Stress and Schizophrenia: Some Definitional Issues

by Bonnie Spring

Abstract

This article discusses definitional ambiguities in research on the role of stress in the etiology of schizophrenia. Implications of the change to *DSM-III* criteria are considered, as is the question of whether prior research samples have overincluded acute schizophrenics. It is suggested that the problem of defining schizophrenia's time of onset is one of the thorniest in this literature.

Three different operational definitions of stress are examined. Stress may be considered a response involving disruption in homeostasis, or as a stimulus with objectively specifiable properties. Stress is also defined interactionally with reference to characteristics of the individual and the surrounding life context. Relative merits of the three definitions are evaluated, and an attempt is made to clarify the differentiation between formative and triggering effects of stress. Further study of the impact of remote life events on vulnerability is encouraged.

Is stress a precursor of schizophrenia? Professional opinions on the question have completed several full cycles over the course of the past century. The fact is that stressful life events have been found to account for somewhat less than 10 percent of the variance in the onset of schizophrenia and depression (Andrews and Tennant 1978). However, it has been suggested that problems of sample size (Rabkin and Struening 1976) and sample selection (Dohrenwend and Egri 1981) may have rendered this estimate overly conservative. This article suggests that conceptual issues in defining stress and dating illness onset have never been adequately resolved. Therefore, both the magnitude and the importance of the association between stress and schizophrenia may warrant reexamination.

Defining Schizophrenia

For more than a century, psychopathologists have attempted to come to grips with the major problem of the schizophrenia researcher: heterogeneity. Schizophrenics are a diverse group in terms of their symptoms, premorbid social functioning, and even precipitating circumstances. It has long been hoped that if a core group of "true" schizophrenics could be isolated based on homogeneity of these presenting features, a unitary etiology might perhaps emerge. It may be worth questioning whether such a fantasy bids fair to come true. If we carefully reduce the schizophrenic populace into a nuclear, core, or process group, and various other peripheral clusters (e.g., schizotypal, reactive, schizoaffective), will we find a uniform causal pathway for the nuclear group? Or will we continue to rediscover the historic truth—that the schizophrenic clinical picture emerges as a final common pathway along many different etiological high-


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ways. If, in the final analysis, schizophrenics persist in arriving at their illnesses by divergent routes, we would have to admit that the question beginning this article is naive. Indeed, it might never be possible to develop a formulation of the role of stress in schizophrenia that applies with any validity to the majority of schizophrenics. Stress might be a sufficient cause for some schizophrenias, a necessary cause for others, and an irrelevant factor for still others. Progress in explaining the role of stress may only come by way of evaluating subgroups of schizophrenics who are homogeneous with respect to etiology as well as presenting clinical features. The role of stress may truly be different for each subgroup.

Although we are faced with the possibility that the problem of etiologic heterogeneity may be here to stay, we should consider what efforts have been made to arrive at clinically homogeneous schizophrenic groups. Interestingly, presence of a precipitating stress has historically been a feature used to contraindicate the diagnosis of true schizophrenia, even though typical psychotic features might be present. Thus, Langfeldt (1956), Schneider (1959), and Mayer-Gross, Slater, and Roth (1969) distinguished between schizophrenia on the one hand and the "emotion psychoses" or schizophreniform illness on the other—the latter arising in response to exogenous factors but mimicking "endogenously produced" schizophrenia. Kety et al. (1968) and Pope and Lipinski (1978) continue the historic tradition that equates schizophrenia with biological causation, insidious onset, and chronicity. These researchers question whether "acute schizophrenias" of sudden onset and discernible precipitants should be regarded as schizophrenias at all. The chronicity element has now been incorporated into the nomenclature of the new DSM-III, wherein schizophrenia cannot be diagnosed unless signs of the illness have persisted for 6 months. Illnesses of shorter duration are classified as "schizophreniform disorders," and those that appear transiently as a result of a psychosocial stressor are called "brief reactive psychoses." However, neither is considered a subtype of schizophrenia.

