Psychogenesis Versus Biogenesis: The Issues and the Evidence

Bernard Rimland, Ph.D.

Editor's Note

Dr. Bernard Rimland's article, reprinted here, presents a logical and valuable review of the nature nurture argument. Those favoring a psychodynamic view now must present the scientific evidence that their hypothesis merits any further examination.

A transfer of all the research funds being wasted in psychoetiologic research to bio-chemical or Orthomolecular research will hasten the day when schizophrenia is brought under control. This article is so relevant to psychiatry and especially to schizophrenia that it is reprinted.

We will be pleased to publish letters or notes from readers who feel they have some scientific facts which can clarify the issue.

-A. Hoffer, M.D., Ph.D.

This paper is a highly condensed version of material which will appear in a forthcoming book, *The Psychogenic Hypothesis.* Because references to the literature will be available in the book, and would be unduly space-consuming here, I have limited the number of words cited. Some of the documentation not included here may be found in my book *Infantile Autism,* especially in Chapter 3.

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Orientation

"A voice out of the past which speaks of the future" is perhaps an appropriate description of the paper by Bernard Rimland.

The assumption that all psychopathologi-cal behavior is based on constitutional-genetic determinants was considered a tenable hypothesis in the nineteenth and early twentieth centuries. Since the rise to prominence of Freudian concepts, however, functional explanations of pathology have tended to put organic-oriented explanations into disrepute. Thus, to speak out publicly about such hypotheses has usually meant relegation to research oblivion.

Dr. Rimland's ideas are bold and controversial. He assumes that all personality disorders are organically based and that psychosocial influences are minor in the development of these disorders. He presents the arguments for both sides clearly and objectively before marshalling an impressive array of evidence against functionally based theories. The sacred cows of current beliefs about mental illness are systematically destroyed by his arguments and his manner of presentation.

Not content simply to criticize existing functional theories, the author offers strong evidence of biogenic causation in mental illness. If "environmental upheaval" is far

more serious than "social upheaval," as Rimland concludes, he is becoming the voice of the future in pointing to the coming directions of research.

Introduction

Millions of people throughout the world are so disturbed in thought and behavior that we call them "mentally ill." We know the cause of the disorder in many of these people: infections of the brain, tumors, toxic effects of chemicals or drugs, vitamin deficiencies, head injuries, and metabolic disorders are among the recognized causes of mental illness. In the case of millions of other affected persons, however, no specific cause can be ascribed. To these latter cases many psychiatrists and psychologists attach the label "functional" or "psychogenic" mental illness, indicating their belief that no physical or chemical impairment accounts for the disordered behavior. Rather, they claim that the disorder is a consequence of faulty relations with other people, especially in early childhood.

In using the terms "functional" and "psychogenic" the professionals explicitly assume that the patient has no biological defect to which his disorder might be traced, and they implicitly assume that there is in actuality a general class of disorders correctly called "psychogenic." That is, they assume that mental illness *can* be caused by faulty interpersonal experiences.

It is the purpose of this essay to question that assumption; to ask, "Why do psychiatrists and psychologists believe there are people whose mental disorder is functional rather than organic? Why do they reject the plausible premise that the 'functional' cases differ from the organic cases only in that our knowledge is at present too limited to identify the 'organic' defect in the 'functional' cases?"

The concept of psychogenic mental illness is so widely accepted today that most readers may regard these questions as too naive to deserve consideration. Yet, I maintain, they are not. These are extremely important questions which must be asked— often and insistently. Though the questions need asking, I think it is not yet possible for us to provide more than a fairly good guess (or should I say prediction?) of the ultimate answer to the question, "Is there a sound basis for the widespread belief in 'functional' mental illness?"

Let me emphasize, before we enter into any very detailed examination of the matter, that our task, at this stage, is more like that of a bettor at a race track than that of a juror in a court of law. That is, our task is not to reach a *conclusion*—any conclusion would be premature—but to make a *prediction*, albeit a prediction based on the available evidence. We know so little about mental illness, how to define it, what causes it—and for that matter, about how the normal brain functions—that to try to solve this problem may appear as futile as to try to describe a rainbow to a man born blind. Yet the problem of determining causation of mental illness is obviously an important one. Not only do our ideas about causation bear directly on how vast expenditures will be made in research and treatment, but they also have important implications for such everyday human affairs as child rearing, the management of criminals and delinquents, and even our attitudes toward ourselves and others.

What will the textbooks say 50 or 100 years from now about the causes of what we now term "functional" psychoses? Will they refer to the psychogenecists of the 1960's in the same half-amused, half-pitying way our current texts refer to the nineteenth century physicians who considered paresis a "moral disease"? Or will those who insist on the primacy of biological factors be seen in the wisdom of retrospect to have been foolishly misguided? How will the electics fare in retrospect—those who say it takes *both* a faulty constitution *and* a history of adverse social relationships to

cause the disorder? Is it logically inescapable, as some seem to believe, that the eclectics *must* be right?

The outcome of the search will not be a function of how popular each choice is with the current experts, nor of how confident each authority feels in asserting that he is right. The history of science is replete with instances of respected authorities who turned out to be very wrong.

I have stressed the tentativeness of the present picture to encourage the reader to consider my own prediction with an open mind. I predict that research will ultimately show psychosocial influences to have minor—if any—relevance in causing the limited disorders called "neuroses," and even less relevance in causing the severe disorders known as psychoses. This view is today an exceedingly unpopular one, unpopular both in the statistical sense of being relatively rare or uncommon, and in the affective sense—to doubt any long-held belief, perhaps particularly the belief in psychogenesis, makes people angry. Nevertheless, a number of years of close consideration of the available evidence has caused me to doubt that faulty interpersonal relations will appear in the textbooks a century hence as a significant factor in the cause of mental disorder. At the very least, I predict that it will be seen to have been grossly overrated as a causal factor.

My own professional training was similar to that of most psychologists. I was led to believe that psychosocial causation of mental illness was a fact established beyond doubt. I also learned (and many present textbooks continue to give this impression) that the few die-hards who questioned the psychogenicity of much mental illness were not only biased, old-fashioned, and irrational, but motivated by evil, antihumanistic intentions as well. This being so, I was later distressed to find occasional statements in the literature which suggested that my beliefs might be incorrect, and that what I was then teaching the next generation of students might be no more than myth. For example:

If the experiences of childhood importantly influence the later personality, we should expect to find some correlation between such experiences and the later occurrence of mental disorders. In fact, no such correlations have ever been shown (Stevenson, 1957, p. 153).

There are no data to prove that . . . there is a class of "functional" mental illness that is produced by emotional disturbance alone. (Hebb, 1949, p. 271)

There seems to be no clearly demonstrated instance of either a cultural or social factor being known to be a predisposing factor in mental illness . . . The absence of clear-cut evidence does not show that the hypothesis is incorrect but only that it has not been demonstrated even once. (Milbank Memorial Fund, 1961, p. 379)

Psychologists have reasoned that the experiences the individual has in his early life at home . . . are major determinants in . . . the development of Psychopathology. A review of the research of the past 40 years failed to support this assumption. No factors were found in the parent-child interaction of schizophrenics, neurotics or those with behavior disorders which could be identified as unique to them or which could distinguish one group from the other, or any of the groups from the families of the controls. (Frank, 1965, p. 191) Statements such as these surprised me. If they do not surprise you, read them again. If you remain unsurprised you are either an unusually sophisticated psychologist, or you are reading this chapter some years after it was written.

Upon finding assertions so discordant with my beliefs, and with the beliefs of the vast majority of other psychologists (in-

eluding virtually all textbook authors), I decided to take a long, hard look at the research evidence myself.

My prediction that psychogenicity of mental illness will eventually be abandoned as a tenable hypothesis results from the negative outcome of my search for unambiguous or even strongly suggestive evidence favoring the hypothesis, and from my discovery that the belief in psychogene-sis is founded on some rather amazing misinterpretations of the negative evidence. On the other hand, I found what I consider to be a good deal of solid evidence favoring biological causation even in those cases called "functional."

I don't imagine I can change the minds of many readers in the few pages allotted to me here. Nor do I pretend personally to have a very thorough understanding of this very complex matter. But I do want the reader to share my doubt of what is usually presented as fact. I feel strongly that if we accept as true anything that purports to be based on science rather than on faith we should be able to say why—to state the basis and cite the evidence for our belief. And I feel that the current high level of belief in psychogenesis has resulted from an unfortunate suspension of critical judgment—amounting almost to ideology— among people who regard themselves as scientists.

