The metamorphosis of schizophrenia: from chronicity to vulnerability

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SYNOPSIS On the evidence of long-term follow-up studies of cohorts of patients in Europe the outcome of schizophrenia appears to be changing from chronicity to an episodic course with a more favourable outlook. While the reasons for this change are unclear it is suggested that schizophrenia is characterized essentially by a state of vulnerability to the disorder. This vulnerability may or may not give rise to an episode of illness, depending on the incidence of triggering life event stressors and on the moderating influences of social networks, ecological factors, and premorbid personality. The traditional view of schizophrenia as an essentially chronic condition reflects not so much the natural history of the disorder as iatrogenic influences, lack of satisfactory extra-mural care, the accumulation of a relatively small proportion of truly chronic illnesses and the failure to recognize the termination of an episode of illness in patients with poor premorbid personalities. Evidence for the episodic nature of schizophrenia is presented.

INTRODUCTION

It is uncertain whether schizophrenia has afflicted mankind since antiquity or whether it arose as a mutation in the nineteenth century (Torrey, 1980). Our perception of its nature has been changing since the concept emerged from the general category of madness or insanity during the mid-nineteenth century. This metamorphosis, however, has been a gradual process, leading to an awareness that the outcome of the disorder is no longer as poor as Kraepelin maintained. Recent long-term follow-up studies in Europe have lent further support to the idea that the disorder is more benign than was originally believed.

What are the major changes in our view of schizophrenia as we enter the 1980s? What produced these changes and what are their implications for the future? The major change has been in the sphere of neither aetiology, nor treatment, nor diagnostic schemata, although advances have been made in all these areas. It would seem, rather, that the disorder itself has undergone a benign metamorphosis, as has occurred with some infectious diseases. Thus, Kraepelin at first regarded chronic deterioration as a criterion for dementia praecox and only later agreed to lower the estimate of deterioration to 70%. Manfred Bleuler, the son of Kraeplin’s colleague, by contrast, categorized only 6% to 15% of his probands as persistently chronic.

The reasons for this improvement in outcome are still obscure. There would seem to be several contributory causes: (1) the opening up of the formerly custodial hospitals; (2) the widespread introduction of chemotherapy, sociotherapy, and psychotherapy; (3) the birth of community mental health centres; and (4) the increase in tolerance towards aberrant behaviour in the community. In addition, account must be taken of the sociological and demographic shifts in the population – for example, changes in birth-rate and socioeconomic cycles and the politicization of the mental health movement (Brenner, 1973; Hare, 1974; Kramer, 1978; Zubin & Burdock, 1965).

In this paper the evidence for the change in the character of schizophrenia will be presented first, followed by a brief overview of progress in techniques of description and in the knowledge of aetiology. The level of understanding that has been reached in both will be outlined, and the gap
between data and theory will be delineated. Finally, a vulnerability model of schizophrenia will be proposed.

THE BENIGN METAMORPHOSIS OF SCHIZOPHRENIA

It is maintained that two types of changes have occurred in the nature of schizophrenia: first, the disorder has become more benign in outcome; secondly, it has been transformed from a persistent state to one which is more or less episodic. These two developments are no doubt interconnected, but they will be discussed separately here.

The nature of this change is difficult to demonstrate in the United States because the return of patients to the community without proper planning has resulted in their deterioration and has complicated the therapeutic picture. In some European countries, however, the process has existed for a longer period and the changes have been well documented. Thus, in a summary of major follow-up studies, Shapiro & Shader (1979) include only three studies from the United States, all published in 1939, before the current developments in the evaluation of diagnosis and outcome. Furthermore, none of these studies was carried out longitudinally by the same psychiatrist over the patients' lifetime.

By contrast, long-term European studies tend to be more satisfactory. Notable among them is Manfred Bleuler's lifelong follow-up of 208 schizophrenic probands and their families (Bleuler, 1978). In view of the rigorous diagnostic tradition at the Burghölzli Hospital and the close personal attention which Bleuler gave to his patients, the diagnostic uncertainty that shrouds many earlier and concurrent studies can be discounted and the patients' relatively favourable outcome is all the more striking.

Bleuler describes in detail the basis for his diagnosis of schizophrenia (pp. 15-16). The features characterizing all cases diagnosed as psychotic, before a diagnosis of schizophrenia was entertained, constitute his research diagnostic criteria:

1. Confusion to a degree where the patient's train of thought would be incomprehensible to a normal person, or at least difficult to understand, and this not only in relation to topics of severe emotional impact.

2. A total incapacity for emotional empathy that had no relation to the patient's actual condition on any normal psychological basis.

3. A state of abnormal excitement or stupor of intense magnitude that could not be explained psychologically and might last more than just a few days.

4. Illusions and hallucinations of long durations.

5. Delusions.

6. Total and abrupt changes in activities, commonly either by total neglect of matter-of-course obligations or by senseless, unprovoked acts of brutality against others.

7. The firm conviction on the part of normal family members and intimates that the patient had suddenly become completely different, and that he could no longer be understood.

All the probands who were subsequently diagnosed as schizophrenic had to demonstrate at least three of these characteristics.

Secondly, after excluding organic causes such as brain disease or injury, endocrine disorder, toxic conditions, or severe physical illness, a schema for a diagnosis of schizophrenia was made on the basis of the generally accepted positive criteria such as:

1. 'Keeping a double set of books', in the sense that a normal degree of intellectual accomplishment potential was evident along with severe psychotic manifestations.

2. Definite schizophrenic distractedness in thinking, that is clearly distinguished from other kinds of confusion such as: loss of train of thought, disjointed thinking of the acute exogenous reactive type; results of labile attentiveness; results of failure to comprehend a situation; organic disorder with perseverations; confabulations and verbose rambling; dream-like thinking in exceptional hysterical manifestations; monodectic thinking in times of dejection.

3. Severe inner strife reflected in all affective expression (voice, gesture, movement, direct verbal reports about the emotions), so that the customary capability for affective contact was lost.

4. Severe manifestations of depersonalization, such as transivitism, obedience automatism, identity loss, thought hearing, the feeling of complete personality change (not being a person any more); but only when several of the same manifestations are identifiable in the same
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patient, not when only one of them was in evidence, as sometimes happens without schizophrenia disorders.

(5) Severe catatonic muscular symptoms while the body is healthy and the mind alert.

(6) Delusions of the type frequently found in the schizophrenias but rarely in other illnesses – for example, constant, manifold and disordered relationship delusions without any corresponding mood change.

(7) Hallucinations as they are frequently found among schizophrenics but rarely in other patients – for example, grotesque sensations of irradiation, hearing everything the patient plans to do, etc.

(8) Secondary severe memory illusions and hallucinations, while primary memory function remains intact.

All the probands exhibited several of these characteristics, but the diagnosis of schizophrenia was oriented more towards the entire syndrome than towards the sum of individual symptoms.

Since the course and outcome of schizophrenic illness in a given individual fluctuates over time, Bleuler regarded outcome as stabilized only when the patient’s status remained relatively unchanged for at least 5 years. Nearly two-thirds (63%) of first admissions and their siblings who had experienced at least one episode came into this category. The remainder are not included in the outcome statistics, but a majority of these underwent episodes of schizophrenia and improved sufficiently to enter the ‘mild’ or ‘recovered’ category. Hence, the overall outcome would have been even better if they had been included. It might be claimed that the improved outcome in Bleuler’s patients is attributable to the attention he paid to them. This, however, in no way diminishes the trend, since the natural course of an illness may be approximated more closely under the most favourable environmental conditions.

Bleuler (1941) reported an earlier comparison group 10 years earlier, consisting of schizophrenic probands from his New York and Swiss (Pfaffers Clinic) investigations who had entered hospital as first admissions, and their schizophrenic relatives who had been hospitalized at least once. This group seems comparable with the groups included in the 1972 study. The 1941 study followed up the patients who were admitted in the period of 1933–6 to the Burghölzli clinic, to the Pfaffers Clinic, and the 1929–30 New York sample. The 1972 report followed up the patients who entered hospital a decade later, in the year 1942–3. Although these two cohorts were only a decade apart, a crucial difference in outcome emerges when the proportions of patients by each stabilized end-state are analysed (Table 1).