Ironically, constraints of experimental design may have led stress researchers to select as schizophrenic probands primarily those patients who would no longer qualify for schizophrenic diagnoses according to DSM-III. If one wishes to determine whether stressful life events precede the occurrence of schizophrenia, it is desirable that the illness onset can: (1) be clearly dated and (2) have occurred recently so that surrounding life circumstances can be accurately remembered. However, the gain in precision that these selection criteria afford may be offset by the cost of having a sample population that is unrepresentative of schizophrenics in general. In searching for schizophrenic cases with recent, datable onset, Brown and Birley (1968) excluded 60 percent of a schizophrenic hospital population. By definition, first-break cases with recent onset—less than 6 months ago—would now be seen as "schizophreniform" rather than schizophrenic. However, it should be pointed out that there are disadvantages in attempting to do research on stressful precipitants of a "true" schizophrenia that is more than 6 months old, since Uhlenhuth et al. (1977) found that 5 percent of life events are forgotten per month over an 18-month period.

Clearly, we need to address the problem of defining illness onset. The onset of schizophrenia has in the past been most commonly dated by the appearance of psychotic symptoms. An implicit criterion is that the patient must seek care or somehow come to the attention of mental health or medical facilities, or we can never learn of the onset of his psychosis. As the revised version of DSM-III points out, prodromal features often antedate psychotic symptoms. It is, therefore, basically an arbitrary decision whether we define onset as the appearance of prodromal or psychotic symptoms. Logically, it would be best if we could define onset as the point when there is a veritable certainty that a schizophrenic psychosis will occur. When schizophrenia is already a foregone conclusion, we might say that it has, in essence, begun. But when has this point been reached? Some theorists might trace onset to the point at which a very high level of vulnerability has developed, since vulnerability beyond a certain threshold will inevitably translate into disorder. Only the timing of this occurrence remains uncertain. Brown, Harris, and Peto (1973) suggest such an approach. However, according to a different approach—the diathesis-stress model—vulnerability implies no such inevitability. All will depend on whether, not just when, a triggering event causes vulnerability to germinate into disorder. But what
does a trigger initiate—the prodromal syndrome or the psychotic syndrome?

An analogy might be drawn to the following problem: When has an individual lost a job? Is it at the point at which his performance deteriorates to a totally unacceptable level? Some later time when this fact reaches the attention of his supervisor? The point at which the trusted supervisor informs the employer? The employer’s memo to the personnel department asking that termination be initiated? The date of the notification sent to the employee? The time when the employee realizes he is leaving? Or the precise date when he cleans out his desk and goes? By analogy, we conventionally date the onset of schizophrenia to this last, desk-clearing behavior. We could for all practical purposes date it much earlier. Also by analogy, if we looked for the immediate precipitating cause of job loss defined as actually leaving one’s post, we would identify the cause as receiving a notification from the personnel department. Actually, this mechanical cause is only the last in a sequence of causal events. The causal factors of greatest importance occurred much earlier.

In psychopathology research, the problem of dating schizophrenia’s onset is often treated as a methodological pitfall to be circumvented by improving interrater agreement. In actuality, it is a conceptual problem. Schizophrenia is a disorder that often develops by gradual accretion. It is difficult to determine when behavioral eccentricities have passed the threshold into a paranoid or schizoid personality, when these have shaded into a prodromal syndrome, and when this, in turn, has met the criteria for frank psychosis. The issue is further complicated by the fact that not all schizophrenias progress through this sequence of manifestations. The “acute schizophrenias,” appear to descend suddenly without warning signs, although, as discussed earlier, there is a question as to whether such an uncomplicated onset is compatible with a diagnosis of true schizophrenia.

Clearly, present procedures for defining onset are basically arbitrary. If we wish to maintain criteria that apply to acute schizophrenics as well as to other subtypes, then we must use psychotic symptoms as the benchmark, since these are the only features manifested in common by all the subtypes. If we exclude cases that begin acutely, then it becomes possible to consider earlier markers of onset. However, we are on equally unsure footing when we propose alternative criteria. Logically, we could use the first manifestations that indicate with absolute certainty that a schizophrenic psychosis will ensue. Can we identify such markers? The appearance of a schizoid personality is clearly unsuitable since many individuals manifesting such traits never develop schizophrenia (Bleuler 1978). We do not yet have adequate ways to assess the level of vulnerability quantitatively, and to test whether high vulnerability is the precursor of an inevitable episode of schizophrenia. The implications of the appearance of a prodromal syndrome are as yet untested.