As an offshoot of the original problem, the problem of the belief system itself has intrigued me. Why is it that so many psychologists, psychiatrists, and other professional workers are convinced that there is such a thing as functional mental illness? Is is possible (Heaven forbid!) that *I* am wrong, and they have good reason for arriving at a view much different than mine? Self-deception should never be ruled out lightly.

On the following pages I have tried to present, as clearly and succinctly as I can, the major issues and assumptions that I feel underlie belief in the psychogenesis of mental illness. In conjunction with the discussion of these issues, I will present a sampling of the research evidence which bears on the problem.

Because of space limitations, most of the discussion will be confined to *severe* mental disorder—the psychoses. By limiting our concern primarily to severe disorder we can avoid becoming enmeshed in what I refer to further on as "the continuum fallacy." However, after having considered in detail some of the errors entailed in attributing psychological cause to the severe behavior disorders, we will be in a better position to discuss causation of the less severe disorders. The reader will find, I believe, that much of our discussion has implications relating to the causes not only of mental illness, but of individual variation within the normal personality range as well.

THE ISSUES

Much of the confusion regarding psychogenesis stems from fuzzy thinking. Let us start by defining terms as explicitly as we can, and by recognizing the fact when we cannot be completely explicit.

The Concept of Biogenesis

A biogenic mental disorder is a severe behavior disorder that results solely from the effects of biological factors, including both gene action and the effects of the physical-chemical environment. Biological factors may exert their effects prenatally, during labor and birth, and at any subsequent time. There are many examples: paresis, a consequence of syphilitic infection; pellagric psychosis, which results from a lack of certain vitamins; and various permanent and transient effects of such substances as alcohol, LSD, and amphetamine. An important point here is that we *know* that such biological factors can cause severe behavior disorders. We may not be

able to say of a certain specific individual whether or not his disorder is biogenic, but we know that the *class* of biogenic disorders is a real one.

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The Concept of Psychogenesis

Psychogenesis is harder to define, partly because very few writers have been very explicit in articulation, what the word means. Psychogenic or functional mental illness refers here to severe behavior disorder *purportedly* caused by adverse experience in the psychosocial environment, that is, by *socially meaningful stimuli* whose point of entry is the *sense organs* of the individual. In practice, the definition is usually tacitly limited to refer only to adverse interpersonal interactions. The distinction that psychogenic variables must input through the sense organs has not been made before, so far as I know, but it is important and I wish to make it explicit. The individual is assumed to be organically intact, or organic problems are assumed not to be the direct cause of the behavior disorder. The body is regarded as normal, and the abnormal behavior stems from consciously, or more often, unconsciously remembered experience.

The age at which the supposedly pathogenic events took place varies somewhat from one psychogenic theorist to another, though physical or psychological mishandling of the infant by the mother, usually in a vague and undefined way, is a commonly held view. Other psychogenic theories focus upon the developmental years, and refer to loss of a parent or inconsistent or self-contradictory communication patterns within the family as creating confusion. Somewhat more plausible, though still very weak from the evidential view, are the theories which focus upon the circumstances immediately preceding the breakdown. Even here, the fact that some persons break down readily under stress, while others endure far greater stress without breaking down, is often attributed to differences in child-rearing practices.

There is a rather trivial sense of the words "biogenic" and "organic" in which the distinction between biogenic and psychogenic disappears: since all learning and memory take place in biological organisms, even "functional" disorders can be reduced to a biological basis. Let me make it clear that my objection to the validity of the concept "psychogenic" is *not* based on this rather sophistic argument. It is instead based on the empirical position that there is little or no scientific *evidence* that one's social experiences do in fact cause or predispose one to become mentally ill. Stated somewhat differently, what I object to is acceptance of the assumption that the critical *difference* between mentally ill and non-ill persons resides totally or partially in differences in their social (largely familial) experiences, that the illness of affected persons could have been averted had they been raised in a "better" social environment, and that people who are not mentally ill would be ill if their social (largely familial) experiences had been sufficiently adverse. I contend that the bulk and perhaps even the entirety of presently available evidence contradicts the view that one's social experience has any important causative effect on whether or not he will become mentally ill.

An important distinction between the biogenic and the psychogenic concepts is that the latter can only be tentative in any given case. We can only be inferring when we say a patient has psychogenic or functional mental illness. This is so for two reasons: In the first place, one may question the *assumption* that there is, in reality, a class of disorders legitimately called functional, in contrast to the biogenic class, which demonstrably does exist. Throughout the history of science and medicine, firmly accepted immaterial causes of phenomena have been discarded when physical causes were discovered. To label an illness functional *is* obviously tenuous if tomorrow a virus, a vitamin deficiency, or some other biological factor may be discovered as the true cause.

If the reality of class of functional disorders may be questioned, use of the terms "functional" or "psychogenic" becomes even less defensible at the individual case level. Aside from not being sure there is such a category as "functional," one faces the additional hazard that the patient may later turn out to have an identifiable organic defect sufficient to account for his odd behavior. The literature of psychiatry is replete with cases of patients called psychogenic and given psychotherapy, only to succumb to an undetected brain tumor or degenerative CNS disease. Ross (1959) gives an example of this. A young girl had been given intensive physical examinations at three large medical centers. All findings were negative. Intensive psychotherapy was given to remedy her mother's "intellectualized" affection, which supposedly caused her strange behavior. When the girl suddenly died, a postmortem examination of her brain revealed massive degeneration which the neurological examinations had failed to discover. Malamud (1959) gives several similar examples.

Considering that present neurological and EEG methods often fail to discover even *gross* brain pathology which is clearly visible upon postmortem examination, and considering our virtually complete ignorance of how the normal brain operates (a famous neurophysiologist was quoted recently as saying, "We know *zero* about how the brain really works"), it would seem presumptuous to label any given case "psychogenic"—even if we were sure that some cases were psychogenic. Each of the ten billion neurons in the human brain is far more complex than any transistor or vacuum tube. We don't know how a neuron works, and we certainly have no instrument for determining the adequacy of even one of these neurons. To proclaim a behavior disorder "functional" under these circumstances seems as unreasonable as applying the same label to a malfunctioning television set when one lacks a tube tester, voltmeter, wiring diagram, and an understanding of electronics.

The terms "functional" and "psychogenic," if used at all, should be applied very tentatively, and then only *after* it has been shown that the category is not an imaginary one. Our present task is to try to predict whether the category will in time prove to be real or imaginary.

The Concept "Environment"

In discussing the concepts "biogenic" and "psychogenic," I distinguished between the physical-chemical and the psychosocial environments as the inferred sources of adverse effects. Many writers fail to make this discrimination and erroneously ascribe all adverse environmental (non-genetic) effects to the psychosocial environment. This failure represents an important source of the belief in psychogenesis. Examples are numerous. A striking one is Bettelheim's (1959) case of a psychotic girl whose illness he regarded as functional. In attributing her problem to the *presumed* effects of lack of mother love, Bettelheim ignored four

known causes of behavior disorder. The girl had been conceived and raised by her Jewish parents in a tiny, dark, cramped hole beneath a farm building in Poland in World War II. German soldiers were nearby (they sometimes fired shots into the building), and the mother had to smother the child's cries. Bettelheim emphasized such psychological factors as the mother's dislike of the father and the child's being unplanned, not deigning to mention such established adverse influences on the child as (1) prenatal development in an unbalanced endocrinal environment due to maternal stress; (2) extremely poor pre- and postnatal sanitary and (3) nutritional factors; and (4) extreme postnatal sensory deprivation. Each of these factors is known to have *demonstrable* effects on the young. One would think biological factors such as these at least warrant mention.

The relative potency of the physical and social environments may be compared by considering the "sensory deprivation" studies. When one's sensory input is sharply curtailed, such as by submersion in a water tank (with a breathing tube, of course!) in a silent and dark room, he is unable to tolerate the experience longer than 8 hours at most. By contrast, if a person is isolated only from *social,* as opposed to *physical,* stimuli, he can endure indefinitely, although he may become lonely. Hermits, forest rangers, marooned sailors, prisoners, and life-raft survivors are among those who have undergone lengthy *social* deprivation with no evident harm.

