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TOWARD AN INTEGRATED THEORY OF SCHIZOTAXIA, SCHIZOTYPY, AND SCHIZOPHRENIA

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METHODOLOGICAL PROLOGUE

Since Dr. Millon is inviting some experts to critique my theory in this issue, I can save time all around by setting out briefly (and therefore somewhat dogmatically) my philosophy of science or, as I would prefer to call it, "metatheory." The new version of philosophy of science in the younger generation differs from the logical positivism of my youth by being more empirical and historical, that is, it treats philosophy of science as the empirical theory of theories which, of course, includes rational reconstruction of the history of science. Despite remnants of positivism, I could be best described as a semi-Popperian or a Lakatosian. This means that I am not an

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The editor's invitation to update and elaborate my theory of schizotaxia, schizotypy, and schizophrenia specifically relieved me from attempting a review of the vast literature, and I have cited only work clearly relevant to the theory, attending to integration and accessibility rather than scholarly priority, when no injustice results. To make the presentation self-contained for the invited critics, considerable overlap was allowed with material in Meehl (1989b, 1990b), to which readers are referred for more detailed treatment of some topics (e.g., relation of diagnosis to course). Given the differing lengths, major focus, readership, availability, and opportunity for criticism of these different publications, this overlap was agreed upon antecedently by the editors involved, Freedman, Millon, Rabin, and Zucker.

inductivist, and do not require "support" for a conjecture, but merely that the conjecture be, in principle, capable of falsification by facts. Even this is too strong, because one may not be able to specify the conditions for testing at a given point in time. But one should at least be able to say what else we would have to know (e.g., auxiliary theories, or new measuring instruments) in order to make the theory testable. So when people ask me, "Where do you get those ideas about schizotaxia in your theory?" my standard answer is, "I make them up out of my head. Where do you get your theoretical ideas from?" Thus a critic who is a hard-boiled inductivist will not be relaxed about my conjectures; but he shouldn't waste his time pointing out to me that it is conjectural since, not sharing his philosophy of science, that doesn't bother me. We would be at cross purposes to argue about that. Like any Popperian, I want the tests, when available, to be strong tests. Hence I am not impressed with the mere showing of statistical significance between groups, except in certain special cases that I will illustrate below. I view the convergence of different lines of evidence, especially their convergence on a certain numerical value of a theoretical variable, as far more important in the history of science than exact tests of significance or even exact confidence intervals (Atkinson, Furlong, & Wampold, 1982; Bakan, 1966; Carver, 1978; Chow, 1988; Dar, 1987; Hedges, 1987; Lykken, 1968; Meehl, 1967, 1978, 1990a, 1990c; Morrison & Henkel, 1970; Rozeboom, 1960; Salmon, 1984; Serlin & Lapsley, 1985;).

I am a realist rather than a fictionist or instrumentalist, that is, I believe the aim of science is to find out the way the world really is; so that, rather than theories being a means to an end ("to predict and control" as the usual psychologist puts it), for me the facts are the means and the theory is the end. I therefore make the Popperian distinction between *verisimilitude* ("truth-likeness," closeness to the truth) and *corroboration* (the strength of the evidence for the theory, how many, and how stiff, empirical tests it has passed). Verisimilitude is ontological; corroboration is epistemological. Verisimilitude is the world as known to Omniscient Jones; corroboration refers to how we come to know whatever we do know.

In the life sciences I consider it appropriate to move from one level of analysis to another and therefore to mix levels of explanation. *Example*: It is not a methodological sin to explain the *content* of a schizophrenic hallucination psychodynamically, while recognizing that a person with those psychodynamics would not be a schizophrene and would not be prone to hallucinate if he had different genes and brain chemistry.

Accepting the consensus of contemporary logicians and philosophers of science, I am not an operationist in the sense that psychologists use the word, because I recognize that only a proper subset of theoretical terms in any science are tied directly to observational concepts, whereas the rest are related indirectly to data by sometimes long (and frequently rather tenuous) derivation chains. The theoretical concepts that are not operationally defined are defined "implicitly" or "contextually" by their role in the theoretical network. Since that network is incomplete, the direct or indirect tie of theoretical concepts to observables is an extensible list, which means that we deal with open concepts (Cronbach & Meehl, 1955; Meehl, 1972a, p. 21, 1973a, p. 195, 1986a, 1990b; Meehl & Golden, 1982; Pap, 1953, 1958, chap. 11, 1962, chap. 3). Lakatos once said to me, "Meehl, all concepts are open, it's just that some are opener than

others!" But the metatheory of open concepts is particularly important in fields like psychology and behavior genetics because the network of laws is extremely incomplete. A second source of openness is that the strands in the nomological network (the laws connecting the nodes, which are the concepts or theoretical entities) are themselves not strictly nomological but are statistical or probabilistic in nature (Meehl, 1978). Further, when concepts are defined implicitly by their position in the network, there is a third kind of openness that is present, even if one were to take the net to be complete and the strands of the net to be tight rather than loose, to wit, the answer to the question, "What is the inner nature of this entity?" Some psychologists reject that kind of question as unscientific or metaphysical, which is a mistake, since ascertaining the true inner nature, the composition and structure and inner workings of an entity, is frequently a major breakthrough in all sciences. Prior to the Crick and Watson discovery, the concept "gene" was defined contextually by a rich mathematical and cytological network, but the inner nature—chemical substance and structure—of the "gene" was unknown (Meehl, 1977). It follows from this view that I am not a behaviorist in any but the broadest sense of the term, namely, the way I know about other persons' mental events is from their speech and gestures, since I don't have direct access to their inner life. But that truism is hardly definitive of a special position today. I have no scruples about using mentalistic language, provided it is tied (however loosely) to behavioral dispositions or life history antecedents whose causal efficacy has been empirically corroborated to some degree. Methodological behaviorism of this sort simply says that mentalistic constructs should be behavior relevant, they should be at least probabilistically linked to behavioral dispositions or environmental events in the life history or current stimulus field. That is not the same as requiring that they should be *behavior equivalent*, completely reducible to behaviors or behavior dispositions (Meehl, 1973a, pp. vii–xxii). Thus I continue to subscribe to a distinction (McCorquodale & Meehl, 1948) between hypothetical constructs and intervening variables, and am quite contented to use both kinds in theorizing.

Finally, the explicit aim here is theoretical understanding, finding out about the way schizophrenia works if we can, rather than helping schizophrenic patients. There are many interventions, even in organic medicine, that are not theory based and that are helpful; and there are true theories which, at a given stage of technology, do not lead to prophylaxis or cure. One of course hopes that a theory will have useful effects, and one is justified in scientific work at the taxpayers' expense by their hope to that effect. But given that social role as ethically and politically valid, the scientist's aim, within the pursuit of theoretical science, is not instrumental.

Given those overall methodological points, there are some points specific to the strategy of schizophrenia theorizing that I must briefly mention. I think one should start with the big trends rather than with minor correlates and variations, except when those latter may have some special theoretical suggestiveness. As an example, consider the allegation that malocclusion, while clearly not pathognomonic either as an inclusion or exclusion test, has a somewhat higher statistical frequency among schizophrenes. That kind of thing, like the nailfold capillary anomaly, is important, although a

weak statistical trend, because of its interesting bearing on the pleiotropic gene influence, and because it may provide a hint as to the biochemical or developmental causal pathways.

There are some kinds of psychisms or behavioral connections which we know only a little about in terms of direct study of the brain, but which we are able to put in the CNS as part of a block diagram. I feel quite free in conjecturing about certain things that we know the brain somehow has to do, although of course if we have direct evidence (e.g., positive or negative reinforcement centers in the limbic system), that makes it all the better.

In the context of discovery (Reichenbach, 1938) I think it is appropriate to put some reliance on strong statements by seasoned, gifted clinicians. Anything that Bleuler (1911/1950) or Kraepelin (1909–1913/1971) says, I take with great seriousness. One must distinguish between the statements, "No quantitative evidence, using up-to-date statistical methods, *supports* this clinical impression of Bleuler's" and "We possess quantitative evidence that is *adverse* to what Bleuler said." I notice among some superskeptical scholars (not so often among clinicians) a tendency to conflate these two. That doesn't mean we should not bother getting quantitative evidence on something Bleuler or Kraepelin said from their extensive clinical experiences. It just means that the fact that nobody has done so in a fully satisfactory way should not count as *negative* evidence against a brilliant clinician's generalizations.

Although my own psychotherapeutic practice has largely shifted from psychoanalysis to rational emotive therapy in recent years, I still consider myself strongly psychodynamic in theoretical orientation, and I usually tell students that I am now a "40% Freudian."

Finally, with respect to indicators of the conjectural schizotaxic defect, I favor focusing attention on those at a somewhat lower level of functioning (soft neurology and psychophysiology) rather than social behavior, or even psychometrics, on the ground that the former kinds of behavior are closer to the DNA and so have, by and large, been subject to less influence from complex social learnings contributing to individual differences variation and, hence, to increased statistical overlap.

TAKING A FRESH LOOK

Whitehead said that it requires a very unusual mind to undertake the analysis of the obvious. A parallel comment, not about a mind but a mental set, is that it requires unusual effort to conjecture creatively about the overly familiar. The problem with almost all scholarly clinicians when looking at schizophrenia is its phenomenological familiarity, which goes back many years for most of us. I, for instance, first read a description of schizophrenia in McDougall's *Principles of Abnormal Psychology* at age 15. Almost any psychology major will, as a sophomore, have read at least two or three pages of text in the typical general psychology textbook, have heard part or all of an hour's lecture, and maybe have seen a videotape of a schizophrene. Before they graduate, almost all will have taken a class in abnormal psychology where they will have spent several class days on the topic, and they will have read a score or more pages about the syndrome.