Given the complexity of schizophrenia’s onset, perhaps the best that could be done is to examine separately those factors immediately preceding the onset of the schizoid or paranoid personality, the schizophrenic prodromal state, and the schizophrenic psychosis. Only in this way is it possible to avoid the pitfalls of mechanical and superficial causal inference described earlier in the job loss analogy, and to understand any sequence of causal factors that may bring about schizophrenia.

Defining Stress

In order to investigate whether stress is causally related to schizophrenia, it is essential that stress be defined and measured independently of other factors related to the onset of schizophrenia. If the occurrence of the predictor/independent variable (stress) is influenced by the criterion/dependent variable (schizophrenia) or vice versa, our ability to draw inferences about causality is severely restricted. In offering any suggestions for disentangling measurement of stress and schizophrenia, it may be best to start at square one—with how stress is defined for the purposes of research. Three major categories of definition appear in the contemporary research literature.

Stress as a Response. Stress may be defined and measured by a disruption or alteration in biological, physiological, emotional, or behavioral homeostatic functioning. Measures of autonomic nervous system arousal, changes in endocrine function, reports of emotional distress, and disruptions in coping behavior have all been used as indices of stress. The tendency to define stress in terms of disruptions in homeostasis is rooted in
modes of thinking from the biological sciences. The classic response definition of stress is: A state manifested by a specific syndrome consisting of all nonspecifically induced changes within a biologic system (Selye 1956). Response definitions of stress have some face validity in terms of colloquial usage. We often conclude that we are under stress based on signs of autonomic nervous system activation (e.g., rapid heart rate, perspiration, dizziness, flushing), a feeling of emotional distress, or the fact of performance deterioration.

Unfortunately, response definitions of stress are of limited value for research on stress as a cause of schizophrenia, because the measurement of independent and dependent variables is severely confounded. If behavioral disruption or disordered coping is taken as the index of stress, it is likely to be confounded with the criteria for diagnosing schizophrenia's onset, as well as with early signs or prodromal features of the disorder. In the DSM-III diagnostic criteria for schizophrenia, one required sign is significant impairment in two or more areas of routine daily functioning—e.g., work, social relations, or self-care. Thus, the diagnosis of schizophrenia of necessity implies behavioral impairment. Moreover, Phillips (1968) has proposed that longstanding inefficiencies in social functioning are prime components of vulnerability to schizophrenia. Since the onset of psychotic symptoms often occurs against a backdrop of disordered coping, thereby provoking even further deterioration of daily functioning, labeling either the backdrop or the further plunge "stress" adds no new information and certainly no explanatory power.

If biological responses are used to index stress, similar problems are encountered. Patients in an episode of schizophrenia are characterized by a host of abnormalities in physiological arousal (Mednick 1958; Venables 1964; Broen and Storms 1967; Kornetsky and Eliasson 1969). If such signs are detected shortly before psychotic symptoms appear, the findings may merely signify that the episode has begun but not yet reached full clinical bloom. Moreover, since there is some evidence (Mednick and Schulsinger 1968) that individuals at risk for schizophrenia show unusual patterns of arousal, the researcher who calls these signs of stress may be mistakenly examining stable components of the predisposition for schizophrenia.

Indexing stress by anxiety or subjective reports of distress cannot take us much further, particularly since a sense of panic and subjective discomfort is one of the first symptoms of encroaching schizophrenia (Docherty et al. 1978).

**Stress as an Interaction Between Stimulus and Moderating Factors.** An interactionist approach, based in social science modes of thought, defines stress only in relation to the characteristics of the individual and the surrounding life context. A situation or an event is considered to be a stress if it is perceived as such by the individual, or if in the judgment of a rater, it exceeds the available resources for coping. The common theme in the interactionist approach is that it is impossible to define stress objectively, without regard to the person or life context on which it has an impact. Thus, the effects of any life event are believed to be controlled and moderated by the idiosyncratic cognitive or perceptual structures of the individual (Mechanic 1967; Lazarus 1974), and by the general environmental supports available to the individual (Brown 1974).

