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NEUROBIOLOGICAL ASPECTS OF PSYCHOPATHOLOGY

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DEDICATION

This paper is dedicated to the memory of Paul H. Hoch, to whose leadership this association owes its continued existence and whose impress is still felt in our activities and programs. Elsewhere I have indicated his contributions to our association; here I wish to indicate the debt owned him by the Biometrics Research staff, a unit which he fathered and whose program this paper reviews. Above all, however, we all valued his deep and abiding friendship and collaboration and his deep compassion for the mentally stricken, in whose behalf he labored to the very last moment of his all too short life.

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THE BIOMETRIC APPROACH TO PSYCHOPATHOLOGY — REVISITED

by JOSEPH ZUBIN, Ph.D.

Biometrics Research, New York State Department of Mental Hygiene, and Columbia University

Seventeen years ago, in a presidential address before this association, I suggested a program for research in psychopathology based on biometric methods, in contrast to the clinical method generally in use. Only the mere outline of the method was presented, but stress was placed on its underlying theme—objectivity and quantification. Though some data were already available, the proposal was more in the nature of a blueprint than of a practical program.

Since then, however, the biometric approach has spread to several other centers and under various guises has become a permanent part of psychopathology. If my presidential address assisted in its birth, this talk tonight may signal its adolescence. While the ultimate usefulness of the approach must be left to history, it would not be amiss to report the developments that have taken place.

The implementation of our program began in 1953, about two years

The Paul H. Hoch Lecture, delivered before the American Psychopathological Association, February 17, 1968, at the Park Sheraton Hotel, New York, N.Y.
after my description of it to this association, with a project on prognosis in schizophrenia that applied a battery of objective techniques to the prediction of outcome. In 1956, when the man whose memory we are honoring today became Commissioner of Mental Hygiene of the State of New York, I was given the opportunity further to implement the biometric program. It had become quite clear that our small permanent staff and the somewhat larger grant-supported staff of students who had in the meantime obtained their Ph.D’s were insufficient to deal with the entire biometric problem. There was need for more personnel and more funds. Thus we were fortunate in attracting more students from Columbia University, in the departments of psychology, psychiatry, sociology, anthropology, and mathematical statistics, and in being granted research funds from the NIMH.

In addition, several skilled and imaginative researchers, trained elsewhere, had joined our staff. There is hardly an idea, concept or paper written under the aegis of Biometrics Research that does not owe much of its value to one of the staff that has joined our ranks in the past 15 years. The dictum of one of the ancient sages characterizes well my estimate of their share in this common effort: “Much have I learned from my teachers, and from my colleagues more than from my teachers, but from my pupils more than from all of them.” (From the Talmud)

My purpose today is to compare the blueprint offered in the early 1950’s with the accomplishments of the biometric approach, both in our group and elsewhere, during the succeeding fifteen years.

From our 1950’s review of the available literature on the biometric approach—i.e., the objective measurement of behavior—in psychopathology, it became quite clear that the available instruments for assessing patient behavior for diagnosis and for prognosis were far from ideal, leaving much to be desired from the point of view of reliability and validity. Clinically used tools like the Rorschach, TAT or other projective techniques turned out under inspection to be less like tests and more like interviews. Reliable and valid instruments such as intelligence tests, though useful for measuring intellectual functioning, were of little value in assessing psychopathology other than in the field of mental retardation. Personality tests of the self-reporting type, attitude tests, interest tests, and value tests were of limited use with mental patients.

As a consequence, we turned to the classical categories of human behavior and the methods for their elicitation and constructed a
Mendelejff-like table consisting of physiological, sensory, perceptual, psychomotor, and conceptual responses which can be elicited under idling state, energy, and signal stimuli (Table 1).

This taxonomy of responses and of stimuli or methods for their elicitation proved heuristic in our prognostic study of schizophrenia, but the heterogeneity of the patients classified as schizophrenic soon led us to realize that before success can be attained in assessing deviant behavior we must have a more reliable method of classifying patients.

At the present time, overt behavior and self-reported symptoms are the only bases we have for suspecting, identifying and diagnosing most of the mentally disordered. The primary tool for observing and identifying is the clinical interview. By mental disorder we mean any progressive condition which unless therapeutic intervention takes place leads to premature death, severe reduction of efficiency, severe limitation of happiness, or to all three of these ends. Some of the mental disorders are stationary, and we designate them as defects.

Our review of the literature of reliability of diagnosis led to a conclusion similar to Mark Twain's response when asked how he liked Wagner's music: "It is better than it sounds." But despite agreement of 70-80 per cent in the gross diagnosis of schizophrenia, for example, sufficient heterogeneity was left to cause concern. Other conditions such as neuroses are much less reliably diagnosed. Consequently, we turned our attention to the study of clinical diagnosis itself and to the tool most often used in arriving at it—the interview.

But the behavior exhibited by a given patient could be due to a variety of etiological factors. It was not sufficient to know the behavior alone; its antecedents were of the utmost importance. Since etiological knowledge is still poorly developed in psychopathology, we resorted to the description of potential or "ideal" etiologies in the form of scientific models. These have arisen on the basis of the phenomenological observations made by countless observers throughout the 33-century history of psychopathology. Among these were demonology, witchcraft, sin, familial degeneracy, brain disease (Griesinger), metabolic disorder (Kraepelin), genetics (Rüdin), development (Freud), learning (Meyer), focal infection (Cotton), social-cultural stresses (Durkheim), and many others. Out of the welter of possibilities, we chose six to work with: ecology, learning, development, genetic (heredity), internal environment, and neurophysiological (brain function).

