continuum versus dichotomy in theories of schizophrenia*

Philippe Khouri

"The etiology common to the onset of a psychoneurosis and of a psychosis always remains the same. . . ."


There is a sharp difference of opinion in the psychiatric literature over whether schizophrenic disorders exist on a continuum with other mental disorders and normality, but this issue has in the past been overshadowed by another controversy—the role of genetic vs. environmental factors in the etiology of schizophrenia. This paper argues that the division of theories of schizophrenia into those emphasizing genetic and those emphasizing environmental models overlooks similarities between certain genetic and certain environmental models. Arranging the theories of schizophrenia according to the concept of a continuum of disorder allows for a new perspective, by which mono-genic theorists (e.g., Böök 1953, Heston 1970, Kallmann 1946, and Slater and Cowie 1971) and certain environmental theorists (e.g., Alanen 1966 and Lidz 1973) form one group; while proponents of polygenic models (Gottesman and Shields 1973) and other environmental theorists (e.g., Bateson et al. 1956 and M. Bleuler 1965 and 1972) form a second group.

The concept of a continuum of schizophrenic disorders is interpreted in various ways. At times, it is used in the context of phenomenology. This type of continuum refers to the lack of symptom specificity in schizophrenia—a lack that has been cited by Kraepelin (1971), Meyer (1948), Sullivan (1974), M. Bleuler (1965), and more recently Strauss and Carpenter (1974). A contrasting position has been taken by Schneider (1959) who views certain symptoms as specific and pathognomonic to schizophrenia. At other times, the concept of a continuum is applied to the outcome or prognosis of schizophrenic disorders. This type of continuum refers to a variability in outcome possibilities in contrast to a unidirectional deteriorative outcome for schizophrenia (E. Bleuler 1950, M. Bleuler 1972, Meyer 1948, Strauss and Carpenter 1974, and Sullivan 1974).

Etiological Continuum vs. Etiological Dichotomy

Another context for the use of a continuum of schizophrenic disorders is etiology. In the following discussion the concepts of an etiological continuum and its opposite, an etiological dichotomy, will be explored. Those who hypothesize an etiological dichotomy posit the presence of specifiable etiological factors which operate in schizophrenia but not in other disorders. Some other theorists assume that there are no such specific factors. They postulate a number of relevant etiological factors with which the nonschizophrenic population has often had contact. Thus, they hypothesize an etiological continuum. Though for genetic theorists these factors are the "schizophrenogenic" genes and for the environmental theorists these factors are a variety of influences (often interpersonal), the common underlying assumption of the etiological continuum models is that any difference that exists between schizophrenics and nonschizophrenics is more quantitative than qualitative. In other words, people become schizophrenic because they have too many of the schizophrenogenic factors; there are members of the population who have had some—but not enough—of these factors and thus escaped having the illness. The

*Reprint requests should be addressed to the author at Rm. 2N-220A, Bldg. 10, NIMH, 9000 Rockville Pike, Bethesda, Md. 20014.
polygenic/multifactorial model of inheritance, as proposed by Gottesman and Shields (1973), is an example of a genetic continuum model, while the double-bind theory of Bateson et al. (1956) and the theory of M. Bleuler (1965 and 1972) are examples of environmental continuum models.

Theories of an etiological dichotomy posit a qualitative breach between schizophrenics and nonschizophrenics because putative factors in the etiology of schizophrenic disorders, whether genetic or experiential, are said not to occur in the nonschizophrenic population. The monogenic/major gene hypothesis of disease transmission proposed by Slater and Cowie (1971), Heston (1970), and others exemplifies a dichotomous genetic model. Lidz’s (1973) “Schizophrenic families” theory and Alanen’s (1966) similar hypotheses are examples of dichotomous environmental models.

The sampling of theorists who emphasize environmental factors is small because for many theorists it was difficult to find answers to the specific question asked: Are the etiological factors important in schizophrenia found also among nonschizophrenics, or are they limited to schizophrenics? One possible explanation for this difficulty is that some environmental theorists tend to focus on the individual and his immediate human environment. They attempt to answer the question: What made this person ill? They do not often concern themselves with what has become of others who may have had these same adverse influences (Arieti 1975). However, it was possible to find answers to this question (or at least allusions to it) in the writings of Bateson et al. (1956), M. Bleuler (1965 and 1972), Lidz (1973), and Alanen (1966)—some of the most prominent contemporary writers on schizophrenia.