At times even biologically sophisticated people make the error of equating "environment" with "psychosocial environment." The cases of identical twins of which only one is schizophrenic are sometimes incorrectly cited by those who commit this error. They conclude that while inheritance may be relevant, the normality of the co-twin somehow proves that social factors play a role in causing the disorder. Actually, of course, one of the twins might have suffered adverse biological effects in the uterus, at birth, by postnatal infection, and so on, and the schizophrenia might have nothing to do with his psychosocial experiences. A recent review of the literature in fact showed that of 26 pairs of identical twins of which only one twin was schizophrenic, in 19 cases the schizophrenic twin had been the lighter of the two in birth weight—a statistically significant difference (Pollin, Stabenau, & Tupin, 1965). (This, incidentally, is similar to the finding on IQ scores of identical twins: the greater the birth weight difference, the lower the IQ score of the lighter twin.) However, Pollin, Stabenau, and Tupin show the usual preference for a psychogenic explanation: they suggest that the mother's special solicitude for the weaker child led to his schizophrenia. This type of explanation troubles me. If the heavier twin had turned out to be the more prone to schizophrenia, it could be said his mother's solicitude for the weaker sib caused him to feel rejected and to withdraw. Either or both of these explanations would be more convincing if there were some independent evidence showing that a mother's attitude has *any* causal relevance in the development of schizophrenia in her children.

When a psychogenecist cites evidence that a mental disorder is "environmental," he has in most instances ruled out the physical-chemical environment merely by ignoring it.

The "Moderate" Approach— Biogenic and Psychogenic Causation

The proposition that *both* an individual's biological makeup *and* his psychosocial history are important in determining whether or not he will become psychotic is so widely accepted that it has earned "sacred cow" status. One hardly dares question it. It may even be true, but I doubt that it is.

As in the case of many other self-evident "truths"—"The sun revolves around the earth," "A heavy object falls faster than a light one," "A heavier-than-air machine cannot fly," "The atom is indivisible"—neither plausibility nor wide acceptance, even by the scientifically trained, confers validity. Nor is validity conferred by stating the reasons why such propositions *must* be true: "If the earth were round, people would fall off it." "The atom cannot be divided because it is already the smallest particle of matter." These matters are empirical ones that can be evaluated only on the basis of scientifically valid evidence. The scientist's job is to question and test assumptions, not merely to accept those that are plausible or widely believed.

The "it takes both" position has no better claim to acceptance on faith than the alternate purely psychogenic or biogenic positions. It is quite conceivable that schizophrenia is as purely organic (nonfunctional) as paresis. I will accept the idea that social interactions contribute to the causation of schizophrenia, just as I will accept the idea that a cosmic ray striking one's navel is a contributing cause of schizophrenia, when I see good evidence that it is so— not before.

Let us look at the "it takes both" position more closely. It is of course true that genetic mental disorders, such as those associated with hypothyrodism and phenylketonuria, are dependent upon certain kinds of environments. These two disorders can be corrected by altering the environment with regard to the amount of thyroid and phenylalanine the patient ingests. It thus cannot be denied that these problems reside not only in heredity but also in the environment, providing you mean the physical-chemical environment. The misconception that psychosocial factors *must* play a role in the cause of mental illness can be traced in part to the confusion concerning to what the word "environment" refers, that is, to the tendency to ascribe the same potentialities to the psychosocial as to the physical-chemical environment. The well-known geneticist, Dobzhansky, in his book *Heredity and the Nature of Man* (1964), makes a statement that many would misconstrue so as to cause the kind of confusion I describe: He explains (p. 18) that the frequent dichotomization of human traits into hereditary and environmental is "false and misleading." Using skin color to illustrate the point that heredity cannot be separated from environment in its effects, he says that one could be strongly tanned by outdoor life or bleached by living indoors, "yet nobody doubts that the skin pigmentation is influenced by heredity." Exactly the same holds true for mental illness, many will claim, "you must have bad heredity and bad environment!" But note again that it is the physical-chemical environment which is important in Dobzhansky's example, except for such brief and transient changes as blushing and blanching. If one asserts that the psychosocial environment has much effect on long-term and significant changes in skin color, I will willingly listen, but it will be his burden to prove the point. Similarly in the case of behavior disorders, I want to see evidence that the social environment is influential in causing the disorder; don't just assert it *must* be so. (I might add, on the subject of skin color, that freckles make perhaps a good analogy to schizophrenia. Only a small fraction of the population is genetically predisposed to become freckled, but whether they do or not depends on the environment—the physical-chemical environment, to be sure.)

The analogy with skin color (and freckles) serves to illustrate the point I had in mind, but I hasten to admit that it may be misleading. After all, behavior disorders involve nervous system functioning, and unlike skin coloration, the nervous system *is responsive* to the social environment.

True enough. Even a child who is organically intact will not learn to speak unless he is exposed to speech. Thus, within the *normal* range of behaviors, the social environment can be said to have a demonstrable effect on the emergence of certain behaviors. Our basic question is: Does the same hold true for *abnormal* behavior, such as mental illness? My position is that I doubt that it does, but the question is one that must be answered empirically, and not, as my illustration was intended to show, one that can be answered by uncritically saying, "It takes both." The distinction between normal and abnormal is obviously crucial to this point, and it is not being taken lightly. It will be discussed in detail a little further on.

The "both" position has certain dangers which warrant special mention. (1) It is seductively attractive. Many people seem to derive notable satisfaction from proclaiming, "Mental illness cannot be entirely biological nor entirely psychological. We must reject both extremes. It must be both." Attractiveness notwithstanding, "both" may be an entirely wrong answer. (2) The "both" position often pays lip service to biogenic possibilities, then slides too quickly to what is in essence a wholly psychogenic position. "It takes both, but since we can't do anything at present about the biological part, let's concentrate on the other." Thus society is led to massive expenditures on completely unproven remedies and preventatives such as psychotherapy (which we will discuss shortly) and ultra-permissive child rearing.

In summary, the "both" answer is neither harmless nor *ipso facto* correct. Like the purely biogenic and psychogenic positions with which it competes, it must be judged on evidence.

The Concept of Causation

There are various ways of conceiving of causation, as evidenced by such adjectives as "precipitating," "predisposing," "necessary," and "sufficient." Despite the variety of ways in which psychosocial influences *might* be causative of mental disorder, I know of no reason to believe that they are *in fact* causative. Most studies which purport to demonstrate a causative relationship merely demonstrate a correlation. A surprisingly large number of writers on mental illness have apparently never heard of, or do not understand, the admonition, "Correlation does not imply causation." The studies on maternal deprivation, on so-called schizophrenic mothers, and on social class differences in mental illness, for instance, have all been criticized severely and justifiably because their authors accepted simple correlation as evidence for psychosocial causation, when competing hypotheses, such as the biogenic one, could equally well or better account for the findings. For example, many writers, knowing that schizophrenics are found disproportionately often in slum areas (that is, schizophrenia correlates with socioeconomic status), conclude that poor social conditions must *cause* schizophrenia. Several recent studies, however, such as Dunham's *Community and Schizophrenia* (1965), show clearly that most schizophrenics have *migrated* to the poorer parts of cities, quite possibly as a *result* of their disability.

In addition to these rather commonplace points, the problem of psychogenesis versus biogenesis presents some rather unique and interesting features regarding causation. One of these concerns use of the concept for the individual as against the group. For instance, if we consider the hypothesis that biogenic and psychogenic factors *are* equally involved in causing schizophrenia, the following possibilities are still open:

1. In half the population the disorder is entirely psychogenic, in the other half, entirely biogenic.

2. In each individual in the population, psycho- and biogenesis contribute equally.

3. The relative contribution of psycho-and biogenesis within individuals varies between rather wide limits, and the average contribution of the two factors is equal for the total group.

Positive results in the studies of psychogenesis would require us to try to untangle this complex problem. The strongly negative findings we actually have spare us this task.

Another problem stems from the difficulty attached to proving a negative proposition. When one hears or reads of a case of functional or psychogenic disorder, the label "psychogenic' 'is intended to indicate that some special features of the individual's psychosocial environment are considered to have caused the disorder. The burden of demonstrating the systematic exclusion of organic causation would seem to lie with the one who attached the functional label. This, as we have seen, is not possible, since neither do we understand how the brain works in general nor do we have instruments adequate to determine whether any individual brain is functioning properly. Thus, the assumed pathogenic features in the environment are pointed to. But for each person who develops a behavior disorder in an adverse environment, there are easily dozens in similar or more adverse environments who do *not* manifest the disorder. At this point, it seems to me, the psychogenecist is forced into postulating an organic or constitutional weakness in the patient. Once this concession is made, it would seem much more economical, scientific, and straightforward to attribute the *entire* disorder to the constitutional weakness, rather than to postulate nebulous, undemonstrated, and quite possibly purely imaginary factors in the psychosocial environment as having caused or contributed to the disorder.