Most medical students, some by admission requirement, will have taken a general psychology class before going to medical school, and will begin seeing schizophrenic patients interviewed by their second year. Now this is all to the good so far as developing clinical skill is concerned. But the point is that we get familiar with the syndrome; so when we reach the stage in our education and scholarly careers when we try to conjecture about the pathology and etiology of this illness, we have become *too* thoroughly accustomed to the syndrome, and hence are insufficiently struck by the strange cluster of "unrelated" phenomena that it presents. Consider the following list: interpersonal aversiveness, gross or subtle aberrations in syntax and semantics, aberration in tracking a sinusoidal target motion, a (±) dysdiadochokinesia, inappropriate affect, ambivalence, and a tendency to dereistic thinking. Speaking descriptively, or even with low level common sense psychologizing, what on earth do these diverse things have in common? On the face of it, the answer is "nothing."

This is important. It's the kind of fact I call a "big fact," one that should capture our theoretical attention and occupy a major role in determining our theoretical strategy. Schizophrenia in this respect is quite unlike the mood disorders. A thoughtful layperson who has never heard of cyclothymia would be apt to describe pretty well what a manic person would be like if we ask the question properly: "Have you ever been what you might call unusually 'up' in mood? Well, think about how you thought and felt and acted when you were feeling that way, and try to imagine how a person would think, feel, and act who was three or four times as 'high' as you have ever been." The same is true, probably even truer, of depression. Again, I don't think a bright layman would do a bad job describing the organic brain syndrome if you gave him a lead such as how he thought, felt, and acted when he had been going too long without sleep, or had to perform suddenly upon being rudely awakened from a deep sleep, or after having too much to drink. And I suspect you would get a pretty good description of the compulsive neurosis if you asked a layperson to characterize the thoughts, feelings, or actions of somebody who was, say, "a super solid-gold fussbudget." The same would be true for anxiety state, and to some extent the psychopathic deviate, although that one gets a little harder because the layman would not be likely to distinguish genuine warmth from the superficial charm of the psychopath that springs from lack of social fear.

Without arguing just how many and how well various other syndromes in psychopathology could be inferred by the layperson from a cue or two about its "essence," I think the point is fairly obvious that schizophrenia has a special kind of qualitative heterogeneity, a lack of any obvious phenomenological unity, at the descriptive level, or even one step up inferentially from the behavior to the momentary regnant psychisms. Once one steps back enough to contemplate the cluster afresh, one realizes it is not easy to make any plausible connection, speaking "psychologically," among the facets of the syndrome. We are, for instance, used to seeing social fear (or, better, interpersonal aversiveness, not implying an introspectable anxiety signal) combined with that special oddity of language that is in my view (as it was in Bleuler's) pathognomonic. But when one reflects on it, there seems to be no particular reason for such a correlation. Why shouldn't semantic oddities be associated, say, with hyperactive extroversion, or

with a compulsive avoidance of being alone? What on earth could be the connection between having an attenuated inhibition of the P50 cortical evoked potential (Freedman, Adler, Baker, Waldo, & Mizner, 1987) and having ambivalent cathexes? I don't deny that there are some more or less obvious connections between certain pairs of elements in the syndrome, but my point is that by and large it can't be done. This simple descriptive fact about the *qualitative heterogeneity of the syndrome* is the jumping off point for my theory of schizotaxia.

When there is no common phenotypic quality, and when even most pairs or triads of the syndrome's elements can be related only by procrustean forcing or reliance upon our familiarity with the cluster, I conclude that it is hopeless to derive the syndrome at one level of causal understanding. So it seems to me the strategy should be one that permits us to move up and down in the conceptual hierarchy of the life sciences in explaining these heterogenous effects. *Example*: Around the turn of the century "organicist" psychiatrists had a tendency to explain the phenomena of catatonia by neurologizing, that is, saying things about ballistic movement, antagonistic innervations, analogies to the effects of certain drugs like bulbocapnine in animals, vague similiarities to the movement in Wilson Disease, and the like. I do not deny that there may be an important element of soft neurology that *helps* to produce a subset of the clinical phenomena of catatonia. In fact I would tend to argue favorably along those lines, since I view schizophrenia as fundamentally a neurological disorder. But Bleuler (1910/1912), in his monograph on the theory of schizophrenic negativism, shows there is a clearly evident purposive, molar level aspect to many catatonic phenomena that should be apparent to any clinician, whether he is psychodynamically oriented or not. There are many ways to show this, as he does in this book and in the classic *Dementia Praecox* (1911/1950). Perhaps the most obvious is the appearance, in the same patient and sometimes in fairly rapid alternation, of behaviors that are, speaking purely topographically in terms of effector activity, "opposite" in quality, such as mutism versus echolalia, or negativism versus command automatism and echopraxia. Sometimes the very *content* of the response has to be understood purposively, as when the patient does the "opposite" of what he is ordered to do, or what others are doing, or what is normally done at a particular time. Hearing the dinner bell, he proceeds to turn around in the hall and walk in the opposite direction from the cafeteria. It is unnecessary to pile up examples, the point being that such things cannot reasonably be attributed to anything analogous to the intention tremor of multiple sclerosis, or the nonintention tremor in Parkinson Disease. We have to view this behavior as molar and purposive, whether we talk goal language or reinforcement theory language, in characterizing what constitutes "a reinforcing state of affairs." A subgoal definable as "doing the opposite of what the nurse wants you to do" cannot be properly understood—I would say cannot be properly *described*—as some kind of neurological inhibitory or coordination defect.

I further believe that one can list some poor strategies in theorizing about schizophrenia that are tempting and still fairly common. The commonest flies in the face of the striking fact of phenomenological heterogeneity. We select some aspect of the syndrome that has a high statistical frequency

(strangely enough, we don't require it to be universal); we identify a brain region or function that is known, or conjectured, to be strongly related to that behavioral aspect; then we conjecture that inferred state to be the specific pathology and go on from there.

I need not step on toes of current theorists to illustrate this, because I can go back to the widely discussed theories to which I was exposed as a psychology intern at the University of Minnesota Hospitals in the early 1940s.

Example: The apparent efficacy (exaggerated, as we now know) of insulin treatment, and the fact that the brain cells can only burn carbohydrate, plus a few clinical observations about schizophrenes who are compulsive candy gobblers (one is reminded of schizotype Hitler and his fondness for sweet cakes), led to a conjecture that the fundamental problem of these patients was a disturbance of carbohydrate metabolism. I remember being puzzled as to how such a theory would explain specific oddities in syntax, or why inadequately nourished cerebral neurons should lead behavior to drift in the direction of social fear, or excessive preoccupation with fantasy!

Example: The aversiveness of schizophrenes, while usually considered most striking in the area of social relations, is somewhat more general. The pan anxiety characteristic of the atypical form described by Hoch and Polatin (1949) suggests a conjecture that the aberration is something wrong with the anxiety system, such as an abnormal anxiety gradient parameter, or an excessive conditionability, or perhaps a resistance to Pavlovian extinction. From my clinical experience with different kinds of patients, I never saw much plausibility in this theory either. One has seen many patients with anxiety neurosis who are both chronically and episodically more anxious than the majority of schizophrenes, at least by the obvious operational indicators of that affective state, but who lack other features of schizophrenia that are equally striking or, to some of us, even more so. I have a patient who is so anxious than even on diazepam he has to take a swig of vodka from his pocket flask in order to get up his courage to drive from his office to a session, and after a session the chair arms are literally dripping with palmar sweat. But this man is devoid of *any* features of cognitive slippage, oddities of speech or manner, inappropriate affect, or social withdrawal; and despite the terrible anxiety, he is a successful inventor and business executive, an excellent salesman, and popular among his peer group. I find it hard to believe a theory that makes anxiety the focus of the schizophrenic's problem. Differences in drug efficacy is additional evidence against an anxiety theory; diazepam does little or nothing for schizophrenes, whereas the major tranquilizers (now properly described not as tranquilizers but antipsychotics) don't do much for the anxiety patient. The end state of a chronic schizophrene is frequently one in which anxiety is less present than in normal individuals. Understand, I am not saying that nobody could make some kind of case by sufficiently complex "ad hockery" and additional auxiliary assumptions, although I believe one would find himself, however ingenious, running into contradictions with the clinical material. I am not arguing a killing falsification with these examples, I am simply suggesting that this is not a very good strategy, it is not an approach that has a sufficiently strong antecedent probability to be pursued in research.

