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Chapter 7: Schizotaxia as an Open Concept

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Schizotaxia as an Open Concept

Paul E. Meehl

AUTOBIOGRAPHICAL PROLOGUE

Born in Minneapolis (1920), an only child of affectionate and proud middle-class parents, I had a relatively happy childhood that was traumatically interrupted at age 11 by my father's suicide. At age 14, suffering considerable psychic distress, I read Menninger's *The Human Mind,* which had a tremendous healing influence and changed my vocational goal from lawyer to psychotherapist. Entering the University of Minnesota the year Murray's classic *Explorations* was published [1938], I found myself majoring in a psychology department that was strongly behavioristic, quantitative, "tough-minded, "and rather anti-Freudian. Coming to the field via my high school psychoanalytic reading, this academic environment forced me to think hard and long about methodological questions that most psychologists take for granted. My book on prediction (1954) was a good book partly because of the cognitive dissonance history behind it. Despite the overall flavor of the Minnesota department during my BA and PhD student days (1938-1945), chairman Richard M. Elliott urged us to read Allport and Murray, and a young instructor, Robert E. Harris, "spoke Murray-ese" in characterizing personalities. To this day I find it natural to think of my students, colleagues, and patients in terms of the Murray needs. (I once surprised a sociologist patient by interpreting his behavior as excessively controlled by *n Counteraction*.)

While my early research was on latent learning in rats and on the MMPI, I retained my teenage psychodynamic interest and so took the trouble to have an analysis (85 hours with a Vienna-trained Freudian and 300 with a Radovian), then did a couple of controls and attended a continuous case seminar. My supervisor introduced me to Sandor Rado's little known writings on schizotypy, and about the same time the genetic evidence was becoming impressive,

I was treating schizotypal and neurotic patients and was struck by the differences between them. I had as a graduate student read Rosanoff's early work on twins and had imbibed broadly "hereditarian" views from Donald G. Paterson and comparative psychologist William T. Heron. I concluded, partly from the quantitative research but also, some think strangely, from my therapeutic experience, that schizophrenia was a hereditary neurological disease, a view that shocked most psychologists when I propounded it in my American Psychological Association (APA) presidential address (1962b). I am gratified to see that this heretical conjecture has now become a commonplace among informed persons.

From 1955 to 1970 I did a lot of empirical research on this theory, almost all unpublished, because I realized it didn't really *prove* much of anything. I thought—and still think—that what we needed to test a dominant gene theory of a pervasive schizotaxic defect were (1) better indicators, closer to the DNA than to psychometrics or social behavior, such as psychophysiology and soft neurology, and (2) mathematically stronger taxometrics for combining indicators. So I have devoted recent years to the invention of several such taxometric methods, which I conjecture are better than most of the conventional cluster algorithms. All of my work has been influenced by contact with philosophers of science in the Minnesota Center for Philosophy of Science, of which I was a cofounder (with Herbert Fiegl and Wilfrid Sellars) in 1953. Much of the sequel reflects that intellectual influence. I have sometimes regretted becoming a clinical psychologist, because the field's scientific quality is so poor compared, say, to genetics or statistics. But in recent years I have found it possible to work on problems of psychopathology along a mixture of genetic and mathematical lines, which for a person of my tastes and talents is not a bad compromise. By and large, I am having fun at it, and—as my parents taught me by precept and example—if you're not having fun, you might just as well be dead. By far the largest part of my adult behavior has been controlled by a fusion of *n Cognizance* and *n Play,* with assists from *n Recognition* and *n Autonomy.* Readers interested in more details may consult my autobiography (Meehl, 1989).

THE DIAGNOSTIC PROBLEM: TWO CLINICAL CASES

To motivate a discussion that will be largely methodological, I begin with two brief clinical examples. These were patients seen in the Tuesday morning Grand Rounds in the department of psychiatry, a year or so ago, and I write from memory without having gone back to study their charts. As you will see from how I use them, it is not important whether my recollections are entirely accurate, since my discussion is about a loose and debatable syndrome with considerable sign and symptom substitutability. The first patient is a 40-year-

old male who presented with complaints of mild confusion, "a dizziness in the head," and inability to work because of "trouble with the hands and feet." On admission the examining resident recorded cataleptic-like partial paralysis of all four extremities, minimal clouding, and anxiety normal for the symptoms. The paralyses disappeared for some hours, then reappeared for a couple of hours and were not clearly in evidence when see in the case conference, although there was a hint of waxiness manifested as slow drop. The patient walked with a somewhat retarded gait, and there was a little stiffness when the examiner handled his arms. There was nothing diagnostic about his interview behavior other than a couple of odd semantic choices, noted by some of us but not by others. Unfortunately, the patient had completed no psychometrics and was subsequently discharged without having done so. I included "atypical catatonic schizophrenia "in my differential diagnosis, but only two other clinicians present agreed with me that this should even be considered. The appropriate affect in the interview and the absence of any clear-cut thought disorder (despite the ± odd semantics that some observers didn't agree were particularly odd) and a previous history of adequate work and social adjust–ment made the schizophrenia possibility seem reaching far out in left field to most of those present.

In the second case I again found myself in the minority (this time a somewhat larger one), the patient being a 30-year-old female who presented with complaints of anxiety and depression and who had for some years lived partly in a childlike, dependent way upon her parents and since then alone on relief because she was unable to hold a job and also was almost phobic about leaving her apartment. The history was of a chronically withdrawn, listless, marginally functional introverted person without any psychotic signs either in the history or when seen. She had been in therapy with several practitioners, one of whom was present at the conference and reported that she was a very difficult patient to deal with as she elicited many countertransference problems on his part. While he didn't know how to diagnose her, he was convinced that she was a hard case with a poor outlook for change. Several of us were in-clined to such diagnoses as "borderline" (Kroll et al., 1981) or "Hoch-Polatin Syndrome," a suggestion that engendered a heated debate about whether there was such an entity, and if so, what did it have to do with schizophrenia? There were claims and counterclaims from our fallible memories as to what the follow-up data showed on the original cases described in Hoch and Polatin's classic article (Hoch, Cattell, Strahl, & Pennes, 1962; Hoch & Polatin, 1949). Those who recalled (correctly) that 40% were subsequently hospitalized (one to nine times) and 20% developed a florid schizophrenia construed this as supporting the entity's reality. Opponents urged that this "*only* one in five" figure tended to cast doubt on Hoch and Polatin's concept.

This second patient's mother had at one time been hospitalized in a state hospital for several months, where she had received shock treatment, but—to

my chagrin, because it happens constantly in case conferences—the presenting resident had not taken the trouble to find out what the mother's diagnosis was. Here also we obtained no psychometrics, and I have chosen both of my examples partly for this reason. The question is not whether the minority clinicians were right in thinking that these two patients might have been schizophrenes but to illustrate a ubiquitous problem in understanding the concept of psychiatric diagnosis. The conference majority was right in arguing that neither of these patients showed much *descriptive* resemblance to the paradigm patients labeled "schizophrenic" by the great Eugen Bleuler (1911/1950) or much less the "textbook" syndrome delineated by the equally great but (in some circles) much maligned Emil Kraepelin (1909-1913/1971). Aside from the diagnostic merits, especially because (as so often happens) there was no elicitation of sufficient additional material, either historical or psychometric, to warrant pursuing the discussion, what were we disputing about? In order to know what additional evidence we would need, and why, one must first ask what the argument was about, what the issue was.

Some would say that such a dispute is merely semantic, a matter of words, a view that I wish to combat vigorously as being a kind of pseudosophisticated cop-out common in social science. I wish I could devote a whole chapter to this one mistake, but I confine myself to expressing with Sir Karl Popper (1959) my agreement with Immanuel Kant, who said, "I for my part hold the very opposite opinion, and I assert that whenever a dispute has raged for any length of time… there was, at the bottom of it, never a problem about mere words, but always a genuine problem about things." There are, of course, pointless semantic disputes in social science, but this is not one of them. There are two main goals involved here. The practitioner wants a diagnosis because he believes, perhaps wrongly, that having it will help him deal more effectively with the patient's problem, to make a prognosis and to select treatment. The other goal is that of the theoretician. For example, a clinician doing research in the behavior genetics of schizophrenia is asking a theoretical question that comes down, ultimately, to a question of causality. With regard to prognosis and treatment it is not immediately obvious that the introduction of the diagnostic rubric "Hoch-Polatin Syndrome" will be helpful even if the entity does exist, first, because practitioners who use this concept usually report that the drug treatments of choice for florid schizophrenia (phenolthiazines) are not effective with this syndrome, a clinical impression that fits the growing theoretical conviction among psychopharmacologists that these drugs are not antischizophrenic drugs but antipsychotic drugs. Second, there is the fact that the psychophysiology and soft neurology of schizophrenia do not disappear in drug-induced remissions. The prognostic implications for a patient who is clearly unlike Kraepelin's *dementia praecox* cannot be directly transferred to such an atypical form.

Furthermore, it is not known to what extent predictions, including "treat-

ment of choice" predictions, are better mediated by the introduction of such a rubric than by straight statistical prediction from the relevant diagnostic data, that is, by bypassing the nosological taxon. It is interesting that psychiatrists tend to take this mediation for granted, as do psychologists sympathetic to the categories of DSM-III, despite the absence of either mathematical or empirical proof to that effect. My colleague and former student Will Grove has shown in recent mathematical and Monte Carlo work that it is only under certain fairly extreme conditions of taxonic validity and low symptom correlation that the introduction of this mediating category can be expected to improve upon a straight statistical prediction from the evidentiary base.

THE METACONCEPT "TAXONICITY"

A more general question that one might expect to have been settled long since, philosophically and mathematically, but that I find is not, is what do we *mean* when we postulate a taxon (disease entity, type, species, "natural kind") as a way to conceptualize the relationship between a set of signs, symptoms, life history facts, and test scores? What does it mean to say of a data domain in psychopathology that the latent structure is taxonic rather than factorial? I believe that bipolar affective illness and schizophrenia are true taxa, whereas garden-variety borderline intelligence and high social introversion are not. The latter being dimensional in nature, any class words or categories we might use, as when in ordinary speech we use the category term "introvert," are merely convenient demarcations of a continuous region in the descriptor space. It turns out that to construct a definition of "taxonicity" is a good deal harder task than one might have supposed.

In my seminars in philosophical psychology and classification in psychopathology, we have to deal with the metaquestion, what *is* taxonicity? When is an alleged taxon really a taxon, rather than an arbitrary class, perhaps even one that the classifier himself treats as somehow "nonreal"? Students want to be given a clear idea of what a taxon is before leaping into the mathematics, or even considering the general methodology of how one goes about searching for taxa. This is particularly true of students who have been indoctrinated with a philosophy of science that is at least a half century out of date! While it is true in some sense that definitions, being stipulations about the use of language, are "conventional," it is not true that in any respectable science they are "arbitrary." As Carnap pointed out in his paper on the probability concept (Carnap, 1945), one should perhaps use the term "explication " for the kind of definition that occurs in empirical science. The stipulation about the choice of a *term* is a rather minor and uninteresting aspect of the conceptual problem of definition, that is, the formulation of a *concept* that will be theoretically powerful rather than useless or counterproductive. I have found it pedagogically more effective to go directly to a consideration of empirical examples

from fields like medicine and genetics, sometimes with but often without the taxometric math, explaining only preliminarily that the metaquestion, what is a taxon? will be clarified as we proceed. This is sometimes referred to by logicians as an implicit or contextual definition, rather than an explicit one in the old-fashioned sense. I hope this does not sound obscurantist—definitely not my intellectual style. I will therefore do here briefly the same thing I do to satisfy the inquiring students, where the incomplete satisfaction may lead you to reflect further on the taxonicity concept and perhaps tempt those with mathematical expertise to make contributions that are sorely needed.

There are roughly three ways of explicating the open concept "taxonicity." They are not mutually incompatible, but their precise relationships remain unclear. The first way proceeds by offering rough synonyms and familiar examples, and this sometimes turns out to be adequate as a basis for developing the statistics of a taxometric search method. A taxon is a class, that is, a collection of individuals (stones, honeybees, mental patients, daffodils) that is—in some sense to be explained—not arbitrary. It is often convenient for ease of communication and administrative purposes, especially where a yes or no pragmatic judgment must be made as to a proposed action, to demarcate regions on a continuum or in a continuous descriptor space by a class name, without any intention of claiming that the demarcation is other than arbitrary. ("Arbitrary" here does not mean whimsical or totally unmotivated; it has to do with certain utility considerations.) When we classify some students as "underachievers" or say that a person is "obese," we are aware that this is a category term used for short to refer to some region of a quantitative dimension. So terms like "introverted," "bright," "energetic," "underweight," "clumsy," and "dominant"—in fact, almost all of the trait names in ordinary English, as well as most of them in the technical language of psychopathology—are shorthand for regions on a quantitative dimension or volumes in a descriptor hyperspace.

When we attribute empirical taxonicity to a concept, we have in mind something stronger than that, although it is difficult to say precisely in what that stronger meaning consists. In ordinary language we say that we are intending to refer to a *natural kind,* such as a biological species, a disease entity, a personality type, a Mendelizing mental deficiency syndrome, a "qualitative" as contrasted with a merely "quantitative" difference between groups of individuals. To use an expression from Plato, "We want to carve nature at its joints." One sometimes hears reference to a taxon, species, or disease entity as presenting a question of kind and not of degree, but this locution is somewhat careless; what one should instead say is "a difference of kind *as well as* degree," since obviously some of the indicators of membership in a taxon are themselves matters of degree. (When the point is pressed by careful inspection, they almost always are.) It discourages me to find that even graduate students sometimes offer the cliché objection to a concept like schizophrenia or sociopathy, "How can you talk about schizophrenia as an entity

when there are all degrees of its symptoms and traits?" This illustrates how even bright people can be led to say dumb things by undergraduate miseducation. Meningitis is a disease entity with a characteristic pathology, and nobody familiar with it would deny that it is a genuine categorical entity in a way that obesity or borderline IQ are not entities. Patients with meningitis have fever, headache, clouding, Kernig's sign, Brudzinski's sign, and so forth. Some of these indicators of the meningitis entity are quantitative, others qualitative (although even Kernig's and Brudzinski's signs *could* be considered merely extreme degrees of leg or neck stiffness!). The confusion here is failing to distinguish between the question whether an indicator is quantitative and whether the set of indicators reflects an underlying causal state of affairs that defines a nonarbitrary class. *Whether the fallible indicators of a putative taxon such as a disease entity, personality type, or biological species are singly qualitative or quantitative in character tells us nothing about the presence or absence of a latent taxonic structure.*

Sometimes this rough synonyms-cum-examples explication of taxonicity suffices to get on with the scientific job. But in other contexts—including, alas, psychopathology—it is rarely sufficient. Reflection on the classification of Mendelizing mental deficiencies and disease entities in organic medicine has led me to focus attention on *causal agency* as a criterion. Mendelizing mental deficiencies may be only slightly distinguishable, if at all, by psychometric patterns, and distinguishable with somewhat higher accuracy by certain anatomical correlates in skin, bones, hair, and the like. These pathological taxa are often specified with high precision when we know the biochemical path, sometimes even the chromosomal locus and codon substitution of a mutation. In stressing causation I have no wish to dogmatize by imposing this semantic preference upon others who experience it as narrow. In teaching or discussing research plans with colleagues, I therefore attach the adjective "causal" to the noun "taxon," since it doesn't matter whether you and I prefer the same semantics so long as we understand each other. In organic medicine and genetics, the taxonicity of a disease entity is closely tied to the notion of specific etiology, but unfortunately that notion is not as clear-cut as one might initially suppose. I have elsewhere set out an explication of *strengths of causal influence* (Meehl, 1977), and I find that scholarly clinicians in my environment use the term "specific etiology" for only the first three strongest causal influences and resist using it for weaker ones. (I may add that most physicians, including scholarly ones, have not reflected much on these matters.) Assume we have set up criteria for an identifiable syndrome of indicators (itself a part of the task of taxometrics). Let us refer then simply to "the probability that Individual X develops the disease." We presuppose what Mackie (1965, 1974) calls a "causal field," such as that the individual receives sufficient calories to stay alive to adulthood and similar kinds of things usually not mentioned as part of the equation. Given that (usually unmentioned) causal field, we imag-

ine a causal equation (not a mere statistical prediction function, although if known it would serve that job beautifully) that gives the probability of disease as a function of causal influences. Suppose one of these causal influences is intrinsically dichotomous, that is, a qualitative, present-or-absent factor, such as whether there is or is not a mutated gene at the Huntington locus. If the probability function equals 1 with that factor present and zero with it absent, we could take the factor in question out of the equation and treat it as a multiplier having the two values 1 and zero. Then regardless of the joint values of all the other causal factors that might, for instance, influence the intensity or age of onset or the rapidity of course or the preponderance of certain elements of the syndrome over others, we have a necessary and sufficient qualitative condition. The dominant Huntington mutation is an obvious example.

