with differing initial vulnerability factors or patients who have developed different electrodermal response patterns from a similar initial anomaly.

The indications that some information-processing deficiencies among schizophrenic patients are associated with a family history of schizophrenic disorder (see Nuechterlein and Dawson 1984) suggests the possibility that at least certain information-processing deficits might be vulnerability indicators for forms of schizophrenic disorder with stronger genetic or familial transmission patterns. Their role in other forms of schizophrenic disorder might be quite different. The possibility that any given vulnerability factor may be relevant to only a subgroup of schizophrenic patients has recently received increasing attention as part of the current emphasis on biological heterogeneity in the etiology of schizophrenia and other major psychiatric disorders (Buchsbaum and Rieder 1979; Buchsbaum and Haier 1983).

**Outcome Components.** The schema in this article is an organizing tool for examining some possible vulnerability and stress factors that may contribute to the periods of psychotic symptoms in schizophrenic patients. Outcome in terms of social and work adjustment in schizophrenic disorders is apparently related only weakly to symptomatic outcome (Strauss and Carpenter 1974, 1977). Thus, the differential contribution of various vulnerability and stress factors to social and work adjustment deserves separate evaluation. Some of the potential vulnerability factors suggested in these articles, especially those involving social coping and competence, may be more strongly related to social and work adaptation following psychotic periods than to the periods of positive psychotic symptoms themselves. Future studies of the onset and course of schizophrenic disorders would benefit from clearer differentiation of the social, work, and symptomatic dimensions of outcome suggested by Strauss and Carpenter (1974, 1977) to allow further evaluation of their differential predictors and determinants.

**References**


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Videotapes on Schizophrenia Available

The Video Center of the George Warren Brown School of Social Work in cooperation with several community and mental health organizations has produced four videotapes on the following topics relating to survival issues for chronically mentally ill persons and their families in the community.

Coping With a Chronically Mentally Ill Relative in the Community—The two videotapes on this topic were produced in cooperation with the Alliance for the Mentally Ill, St. Louis Chapter. Each videotape presents the experiences of a family which has had some success surviving the multiple problems arising from caring for a mentally ill relative in the community. The videotapes are intended for an audience of parents and relatives of chronically mentally ill persons who could benefit from a vicarious sharing of experiences with the families on the videotapes.

Psychosocial Rehabilitation: Two Agencies Based on the Fountain House Model—These two videotapes were produced in cooperation with the Missouri Department of Mental Health, Independence Center, and Places for People, St. Louis, MO. Each videotape presents a psychosocial rehabilitation agency from the point of view of its members. The tapes are intended for professional audiences as well as for families and mentally ill persons who could benefit from knowing what it's like to experience psychosocial rehabilitation "from the inside."

For more information about the rental or purchase of these videotapes, please contact: Dr. David Katz, Video Center, Box 1196, Washington University, St. Louis, MO 63130.