Possible Implications of the Vulnerability Hypothesis for the Psychosocial Management of Schizophrenia

Joseph Zubin, Ph.D.

Biometrics Research, 151R
Veterans Administration Medical Center
Highland Drive
Pittsburgh, Pennsylvania 15206 USA
and
Department of Psychiatry
University of Pittsburgh School of Medicine

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The term "management" might lead one to infer that schizophrenia is a disease which is life-long, has no cure and the only option open to the therapist is try to manage the patient so that he can cope as well as he can with his predetermined fate, as is the case with Huntington's Chorea or Parkinsonism.

I was, therefore, glad to note that Professor Wing has worded his title to refer to 'long-term' course rather than 'life-long' course. I would, however, go even further, and state that even long-term course may not be a necessary outcome of schizophrenia. The long term course may be largely an artifact and not a natural part of the course of the disorder, but due to the impact of iatrogenic, nosocomial and ecogenic forces. These sometimes stand in the way of the natural healing process that applies to schizophrenia as it does to other mental disorders, as well as to some physical disorders.

The assumption that schizophrenia is necessarily a long-term disorder leading to deterioration or to severe reduction in quality of life may be a relic of a previous era which persists despite the evidence to the contrary. The generally benign outcome of schizophrenia which has been demonstrated by three outstanding long term European studies, i.e. the studies of Manfred Bleuler (1978), Luc Ciompi (1980a), and Gerd Huber (1980) has not yet been fully appreciated. Manfred Bleuler has pointed out that the reason why Eugen Bleuler, Emil Kraepelin, Kurt Schneider, and their colleagues endowed schizophrenia with such a jaundiced outlook, was the biased sample of patients they saw. These patients were drawn largely from those whose families had
given them over to institutionalization and who were already on a chronic course. Kraepelin saw the task of the clinician as consisting mainly of diagnosing, providing a prognosis, and leaving the disorder to take its natural course (Zubin et al., in preparation). In the USA too, the average practitioner is exposed continuously to the recidivists in his practice and forgets about those who had only one episode and were never readmitted! (Apparently from 23 to 39% of schizophrenic patients seem to fall into the latter category and the proportion having an episodic course with final remission is from 35% to 78%. (Zubin, Steinhauer, Day, van Kammen, in press).

The reason for this cultural lag in the acceptance of the more benign character of schizophrenics, is perplexing. It may lie in the cultural persistence of the "Degeneration Theory" which reigned supreme during the 19th century. It suggested the presence of a familial degenerative strain that became increasingly more severe over successive generations, eventually causing extinction of the line (Zubin et al., in preparation). Although science has long ago disproved this theory, the folklore may still retain it, and may influence patients, family members, the general public, and even some clinicians to regard schizophrenia as a degenerative disease. This hypothesis may have had some influence on Kraepelin's thinking about the etiology of mental disorders including dementia praecox, manic depressive psychosis, hysteria, and even some physical disorder, since, as Akerknecht (1968) points out, "Kraepelin, although much influenced by it (degeneration theory) expressed numerous reservations."
We all recognize that the model we adopt for the etiology of schizophrenia is bound to determine the management we apply to it. There are at least seven scientific models for this etiology (ecological, developmental, learning theory, genetic, internal environment, neurophysiological and neuroanatomical) (Zubin et al., in press). These can be classified into two major divisions: biological and environmental. I have selected three representative subsets of the seven etiological models and one superordinate model - the vulnerability model, and will limit our discussion to the management policies of each of them. In order to focus the discussion, I will target primarily the management of negative and positive symptoms (PNS) by each of them and contrast their approaches.