Suppose we have an intrinsically dichtomous causal factor such that the disease probability is zero if the factor is absent but is not equal to 1 with the factor present (whether disease occurs being dependent upon other factors); we then speak of a *qualitative sine qua non,* of a necessary but not sufficient condition for the disease. Clinicians readily agree that these are both cases of specific etiology.

Finally, suppose there is a causal agent that is not intrinsically dichotomous but quantitative in character, where the probability of disease is a step function of this agent, such that to the left of the step it has the value zero and then to the right of the step it has a non-zero value that may, however, not be flat but continue to rise as a function of the combined action of the other causal variables. Here, although the factor is not intrinsically dichtomous or qualitative in nature, I find most scholarly clinicians of my acquaintance willing to label this as specific etiology.

But any "weaker" influence than that, such as the most powerful single factor (e.g., caloric intake as a factor in obesity), they think should merely be seen as a case of strong influence and not labeled specific etiology. You should not assume from these examples that the notion of specific etiology has to be closely tied to the organic medical model. A specific major life history event would do as well, the only problem there being that it is difficult to show that there are many such. For example, if Freud's early theory (Freud, 1896/1962) about the specific life history events pathogenic for hysteria and obsessional neurosis had been empirically correct, rather than a mistake on his part between historical events and infantile fantasy, they would have been perfectly good examples of specific etiology. You recall he thought that a condition for either of these neuroses was a prepubescent sexual experience involving overt genital friction. If the patient was passive and the affects of fear and disgust predominated, the later consequence would be hysteria; if the patient played an active role, was the seducer, and the affect at the time was mainly one of pleasure, we have the antecedent for an obsessional neurosis. This, he thought, was why hysteria was commoner in females. Now that event was not a germ,

or a gene, or even a mechanical trauma like the popular "being dropped on the head as a baby." But if it had held up empirically, it would be a perfectly good example of specific etiology.

To give a nonmedical example of the taxonicity problem, as an undergraduate I was acquainted with a number of Trotskyists (Minneapolis was one of the centers of the Trotskyist movement in the 1930s and 1940s), and Trotskyism is a very tight syndrome. It is a clearer taxon than many of the taxa of psychopathology, including even the good ones, let alone the rather arbitrary ones sometimes found in DSM-III. I found two signs that were jointly pathognomonic of Trotskyism as a political syndrome. If a person said that (1) the Soviet Union was a workers' state and (2) Stalin was a counter-revolutionary bureaucrat, I could be 100% sure he was a Trotskyist, and a dozen other traits could then be predicted with high confidence. Being a Trotskyist is not due to a germ or a gene, but it's a nice statistical taxon. In some instances, it would not be due to a specific etiology, because one of these events might be reading a certain book; another event might be having married a Trotskyist. Etiology here is not specific, even though each of the candidate causes may have an almost dichotomous character.

In discussing with my colleague Auke Tellegen causal taxonicity as a fruitful concept for analytical purposes and for evaluating taxometric search methods, I have come to use the phrase "Tellegen case" for a puzzling kind of example. Suppose that a federal judge, in his infinite wisdom, decides that the borderline IQ kids in special schools must be mainstreamed in an affluent Minneapolis suburb such as Edina. If one were to administer an omnibus intelligence test to the Edina pupils, given these circumstances, one would get a clear, unmistakable strong taxon from the data, no matter which of the available taxometric methods was employed. You don't need any research to figure that one out from the statistical armchair. But if we are working in the borderline IQ range, avoiding Mendelizing or karyotypic mental deficiencies or developmental anomalies like microcephaly, we are simply dealing with the low end of the polygenic distribution of the heritable component of *g,* plus environmental deprivation effects. So category words like "dull," "normal," or "borderline" are only loose class names for *regions* on the intelligence dimension. Tellegen points out that this is a pseudotaxon, not a true taxon in any interesting sense, causal or otherwise. He goes on to point out that several—perhaps all—taxometric methods would tend to yield this misleading result.

He makes a good point, and the Tellegen case is an important test case to reflect upon in discussing the metatheory of taxometrics. But the example doesn't trouble me as much as it does him. For one thing, suppose we adopt a convention that "interesting" or "real" taxonicity means causal taxonicity and combine that stipulation with the above mathematical relations, so that we require a dichotomous or step function causal agent responsible for producing the taxon. Then, while the Tellegen case is not a true taxon from the stand-

point of a geneticist or developmental psychologist, it is a taxon from the standpoint of a political scientist or jurisprude. In those disciplines, an injunction by a federal court is a powerful causal agent, not a matter of degree; that is, the judge has either issued an injunction regarding educational mainstreaming, or he hasn't. It seems arbitrary to say that we can only identify taxonicity with causality at one level in Comte's famous pyramid of the sciences. For example, to say that Freud's specific etiology of hysteria doesn't count because it's a life history event, and we only count genes or germs, would be quite arbitrary. Higher up in the pyramid of the sciences, we deal with the disciplines of sociology, economics, political theory, and law, and for scholars in those disciplines concepts like "Trotskyism" or "bankruptcy" have a taxonic character.

Second, it is a mistake to require of any statistical method aimed at identifying a substantive—especially causal—state of affairs in the empirical world that should be capable of functioning as a litmus test of theoretical truth or falsity, as a kind of automatic truth-grinding machine for "verifying" causal conjectures. It is perfectionistic to demand that of any taxometric search method. If a taxometric method reveals that 10% of Edina schoolchildren belong to a low IQ taxon in the hypothetical Tellegen case, we remind ourselves that taxometric investigation always involves an interplay between the statistics, which may be a programmed algorithm and in that sense "mechanical," *and all relevant theory and evidence.* There is a temptation—which I do not attribute to Tellegen—for social scientists to treat statistical methods as if they were automated truth-grinding machines. One must consider all relevant aspects of construct validity given by the item content, psychometric, demographic, and life history facts about the individuals, where the sample was obtained from, and whatever general psychological theory and even commonsensical knowledge is available. Our Edina researcher, having identified the 10% low-IQ taxon members, could easily discover a big explanatory fact about them by checking their home addresses, which would (unlike those of the complement class) be outside Edina. No sane investigator would ignore this information or fail to follow it up by inquiry.

A third approach to Tellegen's problem focuses on the mathematics, saying stipulatively, "A set of numerical data is formally, numerically taxonic if and only if they satisfy such and such taxometric equations." In explaining my taxometric methods to graduate students, I begin with a convention: "For the next two hours, we will call a class of patients taxonic if their indicator scores act configurally like Meehl's taxometric bootstrapping methods say they should." Adding a small amount of interpretative text to tie the mathematical machinery to empirical data is an acceptable explication of theoretical concepts, employed in all sciences. But if you do it that way, the question arises, what is the relation between numerical taxonicity (satisfying the postulates of a certain formal calculus) and the interesting substantive

questions about causality, which is what we're up to in much of our psychopathology research?

Considering a set of quantitative fallible taxon indicators, starting the reasoning with the taxon members already specified, it is easy to go in that taxon-to-indicator direction. Good indicators of the taxon will be those measures that when distributed separately for the taxon members and the complement class yield two clearly separated (although overlapping) unimodal frequency curves. Imagining ourselves to have Omniscient Jones's knowledge of the latent situation, we assert, "There is some attribute *A* that is either intrinsically qualitative (yes/no character) or is a quantitative attribute with respect to which the probability of taxon membership is a quasi step function with $p = 0$ to the left of the step. *A* may be a life history event, an inner state or structure, even a manifest disposition. When we partition the population into the two sets *A* and *–A,* then the quantitative indicator X_1 will show two unimodal distributions, as will indicator X_2, and so on for all of the indicators that have taxonic validity."

Going in this direction, it's easy. But when we don't have Omniscient Jones's knowledge and have to start with a set of fallible quantitative indicators, how do we define taxonicity? It won't do to say that taxonicity exists if there is a way to partition the population into two subpopulations satisfying the above distribution criteria, because there will always be a way to do that, in fact a very large number of such ways. The existence of a large number of such possible partitions does not depend upon the existence of a taxon since it can, of course, be achieved in a hyperspace of purely factorial ("dimensional," "noncategory") data, provided the indicators are correlated. All we have to do is divide the group at an arbitrary cut on any valid indicator, and when we distribute the other fallible indicators within the two resulting subpopulations, what we get will "look rather taxonic."

Statisticians writing about the "decomposition problem" bypass this troublesome point by simply referring to the decomposability of a manifest frequency distribution into weighted sums of component distributions. But such a mathematical decomposability will always exist, and in a very large number of ways. I am not relying here on the cardinality of the continuum or other such mathematical nicety. I mean that even if we confine all numbers to integers (for example, we don't express any individual's IQ with decimals), there are not an infinite number but an extremely large number of ways to decompose any well behaved frequency distribution. For example, imagine a smooth curve based upon a billion cases that can be exactly graduated (no frequency "error" in the unit intervals) by a frequency function $f(x)$. Choose an arbitrary taxon rate P lying in the probability interval $0 < P < 1$. Define another arbitrary frequency function $g(x)$. Then define, in terms of these, a function

$$h(x) = \frac{f(x) - Pg(x)}{Q}$$

Then if $f(x)$ and $g(x)$ are well behaved frequency functions, so will $h(x)$ be. Since we can choose $g(x)$ in many ways and still satisfy the conditions for being a frequency function, and we can assign the arbitrary taxon rate P in many ways, we can therefore derive our $h(x)$ in many ways. But from the above equation we have immediately

$$f(x) = Pg(x) + Qh(x)$$

That is, the manifest frequency function $f(x)$ is decomposable. If we restricted the choice of $g(x)$ to, say, Pearson's 12 curve types and carried their parameters out to two decimal places, and the same for the arbitrary P, there are over 10^{11} possible decompositions of the manifest frequency function $f(x)$. So the decomposability of a single indicator variable cannot be a test of latent taxonicity, since the answer to the decomposability question is always affirmative. Of course, we concocted a hokey function $h(x)$ so that it *would* serve as a component, but that's not an objection to my point, since if $g(x)$ is a "plausible" frequency function to start with, there needn't be anything outlandish about $h(x)$. The point is that we have not given a mathematically rigorous criterion of taxonicity when we simply say, "Imagine the manifest frequency function to be decomposable into two functions when the latter are appropriately weighted by taxon and complement frequency constants." In order to give a formal definition of taxonicity in terms of the decomposability criterion, one must impose some additional constraints upon what kinds of functions will be admitted as candidates. We would like to make some sort of link, even if it's only a loose one, between a formal criterion of taxonic structure and a "theoretically interesting" concept of taxonicity (e.g., causality? developmental origin? source?). We would hope to attain partial insight into what it means to be a "true taxon" or "natural kind" by contemplating the properties of the formalism. Could we motivate the choice of a mathematical generating procedure by reference to a suitably broad specification of how natural kinds, so to speak, "come into being"?

It naturally occurs to one that perhaps Karl Pearson's famous but little used family of curve types might serve this heuristic role (Craig, 1936; Cramer, 1946; Elderton & Johnson, 1969; Johnson & Kotz, 1970; Kenney, 1939; Pearson, 1894, 1895; Rietz, 1927; Stigler, 1986). Pearson starts with the

old notion that the probabilities of a number of causal factors, operating positively or negatively and independently of one another, yield on a particular occasion (say, the measurement of a length of a table or the determination of a person's height by his genes) when $p = q$ a frequency polygon $(p + q)^n$ extremely close to the Gaussian function. If we allow an asymmetry between p and q, we get frequency polygons of varying amounts of skewness. Then Pearson further relaxes the conditions for generating the terms of the series for the discrete case by allowing p to vary as we draw, say, marbles from an urn that contains a mix of white and black marbles in a certain proportion. He opts for the obvious setup of a finite urn sampled without replacement, so that the probability of drawing a white marble in a given point in drawing m marbles depends upon what proportion of those left in the urn are white and therefore depends upon where I am in the sequence and how many white marbles I have drawn up to this point in the drawings. As is well known, a finite urn model sampled without replacement yields us a discrete series somewhat different from the binomial, symmetrical, or skewed series, called the hypergeometric series. By considering the slope connecting two points in the polygon and some high school geometry and algebra, Pearson shows that the slope expressing the difference in height of successive terms of the hypergeometric series per unit increment on the abscissa is equal to the height of the midpoint multiplied by a fraction whose numerator contains the distance from the mode of the polygon in the first degree and whose denominator is a quadratic in that distance. Moving to the continuous case from this frequency polygon, he obtains the differential equation

$$dy / dx = y(m - x) / (b_o + b_1 x + b_2 x^2)$$

It turns out that by suitable assignment of the four parameters of this "generalized frequency function," individual differences data from a wide variety of measures in the biological and social sciences can be satisfactorily graduated.

Pearson was mainly interested in heterogeneity and considered that if you could not closely graduate a set of data from the biological or social sciences by appropriate assignment of the four parameters in his generalized frequency function, this would suggest that you had a population mixture of two natural kinds, and you should attempt to decompose the manifest frequency function into two component functions appropriately weighted by the taxon rate. To a psychologist there is another likely source of maldistribution that, so far as I am aware, did not interest Pearson, namely, a systematic bias, when constructing a psychometric device such as a personality inventory, in the distribution of item difficulties on the underlying psychometric factor. It is easy to see this if, for instance, in building a scale of paranoid sensitivity, a clinician was pretty good at inventing items for individual differences in the nonpathological range, of the sort that go with social introversion and the like, and was also expert at describing psychotic distortions but had trouble thinking up item

content in the borderline region (e.g., in early schizophrenia or in a severe neurotic with social phobic distortions not quite amounting to ideas of reference, but close to it). Then if the true distribution of paranoid sensitivity closely fitted one of Pearson's 12 curve types, an inventory used to measure it with poor representation of items in that borderline region would not be satisfactorily fitted by a Pearson curve.