In Table 1, genetic and environmental theorists are grouped according to their shared assumptions regarding an etiological continuum. These assumptions are either stated clearly or implied in their writings. Within a group, investigators may have (and frequently do have) drastically different views on the phenomenology, outcome, or “nature” of schizophrenia; but an attempt is made to present evidence that, despite these differences, they share common assumptions about the specificity or lack of specificity of etiological factors.

**Etiologically Dichotomous Models**

Proponents of this type of etiological model have tended to conceptualize in terms of “schizophrenia” or “schizophrenic” rather than the “schizophrenias” or “schizophrenic disorders.” Though variation in the clinical picture is recognized, a unifying factor is presumed to exist—that of etiology. Kraepelin began this tradition by stating that hebephrenia, catatonia, and paranoia should be grouped together and put aside from other disorders because they were all products of the same etiology. Kallmann (1946), Böök (1953), Heston (1970), and Slater and Cowie (1971) have similar views, presenting a genetic abnormality as the etiological factor. The major gene hypothesis as proposed by these theorists may be described as follows:

- Schizophrenia is a disease that has a genetic basis. The genetic basis is an autosomal gene with variable penetrance. (There is disagreement as to whether this autosomal gene is dominant or recessive.)
- Not all individuals who have the schizophrenogenic gene become schizophrenic. In Slater and Cowie’s (1971) model all homozygotes and 22 percent of heterozygotes are expected to manifest schizophrenia. The

### Table 1. Genetic and environmental etiological models of schizophrenia from the continuum/dichotomy perspective

<table>
<thead>
<tr>
<th>Model</th>
<th>Specificity/dichotomous</th>
<th>Nonspecificity/continuum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monogenic/major gene hypothesis, e.g., Böök (1953), Heston (1970), Kallmann (1946), Slater and Cowie (1971)</td>
<td>Polygenic/multifactorial hypothesis, e.g., Gottesman and Shields (1973), Reich et al. (1972 and 1975)</td>
<td></td>
</tr>
<tr>
<td>Polychromic/multifactorial hypothesis, e.g., Gottesman and Shields (1973), Reich et al. (1972 and 1975)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disharmony of personality tendencies, e.g., M. Bleuler (1972)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Environmental</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schismatic and skewed families, e.g., Lidz (1973)</td>
<td>Double-bind situation, e.g., Bateson et al. (1956)</td>
<td></td>
</tr>
<tr>
<td>Chaotic and restricted families, e.g., Alanen (1966)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 M. Bleuler’s interactional model assumes that both genetic and environmental factors are nonspecific, the genetic predisposition being a disharmony of “normal” genes rather than pathological genes.
phenotypic fate of those heterozygotes who do not develop schizophrenia is thus a problem. One answer has been to attempt to find cases of "near-schizophrenia" variously described as "eccentric personalities," "schizoid psychopaths" (Kallmann 1946), or "schizoid personalities" (Heston 1970).

- The presence of this major gene is necessary for the development of schizophrenia. Individuals who do not have the schizophrenogenic gene do not become schizophrenic regardless of the presence of modifying genes or environmental stresses. Thus, there is a qualitative difference between schizophrenics and the rest of the population by virtue of the presence of schizophrenogenic genes.

Lidz (1973) and Alanen (1966), who argue from the environmentalist side, appear to reach a similar conclusion about etiological specificity, though they might object to being grouped with some of those listed above. In his latest book on schizophrenia, Lidz (1973) articulates his approach clearly. He starts by stating that "a great deal concerning the etiology of schizophrenic disorders is now firmly established" (p. 6). He then proceeds to refute any specific genetic basis for schizophrenia and develops his theory, based on observations (not controlled clinical studies) of families of schizophrenics whom he has followed over several years. His conclusions could be summarized as follows:

- Schizophrenia is "one of the potential fates to which man is subject... due to... the many potential hazards that beset his path from infancy to maturity" (p. 6).
- One of those hazards is exposure to a schizophrenogenic family. The family is the medium for the acquisition of language and socialization skills. Disturbances in these areas characterize the disorders called "schizophrenia."
- Not all kinds of "flawed and deprived" family settings are responsible for schizophrenia—only "specific distorting influences" make a family schizophrenogenic (p. 23). He describes these "influences" in terms of schismatic and skewed families and gives clinical examples.
- In this model, schizophrenics differ from non-schizophrenics in a qualitative sense because of the specific nature of the etiological factors to which they have been exposed. Schizophrenic offspring are found only in schismatic and skewed families (p. 49).
- The specificity of etiological factors is such that only schizophrenia develops in the offspring rather than other psychopathological conditions (p. 23).