As is the case for the response definition of stress, the interactionist definition incorporates conventional wisdom. One person's stressor is another's challenge. This approach makes excellent common sense and splendid clinical sense. When used in combination with other definitional approaches, it can yield important findings. However, when used alone, no definition of stress is more pernicious to the goal of drawing a causal relationship between stress and schizophrenia.

If stress is defined by the perceptions or cognitive interpretations of the individual, then measurement of stress is hopelessly confounded by phenomena that may be symptoms or predisposing factors for schizophrenia. Cognitive idiosyncracies are the most salient feature of schizophrenia, and the tendency to feel overwhelmed by many events may well be an early symptom of illness. Moreover, eccentricities in cognition or perception may predispose both to schizophrenia (Mednick and Schulsinger 1968; Strauss, Harder, and Chandler 1979) and to the tendency to construe life events in an unusual way. Any real relationship between life events and schizophrenia might be obscured by a predisposition on the part of the preschizophrenic individual either to overreact to minor events or to underreact to real danger. The direction of the bias in meas-
urement could not even be specified with certainty. Brown differs from these theorists in emphasizing environmental resources and explicitly attempting to exclude subjective appraisals of stress (Spring and Coons, in press).

**Stress as a Stimulus.** Finally, stress may be defined in terms of stressors, or stimuli whose properties are objectively specifiable, and whose probability of occurrence is independent of the actions or characteristics of the individual on whom they have an impact. This is the definitional framework used in laboratory experiments on stress wherein noxious physical (e.g., noise, heat, crowding) or psychological (e.g., failure, competition) stimuli are imposed on randomly assigned subjects. It is also the basic definitional approach used in most studies of stressful life events, although it must be acknowledged that there is some tentativeness to the assumption that life events “assign themselves” randomly across individuals. If there is any validity to this assumption for at least some types of events, then this is the only definition of stress suitable for drawing causal inferences, because it is the only one for which independent and dependent variables are likely to be unconfounded.

In life events research, there is controversy over whether events are most validly seen as falling into discontinuous categories or as falling along a continuous dimension of magnitude. In laboratory research, it is unlikely that heat and noise stressors would be conceptualized as falling along the same continuum. Similarly, in life events research, there is some evidence that particular types of stressors may have specific causal impact for certain disorders—for example, exits from the social field and depression (Paykel et al. 1969). However, events are also commonly scaled along a continuum of magnitude.

What assumptions are made in attempting to specify objectively the properties of life events? Along what dimension should magnitude be scaled? Holmes and Rahe (1967) originally scaled life events in terms of the amount of readjustment they would require the average person to make. Paykel, Prusoff, and Uhlenhuth (1971) subsequently scaled events for the degree of upset they would normatively engender. The assumptions underlying such scaling procedures are borrowed from models of stress in the physical sciences. For example, in Hooke’s Law and Young’s Modulus of Elasticity, a stressor (e.g., a weight) is quantified according to the load it places on an object (e.g., a string). The degree of distortion provoked in the object is assumed to be linearly related to the physical magnitude of the stressor. This assumed parallelism between stimulus and response dimensions provides the rationale for having subjects provide normative magnitude ratings of stimuli along a hypothetical response dimension. Even though life events are scaled along a putative response dimension, it is important to note that the weights were neither literally derived nor have they ever been successfully validated against direct measures of response disruption or symptoms. Indeed, the fact that correlations with most outcome variables are as high for simple counts of events as for summed magnitude scores (Rahe 1974) suggests that the hypothetical magnitude dimension may lack validity. Although this is disappointing from the standpoint of the assumptions of the model, it also implies that stress scores are at least partially independent of symptoms and behavioral outcomes. Precisely such independence is required to test hypotheses about the causal relationship of stress to schizophrenia.