It is well known (Pearson proved it in his original paper) that it is impossible to assign parameters to two Gaussian functions so that their weighted sum will be Gaussian, except for the degenerate case where they have the same mean and standard deviation but merely different sample sizes. I do not have a rigorous proof, but looking at Pearson's equation above, it seems obvious (and my statistician friends agree with this) that a more general statement is that no weighted sum of two Pearsonian functions can be Pearsonian. That interesting mathematical fact suggests a line of reasoning connecting Pearson's curve family with our intuitive idea of a natural kind, and perhaps connected with a dichotomous or step functional causal originator of a curve of individual differences among organisms. If your frequency data cannot be satisfactorily graduated (not merely a significant chi square on frequency discordance, but not even a "close" fit), this suggests that your data have originated in a latently taxonic causal situation. If one can decompose a non-Pearsonian manifest distribution into two Gaussian (or nearly Gaussian) components, this is usually taken as quite impressive corroboration of the taxonic conjecture, although of course it does not constitute a deductive proof. Suppose we find that a decomposition of one fallible indicator's non-Pearsonian distribution into two Pearsonian components is possible, and then we examine a second quantitative indicator and find that we can decompose it into two components (although their Pearson types may not be the same as the first). If the compositional weights assigned are approximately the same for the two indicators, this would constitute a strong corroboration of the taxonic conjecture.

There is a troublesome point here arising from the way in which Pearson derived his function via the hypergeometric series. In the case of the symmetric or asymmetric binomial, it is easy to think of physical circumstances in which multiple causes operate in the way the mathematical model says, and also cases in which the causes are not independent. But the idea of sampling from a finite urn without replacement has, so far as I know, no plausible physical realization in social or biological science. However, you can't go from Pearson's generalized function back to the hypergeometric series. You can only go in the other direction. The movement from the hypergeometric series to the generalized frequency function did not, in Pearson's derivation, consist solely in moving from the discrete to the continuous situation. The discrete case formula for the slope of the polygon segment has an inner structure involving certain required relationships among four numbers (the number of marbles in the urn, the proportion of white marbles, the number of marbles

thus far drawn, and the proportion of those drawn that are white). But when Pearson moves to the continuous case, he pays no attention to those interrelationships. He simply observes that in the discrete setup the variable x (distance from the mode of the frequency polygon) appears in the first degree upstairs and as a quadratic downstairs. In that sense, therefore, the Pearson generalized function is more than a continuous form of the discrete; it is also a generalization freed of certain internal constraints upon the way in which the discrete case coefficients were arrived at from the finite urn model. So Pearson's generalized frequency curve does not require the idea of sampling marbles from an urn without replacement as its "structural" or "generative" basis. The easiest way to see that, other than contemplating the discrete case coefficients, is to learn that Pearson is able to fit a U curve to data that require it, with his generalized function. But that is impossible with the hypergeometric series, because it is what the engineer calls "restorative." That is, if, as you draw your set of marbles, you are running ahead of the game in the proportion of whites, the probability of a white falls below that of the initial urn parameter value, and if you are running behind expectancy in terms of the original urn content, your odds of drawing another white marble go up. That means that at any point of the drawing, there is a restorative tendency pulling you back toward the middle, that is, toward the white marble expectancy of the urn before you started drawing. Therefore, the hypergeometric series has to be "constricted" in comparison with the binomial of the same initial white marble rate. Such a restorative generating process cannot, of course, yield a U-shaped distribution, where the ends have higher probabilities than the middle region.

What one would like would be to find the "most general" way of reaching Pearson's generalized frequency function and then to examine that generative process with an eye to the question what kind of physical generative process, giving rise to individual differences among organisms, would correspond to this mathematical generative rule? I have not done a literature search, but query and correspondence with statisticians lead me to the conclusion that nobody has as yet been interested to attempt this most general investigation. I repeat, the point is not to produce a rigorous deductive argument that any time you can fit data with a curve from the Pearson system you can dismiss taxonicity, and any time you can't you can infer taxonicity. That is a philosophical mistake, whatever the mathematician might make of it. It goes without saying that there is no such thing as an absolutely necessary deductive proof of any substantive theory, especially a theory about causal origins, from a mathematical statement of the observable data. That's just not the way any empirical science works. But it would be interesting to know how the most general setup capable of yielding Pearson's function might be restated in terms of the most general kinds of imagined physical processes or entities we could cook up. From a Popperian point of view—or Wesley Salmon's principle of "Damn Strange Coincidence" (Salmon, personal communication, 1980; 1984)—it

might turn out that such a general, almost metaphysical statement of the generative conditions for a Pearsonian frequency curve of individual differences, the mathematics being interpretable by a very general embedding text (referring in highly abstract ways to unspecified kinds of repeated events occurring together and the like), could corroborate substantive taxonic conjectures by an empirical finding that two or more quantitative indicators (1) are each distributed non-Pearsonian but (2) can each be decomposed into two Pearsonian components (3) having component weights the same (within tolerance) for all the indicators.

In almost any discussion of research strategy or data interpretation, one will hear plausible statements like the following: "You cannot study the genetics of schizophrenia until agreement exists on a *definitive* set of diagnostic signs." "To add a new symptomatic indicator to the list constituting a syndrome, or to justify a shift in the diagnostic weights within the received list, either (1) is an arbitrary redefinition or (2) requires nonsymptomatic criteria to validate it." "To rediagnose a case because its subsequent clinical course disconfirms expectation is an arbitrary [or, 'circular'] act." "To say that 'true schizophrenia' refers to the genetically determined cases and all others are phenocopies is viciously circular." "We cannot assign differential diagnostic weights to the elements of a syndrome unless we have an external criterion, as in neuropathology." "Since all classifications are arbitrary anyway, and mental patients differ from normal persons in ways that exist in all degrees, it makes no scientific sense to ask whether an entity like schizophrenia 'really exists,' and the use or avoidance of this concept is a matter of preference only." "It is inadmissible to explain a given symptom as caused by a disease D unless we can define the term 'D' independently of its symptoms. Otherwise we would be mixing empirical relationships and meaning stipulations." "Any diagnostic cutting score on a continuous indicator variable will be arbitrary, a matter of semantics or convenience." "I can find you a so-called 'schizophrenic' who is more similar symptomatically to some manic-depressives than to most schizophrenics, which proves there is no such entity as schizophrenia." "To speculate that a particular person has the disposition to schizophrenia even though he has survived the morbidity risk period without becoming clinically schizophrenic is scientifically meaningless."

None of these familiar remarks is expressed in technical philosophese, but they are all methodological in nature. *And they are all erroneous.* The last one, for example, imposes a criterion of empirical meaningfulness whose grave disadvantages were already shown by logician Carnap more than 50 years ago (Carnap, 1936-1937/1953, pp. 461-463), when the philosophy of science was far more "operational" and "positivistic" than today. I doubt one could find a single contemporary logician or historian of science who would accept the remarks quoted (Meehl, 1972c, 1986).

SYNDROME AND DISEASE: METACRITERIA FOR ASSIGNING WEIGHTS

Let's go back to Kraepelin. In the 1899 (sixth) edition of his textbook of psychiatry (although he had adumbrated it in the fifth edition 3 years earlier), he sets out for the first time his concept of the entity *dementia praecox.* It was a composite of entities previously described by himself and others, namely, the *démence précoce* of Morel in 1857, the catatonia or *tension insanity* of Kahlbaum in 1874, the hebephrenia of Hecker in 1871, and Kraepelin's own concept of *dementia paranoides.* As you know, this synthesis involved at least as much emphasis upon history as upon symptomatology in that it was a presumably endogenously determined dementia occurring in adolescence and young adulthood and running a malignant course, the latter big fact differentiating it from the favorable prognostic outlook of the other great psychotic group that we now call the major affective disorders. What Kraepelin calls *"the* common characteristic" of dementia praecox—and I take it, since he was a compulsive Teutonic nosologist, he means the *only* common characteristic—is set out briefly and with little descriptive precision, namely, "a peculiar destruction of the internal connections of the psychic personality." It is true that in subsequent editions of the textbook he no longer made early life onset or terminal dementia strictly *definitive,* since he was forced to admit that some cases that met the rest of his criteria made an apparent clinical or social recovery, and that some patients first showed manifest illness in their 30s or even 40s. But the point is that two aspects of history, onset and course, played a core role in his conception of the disease. Bleuler (1911/1950) extended Kraepelin's conception, and, as I read him, considerably more than most British and Continental (or conservative American) psychiatrists are willing to admit. Thus, for example, he flatly states, both in the 1911 classic and in his *Textbook of Psychiatry* (1924), that nearly all cases of chronic intractable hypochondriasis are in fact schizophrenes.

Karl Menninger complained years ago of psychiatrists who rediagnosed patients they had at first labeled dementia praecox or schizophrenia when the patient appeared to make a good social and even clinical recovery. Today we have pretty well accepted the rule of thumb "one third." That is, of patients having a clearly recognizable florid schizophrenia attack, we can expect one-third never to recover from the attack, one-third to recover but have one or more subsequent attacks, and one-third to recover and remain well. Bleuler said that perhaps a fifth of patients make a social recovery, but while he held that Kraepelin was overly pessimistic and succeeded in influencing Kraepelin in this respect in his subsequent editions, Bleuler does make the flat statement —and he had seen not hundreds but thousands of schizophrenic patients and had been able to follow their course over many years—that he had never seen a case that he thought when first carefully diagnosed was an unquestionable

schizophrene who upon making what we would ordinarily call a clinical recovery had a complete restitutio ad integrum. In fact, he said, a good clinician does not usually have to look very closely at a "recovered schizophrene" to see unmistakable residues of the disease.

How do we go about deciding when history, as distinguished from the presenting syndrome and the psychometrics, deserves a high weight or even, as clinicians who rediagnose on the basis of long-term course, a definitive status? Whatever else you can say about this problem it is not "merely semantic," and it ought not to be whimsical or arbitrary. The logical positivists' point about the conventional character of definitions has been abused by some psychologists in a way that logicians would not countenance, namely, arguing that because a definition is ultimately stipulative about the use of language, that therefore one definition is just as good as another. As far as I'm aware, no logician, not even the Vienna positivists in their 1928 heyday, maintained such a silly thesis. I put it to you that the evidentiary weight that history deserves among our diagnostic criteria rests epistemologically upon precisely the same basis as neurology or mental status or biochemical tests or an MMPI profile; to wit, it is a portion of the evidence whose relevance must be decided empirically, that latter including corroborated causal theory, if such exists. I have heard psychologists poke fun at psychiatrists for modifying diagnoses in the light of course, as if this were some kind of vicious circularity. They are mistaken in this. The epistemic situation of a diagnostician in the absence of a well confirmed causal theory of a disease entity is exactly analo–gous to that of a psychometrician factor analyzing an omnibus intelligence test, even though the mathematics has a different structure. You start with the initial observation that certain signs, symptoms, test scores, and life history facts show a non-chance tendency to be associated in the population of patients and normal people. In organic medicine, the fact of the existence of things that "run together" (the Greek for these words being *syndrome)* is usually fairly obvious, and physicians have identified such entities many centuries before Karl Pearson invented chi square and the correlation coefficient. Doing it without formal statistics and frequently with reliance upon one's clinical memory means, of course, that some pseudosyndromes will be identified. But that fact does not disprove the basic epistemic point that when symptoms S_1, S_2, … S_n are found empirically to cohere, so that when patients have a pair of them they are likely to have several of the others, we conclude that we are onto some kind of disease entity. As I have pointed out elsewhere (Meehl & Golden, 1982), there isn't anything metaphysically profound or methodologic–ally suspect about this. It's close to common sense and the universal experi–ence of medical practitioners. Dr. Fisbee discovers a new syndrome not in the books, consisting of headache, fever, pink ears, and purple tongue. Initially all he has is the statistical confluence of the signs and symptoms. But he also has a sensible medical conjecture, namely, there is something going on inside the

patient that gives rise to this syndrome. He doesn't believe that the pink ears are themselves directly causative of the purple tongue or the headache, and so he conjectures—a plausible conjecture warranted by the whole history of medicine since Hippocrates—that these things that go together do so because of a common cause, and that that common cause is not witchcraft or sunspots but an inner state of some sort. In organic medicine that state is called *pathology*.

Now the question is, which signs, symptoms, test scores, or history facts ought to receive more weight in diagnosing the new entity? In medicine that is ultimately decided by the correlation each of them has with the defining pathology. If two similar pathologies are associated with different etiologies (as, for example, two different germs causing meningitis), the weighting of symptoms will also take that different etiology into account. The problem in "functional" mental disorders is that the clinicopathological conference does not have somebody who comes in with the patient's psychic institutions on a tray, or with slides, the way they do in internal medicine, and who points to the "holes in the patient's superego" or the "latent homosexual bubbles from his id." The closest analogy to the pathologist's report on tissue is trait organization and psychodynamics; the analogy to the etiology is genes and the social learning history (Meehl, 1973b, p. 287). Until a well corroborated causal theory of something like schizophrenia or the sociopathic personality is available, we cannot offer *criterial* definitions of the sort we do in neurology or internal medicine, where one says that whatever may be the symptoms and signs, a necessary condition to have paresis is the characteristic (I was taught *unique)* cortical changes of the paretic brain and the presence of *Treponema pallidum* as the causal agent. We do not have the equivalent of this in psychopathology.

Does this mean it is impossible to say anything rationally persuasive about the differential weights for elements of the syndrome and facts of the life history, in discussing an entity like schizophrenia? It is easy to become discouraged and think this, but things aren't quite that bad. I look again to organic medicine for enlightenment, recognizing that some modifications—especially souping up the mathematics—will be necessary. In the delineation of medical syndromes, including those for which the pathology and etiology are unknown or speculative, one is struck by an interesting historical fact. A medical writer does assign more diagnostic weight to some facts than others, which is puzzling when he has no pathological, defining criteria against which to validate them. Suppose I say that Fisbee's Syndrome has as elements purple tongue, pink ears, fever, and headache, but that the pink ears, while perhaps not pathognomonic, are "almost invariably present," whereas the fever may be minimal or absent. This seems a strange thing to say if at this stage of my knowledge all I have to *constitute* Fisbee's Syndrome is its signs and symptoms. This kind of epistemic situation leads some hard-nosed behaviorists to

say that the syndrome is circularly defined, which is a stupid thing to say, because the *fact of the syndrome* is a statistical fact, not a definition. When we "define" the syndrome, if we are careful, we scrupulously refrain from a literal identification of the disease with its signs and symptoms. Such an identification would be impossible here, since it would mean that none of the elements listed could ever be missing. That would require that the pairwise correlation of the elements be perfect in order for a syndrome to exist. Since that is plainly false for several thousand well understood diseases, it would be a foolish move to say that a syndrome can only exist if its elements are pairwise perfectly correlated. Most syndromes in organic medicine are like syndromes in psychopathology, namely, they are "loose syndromes" (Meehl, 1973a). That is a fact of nature, not a matter of clinicians' carelessness, laziness, or illogicality. *The background conjecture is the same in psychopathology as it is in organic medicine, even though the theoretical entities differ, and psychologists have been forced to develop more sophisticated search methods than Hippocrates or Osler.*

Basically, the notion is simply that if two things go together in a nonchance way and cannot (on common sense or theoretical grounds) plausibly be supposed to be causing each other, then there is some third thing that causes each of them and brings about their covariation. It is that unknown latent something that we have in the back of our minds when we undertake research on a syndrome so as to understand it better. We anticipate that when we understand it well enough, we will be able to substitute an explicit definition, *in these causal theoretical terms,* for the implicit or contextual definition we have to offer initially by referring to the syndrome's internal statistics. It puzzles me that some psychologists have a hard time grasping this simple methodological point. I have heard it said that to invoke schizophrenia as a cause of hallucinations is circular or, even worse, like explaining a hysterical symptom in terms of witchcraft. The trouble with those fetching analogies is that they are themselves circular, because they presuppose a negative answer to the question whether there is some shared pathology and etiology in cases of schizophrenia. The trouble with the witchcraft theory of a paralyzed arm is not methodological but factual; that is, there aren't any such things as witches. The witchcraft analogy could be used in this way to disparage the biochemist's view that milk is curdled by little creatures called *Lactobacillus bulgaricus,* holding that the latter concept is no more methodologically respectable than the medieval housewives' view that milk is curdled by the brownies. Such malicious analogies are useless, and I have found that pseudosophistication is far worse than naivete because it's harder to cure. The reason we prefer the *Lactobacillus bulgaricus* to the brownie theory is simple—namely, there *are* tiny organisms that secrete the enzyme that curdles milk, and there *aren't* any brownies. If there were any brownies, it would be perfectly all right to explain things by invoking them.