First, let us give a brief description of the three chosen models. The first is the biological or disease model inherited from Kraepelin's days and combines the four biologically based etiological models (genetic, internal environment, neurophysiological and neuroanatomical). The disease model, simply stated, postulates that schizophrenia is a disease similar to diabetes in so far as the disease is a permanent characteristic of the patient to which he has to adjust with the help of his physician in order to minimize the hazard to health and happiness which the disease presents. At best, the patient is a permanently sick person who once in a while may undergo an episode of health. In addition, the prevalent attitude towards the disease model inherited from Kraepelin is that it is a degenerative disease leading to deterioration or severe reduction of efficient living.
The etiology of the disease is presumed to be genetic or at least organic, and the treatment is aimed at reducing the symptoms by means of a suitable neuroleptic or other biochemical means, since no cure is available at this time. In other words, the goal of therapy is to reduce the impact of the symptoms, but the disease itself persists even if it goes into remission, presumably affecting behavior through residual symptoms. The disease model is represented here by one of the most vigorous proponents of the biological model - Crow (1980). He postulates two distinct subtypes of schizophrenia based on PNS. Type I is largely characterized by positive symptoms with biochemical dysregulation due probably to dopaminergic activity. Type II is largely characterized by negative symptoms that are associated with gross structural abnormality (cerebral atrophy, enlarged ventricles). MacKay, adds further that the negative symptoms may reflect chronic dopaminergic underactivity while positive symptoms emerge when there is a burst of dopaminergic overactivity. He postulates further that the negative symptoms are chronically present but that positive symptoms are periodically superimposed on them. According to this disease model PNS forms a permanent feature of schizophrenic behavior.

The second is the environmental model inherited from Sir Aubrey Lewis, represented by John Wing. It is essentially the ecological model described earlier. Professor Wing is a hard act to follow, but I am fortunate in so far as he has already laid down the basis for the model. It is firmly rooted in Anglo-European traditions of descriptive psychopathology and more recent thought about the role of social factors in psychiatric disorders. It is primarily concerned with
socioenvironmental factors affecting the onset, phenomenology, course and outcome.

The third model is the behavioral model emerging from and encompassing the developmental and learning theory models and represented here by Kurt Salzinger's (1984; 1981) and Wallace and Boone's (1983) approach. It postulates that there is no schizophrenic disorder except that which is seen in the behavior of the patient and that these behaviors, PNS, can be targeted individually for elimination by means of behavior modification techniques.

The superordinate model which attempts to provide living space for all the models is the vulnerability model. It stipulates that regardless of which of the seven etiological models may be operative in a given case, the patient is essentially a vulnerable individual. This vulnerability may exist only as a risk or may be elicited into an episode if sufficient stress and strain impinge on the patient. In order to identify vulnerable individuals, regardless of whether they suffer an episode, a series of studies to find markers of vulnerability are in progress (Zubin, in press a). Beginning with patients who have demonstrated vulnerability by developing an episode, a characteristic is found that differentiates them from the general population and from non-schizophrenic patients. They are then followed up to determine whether the marker persists after the episode terminates and whether it is also found in unaffected first degree relatives. Those markers that persist after the end of the episode and are also found in first degree relatives with a risk higher than in the general population, are
regarded as vulnerability markers. If they vanish with the end of the episode and are absent in unaffected first degree relatives, they are regarded as episode markers. Since the advent of the drug era and of the new psychosocial therapies, the focus of attention is no longer the episode itself because remission of acute symptoms is usually so readily attained. Now the focus has been shifted to the attempt to prevent future episodes or recidivism. This has become the goal of a study of recidivism in schizophrenia conducted by our Biometric Research Unit.

The vulnerability hypothesis, in contrast with the biological models, does not imply a chronic disorder but a permanent vulnerability to develop the disorder. Episodes are postulated as time limited. When the episode terminates, the individual usually returns to his premorbid level of adjustment to every day living. Thus, schizophrenia is postulated to be an episodic illness in the same sense as depression, epilepsy or allergy. However, even a vulnerable individual who has undergone a sufficiently stressful life event to elicit an episode, need not necessarily develop an episode. There are at least three protective factors which may abort the episode if they are favorable, and aid and abett the episode if they are unfavorable. These are: (1) the social network, (2) the ecological niche which the patient occupies and (3) the premorbid personality including his intelligence, and personal competence and learned coping skills. One way of epitomizing the difference between the vulnerability model and the disease model is to indicate that while the disease model regards the patient as essentially ill, but may have intermittent periods of wellness, the vulnerability model regards the patient as essentially well, but may have one or more
intermittent episodes of illness.

We can now turn to the therapeutic strategies which each of these models dictates, but before doing so, let us define PNS. Briefly stated, positive symptoms are behaviors that schizophrenics engage in but normals do not. Negative symptoms, when inverted to their opposites (e.g., "withdrawal" to "sociability") are behaviors which normals engage in but schizophrenics do not, or only in a diminished manner.

In a recent review of a symposium on PNS, after surveying the contributions of the seven outstanding contributors to this field, I found that there was a consensus on the following results (Zubin, in press):

1. Positive symptoms generally respond to neuroleptic treatment while negative symptoms generally do not, though Goldberg (in press) makes out a case for the responsiveness of even negative symptoms to neuroleptics.

