Stressful events have been widely accepted as a cause of a variety of psychiatric disorders, including affective disorders, most neuroses and schizophrenia. In the first two instances, the published data are extensive (see reviews by Lloyd, 1980; Paykel, 1978) yet the findings are far from conclusive (Tennant, 1983). In the case of schizophrenia, the empirical evidence is indeed scant (reviews by Dohrenwend and Egri, 1981; Day, 1981; Rabkin, 1980), but in the last few years a little more empirical evidence has emerged from Leff and his colleagues in London, which has not been critically reviewed. The present review therefore reassesses the evidence that life event stress may play a causal role in the onset or relapse of schizophrenic episodes.

Most of these studies are methodologically unsound. The major limitations include the following:

1. Life event inventories have been most widely used, and these usually lack sensitivity in definition of items, include a limited number of items, and make no provision for excluding those events which may be caused by illness or the prodromata of illness (i.e. the events are not independent of illness) and as discussed in several reviews (e.g. Sarason et al., 1975; Jenkins et al., 1979; Tennant et al., 1981).

2. Problems in recall of events, both the progressive fall off of recall with the passage of time (which, it is hoped, will affect cases and controls equally) and 'effort after meaning', which will be most marked in studies using normal controls as a comparison group.

3. Problems in defining 'onset' of illness: many studies have equated the hospitalisation of the subject to the date of onset, so these studies can only assess, in fact, whether life events precipitated hospitalisation in schizophrenics rather than the disorder itself.

4. The 'schizophrenics' studied are often not representative of schizophrenic admissions. Because of the need to ensure that events preceded illness onset, a number of studies have included only 'schizophrenic' patients with acute onsets. Using DSM-III criteria, most of these patients would not be regarded as schizophrenic but rather as having schizophreniform, schizo-affective or brief reactive psychoses. Most studies were published prior to the advent of DSM-III and have used ICD or similar criteria. Any reference to 'schizophrenia' in this article implies the latter 'broad' criteria, with all their limitations.

5. It is not clear whether events provoke psychotic symptoms or only affective and neurotic symptoms in subjects who are already psychotic or in the prodromal phase of illness. One study (Schwartz and Myers, 1977a) addressed this issue indirectly and showed that life stress had a stronger association with affective and neurotic symptoms, than with psychotic symptoms in a sample of schizophrenics.

The ideal method to assess stress and schizophrenia would therefore be a community-based prospective study of incidence. Because of the low incidence of the disorder, such an approach is not feasible. Two approaches thus remain. The first is the quasi-experimental approach, in which a single and extremely stressful experience is assessed in relation to illness; these include, for example, combat, natural disasters, and migration. Studies of combat seem to indicate that, if psychoses occur, they are both infrequent and brief (Wagner, 1946). For migration, there is good evidence that
hospitalisation for schizophrenia is increased (Odegård, 1932), although it is not certain whether migration was the cause or effect of illness. Furthermore, some studies show that migration (within the same country) can be associated with lower hospitalisation rates (see review by Murphy, 1977).

One interesting quasi-experimental study presents acceptable evidence that stressful life changes may provoke schizophrenic episodes. Steinberg and Durell (1968) showed that admission for schizophrenia was more likely in the first month after army induction than in subsequent months; the findings applied to both volunteers and draftees. Presuming an adequate exclusion at intake of frankly psychotic subjects, the study is one of the few presenting any worthwhile evidence that stressful life changes contribute to the onset of schizophrenia.

The second type of study is the case-control design, examples of which will be discussed in three groups. The first group of six studies compared schizophrenics with depressives. Three assessed events in relation to hospitalisation rather than the onset of disorder (Beck and Worthen, 1972; Eisler and Polak, 1971; Lahniers and White, 1976), and only the first of these showed any difference, with depressives experiencing more stressful events. Of the three studies that examined events in relation to onset (not hospitalisation), two reported more events in depressives (Clancy et al., 1973, Jacobs et al., 1974), while Leff and Vaughn (1980) found no difference in the frequency of undesirable independent events in depressive neurotics and schizophrenics. The latter were the only researchers to ensure that events were not illness related. The above findings have no bearing on whether or not life events precipitate schizophrenia; such a question can only be answered by studies using normal and appropriately matched controls.