The prevailing theory of schizophrenia when I was a student was that the specific etiology that would some day become the "operational definition" of the disease was something about having a battle-ax mother, a historical event whose residues are now preserved in the patient's schizoid psyche. The statistical evidence having largely discredited that theory (which has nothing wrong with it had it turned out to be factually correct), we now have good support for a different theory, namely, a schizophrenic genetic makeup, whether a major locus, or two, or polygenic with a threshold being still a matter of empirical dispute (Gottesman & Shields, 1972, 1982). If we remain stuck at our present level of information for some time, where evidence is clear to an unbiased mind that the thing is heavily genetic, but we don't know the biochemistry, or even whether a major gene is involved, how would we go about assigning weight to a historical fact, such as unusual shyness in school or—a favorite one with me that emerged from my colleague Garmezy's research years ago—doing well in school until you hit fractions in the third grade and then rapidly declining in school performance? Or how about having a first-degree relative diagnosed schizophrenia? Or a complete symptomatic recovery, where even Bleuler might be hard put to point to the residual scarring?

Well, if that's all we know, we have to rely upon the internal statistics of the syndrome, of course including psychometrics and ward behavior as part of the data. The first question to ask is a mathematical one, namely, is this entity taxonic, that is, does it represent a *type* or *taxon* (a natural kind), or are we merely locating the patient in a hyperspace defined by the relevant continuous descriptors? I would like to think that I have solved the statistical problem of answering that question with some of my own recent taxometric methods, but that would be a bit on the grandiose side. I will content myself with saying that *some such methods* as those that my colleague Robert Golden and I have been working on in recent years will provide the answer (Gangestad & Snyder, 1985; Golden, 1982; Golden, Campbell, & Perry, 1987; Golden, Gallob, & Watt, 1983; Golden & Meehl, 1978, 1979, 1980; Golden, Vaughan, Kurtzberg, & McCarton, [1988]; Meehl, 1973a, 1979; Meehl & Golden, 1982). The question as to the precise relationship between satisfying statistical criteria of latent taxonicity and corroborating an interpretive text speaking about causes presents grave difficulties, but I am optimistic. Whether you happen to like cluster analysis or latent class analysis and, if the latter, whether you are favorably impressed with such of my methods as have yet been published, is not the point. I only want to persuade you that the essential idea of establishing taxonicity by means of a suitable type of cluster or latent class algorithm corresponds, in a modern sophisticated way, to the historical fact that physicians identified many real diseases before the creation of modern pathology and bacteriology by such greats as Virchow and Koch. The metatheoretical reasoning in taxometrics is essentially the same as in factor analysis, multidimensional scaling, and other psychometric procedures that infer the operation

of latent causal entities by studying the statistical structure of covariant facts. Having said this against abusers of the tricky word "circularity," let me say equally clearly that I do not wish to exaggerate the power of purely correlational methods, especially those confined to cross-sectional data, in testing substantive causal theories. Adding the time dimension (we don't let the causal arrow operate backward in time!) and, even more, including in our evidence portions of the theoretical network tested in other ways (e.g., experimentally, or derivable from some other discipline such as genetics) are usually necessary in resolving theoretical disagreement arising from purely correlational information. Of course everybody (except perhaps B. F. Skinner) agrees that one of the merits of a good theory is suggesting new facts to collect or new ways of analyzing old facts.

THE THEORY OF SCHIZOTAXIA

Let me take my theory of schizotaxia as an example (Meehl, 1962b, 1972a, 1972c). I conjecture the existence of a *schizogene* (a few of us, especially in Minneapolis, are not convinced that the major locus theory has been slain) that is an autosomal dominant completely penetrant for an endophenotypic neurological trait I label *schizotaxia.* This is a special kind of neural-integrative defect (about which I have a detailed physiological conjecture) that is, strictly speaking, *all* that can be "inherited." I do *not* say that schizotypy (the personality makeup predisposing to schizophrenia) is inherited, although that statement is commonly attributed to me in general and abnormal psychology textbooks. Schizotypy as a personality makeup is based upon social learning by an organism with the schizotaxic central nervous system (CNS) defect. Schizophrenia is a decompensation of the schizotypal personality that, given the available genetic statistics, I assume occurs in only around 10% of them. I do not look upon schizotypy as a kind of "attenuated schizophrenia," which I think inappropriate for a sophisticated genetic-learning theoretical model. Therefore I also tend to avoid the common locution "spectrum disorder." An analogy: Consider gout, whose sine qua non is an elevated plasma uric acid titer. Less than 10% of men having the gout genome and therefore the elevated uric acid titer develop clinical gout; no one knows why. If we take the medieval physician's tetradic definition of "inflammation" (rubor, dolor, turgor, and calor), we do not expect the gout-free monozygotic (MZ) twins of clinically gouty males to have "just a teeny bit of gout." We do not anticipate their being ill with a member of a gout specrum. What we do anticipate in an MZ twin, and in a sizeable number of dizygotic (DZ) twins, is the elevated uric acid titer.

There is nothing mysterious about this, statistically or causally. It's simply that the closer we are in the causal chain to the DNA, the larger the correlation between a fallible indicator and the mutated gene becomes. One therefore

expects the psychophysiology and "soft neurology" of schizotaxia to be more powerful indicators of the schizogene than, say, social anhedonia or subtle semantic manifestations of thought disorder. Since on such a theory there are several presumably polygenic potentiators of schizophrenic illness in the schizotypal personality (I have elsewhere listed a dozen or so, each of which I would bet on with high confidence, purely from my clinician's armchair), we have no way of knowing in advance which of these polygenic potentiators is more causally significant, and therefore we do not have any *theoretical* basis for assigning to some of them more diagnostic weight than others.

I no longer anticipate any molar behavioral indicator, psychometric or interview, to be pathognomonic of schizotaxia, although I do still believe in a one-way pathognomicity (that is, an inclusion test) in the characteristic schizophrenic thought disorder. The example I gave in my 1962 APA presidential address of a patient who, asked why he was in the hospital, said, "Well, doctor, naturally, I'm growing my father's hair" seems to me as good today as it did then, as would that of a patient who, when told by the examiner "I sometimes have trouble following your talk," replies with "Oh, yes, doctor, that's because I often speak Echo Monster Head Affair Language." But the *absence* of thought disorder, even of a subtle type hard to detect on interview or psychometrically, is not, I believe, valid as an exclusion test for schizotaxia. Of course, many competent clinicians would reject it as an exclusion test even for schizophrenia of the relatively intact paranoid variety.

It may be that a sufficiently subtle and mathematically souped-up measure of semantical or syntactical aberrations will someday be a powerful sign of the taxon, but I'm inclined to doubt it, and nobody has yet succeeded in devising one. One ingenious and indefatigable researcher, Oltmanns, after several years of work along the "subtle semantic" cognitive slippage line, has for the time being abandoned that approach to finding strong schizotypal indicators (Berenbaum, Gottesman, & Oltmanns, 1985; Kagan & Oltmanns, 1981; Neale, Oltmanns, & Harvey, 1985; Oltmanns, personal communication, May 19, 1987; Ragin & Oltmanns, 1986). I confess to some resistance in accepting this conclusion, partly on (weak) theoretical grounds, partly because of the central role of associative loosening in Bleuler's minimally theoretical clinical description of the diagnosable syndrome. It is puzzling that a core feature of the disease should be totally absent in compensated schizotypes, so I persist in entertaining a faint hope that we haven't hit upon a sufficiently sensitive (schizospecific!) way to measure it.

The main problem with molar indicators of compensated schizotypy is their "causal distance" from the DNA. Speaking physiologically, we deal with molar outputs that result from numerous serial and converging chains of brain processes, acting as potentiators or inhibitors of one another, sometimes involving two-way influence (positive or negative), presenting possibilities of step-functions and sign reversals, and with the parameters of *all* their linkage

functions having large normal range (polygenic and acquired) individual differences. Speaking psychologically, anything molar has a complex learned form and content, with individual differences arising from the different life histories superimposed on (and partly due to) genetic differences. If we did not have available the mass of inconclusive or inconsistent studies to confuse us, we should be able to predict their messiness from our clinical armchairs. Consider, for example, the salient trait of anxiety proneness. Psychodynamically, one thinks immediately of the 20 or so defense mechanisms that the anxiety signal puts into action and realizes that all 20 can plausibly be supposed to have different influences, qualitatively and quantitatively, on various cognitive functions. We cannot even assume that all schizotypes will be made anxious by the same social stimulus, or that, if made so, they will engage the same defense mechanism at different anxiety levels or in different social contexts. The obfuscating role of problematic auxiliary theories, including minitheories of psychometrics, is so great that the conceptual task of parsing studies verges on the impossible (Meehl, [1990b]).

I do not suggest that we cease inquiry into molar level indicators, since a few of them may turn out well despite these pessimistic considerations. "Chaotic sexuality," for example, while listed in my schizotypal checklist (Meehl, 1964) as a fairly important indicator of decompensated schizotypy, I should not have expected to show up in the compensated range, but Frost and Chapman (1987) looks encouraging.

We want to select indicators of the schizotypal personality (Gunderson, Siever, & Spaulding 1983; Meehl,1962b, 1964, 1972a, 1972b, 1972c; Morey, Waugh, & Blashfield, 1985; Rado, 1956, I960; Rado & Daniels, 1956; Siever & Gunderson, 1983). If one is unpersuaded that there is such an entity, then say merely "the schizophrenia-prone person" (Chapman & Chapman, 1985, 1987). We are faced with a vast, confusing, and often inconsistent literature that contains several hundred candidates for indicator status. One has the impression that whether a certain line of investigation has been pursued did not depend solely on satisfactory replicability. We should not reject purely clinical suggestions in the early literature merely because, when Kraepelin and Bleuler were writing, present day standards of "hard data" quantification and statistical method had not yet evolved. It's one thing to ask whether the observations of an astute and seasoned clinician have been subjected to modern quantitative scrutiny. It's another—smacking of perfectionist, nay-saying scientism—to say that if that has not been done, that we can relegate these impressions to the trash can.

The choice of indicators for further research ought to be based upon a combination of skilled clinicians' consensus (I never lightly dismiss anything the great Bleuler says in his 1911 classic) with what quantitative replications exist, plus a liberal dose of theory. The trouble with this latter is that we have had a great proliferation of theories in the 90 years since recognizing dementia

praecox as a diagnostic entity. I have read somewhere that if you count mini-theories, there are over 100 published theories of schizophrenia. It's discouraging that none of them has commanded even a preponderance of assent. If we press the point, about the only thing we can say for sure about schizophrenia theory is that there is something genetic about its etiology. We are almost at the stage where a second conjecture commands sizeable assent, namely, it is in some sense a "neurological" disorder. In my APA presidential address over a quarter century ago (Meehl, 1962b), I concluded by predicting that taxonomic statistical methods applied to carefully chosen fallible indicators would show that schizophrenia is fundamentally a neurological disease of genetic origin. That was rank heresy in the psychological profession (and even to a considerable extent in psychiatry) in 1962, and I am in the pleasing position of seeing (if I see straight) my rash prophecy increasingly confirmed. I hope to present details of my current theory of schizophrenia in an issue of Dr. Millon's journal [Meehl, 1990a], so I shall only adumbrate that fuller presentation here.

My strategy is different from that followed by almost all previous theoreticians. The usual approach to this puzzling disease has been to focus attention upon one striking feature of the phenotype, chosen by its relative specificity or its qualitatively striking character or its social importance. One then takes this "core, striking" phenomenological feature as basic and attempts to derive the rest of the syndrome from it. This can be done in psychological (behavioral or experiential) terms, or it can be done by identifying the allegedly core feature with some specific region of the brain or some component of the body chemistry and then working back from that conjectured physiological core to attempt derviations of the rest of the loose syndrome. I think this strategy is a mistaken one, and that's why none of these theories has commanded the assent of more than a small minority of practitioners and researchers at any given time. When you approach it this way, you have a fairly satisfactory "explanation" (although sometimes rather tautological) of the one big feature that you selected for attention as being the essence of the disease, but then when you start trying to explain the other elements of the syndrome that, even if not statistically as core as yours, you have to admit are fairly high-weight indicators and certainly have a frequency sufficient to require any theory to explain them plausibly, you have to do an awful lot of fancy footwork and ad hockery, which theorists of other persuasions find implausible.

I think that the psychological source of this strategic mistake is a simple one, namely, that most of us hear about the schizophrenia syndrome in an abnormal psychology class as undergraduates. Many of us first heard it earlier, as sophomores, when described in lecture and several pages of text in a general psychology class. So that by the time we are practitioners and researchers, we have become thoroughly accustomed to this collection of signs, symptoms, traits, and life history characteristics. For this reason, "familiarity breeding blindness," we are unable to take a fresh look, and we have come to take what

is really a very odd, heterogeneous collection of aberrations for granted. Clinicians don't realize, unless they force themselves to step back and try to be naive observers who never heard of this strange entity before, what a phenomenologically unconnected, qualitatively diverse range of aberrations constitute the loose syndrome at the descriptive level. I won't try to prove it here, but if you will reflect, I think you will agree that this is truer of schizophrenia than it is of, say, the major affective disorders or anxiety neurosis or organic brain syndrome or even the psychopath. A layman could derive much—I am inclined to think almost all—of the syndromes of hypomania or depression if asked to imagine how he would think, feel, and act if he were a lot sadder than he's ever been or "up" more than he's ever been. But if you try this with schizophrenia, selecting something like, say, an extreme of social introversion, and expect a layman or a personality psychologist to derive from that, thinking phenomenologically or behavioristically, the body image distortion, the soft neurology, the hypohedonia, the subtle semantic aberrations, or psychophysiological indicators like the eye-tracking anomaly or reduced secondary inhibition of the P50 auditory evoked potential, he would not be able to do it. My point is that we should take that qualitative diversity seriously as the *first big fact that must be faced in adopting a strategy of theory construction*.