2. Positive symptoms tend to be independent of intelligence while negative symptoms show an inverse correlation with intelligence, especially in patients who show structural brain impairment and have a poor premorbid personality.

3. Negative symptoms seem to persist and thus become more apparent in long enduring episodes or multiple episodes, while positive symptoms have shorter durations and seem generally to wax and wane with the
episodes.

4. Positive symptoms are aggravated by amphetamine injections presumably because dopamine transmission is facilitated. The effect on negative symptoms is either absent or non-spectacular.

5. Negative symptoms correlate with deviations in brain structure, especially in poor premorbid.

6. There is general agreement that both negative as well as positive individual items are not specific to schizophrenia. Whether combination of items into syndromes will show greater specificity remains to be seen.

7. There is some agreement that positive symptoms are associated more with neurochemical rather than structural anomalies in the brain.

8. It is generally agreed that there is difficulty in distinguishing between negative symptoms and depressive symptoms and criteria for schizoid personality.

9. There is general agreement that positive and negative symptoms are independent and that there may be 4 categories of schizophrenics with regard to the presence and absence of both types of symptoms: (1) both present, (2) both absent, (3) negative present but not positive, and (4) positive present but not negative. The majority of patients seem to fall in the first category.
It was not possible to find a model for integrating these findings which would cast light on the etiology of PNS, but the following conjectures were made by the authors:

1. They are the result of the impact of the psychosis, leading to reduction of normal behavior because the individual has been overwhelmed by the disruption produced by the psychosis.

2. The effect of antipsychotic drugs e.g. akinesia and sedation.

3. The effect of institutionalization.

4. Residual effect of the psychosis after the episode ends.

5. Effect of depressive episodes accompanying schizophrenia.

6. Reemergence of the premorbid negative personality traits at the termination of the acute episode.

The disease model is geared to deal with the acute positive symptoms by means of psychopharmacological intervention, lending some credence to the biological sources of the episode. One management strategy dictated by Crow's model for Type I patients with positive symptoms is to utilize reduced dosage treatment as a means of lessening dangers of relapse and tardive dyskinesia. Presumably, the danger of relapse under no medication and even under medication must be monitored so as to prevent it. Similarly, the danger of continuing in the episode
or even exacerbating it must be carefully monitored. Challenging techniques such as the amphetamine or ritalin type of biochemical challenge can be used to probe for the presence of the episode or its termination. Similarly, certain behavioral techniques (Zubin and Steinhauer, 1981; Zubin in press b) can serve the same purpose. As far as the negative symptoms go, since they are regarded as invariant, and especially resistant to drug treatment, there seems to be no other option except psychosocial intervention.

The behavioral model seems tailor-made to deal with PNS since it regards the disorder as consisting primarily of the presenting symptoms without any underlying general disorder. The behavior modifiers can train their guns on each symptom in turn and devise methods for eliminating it. According to this approach, the title of this symposium should be "Remedying Schizophrenic Behavior" rather than "Management of Schizophrenia." Salzinger (personal communication) describes the behavior modification approach as follows: "Negative symptoms, or as I prefer to call them, behavioral deficits, in schizophrenia are taken care of by behavior modification, in the main, by shaping behavior and by imitation learning. Shaping means that one reinforces ever closer approximations to the desired response. For example, if you wish to produce social behavior in a person who has none, you would first reinforce looking at other people, then listening to them, then saying something brief like "hello" and then a short conversation, and then have them speak to other people for longer periods of time, eventually getting them to telephone people or approaching strangers at a party to socialize with them. Imitation learning could be introduced by reinforcing the patients to
first observe the model and then to imitate rather simple responses of short duration, and eventually more simplex behavior of longer and longer duration; finally, one can then reinforce the spontaneous occurrence of the behavior that was originally emitted only as an imitation.

Positive symptoms would have to be dealt with by the following means, according to behavior modification: Punishment plus reinforcement of other, usually incompatible responses, that is, responses whose appearance would make the occurrence of the undesirable response impossible. Extinction is of course another method and can be used if a patient is emitting deluded speech, with positive reinforcement used when the speech is nondelusional. One can also employ a discriminative stimulus for other behavior when a patient emits undesirable behavior, as for example, by starting a conversation about the next meal, or about last nights baseball game, or about an election coming up, etc., whenever the patient becomes delusional or hallucinated." Salzinger has provided an explanatory hypothesis underlying this approach under the name of the Immediacy Hypothesis. This hypothesis essentially states that most of the deviant behavior of the schizophrenic can be explained on the basis of the assumption that the immediate stimuli in the environment are imperative over the non-immediate.