Using objectively specifiable life events to define stress is a first step in evaluating stress independently from schizophrenia. At least this strategy attempts to disentangle the independent variable from the welter of disturbances in arousal, disruptions in coping, cognitive eccentricities, and peculiarities of lifestyle that are known to be associated with the dependent variable. Of course, it remains open to question whether the scaling procedures currently in use do yield anything resembling an objective metric for stressors. The rationale, derived from principles of magnitude estimation in psychophysics (Stevens and Galanter 1957), is solid, but there is no corresponding physical metric that could be used to validate life events scales.

Although I have argued that objective measures of stress are the ones primarily useful in investigating whether stress is a cause of schizophrenia, I would now like to suggest that for descriptive purposes it may be useful to make tandem use of subjective and objective stress ratings. If subjective distress were the only measure of stress, then any link with symptomatic distress might be explicable by response bias. However, if both subjective and objective scorings were used, then empirical
support might be found for the clinical observation (Beck and Worthen 1972; Donovan, Dressler, and Geller 1975) that schizophrenia often follows subjectively traumatic but objectively trivial events.

Disentangling Measurement of Stress and Schizophrenia

Even if the definitional issue were to be resolved, the research problem of disentangling stress from other factors related to the emergence of schizophrenia can only be described as mind-boggling. Potential confounds exist at many levels: in the implicit or explicit use of information about stress to make a diagnosis of schizophrenia; in the fact that the schizophrenic illness can provoke stress; in the fact that prodromal conditions may also provoke stress; and finally, in the possibility that aspects of vulnerability present long before the emergence of illness may also influence the occurrence of stress.

When information about stress is used in deciding whether a diagnosis of schizophrenia is warranted, a test of the hypothesis that stress causes schizophrenia can only lead to spurious results. The dominant belief in contemporary medicine is that schizophrenia is a biological disorder that arises endogenously. Hence, it is likely that some proportion of cases presenting typical schizophrenic symptomatology may be diagnosed nonschizophrenic, if there is evidence that the illness was preceded by stress. Therefore, to some unknown degree, prevailing contemporary theoretical beliefs about the etiology of schizophrenia impose such a selection bias that the presence of a stressful event leads diagnosticians to ignore the presence of schizophrenia. It might be argued that diagnostic criteria should legitimately include information other than symptoms (Feighner et al. 1972; Pope and Lipinski 1978), so that stress might be a justifiable parameter in the diagnostic process. If this premise is accepted, however, it must also be acknowledged that none of the parameters assessed in the diagnostic appraisal can be used to validate the diagnosis etiologically, concurrently, or predictively. Only by quintessential circularity can we test whether stress is an etiological factor in schizophrenia when diagnostic assignments are, in part, implicitly based on evidence of recent stress.

It is likely that information about stress is used implicitly rather than explicitly in determining whether a schizophrenic diagnosis is warranted. Therefore, the confounding is not really eliminated by the usual procedure of deleting stress from the list of explicit criteria for diagnosing schizophrenia or dating its onset. The only real solution may be to assure that diagnosticians are blind to information about recent life events. The counterpart strategy is to keep life events interviewers blind to diagnosis, but this is rarely feasible. These solutions are clearly not undone by the usual research design, in which the same clinician administers interviews about life events and symptoms, but is then asked to evaluate stress and diagnosis independently.

A second problem of nonindependence adheres in the fact that the schizophrenic's peculiar behavior during an episode is capable of provoking a storm of stressful occurrences. Since these stressors are consequences of the illness, we must find some way of separating them from earlier stressors that might have caused the episode. The strategies generally adopted to achieve this end are twofold: an attempt at carefully dating the onset of the episode and efforts to eliminate stressors that might have been brought on by unusual behavior signifying the onset of disorder (Brown and Birley 1968).

However, the Brown and Birley solution may fall short of the mark of attaining independence for two reasons. One is the conceptual problem of dating onset. The other is that although the patients' preepisode behavior may not be grossly unusual, it may be influenced by the underlying vulnerability or by prodromal features in such a way that the behavior alters the probable incidence of stress. It would be difficult to say in this case that the stressors cause schizophrenia. Rather, they might be epiphenomena in a cycle whereby the vulnerability gives rise both to schizophrenia and to stress.