Bleuler defined the cases with severe chronic end-states as mute or very confused in speech. They either did not work at all or at best performed purely mechanical chores and required constant care. The patients with moderately severe end-states had preserved their faculties, as demonstrated by normal behaviour in front of visitors. Though totally uncommunicative, they performed hard work or cared for others, but their work performance was appreciably lower than before the onset of their episodes. The patients with mild end-states exhibited definite schizophrenic symptoms, showed generally normal behaviour, performed useful work, lived either outside the institution or on quiet wards. Patients classified as recovered were fully employed in gainful work and had resumed their former roles in society, though some showed residual defects.

Bleuler’s data show that the proportion of severely chronic end-states fell by 50% and that

<table>
<thead>
<tr>
<th></th>
<th>1972</th>
<th>1941*</th>
<th>Ratio 1972/1941</th>
</tr>
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<tbody>
<tr>
<td>Severe chronic</td>
<td>12</td>
<td>15</td>
<td>0.50</td>
</tr>
<tr>
<td>Moderately severe chronic</td>
<td>16</td>
<td>17</td>
<td>0.85</td>
</tr>
<tr>
<td>Mild chronic</td>
<td>31</td>
<td>38</td>
<td>1.00</td>
</tr>
<tr>
<td>Recovery</td>
<td>25</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100</td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Stabilized end-state among first admissions and their affected siblings, 1972 and 1941


* Relatives of 1941 probands hospitalized at least once.
of moderately severe end-states by 15%, while the proportion of patients with 'mildly chronic' outcomes almost doubled. The disorder appears to have become more benign first in the severely chronic, then in the less severe cases, but it may be noted that the rate of full remissions is unchanged. A similar observation is reported by Achaté (1967), who compared patients first admitted in 1950 with first admissions in 1960. The percentage of cases attaining at least a social remission was 31% for the 1950 series and rose to 51% (an increase of nearly two-thirds) for the 1960 series.

Studies of the outcome of schizophrenia in cross-sectional studies over the last century indicate that about one third of the patients either recovered or improved, one third deteriorated, and one third remained unchanged (Staudt & Zubin, 1957). Bleuler's longitudinal enquiry modifies this expectation, and Bleuler himself summarises the benign character of the change in schizophrenia today by pointing out that between two-thirds and three-quarters of the patients progressed towards more favourable outcomes, while in only one quarter to one third was the outcome unfavourable. He surmises that psychiatrists of previous generations assumed that if all schizophrenics were to outlive the natural course of the disorder they would present a state of deterioration. Today, he asserts, the survivors would all eventually recover. Bleuler attributes the negative outlook on the part of his predecessors to the fact that they observed only the patients with poor outcome, who either never left the clinic or who returned to it soon after release. His father and his colleagues did not see again these patients who improved and recovered, and failed to consider them in evaluating the overall outcome. This bias has been difficult to overcome.

Bleuler is not alone in pointing to the more benign nature of schizophrenia today. Ciompi (1980) followed up a cohort of 289 patients in Lausanne, and a similar long-term study was conducted by Huber et al. (1980) in Bonn on 502 schizophrenics over an average period of 21-4 years. The results of the studies by Bleuler, Ciompi and Huber, all of them closely comparable in methodology and design, are shown in Fig. 1.

Bleuler's and Ciompi's outcome data in terms of end-state were identical for recoveries (30% and 29.7%, respectively) and similar for severe chronic end-states (15% and 19.7%, respectively). Huber did not report the end-state categories used by Bleuler & Ciompi, and reported only favourable (recovered and mild chronic) and unfavourable (moderately severe and severe) categories of outcome. Overall, some 56% of the first admissions in these studies enjoyed a favourable outcome. Even more striking is the similarity between the proportions of patients developing a severely chronic end-state in the Burghölzi and the Lausanne Hospitals: 15% for the former and 19.7% for the latter.

Based on a review of 10 long-term follow-up studies of schizophrenics, Shapiro & Shader (1979) also report a trend towards a more benign outcome. Beginning with only 2-5% complete recoveries in the first part of the twentieth century, the rate gradually rose to a median of about 13% before 1960 and to 30% after 1960. Wing (1966) in England similarly found that three-quarters of the first-admission schizophrenics whom he followed up showed no or only a mild disability after 5 years. This relatively benign outcome is not limited to first admissions. Among the patients with poorest prognosis, those whom Bleuler would have classified as schizophrenics with moderate or severe chronicity, 53% showed a favourable outcome, i.e. a mild or no disability. In the United States Paul
& Lentz (1977) demonstrated that severely chronic patients, rejected by a rehabilitation project because of their apparently hopeless outlook, improved remarkably with psycho-social therapies. Ninety-one per cent stayed continuously in the community; only 5% required rehospitalization in a 2-year follow-up; and 10% were able to live independently out of hospital.

In trying to account for these findings it is necessary to consider advances that have been made in the spheres of description, research and treatment.

ADVANCES IN TECHNIQUES OF DESCRIPTIVE PSYCHIATRY

The development of standardized, reliable tools for administering and recording interviews and applying diagnostic criteria (APA, 1980; Wing et al. 1974) has constituted a major step forward. Despite the elegant clinical systems of classification advanced by the founders of modern psychiatry, diagnosis became a topic of little more than academic interest to most American psychiatrists during the first half of this century, principally because it did not lead to practical applications. Only with the development of new forms of therapy from the 1940s onwards was there a revival of interest in diagnosis. Improved descriptions of clinical states became necessary to assess changes due to drug treatment and other types of therapeutic intervention. An important stimulus to this awareness was provided by the binational US–UK Diagnostic Project (Cooper et al. 1972) and the International Pilot Study of Schizophrenia (WHO, 1979), both of which depended on the construction of reliable interviewing instruments and of standardized criteria for diagnosis.

As Wing (1978a) has pointed out, the two international studies suggested that the clinical symptoms associated with schizophrenia can be categorized into positive and negative symptoms, in accordance with Hughlings Jackson's (1931) classical distinction. According to Jackson:

Disease is said to 'cause' the symptoms of insanity. I submit that disease only produces negative mental symptoms answering to the dissolution, and that all elaborate positive mental symptoms (illusions, hallucinations, delusions, and extravagant conduct) are the outcome of activity of nervous elements untouched by any pathological process; that they arise during activity on the lower level of evolution remaining.

The positive schizophrenic syndrome consists of delusions, hallucinations, incoherent speech and other florid and productive symptoms. These tend to respond to treatment, and to recede with the end of the stressful period induced by the episode. The negative syndrome, sometimes regarded as the clinical poverty syndrome, is characterized by emotional apathy, slowness of thought and movement, under-activity, lack of drive, poverty of speech, and social withdrawal. These symptoms more often characterize the chronic state and are not generally affected by the treatment (Strauss et al. 1974). Crow (1980, 1981) has postulated that the two syndromes (the positive or Type I, and the negative or Type II) seem to be related to two underlying processes associated with different prognoses: a diagnosis of the Type I syndrome predicts potential response to neuroleptic drugs, while the Type II syndrome carries a poor long-term outcome irrespective of drug treatment.

On the basis of CT scan evidence the Type II syndrome also seems to be associated in some chronic patients with increased ventricular size. A relation between enlarged ventricular size and schizophrenia has been claimed by several workers, including Johnstone et al. (1976) and Weinberger et al. (1979). Weinberger et al. (1980) also found that enlarged ventricular size was associated with a poor premorbid personality and they supported Crow's observation that enlargement of the ventricles was associated with poor response to treatment. Andreasen et al. (1982) have provided confirmatory evidence for the relationship between ventricular size and negative symptoms and have also reported that schizophrenics with smaller ventricles are characterized by positive symptoms.