I recall that sometime in the 1930s, psychologists and psychiatrists overly preoccupied with the introversion dimension (as understood and psychometrized

by Americans, quite different from Jung's conception of introversion) sometimes thought of schizophrenia as simply a pathological deviation at the introvert end, and saw manic–depressive disease as located at the extrovert end. (One introversion test, the Neyman–Kohlstedt, was actually derived by item analyzing responses of schizophrenes and manic depressives.) Here again we have what is a descriptively striking feature of the clinical condition, social aversiveness and withdrawal from social interaction with attendant subjective states of shyness, anxiety, irritation on being required to participate in group activities, and the like, that is doubtless an important feature of the illness in most patients, although I think not in all when one looks and listens with sufficient care. This introversion theory is perhaps in somewhat better shape than the previous two, so long as we remain at the level of molar behavior (Littman & Rosen, 1950; MacCorquodale & Meehl, 1954; Tolman, 1932) and focus on what is clinically so important to family, friends, employees, that is, the social sector. It gets harder, even at the level of molar behavior, when we try to explain the peculiarities of thought or the distortions of perception, although with some procrustean forcing that can be managed. But while the thematic features can be understood fairly well psychodynamically in the framework of interpersonal aversiveness as the core problem, the equally striking formal features of the cognitive slippage do not lend themselves to this kind of explanation. Of course, if we require explanation of the soft neurology and psychophysiology, or anatomical signs such as facial asymmetry and the nailfold capillary bed anomaly, the "excessive introversion" theory does nothing for us.

Example: Another theory entertained during the 1940s focused on what was then called the "reticular activating system" (now generally called the arousal system or simply the RAS). It was suggested partly by the fact that the RAS had only recently been identified and appeared to have rich ramified connections at all levels of CNS activity, combined with the well-known clinical observation that many schizophrenes (perhaps all at some stage of the disease) show deviations in "arousal," either at the high or the low end, the low end preponderating in advanced chronic cases and the high end in the early stages. Here is a poor strategy that makes a somewhat more convincing case than the others I have mentioned. The striking deviations in arousal at the descriptive level are, so to speak, "immediately explained" by this conjecture, and it does not take too much auxiliary hypothesizing to fit in some other features of the syndrome in a fairly plausible way. Thus, allowing for a certain looseness in derivation (such as is unavoidable in the early stages of theorizing about any complicated phenomenon), almost any aberration in soft neurology could be explained by a RAS defect theory despite our inability to spell out the exact neuroanatomical pathways or specify quantitatively the relative strengths of their influences. In those days, a psychologist favorable to broadly Hullian views would have been able to make some plausible inferences about the disturbances in more complex cognitive processes (e.g., perception, speech forms) by employing mechanisms in Hull's theory of learning that dealt with the effect of drive. Differences in habit strengths could be potentiated into larger or smaller differences in reaction potential, because the drive factor

was suppose to operate multiplicatively upon each habit strength and hence could inflate or deflate the manifest difference. So although this conjecture began with a combination of a novel fact about the brain plus a fairly central aspect of the symptomatology, and proceeded to go from there to explaining qualitatively diverse phenomena, it was able to do a somewhat better job without undue forcing. Anticipating the defense of my own theory, I point out that the RAS theory's ability to do this came in large part from the anatomical fact of widespread and multilevel neural connections, that is, we have an anatomical system whose functional connections should be capable of "influencing almost everything that happens to some degree."

Example: Among the three conjectures in my presidential address to the American Psychological Association (Meehl, 1962a) was to take the schizotypal anhedonia as fundamental. I suggested that, "What is *phenomenologically* a radical pleasure deficiency may be roughly identified *behaviorally* with a quantitative deficit in the positive reinforcement growth constant, and each of these—the 'inner' and 'outer' aspects of the organism's appetitive control system—reflects a quantitative deficit in the limbic 'positive' centers. The anhedonia would then be a direct consequence of the genetic defect in wiring. Ambivalence and interpersonal aversiveness would be quantitative deviations in the balance of appetitive–aversive controls" (p. 832). I had to confess in that paper that, "Cognitive slippage is not as easy to fit in" (p. 832); nevertheless I made an effort to derive it. I went on to indicate that the hypothesis had some other troubles such as the fact that schizotypes seem to derive adequate pleasure from esthetic and cognitive rewards; their anhedonia is heavily concentrated in the interpersonal domain. Looking back, it seems to me that I was overly influenced by Rado's (1956, 1960; Rado & Daniels, 1956) emphasis on anhedonia in the schizotype and allowed myself to be seduced into employing the very strategy which I am here saying is not an optimal one.

Example: Given the influence of Rado on my thinking, I find it puzzling that I did not spend more time on his theory in which the primary (genetically determined) hedonic defect is combined with a "proprioceptive diathesis," because when you put the two of them together—it is not clear whether he thought these were pleiotropic effects of a major locus mutation, or two different genes—and, again, allow yourself a certain looseness in theorizing of the sort we can't stay away from when we talk psychodynamics, quite a bit of the schizophrenia syndrome can be explained. Without going into details, the general idea was that the "action self" develops via the child's experiences of successful (i.e., pleasure getting) instrumental functions, so that, as Rado put it, "pleasure is the cement of the developing ego." But that cementing of the action self by performance involves important kinesthetic feedback from one's own behaviors, even in the primitive sense that one learns to differentiate self from nonself partly on the basis of what experiences one can produce by appropriate movements manipulating the external world. So fairly direct consequences of this kind of theory are the aberrations in body image, the peculiar experiences unique to the schizophrene (except for drug induced and perceptual isolation effects?), and the frightening breakdown of ego boundaries. I shall say more below about what I think Rado was correct in observing (and other people have done, such as Paul Schilder), that there does seem to be

something especially anomalous about the "spatial–kinesthetic–vestibular" system in the schizophrene.

Example: Finally, a second theory I suggested in 1962 that is not currently my favorite but which is advocated (for instance by Freedman and his colleagues, 1987), I would look upon today as still a live option and would not take much more than even bets on my own preferred view over it, given the present evidence. An inhibitory defect, if conjectured to be ubiquitous in the CNS, is capable of doing a good explanatory job precisely because of the anatomical and functional ubiquity involved.

Another strategy detrimental to successful theorizing is to identify oneself as being a sociotrope or a biotrope (Hollingshead & Redlich, 1958). Schizophrenia theory has undergone cycles starting with strong organicist prejudices pre-Freud, followed by the dominance of psychodynamics (especially in the United States), a complete neglect of genetic factors, and either disregard for the soft neurology or an assumption that it could be explained psychosomatically. Today, the prestige of psychoanalysis is reduced, and there is correspondingly less interest in psychodynamics (though the latter is not necessitated by recognizing the inefficiency of classical analysis as a healing technology); the evidence is persuasive for a genetic predisposition, and the presence of striking psychophysiology and soft neurological phenomena; so people of the younger generation are again overwhelmingly biotropes. I think that most psychiatrists, and even some psychologists, are still dualists at heart, although they would vigorously deny any such attribution. Suppose a psychiatry department regularly requested a consult from neurology or otolaryngology, specifying a run in the Barany chair with sophisticated apparatus for studying postrotatory nystagmus, with and without ethanol (Angyal & Blackman, 1940, 1941; Levy, Holzman, & Proctor, 1983). We would probably be asked by the other department why we thought the patient had a disturbance of the vestibule or a lesion in the cerebellum. Since I am not a dualist, and I believe I have thoroughly assimilated the cliche that people say but don't deeply believe, namely that *the mind is the brain in action*, I cannot classify myself even roughly as a sociotrope or a biotrope, and I am glad of that.

Another strategic mistake is to focus attention upon some small trend because it was unexpected, or because it has a certain intrinsic psychological interest. Years ago a colleague, who was a pretty staunch environmentalist, objected to Kallman's genetic inferences, pointing out to me that a study had shown that the homosexual content of Italian male paranoid schizophrenes tended to be manifest, whereas the same (inferred) content for Irish male patients with paranoid schizophrenia tended to be disguised. He rightly said that this could hardly be due to Italian or Irish genes, but presumably reflected features of the two cultures, such as degree of general sexual puritanism, childrearing practices conducing to repression and denial as defense mechanisms among the Irish, and the like. I readily agree that this cultural factor is intrinsically interesting to the theoretician, and would be the proper focus for a sociologist interested in the epidemiology of mental disorder. However, for a psychologist or geneticist whose aim is setting up a general theory of schizophrenia, the *prime* task is to ascertain the specific etiology, if any exists. In the context of discovery, the cultural influences upon psychodynamics that determine latent and manifest content,

whether of dreams, waking fantasy, parapraxes, or delusions and hallucinations, does not speak either for or against a specific genetic etiology (Meehl, 1972a).

Another strategic mistake which is less obviously mistaken—and which has been pursued by able people with the result that facts were unearthed that would not have been without the strategy—consists of theorizing about the nature of the schizotypal aberrations in complex tasks when the mechanisms involved in those tasks are not at all well understood for normals; or, if the mechanisms of a complex task are putatively understood at least fairly well, it is known that the parameters vary widely over experimental modifications of the task, and also as a result of individual differences. Since there has been some payoff, I hesitate to denigrate that strategy outright, but will merely say that the net theoretical yield that is well enough corroborated to command universal assent seems to me rather poor in relation to the researcher brains and taxpayer dollars expended. I cheerfully admit that this is a judgement call on which competent persons (and, I must add, persons more thoroughly immersed in the literature than I am!) may disagree. Without arguing the point at length, I might refer to one excellent summary by a skeptical but optimistic investigator, Raymond A. Knight (1984). I think this paper, coming from one who himself has pursued creatively and assiduously the understanding of cognitive deficit in schizophrenia, suffices to make my point.