Thus, while normals can carry through intended activities despite the intrusion of new stimuli, schizophrenics are distracted from their intentions by such intrusions and attend to the intrusion.
Wallace and Boone (1983) have given several examples of how behavioral treatment may be applied to improving social skills by eliminating some of the negative symptoms. Thus, the problem of lack of conversation skills was dealt with by reinforcing continuous speech with an interlocutor. Eye contact and voice volume were improved by an assertion-training format in which improved behavior was reinforced. Training in recognizing possible options for responding in challenging situations was improved by a modified assertion-training format. Community-living skills and problem-solving skills were taught by a specially devised training program. Thus, by targeting the various negative symptoms, considerable progress can be made in their elimination.

With regard to the psychosocial model, Professor Wing has already covered it amply. The only thing left for me to do is to contrast its approach with that of the vulnerability model. Wing and Brown (1970) divide up the handicaps that the schizophrenic suffers from (among which are included the negative symptom) into three distinct types: (1) premorbid (low IQ, difficult personality social withdrawal) which they regard not so much as premorbid as possibly prodromal, although the considerable evidence for low IQ being premorbid, contradicts it; (2) primary disabilities, consisting of negative and of positive symptoms; (3) secondary disabilities—these stem not from the illness itself but from the social and medical consequences of having been ill. These include handicaps due to institutionalization, social stigma, loss of self-confidence and self-esteem, induced dependence and other inadvertant difficulties resulting from the patient's social interaction
with members of the medical profession, relatives and the general public. One of the basic tenets of this model is that schizophrenic patients are biologically vulnerable to both under- and over-stimulating environments.

The major points of difference between this model and the vulnerability model lie in the interpretation of premorbid handicaps, negative symptoms, and chronicity.

Regarding premorbid personality, Wing regards deviant traits that are present before the episode erupts as prodromal while the vulnerability model regards them as independent of the episode. Though there is an association between premorbid personality and the development of an episode, this association is rather mild (Zubin, Magaziner and Steinhauer, 1983). Thus, while the risk of schizophrenia for extremely deviant premorbid personalities is 9.36, and the risk for mildly deviants is 0.69, the risk for normal premorbid personalities is only 0.67. However, 90% of even the severely deviant personalities do not develop episodes, presumably because they are not vulnerable to schizophrenia. Hence, to regard extremely deviant personalities as prodromal runs the risk of being wrong 90% of the time.

The vulnerability model is presented with two dilemmas by FMS. First, the vulnerability hypothesis does not allow any room for the occurrence of chronicity, since every patient is suppose to emerge from his episode sooner or later. How to explain the frequency of chronicity in our daily practice is one dilemma. Secondly, since negative symptoms
are associated with chronic states, how can they be explained? There is no problem with positive symptoms, since the vulnerability hypothesis accepts them as characteristic of the episode and they wax and wane with it. But negative symptoms and chronicity present a problem.

While Wing regards some of the negative symptoms as intrinsic, the vulnerability hypothesis assimilates this syndrome completely into Wing's secondary or external disabilities. Thus, vulnerability theory regards negative symptoms as neither inevitable (Kraepelin) nor intrinsic (Wing). Instead, it regards them as essentially due to an artifact, developing as a social consequence of being labelled and treated as a schizophrenic by medical specialists, relatives, close friends, and other members of the patient's social network.

Another point of difference between the vulnerability and behavioral model on the one hand, and the disease and Wing's psychosocial model on the other hand is their negative attitude towards the episodic nature of schizophrenia which the vulnerability model stresses. If it be periodic, how explain chronicity? I was glad to find that Professor Luc Ciompi has also struggled with this problem (Ciompi, 1980a). Our combined arguments pro and con the assumption that

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1 The figures quoted here are based on the combination of two studies - M. Bleuler's study of premorbid personality in his probands and Essen-Moller's study of the general population in his Lundby study. The assumption that the two samples are drawn from similar populations may not be tenable, and the equivalence of the personality determinations by the two investigators not entirely acceptable, but it is the best data I can find for relating premorbid personality to the occurrence of an episode of schizophrenia.
chronicity is an indigenous component of schizophrenia are as follows:

Arguments Against Chronicity as an Indigenous Component of Schizophrenia

1. Not Universal: Not all schizophrenics develop chronic states. M. Bleuler found no more than 10% continuously hospitalized after first admission in his follow-up study.