These workers (Andreasen, 1982; Andreasen & Olsen, 1982) have also developed a scale for negative symptoms and have proposed a classification of schizophrenia into three subtypes: positive, negative and mixed, according to the prominence of negative and positive symptoms in the patient. Strauss et al. (1974) suggest the inclusion of a different category, namely disorders in relating. These conditions seem to be of ontogenic origin and may reflect such deviations as absence of intimacy in schizo-

One caveat with regard to descriptive criteria is required. Structured interview techniques and research diagnostic criteria have become so widespread that there is a danger of standardizing diagnosis prematurely, thereby preventing further growth and development in this area. Though standardization imposes uniformity and comparability, it can also lead to a rigidity which may rule out innovative findings and further developments. Thus, it is essential to return repeatedly to careful clinical observations if advances are to be made in this sphere.

ADVANCES IN THE AETIOLOGICAL UNDERSTANDING OF SCHIZOPHRENIA

The aetiology of schizophrenia remains shrouded in mystery, even though the last few decades have seen many attempts to penetrate the veil. At least six scientific models of aetiology have been identified (Zubin, 1972), ranging from the molecular biology type exemplified by the genetic model, to the field theory type exemplified by the ecological model. Between these two poles lie the internal environment and neurophysiological-anatomical models, which lean towards the genetic pole, and the learning theory and developmental models, which lean towards the ecological pole.

The concept of vulnerability

In parallel with these two broad types of models a dichotomy of opinion has arisen. On the one side are those who regard schizophrenia as an essentially biological disorder in which psychosocial factors (ecological, developmental, and learning) play a minor or even trivial role; on the other side are those who regard it as an essentially psychosocial disorder in which biological factors play a minor role. The vulnerability model (Zubin & Spring, 1977; Zubin & Steinhauer, 1981) has been introduced as a compromise between these two extreme views. It assumes that schizophrenia occurs only in a vulnerable individual, but that vulnerability requires a life event stressor (either exogenous or endogenous) to trigger an episode. Perhaps the greatest difference between the two extremes and the middle ground of vulnerability is the attitude towards recidivism. According to the biological and to some extent the psychosocial approaches, the mental health of a vulnerable person is merely a temporary respite in the course of continuing disorder. According to the vulnerability model, on the other hand, the episode may be a temporary interruption of an essentially healthy life.

The vulnerability model also differs from the others with regard to the assumed causes of recidivism. Though widely employed for the purpose, rehospitalization is not a satisfactory index of relapse since every new episode does not necessarily lead to hospitalization and not every admission to hospital is due to the development of a new episode. However, until objective indicators for the initiation and the termination of an episode become available, rehospitalization remains an indispensable measure. According to the biological model, recidivism is brought about by either an untimely release from hospital which exacerbates an incompletely resolved episode of illness, or by the failure of chemotherapy, or by non-compliance with the post-release drug regime, or by the natural oscillations in the course of the disorder. According to the psychosocial model, recidivism may be due to untimely release, as in the case of the biological model, or to the return of the patient to a noxious premorbid environment, or to a failure to transfer the improved behaviour learned in the hospital to life outside hospital, or to a failure in vocational readjustment, or to the demanding nature of the environment and its intrusive qualities.

According to the vulnerability model, by contrast, recidivism is due to the impact of life event stressors which elicit a new episode of illness unless some moderating variables intervene to prevent it. If it were not for the life event stressors, no such episode would occur. The vulnerability hypothesis challenges the possibility of intrinsic undulation as a cause unless recurring life event stressors bring it about. It also denies non-compliance with treatment as a cause, since new episodes of illness can develop in compliant patients. Families with a high degree of expressed emotion towards the former patient may also be a source of stress and thus engender recidivism (Leff & Vaughn, 1981).

The general scope of the vulnerability model is shown in Fig. 2. To cause an episode of illness a trigger, in the form of an exogenous or
endogenous life event stressor, must exceed the tolerance threshold. Once this threshold has been breached, a crisis develops which, in the vulnerable individual, develops into an episode of illness. The episode continues until the stress and its aftermath dissipate, though vulnerability to future episodes persists. When the episode terminates, the patient returns to his premorbid level of adjustment.

The episodic nature of schizophrenia

The vulnerability hypothesis thus assumes that the concept of schizophrenia does not imply a chronic disorder so much as a permanent vulnerability to develop the disorder. Viewed in this way, schizophrenia becomes an episodic illness in the same sense as depression, epilepsy, or allergy. Since there are no data on episodes that do not lead to hospitalization, rehospitalization data may be used to determine the intermittent character of such episodes.

The data for the frequency of rehospitalization in Bleuler’s (1978) study are shown in Table 2.

About one third of the first admissions had only one hospitalization, from which they were released and never rehospitalized. In Ciompi and Müller's follow-up study nearly one half of the patients had one hospitalization; only 22% remained hospitalized after one or two releases, while the remainder oscillated between the hospital and the community.

That the majority of cases (about 80%) had either one hospitalization followed by release, or multiple hospitalizations followed by release, lends credence to the episodic nature of schizophrenia. While rehospitalization has been viewed historically as indicating an exacerbation of an underlying illness, our view is that rehospitalization is often related to the onset of a new episode following a period of recovery and freedom from symptomatology. Ciompi (1980) reports the duration of hospitalization as less than one year for 47% of his follow-up group, with two-thirds being hospitalized for no more than 6 months; only one quarter were hospitalized for more than 20 years, and a large portion of this group might well have been the product of iatrogenic factors. Since the samples consisted equally of acute and chronic patients, these results cannot be attributed to a skewed distribution of admissions. Further support for the episodic nature of schizophrenia comes from Bleuler’s report that the proportions of cases in each outcome or end-state group (Table 1) remain constant even though the patients vacillated from one status to another. This constancy for the group, but inconstancy for the individual, is readily explained by the vulnerability hypothesis, in terms of which the proportions remain the same for the group because the waxing and waning of episodes in the individual results in a dynamic equilibrium for the group as

Table 2. Frequency of hospitalization among first admissions during the total follow-up period

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>%</th>
</tr>
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<tbody>
<tr>
<td>Never released</td>
<td>6</td>
<td>8.8</td>
</tr>
<tr>
<td>Released and rehospitalized and never released again</td>
<td>9</td>
<td>13.2</td>
</tr>
<tr>
<td>Rehospitalized more than once followed by release</td>
<td>32</td>
<td>47.1</td>
</tr>
<tr>
<td>Released and never rehospitalized</td>
<td>21</td>
<td>30.9</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Based on Bleuler (1978), table 4.11, p. 236.
a whole: as one patient enters an episode, another emerges from an episode, like ions in a solution.

An analysis of Bleuler’s data of the different types of courses associated with the schizophrenic disorders lends more evidence in favour of the episodic hypothesis. These data indicate that only one third of the first admissions in the 1972 study exhibited a simple undeviating course, while about two-thirds (64%) followed an undulating course, with only 3% tending towards a severe outcome, 22% towards a mild outcome, and 39% towards a recovery. Thus the illnesses of about two-thirds of these patients were episodic, most of them with a relatively favourable outcome.

Why the persistence of chronic schizophrenics?
If it is assumed that schizophrenia is essentially an episodic disorder, how can one explain the persistence of so many chronic schizophrenics in hospitals and clinics? There are several possible explanations. First, chronic patients accumulate over time, even though proportionately their number may not be very large. Many of them - nearly 30% according to Bleuler - remain in hospital because they have nowhere else to go. Many suffer from the result of iatrogenic and nosocomial influences which keep them in hospital because the social behaviour learnt in the hospital or at home during the illness impairs their extra-mural adjustment. There is also a group of patients who were never able to make a satisfactory adjustment to society: when they return to their premorbid level, they remain ‘pseudo-patients’ even though the episode has terminated. In addition, there may be a small number, not more than 6–10% according to Bleuler, who are chronically ill or who have episodes in rapid succession (see Spring & Coons, 1982, for other explanations of chronicity).