Let me give some examples of the wrong strategy that go very far back (except for my own 1962 mistake), so hardly anybody today would have any investment in them. While I was an extern in the early 1940s, psychiatrists in my surround were talking about the probable crucial role of what was then called the reticular activating system (RAS) in the etiology of schizophrenia. Why were they talking about this? Well, its very existence as a discrete anatomical system had only been demonstrated in the late 1930s, and that interesting finding from the neuroanatomist was combined with the obvious clinical fact that many schizophrenes show a deviation from the norm in their general level of arousal. The majority were on the low side, with some chronic deteriorated back ward schizophrenes almost as vegetable-like as a severe organic brain syndrome patient; others, especially in the early stages (and dramatically in the syndrome of *catatonic raptus*), showed a hyperaroused state. I'm not saying that it was stupid to call attention to this interesting possibility. I'm merely illustrating what I call the "specific defect" error in strategy. One could understand *some* psychometric and psychomotor characteristics on the basis of aberrations in the arousal system. But when one asked why should arousal result in the unique kind of thought disorder that is not found in an excited manic or a hyperaroused anxiety neurosis, or what arousal should have to do with the interpersonal aversiveness so characteristic of this disease, or even why it should be that some of the severest shattered mentation with gross thought disorder should be found in calm patients (I view the characteristic thought disorder in a nonconfused, nonexcited state as one of the

two pathognomonic inclusion tests for schizophrenia), it was hard to explain these things on the arousal basis.

Then I remember a suggestion current around 1940 that the basic problem was a disturbance of carbohydrate metabolism, suggested by the apparent efficacy of insulin treatment and the tendency of some schizophrenes to gobble sweets (one thinks of schizotype Hitler!) and the fact that the brain doesn't know how to utilize anything but carbohydrate in its energy consumption. Or again, because of the spread of interpersonal aversiveness that is *socially* the most outstanding feature (we notice that more than we do soft neurology in our relations with other persons), and assuming that aversiveness ultimately comes down to a matter of conditioned anxiety, we had theories shortly after World War II that focused upon some quantitative aberration in the anxiety parameter (say, the steepness of the conditioned anxiety gradient). This works fine for social fear, but when one considers that potent anxiety reducers like Valium don't do anything for schizophrenia, that relatively nonanxious chronic schizophrenes are likely to show more severe thought disorder than acutely anxious early stage cases, and that one can think of many neurotics who manifest extremes of anxiety more than the great majority of schizophrenes but who do not show even a hint of schizoid cognitive slippage, or any of the psychophysiological or soft neurology features of the syndrome, this conjecture requires more extensive ad hockery than we want to permit.

Coming closer to home, in 1962 under the influence of my Rado-trained analyst (from whom I learned a great deal about schizotypy), I attached more diagnostic importance to hypohedonia than I now think appropriate, and I even made what I now view as a foolish, unplausible attempt to derive the schizophrenic syndrome from a genetic hedonic deficit. I believe that all such theories are bound to fail.

If we begin by focusing our attention not on a particular facet of the syndrome that has high statistical occurrence or clinical importance or social impact or striking quality but instead attend to the near ubiquity of the defect, we come to a different kind of theory. I do not of course maintain that absolutely everything you can study in schizophrenes is impaired, but I suggest to you that *almost* everything is impaired to some extent, even those functions that Bleuler in his monograph describes as not so, such as attention (cf. Knight, 1984). We don't have to decide exactly which subset of all of the hundreds of empirical findings in the literature will hold up under efforts of quantitative replication to make the general statement that many, many things are haywire in these people compared to how many different kinds of things are haywire in neurotics, those with major affective disorders, or psychopaths. In almost all behavior domains or sectors and at all levels of molarity from tendon reflexes up to the most integrative, complex, learned social behavior, if you look carefully, the schizophrene is likely to show some departures, large or slight, from the norm. That to me is the big fact, and the one that suggests

the kind of theoretical strategy I favor. The most obvious, straightforward conjecture suggested by ubiquity of aberration at different levels of molarity and different life sectors is that *whatever is wrong with the schizotaxic CNS is wrong throughout; that is, the schizotaxic nervous system is aberrated in every anatomical region and at all levels, from the sacral cord to the frontal lobes.*

What might this defect be? It is not a defect of inputting, nor storage, nor retrieval, nor of insufficient complexity, all of these being defects that would prevent the extraordinary intellectual achievements of some world-renowned schizotypes (e.g., Newton, Goedel). I conjecture a quantitative aberration in the synaptic transmission function called *hypokrisia.* Consider a pattern of arrival times for presynaptic spikes at the terminal knobs, representing this pattern over the synaptic scale by a point in the synaptic signal space whose coordinates are arrival times at the knobs. Assuming concurrent stimulation of the axon hillock, each such point is associated with a Lorente de Nó optional transmission probability. These probabilities define a hypersurface in the synaptic signal space. My conjecture is that this hypersurface is both (1) elevated and (2) flattened—exaggerated but with fewer differential hills and valleys—in the schizotaxic CNS. This results in single-unit "synaptic slippage," which in turn causes slippage in intermodule control functions, and so on up the hierarchy of CNS systems. The hypokrisia itself I attribute to a deficient amount of some substance X whose function is to stabilize the cell membrane or facilitate its repolarization in stimulated subregions in the orthotaxic brain. So you see I am doing precisely what disciples of Skinner consider a cardinal sin, to wit, reattributing to the CNS innards a descriptive property of the behavior, by generalizing Bleuler's fundamental trait of *associative loosening* to the physical system underlying associations, specifically, functional connections at the synapse. Contrary to the Skinnerians, while the history of science shows examples where this kind of partly repeated attribution at another causal level has been useless, in other cases (e.g., magnetism, gas pressure, population genetics) it has been extremely profitable and turns out to be the literal truth of the matter. Strangely enough, while Bleuler does mention several phenomena of soft neurology in his chapter on the physical symptoms of the disease, he does not (so far as I can find) connect these with that "loosening" he believes to be fundamental at the molar level of behavior and experience.

How this loosening in synaptic control will affect a given molar level and behavior domain cannot be rigorously derived without knowing details of the CNS systems of molar domains among normals, which we do not presently have. Hence, explanations of why some cognitive functions are more impaired than others in the experimental literature of psychologists studying schizophrenia are usually premature and result in what appear to be inconsistent findings (Knight, 1984). But I suggest that less detailed, broadly characterized trends are understandable. Why does a ubiquitous integrative defect require

less ad hockery than that required by theories that focus on a specific domain or subsystem and are then faced with the terrible task of deriving the qualitative diversity of consequences? The methodological point involved here is this: Because of the syndrome's diversity, playing it in the customary way requires *many different unrelated ad hoc auxiliaries* (Meehl, 1978). It might seem that postulating a ubiquitous defect throughout the nervous system at all levels and in all domains would be in the same boat with regard to ad hockery, but this is not true. Because instead of having the former difficulty, how to derive dissimilar symptoms S_1 S_2, … S_n from special defect D_s? we have the opposite problem in my kind of theory, why doesn't ubiquitous defect D_u impair all functions (levels, domains) equally? But this second question is much less embarrassing. The reason is that we already have independent knowledge, apart from psychopathology, concerning many of the required auxiliary hypotheses. Also, the general fact of partially independent and variably influenced subsystems is well known outside of psychopathology, so it is not ad hoc as a general principle. Classical psychometrics provides the paradigm. For example, in omnibus intelligence tests we have a general intelligence factor, several group factors, specific factors, and error in our psychometric equation. We don't have to know anything about schizophrenia or about abnormal behavior to know that common knowledge and a huge mass of psychometric research on abilities and personality traits shows that whatever may be the detailed character of the cerebral subsystems involved, it is an empirical fact that a general factor, even if as powerful as we find in something like *g,* does not saturate all kinds of performances equally. This kind of standard psychometric model is the model we should adopt in thinking upon findings like soft neurology in the schizotype. This view, which, I repeat, *does not involve assuming anything beyond what we already know from a huge mass of data about the psychology of individual differences among normals,* leads to expectations that a ubiquitous CNS aberration (1) should influence all, or nearly all, levels and sectors somewhat, (2) should influence some levels and sectors more than others, and (3) should be statistically "fuzzy" because of individual differences in group factors and specifics. There is nothing ad hoc about any of these expectations.

One would like to say a little more than this about different behavior domains, and I think that we can make some plausible predictions of a probabilistic nature as to which domains we expect to be more impaired, *on the average.* We expect to have more impairments among domains (1) involving relatively distant subsystems and hence spike transmissions as the main process, (2) requiring complex integration of spike-transmitted outputs from diverse controlling subsystems, (3) demanding finely tuned multiple control by several converging subsystems as in (2) (this a conjecture that might be expressed mathematically in terms of values of the partial derivatives of multiple input control functions), (4) providing rare opportunity for develop-

ing corrective or substitute adjustments (for example, not like the color blind who learn to rely on other cues), (5) where growth of a molar aberration is autocatalytic because two or more functions interact with positive feedback (for example, we have distorted social cognitions leading to aversive drift leading to further distorted cognitions, etc.), (6) where person and environment interactions are like the intrapersonal situation (5), and (7) where the external reinforcement schedule is highly stochastic. Thus the most stochastic of all reinforcement schedules in human life, as I pointed out in my 1962 lecture, is the social, and hence it is not surprising that the most dramatic impairment in the schizotype often is in the interpersonal domain.

It would be pretentious to undertake any kind of high-level quantification of the above list. But I think the reader will agree with me that it is at least not empirically empty of consequences. So my contention is that the postulation of a ubiquitous integrative neural defect such as schizotaxia (whether or not its micro level is spelled out in detail, as in my concept of hypokrisia) does not involve the tremendous amount of unrelated ad hockery needed in going from a more special defect of a particular subsystem like the reticular or the limbic or the dopamine or whatever; rather, it appeals to already independently known facts about individual differences such as are familiar from classical psychometrics. Deriving the aversive drift and the hypohedonia from the concept of hypokrisia is a much more complicated—and, I admit, loose—derivation that I do not have space or courage to present here but will try to do in a longer paper now in preparation [Meehl, 1990a].

My colleagues in behavior genetics are puzzled by my avoidance of the term "penetrance" in discussing the theory of schizophrenia (Meehl, 1962b, 1972a, 1972c, 1973a). They sometimes think I dislike the concept because it is an abusable fudge factor, but that is not my point at all. There is nothing wrong with a fudge factor if the relation of the number of equations to the number of unknowns makes the penetrance coefficient overdeterminable from the data. While I sometimes allow myself to speak of "clinical penetrance," by which I mean the proportion of schizotypes who develop a diagnosable clinical schizophrenia, this is a harmless concession to the conventional speech of geneticists, and I have a methodological reason for preferring a different terminology. Genetics texts introduce "penetrance" first and then define "expressivity" as a secondary, derivative notion—that is, "*If* it shows in the phenotype, *then* by how much quantitatively?" This order is both epistemologically and mathematically backward. The proper prior concept is expressivity, the quantitative distribution of an indicator among subjects having the schizoid genotype. This distribution is an objective fact about the empirical world, although we may have a hard time ascertaining it, whereas the penetrance coefficient is the result of our choice of cutting score. The only nonarbitrary penetrance is 100, that is, when the two expressivity distributions have zero overlap. There are as many penetrances as there are cutting scores for a

single quantitative indicator. But the situation is worse than that, because most syndromes—certainly all syndromes in psychopathology—are fairly loose in the statistical sense; so we first have arbitrary penetrance cuts on each trait, and then we have a system of such cuts assigned over the several traits of the phenotypic syndrome. That's a different situation from having a scarlet eye color in the fruitfly.

In my theory, what the schizogene does is to produce a certain aberration in the synaptic control parameters, an *endophenotypic* trait at the level of a single nerve cell, found throughout the CNS of schizotaxic persons. That is *all* the schizogene directly produces causally. In textbooks and articles I am said to hold that what is inherited is the schizotypic personality, but I never committed such an absurdity, and I think my writing on that is clear. All that is, strictly speaking, inherited is the schizotaxic brain, upon which the schizotypal personality develops by a complicated process of social learning. The phenotype is in turn expressed, in varying quantitative amounts, by various psychophysiological and soft neurological exophenotypic indicators. Whether any of the latter are truly pathognomonic for the schizotaxic brain I do not know. But I take it for granted that the longer the causal chain and the more different kinds of causal links are in that chain between the DNA and an exophenotypic manifestation, the lower the statistical "pathognomicity." It is theoretically unlikely, on a model such as this, that one could invent, say, a psychometric measure of cognitive slippage or hypohedonia that would have quasi-pathognomonic status with respect to the dominant schizogene I conjecture, or, in a polygenic model, a high correlation with the polygenic variable or high sensitivity for having passed a polygenic threshold of the kind conjectured by Gottesman and others.

I find some behavior geneticists, even those whose Ph.D. is in psychology, minoring in genetics (and almost always among those where it goes the other way), still refer to "the trait" in discussing schizophrenia or schizotypy. This is an unfortunate usage because schizophrenia and schizotypy are *loose syndromes,* composed of elements some of which have no content overlap with the others. This is partly what makes schizophrenia so baffling (and interesting!) compared, say, with the major affective disorders. There isn't anything behaviorally in common between subtle semantic or syntactical aberrations in speech, extreme social aversiveness, and a tendency to hear voices—let alone a ± dysdiadochokinesia or the famous reduced specific dynamic action of protein that the Worcester group found when they fed schizophrenia patients a meat breakfast! Even the psychophysiological and soft neurology indicators, which I am happy to see have now been revived in research interest and are spoken of more commonly among clinicians (they went in abeyance during the heyday of psychodynamic and social views of schizophrenia), are far from perfectly correlated. Why should we be surprised at this? It *would* be surprising if the correlation between different neurological and psychophysiological

indicators was feeble compared with that between social and personality measures, which attain the values they do as the end result of a long stochastic process involving thousands of life experiences in social learning, whereas the former are closer in the causal chain to the schizogene and influenced by fewer nuisance variables. We take the existence of group and specific factors as a matter of course in all domains of psychometrics, whether we're studying intelligence or mechanical ability or social effectiveness or whatever. We should also assume, given the terrible complexity of the brain, that there are neurological equivalents of group and specific factors, involving different cerebral subsystems, despite our conjecturing a pervasive CNS functional aberration (analogous to Spearman's *g*), as in my theory.

Considerations of this sort also lead me to avoid the term "modifier" in expounding my theory. If the schizogene gives rise, either by a biochemical lesion or, as I think less likely, but a live option, a fine structure, microanatomic aberration (e.g., something funny about the distribution of terminal knobs over the synaptic scale), we have a brain that is predisposed to certain quantitative aberrations in the acquisition of all sorts of behavioral first-order dispositions such as social habits, verbal, perceptual, and motor skills, and so forth. The same group and special factors that would appear in a factor analysis of abilities, skills, and personality traits in a normal population clear of schizotypes will be operating to produce individual differences within the schizotypal taxon and will thereby lead to some indicators being more statistically high-weight for the schizogene than others. You don't need any research to know this a priori. It would be a miracle if it were not the case, extrapolating from our general knowledge of psychology, physiology, and the statistics of individual differences. In my theory of schizotaxia, the basic neural integrative deficit leads, by a chain of reasoning that I cannot develop here, to the prediction of *aversive drift,* and this aversive drift would have *on the average* a greater push in the highly stochastic domain of interpersonal reinforcement schedules than in, say, the simpler high-probability ones of reaching for a seen object or even working on crossword puzzles.