Alanen (1966) investigated the families of 30 schizophrenics and those of 30 neurotics. He found a qualitative difference between the family disturbances in the two series—10 "chaotic" families in the schizophrenia series as compared to none in the neurosis series. From this dissimilarity, he came to his conclusions about etiological factors. His position may be summarized as follows:

- There is no need to invoke any specific genetic predisposition (i.e., monogenic transmission). If any genetic basis exists, it is "diffuse rather than strictly specific" and may consist "in a heightened inborn passivity or in an inborn inclination to introversion" (p. 551).
- "The pathogenetic influences of the family environment" are the critical factors that produced "the disorders in the children" (p. 547).
- Those pathogenetic influences in the families of schizophrenics (the etiological milieu) are described as "chaotic" family interactions reflecting one parent's intense irrationality and incoherence. These influences are absent in families with neurotic offspring.
- In reference to etiological specificity, he states:

Thus, evidence is accumulating in support of the following view: the disorders met in the patients' environments of growth... differ depending on the disorder type; and they seem to differ to an extent justifying the conjecture that they are at least comparatively specific. [p. 550]

In conclusion, regardless of whether or not there is an overlap at the phenomenological level between schizophrenics and the rest of the psychiatric and non-psychiatric population, the dichotomous models discussed above do set apart schizophrenics from the rest of the population at an etiological level, because persons destined to become schizophrenic are assumed to have special etiological influences.

**Continuum Etiological Models**

These etiological models differ from the previous ones because they do not assume any one factor specific for the etiology of schizophrenia. The same factors operate in schizophrenics and nonschizophrenics, and these models emphasize quantitative differences rather
than qualitative ones.

The polygenic/multifactorial model represents a genetic continuum model. It is used by Gottesman and Shields (1973) and Reich and his associates (Reich, Cloninger, and Guze 1975 and Reich, James, and Morris 1972) to explain the familial distribution of schizophrenia. It uses the normal curve, which plots the distribution of traits like height, weight, and blood pressure—traits that are continuously distributed in the general population. The concept of the normal curve has been adapted by these theorists for use with non-continuous traits such as liability (figure 1). Falconer (1965) first developed the concept of liability to account for the sum total of genetic and environmental causes of a disorder—in that case, schizophrenia. The assumptions of the polygenic model can be summarized as follows:

- The liability to develop schizophrenia is normally distributed.
- Schizophrenia appears when its threshold on the liability scale is passed.
- The heritability of the liability is 85 percent. In other words, genetic factors are the most important contributing factors for the development of schizophrenia.
- The genetic basis is necessary for the expression of the disorder and consists of many abnormal genes whose distribution in the general population is continuous and normal (statistical sense).
- A corollary of the above assumption is that the putative abnormal genes exist in persons who are not schizophrenic, but in a lesser amount. This accounts for their asymptomatic status.
- From a genetic standpoint, any difference between

---

**Figure 1. The polygenic/multifactorial model**

![Image of normal distribution curve with liability threshold]

1The model shows the distribution of the liability in the general population with one threshold for schizophrenia.
Schizophrenics and the rest of the population is essentially quantitative rather than qualitative.

Arguing from the environmentalist side, Bateson, et al. (1956) have presented the double-bind theory as an alternative etiological model for genetic theories. The theory represents an environmental continuum model and assumes the following:

- Schizophrenic symptoms are not a reflection of disease processes in the medical sense of the word but a reflection of communicational disturbances between the “victim” (in this case the schizophrenic patient) and another member of his family (usually the patient’s mother).