The problem presented by the supposedly chronic patients is further complicated by recidivism. Patients who fall into this category were rarely released in the past but they now constitute the bulk of the ‘chronics’ who move in and out of hospitals. In terms of the vulnerability hypothesis, they include two types: those who were released before the end of an episode of illness and suffered a relapse, and those whose illness terminated but who then developed a new episode. It is not always easy to distinguish between a relapse and a new episode, but new episodes might be delineated if, during an interval of at least 3 months, the individual fails to meet pre-determined criteria of illness – for example, the Research Diagnostic Criteria (RDC) (Spitzer et al. 1975) - at one point in time but does so subsequently.

The assessment of vulnerability
The vulnerability model consists of the following parameters: (1) the degree or measure of vulnerability, (2) the impact of the life event trigger, (3) the development of a crisis, (4) the development of the episode, and (5) the disappearance or subsiding of the episode.

Efforts to measure vulnerability are at an early stage of development. One aim is directed at finding markers to identify vulnerable individuals (Spring & Zubin, 1978; Zubin & Steinhauer, 1981). With regard to life event triggers, schedules such as the Social Readjustment Rating Scale developed by Holmes & Rahe (1967) are not always useful for research in schizophrenia. This approach must be revised in order to distinguish life events that are stressful from those that do not have such an impact. Since one of the criteria for the impact of life events is the amount of change it induces and the degree of adaptation it demands, it might be profitable to monitor the individual’s daily life to assess changes in daily activity induced by particular events. The current confusion will be clarified when the role of moderating variables is better understood. Such variables are clearly significant, for two equally vulnerable individuals who have undergone equally stressful life events may not both develop episodes. In such circumstances, it is postulated that moderating variables either cushion the impact of the stressor or permit it to operate unimpeded.

Even with the most advanced model of vulnerability, that based on genetics, it cannot be expected that all individuals who carry the genotype will develop the phenotype, since the penetrance of the gene or genes is never 100%. According to some calculations, only 26% of schizophrenic genotypes become phenotypes (Zubin & Steinhauer, 1981). What prevents the remaining three-quarters from developing overt illness? While most vulnerable individuals will experience a life event stressor sufficient to trigger an episode, the moderating variables may
intervene to prevent the phenotypic development even when a potential triggering life event stressor strikes.

A variety of factors modulate the impact of life events. First, an individual's ecological status may either cushion or exacerbate the impact of a triggering event, depending on the structure and/or the degree of support provided by the environment. Secondly, personality-structure can be vital. Each of these factors will be discussed in turn.

Ecological influences fall into two groups: those which are social and come under the general headings of social status, network or support; and those which are physical and characterize such aspects of the physical environment as type of housing, crowding, noise and pollution. The physical-social boundary is often not clear-cut and is used here only for operational purposes.

SOCIAL FACTORS

Studies of the role that social networks and supports play in psychopathology are relatively new, though the social aberrations that characterize psychopathology form the primary basis for the recognition of mental disorder. Social workers, clinical psychologists, and psychiatrists have long recognized the importance of the social network provided by family and friends, but until recently formal studies were few in number. The difficulty of most such studies is the lack of a baseline in the general population, for most available information has been obtained from deviant populations.

There is, however, growing evidence in favour of the importance of social networks and supports in protecting individuals from psychiatric disorders, from physical illness, and even from death. In a general population study of social relationships and neurosis, Henderson et al. (1980) associate a lack of social bonds and the perceived benefits that would have been supplied by these bonds with an increased prevalence of neurotic disorders, regardless of exposure to adverse experiences during the preceding 12 months. Brown et al. (1972, 1975) have provided evidence on the protective role played by a confidant or intimate companion in preventing psychiatric disorders in urban women. Women who experienced a severe problem and had a confidant (i.e. husband, boyfriend, female companion) were significantly less likely to become mentally ill than those without such a companion. The lack of this person in itself does not provoke the disorder, for the possession of a confidant did not discriminate 'cases' from 'non-cases' among women who had experienced no severe events or difficulties.

Cassel (1976) has presented data from several studies indicating that social networks and supports play a role in preventing various physical illnesses. Survival itself, as evidenced by mortality statistics in the Alameda County 9-year follow-up study (Berkman & Syme, 1979), is associated with the quality and extent of the social network. Even after controlling for such determinants of mortality as smoking, obesity, alcohol consumption, physical activity and socio-economic status, supportive social networks were still related to lower mortality rates.

According to Hammer et al. (1978), the following factors are associated with a higher risk of schizophrenia: migration, social marginality, lack of acculturation, underprivileged ethnic origin, and social isolation. These factors are important in both the development of social networks and their disruption. Schizophrenics tend to have smaller social networks, and those with low density contacts (fewer interactions between friends) and low social networks tend to be at a higher risk for rehospitalization (Sokolovsky et al. 1978). In one ecological study people who were not living alone and who had a history of mental hospitalization were rehospitalized at a higher rate if they lived with many other individuals (Magaziner, 1980). It has been suggested (Wing, 1978b) that 'overstimulation' (a logical result of having many people in the same household) can lead to florid symptoms in vulnerable individuals who are unable to withdraw from the setting. The larger number of people in the household also may be associated with more expressed emotion in the home which, in turn, is related to relapse among schizophrenics (Wing, 1978b; Leff & Vaughn, 1981). One of the most important parameters with regard to outcome is the character of the family to which the patient returns, and the degree of family concern for the patient is often crucial. Data on family concern based on Bleuler's (1978) follow-up study are shown in Table 3.
Table 3. Family concern for patient (probands) by end-state

<table>
<thead>
<tr>
<th>End-state</th>
<th>Devotedly cared for</th>
<th>Rejected</th>
<th>Total</th>
<th>Devotedly cared for (%)</th>
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<tbody>
<tr>
<td>Recovery</td>
<td>10</td>
<td>1</td>
<td>11</td>
<td>90.0</td>
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<tr>
<td>Mild and moderately severe</td>
<td>27</td>
<td>17</td>
<td>44</td>
<td>61.4</td>
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<tr>
<td>Severe</td>
<td>17</td>
<td>7</td>
<td>24</td>
<td>70.0</td>
</tr>
<tr>
<td>Total</td>
<td>54</td>
<td>25</td>
<td>79</td>
<td>68.3</td>
</tr>
</tbody>
</table>

Based on Bleuler (1978), table 4.28, p. 287.

The 'devotedly cared for' seem to have an edge on the 'rejected' with regard to outcome. However, some of the 'rejected' may have been more severely ill and therefore might be expected to have a worse prognosis. Although the overall value of chi-square for the entire table is not statistically significant, the recovery rate alone shows a 10:1 advantage for the 'devotedly cared for' (χ² for the row = 4.3, P < 0.05). Some of the 'devotedly cared for' may have received too much care, which, according to Wing (1980), would militate against improvement. Leff (1978) has shown that patients exposed to a hostile environment relapse more frequently than those who returned to more favourable home situations. As Brown et al. (1972) have observed, the amount of expressed emotion in a patient's key relative is associated with the extent to which the relative, especially a parent, is isolated. Those relatives with fewer outside contacts had higher expressed emotion scores than those with more frequent contacts of this type. This relationship between expressed emotion and a person's association with an outside social network provides further evidence of the need to investigate the social networks of both schizophrenics and those related closely to them (Hammer et al. 1978).

The social network or support system therefore seems to moderate the development of schizophrenia. In the vulnerable individual, a well-developed social network may absorb the impact of a life event stressor (triggering event) and prevent an episode, while a poorly developed network may offer no such protection. However, in tracing the relationship between social networks, life event stressors, and episodes in the vulnerable individual, the role of social networks may be difficult to predict as it may have two contrary effects. An individual with a well-developed social network is likely to experience more life events during typical daily activities, since the number of possible interactions is a function of the size or interrelationship of the network itself. On the one hand, this tends to increase the risk of an episode in a vulnerable individual. On the other hand, a well-developed social network tends to cushion the impact of life event stressors, and thus tends to reduce the risk of developing an episode of illness. How may these two contrary effects be reconciled?