Since patients often "slide" toward illness by a very gradual process, it is important to recognize as possibly nonindependent any stressors whose occurrence is influenced by the developing features of the preschizophrenic on this gradual slide. Whether we perceive such events to be associated with the early symptoms of the onset of illness will basically be an arbitrary function of whether we choose early or late criteria for defining onset. If we define onset at the beginning of the slide, these events will seem to be secondary consequences of illness. If we de-
fine onset at the very end of the slide, with the appearance of psychotic symptoms, then such events may appear to be associated with the manifestations of high vulnerability.

It is now apparent that as far back as childhood, there may be certain behavioral features associated with preschizophrenic conditions. Watt et al. (1970) found that a substantial proportion of preschizophrenic boys showed poor school performance, emotional instability, and aggressiveness. Many preschizophrenic girls appeared oversensitive and introverted. Each of these behavior patterns might be expected to enhance the occurrence of stress.

Fontana et al. (1972) have noted that life events are often goal-directed; that is, they may be brought on by the individual to achieve certain ends. Some of the events immediately preceding the onset of schizophrenia may illustrate this principle. Leff (1976) has suggested that in the years immediately before illness, schizophrenics show an increasing tendency to withdraw from contact with individuals close to them. Instead of fatefuly befalling the patients, some of the events in the period before onset may be willfully provoked in pursuit of social isolation. To a neutral third party with certain preconceptions about life, these events may seem unpleasant and stressful. To a preschizophrenic who possibly harbors different preconceptions about the value of social isolation, they may seem well worth the cost.

Leff (1976) reviews evidence that in the few years before a first schizophrenic attack the individual often leaves his family of origin in a small town or rural area and moves to a single-person household in the “transitional zone” of a city. Relocations or changes in residence have been found to be significantly more prevalent for first-episode preschizophrenics than for controls during a comparable time period (Jacobs and Myers 1976). Events involving other people (Schwartz and Myers 1977), and family-related events (Jacobs and Myers 1976) also appear in significant excess during the time period immediately preceding a schizophrenic episode. Clearly, the preschizophrenic who becomes embroiled in interpersonal conflicts and changes in residence may experience stress and suffer a disruption of social support. However, it may also be the case that these events are secondary consequences of preschizophrenic behaviors and goals.

The Nature and Timing of the Maximum Stress Effect

In research on stress and schizophrenia, two intriguing questions remain unanswered. The first of these is when we might expect stress to have the greatest impact on the probability of schizophrenia’s occurrence. Are the most momentous stressors those that are recent or those that are remote (i.e., occurring earlier in the individual’s developmental history)? The best known studies of stress and schizophrenia have concerned events immediately (within 2 years) preceding the appearance of schizophrenic disorder (Brown and Birley 1968; Brown et al. 1973; Jacobs, Prusoff, and Paykel 1974; Jacobs and Myers 1976). These studies have suggested that schizophrenics report more recent stressful life events than normal controls, but fewer than depressives. Findings generally indicate that stress is frequently a precursor of schizophrenia, although it is not necessary in all cases. Results do suggest that recent stress might play a causal role in the initiation of schizophrenia. However, as has already been pointed out, this inference is limited by possible atypicalities in the schizophrenics who comprised the research samples, as well as by difficulties in pinpointing schizophrenia’s onset.

An entirely separate question is whether remote stressors occurring early in the lifespan may also play a causal role in schizophrenia. There is an increasingly impressive body of evidence to suggest that preschizophrenics suffer more than the normal incidence of pregnancy and birth complications (McNeil and Kaj 1978) as well as early parent loss (Wahl 1956; Garmezy 1974a, 1974b). Might such remote stressors be involved in schizophrenia’s etiology, and if so, what might the mechanism be?