I would make here a general comment suggested by critical literature reviews such as Knight's. Suppose an advanced doctoral student comes to me for advice about accepting a job in which the research facility's focus was on the theory of schizophrenia, a subject about which the student knows relatively little. He needs to "bone up" on schizophrenia in three months. Query how to go about it? The student is bright, well organized, and hard working; but three months is a pretty short time, and you can only read so many pages a day. If I want to provide him with an apperceptive mass which would prepare him for his new research environment, how would I advise him to spend that precious three months? Alas, although I am a psychologist (and one in a strongly quantitative and experimental tradition as clinical departments go), I think I would *not* set him to reading the vast literature produced by psychologists who were engaged in what my colleague Garmezy once referred to as "merely smuggling clinical phenomena into the lab." I don't mean that I would not have the student read anything about this, but I would content myself with having him read several summaries such as that of Knight, and maybe a carefully selected subset, quite small in number of original papers cited therein. A larger part of his three months time I would have him spend reading and rereading Bleuler's 1911 treatise and Kraepelin on dementia praecox. He should have extended conversations with schizophrenes carefully chosen to represent different subtypes and different stages in the natural history of the disease, spend several hours conversing with (or in the company of) a chronic back ward state hospital deteriorated schizophrene, and similarly spend several hours with a young first episode catatonic. I would have him read both volumes of Gottesman and Shields (1972, 1982), the foster child paper by Heston (1966), and then I would have him read a recent summary of the soft neurology and psychophysiology findings. Relying on these readings to

convince the student that we deal with a genetic neurological disorder, I would conclude my crash course of instruction by having him read Arieti (1955), Fromm-Reichmann (1950, 1952), Karon and VandenBos (1981), Lidz and Fleck (1985), and Sullivan (1953, 1956, 1962). Cognitive dissonance would be averted by telling him, "These experienced, perceptive clinicians discerned important truths about the schizophrene's psychodynamics, which some biotropes wish to ignore. One can accept 80–90% of what they say, merely deleting their needless conjectures that the family pathology and resultant intrapsychic dynamics constitutes the specific etiology. The latter is a gene." Much as I dislike to say it, I cannot convince myself that the contributions of my psychology brethren to the description and the understanding of this disease have been as great as those of the great psychiatrist clinicians. I believe one reason for that is that the psychologists' preoccupation with the experimental method has led them to be overoptimistic about what it can and cannot show.

I find it especially puzzling when psychologists, in their effort to "smuggle a clinical phenomenon into the lab," do so in a way which, if the smuggling effort were a failure, would not convince the profession (perhaps even the investigator himself) that the phenomenon as described by seasoned clinicians was unreal. Citing specific examples would be invidious and take time, so I will content myself with saying that I have read papers in which such an effort was concluded by a series of ad hoc explanations of *why* the clinical phenomenon failed to appear under "controlled conditions."

Example: I read a study years ago in which the question was, to what extent does anxiety, mobilized by some kind of interpersonal threat, impair cognitive processes in schizophrenic and nonschizophrenic subjects? The predicted finding was absent or very weak, but that did not lead the author to conclude that socially induced anxiety does not affect schizophrenic thought or perception, despite the unimpressive laboratory results. One need not be a Popperian to wonder what was the point of such an experiment if it were to be believed by the investigator himself, let alone clinicians reading his paper, only if it "came out the way it should"? Anybody who talks to a superintendent of a state hospital about visiting classes in abnormal psychology knows that such administrators usually have ambivalence about such visits. On the one hand they like future taxpayers to see what the needs are, and there are a few patients who respond positively to this mass of strange faces peering at them; but there are still more—especially before the era of phenothiazines—who become very upset, whose delusions, hallucinations, catatonia, or malcommunication worsen, sometimes for a matter of days, after the visit. No psychotherapist who has treated schizophrenes has any doubt about this, and sometimes we see a striking increase in katathymic thought in the midst of a session due, say, to an instance of therapist clumsiness such as looking at one's wristwatch, or making a tactless interpretation of transference. Perhaps the most striking of these is the "thought deprivation" that Bleuler considered pathognomonic. The point here is not that I believe clinicians are never wrong, a view nobody familiar with my writings could attribute to me! The point is that when seasoned clinicians, holding different causal theories and relying on different helping procedures, show unanimity in reporting a phenomenon

that is minimally theoretical, close to the "purely descriptive" level, we (properly) do not consider their clinical experience to have been "refuted" by an experiment which purports to get at the processes in question but which we cannot be confident did so because of our incomplete knowledge of the *normal* case. For a general methodological discussion of this problem in the soft areas of psychology see Meehl (1971, 1978, 1990a, 1990c).

I infer, from the track record and theoretical reasoning, that theories which focus on a conspicuous molar trait (anxiety, anhedonia, social malcommunication, arousal) will require excessive stretching and multiple ad hoc auxiliaries in order to derive the other ("dissimilar," unrelated) elements of the syndrome. I conjecture the same will be true of theories that start with such a focus on a molar property, and then go on to a particular subsystem of the brain, or a particular neurotransmitter, or some specified feature of general body chemistry. An important corollary is that, adjusting for an estimated upper bound on the percentage of misdiagnosed cases, based upon a combination of the genetic data on MZ twins and reliability studies of objective methods (e.g., SADS), a molar-level phenomenon that is not found in substantially *all* schizophrenes is a poor bet to start with if we are searching for a theory about the specific etiology of the disease, if such exists. I do not say that the sign in question must be both present in all schizophrenes and absent in all controls, because a specific etiological agent is only a sine qua non, a necessary but not sufficient condition (Meehl 1972a, 1977), and consequently may be only one-way pathognomic, that is, as a exclusion test. This is especially true when we have a genetic disorder in which the concordance for MZ twins is only a little over 50%. A descriptive property which merely shows a *trend*, a statistically significant difference between schizophrenes and psychiatric or normal controls, may be of epidemiologic interest for screening purposes, and may be an important fact in elaborating a theory of schizophrenia that goes beyond identifying the basic etiology and pathophysiology; but it is not a good candidate if our aim, in a given stage of knowledge, is to find out what is *specific* to these patients that is not found in normal people, manic depressives, anxiety neurosis, psychopaths, etc. One already knows, on the basis of such modest concordance for MZ twins, that numerous environmental causal factors operate in determining who falls ill. One also knows, from the statistics on first-degree relatives compared to those for MZ twins, that there must be important polygenic potentiators, presumably nonspecific to schizophrenia but playing a critical role in determining which schizotypes decompensate. So it is obvious from the armchair that quite a few "factors" (usually observed current dispositions and life history events or conditions that act as proxies for *factors* in a causal sense) will show sizable differences between schizophrenes and controls. In my theory I have listed a dozen or more plausible polygenic potentiators. Some of these may play such a powerful role in precipitating illness among schizotypes (90% of whom, on my theory, remain lifetime compensated) that it would be easy to get statistically significant differences between schizophrenes and controls on such factors. The excessive reliance by psychologists on significance testing is partly the reason for mistaken reasoning about this matter (cf. references above on significance testing).

THE UBIQUITOUS CNS ANOMALY APPROACH

If one does not adopt the poor strategies, what would be the alternative? We get a wide variety of malfunctions in all systems, at all levels, and all domains, although in some systems specific processes appear relatively unimpaired, at least with the sample sizes and measuring devices we have available. I commence with this crude generalization, based admittedly on skilled clinicians' impressions as much as upon quantitative studies, that "something is haywire everywhere, or almost everywhere, with these patients." Moving down in explanatory levels from behavior (including neurology) to the CNS, I conjecture that whatever is wrong with the schizotaxic CNS is ubiquitous, a functional aberration present throughout, operating everywhere from the sacral cord to the frontal lobes. (I will drop the repetitious "I conjecture" in what follows and use the simple indicative.) More specifically, this "something wrong at all levels" is something wrong about the way individual neurons work, *a functional parametric aberration of the synaptic control system*. It is almost certainly not a defect of sensory inputting, or storage, or retrieval, or "achievable complexity," each of which would be hard to fit with the high intellectual achievement of certain famous schizotypes (Newton, Goedel, perhaps Einstein), or with the well-known fact that if you can motivate him you can get a chronic back ward schizophrene to play a pretty good game of chess or Scrabble®. Roughly, one thinks of the defect as an integrative defect analogous to dyslexia, tone deafness, stammering, "normal range" but extreme motor discoordination, the specific spelling defect sometimes found in persons of high IQ and good verbal fluency (DeFries, Fulker, & LaBuda, 1987; Lytton, Watts, & Dunn, 1988; Stevenson, Graham, Fredman, & McLoughlin, 1987), and functional phonology (articulation) disorders, some of which apparently have genetic etiology (Byrne, Willerman, & Ashmore, 1974; Neils & Aram, 1986; Parlour & Broen, 1989; Tallal, Ross, & Curtis, 1989). Unlike these, which are presumably restricted to cerebral subsystems, the integrative defect in schizotaxia is ubiquitous. The neologism "schizotaxia" is from *schizo* and *ataxia* (the Greek word for a disturbance in arrangement, array, marshalling, ordering), eliding the alpha privative.