On the other hand, the polygenic system for garden-variety, normal range individual differences in social introversion, which is now well corroborated by numerous investigations, presumably operates in a quite different way, having no overlap with the causal chain involved in schizotaxic aversive drift. Similarly, polygenes for the general intelligence factor initiate another complex *but parallel* causal pathway. There isn't anything in the theory that suggests that these pathways overlap or that the genes for intelligence somehow get in the way of the schizogene's effecting the aversive drift. Even social introversion, which must be involved in some complicated way with the tendency to aversive drift, is not something that *intervenes* in the causal chain between the gene and the phenotypic expression, as in textbook examples of epistasis or polygenic modifiers. Rather, we have a schizogene leading via

social learning processes to more or less specifically schizotypal personality traits *themselves manifest in the phenotype.* (Consider, for example, the "friendly schizotype" we have all seen, who interacts more than a normal person, and far more readily than a nonschizoid introvert, with inappropriate social approaches to total strangers.) Finally, whether a schizotype makes his way in the world or ends up in a mental hospital might be dependent upon his IQ, which is a cluster of traits having, again, negligible overlap with schizoid cognitive slippage or schizoid aversive drift or garden-variety nonschizoid introversion. The point is that these are exophenotypic dispositions controlled by different genetic systems that do interact with the environment and alter the probability of decompensation into a clinical schizophrenia. That causal situation is so different from what is implied by the geneticist's term "modifier" that we need a separate word to designate the change in illness probability, and that's why I prefer the word "potentiator." One consequence of this kind of reasoning is a research strategy that focuses heavily on family studies of neurological and psychophysiological indicators that have been shown to be related to schizophrenia risk, in preference to the more clinically relevant abnormalities of interpersonal behavior.

ENVIRONMENTAL INFLUENCES AND COMPLEXITY OF THE CAUSAL CHAIN

The methodological difficulties in attempting to infer causal influence from the kind of reconstruction of a life history that we ordinarily have available in clinical work are illustrated beautifully (and discouragingly) by the ascendance and decine of the concept "schizophrenogenic mother." After World War II, when I was a young clinician, the notion that one or another kind of battle-ax mother (described in a variety of ways but at least recognized by all as malignant) played a crucial role in the development of schizophrenia was taken seriously by my fellow students and teachers. This was true even in the Midwest, which was less psychodynamic than either coast, where the doctrine was taken as almost axiomatic. In recent years, partly as a result of the adoption studies but also due to the general decline in psychodynamic emphasis in favor of biological approaches to psychopathology, many psychiatrists and psychologists (the majority in my environment) have concluded that the notion of a schizophrenogenic mother is totally without merit. I believe both the old position and the new one are mistaken, and it is useful to examine how these two opposed mistakes have come about.

I begin with the older one. Suppose that there is no such personality type as the schizophrenogenic mother, however described. Putting it more strongly, assume that, typology aside, the behavior of mother has literally zero influence upon whether a child with the genetic disposition to schizophrenia subse-

quently develops the clinical illness, a doctrine with which I must disagree although I grant that it is closer to the truth than the purely environmental interpretation that used to prevail. How did such a notion arise and capture the allegiance of so many clinicians? I remember that during the 1950s, my reading of the genetic data then available, combined with the rather feeble and inconsistent results of efforts to support the schizophrenogenic concept by quantitative studies, led me to question increasingly the alleged impact of the mother's child-rearing practices and attitudes. Students and colleagues strongly committed to the notion expressed bafflement upon hearing my skepticism, knowing that I was doing quite a bit of psychotherapy at that time and that a sizeable fraction of my patients were schizotypes. "Surely your schizotypal patients bring out material like mine do, unless you are somehow blind to it or turning it off by your interview tactics." It wasn't that my schizotypal patients didn't talk about their childhood families (including their mothers) pretty much the same way the psychodynamic literature reports from other therapists. Rather, it was that my Minnesota training from hardheaded skeptics like Donald G. Paterson and Starke R. Hathaway, not to mention Bill Heron in comparative psychology, had sensitized me to the grave dangers of the anecdotal method, as the old animal psychologists used to call it. I was uncomfortably aware that the anecdotal method does not cease to be anecdotal when we rechristen it with the honorific phrase "clinical experience." I did not doubt, and I do not doubt now, that seasoned clinicians know a lot of things that fresh-baked ones and laymen don't know, but I was properly educated to realize that experienced clinicians also "know" all sorts of things that are not true (Meehl, 1987). Absent quantitative research, one cannot be confident as to what part of clinicians' lore is valid and what part is superstition. It is easy to list from the armchair, simply relying upon our general knowledge of the psychology of testimony and the fallibility of human observation and memory, not to mention the recent research on the inefficiency of many human cognitive processes involving complex judgments, at least seven factors that could give rise to the concept of the schizophrenogenic mother from one's clinical experience, even if the notion, as I am presuming arguendo, was totally devoid of merit. I list the factors briefly without argument, since they are all obviously possible and seen to be antecedently probable as soon as one writes them down:

1. Mother actually behaved ambivalently, inconsistently, and unlovingly because she herself was schizotypal, and this historical fact appears in the patient's psychotherapy productions even though the fact was devoid of causal influence on his illness.

2. Since our schizophrene was schizotypal as a child, he was not a rewarding child for the caregiver, who consequently developed ambivalent attitudes toward him, which are now reported in therapy.

3. The child experienced quite normal child-rearing attitudes and practices, which, of course, invariably included some inconsistencies and a mixed positive and negative

social reinforcement schedule, but being schizotypal he experienced them as more aversive and cognitively misinterpreted them more than a normal or neurotic child would have.

4. Our adult schizophrene remembers selectively and distortedly his childhood experiences of mothering, which were not in fact unusual.

5. Although none of the above factors was operating initially in the sessions, the therapist, because of his theoretical orientation, reinforces talk along the lines of the schizophrenogenic mother, probably overtly, certainly in subtle ways. Thus, for instance, he will show more interest in an episode about bad mothering than he will in an episode about bad teaching in junior high school.

6. Because of his theoretical commitments, the therapist remembers episodes selectively and distortedly, although a content analysis of the patient's discourse would not show it to be different from a randomly chosen nonschizophrenic patient.

7. The therapist construes correlations from listening to the content as "obvious" instances of psychological causality, forgetting that while nonchance covariations prove that some kind of causation, in one direction or another, is at work, they do not tell us without further argument what is causing what.

It's important to realize that none of these seven factors leading to a net therapist impression about schizophrenogenic mothers is incompatible with any of the other six. So it is possible, and I should think to any sophisticated psychologist highly likely, that all of them are at work. Speaking dichotomously, in terms of "Did the patient's mother act schizophrenogenically by my criteria or not?" and imagining that each of these seven methodological bloopers generates a spurious 10% increment in the apparent frequency of such mothers among schizotypal patients, we can easily reach an impression that 70% of our patients had such mothers. If the kinds of things we're listening for appeared in, say, one third of our neurotic clientele, then adding 70% to 30% we might say with a straight face that our clinical experience as psychotherapists proves that 100% of schizophrenes had such mothers.

It is not difficult to concoct reasonably good quantitative tests of these alternatives, although some of them involve quite a bit of effort and it's probably not worth the trouble in today's climate. Example: Our second misleading factor is that a normal mother is simply reacting to the troublesome features of a schizotypal child. On this hypothesis, while a good measure of maternal attitudes and child-rearing practices would show significant differences between the mothers of subsequent schizophrenes and mothers of normal controls, we might expect to find similar sizeable differences in the attitudes of mothers of brain-damaged children whose pathology arises from birth injury and the like, over which the mother had negligible influence; at least one study found that to be the case. Of course that doesn't prove the negative, but it does mean that any investigation of this question by quantifying maternal attitudes has to include other kinds of pathological children in addition to normals, as controls. For example, suppose we have developed

sufficiently powerful psychophysiological and soft neurology indicators of the schizogene that we can identify which of a proband's siblings are schizotypes. (It obviously won't do on any sophisticated theory, not just mine, to classify those siblings who are not diagnosably schizophrenic as normal, since the decompensation rate is, on a dominant gene theory, as low as 10%.) We could compare the proband's, the compensated siblings', and normal siblings' retrospections of mother's behavior, both toward them and the preschizophrenic proband, by content analysis of interviews or perhaps even by a sufficiently sensitive and reliable memory-based questionnaire.

Now let's look at the other side of the coin. Biotropes tend to assert that we *now know* that maternal attitudes and child-rearing patterns have zero influence on schizotypal decompensation probability. Being a psychologist, I cannot believe this, on extrapolative theoretical grounds. We have here an interesting methodological problem involving a principle the logicians call "the Total Evidence Rule" for empirical questions. I emphasize that it is a rule of inductive logic, not a particular (debatable) philosophy of science position. It is a principle that I find social scientists sometimes tend to ignore. Beginning with the admitted fact that the concordance even for MZ twins is only around 50 to 60% (depending on Weinberg adjustment and other sample differences), *something* environmental must account for the difference between those who become schizophrenic and those who remain compensated. If we don't believe it's a virus or sunspots or witchcraft, what on earth might it be? One can divide the environmental influences, without being able to specify them individually or even in narrow subcategories, into two broad classes: (1) environmental stressors on the adult as precipitators of illness, and (2) environmental shapers in the childhood developmental period that yield an adult schizotypal organization more or less susceptible to stress. I cannot think of any good theoretical reason, nor am I aware of any hard quantitative data, that would lead me to prefer current stressors over childhood shapers, or the other way around. It has not been easy to show an effect of current stressors as precipitators for schizophrenia, and that debate continues, just as it does for adult precipitators of depression, a more likely candiate for such influence. But the Total Evidence Rule does not permit me to reason as if I *had no other information* about how the human mind is shaped by experiences. Merely because I am talking about schizophrenia, a mental disease, I cannot elect to ignore any general information we have about how persons become the kinds of adults that they are. From common sense and everyday observation, not to mention literally thousands of research studies by developmental psychologists and others, we know that adult personality is influenced in a variety of ways, sometimes greatly and other times slightly, by the threefold influences of one's childhood, "precept, example, and reward," as our grandparents would have called them. Children raised in German households do not grow up speaking French. Catholics usually come from Catholic households.

People's table manners, their political and religious ideas, their notions of what is a normal kind of family relationship, their ways of handling forbidden wishes, whether of a sexual or aggressive nature, their expectations of other human beings' trustworthiness, their altruism or egotism, their avocational interests, their degree of sociability, their way of expressing aggression or countering it—these and hundreds of other aspects are influenced by identification or modeling, by differential reinforcement, and even, although less than it used to be supposed, by verbal instruction as to "how one ought to be." The notion that because we have moved into the domain of psychopathology, all of what we know about social learning goes by the board, just doesn't count anymore, seems to me preposterous on the face of it. To take a simple example, while schizophrenia is a genetically determined neurological disease, it has a *content,* and that content is socially learned. As Bleuler says, "You can't have a delusion about Jesuits if you never heard of Jesuits!" It would be strange to say that while we can infer directly from the MZ twin concordance data that whether you become schizophrenic or not, given the schizotaxic genotype, depends upon nongenetic factors, yet those nongenetic factors do not include *anything* about the way the primary nurturing figure treated you during the early stages of development. I can't believe that as a psychologist, and it's going to take a tremendous mass of powerful, clearly interpretable, and consistent data of a negative sort to persuade me of any such strange doctrine. I shall listen open-mindedly to such negative evidence, but I am going to require that it make a very strong case before I buy it. So, for example, the fact that my colleague Heston's adoptees (Heston, 1966, 1970) show a Weinberg-adjusted lifetime risk not different from that of cases reared by a schizophrenic mother is not a strong refuter, when one computes the confidence belt for a proportion based on $N = 47$.

The very imperfect MZ concordance is puzzling given the sparsity and feebleness of environmental factors and presents a major conceptual problem. This does not depend upon whether one is betting on a major locus or a polygenic theory. I suggest that it becomes somewhat less puzzling if we think about the psychodynamics of an individual from an idiographic viewpoint, given Freud with a big dash of Allport, and some Murray in between. Efforts to find environmental correlates of higher risk have quite understandably focused on what we might call "generic factors," such as infantile illness or injury, school experience, social class of parent, and the like. On my view of the disorder, I would assign a crucial role to the occurrence of certain sorts of adverse events, some of which are essentially independent of the personal characteristics and social status or other demographic features of the individual, literally matters of "bad luck" or "pure chance," that are sequenced, highly autocatalytic, and represent extreme examples of divergent causality (London, 1946; Meehl, 1978, p. 809). Something takes place that was not even unconsciously produced or rendered probable by the behavior of the schizo-

typal individual, a pure happenstance that altered his momentary psychological state and hence his readiness to misperceive along certain lines; then something else takes place, equally independent, in both the causal and statistical senses, of the first event, that impacts him because of an idiographic significance *that it would not have for another equally schizoid person*, and that it would not even have for this individual, despite its special idiographic content, if he were not in the state he's currently in. Since such events and states as social misperception and social anxiety are two-way autocatalytic (the more you distort, the more frightened you become, and the more frightened you become, the more you tend to distort), it doesn't take any fancy, implausible ad hockery to understand how one member of a MZ twin pair may fall ill and another one remain well despite sharing not only the specific schizogene but also all of the potentiating polygenes. Sometimes schizophrenic decompensation is the absorbing state of a random walk (Meehl, 1978, pp. 811-812). *The conventional social science search for the standard familiar generic variables will not elucidate such processes*. To exemplify this, I cannot do better than quote a previous paper of mine.