While these models are conceived of as independent for heuristic
TABLE 1.—Examples of Measurable Activities

<table>
<thead>
<tr>
<th>Level of observed behavior</th>
<th>Stimulus variables</th>
<th>Energy variables</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Idling state</td>
<td>Appropriate energy</td>
</tr>
<tr>
<td>Variable</td>
<td>Function</td>
<td>Variable</td>
</tr>
<tr>
<td>Conceptual:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>Reverie and fantasy</td>
<td>Uniformly diffused light</td>
</tr>
<tr>
<td>Psychomotor:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>Spontaneous movement</td>
<td>Painful stimulus</td>
</tr>
<tr>
<td>Perceptual:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>Spatial and temporal orientation</td>
<td>White noise</td>
</tr>
<tr>
<td>Sensory:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>Background noises; cortical gray</td>
<td>Light of graded intensity</td>
</tr>
<tr>
<td>Physiological:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>—</td>
<td>BMR; basal EEG; basal PGR</td>
<td>Increase in carbon dioxide concentration</td>
</tr>
</tbody>
</table>

*From ref. 2.

purposes, they are in reality interdependent to a greater or lesser degree. This interrelationship is indicated by a Venn Diagram in Fig. 1.

Until recently, the first of these models—the ecological—has been referred to in our papers as the social-cultural, the social-cultural-physical, or even the social-cultural-physical-environmental model. We have wanted to refer to a very wide range of variables or factors, ranging from social class, family income, urbanization, etc., to geography, diet, climate, and so on, but the term has clearly become unworkable. Without attempting to move into the realm of general systems theory, which has recently become modish in social science, we here use the term “ecological model” to express the idea of the individual’s oc-
as Functions of Stimulus Variables*

<table>
<thead>
<tr>
<th>Configurations</th>
<th>Signs</th>
<th>Symbols</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Function</td>
<td>Variable</td>
</tr>
<tr>
<td>Aircraft forms or silhouettes</td>
<td>Recognition of identity of forms</td>
<td>Practical trouble-shooting test</td>
</tr>
<tr>
<td>Star-shaped maze</td>
<td>Mirror tracing</td>
<td>Classical delayed response stimuli in animal experimentation</td>
</tr>
<tr>
<td>Rotating Benham Disc</td>
<td>Subjective color experience</td>
<td>Usual visual alternatives in animal discrimination experiment</td>
</tr>
<tr>
<td>Patterned light stimuli</td>
<td>Visual threshold</td>
<td>Infant's faint cry</td>
</tr>
<tr>
<td>Photic driving</td>
<td>Change in EEG pattern</td>
<td>Bell ringing in Pavlovian conditioning</td>
</tr>
</tbody>
</table>

ocupying a “niche,” relative to the cultural and physical environment—that is, being in a particular place at a particular time.

The ecological model and the learning model refer primarily to exogenous factors impinging on the individual. The developmental model is partly exogenous, influenced by ecological and learning factors, and partly endogenous, reflecting maturation. The genetic, internal environment and neurophysiological models operate entirely within the skin but they are mutually interrelated as well as influenced by the ecological forces via learning and development.

We have thus far devoted a great deal of effort to measuring the deviant responses arising from the variety of factors tentatively subsumed
by each of the scientific models that were proposed for explaining the aetiology of psychopathology. The classification of these responses has been going on for a long time, and much more is known about responses than about the stimuli that give rise to them. In fact, the search for the stimulus is one of the perennial problems in behavior analysis. It is also quite likely that the number of different stimuli that can elicit these responses is greater than the number of responses observed. (This, however, may simply reflect the greater ease in observing and classifying responses than in observing and classifying stimuli, which are by definition what is being sought. It is probably true that the number of ways of classifying responses is large indeed, thus increasing the number of responses that might be considered.) But in order to provide good scientific models in which the links between stimuli and responses can be ascertained, we need a taxonomy of the stimuli that control deviant responses. This is one of the burdens of our current effort.

We shall now take up each of the models in turn, describing its assumptions, the causal agents presumably salient to it, the deviant behaviors associated with these agents, and the techniques provided for measuring them. For heuristic purposes, we deal one at a time with the causal factors of the model under discussion, assuming that the factors assigned to the other models are not also involved in the disorders under examination. Thus, when we deal with the ecological model, we will assume that it alone is responsible for the particular deviation, and that the basic capacities involved in development, learning, genetic expression, internal environment, and neurophysiology were intact to begin with. When these models become sufficiently specified we can turn to them for experimentally investigating the aetiology of the mental disorders. Considering these six models and the hypotheses each engenders for exploring a given question, we can continue experiments to test the tenability of each of them.

1. The Ecological Model

The human ecology model is built on the assumptions that all mankind is vulnerable to mental disorders and that, given sufficient deprivation, stress-producing loads, or other alterations in our environment, our behavior will be altered to the point where our ability to continue living normally as independent individuals in society is endangered. The evidence for social-cultural-environmental pressures as aetiological agents comes largely from studies of socio-economic status,
isolation, educational and social deprivation, and social-cultural change due to migration or rapid acculturation. Even the most sanguine environmentalist will not be satisfied with merely pointing to the above-mentioned factors as "causal" agents, but will try to determine just how the malignant factors bring about their deleterious effect.

To cope with the stimuli assumed to operate under this model, we need techniques and methods that will delineate the various environmental forces that underlie the production of psychopathology. Our handicap here is tremendous, because even preliminary descriptive work is yet to be done. We do not have a taxonomy of ecological factors that is suitable for psychopathology, nor do we know the links between these global forces and the proximate forms by which they may bring about psychopathology. Except for such relations as radiation affecting behavior through the mediation of genetic factors, or nutrition affecting behavior through metabolic deficiencies, we have thus far failed to specify the parameters of the environmental factors eliciting deviant responses.*

Recent work by Richard Wolf has illustrated how the correlation between social status and intelligence, which is usually found to be between .20 and .40, can be boosted to as high as .69 if the parameters of the socio-economic environment that have a bearing on intellectual potential are measured and included in the multiple correlation. Similarly, the correlation between social status and achievement, which is usually found to be .50, can rise to .80 if the parameters of social status pertinent to achievement are identified and measured. How the factors presumably underlying low socio-economic status will relate to the occurrence of psychopathology when their parameters are explicated and measured, no one can tell, but arguments such as those provided by the

