Figure 1. An oversimplified diagram to clarify the relationship of life events to illness. Source: From Dohrenwend BS and Dohrenwend BP, eds., Stressful Life Events: Their Nature and Effects. Copyright © 1974, John Wiley & Sons, Inc. Reprinted by permission of John Wiley & Sons, Inc.

"normal" responses to life events has all too often foundered on a confusion between the various uses of the term normality. For this reason alone the growing interest in predisposition as a key factor for the psychopathological investigation of disorders (9) represents a promising step for future studies. Once more, however, it should be emphasized that the paradigm adopted is essentially epidemiological. For the integration of clinical and research studies the epidemiological method constitutes the scientific core of social psychiatry as a major field of inquiry (10).

References
5. Cooper B. Reactivity: A Psychiatric Concept and Its History; unpublished.

Commentary by Joseph Zubin, Ph.D.*

It is quite apparent that Bebbington's limitation of his discussion to depressive disorders is unnecessary since it could equally apply to schizophrenia and to other mental disorders and even physical disorders. The role of psychosocial fac-

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tors and stress in all of these conditions has been documented adequately (1,2,3). It is interesting to note that the statement he quotes from Jaspers speaks of psychopathology in general rather than of depression.

The basic thrust of this paper is to distinguish the disorder known as depression from the concept of emotional stress that may or may not be a forerunner of the disorder, according to the author. It is, perhaps, a little more difficult to distinguish between emotional stress and depressive disorder because there is often a seeming confluence between the normal aspects of emotional stress and the more severe symptoms of psychopathological depression, a confluence which is not so readily present in the emo-
ever, that the connection between emotional distress and depression may be more continuous and linear than the corresponding connection between emotional distress and a schizophrenic episode.

I have attempted to deal with the role of psychosocial factors in the development of schizophrenia in a fashion similar to that of the author’s approach to depression. It is interesting that the author chose to attribute to Kessler the conceptual framework for the diathesis stress or vulnerability model which actually goes back somewhat earlier historically (4,5) and was finally modified and amplified by Zubin et al. (2,6,7).

The essence of the vulnerability model is that an episode of illness will develop in a vulnerable individual who is subject to sufficient endogenously or exogenously produced life event stressors to exceed his level of tolerance, as shown in Figure 1.

The source of the vulnerability may lie either in the biological domain (constitutional makeup) or in the environmental and psychosocial domains. The biological domain includes the genetic, internal environment, and neurophysiological models, whereas the environmental domain consists of the ecological model, and the psychosocial domain of the developmental and learning theory models. Another model has recently come to the fore as a revival of the formerly discarded neuropathological model via the CAT scan data showing enlarged ventricles of the brain in some schizophrenics and the evidence from cerebral blood flow studies that show differences in brain functioning (8).

No integration of these seven models had been provided until development of the concept of vulnerability, which may be considered the common denominator of all these models. If they were able to be squeezed into a goblet, the resulting elixir would characterize a vulnerable person, no matter how he arrived at his vulnerability.

A consideration of even the most highly developed of the etiological models, the genetic, leads to the conclusion that it is neither sufficient nor necessary for the development of a schizophrenic episode nor of a depressive episode. Witness the fact that only 40% of monozygotic pairs of twins are concordant for the expression of the schizophrenia genotype into a phenotype, that the penetrance is only 0.26 (6, quoting R. Golden), and that many schizophrenics have no family history of the disorder. What prevents the development of the phenotype in the vulnerable? Insofar as most individuals are sufficiently bombarded by insults from the chemical, physical, and social environment to warrant the development of sufficient stress, it cannot be the absence of life stressors that prevents episodes in the vulnerable. While the nonvulnerable (or mildly vulnerable) can absorb the stress homeostatically and develop at most a self-contained crisis, the vulnerable will develop an episode unless certain protective factors are available. Among those that suggest themselves are social networks, ecological niches, and premorbid personalities. Thus, even vulnerable individuals do not have to develop episodes if certain protective factors are present permitting them to cope adequately despite adversity.

What are we to make of the finding that life events account for only 10% of the variance of symptomatology (9,10)? It should be noted that we regard life events not as causes but as triggers that set off an episode in the vulnerable. Life events may be causal in early life development and may induce high vulnerability in a person on their own account, but these are remote
life events that may be of a persistent nature (e.g., living in deprivation in a ghetto). They are not the trigger that sets off an episode but are the underlying causes producing vulnerability.

The life events that serve as triggers are the immediate (within 3 months) events eliciting the vulnerability of the person. Why do we not find triggers for each episode? The fault may lie in the methods we have adopted for tallying life events and how we define them. Thus far, we have been dealing only with the dramatic life events (death, loss of jobs, marriage, etc.) and have utilized the Holmes and Rahe (1) schema for assessing the degree of stress produced by the events or the panel method of Brown (11). However, at least in schizophrenia, if not in depression, these dramatic life events may not be the only factors. Much more subtle elements in daily life may serve as the stress-producing trigger, such as imagined insults, dreams, nightmares, missed opportunities, expectations that did not materialize, disappointments, etc.—things which never get recorded. It is the impact of such events and the need to cope with them through either accommodation or assimilation (Piaget) that makes for the triggering force.