The Fallacy of Confusing Content and Cause

The answer to the question just raised-Why postulate psychogenic causes if organic cause cannot validly be ruled out?— resides, I think, in an erroneous equating of the conceptual content of a disorder with its cause. If a person raised in a French home becomes psychotic, his bizarre speech is ordinarily in French. If he is raised in a deeply religious home, his hallucinations and concerns may well relate to religious matters. Raised in a home stressing wealth and power instead, the same person might dwell on these matters in his rambling. This is quite understandable, even if one is willing to regard the case as purely biogenic. Yet who has not read or heard of cases where the *content* of the disordered person's concern was used as evidence that the disorder was psychogenic?

Inferring cause from content is a particularly important source of the widespread belief in the psychogenesis of mental illness, because this type of reasoning is often presented in an apparently scientifically approved way in the public press.

The case of Marilyn Monroe is a good example. I have read many popular accounts of her repeated episodes of mental disorganization and depression, long-term psychoanalysis, and eventual suicide. Most of these accounts stress her unhappy early marriage and her concern with her fading beauty. Little heed has been paid to possible biogenic causes, although her own hospitalization for "breakdowns" and the fact that her mother had been institutionalized for many years suggest possible genetic causation. Has the "fading love goddess" aspect any real relevance? No matter. The public finds the psychogenic material more interesting, and thus the belief in psychogenic factors is reinforced.

Charles Whitman, the young man who in 1966 shot 14 persons from the University

of Texas tower, presents a similar case. Massive publicity was given to his dislike for his father, which was regarded as the psychogenic cause, but very little publicity was given to his malignant brain tumor, to the remarkable change in his personality which took place before the shooting, nor to the large quantities of psychoactive drugs he had been taking.

Much has been written about the man who piloted the airplane that dropped the atom bomb on Hiroshima. His subsequent mental illness is widely believed to be the consequence of guilt. Little mention is made of his long-term record of mental instability, nor of the failure of the remainder of the men on the several atom-bombing missions to become mentally ill. Was his Hiroshima experience at all relevant?

The fact that a mentally ill person was or was not a "love goddess," had too much or too little affection for his father, or felt deeply guilty because he killed in wartime or because he evaded the draft—all these and many more things may be part of the *content* of the patient's consciousness if he does or does not become mentally ill. To claim, as is commonly done, that these things somehow *cause* the illness involves, I think, quite an unwarranted assumption.

The Post Hoc Explanation Fallacy

Closely related to the last point is the ease with which plausible psychogenic explanations can be concocted—and accepted—after the fact. Students are readily convinced by textbooks case histories which make it appear logical that the sophomore should be found running naked in the snow claiming he is the reincarnation of Joan of Arc. After all, he *was* an only child whose parents insisted on his getting good grades and becoming a doctor and . . . Hah!

Quite often, in lecturing on the supposed psychogenicity of mental illness, I have illustrated the fatuousness of *post hoc* psychodynamic explanations by singling out in turn several members of the audience at random and asking.

You, yes, you in the third row, with the red shirt. Suppose you were to suddenly become psychotic tomorrow morning at 10 o'clock, because I picked you out at random and shot you with my imaginary mind-disintegrator ray gun. And suppose someone investigated your case to find a plausible psychological explanation for your cracking up. Could they do it? For instance, your fiancee may have told you she is leaving to marry a fat short-order cook, or maybe your wealthy granduncle died and is leaving all his money to found a monastery, or the dean's office may have said you are about to be expelled, or your mother used to say she loved your baby brother more than you, or . . .

After only a moment's reflection, everyone I've ever asked this question agrees: "Yes, if I become psychotic, there would be no trouble in ascribing a logical environmental (psychosocial environmental) cause." (If you doubt this, ask yourself the question.) This being so, how much credence should one give the textbook examples, the TV shows, the newspaper accounts, and the other sources of the prevailing belief in psychogenesis?

Looking back at my own training, which like that of most psychologists, instilled in me the belief that psychosocial causation of mental illness had been scientifically established, it seems to me that I was very much influenced by those fascinating case histories which appear in small print in the textbooks. The author used them with assurance to *illustrate* the operation of psychological factors in a given case. We students never questioned the author's desig-

nation of the case as "functional." The possibility that there might be an undetected organic disorder, such as a metabolic dysfunction, was never even mentioned, nor did I appreciate the now obvious point that plausible after-the-fact psychodynamic explanations of any behavior—either normal or abnormal—can be devised by any person with even modest imagination.

As part of a research study on normal males, Renaud and Estess (1961) conducted intensive clinical interviews with 100 "above average" young men. Renaud and Estess reported that they were quite surprised to find just as much supposedly "pathogenic" personal history material in this superior group as they were accustomed to finding in clinically abnormal persons. Needless to say, I was not a bit surprised.

Psychologists aren't the only experts in concocting after-the-fact explanations. Following an election, or football game or a squiggle on the stock market curve, the experts come out in full force to explain why what happened was inevitable and should have been anticipated. This is part of human nature, I suppose, and it is not very surprising. It is not science, however. Science requires that one demonstrate understanding of a phenomenon by predicting it; postdiction is quite insufficient. That *post hoc* explanations are so easily devised does not mean they are invariably invalid, of course. It does mean that we should look beyond them for evidence of validity.

The Continuum Fallacy

Most textbooks in psychology, psychiatry, and sociology present the psychogenic viewpoint, or the combined psychogenic and biogenic viewpoint, as though pychogenicity was of established rather than hypothetical relevance in mental disorder. An argument often used to advance this position is that the continuity of troublesome behaviors—the fact that behavior forms a spectrum ranging from a mildly offensive habit to the widely assaultive behavior of a schizophrenic—somehow demonstrates that these kinds of behavior merely represent differences in degree and not in kind. This is an unusually appealing type of argument, and it is, in my opinion, at the root of a large proportion of the fuzzy thinking that characterizes what are called the social sciences. It is demonstrably specious. It embarrasses me to admit that as a student I accepted this reasoning uncritically, and as a teacher I espoused it enthusiastically. In general, the idea is that if it is difficult to make a distinction between two neighboring points on a hypothetical continuum, no valid distinctions can thereafter be made even at the extremes of the continuum. There are thus persons who would argue that the existence of several variations of gray precludes a distinction between black and white. Hokum. While I will agree that some patients in mental hospitals are saner than some non-patients, and that it is sometimes hard to distinguish between deep unhappiness and psychotic depression, I do *not* agree that the difficulty sometimes encountered in making the distinction between normal and abnormal necessarily invalidates all such distinctions.

Many books and articles are devoted to asserting that mental illness is merely a myth, and that we are faced with only a distribution of normal personalities having more or fewer quirks, or having habits which conform to others' expectations in varying degrees. Yet even the most psycho-dynamically inclined psychologist who believes this will, seldom be found driving his auto without lights at 11 p.m., however imperceptible may have been the change from the bright daylight of 3 p.m. to the complete darkness of 10 p.m. Similarly, we may guess that no "mental-illness-is-a-myth" psychologist swelters in his long underwear in July, even though the change of

temperature from winter cold to summer heat is not only mere matter of degree(s), but an uncertain and over-lapping one at that, since some days in May are colder than some days in February.

If the continuum fallacy had misled investigators of overtly physical illness as it apparently has misled investigators of "mental" illness, modern medicine would not exist. Instead, we would be advised that since such measures as temperature, blood pressure, and white cell counts all fall along continua having no natural dividing points, there are really no such things as fevers, hypertension, or infections. These are merely gradations or variations from average, and they thus have no special significance. ("Besides, where are you going to draw the line?")

It should be evident that the distribution of phenomena along smooth gradients can lead the unwary to erroneous conclusions. The fact is that there *are* people who are mentally ill, many millions of them, and they exist in every land on earth, the continuum notion notwithstanding.

The Parallel Planes Concept

Since the continuum fallacy obviously may lead to some rather absurd conclusions, it needs to be viewed with skepticism. (One can use this pseudologic to demonstrate that the nose is the same as the ear: after all, each blends smoothly into the skin of the cheek, and surely no one wants to draw the line arbitrarily!) However, my criticism of the continuum concept should not lead us to reject it prematurely. I demonstrated that it was not necessarily a valid view; I did not demonstrate that it is invariably an invalid one.

Despite my criticism, I find that it *is* helpful to conceive of a population as falling along a continuum in terms of the psychological normality of its members. The distribution along the continuum may or may not be a "normal" curve in the mathematical sense; for our purpose it doesn't matter. At one end of this distribution are people whose behavior is so peculiar that protective custody is required for their own sake and for the welfare of others. A little closer to the main body of the population are individuals who are less disordered in their behavior and for whom custody is problematical, and so forth. At the other end of the distribution are people who are so completely rational and in control of themselves that there is not the slightest doubt about their stability. Since there are no gaps in this distribution, it *is* hard to draw the line. Who is to say that the most deviantly behaving people are not just a little different in *degree* from the others?