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*It is, of course, the case that social science has developed a number of sophisticated taxonomies in what I am calling the "human ecology" realm. We have classification systems for economic modes, social organization, kinship, cultural complexity, and so on. But in relating psychopathology to these, we generate a rather frightening plethora of "intervening variables"; conspicuously, we lack parametric control. For example, in face of the often-demonstrated fact that the prevalence rates of major psychiatric disorders are quite constant across cultures, we find ourselves invoking such constructs as "individual stress" or "personality" to explain differences between the subgroups within a culture. These may be just the right middle-level constructs—they may pay off. But we will not know until they can be anchored objectively and measured either in the causal or consequential realm. If, indeed, such constructs can be objectified, we may find that the more molar taxonomies no longer serve our purpose.
Dohrenwends lead one to suppose that at least transient, if not permanent, psychopathology is highly related to the stresses and strains of the environment. Perhaps persistent noxious stress can even lead to permanent psychopathology.

Until the day arrives when we have these variables under control, we may have to continue to resort to the blunderbuss of interviewing and observation as our basic approach to uncovering the aetiology of the disorder. The difficulties of the interview technique in aetiological investigations stem from the fact that with it we cannot separate causal agents from their effects, because often the two interact in the response of the patient. We cannot distinguish antecedent from consequent nor can we distinguish distorted from non-distorted reporting. This tendency to lean on the interview for determining both the causal factors underlying the disorder as well as the response of the patient to the disorder leads to a circularity which at the present time seems unavoidable. However, we can at least objectify the interview and make it more comparable from patient to patient and interviewer to interviewer; furthermore, we can provide specific interviews with informants (e.g., "significant others") for specifying the particular environment and its potential assets and liabilities for adjustment by the patient.

In summarizing the ecological model, we might point out that the current revolution in management in psychopathology with regard to hopefulness of treatment, reduction of patient population, rehabilitation, etc., is to a considerable extent a social, cultural and physical change in-
volving changes of attitude on the part of patient, family, therapist, social planners, and even taxpayers. Hence, though the role of environmental forces in aetiology of some disorders is not entirely clear, they are of great importance in detection, treatment and rehabilitation.

2. The Developmental Model

The developmental model of aetiology is built on the assumption that mental disease develops as a result of some specific deprivation or interference during a critical period in development, when the resulting deficit is crucial. Identification of the critical periods of development is still moot, with research covering the entire ontogenetic range: foetal and neonatal periods, childhood, adolescence, adulthood, middle age, and old age. Moreover, the values of the variables that may affect behavior at the critical junctures are still to be specified. At present, such obvious factors as toxemia during the gestation period, restricted early experience, limited peer interaction during early childhood, deviant friendship patterns during adolescence, poor psychosexual adjustment patterns, poor vocational adjustment patterns, unsatisfactory role development in family, vocation, and society, and social isolation in old age can be tabulated as important potential causal agents. How to measure degree of toxemia, extent of peer relationship, pattern of friendship, etc., is still beyond us. Even the categorization of family interactions in terms of degree of relationship between its members shows no universal agreement, as shown by the diversity that exists between Bateson’s, Lidz’s, and Wynne’s nomenclature.

Since the developmental model represents the unfolding of the individual, it seems that in this model the nexus between personality and psychopathology might be found. In tracing back toward this nexus, we are really entering into the anamnesis or history of the individual, and this aspect of the investigation of psychopathology is one of those most urgently in need of further research and instrumentation, as is prospective research in this area.

If we define personality as the systematic aspect of a person’s behavior, and psychopathology as those aspects of his systematic behavior attributable to illness, an important question arises regarding the possible connections between premorbid personality and psychopathology. Elsewhere I have discussed these three possibilities: that personality and psychopathology are one and the same and no
distinction can be made between them, even in the premorbid stage; that psychopathology represents an interaction between the premorbid personality and the noxious aspects of the illness; or that they are independent of each other—i.e., anyone can become mentally ill regardless of his premorbid personality. Until recently, the literature gave no definitive preference for any one of these three alternatives, but a recent series of studies using Sjöbring's method of personality evaluation has thrown its weight definitely on the side of the independence hypothesis.

Sjöbring's system postulates that individuals are either normal or suffer from some underlying neurophysiological "lesion." The lesion cases are the result of damage due to noxious influences—single-gene substitutions of a noxious for a wholesome allele, genic mutation, birth injury, accidents, etc. Virtually all serious mental disorders are regarded as lesion-al, though of course even lesion cases have personality characteristics other than those imposed by their lesion. But normal personality can be subdivided into four dimensions (considered genotypic by Sjöbring): capacity, validity, solidity, and stability.

Essen-Möller applied the Sjöbring method in a study of 2550 individuals in "Lundy" outside of Lund, Sweden in 1947, and was able to assess 98.8 per cent of all individuals in the district over 15 years of age. The same population was re-examined by Olle Hagnell ten years later, in 1957.

The principal finding of interest in the Hagnell study, on the relation between personality and psychopathology, is that 1947 Sjöbring ratings did not predict mental illness incidence in the following ten years. That is, 1947 Sjöbring "deviants" in the (sub- and super-) categories of his four dimensions did not become mentally ill in greater proportions than did members of the population as a whole. This does not mean, of course, that those who have become mentally ill will demonstrate "normal" personality in the Sjöbring system. It appears that psychopathology and premorbid personality are distinct and independent things. Whether the premorbid personality has prognostic significance once the disorder strikes remains to be determined. What also needs to be investigated is whether psychopathology, once it has appeared, takes on a coloration that can be related to the personality that pre-existed, or whether the personality can be said, in some sense, to be suspended for the duration of illness.

Though Sjöbring personality deviation does not "predict" mental illness, there is an aspect of the Hagnell 1957 study that suggests that
Sjöbring deviation may predict other illness. Of 22 women who developed cancer between 1947 and 1957, 20 women had been classified in 1947 as “substable.” There was no such relation demonstrated for men with cancer. Moreover, the relation seemed “linear,” in that the observed/expected ratio (when corrected for age by the Weinberg method) for each scale point on the stability dimension was progressively greater as the stability scale descended. However, the n’s were too small at the lower scale values for statistical significance to be demonstrated.