A Skinnerian colleague complained that I was simply reattributing Bleuler's core trait of associative loosening to the neuronal systems. With this criticism I cheerfully agree, since, despite Skinner, I assert that in the other sciences it has sometimes been profitable to engage in such lower-level replication of properties if you're careful about how you do it. Magnetism provides a clear and easy illustration of this point. The molar magnetism of a soft iron bar is attributable to the lining up of micromagnets called "domains"; the magnetic poles of the domains are determined by the north/south pole orientations of the iron atoms in each domain; and, finally, the north and south poles of the atoms are based upon the positions and motions of the elementary particles which have magnetic moments. It is not necessarily sinful to analogize at a lower level with explanatory intent. It depends upon where you go from there, and whether your general knowledge of the system is such as to make the hierarchy of properties physically plausible. I can put it crudely thus: Like Bleuler, I think the most striking fact about these patients that makes them unique in psychopathology is

their "slippage" (Bleuler's *associative loosening*), both the "cognitive slippage" and the cognitive–affective slippage. You cognize and emotionize with your brain cells, and if the big striking fact is molar machine slippage, it's a good guess that this corresponds to a microslippage in the gears. And what are the gears? The gears are subsystems of nerve cells, whose engearment with one another is a matter of synaptic control. Omitting graded potentials, electronic inductive effects in the nerve trunk, and the like, the big thing that the neuron does to another neuron is contribute to making it spike. If one considers the parameters of the synaptic control process in terms of a set of arrivals of presynaptic impulses over the synaptic scale, there is something wrong with those functional parameters that leads to an aberration in the postsynaptic cell's spike probability. In a nutshell: *The reason for the molar slippage in the schizophrene is that the schizotaxic brain has slippage at the synapse*.

We have to make this synaptic control function aberration rather subtle, because otherwise there would be too gross an impairment in all functions, rather than the different degrees of impairment described by clinicians and experimental psychopathologists. Confining ourselves to spike control only, and considering an idealized postsynaptic cell, we associate each terminal knob of the presynaptic fibers that terminate on that cell with a coordinate in a representational hyperspace, so that the space has as many dimensions as there are knobs. The neurophysiologist informs us that the arrival of a presynaptic impulse at a knob produces (by a combination of neurotransmitter secretion and maybe some electromagnetic effect?) a localized depolarization of the cell membrane, which then repolarizes. If the spatiotemporal pattern of such presynaptic spike arrivals is such, by proper quantitative combination of time and place, to produce a sufficiently large area of depolarization, and provided also—what seems to be a necessary but not sufficient condition—an approximately simultaneous spike arrival at the axon hillock, the cell reaches threshold and fires. Let us assume with Lorente de Nó that there is "optional transmission" for a given presynaptic signal pattern, so our output variable is the optional transmission probability of a spike. We assign the arrival times as values of the coordinates associated with the presynaptic knobs. Then, in a cell not in refractory phase at arbitrary time zero, to each such spatiotemporal presynaptic spike arrival configuration (i.e., to each point in the synaptic signal space) there corresponds a particular optional transmission probability. Representing these probabilities as a continuous hypersurface in the synaptic signal space, the schizospecific parametric aberration in synaptic control function consists of a geometric change in the properties of that hypersurface; namely, it is (a) elevated and (b) flattened, i.e., there is a dedifferentiation or washing out of the hills and valleys in the topography of the hypersurface.

This parametric functional control aberration I call *hypokrisia*. One does not solve scientific problems by making up words, but I am making up a *concept*, and I need a word to designate it, to differentiate it from other plausible conjectures as to what is wrong with the CNS brain. The word comes from the Greek words meaning an insufficiency of separation, differentiation, or discrimination. Obviously one might go along with the conjecture that schizotaxia is a certain kind of neural integrative defect,

and not accept this specific microexplanation about the synapse. For example Freedman et al.'s (1987) conjecture that it is a defect in inhibitory function, perhaps a defect involving only one of the several neurotransmitters that are inhibitory, would result in an "integrative deficit," since the particular coordinated pattern of excitation and inhibition is what constitutes an integrative process. But that would not be the same as my hypokrisia concept.

When pressed by colleagues to speculate further about the basis for the hypokrisia (which I am loathe to do, since neurochemistry is not a subject in which I have any competence), I do have a tentative view. If, as conjectured infra, we deal with a major locus and, hence, a mutation; and, hence, a missing or aberrated enzyme which normally controls the synthesis of some neurohumor; then we deal not with an active substance but with an inactivated one, that is, we have not a toxin but a deficiency disease. That mysterious Substance X, (which someday the neurochemist will, I trust, identify) has in the normal brain the function of either retarding the spread of the depolarization from the points of presynaptic spike arrival, or facilitating repolarization, thereby holding down the probability of a critical mass adequate for spiking being developed. However, I must admit an alternative would be something not biochemical but micro-anatomical, concerning the statistical geometric distribution of end knobs over the synaptic scale. This latter should perhaps be investigated first, for obvious search technology reasons.

Returning to what was said above about bad strategies, just how hypokrisia may be expected to affect molar level functions cannot be rigorously derived without more details of the CNS systems for the various molar domains among normals than we presently know. Hence *detailed* explanation of why some cognitive functions are more impaired than others would be theoretically premature, and research on that will have sometimes an appearance of inconsistency as discussed by Knight and other reviewers. *Analogy*: I have a very complicated mechanical device which I inform you operates by a combination of gears and pulleys. I know something about some subsystems but almost nothing about others. I also know that even those subsystems that I understand in some detail as to their structure and function show individual differences, for example, in the coefficients of friction of the pulleys. In explaining why some specimens of the assembly line malfunction, I conjecture a ubiquitous aberration in property of the gear teeth, leading to the greater possibility of a certain amount of "mechanical slippage." I point out further that under these conditions matters involving delicate timing might be quite grossly impaired. But if now we ask why Function A, which this machine normally performs, is grossly impaired, whereas molar Function B is hardly at all impaired, its malfunctioning being detectable only by the most precise tests of high statistical power, whether that question is answerable or not depends upon the detail in which we have figured out the subportions of the machinery involved in these two molar functions. This is why I consider efforts at quasi-complete theoretical understanding of the aberrations of thought, perception, memory, feeling, attention, and so on among schizophrenes to be more often than not premature and undoable at the present stage of our knowledge.

However, if we are careful not to become grandiose and over extend ourselves in our ignorance of the brain's details in the normal case, less detailed broad trends are perhaps understandable relying on auxiliary theories already in fairly good standing. Let me try to show why postulating a ubiquitous integrative defect, operating at all levels of the CNS and hence potentially permeating all levels and sectors of behavior, can be defended with less ad hockery than is required by the other theoretical strategy, which focuses upon some common or clinically striking defect and postulates it as the specific etiology. Essentially, the point is a methodological one. Because of the syndrome's qualitative diversity, one must with that latter approach postulate many different and unrelated ad hoc auxiliaries, called by Lakatos (1970) ad hoc_3. They may be content increasing, and they may even be empirically corroborated, but they are, so to speak, "extraneous to the core ideas of a theory," sort of adjoined or pasted on to save the appearances. And even then it's often pretty thin, as in my 1962 overemphasis on anhedonia, which permits only the feeblest ad hoc_3 "derivation" of other (nonaffective) symptoms, such as neologisms or soft neurology. It might seem that the ubiquitous defect postulate is in the same boat, that instead of the difficulty "How derive dissimilar symptoms S_1, S_2 . . . S_m from special defect D_s?" we have the opposite difficulty, "Why doesn't ubiquitous defect D_u impair all functions (levels, sectors, domains)?" The reason that this second question, posed to a ubiquitous defect theory, is not nearly as dangerous a temptation to ad hockery as the first question is simple, namely, we already have independent knowledge concerning many of the required auxiliary hypotheses, although it is often less detailed quantitatively than one would wish. Furthermore, the *general* fact of partially independent and variably influenced subsystems is known outside of psychopathology, hence is not ad hoc merely because we carry them into psychopathology. *Classical psychometrics provides the paradigm*. Thus, omnibus intelligence tests regularly show the general factor *g*, group factors, specific factors, and error variance. If we look into other subdomains of functioning, such as motor skills or mechanical aptitude or social competence, we again find a hierarchy of factors at work. Thinking along these lines, what does one expect to follow from a ubiquitous CNS integrative aberration due to a cause like hypokrisia? Such a defect should:

1. Influence all, or nearly all, levels and sectors somewhat.
2. Influence some levels and sectors more than it does others.
3. Be quantitatively "fuzzy," because of individual differences in group factors and specifics.

There is nothing ad hoc about any of these expectations.

Which behavior domains are likely to be more impaired, *on the average*?

A. Domains involving relatively distant subsystems and hence relying on spike transmission for processing information. So we do not look for malfunctioning in aspects of visual perception that are based on nonspike transmission (e.g., graded potentials).
B. Domains requiring complex integration of spike-transmitted inputs from several different controlling subsystems (e.g., multiple sensory modalities).

C. Domains demanding finely tuned multiple control by the subsystems in (B). One might express this conjecture in terms of the values of partial derivatives of the multiple input control function. Let the excitatory level (conceived roughly as average frequency of spikes over all neurons belonging to a module) be $y = F(x_1, x_2, \ldots x_m)$, where the xs are excitation levels of the multiple modules "driving" the one in question. Then taking the partial derivatives $\partial F/\partial x_1$, $\partial F/\partial x_2$, … $\partial F/\partial x_m$ of the excitation function of the "controllee" module with respect to the input excitation levels, the magnitude of these partial derivatives indicates the relative potency of each input. A disturbance in synaptic control of a minimal kind might make itself known by either a reduction in the magnitude of those partial derivatives (i.e., the control is less "sharp") or by a reduced separation between them, probably both.

D. Domains providing rare opportunities for development of corrective or substitute adjustment mechanisms. *Example*: There are color blind males who never realize they are color blind until they try to enlist in the Navy and fail the Ishihara. This is possible because they learn to rely on other cues, and because—especially for males—accurate color designation is not a culturally emphasized skill. Contrast this with an aberration in postrotatory nystagmus in the schizophrene. Neither normal nor schizotaxic persons spend time riding around in Barany chairs, and most of us have negligible exposure to stimulus input for nystagmus, except on an occasional visit to the state fair!