In determining whether a life event has enough clout to incite an episode, some criterion of stress must be accepted. For example, we might define a stressful event as one which is sufficient to disrupt the social network. When this source of support fails, the stress displays its full force and brings about an episode. Another definition might be that the event is sufficiently stressful to disturb the physiological, psychological, and social functioning of the individual. This disruption can be monitored by noting whether it disrupts the usual everyday routine behaviors of eating, sleeping, seeing friends, keeping up with appointments, etc. Such a series of ratings might indicate an underlying stressful life event even if the person does not report it.

In a recent attempt at tackling this problem in outpatients, to determine whether they will relapse or have another episode, we developed the following strategy. On each visit to the outpatient clinic, the patient was asked how well he slept last night, how well he ate, how well he took care of his daily schedule, etc.—a list of some 30 parameters. He was also asked what kind of a day he had, and whether anything special happened. The interviewer also rated the kind of day the patient had. These ratings might serve as a seismograph of the impact of stress and the Richter-like scale they provide may yield a measure of the stress.

Preliminary data suggest that several of the individual items taken together are predictive of global evaluations made by the interviewer. Often, even when no traumatic events were reported, the ratings showed a decline and in such cases, sometimes when confronted, the patient would recall a traumatic event such as a disposess notice which he had not reported previously. This technique is still in its experimental stages but it seems to be promising.

One of the corollaries of this model is that the episodes are time limited and when they end the patient returns to his premorbid level. If the premorbid level was adequate, we regard the patient as improved or cured. If he failed to cope with his life exigencies premorbidly, he may be regarded as chronically ill even though the episode has ended, because he still appears to be incapable of coping with life's exigencies. But in reality he is a pseudo-chronic, since he has returned to his poor premorbid level. While this does not explain all of chronicity, the proportion it explains needs to be determined by adequate premorbid and follow-up studies.

Not all the chronics can be regarded as belonging to this pseudo-chronic category. Some of the long-term follow-up studies in Europe (12) found that fully 80% of the patients were never continuously in the chronic category. The small proportion of chronics who persist in their episodes accumulate over the years and give the false impression that schizophrenia is largely chronic. However, whether chronicity is an essential part of the natural development of schizophrenia or a psychosocial artifact is debatable. The characteristics of the chronic schizophrenic, especially the negative symptoms, are also found in residents of prisons and understaffed nursing homes. Furthermore, family history is not related to outcome nor is it associated with diagnosis, but it is related to the psychosocial factors which tend to maintain chronicity (deprived status due to poor social network, ecological niche, or premorbid personality). Chronic schizophrenia seems to bear no relationship to organicity, biochemical, or somatic factors.

It is clear that the two disorders, schizophrenia and depression, are parallel in many ways and, in fact, affective disorders lend themselves more readily to the proposed vulnerability model insofar as they are more easily perceived as episodic and have probably a smaller proportion of long-term chronics.
The central issue addressed by Bebbington is how to distinguish between depressive illness and normal response to stress. He posits two requirements for this distinction to be valid: (a) that the two conditions be separable on symptomatic grounds, and (b) that they have different relationships to adverse circumstances.

I am hesitant to accept these requirements as validating criteria for this distinction. Employing the first, we would say that there is no distinction between hypertension due to chronic glomerulonephritis and hypertension due to renal artery disease. These two conditions have identical symptomatic presentations but have different etiologies and different treatments.

An example closer to home is the validity of situational ("reactive") depression. In the NIMH Clinical Research Branch Collaborative Program on the Psychobiology of Depression (1), we could discover no differences in symptomatology between patients with situational (i.e., reactive) major depressive disorder (as judged by the examining clinician) and those with major depressive disorder that did not qualify as situational. Whether the presence or absence of precipitating factors makes a real difference in the condition will require further investigation, but clearly the requirement of a symptomatic difference would not be discriminating. Unipolar and bipolar depression often cannot be distinguished on symptomatic grounds, but few would argue that they are not different.

The second criterion is that "illness and distress should bear different relationships to adverse circumstances," a position similar to that taken in earlier publications (2). By this I believe he means that a linear relationship exists between social adversity and emotional distress, but not between social adversity and depressive illness. George Brown and Tirril Harris also interpret this criterion "that a disease could not be seen as caused by stress, and it is this which distinguishes it from distress, which is, by definition, so caused" (3). They argue for a multifactor etiology of disease including stress, in contrast to a "single final cause." I would agree stress is an important factor in most, if not all, diseases, including cancer. Yet no one would assert that no difference exists between distress and cancer.

I would offer a different approach—a set of approaches, actually—to view the interaction among these conditions. Let me begin with a few clarifying definitions.

Non-specific emotional distress can be defined as mental upset that is in direct response to—and caused by—undesirable, untoward life events (i.e., stressors). Upsetness may take the form of dysphoria, anxiety, general discomfort, or other negative states. The stressors may be acute (such as death) or ongoing (such as marital discord). Distress should be distinguished from adjustment, which refers to the emotional and physical response to any event and may include both positive and negative aspects. In contrast, distress refers only to negative emotional responses.

Dimensions of psychopathology are groups of symptoms that assert together around a common psychopathological concept. Examples include depressive symptomatology, anxiety, psychoticism, hostility, and psychosocial specificity. They are often assessed by self-report measures such as the SCL-90, the MMPI, and Beck Depression Inventory (4,5,6). Similar to non-specific emotional distress, dimensions begin in normality and extend into clear abnormality.

Affective disorders include several clinical syndromes composed of dysphoria, associated features, duration, and social or functional impairment. Several different clinical syndromes exist, including bipolar affective disorder, unipolar affective disorder, and melancholia.