The deviant behavioral responses elicited by developmental factors are difficult to separate from the deviations in the factors themselves. For example, how much of the child’s withdrawing behavior is a natural consequence of the type of friendship patterns he is exposed to, and how much of it is endogenous? Here again, we must have independent measures of parameters of the environment that are still unidentified. Meantime, we can point to some of the behaviors that seem to be direct reflections of good or poor development: linguistic or verbal behavior, comprehensibility of speech, greeting, eating, sleeping, and other types of daily behavior accompanying socialization.

Finally, it should be pointed out that certain kinds of developmental models may be considered as special cases of the ecological model. In a model, for example, that postulates weak family structure—e.g., broken homes—as crucial for the development of psychopathology, what is really being suggested is that childhood is an optimal period for transmitting certain effects from the social-cultural environment to the individual. Such a conceptualization may lead one to consider the role of learning in relation to psychopathology.

3. The Learning Model

The learning or conditioning model postulates that the source of the deviant behavior of the patient is to be sought in his reinforcement history and the current behavior-reinforcement contingencies. Because learning is dependent on innate mechanisms like sensory analyzers and unconditioned responses, it is difficult to separate the learning process from them, but for heuristic purposes we shall assume these underlying mechanisms not to be deviant to begin with and discuss only the maldevelopment due to the learning process itself. (This caveat also holds for the ecological model, but it is of special significance to both the developmental and learning models.)

It seems reasonable to assume that many behavior deviations,
especially in the neuroses and other non-organic conditions, must be acquired in accordance with known learning principles or with those which are still to be discovered. The learning procedures, intentional or unintentional, that our culture utilizes in shaping behavior are gradually becoming known. Some of the basic principles (such as schedules of reinforcement) have been studied extensively in animals and are beginning to be applied to human beings, especially in the area of verbal and other social behavior. The acquisition of adaptive emotional responses is still largely to be investigated in humans. In general, the parameters of the learning process itself are slowly revealing themselves, since the products of such learning, whatever the process may consist of, are easily observable and often measurable.

Some of the aspects of the learning situation likely to result in maladaptive behavior are those due to intrafamilial learning, as postulated, for example, by Bateson’s double-bind model, in which the mother’s ambivalence in her relationship to her offspring produces ambivalent behavior and other types of deviation in him that we recognize as schizophrenic. While Bateson’s double-bind model has aroused considerable interest in psychodynamic circles, it has thus far defied experimental testing of any of its hypotheses. Other more experimentally based models have been provided by psychologists. Thus, Sarnoff Mednick bases his approach on the evidence that the early or acute schizophrenic conditions more quickly and shows greater stimulus generalization (less steep gradients). These are related to the higher level of arousal or drive that is attributed to an “innate” factor in early schizophrenia. Alternately, in studying schizophrenic behavior, one can postulate, as Kurt Salzinger has recently done, the interaction of learning processes with an “innate” tendency to respond to immediate, rather than remote, stimuli.

The physical basis of learning also may cast light on deviational possibilities. The identification of biochemical processes involved in consolidation of learning and the finding that certain stimulants injected after a learning episode can exert a retrograde facilitation effect that shows up after the drug has worn off have potentialities for perhaps reducing the difference between learning by retardates and by normals. Similarly, the role of attention (perhaps conceived of as involving nervous-system “efficiency”) in learning of both retardates and schizophrenics has been investigated; according to Zeaman and House one reason why retardates of certain levels fail to learn quickly is the
long trial-and-error period before they select the proper stimulus to attend to.\textsuperscript{21}

Further illustrating the possibility of interaction between models, a study by Salzinger and colleagues demonstrated that the administration of chlorpromazine affected only that class of behavior that was being reinforced.\textsuperscript{18} When verbal behavior in general was being reinforced, it was emitted at a lower rate than when no drug was administered. On the other hand, when self-referred affect statements were being reinforced, only these showed a lower rate of emission, speech remaining unchanged. Finally, when movement in the subject's chair was measured (another class not under the reinforcement contingency), it showed, if anything, a higher rate owing to the administration of the tranquilizer. It was therefore concluded (based on a \( n \) of only 4 subjects) that the effect of the drug was not directly upon the behavior, but was indirect, perhaps through some aspect of the reinforcement process.

Another consideration in the aetiology of deviant behavior is the role played by the original stimulus in producing the deviant behavior—the role of traumatic events, for example, as distinct from the factors maintaining the behavior long after the effect of the initial stimulus has disappeared. The contingencies of reinforcement for specific deviant behavior may serve to maintain it, whether the reinforcement is intended or not.

With regard to measuring the deviation in behavior referrable to learning, the entire gamut of patient behavior is involved; much of it can be observed in the clinic and hospital, and some of it measured under laboratory situations. Here, observational techniques and interviewing under individual or group conditions are available, but standard procedures for the assessment of degree of psychopathology in relation to learning principles are only beginning to be provided on a practical basis.\textsuperscript{12}

It might be pointed out here, that learned behavior, as a basis for detection, diagnosis and elimination of psychopathology, has received a new impetus from some of the successes reported for behavior therapy. No one can deny that, at least at the present time, there is no other way to detect the presence of functional psychopathology except through overt behavior, verbal or non-verbal. But it must also be realized that the same behavior may receive a positive reinforcement in one culture, a negative valence in another, and be completely ignored in a third. This fact may lead us to adopt either a purely relativistic view on mental
disorders or to search for other indicators which may accompany or underlie the pathology. The learning theorists for the most part object to this, saying that the behavior and its functional relationship to the environment is the psychopathology and nothing else is needed. Yet, if we discover that a neonate is incapable of some metabolic process (say metabolism of phenylalanine) and neglect it because the neonate is not demonstrating any pathological behavior, we may lose the opportunity of saving him from mental retardation later. It is in this sense that we should be critical of the statement that we need to pay attention only to current behavior—unless we wish to include all activity of the organism, even the cellular or the segmental, as behavior. In this way, a thorough survey of the various response systems of patients, other than their overt behavior, may permit the detection and diagnosis of even latent conditions that have not yet come to fruition, in addition to providing a more objective indicator of the presence of the illness.