E. Domains where the growth of a molar aberration is autocatalytic, because two or more functions are interacting with positive feedback. *Example*: Distorted social cognitions in the schizophrene facilitate aversive drift in the interpersonal domain to a greater extent than would be true of a solitary activity such as doing crossword puzzles, or the minimally social activity of playing correspondence chess. But one consequence of aversive drift (as explained below) in the interpersonal sector is increased social withdrawal, with the result that corrective experiences, such as the control of distorted cognitive content by consensual validation, is reduced. Hence the tendency to distorted cognitions regarding the interpersonal domain is in turn accelerated, producing more confusion and anxiety, potentiating distortion and further aversive drift, and so on.

F. Domains where the person–environment interactions are like the intrapersonal situation (E), that is, there is positive feedback generating autocatalysis. This is familiar to clinicians for neurotics as well as schizophrenes, although in the schizophrene it is more pronounced. The patient behaves strangely because of the combination of aversive affect and cognitive distortion, which leads others to be baffled, irked, or frightened by him, which leads them (statistically) to treat him in ways that confirm his aversive set, or which puzzle him and lead to efforts at explanation that, again being less subject to consensual validation, move in a "crazy" direction, and so on—a vicious circle of the worst sort.

G. Domains where the external reinforcement schedule is highly stochastic.

We expect the impact to be greater on some domains than on others. *Example*: The clinical lore (supported by some quantitative research, although inconsistently) shows aberrations in what I refer to as the *SKV* ("spatial–kinesthetic–vestibular") *system*. While these functions do not constitute a single CNS system anatomically, they are nevertheless subsystems that must be hierarchically integrated for a human being to locomote, stand straight, orient in space, and have a "normal" perception of his own body (and, psychodynamically, his ego boundaries). Despite the different anatomical systems, there is a kind of molar level unity here, in that space and one's positioning and coordinating in it are involved. When a normal subject knows his location and position (e.g., standing upright or leaning at a angle, nearer to one wall in the room than another), these perceptions are formed automatically by integrating inputs from various modalities: vision, the vestibule, deep and light touch, hearing, and proprioception. The tabetic has more trouble walking at night because in the dark he cannot substitute visual cues for his impaired proprioception due to the luetic damage of the dorsal columns. When visual, kinesthetic, and vestibular cues are made incongruent, as in the famous Ames demonstrations at Cornell, some "normal" people become so anxious that they have to leave the scene. Given the complexity of the CNS integrative task from these multiple inputs, we are not surprised to find schizophrenic deviations in such things as postrotatory nystagmus, three-dimensional space perception (more than if flat!), field dependence, discrimination of lifted weights, positive Romberg, orientation, retention of orientation in passive movement, body image, and size estimates. So this somewhat puzzling SKV cluster is at least partially understandable on the basis of impairment in domains A-B-C, and to some extent D, without postulating Rado's specific "kinesthetic diathesis," as I inclined to do in 1962 and later.

Social impairment looms large in our picture of schizophrenia, although one should keep in mind that this may be partly due to its importance to the rest of us. Aversive drift, bizarre thinking, and (±) dysdiadochokinesia, which one could go throughout life without anybody n episodic rage are both more visible and more important to other humans than a oticing, including oneself. Also, we have no standard metric for comparing "how much social deviation" with "how much SKV deviation" or how much semantic deviation" from the norm. So we cannot really assert, absent such a metric, that schizotypes are more aberrant interpersonally than they are perceptually, motorically, cognitively, or verbally. But if they are more deviant there, this would be easily explained by the above list. For instance, no behavior domain has such a chancy stochastic reinforcement schedule as the interpersonal.

Any "derivation" of the social–affective symptomatology from the schizotaxia core concept will be somewhat loose, what Feigl used to call an "explanation-sketch." Thinking of schizophrenia with different levels of explanation being concurrently and interactively employed, one nevertheless would prefer to derive all of the general statements at one level from statements at levels below. I am not so foolish as to attempt that, and I am aware that the loosest and weakest aspect of my explanation sketch is located at the interface between hypokrisia and what may be called the "first-level

characterization of psychisms." As soon as we can operate at the level of psychisms, dealing with cathexes, ambivalence, reward probability, aversive drift, and the like, we require little or no ad hockery to see how all of the major elements of the syndrome, and many minor ones, can be understood. *Example*: Suppose we can get from the integrative defect at the CNS level of description to a quantitatively inadequate differentiation (fuzzing up, scrambling, "mixing") between competing affective tones associated with perceptual cognitive processes, so that "plus" and "minus" affective tones are more confused in the schizotype than in the normal, or even the neurotic. Then it is rather easy to subsume secondary hypohedonia, Rado's pain dependent pleasure (similar to Freud's "moral masochism"), together with Bleuler's ambivalence—all of these being major features of the molar level descriptive syndrome—under the single heading of ambivalence. Secondary hypohedonia is then simply an enfeeblement of pleasure at the high end of the basic hedonic continuum, pain dependency is a heightening of connections at the other end, so that the whole pleasure–pain continuum is flattened, contracted, or scrambled, however we want to call it; and this is what we mean by ambivalence. If we take note of this ambivalence at the negative end, we label it pain dependency; when struck by the manifestation of ambivalence at the high end, we call it hypohedonia; whereas if we focus on the intermediate range, where we expect nonschizotypes also to show some considerable mixture of affective states (e.g., with respect to a spouse with whom the patient is passionately in love, and intellectually admiring, but who also plays the role of bad mother in the relation), we call it simply ambivalence.

In sketching the derivation of these first level psychisms from the schizotaxic defect, it helps to think in terms of a block diagram of mental functions. It is, of course, desirable that the postulated connections between boxes in the block diagram should have *some* direct experimental and neuroanatomical support, but it is not imperative. Thus, for example, we are pleased to know about effects of direct stimulation of what I have labeled (Meehl, 1962a) "Olds (+)" and "Olds (–)" centers (the latter term being unfair to Delgado, Roberts, and Miller, 1954, whose negative centers were published the same year as Olds' on the positive intracranial stimulation). But even if that were not the case, I would feel free to rely on lower animal experiments and common observation for drawing the block diagram. (Conjecturing freely is one advantage of holding a neo-Popperian philosophy of science!) That the internal representations of certain classes of external (especially social) objects and events acquire positive and negative cathexes is, I assume, not in dispute. It will not come into dispute even if the original clearness about the Olds (+) and Olds (–) centers becomes attenuated by more detailed investigation, as may be currently happening (Gallistel, 1983; Gallistel & Shizgal, 1981). I presuppose that the learning process results in formation of connections between stimulus inputs and psychisms, and between psychisms and behaviors, but I do not adopt a radical behaviorist point of view as regards what is learned. That one can reward and punish schizophrenes and get behavioral changes has been thoroughly documented by the operant behaviorists (e.g., Ayllon and Azrin, 1968). This technological finding does not lead me to deny that schizophrenes also learn intrapsychic connections, say, between an image of

mother's angry face and a feeling of fear. My basic picture of the learning process is that what we learn consists of: (1) Perceptions, (2) habits, (3) memories, (4) expectancies, (5) motives, (6) cathexes. I leave open the question of the distinction between primary and secondary motives, although I take it for granted that the work of Bowlby (1969–1980) and others, as well as studies of the cognitive motive in the monkey (or even exploratory drive in the rat), has refuted the older behaviorist (and Freudian) conception that so-called higher order motives, such as complex social motives, are anaclitic, "derivative," or based upon the laws of secondary conditioning (cf. Eagle, 1984). Finally, I attach supreme importance to the *intrapsychic* connections in the schizotype, not because they are unimportant in the normal and neurotic, but because the derivative fact of schizotypal social withdrawal alters the stochastic parameters relating inner events and external reinforcements (contingent upon effective instrumental behavior) to such a great extent that it approaches Marx and Engels' "transformation of quantity into quality." For this reason I look upon the acquiring, eroding, and transforming of associative linkages between perceptual–cognitive and motivational–affective psychisms within the schizophrene as the primary basis of the aberrant behavioral dispositions. This intrapsychic emphasis does not contradict the fact that the strength of instrumental behaviors can be directly manipulated when the environmental contingencies are brought under sufficiently great control, as in the Ayllon–Azrin token economy.

One of the "big facts" about schizophrenia which any theory must deal with on pain of being grossly defective is what I have called *aversive drift* (Meehl, 1962a). While I no longer view Rado's anhedonia as a defining element of the syndrome, I remain convinced clinically that these unfortunate patients do have an overall tendency to show a drift in many different life domains from positive to negative affective tone. One might view the ambivalence as a fairly direct consequence of the hypokrisic slippage, given the stochastic reward–punishment sequence in the developmental history. Speaking loosely, because of "synaptic slippage" all learned connections are a bit "loose," as Bleuler called it. So the amount of scrambling or mixing between Olds (+) or Olds (–) linkages to a perceptual–cognitive representation in the brain would be enhanced. But why the asymmetry of aversive drift? Working therapeutically with these patients (I am talking here mainly about schizotypes in pseudoneurotic degrees of decompensation, and fairly well compensated schizotypes hardly diagnosable symptomatically) one is struck with the extent to which new persons, places, activities, even self-selected hobbies, start out to be fairly rewarding, despite the common attenuated pan anxiety. Despite successes of the sort that would maintain the behavior and the intrapsychic expectations in normals and neurotics, for the schizotype things begin to take on a burdensome, threatening, gloomy, negative emotional charge. Harry Stack Sullivan (1956) puts this in terms of the schizophrene giving up all "hopeful expectation" about life.