When the life event stressor is not a direct result of interactions with the support network (e.g., traffic accident, physical illness), the network may function in the expected manner, ameliorating or intensifying the effects of the event. Only when the stressor disrupts the social network directly (e.g., death of a friend, argument with a close relative) is the role of the network as an intervening system likely to be called into question since it may represent a triggering, rather than a moderating, factor. It is at this juncture that one must consider other aspects of the network or other interactive dimensions of the vulnerability model, such as ecological status or premorbid personality. Any of these may further modify either the effect of the event or the structure of the social support network.

Social status also may be associated with the character of the social network, perhaps explaining why minority status and low social class, for example, are related to an increased risk of schizophrenia. Minority status may exacerbate a triggering event since there may be few people who can be personally helpful and with whom the individual can identify and interact in a crisis situation. Mintz & Schwartz (1964) have reported, for example, that Italians are less likely to be hospitalized for schizophrenia if they are living in their own ethnically homogeneous neighbourhood, which may act as a prophylactic against deviant behaviour. Wechsler & Pugh (1967) showed that mental hospital admissions came disproportionately from communities in which the probands had few age peers. The work of Rosow (1967) and Rosenberg (1970) on neighbourhood age-composition helps to demonstrate that friendship and interaction are increased in settings with many age peers. Furthermore, Kreisman (1970) found that
schizophrenics had fewer intimate friendships during their premorbid adolescence than did a matched group of controls. The absence of individuals who are of the same age or ethnic background allocates the subject to a minority status which, in turn, may prevent the formation of intimate relationships. Other characteristics such as race and occupation may operate in a similar manner.

Social class also may moderate the stress induced by a triggering life event, though the process by which it operates is complex. Kohn (1973) has hypothesized that 'the constricted conditions of life experienced by people in a lower social class position fosters conceptions of social reality so limited and so rigid as to impair people's ability to deal resourcefully with the problematic and stressful'. Other research workers have also suggested that personal resourcefulness - for example, education - and external resources - for example, family, money - are likely to be most limited in the lowest social classes (Myers et al. 1974; Dohrenwend & Dohrenwend, 1969). When confronted with a triggering event, the vulnerable individual in this class may be at a disadvantage and may be more likely to experience an episode of illness than an equally vulnerable individual in a higher socio-economic class.

PHYSICAL ECOLOGICAL STATUS
Physical ecological factors may also modulate a triggering event, either lessening or enhancing the risk of an episode of illness. Factors such as weather, season, chemical pollution, noise, type of housing, and living density are all regarded as stressors which may increase the chance of inducing psychopathology in vulnerable individuals. However, while the role of social network as a moderating variable has been studied extensively, that of the physical ecological status is only beginning to attract attention.

Proshansky et al. (1978) refer to a person's 'place identity' as being closely related to self-identity with respect to physical setting and including 'feelings, beliefs, behavior tendencies and preferences that express the person's needs for understanding, privacy, control, competence, and security in the physical environment' (p. 10). Accordingly, 'place identity' affects the way in which the individual organizes and interprets the physical world and uses this physical setting in daily affairs to combat stressful experiences. In consequence, following a natural disaster or a move from one city to another, episodes of mental disorder (including schizophrenic episodes) are more likely, even after adjustment has been made to the initial stresses imposed by these changes. Such events may inhibit the support function usually served by living in a familiar setting where resources, services and social relationships are well established.

Chemical pollution has been related to a decline in brain function (Feldman et al. 1980; Brown et al. 1981). In low doses over long periods it may, in turn, limit an individual's ability to cope with stressful events. Noise, in addition to being a stressor, may interfere with the ability to cope with other stressful events, especially if reducing or eliminating this noise is beyond one's control (United Nations, 1980; Glass et al. 1977).

Housing characteristics have also been linked to morbidity behaviour. To the extent that housing facilitates social interaction and control over environmental stimuli then it also may act as a resource or a deficit in the face of a triggering event. Living on the top floor of a high-rise building can, for example, limit access to the street and, in the absence of friends in nearby units, may restrict social interaction and hence exacerbate the impact of a triggering event on a person needing social contact. The same type of housing may be less of a problem for those desiring solitude. Ultimately, as Aitman (1973) has argued, people must be able to control their own interaction and privacy; a housing situation that maximizes this control will probably be most effective in counteracting a stressful event.

The living density of the dwelling or neighbourhood may also serve as an asset or a deficit in that it implies the presence of other people in a finite space. Fifty years of research on the relationship between living density and psychopathology have produced conflicting results (Magaziner, 1980). While high density may lead to pathological behaviour it need not do so since, for example, a dense household can contain many people who may help to buffer a crisis. Alternatively, such a situation may prevent vulnerable individuals from dealing with the crisis by means of their own resources, thus becoming dependent on others in times of stress.
A crowded neighbourhood may signify many nearby potential contacts, goods and services which may serve a supportive function in a time of crisis. If, however, interaction and privacy are difficult to regulate, the high density in the community can worsen a crisis situation.

The person who lives alone may be at an advantage or a disadvantage in a life crisis. Psychiatric disorders are thought to be more common among those living by themselves. Hare (1956a), for example, found in an ecological study that hospitalization for schizophrenia was disproportionately represented in areas of Bristol with a high proportion of solitary residents, regardless of the economic status of the community. Similar ecological relationships have been reported elsewhere (Galle et al. 1972). At the individual level the relationship between stress and mental symptoms appears to be stronger for those living alone than for those living with others (Eaton, 1978). Summarizing the literature, Gove & Hughes (1980) conclude that a solitary life leads to a pathological state because isolates lack the social interactions, supports and networks that give life meaning and value. They also lack the input and structure which involve individuals in daily affairs. This, in turn, may lead to withdrawal and magnification of personal problems.

It is also possible to explain the relationship between living alone and schizophrenia as resulting from a social selection process. Individuals who are vulnerable to schizophrenia may choose to live by themselves. In a second study Hare (1956b) attempted to resolve this question by looking more closely at the hospitalized schizophrenics in his population. Like Gerard & Houston (1953), he attributed the relationship between living alone and schizophrenia to those schizophrenics who were not living with their families—i.e., those who were living alone or with unrelated others. Some of these subjects moved to their isolated environments after displaying symptoms of schizophrenia, while others became ill after living in areas with a high proportion of residents living alone. Thus, Hare concluded that, while some individuals selected their environment due to their illness, for others the environment could have played a triggering, if not a causative, role in subsequent breakdowns.

A later ecological analysis of first admissions to mental hospitals in Chicago during 1960–1 suggests that the relationship between solitary living and psychopathology involves other ecological issues (Magaziner, 1980). After controlling for socioeconomic status and racial homogeneity, Magaziner found that a disproportionate number of admissions came from areas with both a high proportion of residents living alone and a community of low population density. The social interaction, goods and services implied by high population density may counteract the potential isolation associated with living alone. The vulnerable individual may even prefer living in a densely settled neighbourhood which provides the opportunity to be alone or with others at his or her own discretion during a stressful period. The person living alone in a low density community might not have this option and the resulting isolation could exacerbate a crisis. Thus, control over interaction and privacy may provide a useful framework for understanding how solitude, even in a high density area, may moderate the effect of a triggering event in a vulnerable individual. Apparently, both the social and physical aspects of the environment are implicated in the process by which a vulnerable individual may develop a schizophrenic episode.

The discussion of the social and physical aspects of the ecological position has so far been limited to their role as moderating variables in reducing or exacerbating the impact of the triggering stressors in eliciting an episode of illness in a vulnerable person. Whether or not these forces can also serve directly as formative factors in producing vulnerability is an open question. The difficulties facing the investigator in answering this question arise from the possibility that the absence of a good social network, to take one example, may be prodromal rather than etiological.