- Schizophrenic disorders appear as a result of a sequence of experiences that bring about a double-bind situation in which, whatever the victim does, he is the loser. The double-bind theory is based on the “theory of logical types” (Whitehead and Russell 1910), which stipulates that there is a logical discontinuity between a class and its members. In the psychology of everyday communications, an attempt is always made to breach various levels of communicational modes—for example, modes of play, fantasy, nonplay, and humor. The schizophrenic-to-be is continuously thwarted by his mother in his attempts to breach his different communicational modes in a “normal” and “consensually validated” way. The mother’s attitude reflects her own psychological difficulties which are of unconscious origin. The symptoms of schizophrenia appear when certain formal types of breaching are used by the “victim.”

- The future schizophrenic is victimized by the double-bind situation and becomes conditioned to react to all situations in life as if he were in a double-bind even though he may no longer be exposed to a double-bind situation. The notion of “a priming time”—an initial time when the double-bind situation is most effective—is used to explain the occurrence of this conditioned response when the conditioning stimulus (double-bind situation) is removed.

- The double-bind situation is a characteristic of families of schizophrenics, of the relationship between the preschizophrenic and his mother, and of normal relationships. Thus (according to Bateson et al.), any person exposed to a double-bind situation will “respond defensively in a manner similar to the schizophrenic” (p. 254).

In conclusion, a rather complex family dynamics—the double-bind situation—is considered to be causally related to schizophrenia but not limited to families with schizophrenic offspring. Indeed, this family dynamics is universal and only when it is extreme does it lead to schizophrenia.

Some environmental theorists, like Wynne (1970) and Arieti (1975), have been careful not to exclude any significant inborn component in the etiology of schizophrenia, and individual variabilities among the potential schizophrenics are mentioned to account for the differential outcome of individuals exposed to the same factors.

Another example of a predominantly environmental and continuum model is that of Manfred Bleuler. Derived from that author’s experience with schizophrenics and their families over several decades and described in his latest book, M. Bleuler (1972) posits a phenomenological, an outcome, and an etiological continuum. With regard to etiology, he takes a middle-of-the-road position between heredity and environment. He rejects any specific schizophrenogenic gene and considers the inheritance to consist of normal genes (found in the rest of the population) whose combination is “disharmonious” and is reflected in “disharmony of the tendencies of the personality.” The environmental contribution is also nonspecific, as none of the experiences to which the schizophrenic-to-be is exposed are absent from the rest of the general population.

M. Bleuler goes on to explain why, in his opinion, research on the etiology of schizophrenia has ended in a blind alley. “Schizophrenia... [is] ... due to a multiplicity of factors similar to those that play a role in everyman’s development.” Most researchers implicitly assume schizophrenia to be a disease process grafted to the individual’s personality, and this nonheuristic hypothesis, Bleuler maintains, is responsible for the paucity of data regarding the etiology of schizophrenia.

Any discussion of theories of schizophrenia that omits Eugen Bleuler leaves itself open to criticism. The continuum/dichotomy perspective in this paper focuses on etiological models. E. Bleuler (1950) emphasized the phenomenology of schizophrenia and the need “to explain” its symptoms rather than what caused schizophrenia. Nevertheless, E. Bleuler is noncommittal when he speaks of etiology, as he favors a morbid process involving the brain but does not rule out that “the entire symptomatology may be psychically determined... and [may] develop... [from]... slight quantitative deviations from the normal” (p. 461).
Comment

Despite their differences, both genetic and environmental etiological models of schizophrenia make common assumptions about the specificity, or lack of specificity, of putative etiological factors. The monogenic theories (Boök 1953, Heston 1970, Kallmann 1946, and Slater and Cowie 1971) and the environmental theories of Lidz (1973) and Alanen (1966) assume that a qualitative difference exists between schizophrenics and non-schizophrenics when etiological factors are considered. The polygenic model (Gottesman and Shields 1973), the double-bind theory of disease transmission (Bateson et al. 1956), and M. Bleuler's (1972) model, assume—contrariwise—that a quantitative difference exists between schizophrenics and non-schizophrenics when etiological factors are considered.

References


Acknowledgment

Special thanks are due Dr. Ronald O. Rieder for his helpful comments in the revision of this paper.

The Author

Philippe Khouri, M.D., is a Visiting Research Associate, Laboratory of Psychology and Psychopathology, National Institute of Mental Health, Bethesda, Md.