4. The Genetic Model

The genetic model postulates that the basic origin of mental deviation is an inherited propensity. The genetic origins of some types of mental disorders can be demonstrated in the form of certain inborn errors of metabolism, as in PKU or galactosemia, or can be associated with specific chromosomal anomalies, as in mongolism, or can be inferred from studies of consanguinity ranging from absence of any blood relatedness to monozygocity. Comparing hereditary factors with social-cultural factors, it is clear that we have a better measure in consanguinity than we have in environmental similarity. The relationship between degree of consanguinity and resemblance in IQ is quite linear and positive. The relationship between resemblance in environmental factors and resemblance in IQ is practically zero. But this may be a reflection of the fact that we have good measures of hereditary resemblance but few good measures of environmental resemblance. Indeed, the genetic stimuli that give rise to deviant behavior have been detected and described in much better fashion than the environmental factors or those underlying any of the other models that may account for deviation. Among the genetic factors leading to mental deviation are some identifiable genetic anomalies, such as translocation and non-disjunction as evidenced in Down's Syndrome, mosaics, and specific alleles or combinations of alleles that because of their enzymatic activity interfere with normal cell development and functioning. Some of the other
genetic principles that have been employed in aetiological considerations are polymorphism (or the balance maintained between alternative genic structures in given internal or external environments), penetrance, and expressivity of genes.

Genetics may be viewed in terms of biochemical mechanisms by which the genes serve as precursors for the production of certain enzymes, whose absence (or excess) prevents the organism from prospering. There is, therefore, considerable hope that an investigation of the internal environment of the body may reveal the particular metabolic deficiency or excess that characterizes the patient. A particular error of metabolism may, of course, be inherited or acquired. A considerable amount of effort has been spent in the attempt to relate schizophrenia to metabolic error. Certain fractions of schizophrenic blood have produced metabolic changes and changes in such behaviors as rope-climbing in rats, as well as transitory changes in the psychomotor behavior of normal human subjects. Presumably similar fractions from the blood of normals do not produce such changes.

5. The Internal Environment Model

The specific aspects of the internal environment, such as homeostasis, endocrine balance, acid-base balance, electrolyte metabolism, and other internal mechanisms and circulating fluids are too many to mention, but there is again a need for classifying them into stimulus classes that may be useful in relating them to behavior. Here again, their mediation of factors operating in other models, such as the genetic or the neurophysiological, is important.

6. The Neurophysiological Model

The final model, the neurophysiological or brain-function model, postulates that the psychopathological deviation is dependent upon the malfunctioning of the organ that most directly controls behavior—the brain. The nature of this malfunctioning can only be guessed at, but certain behavioral characteristics have been found in some types of mental disorder that differentiate patients from normals in a way that seems to be independent of ecological factors and that presumably reflects brain function (either innately, by endowment, or in the course of ontogeny). For example, slower recovery of evoked potentials to rapidly succeeding stimuli, and slower reaction time when a stimulus modality shift occurs, have been found. The actual brain substrates or processes underlying
these deviations are still to be discovered. Except for certain "textbook" neurological syndromes, we probably know less about neurophysiological factors for the production of deviant behavior than we know about factors or stimuli in any of the other models (except, perhaps, for the internal-environment model).

In order to test the hypothesis emanating from the six models described previously, we must provide techniques and methods for measuring the hypothesized deviant behavior accounted for by each of the models. We have tentatively divided the models themselves into three groups: (1) the ecological model; (2) the developmental and learning models; and (3) the genetic, internal environment, and brain-function models. For the first, the social-cultural model, culture-dependent interviewing techniques seem currently to be the most satisfactory way of assessing deviant behavior. For the second group, culture-fair techniques are being provided. For the third group, culture-free techniques are being developed.

Time will permit a sampling of the methods and results for only a few of the techniques that have proved useful.

Culture-Dependent and Culture-Fair Techniques

For the social-cultural model we had available the interview technique in its structured form to catch culture-bound deviations in behavior attributable to the influence of social-cultural forces.* For the heredity, internal environment, and brain function models, which are by definition less dependent on exogenous factors, we are seeking instruments that would be culture-free and yet reflect deviations produced by the endogenous factors subsumed by these models. For the developmental and learning-theory models, culture-fair techniques were sought in order to detect behavior that, though reflecting social-cultural factors in a given environment, could nevertheless be translated, their equivalents being

*It is, of course, dangerous to assume that interview methods identify deviations in behavior. Psychiatric and psychological interviews inquire into areas for which we have almost no population norms, and even where we have such norms—e.g., employment or marriage—they are statistical norms for which, for the most part, measures of variation are not available.

Furthermore, as Boas pointed out a long time ago, a category of facts can more easily yield to analysis when the social group in which it is manifested has not elaborated a conscious model to interpret or justify it. Consciously applied norms are by definition very poor, since they are not intended to explain but to perpetuate the phenomena—these inaccurate conscious models get in the way of evaluating the condition of the patient.
found cross-culturally. Examples of behaviors that can be analysed cross-culturally are communicative behavior, greeting behavior, bereavement behavior, etc. Deviation from the norm in such behaviors takes on the local coloration of the particular social-cultural environment, but their functional equivalence can be partially established. (True equivalence must also show that the behaviors are of comparable importance in two or more cultures.)

To describe psychopathology in terms of the ecological model and to test some of the hypotheses emanating from this model (e.g., inverse relation of socio-economic status to incidence of mental illness), the most useful tools are interviewing and observational methods. There are a variety of such techniques available (dyadic interviews, group interviewing, participant observation, stress interviews). There are also a variety of methods for analyzing these techniques (content analysis, scaling) and a variety of multivariate techniques for summarizing and dissecting the results. The most frequently used method, however, is the dyadic interview.