It has been observed that pre-adolescents with a lack of intimacy in their friendship patterns tend to have a higher risk of developing schizophrenia later in life (Kreisman, 1970). Such individuals do not benefit from the usual interaction with their peers and may develop aberrant ideas and even delusions that are usually eliminated through the experience of a social network. Whether deprivation of such experience in itself can cause an episode of illness, or merely trigger it in an already vulnerable individual, or be no more than a
prodromal sign, is an issue which can be resolved only by empirical research.

The social network may be viewed as playing the same role in the ecological model that consanguinity plays in the genetic model. Just as the blood-stream sustains the physical body, so the social network sustains the social organism. Once its parameters are defined, the social network might provide a vulnerability marker for the ecological model.

Similarly, the physical, as opposed to the social, aspects of the individual’s ecological status may play a formative role in the development of vulnerability, in addition to its role as a moderating influence on the triggering stressor. Individuals who grow up in limited, disorganized environments where the opportunities for socialization, learning and development are meagre and where nutrition and other physical supports are minimal, may become more vulnerable because of these noxious experiences. Through early exposure, noxious influences are gradually absorbed and encoded in the developing individual who is then more likely to become ill when a triggering stressor occurs later in life.

PREMORBID PERSONALITY

As with social networks, there are few studies of the general population that can serve as a baseline for evaluating premorbid personality attributes in schizophrenic patients. Consequently, most data on the role of personality are based only on psychiatric patients.

The early studies of premorbid personality were prognostic, being concerned not so much with the prediction of illness-episodes as with the prediction of the outcome of such episodes. When it became clear that outcome depended so heavily on factors other than the premorbid personality, research in this area lagged. Interest in the field has been re-awakened because of the possible role played by premorbid personality in predicting illness, both in prospective studies of high risk individuals and in the retrospective studies of records of individuals who developed single or recurrent schizophrenic episodes.

Three possible relationships between premorbid personality and the psychopathology of schizophrenia have been postulated (Zubin, 1965). First, the premorbid personality and the disorder could be identical, with the personality developing along schizophrenic lines. Secondly, the premorbid personality could be orthogonal to schizophrenia, making it impossible to predict an episode of schizophrenia based on a knowledge of premorbid personality. A third, interactional view takes the middle ground between complete identity and complete independence. These three approaches might be seen as deriving from Freud, Kraepelin and Adolf Meyer respectively. The literature contains no decisive evidence to favour any of these three possibilities, but studies by Essen-Möller et al. (1956) and by Pollin & Stabenau (1968) cast some light on their relative tenability, as do recent investigations of high risk children. Essen-Möller et al. (1956) assessed the personality, demographic characteristics and mental status of the 2550 inhabitants in a rural area, designated as Lundby, in the vicinity of the city of Lund in Southern Sweden in 1947. Their focus was on the predictive value of their 1947 personality data in relation to the occurrence of an episode of mental disorder during the succeeding decade. A follow-up of this population was undertaken 10 years later by Hagnell (1966). He found that the system of normal variants of personality developed by Sjöbring (1963) did not relate to the incidence of mental disorders during the follow-up, but that Sjöbring’s pathological variants of personality were associated with the incidence of episodes.

According to Sjöbring, the normal variants of personality may be considered as variations within the normal range, similar to the variations shown by the distribution of intelligence or stature. In fact, one of the components, capacity, was regarded as essentially a measure of intelligence. Sjöbring designated the pathological variants of personality as ‘lesional’. Essen-Möller & Hagnell (1975, p. 149) state:

Lesional factors, in the sense of Sjöbring, are conceived as obstructing or distorting natural development activity. They may be transient or permanent, and may originate from abnormal genetic predisposition or from exogenous influences of the well-known kinds. With the majority of mild lesional factors, we shall be restricted to diagnosing their presence from minute mental traits, in fact extreme dilutions of the generally accepted brain syndromes, like tension, slackness, rigidity, hypersensitivity or insensitivity, lachrymosity, impaired alertness, memory or judgement... Indeed, the recent interest in 'minimal brain damage syndromes', as diagnosed with the aid of
prospective studies and of laboratory findings, is entirely in this line; Sjöbring diagnosed these signs and symptoms by careful clinical observation alone, his ideas on normal developmental variation and his general theoretical concepts helping him to their recognition.

These workers used four groups of confidence (levels): evident (e), probable (p), conceivable (c) and no lesion (n). 'Evident' included traits, severe or mild, that most psychiatrists would at that time have agreed upon as lesionlal; 'probable' incorporated traits that would have been called lesionlal by Sjöbring but presumably not by the majority of psychiatrists; 'conceivable' covered traits that were barely regarded as lesionlal by the examiner but could not quite be excluded from suspicion.

A special analysis was made by Hagnell of those inhabitants of Lundby who were free of any mental disorder at the time of the initial survey in 1947 and who were followed up during the succeeding decade. These individuals ranged from those in whom personality deviation was quite evident to those with a normal personality. Only 5 individuals succumbed to an episode of schizophrenia during the follow-up. So small a sample was insufficient for the investigation of the relation between premorbid personality and the development of schizophrenia. In order to establish such an association it was necessary to establish the distribution of premorbid personality in a large sample of schizophrenics. Such data are available in Bleuler's (1978) follow-up study. If we make the assumption that the distribution of personality types in Bleuler's data is representative of the distribution for schizophrenics in general, we can apply Bleuler's data to the expected number of schizophrenics who are likely to emerge in the Lundby residents in the future.

Unfortunately, it is difficult to establish the precise equivalence between the personality categories used by Essen-Möller and those used by Bleuler (see Appendix). If, nonetheless, we accept the suggested equivalence as valid, it may be concluded that those members of the general population of Lundby who fall into the category of severest deviation (evident personality deviation) have a nine-fold risk (9.36%) over the risk in the general population (1%) for developing a schizophrenic episode. By contrast, the individual risk in the other two categories of normal or mild deviation is only 0.7%.

What conclusion can be drawn from these data? The group with evident personality deviation seems to be most prone to develop schizophrenic episodes, but about 90% of the members of this personality category in the general population showed no evidence of schizophrenia. The other personality categories exhibit rates similar to those of the general population. Thus, although there may be a relationship between premorbid personality and the development of a schizophrenic episode, the small samples in most studies are not amenable to statistical evaluation. Vulnerability theorists are interested in this relationship because, according to their model, all episodes should be time-limited and followed by a return to the premorbid level. If these levels come within the normal range, the patient should return to a state of restitutio ad integrum. However, if the premorbid state is abnormal, it may become difficult to recognize that the episode has ended because of the poor level of adjustment to which the patient returns. Such individuals are usually regarded erroneously as chronically ill and their postmorbid deviations are falsely attributed to the persistence of their schizophrenic episode.

Table 6 (see Appendix) shows that individuals with abnormal personality traits make up nearly 3.7% of the general population; about 9% of these individuals develop schizophrenic episodes. Even before their overt illnesses it is likely that they were unable to cope with life's demands. The episodes do not improve their capacity to cope with their environmental difficulties, and it is easy to understand why they are mistakenly regarded as still ill even though the illness has run its course. They have, in fact, returned to their premorbid level of dysfunction and, even after the illness, their disorders of personality may predispose them to a greater possibility of generating noxious life events and not only to being harmed by them. This may, in turn, increase the probability of developing new episodes of illness more rapidly and repeatedly and thus reinforce the impression of persistent chronicity.

If educational and rehabilitative methods can improve the coping ability of the most severe personality deviants while they are in the hospital or clinic, such treatment might benefit not only the patients but also the large majority of individuals with comparable personalities who do not become ill. The fact that nearly one tenth
of these people are hospitalized or treated in clinics poses a challenge for rehabilitation and prevention.