But suppose we now learn that 10 percent of the population had taken a few cocktails. This subgroup would tend to be concentrated toward the deviant-behavior end of the scale, though of course, the behavior of the silliest of the people who had not been drinking might be more peculiar than the behavior of the most serious and alcohol resistant of those who had. A better way of depicting the distribution of deviancy values, now that we know about the alcohol, would be to draw two separate but overlapping curves, so that the one for the drinking population is displaced to a different plane parallel to and a little in front of or in back of the plane of the first curve.

From our original vantage point, we felt sure there was but one single continuum. From our new vantage point, a somewhat different angle (now that we know about the cocktails), we can appreciate that there were always two separate curves that had overlapped along their base lines and that we were originally in error in not seeing the data in proper perspective. Suppose now that we learned that a second small subgroup of the population had taken a minute dose of LSD. These people—most of them—had appeared near the tip of our

original continuum, acting more bizarrely than almost anyone else in our population. Again project this group to another parallel plane, since they represent a different (though ostensibly overlapping) population. It was only our original inability to perceive these subgroups that led us to believe there was but a single continuum ranging from the solidly rational to the wildly deviant.

Now suppose there is a subgroup of people who are unable to metabolize adrenaline properly, as has been proposed in the schizophrenia theory of Osmond and Smy-thies (1952; see also Hoffer, Osmond, Callbeck, & Kahan, 1957), and whose bodies thus become loaded with the hallucinogenic substance, adrenolutin? We know that an analogous process operates in the metabolic disorder phenylketonuria (PKU), and that if the population we started our hypothetical study with was large enough, it would include several very bizarrely acting people who, if examined, would turn out to be victims of PKU. In fact, Benda has pointed out that some children with PKU are routinely classed as schizophrenics, until the diagnosing psychiatrist is told about the positive PKU test.

The foregoing presentation of the "parallel planes" concept does not, of course, show that any or all mental illness is biogenic rather than psychogenic. It does show, I think, that writers such as Adams, Jackson, Menninger, and Szasz, who call mental illness a myth, do not necessarily have a valid point in the apparent lack of other than arbitrary lines separating people called "normal" from those called "ill." The problem is that we do not yet have laboratory tests for such disorders and are thus forced to rely on behavioral symptoms. There are good reasons, to be presented shortly, to believe that these writers are quite mistaken.

Perhaps at this point I should note the argument, advanced by some, that since bizarre behavior of certain kinds is accepted as normal by certain primitive peoples, especially in their shaman (witch doctor), we Westerners are being provincial in thinking of such behavior among our own people as a sign of sickness. As Leighton and Hughes (1961), among others, have pointed out, behavior appearing psychotic or hysterical to Westerners may be the result of deliberate learning and practice on the part of the shaman, and thus is only superficially similar to the acutely psychotic behavior it resembles. And as Edgerton (1966) has noted, even very primitive African societies have, and recognize as abnormal, severe behavior disorders coinciding in detail with what we call schizophrenia.

The Matter of Diagnosis

It is indeed true, as many proponents of the psychogenic view have argued that there are hardly two informed people who agree on what schizophrenia is in general, or on whether or not a given patient is schizophrenic. They assert that schizophrenia does not exist and that "schizophrenics" are people who have failed to adjust to their social environment. Menninger, for example, in his book *The Vital Balance,* lists the many conflicting classficatory schemes for mental illness which men have devised over the centuries to support his contention that the patients so classified are not ill but have merely lost their mental balance.

It takes but little thought to dispose of this specious conclusion, though the perspective of history should spare us the exercise. A century ago one might have similarly pointed to the hodgepodge of physical disorders known as "consumption." Noting that there were just a few symptoms in common among the patients, and that chances for recovery varied markedly from case to case, one might have erroneously

concluded that such chaotic information could be due only to human diversity (or perversity!) and that no physical cause for consumption could possibly be found. Today we understand our ancestor's confusion, since the "disease" they called consumption included diabetes, tuberculosis, and other now identifiable disorders. Kan-ner (1958) has pointed out that the same problem existed not very long ago with "the fevers," which included malaria, cholera, and diphtheria. So it may be with a "mental" illness such as schizophrenia, which is very probably a conglomerate of separate diseases, each having disorientation of the higher functions of the brain as one of its most prominent symptoms. Obviously, until we know the exact cause of a disease, it is often difficult, if not impossible, to distinguish it from other diseases on the basis of symptoms alone—particularly if the other "diseases" are also a mixture of conditions of unknown cause. How can it be concluded that "mental illness," or "schizophrenia," must be of psychosocial origin because at the present time we are unable to label and classify it accurately? Of course, when the necessary laboratory tests are developed, the biogenic versus psychogenic dispute will be essentially over, though no doubt some will argue that the social environment *caused* the observed physical changes. Judging from the history of science and medicine, however, those who would argue for mystical, dynamic, intangible functional forces will probably lose out.

That we don't now have a test for, say, schizophrenia is clearly no argument for psychogenesis. As Curt Stern has pointed out, we also have no way of detecting Huntington's chorea until the victim reaches middle age and his brain begins to deteriorate, and Huntington's is clearly a Mendelian dominant genetic disorder. Considering the unimaginably small quantity of a substance, such as LSD, that can affect the working of the brain, it is not surprising that there should be a number of biogenic disorders difficult to detect biochemically.

Biogenesis and the Pessimism Problem

I have talked with many people in an attempt to discover why they believe in the psychogenesis of mental disorder. Those who are most frank sometimes espouse a position which logically should have no bearing on the matter: "If you think mental illness is organic, you are giving it up as hopeless. I prefer a more optimistic approach."

One need only cite cretinism, PKU, galactosemia, epilepsy, and diabetes among the many organic diseases with clear mental or behavioral involvement which are readily amenable to medical control. Those who believe that psychological problems are necessarily more hopeful than physical ones seem oblivious to history, which shows that centuries of lawmaking, teaching, preaching, threatening, punishing, explaining, persuading, and cajoling have not resulted in a notably more exemplary Man. Preventive and remedial medicine, on the other hand, have made remarkable strides, even in many disorders that defined solution while they were called "functional."

Is Mental Illness Unhappines Magnified?

It is widely believed that if a person becomes unhappy enough, he will "reject reality" and become severely ill mentally. This assumption seems to underlie a great deal of the belief in psychogenesis. It is a seriously held belief, though one often hears it expressed in a half-joking way: "It's enough to drive you crazy." (I have even caught myself saying that!) A great deal of thought on this matter leads me to doubt that unhappiness is of consequence in bringing about mental illness, though

there is no doubt that mental illness is one of the prime causes of unhappiness.

In addition to this problem, there is the interesting associated problem of people who are unhappy but not disordered being *called* mentally ill. This latter problem is well illustrated in the book *Mental Health in the Metropolis* (Srole, Langner, Michael, Opler, & Rennie, 1962). Based on interviews with nearly 2000 persons in Manhattan, the study reported only 18.5 percent of the population to be mentally "well"!

Inherent in the unhappiness-leads-to-mental illness concept is the idea that all humans are vulnerable to psychosis and will succumb if conditions become sufficiently grim. An interesting refutation of the "everyone has his breaking point" hypothesis is seen in the studies of World War II pilots who flew many missions despite high casualty rates among their companions. After the weakest broke down early in their assignment, the others seemed able to continue almost indefinitely, despite severe loss of weight and other signs of stress (Milbank Memorial Fund, 1961).

More interesting data on this matter come from a study of U.S. soldiers who were formerly prisoners of war in Korea (Strassman, Thaler, & Schein, 1956). Conditions were so intolerable—hunger, beatings, filth, cold, uncertainty—that some prisoners simply stopped eating, curled up into a ball, and died. For them, life was not worth living. Their surviving companions, who reported these cases, said emphatically that those who died in this way were sane and lucid until the end.

Considering the tragic plight of some humans whose sanity never falters, and the enviably favorable life circumstances of many who become psychotic, I find the hypothesis that unhappiness causes or contributes to mental illness patently inadequate. A more plausible hypothesis is that people who are becoming psychotic mismanage their affairs so badly as a *result* of their mental impairment that quarrels, loss of jobs, and other unhappiness-provoking events become common.