2. Prevalence is Exaggerated: The apparent prevalence of chronicity is due to the accumulation of a small proportion of chronics, but even of these, in M. Bleuler's follow-up, 30% remain in the hospital for lack of any other home.

3. General Trend in Outcome is Remission Rather than Chronicity: Long term follow-up studies (Bleuler, 1972; Ciompi, 1980b; Huber, 1980) indicate that the general trend is towards improvement rather than deterioration. Sporadic recovery can occur even after a lifetime of chronicity.

4. Environment Rather than Heredity is Associated with Chronicity: Long term outcome is often independent of family history of schizophrenia - environment rather than heredity may influence the appearance of chronicity. Such moderating variables as social networks, ecological niches, and premorbid personality, if favorable, may mitigate the impact of stressors but, if unfavorable, may exacerbate the impact leading to chronicity.

5. Chronicity is Associated with Psychosocial Factors:
a. Life events play a triggering role in recidivism and in maintenance of chronic episodes.

b. Labelling impact prevents readjustment, especially occupationally.

c. Highly critical families play a role in the prolongation and resumption of schizophrenia episodes.

6. Negative Symptoms sometimes do Improve With Neuroleptic and Other Treatments and do Respond to Amphetamines. (Boronow & van Kammen, 1982).

Arguments in Favor of Indigenous Nature of Chronicity

1. Irreversibility of Negative Symptoms:

a. It has been claimed that the presence of irreversible negative symptoms characterizes chronic schizophrenia, but negative symptoms may be associated with chronicity because they may have been premorbidly present, and when the episode ends, the patients return to their premorbid level including their negative symptoms. As evidence for this possibility, 70% of Bleuler's probands had deviant premorbid personalities including negative symptoms, and about 58% of these poor premorbid states were regarded as attaining a moderately severe, or severe, end state (chronic) (Zubin et al., 1983). Furthermore, deviant behavior can develop due to institutionalization and isolation and may be mistakenly regarded as a natural consequence of schizophrenia. For
example, negative symptoms are also seen in long-term prisoners, neglected residents of old age homes, nursing homes, etc.

b. It is often difficult to distinguish chronic schizophrenics from other chronic patients in the back wards of institutions but these symptoms often disappear under social stimulation (Paul, 1977; Wing, 1970).

2. Biochemical or Organic Basis: No general accepted evidence is available for any specific somatic, biochemical or other organic basis for chronic schizophrenia. Chemical intervention, though effective in the acute phase against positive symptoms, is not generally effective in the chronic phase against negative symptoms; the latter may be more amenable to psychosocial intervention.

3. Ubiquity of Chronicity: It has been claimed that chronicity is ubiquitous and that it transcends differences in environments. However, cross-cultural studies indicate that developing countries have better outcomes and hence less chronicity than developed countries (World Health Organization, 1974, 1978).

Thus, it may be concluded that the proposition that chronicity is not an indigenous part of the course of schizophrenia (except in a small proportion of cases) is quite tenable in view of currently available evidence (Ciompi, 1980a; Cutting, 1983; Zubin, in press; Zubin et al., in press).
It is clear that the vulnerability and the behavioral models differ from the others on one major issue, whether chronicity and negative symptoms are a natural part of the natural history of schizophrenia or are they artifacts of iatrogenic, nosocomial and ecogenic factors.

The question of the intrinsic or indigenous character of chronicity and negative symptoms cannot be resolved until we find the basic cause of schizophrenia, discover the vulnerability markers and follow-up patients sufficiently long to note whether negative symptoms and chronicity are natural parts of the progress of the disorder. Meantime, we can adopt several strategies to determine the indigenous character of PNS and chronicity.

With regards to positive symptoms, it is assumed that they largely wax and wane with the episode. With regard to the negative symptoms, a quandary exists. They usually are associated with so-called chronic states. It is clear that some of these negative traits may have existed premorbidly, and their persistence postmorbidly is due to the return to the premorbid status. But what about the traits that did not exist premorbidly? Are they part of the schizophrenic process?