In the research literature, two types of stress effects have been postulated: triggering and formative. Most researchers of recent events see themselves as investigating a triggering rather than a formative effect of these events on schizophrenia. Conceptually, however, the distinction between the concepts of triggering and formative effects has never been very clear. The formative-triggering distinction is sometimes taken to mean that a factor with a formative effect is a necessary condition for the occurrence of schizophrenia (Rabkin 1980). By contrast a factor with a triggering effect is not a necessary condition—i.e., not a causal factor. Rather, a trigger may influence some ancillary property
of the disorder, such as its timing. Alternatively, based on the mathematical assumptions of their "brought forward time" index, Brown et al. (1973) have argued that the distinction between formative and triggering factors can be made on the basis of which factors are most important etiologically. If life events are more important etiologically than predispositional factors (stable properties of the individual and his environment), then they have a formative effect. If they are not, then, by default, a triggering effect cannot be ruled out, although it cannot be directly confirmed either. As Dohrenwend and Egri (1981) point out, however, the inference that the "brought forward time" index can be used to judge the relative impact of stress versus predisposing factors remains untested, since life events and dispositional properties have never been measured and directly compared in the same study. Finally, the formative-triggering distinction has been defined with reference to whether life events advance the appearance of schizophrenia a lot or a little in time (Brown et al. 1973). Although this latter formulation is again mathematically clever, it is also somewhat teleological and not directly testable.

Quite clearly, the distinction between formative and triggering effects of stressful life events has remained unclear and open to multiple interpretations. It may, therefore, be fruitful to examine the theoretical model that differentiates between these two effects of stress. The distinction is best elaborated in the vulnerability (Zubin and Spring 1977; Spring and Coons, in press) or diathesis-stress (Meehl 1962; Rosenthal 1970) models of schizophrenia. First, it is postulated that some stressors contribute to the formation of the vulnerability or diathesis for schizophrenia. Vulnerability is described as a relatively stable trait of individual difference that mediates the risk or capacity to become schizophrenic. Generally, remote stressors early in life are thought to have the most pronounced formative effects, although it might also be possible that recent trauma could exert formative effects on vulnerability. The vulnerability model also postulates a second triggering or precipitating role of stress. The precipitating role of stress is qualitatively different from the formative effect that augments vulnerability. Vulnerability is postulated to remain latent until it is elicited by a precipitating or triggering event. The precipitant does not add to vulnerability, but rather causes vulnerability to become manifest. Stated differently, a "trigger" precipitates an episode but does not alter the threshold for future responses to new triggering events. An event with a formative effect changes vulnerability and modifies the response to future stressors. The effects of a trigger are reversible, whereas those of a formative event are relatively irreversible. Nonetheless, according to the theoretical model, a triggering event of some magnitude is always needed to bring about a schizophrenic episode, even if vulnerability is very high. Thus, triggering stressors influence not just the timing of schizophrenia, but its very probability of occurrence (Spring and Coons, in press).

Conclusions

The purpose of this article has been to raise some new questions about the role of stress in the etiology of schizophrenia. Currently it appears that stress may play some causal role, but its impact is smaller than we might have wished and certainly less specific than the role of loss in depression. It has frequently been concluded that stress plays only a triggering role at best, rather than a formative role. However, the triggering-formative distinction has remained open to multiple interpretations. Before the nature of the effect of stress on schizophrenia can be clearly discerned, it seems important to pay more careful attention to several issues of timing. For instance, is the onset of schizophrenia defined as that point when the psychotic syndrome becomes manifest, or is it more reasonable to conceive of onset as occurring at some point in the prodromal syndrome? In addition to questions about the onset of illness, the role of remote events in the development of schizophrenia bears further investigation. The search for causal factors which have the potential to trigger episodes needs to be supplemented by greater attention to the effects of remote stressors on the formation of vulnerability to schizophrenic illness. This information should prove useful in our attempts to define the relationship between stress and schizophrenia.

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**Acknowledgment**

This research was supported, in part, by Grant No. MH-32355-01 from the National Institute of Mental Health to Dr. Spring; and by Grant No. MH-31154-04 from the National Institute of Mental Health to Dr. Kety, as well as Grant No. RK-07046 from the National Institute of Health Biomedical Sciences, a William F. Milton Fund Grant, and a Joseph H. Clark Fund Grant to Dr. Spring. The author is grateful to the Biometrics Research Unit, Highland Drive VA Medical Center, Pittsburgh, PA, for consultation.

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