Psychotherapy

I am sometimes asked, "If you don't think the psychosocial environment contributes to mental illness, how do you account for the effectiveness of psychotherapy (or psychoanalysis) in helping victims?" This question, as probably many readers know by now, has a very obvious answer: there is no scientific evidence whatever that psychotherapy helps the mentally ill (psychotics or neurotics), despite the numerous studies which have attempted to show its beneficial effects.

In 1949, in his celebrated book *The Organization of Behavior,* D. O. Hebb briefly reviewed the evidence on the effectiveness of psychotherapy and psychoanalysis and concluded flatly: "There is no body of fact to show that psychotherapy is valuable" (p. 271). A few years later H. J. Eysenck made a more intensive review and came to the same conclusion (see Eysenck, 1964, for a more recent review). The literature on the effectiveness of child psychotherapy has been separately reviewed by several authors (Levitt, 1963; Lewis, 1965) with similar findings. Levitt, basing his conclusion on more than 50 studies involving thousands of children, said the conclusion was "inescapable" that psychotherapy could not be claimed to be effective.

The studies which claim that benefits are derived from psychotherapy seem to be only those in which no control group is used, and in which anecdotal and testimonial evidence make the findings scientifically useless. These are the kinds of studies that medicine (except for psychiatry) wisely learned to ignore long ago.

Space limitations prevent our reviewing the massive research literature on the efficacy of psychotherapy. It is possible only

to note briefly that the proponents (usually practitioners) of psychotherapy have fought back vigorously, but their claims are peculiarly small. Pointing to what they regard as technical shortcomings in the research, they say, for the most part, "Psychotherapy has not been proven useless—it simply has not been proven useful" (Astin, 1961). They have also, as Astin has noted, de-emphasized the "cure" aspects and have instead suggested rather nebulous general benefits, such as self-actualization or, perhaps, happiness, but again proof of efficacy is lacking. The burden of proof of usefulness traditionally rests on the advocates of any treatment. Be that as it may, the failure of psychotherapy (and I do think it is a failure, to put the matter bluntly) to ameliorate mental disorder in children and adults, while it does not prove biogenesis, is precisely what one would expect if the "insight" which psychotherapy is intended to provide had no bearing whatever on the genesis of the disorder.

Contrary to what most psychodynamic doctrine indicates, research shows that a substantial proportion of the mentally ill recover spontaneously, as do many people with the majority of illnesses that *are* widely recognized as physical in origin (Wolpe, 1961).

Since anyone questioning the claims of the psychotherapists is probably considered even more anti-humanitarian than one who questions psychogenesis, let me attempt to redeem myself by adding, as an aside, that I am in general agreement with the position of William Schofield in his book *Psychotherapy: The Purchase of Friendship.* Most people feel a desire to talk to a sympathetic person about their problems, and they should be given an opportunity to do so. To pretend, however, in the face of existing evidence, that such conversation has curative powers, or that the listener needs to be highly sophisticated in psychology or psychiatry, is quite unjustified. In any event, it is clear that the proponents of the psychogenic view cannot turn to psychotherapy research for support of their position.

Behavior Therapy

This discussion has been concerned with "insight therapy," as contrasted with a newer and apparently much more fruitful approach to behavior disorders—behavior therapy. Behavior therapy is based on a learning theory approach to the modification of behavior. In certain forms of neurosis, the symptoms are attacked directly, the old fear of symptom substitution being discarded as a superstition. Based on just a few years' evidence, the results of behavior therapy seem surprisingly good-good enough to have converted me from strong skepticism to rather enthusiastic endorsement.

Certain behaviors of psychotics, like the fears of neurotics, have proven amenable to a behavior therapy approach. The usual method of dealing with psychotics is operant conditioning, but in the case of psychoses, unlike neuroses, behavior therapists seldom if ever claim to have actually cured a patient.

The efficacy of operant conditioning in modifying pathological behavior is thought by many behavior therapists, including some leaders in the field, to indicate psychogenesis of the behavior problem. I doubt, however, that behavior therapy tells us anything about the *cause* of the problem. One can use conditioning successfully on a mongoloid child, a schizophrenic adult, or a decorticate dog. Does this imply in each case that the nervous system is sound and intact?

Does the demonstrated effectiveness of operant conditioning in improving the behavior of a child with, say, infantile autism mean that the autism must have been *caused* by selective reinforcement by the

child's parents of behaviors of an autistic sort, as some writers have suggested? No more than the usefulness of aspirin in relieving a headache means the headache was *caused* by a lack of aspirin.

While I have learned to respect the conditioning techniques of behavior modification as unexpectedly powerful devices for improving the behavior of both mentally ill and retarded children and adults, I have no sympathy for the naive belief of the many "behaviorists" or "Skinnerians" who have leaped to the untenable conclusion that because a mentally ill person can sometimes be taught to discontinue some of his "crazy" actions, he must be a normal person who has merely learned maladaptive habits.

Similarly, I cannot agree with the enthusiasts of the behaviorist approach who argue that because in laboratory-type studies you may be able to manipulate a normal person into temporarily acquiring a very specific behavior of a bizarre sort, *all* bizarre behavior must have been similarly produced. This is as logical as asserting that because you have discovered that natural blondness can be simulated with peroxide, *all* blondes must use peroxide on their hair. Further, this mode of thought ignores the basic question of *why* some persons acquire bizarre behavior in the real world and others do not: the essential problem of biogenesis or psychogenesis. The answer, "Their reinforcement histories differ," is merely an assumption, not a fact. As indicated in the quotations cited earlier, the available evidence contradicts the assumption. To focus on the behavior alone is to commit the error of confusing content and cause. Are the behaviorists willing to face the question: Does anything resembling their carefully contrived laboratory procedures actually occur in real life, and if it does, can it produce enduring changes in personality?

It seems to me that the Skinnerians should have learned from the horrible example of the reudians that there are real dangers in making extravagant generalizations from scanty, albeit interesting, data. Perhaps I should suggest to Professor Skinner that when his classic book *The Behavior of Organisms* is revised, he ought to give it a more seemly title, like *Some Behavior of Some Organisms.*

The Problems of Neurosis

Neuroses present a more difficult problem than the more severe disorders because they are harder to define and harder to discriminate, clinically and conceptually, from mood changes, anxiety, unhappiness, and other psychological variations among normal (non-sick) individuals. It is of more than passing interest to our main concern with possible functionality of psychotic illness that statistics show that neurosis is not simply a way station on the path to psychosis; psychotics, contrary in particular to the views of the psychoanalysts, are *not* ordinarily recruited from the ranks of the neurotics.

I have not devoted nearly as much study to the etiology of the neuroses as to that of the psychoses; nevertheless, since I am frequently asked my views on the topic, I will briefly state my present position, which is fundamentally a behavioristic one.

Those called neurotics fall into three categories: (1) some are temporarily anxious or unhappy because they have a right to be—life has been or threatens to be unkind. If one wishes to call them neurotics (I would not), these *would* be functional neurotics. (2) Some have temperaments or dispositions that lead to what appears to be chronic unhappiness. A good deal of research suggests a genetic element here. I know of no scientific research that shows child-rearing practices to have the causal influence on this condition that the popular and professional literature implies. (3)

Some have a severe and specific problem, such as enuresis or a phobia. Behavior therapists have taken the view that in these cases the symptoms *are* the disease: the idea of an underlying emotional problem is rejected as a myth. As noted above, the danger of symptom substitution is scoffed at by the behavior therapists as merely a deduction from psychoanalytic theory, bolstered by a few anecdotal instances. "Therapy" consists of training designed to eradicate the undesired habits or to teach new ones. Grossberg (1964) has provided an excellent review of this approach and Paul (1966), in his book *Insight vs. Desensitiza-tion in Psychotherapy,* has described an impressive experiment in which the behaviorist approach is shown to be superior to the traditional one.

Insofar as such specific behavior as phobias are learned and usually present a rather circumscribed problem, I might accede to their being called "functional." However, (1) these problems are perhaps better described as bad habits than as mental illness, (2) there is no reason to believe that their occurrence is in any way influenced by the patient's early family life or social relationships, and (3) since so few people are afflicted, the problem, despite its being amenable to psychological modification (which I would term "educational" and not "therapeutic"), would appear to be at its roots a biogenic one. Despite these reservations, I feel that learning theorists have made a substantial contribution in this area.

Weak Inference, or "Don't Confuse Me with the Facts"

In a paper in *Science* that attracted a good deal of attention, John Platt (1964) attributed the very rapid progress made in the fields of high energy physics and molecular biology to the use of a systematic research strategy which he named "strong inference." The strategy consists in carefully spelling out various alternative hypotheses for phenomena of interest, devising and performing studies capable of rejecting the incorrect hypotheses, then employing the confirmed hypothesis in a repetition of the cycle at the next point of uncertainty. The process is akin to finding the shortest path through a maze by carefully planning the steps to take at each point of choice.