One way of answering this question is to determine whether the negative symptoms are mutable. If they are not, perhaps we can accept them as part of the schizophrenic process and not an incidental effect produced by iatrogenic, nosocomial and other ecogenic influences. If they are mutable, the likelihood is that they may have been produced as side effects. But the question remains whether the intervention that
eliminated them represents an efficacious way of treating the disorder rather than only eliminating a side-effect. Only the presence of these side-effects in non-schizophrenic patients who are subjected to the noxious niche which post episode schizophrenics occupy in life, can finally resolve this issue.

Depending upon the presumed source of the negative symptoms, the following strategies based in part on Carpenter (in press) are proposed:

1. If the negative symptoms are a response to the psychosis resulting from self-preoccupation or from a defense maneuver to dampen its impact, they should generally disappear with the recovery from the episode provided they have not been stamped in by nosocomial and/or iatrogenic influences.

2. If they are induced by drug treatment (akinesia, sedation) a change in the treatment regime should eliminate them.

3. If they reflect the understimulation which Wing and Brown (1970) found to be the case in many instances, they can be eliminated by environmental manipulation.

4. If they are induced by the acute psychotic episode or post episodically, proper therapeutic intervention should be found by clinical trial and error, leading to their elimination.

5. If these strategies fail to eliminate the negative symptoms,
they could be regarded as genuine aspects of the schizophrenic process which gives schizophrenia its chronic characteristics. However, before accepting this conclusion it would be of interest to determine whether these negative symptoms are not a reflection of the premorbid personality characteristics.

6. Another question that needs to be tackled is whether negative symptoms are specific to schizophrenia and not found in depression and other psychiatric or non-psychiatric statuses. For example, are they characteristic of individuals who are neglected or isolated through incarceration in prisons or in poorly managed nursing homes. Only comparative studies can answer this question.

All these strategies require in addition a long term follow-up to determine the permanancy of the negative symptoms and whether they return after being eliminated.

As a final point in the discussion of negative symptoms, it must be remembered that though psychosocial variables have not been demonstrated to be as important as biological variables in the etiology of schizophrenia, they have been shown to play a most important role in the triggering of an episode and in outcome. If we adopt the vulnerability hypothesis, good outcome as opposed to poor outcome may be determined not so much by biology as by the type of social network, and ecological niche to which the patient is returned when the episode ends and by the premorbid personality which he resumes when the episode terminates. In investigations of outcome, it becomes necessary that control groups or
comparison groups of other patients or normals must be equated on these psychosocial variable before sufficient conclusions can be drawn. Even on such biological factors as mortality, comparisons that do not include psychosocial variables such as social networks would be unacceptable, since the evidence that social networks are important factors in survival is now well established. For this reason the current data on the higher mortality rates for schizophrenia compared to other mental disorders and to normal controls are highly suspect if no suitable controls are introduced. The same holds true for the other psychosocial variables. Hence, in the investigations for the elimination of negative symptoms, the same principal must be followed and controls for psychosocial variables must be introduced when the efficacy of treatment outcome is being investigated.

SUMMARY

The implication of the vulnerability model for the psychosocial management of schizophrenia has been contrasted with the implication of three other models - the disease model (Kraepelin-Crow), the psychosocial model (Wing), and the behavioral model (Salzinger, 1984; Wallace and Boone, 1983). The discussion was focused on the management of negative symptoms since they are the most likely to be involved in management. A question was raised challenging the place of chronicity and the negative symptoms usually associated with it in the natural history of schizophrenia. The disease model (Crow) regards the negative symptoms as the basis for a subcategory of schizophrenia (Type II) and as inalienable parts of the disorder.
The psychosocial model (Wing) accepts some of the negative symptoms as primary and irreversible but considers the secondary negative symptoms - those that do not stem from the illness but from the social, and medical consequence of having been ill, as not an intrinsic part of the illness and subject to elimination by suitable techniques.

The behavioral model sets its aim on each of the individual negative items and attempts to eliminate them through behavior modification techniques.

The vulnerability model denies negative symptoms and chronicity as integral parts of schizophrenia and regards them as secondary phenomena induced by iatrogenic, nosocomial and ecogenic forces. This controversy can be resolved only by an attempt to eliminate the negative symptoms through further research. But even if this attempt meets with failure, there is still the possibility that those symptoms that are resistant to extinction may be residual symptoms from the premorbid period. If it meets with success, then claim for being intrinsic elements in the schizophrenic process would be reduced but not totally denied. To accomplish the latter, their presence would have to be demonstrated in non-schizophrenics who undergo similar deprivations.
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