Bleuler's data provide no support for the view that individuals who develop schizophrenic episodes differ from the general population in respect of their premorbid intelligence, occupational level, or sexual behaviour, though they tend to exhibit less sexual activity in general. As for the early rearing of the schizophrenic, Bleuler reports a greater frequency of poor home surrounding and adverse childhood conditions among individuals with premorbid schizoid personalities than among those who do not develop aberrant personalities. There is, however, no corresponding increase in the rate of schizophrenia.

The second major study bearing on the relationship between personality and the development of a schizophrenic episode is that of Pollin et al. (1966), who examined the premorbid personalities of pairs of discordant monozygotic twins among veterans. The following personality traits were characteristic of the affected twins: neurotic as a child, submissive (especially towards the other twin), sensitive, serious, worrying, obedient, gentle, dependent, well-behaved, quiet, shy, stubborn, weaker, shorter. The unaffected twin excelled in intelligence, was better at school, more outgoing, livelier, the leader. Whether the characteristics of the affected twin were premorbid personality traits or prodromal features of the illness proved difficult to determine (Pollin & Stabenau, 1968).

A critical review of this study and of further developments in the investigation of monozygotic twins discordant for schizophrenia is provided by Gottesman & Shields (1976).

In prospective high risk studies the premorbid characteristics include shyness, being withdrawn and detached, and lower IQ scores (Neale & Oltmans, 1980). In one recent study still in progress (Watt et al. 1982), the social, emotional and scholastic behaviour among children born to schizophrenic parents was distinguished from that of controls by the following characteristics: greater disharmony (more unpleasant, unpopular, negativistic and maladjusted), less scholastic motivation, more emotional instability, and lower intelligence, but not significantly more introversion. Whether the personality features are independent of intelligence or of early precursors of disorder is not yet known. When intelligence was partialled out by analysis of covariance only the disharmony factor remained significant. The children of two schizophrenic parents, and those who have already demonstrated their vulnerability by developing an episode of mental disorder requiring treatment, are expected to have the greatest risk for schizophrenia. Among these two small groups of very high risk children the most dependable markers for vulnerability to schizophrenia were found to be: disharmony, emotional instability, and low intelligence.

At the present time, therefore, there are no conclusive empirical data to indicate that premorbid personality can serve as a moderating variable in a vulnerable individual who develops a schizophrenic illness following a precipitating experience. Since it is still rarely possible to identify vulnerable individuals who do not become overt schizophrenics, it is unclear whether the premorbid personality exercises cushioning or exacerbating effects. Only an expansion of multiple prospective longitudinal studies of high-risk individuals could illuminate this question.

As shown in Table 6 (see Appendix), an analysis of the distributions of premorbid personality types in Bleuler's (1978) follow-up study of schizophrenia and of similar personality types in Essen-Moller et al.'s (1956) study of the general population in Lundby suggests that about 9-4% of the patently deviant personalities, 0-7% of the mildly deviant, and 0-7% of the normals tend to develop schizophrenic episodes. If these data were to be replicated in a prospective field study, the relation between premorbid personality and the development of schizophrenia would indeed be established. By assuming, further, that the distribution of vulnerability, as distinct from illness episodes, is independent of personality, the moderating role of different personality-types on the emergence of the disorder could be assessed. Thus, vulnerable individuals possessing normal personalities would have the greatest protection against developing an episode, and those possessing deviant personalities the least.

Why should a patently deviant personality tend to raise the risk of developing a schizophrenic episode? A causal relationship seems unlikely, since no more than 9% of such people tend to develop a schizophrenic episode. The assumption is that vulnerable individuals who
The relationship between personality and outcome is shown in Table 5. The data concern the 477 individuals for whom end-states were reported. These end-state categories were subdivided into three groups defined according to Bleuler's categorization: (1) non-aberrant; (2) schizoid, and (3) schizoid pathologically and within the normal range, and with frequencies too small to be meaningful. The data are arranged to avoid cells with low frequencies (i.e., uninformative). The distributions are compared with those expected if the variables were independent (chi-square test).

Table 4. Relation of premorbid personality to outcome by type of therapy

<table>
<thead>
<tr>
<th>Premorbid personality</th>
<th>Non-specific</th>
<th>ECT</th>
<th>Insulin</th>
<th>Metrazol</th>
<th>Lobotomy</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal v. abnormal</td>
<td>+ 0</td>
<td>+ 0</td>
<td>+ 0</td>
<td>+ 0</td>
<td>+ 0</td>
<td>+ 0</td>
</tr>
<tr>
<td>Extraverted or cyclothymic v. introverted, schizoid or shut-in</td>
<td>2 0</td>
<td>9 10</td>
<td>10 0</td>
<td>2 0</td>
<td>91 9</td>
<td>99</td>
</tr>
<tr>
<td>Psychosocial development (presence v. absence of heterosexual contacts or interest)</td>
<td>2 1</td>
<td>1 0</td>
<td>2 0</td>
<td>1 0</td>
<td>6 1</td>
<td>7</td>
</tr>
<tr>
<td>Social history (good v. poor)</td>
<td>43 2</td>
<td>11 0</td>
<td>19 0</td>
<td>2 0</td>
<td>88 0</td>
<td>90</td>
</tr>
<tr>
<td>Work history (good v. poor)</td>
<td>2 0</td>
<td>1 0</td>
<td>1 0</td>
<td>2 0</td>
<td>9 0</td>
<td>10</td>
</tr>
<tr>
<td>Interest in environment (present v. absent)</td>
<td>8 0</td>
<td>31 0</td>
<td>0 0</td>
<td>2 0</td>
<td>16 0</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>127 10</td>
<td>22 0</td>
<td>31 0</td>
<td>2 0</td>
<td>12 0</td>
<td>10</td>
</tr>
</tbody>
</table>

Based on Zubin et al. (1961).

+, studies that resulted in a better outcome for the first-named characteristic of the premorbid personality (e.g., normal v. abnormal); -, studies that led to a poorer outcome; 0, indeterminate studies.

Of the 290 studies in which strong and weak therapeutic effects were related to the premorbid personality, 198 (or nearly 95%) indicated that a good premorbid outcome was associated with a good premorbid personality. In 10 of the studies there was no difference, and only one was there a reversal in this relationship. In 10 or more of the studies, the correlation was somewhat better established. The results of a 1961 survey of the relationship between personality and outcome (Zubin et al., 1961) are shown in Table 4.

While premorbid personality seems to play a modest role in initiating or preventing a schizophrenic illness, its role in shaping outcome is somewhat better established. The results of a normal primary personality mitigates the impact of childhood misery.

...
negative outcome (severe and moderately severe); their outcome is therefore less favourable than that of the first admissions shown in Table 1.

Clearly, a normal premorbid personality does not guarantee full recovery, which is not incompatible with the most abnormal premorbid personality. Other moderating variables must influence outcome, and whether personality itself should be regarded as one such variable, or as constitutionally embedded in the vulnerability of the individual, remains an unanswered question. More accurate assessments of premorbid personality are needed to indicate whether ‘weak’ personalities are associated invariably with high vulnerability, or are also found in low vulnerability subjects. The hypothetical conclusions based on the data provided by Bleuler and Essen-Möller point to the latter alternative.

In most investigations the methods for assessing premorbid personality are by means of various clinical interviews, and observer variation is considerable. Nonetheless, these essentially subjective impressions by different clinicians lead to similar conclusions, a fact which lends some credence indirectly to the method even if the available instruments are so varied.

A critical problem in assessing premorbid personality is the retrospective nature of the evaluation. New techniques have rendered the need to examine the issues more pressing. Thus Weinberger et al. (1980) have shown that, within the group of ‘chronic’ schizophrenics, patients with greater enlargement of the cerebral ventricles tended to score more poorly on assessments of premorbid personality than those with normal CT scans. Though ventricular enlargement is not specific to schizophrenia, a positive relation between these findings and poor premorbid scores lends support to the value of retrospective data in assessing premorbid personality. According to Weinberger, other anomalies also would tend to cluster in vulnerable individuals. Another recent study focuses on premorbid sexual behaviour rather than social adjustment (Leckman et al. 1981).