It is no secret that psychology is not a pacesetter among the sciences. Psychology's sluggish progress is often attributed to the complexity of its subject matter. While this is no doubt a valid explanation, I think another important factor in the failure of psychology and the other social sciences to move ahead is their rejection of the strong inference model in favor of what I will call, by analogy, the "weak inference" approach.

Rather than being guided by their data, psychologists seem determined to cling to certain favored hypotheses regardless of the outcome of the research they may do. It will come as no surprise to the reader that I regard the psychogenic hypothesis as the prime example of this backwardness.

Biochemist Roger J. Williams (1959), among others, has also observed this phenomenon and has seen the need to protest it:

We therefore make a plea for an unprejudiced facing of the facts of heredity. We urge that such facts be accepted with as great readiness as any others. This plea seems necessary in view of the attitude which we have repeatedly noted, namely, that of willingness to arrive at "environmentalistic" conclusions on the basis of slender evidence while rejecting points of view which would emphasize the role of heredity, even though the weight of the evidence, viewed without prejudice, appears overwhelming (p. 16).

My own plea for "an unprejudiced facing of the facts" includes not only hereditary

explanations but those implicating the non-social environment. A few illustrations of weak inference will have to suffice, out of the dozens of examples which could be cited.

Osterkamp and Sands (1962) studied the birth and pregnancy problems, and the incidence of breast feeding, in mothers of schizophrenic children as contrasted with the mothers of less disturbed neurotic children. The more severely afflicted children were found to have more often been the product of a troubled pregnancy and delivery. Breast feeding was found to have taken place *more* often in the severely disturbed group of children. Despite these findings, "the results were interpreted in terms of the mother's unconscious negative feelings toward the infants" (p. 366).

Psychoanalyst Rene Spitz has won fame for his studies supposedly showing that when an infant is deprived of "affective interchange" with his mother, for example, when the infant is hosptalized, he experiences a major deterioration of his personality. Pinneau (1955), on analyzing Spitz's published data, made the interesting observation that of the 59-point drop in the average Development Quotient of the children, which Spitz reported as resulting from the mothers' departure, 43 points were lost *before* most of the mothers were separated from their infants.

Spitz's findings, however, were supported in a later study by Fischer (1952). Or were they? Fischer reported that her sample of "maternally deprived" institutionalized infants performed very poorly on the tests she used, and asserted the children's deficiencies were "environmentally fostered." However, as Pinneau (1955) pointed out, Fischer had chosen for her study the lowest scoring 62 infants out of a group of 189—a group whose mean IQ on the Cattell test was 76.1! Again the data appear to have been collected only as a formality, as a means of "proving" what the researcher knew to be true.

Beisser, Glasser, and Grant (1966), basing their conclusions on structured interviews with parents, reached the not surprising conclusion that "children of schizophrenic and psychoneurotic mothers are seen to have a greater rate of behavioral deviations than children of 'normal' mothers, as judged by the mothers themselves" (p.114). A number of possible explanations are evident, including (1) "sick" mothers may be poor judges of their children, (2) some children may have inherited the mother's tendency toward having behavior problems, (3) the interviewers may have been biased (the report does not indicate safeguards against this possibility). Despite the unmentioned and apparently unconsidered alternate explanations, (especially the second one), it is concluded that the results "provide support for the proposition that the family milieu and the nature and quality of its interactions has a significant contribution to the mental health or lack of it of its members" (p. 114).

Still another example of weak inference is found in the report of a large five-year comparison of psychotherapy with three drugs in a group of 299 women (Brill, 1966). The psychotherapy group was seen at least once a week for an hour, while the drug groups were seen for only 10 or 15 minutes weekly, biweekly, or monthly, over a shorter total period of time. To summarize the rather complex report of findings, in this study, as in scores of others, the psychotherapy group showed no improvement over the other groups (and appeared to be somewhat less improved than the meprobromate group). The author says. "These findings were unexpected. They suggest that the widespread preference for the traditional out patient psychotherapy is based as much on the physician's bias as on its *proven* greater effectiveness" (italics mine). But are these findings accepted, and the simpler, less expensive, more convenient methods recommended? No. "The

findings do not justify any departure from the principle of providing treatment which is based on an understanding of psycho-dynamics and unconscious factors in emotional illness" (p. 253).

I will not lengthen this depressing list with further examples. It should be evident by now that psychologists and psychiatrists believe what they want to believe. Perhaps when the reader sees the additional examples of this sort of "science" that abound in the research literature, he will be reminded, as I am, of the small printed sign that one often sees posted on office walls as an intended joke: "Don't confuse me with the facts, my mind is made up." Or perhaps he may prefer Norman Maier's, (1960) way of saying it. "Maier's Law" is "If the facts don't conform to the theory, they must be disposed of."

A BRIEF LOOK AT BIOGENIC FACTORS

I have said that I believe that psychogenic factors will ultimately be shown to have little, if any, relevance in mental illness, that the bulk of the available research evidence strongly counterindicates the psychogenic theory of mental illness, and that the present high level of belief in psycho-genesis is based largely on a series of irrelevant arguments, unwarranted assumptions, and misinterpreted evidence. This is essentially what I set out to do—to point to the large and alarming gap between what is believed and taught about psychogenesis and what research has actually shown. Many with whom I've discussed this matter have tried to excuse this deplorable state of affairs by saying, in effect, "So what if the evidence for psychogenesis doesn't hold up very well? The evidence for biogenesis is just as weak."

Obviously there is no biogenic evidence for any disorder called psychogenic that will convince the psychogenicists. If there were, the disorder would immediately be reclassified "organic" (as has happened so often in the past) without challenging the belief system. But despite the lack of conclusive biogenic evidence for disorders (like schizophrenia) called psychogenic, it is simply not true that the scoreboard for biogenesis is as vacant as the scoreboard for psychogenesis.

The space remaining does not permit more than brief mention of some of the reasons for believing that biological factors are operative in the causation of mental illness. Let us look briefly at some of the evidence for biogenesis of schizophrenia.

Schizophrenia resembles physical illness in a number of ways. For one, untreated schizophrenia comes and goes, as do many chronic physical illnesses in which remissions and relapses for unknown causes are the rule. For a long time we failed to appreciate this because of our preoccupation with the psychogenic model, which did not lead us to anticipate relatively symptom-free periods. This error has proven disastrous to uncounted thousands of schizophrenics who have been "put away" in the past. Only lately have we begun to realize that schizophrenics, like other ill and incapacitated persons, must be motivated toward constructive activity to avoid the lassitude and deterioration which severely impedes recovery.

The extraordinary success of biochemical methods in treating schizophrenia, while not proof of biogenicity, certainly provides a strong impetus toward that conclusion, especially when it is contrasted with the utterly dismal record of psychotherapeutic methods. It is well known by now that after increasing at the rate of about 10,000 cases per year for many years, the number of mental patients in state and local public hospitals reached a peak of 559,000 in 1955, when the introduction of the new antipsychotic drugs began decreasing the number of hospitalized patients until at the end of

1965 there were 83,000 fewer patients than in 1955. This decrease occurred despite a sizable increase in the total U.S. population.

It is sometimes explained, in apparent seriousness, that all the drugs do is make the patients amenable to psychotherapy. I am reminded at this point of the article which appeared in the April 5, 1966, issue of *Look* magazine, under the title *Breakthrough in Psychiatry.* The "breakthrough," which, judging from my mail following publication of the article, excited many readers, consisted essentially of the "direct analysis" method of psychotherapy for schizophrenia, wherein the therapist exerts his total effort and personality in the task of therapy. Previous employment of psychotherapy with schizophrenics was assumed to be not intensive enough to do the job. Photographs of this remarkably effective new approach were shown, and several dramatic cases illustrating its curative power were provided. Unfortunately, when a 5-year follow-up of direct analysis of schizophrenia was published later that year in the *American Journal of Psychiatry, Look* magazine didn't report it. Unlike the 15,000 or so subscribers to the *American Journal of Psychiatry,* the 7,200,000 subscribers of *Look* were not told that the schizophrenics in the untreated control group did just as well as the "direct analysis" group, and perhaps even a little better (Brossard, 1966; Bookhammer et al, 1966).