It soon became clear to us that such assessment of patients as mental

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**Mean Symptom Profiles of a Psychiatric Patient as Viewed by Two Groups of Clinicians Who Differed on Diagnosis**

![Graph showing symptom profiles](image)

**Factors:** Inpatient Multidimensional Psychiatric Scale

**Fig. 2.**
status examinations afford could be much improved if more systematic interviewing methods were introduced. To this end a series of structured interviews were constructed, which passed through several revisions and are now available in standard form: the Mental Status Schedule (Spitzer, Burdock and Hardesty), Structured Clinical Interview (Burdock and Hardesty), and Psychiatric Status Schedule (Spitzer, Endicott and Cohen). With the help of these instruments, and factor, cluster, and profile analytic methods, a fairly stable taxonomy of patient behavior began to emerge, reflecting less the behavior of the psychiatrist and more the behavior of the patient.

The usefulness of these systematic structured interviews in determining the basis on which diagnoses are arrived at is demonstrated in Fig. 2, reporting an experiment conducted by Martin Katz. One of our structured interviews was videotaped so that it could be presented to groups of psychiatrists for their clinical judgment and diagnosis. In addition to the over-all diagnosis, the psychiatrists were asked to fill out ratings on an inventory for such factors as excitement, paranoid projection, anxious intropunitiveness, perceptual distortion, motor

![Figure 3](image-url)

**Fig. 3.**
Table 2.—Diagnoses (%) of patients at U.S. and U.K. hospitals by the respective hospital and project staffs (Series 1: ages 35-59).

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disturbances, hostile belligerence, apathy and retardation, grandiose expansiveness, and thinking disorganization. The psychiatrists were all seasoned veterans of psychiatry; nevertheless, of the 35 participants, 14 diagnosed the patient as neurotic and 21 as psychotic. An examination of their ratings revealed, however, that the groups differed significantly only in one respect—the rating on apathy. Those who rated the patient high on apathy diagnosed him as psychotic, while those who rated him low on apathy diagnosed him as neurotic. We are planning an objective approach to the estimation of apathy independently of the interview.

An application of similar interviewing methods to two selected hospitals in the United Kingdom (Netherne) and the United States (Brooklyn) has yielded the following interesting results. In the U.K. there is a preponderance of patients diagnosed as suffering with affective disorders, while in the U.S. there is a preponderance of schizophrenics, according to the diagnoses of the local hospitals respectively as well as according to the diagnoses made by the project staff. (See Table 2 and Fig. 3, in which slightly different n’s are involved.)

On the other hand, while the profiles on the structured interviews differentiated clearly the affective from the schizophrenic groups when the hospital clinicians’ diagnoses in England were used, this difference did not appear as clearly when the hospital clinicians’ diagnoses in the U.S. were used.

In general, an average reliability coefficient of at least .90 was obtained on the individual items of the interview.
A comparison of the profiles on the data gathered on the same patient by means of a structured interview and of an unstructured interview indicates that, for some factors, a greater amount of psychopathology was elicited on the structured interview while, for other factors, a greater amount appeared on the unstructured interview (Fig. 4).

In an informal study conducted by Barry Gurland and Martin Katz, a group of clinical psychologists who viewed a video tape of the structured interview found less pathology than a comparable group of psychologists who viewed the unstructured interview of the same patient (Fig. 5).

Culture-fair techniques which we have used for the developmental model are three in number: (1) delayed auditory feedback for children and adults; (2) friendship pattern evaluation for adolescents; and (3) measure of pre-admission isolation for residents of old-age homes and mental institutions.

The delayed auditory feedback technique was applied to children to determine at what age stuttering begins. This was found to be approximately age 5 to 7. William Goldfarb applied the same technique to schizophrenic children. From his study, it appears that his subjects behaved like normal children two or more years younger, not beginning to be affected until age 8 to 9. In adult schizophrenics, where we expected to find that they would be affected less than normals, we were surprised to find that they were affected more.
With regard to the friendship patterns of adolescent schizophrenics, it was found that even in their premorbid stage their capacity to make friends, hold friends and have intimate contacts was much less than that of a comparable group of normals.

In studies of the residents of old age institutions, an outstanding, consistent factor militating against good adjustment regardless of the type of institution was found to be preadmission isolation. This isolation was not correlated with mental disorders of the senium, but it did seem to lead to maladaptive behavior. Its effects may be reversible with resocialization techniques now being developed and tested.

In the learning area, reinforcement techniques were used to determine whether the verbal behavior of schizophrenics could be influenced. It was found that under reinforcement patients produced more affective utterances but, unlike the normal controls, failed to continue for as long a time to emit affective utterances when reinforcement was eliminated during the extinction period. The patients who showed greater effects of reinforcement had better prognoses. When the communicability of patient speech was examined with the cloze technique, it was found that it is more difficult to fill in gaps in a patient’s speech than in the speech of

\[
\begin{array}{ccc}
\text{Unstr.} & 6 & 6 & 9 \\
\text{Str.} & 8 & 2 & 4 \\
\end{array}
\]

\[
\text{----- Unstructured} \\
\text{Structured}
\]

**FIG. 5.**
a normal and, furthermore, that the lower the intelligibility the longer the stay in the hospital.

Culture-Free Techniques

The rest of the chapter will be devoted to the culture-free techniques. In surveying the available experimental approaches to measuring patients' behavior that may be culture-free, we decided to limit ourselves to the responses of patients to controlled stimulation during the first 1000 milliseconds following stimulation, under the assumption that the response follows so quickly after the stimulus that culture cannot directly modulate it. Such responses may be designated as either physiological, sensory, perceptual, psychomotor, or conceptual.*

*The time epoch of 1000 milliseconds is obviously arbitrary, and there is nothing magical about this figure that can prevent culture from getting its licks in, so to speak. Two points might be made in this connection. One is that care must be taken to distinguish between primary physiological or sensory responses, of the sort that can be recorded directly by, for example, electrophysiological techniques (e.g., a single evoked potential, or a heart cycle) and a class of response that may be called reporting responses (direct responses or responses mediated through voluntary muscles). In the latter case, while the input of energy stimuli may be equally well controlled as in the former case, one may have to depend, for example, on a verbal report of flicker or apparent movement or on the pressing of a button whose reliability and latency may depend on factors not controlled by stimulus input in a given trial. In these cases, the validity of the measure must rest on experimental demonstrations that motivation is constant, or that a public language is being used, or that the use of a warning stimulus and a variable foreperiod drives reaction time down to an irreducible level. The correlation between primary or physiological responses and reporting responses is a persistent problem area, and one to which detection theory has made important contributions.