> Consequently we should set our switches in advance not to be completely baffled by a paradox: Failure to find positive environmental correlates of decompensation, despite the clearly established power of environmental influences collectively as shown by the genetic statistics themselves. I don't think it will be too hard for a genetically oriented psychotherapist to make sense of these facts, scientifically frustrating though they would be. Neither a rough measure of familial puritanism, nor a measure of a schizotypal mother's differential seductiveness as between two MZ twin boys, covers the possibility of a critical event such as Twin *A* receiving a completely unexpected low grade in his physical education class (due—let's really run it into the ground—to a clerical error!) several months following his first heterosexual experience. It doesn't take much for a schizotypal mind to connect up these two happenings in some sort of crazy, hypochondriacal, and guilt-ridden fashion. *Without anything else* being "systematically" different between him and his MZ co-twin, his aberrated CNS may take it from there and snowball it into a psychosis that appears, say, a year later. I do not myself find this kind of "happenstance" at all implausible, and therefore I was pleased to see our authors' explicit emphasis upon "chance" factors in their discussion of the environment. The causal model for an integrated theory of schizophrenia would surely involve social feedback loops, autocatalytic processes, and powerful critical episodes initiating chains of divergent causality, perhaps the most important of this third kind of causal meaning of interpersonal events *that may have the same sort of "average value," parametrically speaking, for both members of a discordant MZ pair.* Thus, for example, 6 months after our hypothetical Twin *A* in the preceding example has schizotypically concluded that "sexual drainage" caused him to fail the gym course, both twins are present at a dinner table conversation. The father says, innocently and with no notion of his critical schizophrenogenic role, "If there is anything I cannot stand, it is a boy who is a sissy." Now the schizotypal snowball really gets

> going for Twin *A,* whereas father's casual remark has negligible psychological significance for co-twin *B* (who has neither failed in gym nor visited a prostitute). I find it hard to think of any kind of statistical analysis of case history material or neighborhood characteristics that would tease out this kind of thing. And if one combines these "chance" factors with the possibility (some would say likelihood) that the schizogene(s) can "switch on and off" as a function of intercurrent biochemical states, quite possibly including states induced by momentary stressors, the elements of "psychological-social coincidence" can loom very large indeed. Every psychotherapist who has treated schizophrenics knows that the patients themselves sometimes connect a momentary resurgence of anxiety or confusion with what would to a normal mind be a very minor happenstance; and while I certainly do not wish to rely heavily on these anecdotal connections (see *supra*), neither would I be willing to dismiss them as of no evidential weight. Following close upon father's "sissy" remark, random episode E_1 (a waitress momentarily ignores him in favor of a customer that she knows well) ticks off in our Twin *A* a 2-hour increase in the blood level of norepinephrine. Due to his oddball dietary obsessions, which in turn went back to his reading a pamphlet (which didn't *happen* to fall into co-twin *B's* hands) at age 14, he also is running an unusually high level of organic acid X at the time. These concurrent alterations in the intracellular milieu of the schizogene, and note that they are neither physiologically nor psychologically related, nor attributable to any *systematic* characteristics of the environment, "switch on" the cerebral schizogenes and as a result the patient undergoes an increase in his pan-anxiety, his anhedonia, and his tendency to cognitive slippage. The last straw: While he is in this state, which is a deviation from his usual schizotypal norm, his girlfriend breaks a date with him, speaking rather roughly on the telephone (because she is embarrassed, and in order not to feel defensive she becomes aggressive). *Result:* Snowballing in the aversive direction, dangerously consolidating the mixed-up schizoid complex: "I am bad and weak because being sexually drained I have become a sissy as my father said, which is why waitresses prefer others to me, as does my girlfriend, and hence all women. I'm a hopeless nothing." Twin *A* is now well on the way to clinical decompensation. (Meehl, 1972a, pp. 404-406)

I must emphasize that envisioning these kinds of "idiographic unpredictabilities" and even after-the-fact "unexplainabilities"—not quite the same thing, as Scriven and others have convincingly shown—does not require that we postulate any sort of radical biological or psychological indeterminism. We just have to recognize unblinkingly the rather obvious fact that even a so-called "thorough, in-depth" life history is extremely deficient as regards details, and that we have nothing to guarantee that this deficiency could be made up by *any* amount of ingenuity or expenditure on the part of an investigator concerned to reconstruct the past. The kinds of "influences," "variables," and "factors" that social scientists are usually able to assess are almost always, when carefully scrutinized, classes of variables or kinds of episodes. In terms of learning theory, we deal in most psychological and sociological research with parameters of the elaborate "social Skinner box"

that constitute a person's nurturing environment and adult life. The numbers we can correlate with outcome (e.g., psychosis) are in the nature of average values; they do not point to what may be critical events but rather to stochastic features of discriminative and eliciting stimuli or schedules of reinforcement contingencies.

If I were to present an engineer or physicist with a "molar" problem involving a chair made out of a specific kind of metal, riveted or welded in a specified way, telling him I was going to catapult it from the roof of the physics building at 12:32 P.M., in a direction so it would land on the stone steps of the administration building, and require him to predict whether and how it would fracture, he would probably decline the invitation. A fortiori, he would decline if the object to be catapulted were a century-old oak-case grandfather clock! But that, brethren, is about the situation when we are trying to understand why Twin *A* became schizophrenic and MZ Twin *B* did not.

I may conclude this discussion of "chance" factors determining which twin decompensates by quoting from my paper read at the 1971 MIT Conference on Prospects for Schizophrenia Research, where I said:

> But that there are *some* schizophrenics who act more like *some* anxiety-neurotics than they do like *other* schizophrenics is an important fact to think about and to explain. It is, however, very weak evidence against anybody's theory of schizophrenia, genetic or otherwise. I understand from the mathematicians that there is a clear negative answer to this question; and it is found, for example, in the theory of certain stochastic processes such as random walks. Whether a particle pursuing a random walk ends up (after a specific finite time) in a so-called "absorbing state" [= a state that it cannot leave] is a "dichotomous end-result" comparable to whether a schizotype ends up in a state hospital, enters and leaves, or wins the Pulitzer Prize for poetry; but the mathematics of such situations teaches us that it would be a mistake to presume a dichotomous etiological basis for this important outcome difference. In that connection, I mention in passing that we should not assume, even in the case of MZ twins, that a clinical discordance must always have some "big" life-history factor discernible. That Twin *A* becomes schizophrenic and Twin *B* remains "healthy" may be a random walk problem—I myself would bet that *many* of them are. (Meehl, 1972a)

Some of my biologically oriented psychiatry colleagues don't like examples like this; they ask me how anybody could go about checking on such idiographic hypotheses in a scientific way. All I can say is that this is a problem with the usual psychiatric case history that has been with us for many years and that we have become accustomed to it. But that doesn't mean we should ignore its highly problematic status. The usual psychiatric case history that talks about the patient's parental home in a superficial way and mentions that a relative died of a brain tumor and when the patient flunked a certain course in school is simply not capable of getting at the kind of thing

hypothesized in my preceding example. I do not look upon it as a defect at a conjecture aimed at accounting for such a big paradox as that between the moderate MZ concordance and the relative absence of strong environmental correlates of illness that one may have great difficulty unscrambling it. Even a Popperian, while insisting that a theoretical conjecture should be testable (although not necessarily at this time in this state of the art), does not require that a conjecture concerning a *particular* event should always be testable after it has happened. Thus, for example, we have a rather mild windstorm that does very little damage anywhere in the vicinity except that farmer Jones's silo fell down. We know enough about the general propeties of silos and the laws of mechanics and the properties of winds to come up with some plausible explanations of what might have been the cause for this particular silo's falling down in such a mild storm. But it will usually be the case that we are unable to reconstruct the causal chain from the remnants well enough to decide among these competing alternatives.

The ordinary psychiatric history, or even one filled out by a psychotherapist on the basis of extensive contact and psychoanalytic kind of anamnesis, could be analogized to the following example for an animal psychologist: You are told that Rat R came from a bright strain of the Wistar stock, lived in the Minnesota lab for 3 months, and then was sold to the Kansas lab. This rat received an adequate amount of bran mash both places, but also vitamins in Minnesota. There are three litter mates, two living and well; one has an ear infection and shows equilibrium disturbances. Rat R has been subjected to considerable experimentation in three apparatuses, two of which are the T-maze and the Skinner box, the third unknown. The exact experimental regime is undetermined, except that the experimenter is known to be interested in problems of discrimination learning. At the present time, this rat seems unable to form a simple form discrimination and also shows face washing, defecation, and excitable behavior generally when placed in a Skinner box with the light on. Query: What exactly is the trouble with Rat R, and how did it originate? I would be inclined to take the position of Skinner when he is fending off complaints about applying certain principles in nonexperimental contexts, namely, that you attempt to corroborate regions of the theoretical network by experimental or, lacking that, statistical research, but you do not require that the scientist, in offering a plausible explanation of a complicated particular event, should always be able, so to speak, to "prove the whole theory over again" afresh. Of course, all of these considerations become rather pointless if what occurs is a more or less random turning on and off of the schizogene, analogous to what we assume takes place in the major affective disorders.

Suppose the essential features of my theory were to be corroborated by multiple lines of taxometric investigation. What then would be the clinical situation? For one thing, on such a theory one does not anticipate persistence of long-term outcome as a highly valid indicator of the taxon, certainly not the

quasi-pathognomonic role it played with Kraepelin and still does with some old-fashioned clinicians. On the other hand, one might anticipate a predictive value for early life history data. On my theory, garden-variety social introversion, a nontaxonic factor distinct from schizoid autism and known to be highly heritable, is a strong potentiator of decompensation in the adult schizotype. It might also be a predictor of adverse long-term outcome, *but not because it is anything specific to schizophrenia,* just as in our competitve extrovert culture I daresay extreme social introversion potentiates low IQ as a predictor of economic failure, but that doesn't tell us anything about the relationship between the polygenic introversion variable and the heritable component of *g*. Those who don't understand my theory expect me to say that the severity of a soft neurological or psychophysiological indicator of the schizoid taxon should be predictive of outcome, and they are puzzled when I say not at all, and that I would be disappointed if that should be the case. The confusion arises from a failure to realize that one must think differently about taxometrics than about factor analysis or multidimensional scaling of continuous variables. Take, for instance, the SPEM (Smooth Pursuit Eye-Movement) psychophysiological aberration, which has been shown in several laboratories to be a powerful indicator of schizotaxia, possibly having a valid positive rate pushing 90% at the expense of negligible false-positives (provided one excludes remitted manic depressives who are on lithium). This test remains positive on schizophrenes in remission and has been shown to have a fairly high incidence in their first-degree relatives. Since all such "integrative" functions are good bets on my theory, I expect this one to become shortly a part of our diagnostic armamentarium. The parameter distributions of these eye movement curves are bimodal, but there are still individual differences within the pathological group (Iacono, 1988).

Why should one expect, on a major locus genetic model, the quantified SPEM defect to be correlated with clinical severity or malignant course? All one can say taxometrically is that the higher the deviant score, the more *confidence* one can have in the taxonic classification of the individual. But this is totally different from the notion that whatever polygenic or environmental variables produce individual differences within the taxon (as also among normals) should be correlated with severity of the neural integrative defect. It's a different way to think, and one has to get used to it.

Given the MZ concordance of 55% or less, one expects to find some strong environmental influences determining which schizotypal twin falls ill. With rare exceptions (e.g., seasonality, birth weight, maternal exposure to influenza), efforts to identify such generic potentiating factors have not turned up much. How is this possible, given the sizeable MZ discordance? *Something* must be making the difference, so where could it be if not "in the environment"? Two possibilities suggest themselves. It may be that the schizogene turns on and off randomly, or at least not in response to the kind of environ-

mental factor we usually study in social science. It may, for example, respond to a minor alteration of diet occurring in hot weather—hardly likely to be found in the typical psychologist's or sociologist's candidate list of "environmental influences." Examples are known in medical genetics. Speaking against this hypothesis is the fact that schizophrenes in remission show psychophysiological and soft neurological aberrations, as do many of their first-degree "normal" relatives, children at genetic risk, and preschizophrenic children (Fish, 1975, 1977, 1984; Freedman, Adler, Baker, Waldo, & Mizner, 1987; Heinrichs & Buchanan, 1988; Iacono, 1988). A gene turn-on theory cannot handle this, unless we ad hoc it by postulating a *second* major gene that's responsible for producing episodes of schizotypal decompensation. The other explanation, which I think more plausible, is that a series of adverse events occurring close in time and idiosyncratic in their psychodynamic import *just happens* to befall the unlucky twin (Meehl, 1962a, 1972a, 1972c). He's the victim not of such standard social science systematic factors as rejecting mother, bad school, infectious disease, social class, or whatever, but simply of the binomial theorem.

METATHEORY OF OPEN CONCEPTS

What do I mean in speaking of schizotaxia as an *open concept*? While this terminology was introduced by the late philosopher of science Arthur Pap (1953) in 1951, the concept is older, having been formulated by Rudolf Carnap in his classic paper on testability (Carnap, 1936-1937/1953). The theoretical entity named by an open concept term is defined implicitly or contextually; in a developed discipline like genetics or chemistry, by the mathematical formalism and an interpretative text. But the interpretative statements in that text are rarely explicit definitions and almost never "operational" definitions in the conventional sense. The nomological network consisting of the words and the math (and maybe some diagrams or Tinkertoy models) *jointly* (1) define the theoretical terms and (2) make assertions about the theoretical entities' causal and compositional relations, a heretical thesis propounded by Cronbach and myself in 1955 that I gather is now accepted by most knowledgeable psychologists.

A beautiful example of an open concept in the life sciences is the gene. It was contextually defined until Crick and Watson made the theoretical breakthrough that enables us now to define it explicitly as a *cistron* consisting of a certain sequence of *codons.* This is not an operational definition but an explicit definition *in other theoretical terms*, in this case terms of organic chemistry.

This is not the place, even were it within my technical competence in philosophy of science, to present a detailed explication of the metatheory of open concepts as it has been worked out by logicians and philosophers of

science over the past half century since Carnap's classic paper (but those who are interested may see Meehl, 1972c, p. 21, references). Essentially, the advance made over a simplistic operationism about *concepts* (and a corresponding strict verifiability criterion of scientific meaning for theoretical *statements*) was the recognition that many scientific concepts are not originally introduced by explicit definitions, stating the necessary and sufficient conditions for a concept's applicability to a particular, but rather by a collection of statements (in a logical form called by Carnap "reduction pairs") that represent different observational avenues to deciding on the applicability of a concept. Which one of these is chosen will depend upon the context, and it may sometimes not be possible to use one, so we use another. Thus, for instance, whether an electric current is flowing in a wire is sometimes decided by observing whether there is a deflection of a compass needle in its vicinity; on another occasion we ascertain whether when the wire is cut and the ends placed in a suitable solution, one gets a deposit of, say, silver at the cathode; and on still another occasion, we determine whether it will heat a filament. We do not explicitly define the concept "electric current" by conjoining these three indicators. That is, we do not say, "An electric current *is* (or *consists of*)…," but we say that it is appropriate to assert that a current is flowing when one of these test conditions is applied and the specified test result is observed. The fact that this is not an explicit definition of the full semantic content of the term "electric current" and that the list of indicators is indefinitely extensible is the first kind of openness that was expounded by Carnap in his paper and elaborated by Pap (1953, 1958). In addition to the extensibility of the indicator list, there is a second kind of openness that arises when the theoretician makes a weaker claim in that the test observation relevant to decision is only probabilistic, so that observing or not observing the test result in the test condition raises or lowers the odds but does not definitively prove or refute the attribution. When this softened form is used, as is almost always the case in the life sciences and sometimes even in the inorganic sciences, there is no longer a strict deduction running from the result of one of the indicator tests to another, as there was in the original formulation where probability is not involved. So even with a stipulated list of indicators that we have for some reason decided not to expand further, there is an element of looseness, especially as applied to individual patterns of indicators where some are present and some are absent, the typical situation in making a diagnosis in medicine or psychopathology. Finally, a kind of openness that is not discussed by Carnap and Pap but is of great importance in studying the historical development of a discipline is that the long-term aim of much research is to be able to offer an explicit definition of the theoretical entities that in the early stages are defined implicitly or "contextually" by their role in this network of connections among observables. Thus, for instance, from Mendel until Crick and Watson, the term "gene" was defined implicitly by reference to its role in a statistical system that has an

input side (ancestry) and an output side (phenotypic traits). Prior to Crick and Watson's discovery, one could not say in explicit terms just *what the gene is;* one only said it was a something, presumably of a physical nature, that had a location (shown by linkage statistics) at a certain place on the chromosome, and that it was the causal ancestor of a chain of events in the development leading to various phenotypic manifestations. In drawing a diagram to illustrate the nomological network metaphor, where one connects the concepts represented by open circles with lines or strands of the net, where the nodes of the net are the theoretical entities and the strands of the net are their functional or compositional relations, I was struck by an analogy between those open circles and the vacuous eyes that used to appear in the Little Orphan Annie cartoons of my youth. So I dubbed this third kind of openness "Orphan Annie's Eyes," meaning that one of our long-term scientific aims is to learn enough about the *innards* of an implicitly defined theoretical entity—what parts or substances it is composed of, and how they are put together—so that while still using the contextual definition for many purposes (especially technological ones), we can also provide an explicit theoretical definition of the entity in terms of its inner nature (Meehl, 1977; Meehl & Golden, 1982).