Also adding weight to the biogenic position is the experimental production of psychotic behavior in normal persons through biochemical means. One often hears the assertion that drug psychoses are not really very much like real-life psychoses. This is not so. While LSD may not realistically simulate schizophrenia, reserpine produces depression in some persons, according to Kety (1966), that is practically indistinguishable from endogenous depression. Lemere (1966) observes that amphetamine may mimic schizophrenia, especially paranoid schizophrenia, so closely as to be indistinguishable except for the presence of amphetamine in the urine. As indicated previously in this paper, there is no evidence that any psychogenic factors can produce such aberrant behavior, and still less evidence that they do.

Additional evidence on the biogenic side comes from the stability of the incidence and symptoms of schizophrenia from one century to the next, from one part of the world to the others, and from times of peace to times of war and turmoil. Despite common belief, statistics show the proportion of psychotics to be no greater in 1965 than in 1865, no greater in rushing, bustling competitive countries than in slow-moving underdeveloped lands, no greater in England during the nightly bombings of World War II than in the years before (Reid, 1961).

The least refutable and most consistent evidence for biogenesis, however, is probably that compiled by the geneticists. To briefly summarize the data from a number of studies (Buss, 1966, p. 319), the likelihood that a person will be a schizophrenic is a function of the presence of schizophrenia in his blood relatives, according to the following table:

No schizophrenic relatives	1%
Grandparents, cousins, nephews, and nieces	3-4%
One schizophrenic parent	16%
Both parents schizophrenic	38-68%
Half-siblings	7%
Sibling	5-14%
Fraternal twin	3-17%
Identical twin	67-86%

Various objections have been raised to the above data, none of which to me seem very compelling. A common objection is that the percentage differences between studies cast doubt on the validity of the

research. Yet all but one of the twelve studies which have included identical and fraternal twins show the concordance rate for identicals to be four to six times as great as for fraternals, even though the actual rates themselves vary from one country to another as a result of diagnostic differences and other problems. The kinds of objections raised might explain away concordant rate differences of, say, 10 percent, or even 20 percent, but to assert that errors in the diagnosis of schizophrenia, or in the determination of zygosity in twins could account for the differences of 400 to 600 percent which have been found seems rather far fetched.

I have already discussed another objection: the fact that there are identical twins who are discordant for schizophrenia. Discordance in identical twins shows merely that schizophrenia is not *entirely* genetic. It is not evidence for psychogenesis, since pre- or postnatal differences in physical environment have not by any means been ruled out. The study referring to the significantly lighter birth weight of the schizophrenic member of discordant identical twins has already been mentioned as specifically consistent with the prediction based on biogenic theory. Actually, genetic familial data on *known* physical disorders, such as tuberculosis and diabetes, give results very similar to those reported above for schizophrenia. The question of a psychogenic element in diabetes is seldom raised.

Some critics claim the above data do not show genetic causation. By this they mean that the percentages do not follow the simple Mendelian model for dominant and recessive genes. Genetic disorders do not necessarily follow the Mendelian model.

Among the many embarrassments these data hold for psychogenicists is the difficulty of explaining why a fraternal twin of a schizophrenic is no more likely to become schizophrenic than is an ordinary sibling-about 10 to 15 percent in each case. The family and social environment is certainly more similar for twins, even fraternal twins, than for siblings, who may be much younger or older than the one who becomes schizophrenic.

Another embarrassment to psychogenesis, as Lewis Hurst and Curt Stein have both pointed out, is that a schizophrenic father *is* as likely to have a schizophrenic child (about 15 percent likelihood) as is a schizophrenic mother. Since the mother tends to have much more contact with the child, this is hard to explain on psychogenic grounds, but it is entirely consistent with biogenic causation.

Two studies have been published during the last year which cast new light on the causation of schizophrenia. Both studies involved the use of children of schizophrenic mothers in a control group design. Higgins (1966) compared 25 Danish children reared by their own schizophrenic mothers with a matched group of 25 similar children who were raised apart from their schizophrenic mothers. "It was predicted that the mother-reared children would display greater maladjustment on the various measures than would the reared-apart children. The results failed to support the hypothesis" (p. 166). While Higgins' study is a valuable one, the subjects were children, and we do not know which, if any, will actually become schizophrenic in adulthood.

A landmark study, for a number of reasons, is Heston's (1966) follow-up of children born between 1915 and 1945 to schizophrenic mothers confined in an Oregon psychiatric hospital. Heston was able to obtain follow-up data into adulthood for 47 such children who had been adopted as infants into foster homes in which there was no suspicion of schizophrenia. By comparing this group with a carefully matched control group of children born of non-schizophrenic mothers who had also been adopted in the first few days of life by a

matched group of foster parents, he was able to determine what effect, if any, the genetic element might play in the later development of schizophrenia. The results were strongly in accord with genetic expectation. Five (16.6 percent) of the adopted children of schizophrenic mothers and none of the matched adopted children of normal mothers were found at follow-up to have been schizophrenic. Other psychiatric problems were also found more often in the former group. For example, 8 out of 21 of the adopted children of schizophrenic mothers had later records of psychiatric or behavioral discharge from the armed forces, while only one of 17 of the adopted children of normal mothers had such a discharge record.

The design and procedures Heston used appear to be airtight. Unlike the majority of studies which have concluded in favor of psychogenic causation, Heston's study ruled out competing hypotheses through the use of a control group and through refinements in the experimental procedures, rather than by simply ignoring them.

Further support for the biogenic causation of schizophrenia—by far the most important of the "functional" disorders—is now emerging from a variety of other sources, and some of these carry implications as to the nature of the possible biochemical defect which may be involved in schizophrenia. A good deal of interest has been aroused by a study by Dohan (1966), in which the per capita wheat consumption of five countries during World War II was compared to the number of hospital admissions for schizophrenia during the same period. In the three countries (Finland, Norway, and Sweden) whose wheat consumption was reduced by about 50 percent because of shipping shortages during the war, the number of admissions for schizophrenia was also cut nearly in half. In the U.S. and Canada, where wheat consumption did not change, neither did the incidence of schizophrenia. Although it is indirect, this study is of special interest because there are several studies linking schizophrenia with celiac disease, a metabolic disorder involving an unusual sensitivity to wheat and certain other grains. (On Formosa, natives eating very little of these grains are reported to have a schizophrenia rate one-third that of Northern Europe.)

In a very recent study Dohan (1968) was able to manipulate the severity of psychotic behavior in two groups of schizophrenic patients by secretly controlling their gluten intake.

Further evidence linking schizophrenia and its possible treatment to a metabolic error comes from the adrenolutin theory of schizophrenia of Osmond and Smythies and Hoffer et al. referred to earlier. This theory has met great controversy since it was introduced some 15 years ago. It is of interest that biochemist Seymour Kety, who had earlier been highly critical of this work, wrote recently (1966) that he had found "new and compelling evidence" favoring the hypothesis. Hoffer and his colleagues have reported favorable results in treating many schizophrenics with massive quantities of niacin, one of the B vitamins, in what Kety has described as an ingenious application of the theory.

I find the niacin approach to the treatment of schizophrenia intriguing in view of a study by Kaufman (cited by Williams, 1962), in which massive doses of niacin were found to be remarkably beneficial in providing objectively measured improvement in joint movement in arthritics. Since several studies of large populations of schizophrenics have shown a much lower rate of arthritis than would be expected, some interesting possibilities seem apparent. Could the niacin that normal persons use in CNS metabolism be lost into the blood-stream of certain predisposed individuals, thus producing the cognitive and emotional disturbances of schizophrenia, while protecting the victim from arthritis?

I have described but a few of many studies favoring biogenesis. My primary task, however, is not to support biogenesis, but to expose the tenuousness of the widespread belief in psychogenesis. I trust the reader now appreciates why I predict that the term "functional mental illness" will disappear from use as science progresses.

I am quite in accord with the thinking of Dalbir Bindra (1959), who said, "The available research . . . suggests that the psychodynamic approach, like so many other ideas in the history of science has turned out to be a wrong 'lead'" (p. 138). Like Bindra, I urge that psychologists give serious consideration to abandoning this dead end of research and practice, and turn their talents to endeavors based on logic and evidence rather than on wishful and muddied thinking.

[1] Severe stress seems only to accelerate or precipitate psychiatric failures, rather than product them. An analogy may be seen in the "hot day casualty" phenomenon in any large city, where an inordinate number of deaths occur during brief severe heat spells. Over a longer period, however, such as a month which includes several very hot days, the total deaths are often *not* increased. The reason seems to be that those who succumb were on the brink of succumbing anyway, and the stress merely advanced the date.

[2] See Footnote 1 on the "hot day casualty" phenomenon.

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