The other, more general, point to be made is that no measure can really be said to be culture-free, in that the subject always has a cultural history. No one disagrees that the nervous system is plastic, that even single cells can be "conditioned" (though perhaps in a rather special sense), that physiological levels can be shifted over time, that habituation occurs in sensory systems, that even the simplest "involuntary" reflexes can be facilitated or inhibited by complexly mediated means. As my anthropologist colleague, Dr. Muriel Hammer, points out, the fact that social reinforcement can affect the shape of the cortical evoked waveform, as Grey Walter and Samuel Sutton have reported, forces one to be cautious about "culture-free" measures. However, note that exactly the same considerations apply to psychophysical research. In both areas, the situation may be clarified by observing that, when experiments are properly done, these uncontrolled factors are presumably fairly constant and do not enter unpredictably or selectively into the data. That is, for example, the subjects' experience with the stimuli is approximately equal, motivation does not vary significantly trial by trial, the instructions as to the response or to the reception of the stimulus are standard and sufficient, different experimental conditions are counterbalanced, and so on.
One of the perennial findings in the field of schizophrenia is the retardation in reaction time that characterizes several types of schizophrenics. Impressed as we are with the finding, we have never felt comfortable with it, since it is relatively easy to find differences between patients and normals when motivation cannot be suitably controlled. In our own work we circumvented the problem of motivation by randomizing it—i.e., comparing the reaction time in the same patient under certain types of situations which do not alter his motivation. We found that the reaction time to ipsimodal sequences (where sound followed sound or light followed light) was faster than when a modality switch occurred.

Our first thought was that the reason for this delay in the schizophrenic was due to the fact that he developed certain expectancies, so that having just responded to a light he would expect another light, but the coming of a sound instead slowed him up. In order to eliminate this possibility, we told him what to expect each time and had him verbalize the expectation. The effect persisted. The patients exhibited greater retardation than normals in modality switches even when the basic level of response was equated by covariance techniques.

In a similar situation Dr. Harrington Gosling, one of our staff members, compared the effect of long and short preparatory periods on reaction time of mental retardates and normals. In general, the reaction time of retardates is always longer. However, while both groups respond more slowly when the preparatory interval is short, this effect is much greater for the retarded group. He also examined the effect on reaction time of the relative length of preparatory intervals in two successive trials (Figure 6). Regarding the second trial as the target trial, he found in both of the contrasted groups that when the preparatory interval to the first trial is shorter than the preparatory interval for the second or target trial, reaction time is unaffected by the relative duration of the two preparatory intervals. However, when the preparatory interval for the target trial is shorter than the preparatory interval of the immediately preceding trial, reaction time for the target trial increases as the difference between the preparatory intervals increases. Here again, the effect is greater for the retardates—i.e., their reaction time is more impaired than that of normals by longer preparatory intervals in the preceding trial. This finding, while also not accounted for by motivational variables, is consistent with an expectancy explanation. If one postulated that there is a tendency for subjects to assume that successive preparatory intervals will be of the same length, then a short
preparatory interval coming after a long preparatory interval comes as a surprise and reaction time will be longer. The inverse order, however, would not disrupt reaction time, since with a longer interval in the target trial there is time to recover from the surprise.

In pupillographic research, we have established that:

a) There is a smaller initial diameter in the patient group. While this is a small effect, it has been found reliably in two studies.

b) There is less contraction of the pupil to light in the patient group. This is a large effect, which dramatically differentiates patients from normals. However, this effect has been prominent only in the most recent study.

On the basis of our knowledge of the pupil's neural pathways, it may be said that finding a is consistent with the interpretation of reduced sympathetic activity (smaller initial diameter). Finding b is consistent with either increased sympathetic activity (which contradicts the first statement) or reduced parasympathetic reactivity to light in the patient sample, or is due to a possible artifact. The source of the possible artifact is the report of corneal opacities due to phenothiazine intake by the patient. The results based on our sample, which was matched for initial diameters, do not support the interpretation of reduced sympathetic activity, and in fact they are consistent with either reduced parasympathetic reactivity or the artifact hypothesis.

For critical flicker fusion, it has been recently shown by investigators at the New York Psychiatric Institute that this technique is tapping the criterion that the subject utilizes in making a decision (a conceptual or motivational process) rather than his perception. While differences in critical flicker fusion are to be found between schizophrenics and normals when tested with the classical psychophysical methods of limits and constant stimuli, the differences disappear when a temporal forced-choice psychophysical method is used. (In the latter method, the subject merely states which of several stimuli is different from the other in the set.)

We have been carrying out similar specifications and analyses with our other measures. We have, for example, been working with measures of binaural interaction and visual temporal integration and resolution. We have little to report as yet with respect to the functioning of mental patients on our more refined measures. Our preliminary findings suggest that when adequate care is taken to reduce or control the role of attitudinal, motivational, and criterion variables, and when the task that is
constructed is based on close experimental analysis of the perceptual situation, the differences found between patients and normals are usually small. The large differences to be found between patients and normals are evidently results of factors other than sensory or perceptual ones. That the residual differences are small does not disturb us, except in the sense that we have to exercise infinite care with our equipment and procedures to prevent experimental artifacts from swamping small differences between groups. If we can establish such findings and be sure of their accuracy, we would have a sound basis for moving to theoretical formulations that might open the door to designing measures that would yield large differences between patients and normals. Of course, we may find instead that under these conditions of testing the differences between patients and normals will vanish altogether. Findings of this kind would lead us to give up the search on this level of organization, and we would concentrate on those variables—e.g., motivational, attentional, attitudinal, effects of prior reinforcement history—that we have striven so hard to exclude as factors in our perceptual experiments.