In the light of such findings it seems appropriate to try to establish more systematic techniques for assessing premorbid personality. The multiaxial schema of diagnosis in DSM-III includes an axis for personality that may lend itself to this purpose. If the assumptions of the vulnerability model hold, it would follow that the end of an episode of illness should result in the return to the premorbid level of function and personality structure. For this reason the second axis of DSM-IV should be extended to take account of not only the current but also the premorbid assessment of personality.

**SUMMARY**

Current investigations of schizophrenia offer many new facts, but few new theoretical frameworks for integrating these facts. This imbalance forces one to choose between tentative theory and unexplained facts. We have adopted the former stance. The integrating thread is the vulnerability hypothesis, which postulates that schizophrenia is episodic, and that it occurs in vulnerable individuals when sufficient stress catapults them into an episode.
The purpose of this paper was threefold: (1) to present the evidence for schizophrenia as a condition undergoing a benign metamorphosis, (2) to propose a vulnerability model for schizophrenia to replace the disease model, and (3) to indicate the basic parameters of this model. The model entails: (a) a vulnerability threshold for each individual, (b) life event stressors or triggers that can elicit the vulnerability, leading to an episode, and (c) moderating variables of social network, physical ecological niche, and premorbid personality that intervene either to cushion the impact of the stress and prevent an episode or to permit the episode to occur.

Evidence has been presented for the role triggering life events play in an episode. Data were also presented to show that the moderating variables of social network, physical ecological niche, and premorbid personality either prevent the episode or permit it to develop. There is considerable evidence pointing to the episodic nature of schizophrenia. Recidivism is one of the most pressing problems facing psychiatry. From the vulnerability perspective, recidivism stems not from a chronic underlying condition, but from underlying vulnerability triggered by life event stressors. If we could identify the individuals who are vulnerable to schizophrenia by discovering markers for this disorder and also identify the contingencies that elicit episodes in the vulnerable it might become possible to abort episodes, perhaps even the initial episode, through preventive methods. These could take the form of changing life style, education or other approaches that would safeguard the vulnerable person from triggering contingencies even as has happened in diabetes and other conditions. One of the problems in this approach is that by labelling someone as schizophrenic we often prevent him from resuming his place in society even when his episode is ended. It is hoped that by removing the label, the chronicity and/or recurrence of episodes which the name conjures up, and the self-fulfilling prophecy which it entails, might be eliminated.

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APPENDIX

Comparison of the personality data in the general population of Lundby with Bleuler’s data on premorbid personality (Bleuler, 1978)

Before a comparison could be made between the information from Lundby (Essen-Möller et al. 1956) and Bleuler’s data, equivalent diagnostic schemata had to be established between the personality categories used in these two studies. According to Bleuler, the ‘schizoid aberrant and otherwise aberrant within the norm’ personality refers to individuals with the milder deviation for whom the law makes no allowances, regarding them responsible. The category ‘schizoid pathological’ and ‘otherwise psychopathological’ personality refers, on the other hand, to individuals with severe personality disturbances who may escape legal responsibility for their actions if they commit a crime.

There was no difficulty in equating Bleuler’s ‘non-aberrant’ category with the Lundby ‘non-lesional’ category. The category of ‘evident’ personality deviation of the Lundby group was deemed equivalent to Bleuler’s category of ‘schizoid pathological and otherwise psychopathological’, for although the Swedish ‘lesional’ category generally contains individuals with overt illnesses as well as deviant personalities, the Lundby residents under consideration comprise only those who were free of mental disorder. By definition, they contained no one with mental illness, only personality deviants and normal individuals. Consequently, it would be reasonable to apply those Bleulerian categories which were used to classify schizophrenics in their premorbid state.

The milder deviants of the Lundby group who were classified in the category of ‘probable and conceivable psychopathology’ were equated with Bleuler’s category of ‘schizoid aberrant within the norm and otherwise aberrant within the norm’. The data are shown in Table 6. The ‘lesional’ status was obtained by combining the data for males and females in Table II (p. 150) of Essen-Möller & Hagnell (1975). Since the figures for the categories of ‘evident’ and ‘probable’ were combined in this table, they had to be separated on the basis of the more complete information provided by Hagnell (1966). The data of Essen-Möller & Hagnell (1975) excluded some of the individuals who were included in the Hagnell (1966) data because of incomplete records. An adjustment was made to correct for this discrepancy based on the ratio of the total numbers in the two reports. The exact calculations are available from the authors.

If the distribution of the three levels of personality deviation had been the same for the Lundby residents as for the schizophrenics, there would have been no way of predicting the occurrence of an episode of
Table 6. Distribution of Lundby data on the general population free of mental disorder by 'lesional status' and of the Bleuler data on schizophrenia by premorbid status

<table>
<thead>
<tr>
<th>Lesional status</th>
<th>Lundby No.</th>
<th>%</th>
<th>Bleuler No.</th>
<th>%</th>
<th>Premorbid status</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evident</td>
<td>47</td>
<td>3-7</td>
<td>135</td>
<td>33-9</td>
<td>Schizoid pathological and otherwise pathological</td>
<td>9-16</td>
</tr>
<tr>
<td>Probable and conceivable</td>
<td>667</td>
<td>51-9</td>
<td>144</td>
<td>36-2</td>
<td>Schizoid aberrant and otherwise aberrant within the norm</td>
<td>0-86</td>
</tr>
<tr>
<td>No lesion</td>
<td>571</td>
<td>44-4</td>
<td>119</td>
<td>29-9</td>
<td>Non-aberrant</td>
<td>0-67</td>
</tr>
<tr>
<td>Total</td>
<td>1285</td>
<td>100-0</td>
<td>398</td>
<td>100-0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

illness from knowledge of the premorbid personality. However, Table 6 indicates that the ratio of the proportion of schizophrenics to the proportion of the most deviant members of the general population was 9-16. It seems, therefore, that premorbid personality is of some prognostic value.

In order to relate the Lundby findings to Bleuler's data, it was assumed that the 1285 inhabitants of Lundby who were free of schizophrenia (and of other mental disorders) in 1947 would, during the succeeding years, develop schizophrenic episodes to the extent of 1% of this number. In order to eliminate decimals, the sample size was multiplied by 10, yielding an expected number of 128 schizophrenics. These 128 schizophrenics were assumed to have the same distribution with regard to personality as did Bleuler's schizophrenics. The results of this hypothetical analysis are shown in Table 7. Table 7 indicates that the premorbid personality is associated with the development of schizophrenic episodes. The inhabitants of Lundby whose primary personality was in the most deviant category - 'lesion evident', in the Lundby data, corresponding to 'schizoid pathological' and 'otherwise pathological' in the Bleuler data - have the highest risk for developing a schizophrenic episode (9-36%).

Table 7. Hypothetical table of 12850* mentally healthy members of the Lundby population according to their primary personality status in 1947, of whom 1% would develop a schizophrenic episode

<table>
<thead>
<tr>
<th>Primary personality</th>
<th>General population</th>
<th>Schizophrenics (expected)</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesion evident</td>
<td>470</td>
<td>44</td>
<td>9-36</td>
</tr>
<tr>
<td>Probable and conceivable</td>
<td>6670</td>
<td>46</td>
<td>0-69</td>
</tr>
<tr>
<td>No lesion</td>
<td>5710</td>
<td>38</td>
<td>0-67</td>
</tr>
<tr>
<td>Total</td>
<td>12850</td>
<td>128</td>
<td>1-00</td>
</tr>
</tbody>
</table>

* The total population was multiplied by 10 to eliminate decimals.

REFERENCES


Gove, W. R. & Hughes, M. (1980). Re-examining the ecological fallacy: a study in which aggregate data are critical in investigating the pathological effects of living alone. Social Forces 58, 1157-1177.


Hare, E. H. (1956b). Family setting and the urban distribution of schizophrenia. Journal of Mental Science 102, 753-760.