Why should this be? In the case of new people, or new activities involving other people, we might do a passable job explaining it in terms of the autocatalytic and mutual feedback effects mentioned above, although I am not sure this will work sufficiently well to show why it is so much more pronounced

in the schizotype than in the neurotic patient. Be that as it may, I find it hard to understand with reference to a relatively impersonal activity such as playing a musical instrument, or writing poetry, or a solitary depersonalized hobby. The intensity and pervasity of it in the interpersonal domain is striking, and of course takes on frightening proportions when we move into the realm of schizophrenic psychosis. I think my theory can explain this without making more assumptions about brain system linkages and their growth than we are fairly confident in asserting in the present state of knowledge, quite apart from schizophrenia. For some reason this part of my theory has not attracted any attention, so I don't know what kinds of criticisms thoughtful persons and experienced clinicians might have made against it if they had taken it seriously. That being the case, I am going to explain it again, perhaps better than in 1962.

Despite its conjectural character, I take heart from the fact that one of the marks of an interesting theory, and one of the strong corroborators of an interesting theory if it succeeds, is its ability to explain something that is not, "on the face of it," connected with something else, what Nozick (1974) refers to as the "invisible hand" quality of good theories. So I think my derivation of aversive drift is rather nice, because it succeeds in getting a directional asymmetry on the hedonic continuum without surreptitiously plugging that into the postulates but rather as a direct algebraic consequence of the relation between acquired appetitive/aversive control systems. It goes like this: We assume the development of a control linkage between Olds (+) center excitation and a perceptual–cognitive CNS event (or state, if time-extended), and similar control linkage development between the same perceptual–cognitive event and activity in the Olds (–) center. We can be confident that that linkage growth function is at least decelerated, so that a linkage function (whose exact mathematical form we can't specify today) at least has the properties that $dy/dx > 0$ and that $d^2y/dx^2 < 0$. One can conclude that from the armchair, because it has an upper limit, and unless it has a sharp break—a step function of the kind that we don't usually get in learning processes—it has to start decelerating somewhere in the course of its growth. Less confidently, but on a mixture of theoretical and empirical grounds (going all the way back to Sherrington's recruitment), we could make the conjecture a little stronger (but not necessary for the derivation) by hypothesizing an initial phase of positive acceleration, so that the overall shape of the linkage function starting from zero is sigmoid. The reasoning then is that a heightening (exaggerating) of input from a controlling system of modules (I earlier spoke here of Hebbian cell-assemblies) to the controllee system will be reflected in correspondingly exaggerated feedbacks. If the exaggerated effect occurs in the decelerated region, it will be quantitatively less than if a corresponding exaggerated "driving" of the controllee assembly system occurs in the linear (or accelerated) region. We next combine this with the empirical fact that most reinforcement schedules (especially social ones) are stochastic, and frequently (±) concurrent, so that, for example, in the developmental period mother may be simultaneously nurturing and frowning, to take a simple crude source of mixed object cathexis. Concurrent negative feedback from Olds (–) centers—the "normal" function of which is to turn off or suppress, corresponding neurophysiologically to defensive psychisms that avoid pain—is taking place. Exaggerated negative

feedback in linkage development which is predominantly positive retards the formation of connections to the Olds (+) centers. Whereas in a life domain which is predominantly aversive, after the process has proceeded for a while we are in the decelerated region of the aversive linkage function, but we are still in the linear (or positively accelerated) region of the Olds (+) control function. The hypokrisic exaggeration produces an enhanced positive feedback, thereby tending to consolidate the Olds (–) perceptual–cognitive linkage to an excessive degree. In a word, *facilitation based upon exaggerated driving in one region strengthens linkages that are mainly aversive; whereas in the opposite case, mainly appetitive linkages are retarded in their growth*.

I then conjecture that given a certain linkage, the same kind of quantitative reasoning would apply to the momentary (+)/(–) balancing between systems now linked. Over a period of time, the average effect of this kind of process would be a drift in the aversive direction. We know that the activation of Olds (+) and Olds (–) centers is of two sorts. If delivered contingently it strengthens or suppresses targeted overt behaviors. But Olds (+) activation also has a strengthening effect on concurrent operants, even when intracranial stimulation is given noncontingently. On the other side, an aversive state (and, hence, an operant maintained by it on the basis of molar avoidance learning) can be reduced in strength by concurrent noncontingent stimulation of Olds (+) centers.

There is a puzzling clinical phenomenon one observes in psychotherapy with schizotypal patients, especially of the "borderline," "pseudoneurotic," Hoch–Polatin syndrome, in which the patient reports an aversive psychic state, a kind of *painful mind*, that does not seem subsumable under the familiar negative affects found in normal and neurotic patients. Of course we have the problem of the intrinsically fuzzy semantics of designating private stimuli (Skinner, 1945), but I have a strong impression that there is something over and above that communication problem involved here. If one has treated schizophrenes who are highly intelligent and verbally skilled, and in my clientele I have had a number of such patients who had various amounts of training in psychology, including clinical psychology, it is my experience that searching exploration of the phenomenology leaves one doubtful as to just what blend of negative affects is involved. I am inclined to think that these patients experience a special kind of negative affect that cannot adequately be described as depression, shame, guilt, anxiety, grief, or a mixture of these familiar emotional states. I have had some Hoch–Polatin patients who, in an effort to convey the feeling, said such things as, "No, it's not mainly that I'm sad or afraid, it's somehow that my mind hurts, and it hurts a lot—it's almost as if my head hurts, although it's not really a headache." I don't suppose we will ever know what goes on here until we get below the phenomenology into a more detailed understanding of the affective centers of the brain. I don't think a nonschizotypal therapist can really empathize with this unique sort of painful mind, anymore than one can fully apprehend the strange distortions of body image, the blurring of ego boundaries, or the confusions of chaotic sexuality that appear in schizotypes.

In the 1962 formulation of my theory I ascribed a fundamental role etiologically, and a core position in the descriptive syndrome, to anhedonia.

I did this partly on the basis of clinical experience, partly from reading Bleuler, and partly under the influence of my training analyst, analytic supervisor, and subsequent coinvestigator, Bernard C. Glueck, who had received his psychoanalytic training at the Columbia Psychoanalytic Clinic under Rado's aegis. For both Rado and Glueck, anhedonia was a core feature of the schizotypal personality organization. I no longer ascribe a primary role to hypohedonia (an obviously preferable term, since even the most deteriorated, unresponsive back ward schizophrene can get pleasure out of a few things, such as smoking cigarettes or watching TV), although I still believe that it is found with considerably higher frequency among schizotypes than it is among other psychiatric clientele. In my present view, on the etiological side, hypohedonia is one of a dozen *normal-range* (*nontaxonic*) *individual differences factors* (*dimensions*) that raise or lower the probability of decompensation. I think it important to distinguish primary from secondary hypohedonia. The former is one of the several polygenic potentiators of decompensation (speaking causally). Secondary hypohedonia is a descriptive clinical feature which in some schizotypes is primarily attributable to a basic hypohedonia (Meehl, 1972a, 1974, 1974/75, 1987); and in others, who have not started out with a genetic loading for deficient pleasure capacity, attributable to aversive drift. I do not see how we can tell these apart in the adult acculturated decompensated case, unless we have some more direct physiological measure (Cacioppo, Martzke, Petty, & Tassinary, 1988; Cacioppo, Tassinary, & Fridlund, 1990; Hess, 1975). This is another example of a theoretical distinction that is probably important in drawing a correct causal diagram (see Figure 1 in the next section) but which is not answerable empirically until more auxiliary theories have been tested and high-validity detector instruments are available.

There has been some confusion in the literature dealing with my theory, perhaps due to too condensed exposition under the time constraints of a presidential address, as to the relation between the theoretical constructs hypokrisia, schizotaxia, schizotypy, and schizophrenia. The current psychiatric nomenclature contributes to this confusion because in DSM-III (*Diagnostic and Statistical Manual of Mental Disorders*, Third Edition—APA, 1980) one is not usually diagnosed as having a schizotypal personality if one is being diagnosed as currently a schizophrene, although on my reading of the criteria it is not forbidden. While I understand the motivation for handling it that way in the rubrics, it is unfortunate theoretically because the term "schizotype" comes from the writings of Rado, who delineated the syndrome at length; and for Rado one has a schizotypal personality, one is a schizotype, whether clinically decompensated or not. This is by analogy to other conditions in medicine, where a patient is diabetic whether or not she is presently having an exacerbation of symptoms; and one has a mitral valve disorder even though she may be compensated and free of cardiac symptomatology. The problem arises from the fact that DSM-III was intended to stick as closely as possible to the descriptive features, because it was not feasible to reach etiological agreement among doctrinally heterogeneous psychiatric committee members, despite everyone's understanding that in the better developed branches of organic medicine one *defines* a disease entity by a conjunction of the pathology and etiology (Meehl, 1973a, p. 287). In this sense current psychiatric nosology,

despite being thought of by psychologists as excessively influenced by the "medical model," is not in accord with the most powerful medical model. It is more like an old-fashioned 18th century list of "kinds of fever," demarcated by more or less arbitrary clusterings or clumps of the descriptive syndrome, which often include distinguishing features that are quite irrevelant either in theoretical causal understanding or in therapeutic indications ("Medicine," 1771). I repeat, I am not faulting the constructors of DSM-III for doing the only thing that was feasible to do in our present state of knowledge, given the ideological disagreements within the field (e.g., between organicists and psychoanalysts, hereditarians and environmentalists, polygenic and major gene theorists). I'm merely pointing out that it leads to a somewhat unsatisfactory semantics if one wants to think in a sophisticated manner about causal theories of the various disorders (Meehl, 1972a, 1977, 1986a; cf. Faust & Miner, 1986).