![Graph showing reaction times for PPI<P1 and PPI>P1 with data points for Retarded and Controls.](image)

**Fig. 6.**
One may ask, what profit is there in investigating these partial and small-scale functions or their patterning in the mental disorders? What possible advantage can they offer for understanding the total disorder? Would it not be better to study the totality of the behavior—the total disorder? Our answer is that the total disorder is so encrusted with so much varying life experience that it defies analysis; all one can do is describe it. We have had 34 centuries of such descriptions, dating back to the Hindu Ayure-Veda text (The Caraka Samhita), but understanding still eludes us. By dealing with behavior that is relatively free of prior experience and cultural influence, as we do when we limit ourselves to these brief laboratory tasks, we are attempting to obtain differentials between schizophrenics and normals that reflect a basic substrate of brain functioning in the processing of information. If the difference between schizophrenia and normality can be demonstrated to begin at the information-processing level, for example, we may be able eventually to understand why the world looks so different to the schizophrenic, and yet demonstrate that his behavior is consistent and predictable, though systematically deviant from normal expectancy in some basic substrate. Such differences may yield the culture-free indicators we so desperately need when studying subcultural groups or when making cross-cultural comparisons. By depending on interviewing methods or clinical tests alone, or on techniques highly dependent on prior reinforcement history, such as perceptual constancy or higher mental functions, we cannot escape the cultural bias inherent in such techniques. It is hoped that culture-free indicators may help us detect deviations that would be either falsely occluded or spuriously introduced when culture-dependent techniques are used.

Conclusions

In reviewing these 17 years of work it becomes clear that we must draw several important distinctions in its evaluation. We must first realize that in our search for aetiology we must not neglect or belittle the discovery of indicators that may not be causal in themselves but that may help detect deviation. Thus, we still do not know whether the presence of excess phenylalanine is the cause of PKU, but it certainly serves as a good indicator. Similarly, retardation in cross-modal reaction time or reduction in size of the pupil are probably not causes of schizophrenia but, again, they may prove to be indicators of a condition
that has few if any objective indicators for its detection or for judging its severity or course.

If these objective indicators can be firmly established, there is hope that we can at the very least obtain more homogeneous groups of patients for testing the various aetiological hypothesis emanating from our models.

Work with aetiological models is only in its infancy, yet the weight of the evidence today may serve the purpose of rank-ordering the importance of each model in a given disorder. But it must be admitted that we are richer in models than we are in data.

Claude Lévi-Strauss, in his book *Structural Anthropology*, analyzes the search for a model or models in anthropology in a way that can serve as an epilogue to our own endeavor. He quotes von Neuman as saying: "An almost exact theory of a gas containing about $10^{25}$ freely-moving particles is incomparably easier than that of our solar system, made up of 9 major bodies." Psychopathology is presumably not at the level of unorganized complexity of gas theory, but it surely involves more variables than nine. In fact, we may regard it as a state of organized complexity to which neither Newtonian mechanics nor random statistical properties apply. However, we have a parallel, as Claude Lévi-Strauss points out, in linguistic analysis, which consists of a few thousand morphemes out of which significant regularities in phoneme frequencies and interaction can be obtained by limited counts. Psychopathology may be no more complex than language, but linguistic behavior has the advantage of being recordable with great fidelity and reliability. Thus, in our approach toward a scientific psychopathology, the ground seems to fail where it was expected to be firmest: the facts themselves are lacking, either not numerous enough or not collected under conditions insuring their comparability. Paraphrasing Lévi-Strauss: "Though it is not our fault, we have been behaving like amateur botanists, haphazardly picking up heterogeneous specimens, which were further distorted and mutilated by preservation in our herbarium. And we are, all of a sudden, confronted with the need of ordering complete series, ascertaining original shades, and measuring minute parts which have either shrunk or been lost. When we come to realize not only what should be done but also what we should be in a position to do, and when we make at the same time an inventory of our material, we cannot help feeling in a disheartened mood. It looks almost as if cosmic physics were asked to work with Babylonian observations."
The celestial bodies are still there, but unfortunately the... cultures from which we used to gather our data [for example, in our field, hospitals] are rapidly changing and that which they are being replaced by can only furnish data of a very different type. To adjust our techniques of observation to a theoretical framework which is far more advanced is a paradoxical situation, quite opposite to that which has prevailed in the history of sciences. Nevertheless, such is the challenge...."

A review of the last 17 years of Biometrics Research indicates that though we have made some progress, the road ahead is still a long one. We have provided specific culture-bound, culture-fair, and culture-free techniques to assess deviant behavior. Structured interviews, verbal conditioning and perceptual, psychomotor, and neurophysiological techniques have been and are being adapted to assess psychopathology. We have focused on the need for specifying not only the response, as is done by the above-mentioned technique, but also the stimuli or etiological agents which elicit the psychopathology. Thus, the parameters of the ecological forces which are associated with psychopathology need to be delineated and specified. It is not enough to speak of poverty, deprivation, low socio-economic status, migration, stress, etc. We must contrive methods for demonstrating how these factors impinge on the mental health of the person if we are not to merely join the ranks of the do-gooders and claim that elimination of these hazards will eliminate mental disorder. We must similarly analyze the parameters of the developmental model, the learning theory model, the internal environment model, and the neurophysiological model before we can develop a true understanding of the aetiology of any of the mental disorders. Sheer intervention may produce improvement, but it will be short-lived or will soon exhaust its benefit if we do not analyze the essential components involved.

REFERENCES