The second group (of three studies) has compared schizophrenics with normal controls. The first of these (Schwartz and Myers, 1977a) shed little light on the causal relation of events to schizophrenic episodes, since the patients studied had been discharged from hospital for some time (two to three years) and were simply presumed to be in remission. An interesting, and perhaps paradoxical, finding was that life events correlated more strongly with neurotic symptoms than in the schizophrenics. This finding was confirmed first, by other analyses by Schwartz and Myers (1977b), in which life events were found to correlate more strongly with anxiety, depression and somatic concern than with psychotic symptoms. Second, Birley and Brown (1970) found in their schizophrenics that only severe affective symptoms, namely depressive delusions and preoccupation with death, were significantly related to life events; schizophrenic symptoms were not. The other two studies using normal controls and assessing independent events in relation to onset of disorder had discrepant findings. Jacobs and Myers (1976) found no differences, but Brown and Birley (1968) reported that independent events were more common in schizophrenics in the three weeks before onset than in normal controls. Because the effect was limited to this three-week period the authors regarded events simply as a trigger that determined when a person who was otherwise predisposed to schizophrenia would decompensate. Thus of the only two studies designed to show that events contributed to schizophrenic onset, only one had positive results (Brown and Birley, 1968).

The third group of four studies assessed life events and schizophrenia in relation to two other variables; medication and expressed emotion (EE). Because normal controls were not included, no light is shed on the central question of whether events provoke schizophrenia. Nonetheless, the findings are worth examining. Two studies have assessed life events in relation to medication in schizophrenics. The first of these (Birley and Brown, 1970) was limited by small numbers and a failure to differentiate between first-onset schizophrenics (who had not taken major tranquillisers) and schizophrenics who relapsed (after ceasing their medication). Nonetheless, it was found that events were no more likely to produce an episode of illness in those not taking drugs than in those on medication. The second study (Leff et al., 1973) found that events were more common in schizophrenics who relapsed on medication than those on placebo. They argued that drugs might minimise stress-induced relapse.

The final two studies assess life events in relation to expressed emotion (EE) in schizophrenics (and in one study, depressives as well). Leff and Vaughn (1980) found no difference in life event frequency in depressed subjects from high and low EE families, whereas life events (three weeks and three months prior to relapse) were more common in schizophrenics from low EE families. The authors interpreted these findings as suggesting that either events or high EE caused relapse in schizophrenics. In the absence, however, of normal controls, such a conclusion is not justified; all one can say is that events and EE have different relationships in the
families of schizophrenics compared with depressives. In the second report of two separate studies, Leff et al. (1983) found first, that in unmedicated patients who relapsed, events were more frequent in low EE than high EE families. Second, they found that events were associated with schizophrenic relapse but only in medicated patients from high EE families. Again it is to be remembered that this third group of four studies, with the exception of the last, has no bearing on the association of events to schizophrenia onset or relapse.

Summary

Studies of life events and schizophrenia are few. Most have such methodological limitations that make any causal interpretation of their findings impossible. Of the three studies with appropriate methodology and controls, Brown and Birley (1968) found very recent life events trigger onset, Leff et al. (1983) found events were capable of precipitating relapse, under certain restricted circumstances, while Jacobs and Myers (1976) found independent events appear to have no significant effect. The widely held view that life events can precipitate schizophrenic episodes may be supported by our own clinical impressions; it is as yet not well supported by empirical data. Future research should perhaps focus on other definitions of these psychoses, such as is now embraced by DSM-III. Perhaps then a relation between stressful events and some of the non-depressive functional psychoses might be demonstrated.

References


BROWN, G. W. & BIRLEY, J. L. (1968), Crises and life changes and the onset of schizophrenia, J Health Soc Behav 9, 203-14.


SCHWARTZ, C. C. & MYERS, J. K. (1975b). Life events and schizophrenia II. Impact of life events on symptom formation, Arch Gen Psychiatry 34, 1242-5.


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