When we offer a list of symptoms, signs, character traits, life history events, and psychometric scores as relevant to diagnosing schizotaxia, the imperfection and extensibility of that list represent openness of the first kind. When we go on to say that no one of these indicators is two-way pathognomonic (even the thought disorder is not quite that), we are referring to openness of the second kind, the stochastic as contrasted with the nomological character of the strands connecting the nodes of the theoretical net. Suppose someone then asks, "Well, all right, I understand that you don't offer a strictly operational definition that would enable us to tell for certain whether a particular individual is or is not a schizotype, and that you don't expect to be able to do that until research is farther along than it is today; but surely you must have some sort of theoretical conjecture as to just what this schizotaxic defect consists of in its own nature?"

One might justifiably decline to answer that on the grounds that it is premature and speculative. In thinking about my theory of schizophrenia, I am a Popperian and not shy about conjectures, as long as I have some notions about how you might ultimately go about testing them, even if you can't do so today. When one even says something as vague as "a genetically determined neural integrative defect," despite the vagueness of that language, it is the beginnings of filling in Orphan Annie's Eyes. It excludes, for instance, the notion that schizotypes have the same CNS parameters as normal people but have merely acquired some undesirable ways of behaving and experiencing through aversive social conditionings. And when one states further that, whatever the inner nature of that integrative defect may be, it is the specific defect that predisposes to schizophrenia rather than, say, to dyslexia or stam-

mering or tone deafness or the hereditary kind of spelling defect, he has further narrowed down the concept. One must realize that various degrees of filling in Orphan Annie's Eyes can exist, depending on the theorist's speculative daring, all within the contextual framework given by the *implicit* mode of definition. A Minnesota colleague who goes along with my conjectures that schizophrenia represents the decompensation of the schizotypal personality, that the schizotypal personality is the personality that develops by the process of social learning on a schizotaxic brain, and that the schizotaxic brain is characterized by a specific kind of neural integrative defect that is hereditary, may or may not go on to conjecture with me what the inner neurophysiological nature of that integrative defect is. So two theorists may agree about the schizotaxia concept as an open concept specified by its role in the network but may disagree when it comes to closing the third kind of openness by different ways of filling in Orphan Annie's Eyes. My theory starts at the molar level with the open concept of schizotaxia, defined implicitly by its role in a loose and highly incomplete nomological network (clumsy but more accurately, "stochastological network," in Meehl, 1978). The defining contextual strands include the soft neurology and psychophysiology of schizophrenia, with a weak assist from other "organic" correlates (see, e.g., Shattock, 1950); the admittedly variable but, taken as a whole, convincing "cognitive" aberrations (Knight, 1984); and the presence of these phenomena in relatives, which combined with the clearly genetic etiology leads us to attach the adjective "heritable" to the phrase "neural integrative defect" in defining schizotaxia. So at this level we have a syndrome plus a partial, but specific, etiology. Orphan Annie's Eyes are nearly empty, except perhaps for the semantic overtones of "neural" together with "soft neurological," which suggest a "promissory note" about future discovery—something aberrant about how the brain works that is not merely unfortunate *learned content.* Then comes the conjecture that the integrative defect is ubiquitous, something haywire throughout the CNS, a functional parameter deviant for every neuron, not something only in the limbic system, the RAS, or the proprioceptive system. Next we identify this functional parameter aberration with reduced signal selectivity (hypokrisia) at the synapse. Finally we conjecture that the hypokrisia is due to a biochemical deficiency of a substance whose function in the orthotaxic brain is to resist spread of depolarization of the cell membrane, and that a dominant mutation is responsible.

The methodological point is that at each stage of this progressive conjectural filling in of Orphan Annie's Eyes, a theoretician may call a halt and say that he will go along with me so far but no farther. His dealing with the first two aspects of openness will be unaffected by this disagreement as to the conjectured filling in of Orphan Annie's Eyes. I should emphasize, finally, that in the theory of open concepts, philosophers of science have not meant to suggest, certainly not since Pap's elaboration (1958) of the Carnap paper, that

such openness is only characteristic of the "soft" sciences. A fair statement of the consensus is from a remark made to me in discussion by the late Imre Lakatos: "All concepts are open, it's just that some are opener than others!"

If you can't tolerate working with open concepts, you should study something else instead of psychopathology. The opposite mistake is to wallow in them, a distressing tendency of our age's intellectual fecklessness and subjectivism. What you should do with an open concept is to recognize its openness and use appropriate methods, particularly statistical ones, to tighten it up, keeping in mind Aristotle's dictum in the *Ethics* that one should insist on precision in concepts insofar as the nature of the subject matter permits. I hope I am pursuing that line in my current thinking about taxometric research on the theory of schizophrenia. I might reassure you on this score by pointing out that if you are clever and lucky in your choice of taxometric indicators, moderately valid indicators can be combined in sets of a half dozen or so to yield diagnostic probabilities as attached to individuals in the high .90s. This is plenty good enough both for clinical work and for the testing of strong genetic models.

IMPLICATIONS FOR THE CLINICAL EXAMPLES

Where do these considerations leave us with respect to the clinical examples with which we began? The answer is obvious: They leave us up in the air as to the merits. Both parties to the dispute are wrong methodologically if they claim to settle the merits on present evidence. In our first patient, an attenuated waxy flexibility and faint semantic oddities do not rule in schizotypy, let alone schizophrenia, and his previously adequate life adjustment does not rule it out. Ditto the pan-anxiety, introversion, passivity, and dependency of our second patient. The pro-schizotypal diagnosticians who rely on the follow-up statistics of Hoch and Polatin's cases are making too much of the 20% schizophrenic outcome; those who emphasize the majority that do not develop subsequent florid schizophrenia are making too much of that fact. It's a free country, and everyone is entitled to bet on his own horse. As Mark Twain says, "A difference of opinion is the essence of a horse race." I am not criticizing those who disagreed or those who agreed with me for placing clinical bets, as long as we all realize that the assignment of relative weights to life history factors and the defense of such an entity as the Hoch-Polatin syndrome on the basis of long-term outcome are at present subjective judgments and will remain so until more powerful indicators of the schizotypal taxon are available. In our clinic we have begun (not consistently, alas, but frequently) to run some of the

soft neurology tests, especially the SPEM, on patients in whom there is this kind of diagnostic disagreement in case conference. It is only by a certain loosenesss that we refer to a "pathognomonic" sign or symptom, even when looking to the future. Proof in empirical knowledge is, as the logician reminds us, a matter of degree. All knowledge of empirical matters is only probable, although there are sometimes such mountains of tightly interconnected evidence that we find it impossible *psychologically* to doubt it. We do not countenance the conjecture that the sun is Apollo's chariot or a gigantic red-hot cannonball, but in theory even such quasi-certain knowledge as we have that the sun is a ball of hot hydrogen is open to revision.

THE SUPER BOOTSTRAPS THEOREM

We do, nevertheless, speak of certain signs, symptoms, or biochemical tests in organic medicine as pathognomonic. There is an interesting epistemological question here, namely, how can we get to quasi-certainty, which is what is claimed for a two-way pathognomonic sign, starting out with fallible indicators of an open concept? That deserves a lecture by itself, but the short answer is that we conduct a successful "bootstraps" operation on a set of fallible indicators, and we often conclude, as knowledge advances, by changing the relative weights, as in some of the symptoms (e.g., expansive delusions) of general paresis. (Actually, Noguchi and Moore did not find the spirochete in the brain of every single paretic they studied, although some clinicians think that.) Years ago, I proved a paradoxical theorem in my early work on taxometrics, which I deliberately christened with a challenging name: the Super Bootstraps Theorem (Dawes & Meehl, 1966; Meehl, 1965, p. 37 ff.; 1973c, pp. 216-217; Meehl & Golden, 1982, pp. 143-144). Suppose we have three fallible quantitative indicators of a conjectural latent taxon such as psychopathy or schizotaxia or cyclothymia. Applying certain coherency or consistency tests to the internal statistics of these three indicators may justify entertaining, until further notice, a high confidence that the latent structure meets certain mathematical conditions (which I will not here discuss), such as relative independence of the indicators within the inferred latent categories. Given satisfaction of our consistency tests, we believe we have a fairly accurate estimate of the taxon base rate in a specified population. We infer, for instance, that 30% of the patients in a general psychiatric outpatient clientele are schizotypes, even if they present with pseudoneurotic or pseudopsychopathic symptoms, traits, or signs. Then one can optimize a cutting score on each indicator and can compute the inverse probability, for any given sign pattern, of taxon membership for each subject. The optimal cut having led to three dichotomous signs, we have thereby defined eight pattern "cells," for each of which a taxon probability is computable. The point is that while these

must be fairly valid in order to warrant the whole business, they need not be highly valid or anywhere near pathognomonic, *either singly or collectively*. We do not know, even for sign patterns all three plus or all three minus, which *individual* is a taxon member; and for weaker patterns, such as $x^+ y^+ z^-$, we have only moderate betting odds for each individual. Nevertheless, for each cell we can infer with a small error tolerance what the *proportion* of taxon members in that cell must be. What I showed in the Super Bootstraps Theorem (see Appendix) is that if we now consider a fourth quantitative indicator v, which Omniscient Jones knows has two distributions with zero overlap, we can try various cutting scores on v, each defining a dichotomous sign v^+/v^-, and can count the proportion of individuals per cell that is v^+. We then compute the discrepancy between the v^+ rate per cell and that cell's Bayes's Theorem taxon rate, computed on the three fallible signs. What the theorem shows is that it is impossible to match these frequencies over the eight cells (for that matter, over even two cells, but the more the better!) unless the optimal cut on v which achieves that matching is infallible.

This strange result is nothing more than high school algebra and formalizes what physicians have been doing for centuries when they altered their subjective nonstatistical "clinical" weights on the basis of further experience. That's the kind of procedure that leads us to find a genetic marker for a hereditary disorder where we don't know the biochemistry, let alone the chromosomal locus. So it is not fanciful to imagine that within the next 10 years, even if no lucky or clever biochemist finds the "purple spot" for schizotaxia in paper chromatography, that several psychophysiological and soft neurological indicators will be found, via the Super Bootstraps Theorem, to be quasi-pathognomonic. When that happens—I say *when* and not *if*, advisedly—we should be prepared to find patients presenting with a wide spectrum of social and psychological traits and symptoms, despite their genetic homogeneity as revealed by these nonbehavioral markers. Personally I think to say that this will happen within 10 years is being pessimistic; I would guesstimate 5. At that point one will know with some statistical precision what role such life history facts as age of onset, number and duration of episodes, and Kraepelin's malignant final outcome should be assigned.

There is a theoretical reason for expecting long-term course and outcome to have a privileged indicator status. If the defining pathology-cum-etiology is a fine structure brain condition of genetic origin, we do not expect it to change. If biochemical, that depends on whether we have an off-and-on setup or a steady state modifiable (if at all) by a specific therapeutic intervention. If Thorazine acts mainly as an antipsychotic, ameliorating Bleuler's accessory symptoms but leaving the soft neurology relatively untouched, we expect to find signs of the latter in first-degree relatives who have never become schizophrenic, and we do. This indicates a perdurable "something haywire in the brain" that persists despite fluctuations in clinical status, whatever complex of

causal factors superimposed on the schizotaxic neural integrative defect is responsible for decompensations and remissions. But while these considerations may heighten the prior (thinking Bayesian) for giving a high diagnostic weight to outcome, leading to our being more easygoing in criticizing research along those lines, ultimately the matter is empirical. When enough replicable findings are in, course and outcome will be given whatever indicator weight the taxometric bootstrapping assigns to them. Whether, or to what extent, very strong theoretical arguments, including content validity, should be allowed to countervail the taxometrics is a difficult question I shall not discuss.

BIOTROPES AND SOCIOTROPES

Although I am a practitioner of psychoanalytic and rational-emotive therapy, I have said nothing about psychodynamics, which may seem unbefitting a book honoring Henry A. Murray. But that is not because I dismiss the psychisms at work in schizophrenia; rather, it's that I take them for granted. You see, we Minnesota clinicians firmly resist classification as biotropes *versus* sociotropes, an easy conventional dichotomy we consider simplistic. "The mind is the brain functioning" is for some psychologists a mere cliché, whereas we take it seriously. The theory of schizotaxia as an inherited neurological defect does not contradict a single psychodynamic truth about schizophrenia. Psychodynamics is concerned with learned social and intrapsychic *content,* with perceptual, cognitive, affective, and motivational states and events. They are molar level entities—psychisms—and the dispositions to these psychisms are acquired and activated by life experiences, especially with other persons. The mathematical parameters of the several acquisition and activation functions involved are deviant in the schizotaxic brain, and this constitutes the specific etiology of schizophrenia. But we do not liquidate molar level explanations in favor of brain language. It was a mistake in the 1930-1960 period to ignore the soft neurology or explain it away as due to anxiety or withdrawal. It would be a similar mistake today for biotropes to explain catatonia in purely neurological terms, which Bleuler easily refuted in his masterly *Theory of Schizophrenic Negativism* (1912). Catatonic phenomena are purposive, as when we understand such "opposite" behaviors as negativism and command automatism or mutism and echolalia, as the patient's fluctuating modes of disengagement from his social surround. Similarly, the delusion of the end of the world, sometimes found in the early stages of schizophrenic decompensation, can be interpreted as the symbolic and intellectualized expression of the patient's realization that he is undergoing a withdrawal of cathexis from the internal representations of social objects. The anhedonia, Rado's pain-dependent pleasure, and Bleuler's cardinal trait of ambivalence I subsume under the general heading of ambivalence combined with aversive drift, which is an inter-

mediate level psychism. But I can't derive aversive drift in psychodynamic terms; rather, I move down to a neurological level of explanation in terms of (+) and (–) reinforcement centers in the brain. Again, while the uniquely schizophrenic semantic slippage may depend for its *possibility* rather directly on synaptic slippage, *when* it occurs and with what psychic *content* involves the particular patient's psychodynamics. As Bleuler would say, there is a primary "associative loosening," but the resulting thought sequence is cata-thymically determined. It often seems that the genetic and physiological revolution in psychotherapy has led some biotropes to disbelieve in the reality of mind, but I assure you that I am not among them.

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Appendix

Proof of Super Bootstraps Theorem

For a given cut on *v,* defining a sign *v*(+), let

$p_i\,(+)$ = Observed *v*(+) probability among cases in cell *i*,

$p_j\,(+)$ = Observed *v*(+) probability among cases in cell *j*.

Inferred from patterns of fallible indicators (*x, y, z*) are latent taxon-rates in cells *i* and *j*,

p_i = Latent taxon-rate in cell *i*
p_j = Latent taxon-rate in cell *j*
Let p_t = Valid *v*(+) rate in taxon class
p_c = False *v*(+) rate in complement class

Then, expressing the matching of latent taxon-rate with observed *v*(+) rate in latent terms,

$$p_i\,(+) = p_i p_t + q_i p_c = p_i$$
$$p_j\,(+) = p_j p_t + q_j p_c = p_j$$

Subtracting,

$$\begin{aligned} p_i(+) - p_j(+) &= p_t\,(p_i - p_j) + p_c\,(q_i - q_j) \\ &= p_t\,(p_i - p_j) - p_c\,(\mathrm{p}_i - p_j) \\ &= (p_t - p_c)\,(p_i - p_j) = p_i - p_i \end{aligned}$$ Matched, by hypothesis

Dividing by $(p_i - p_j)$, which will rarely = 0,

$$p_t - p_c = 1$$ [misprinted as = 0 in original publication]

which is impossible unless $p_t = 1$, $p_c = 0$, that is, new indicator *v* is infallible when optimally cut.