Since this paper is about my theory of schizophrenia, I feel free to depart from the semantic usages of DSM-III in exposition. I follow Rado in taking the schizotype as the fundamental construct at the level of psychisms, holding that a schizotype may have any degree of clinical decompensation from being well compensated, so that he does not even appear deviant to acquaintances unless they are very sophisticated observers; to one who suffers "pseudoneurotic" decompensation in the form of the Hoch–Polatin syndrome; to one who, while mainly pseudoneurotic, has micropsychotic episodes (if they are short enough they still fall into the Hoch–Polantin group); to one who is diagnosably *schizophrenia* by Bleuler's standards; to one who is diagnosably *dementia praecox* by Kraepelin's tighter standards. If there is sufficient impairment of the cognitive and executive functions of the ego so that we label the patient as "psychotic," "regressed," or "florid schizophrenia," Rado would use the term *disintegrated schizotypy*. If the patient remains disintegrated over a long time period (he tends to show a diffuse impairment in a variety of functions, including elementary ability to communicate, personal hygiene, massive withdrawal of social cathexis, and the like—the chronic, "back ward state hospital type"), that Rado calls the *deteriorated schizotype*. So they are all schizotypes, and they are schizotypes whether clinically well, pseudoneurotic some of the time, pseudoneurotic all of the time and psychotic once in a while, psychotic episodically with recovery and partial remission, unrecovered psychosis, and all degrees of general mental and social deterioration.

DERIVATION OF MAIN SIGNS AND SYMPTOMS

The conjectural causal relations involved when the person with a schizotaxic brain interacts with his environment are shown in the accompanying diagram (Figure 1), circulated among colleagues and students in 1966 and first published in 1972 (Meehl, 1972a). It is almost self-explanatory, but since it is subject to some misunderstanding or puzzlement, I take this opportunity to provide interpretative text. Some methodological guidelines are in order. When one speaks of "deriving" traits, signs, and symptoms from a substantive causal theory, one takes it for granted that only a "*quasi*-derivation" is possible in the present state of our general

knowledge of how the brain works, and, at the molar level, our imperfect and problematic theories of learning, cognition, motivation, and affectivity. Query for logicians: Just what is the meaning of "loose, quasi-derivation chain" from theories to facts? I still favor the old-fashioned deductive model, because philosophers who have rejected it have never succeeded in explaining what a derivation is when it is *not* a deduction. But a deduction, to go through strictly, has to assert a ceteris paribus clause (Meehl, 1990c). What it comes down to is, roughly, "Assuming such-and-such about the functioning of the brain (or psychisms), *and other things being equal*, how would such-and-such a neurophysiological aberration, leading to so-and-so parametric aberration in how some neural centers control the activity of others, and in the presence of such-and-such a mixed positive and negative reinforcement schedule, lead to the main traits, signs, and symptoms of this disease?" As I have argued above, the fewer auxiliary theories we can get by with, especially theories that are asserted *specifically about schizophrenia* (rather than theories that we take over from general "normal" mental functioning as currently understood), the better. I consider it a merit of my theory that the needed auxiliary conjectures, such as the existence of polygenic individual differences in certain CNS parameters, are not ad hoc to schizophrenia but known to hold for human minds in general.

We focus attention on the "big symptoms," where "big" means some combination of strong statistical presence if not pathognomicity, differentiating the schizotype and schizophrene not only from people in general but from other psychiatric diagnoses, and perhaps a kind of clinical importance, phenomenological strikingness, or oddity. Any theory of schizophrenia which cannot plausibly handle all, or almost all, of its main features, including those that appear only in certain diagnostic subgroups, is seriously defective.

We do not require a theory to explain the "minor phenomena," feebler or less powerfully discriminating signs that are shared with many normal persons or with other psychiatric conditions. This policy of selective explanation does not mean that we never would pay any attention to a low frequency sign. A sign that appeared in less than half of schizophrenes but appeared almost never in controls might be worth attending to, especially if its character is such as to be theoretically illuminating. For example, suppose an anatomical feature such as the dome head, or malocclusion, or the nailfold capillary bed anomaly, showed up in only 30% of schizophrenes but only 10% of controls. If that difference is not artifactual it is suggestive with regard to the endophenotypic causal pathway and should be of interest to the physiological geneticist. In considering signs having less than 50% frequency among diagnosed probands, one should keep in mind that when the theory being studied is a dominant gene theory, which implies a base rate of around 10% schizotypes in the general population, the life time risk of decompensating is only 0.10. Then when we screen out diagnosed cases, even correcting for age, we still anticipate a little less than 10% schizotypes among "people in general." These false positives, from the standpoint of the epidemiologist, are (speaking ontologically rather than epistemologically) *pseudofalse positives*. This means that a difference of only 20% in the above example could in reality amount to a difference of nearly 30% and to a high likelihood ratio $\simeq 30{:}1$. If there were a scarcity of strong indicators of

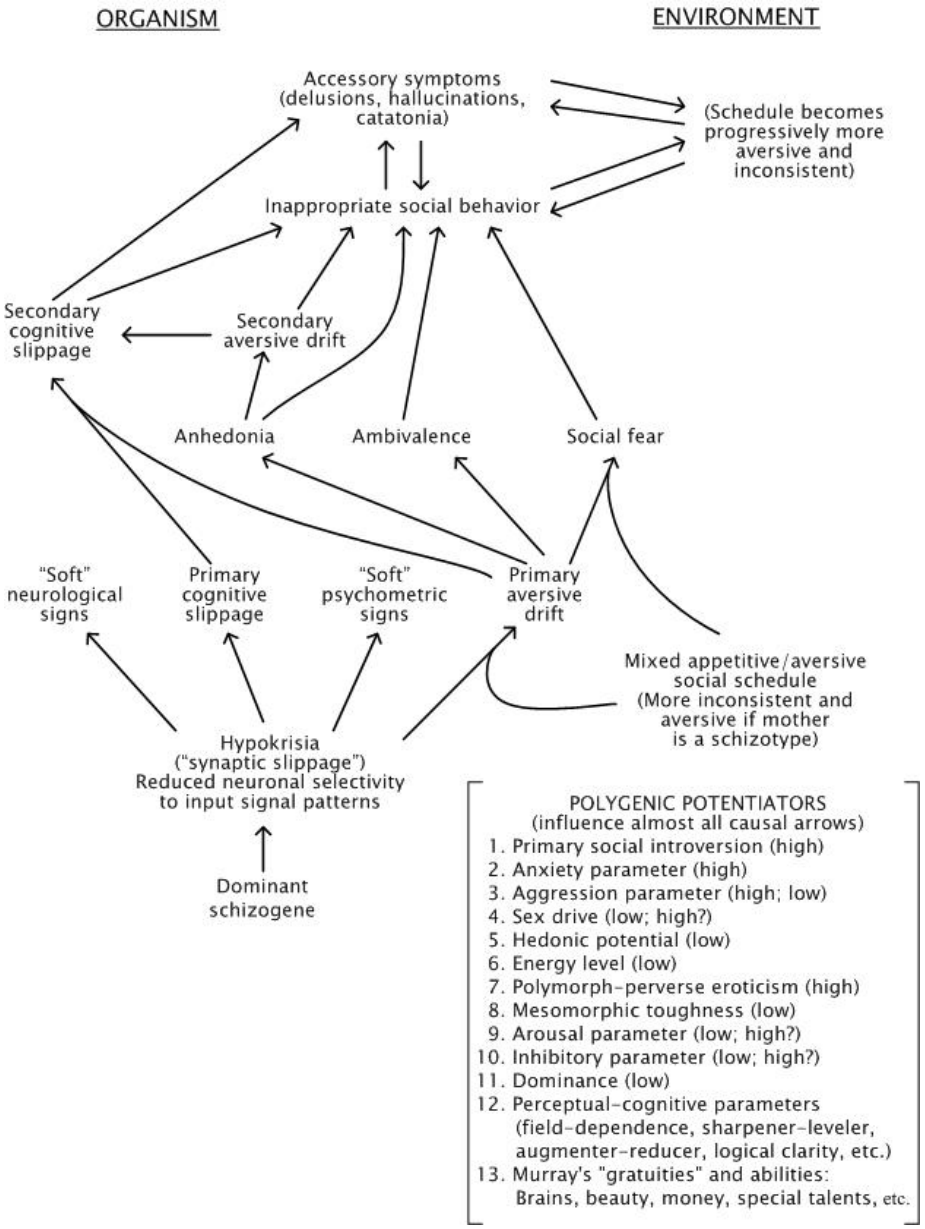

Figure 1. Causal chains in schizophrenia, minimum complexity. (Meehl, 1972, p. 16; 1973a, p. 190; 1989b, p. 941)

compensated cases, one could still employ Bayes's Theorem to achieve fairly good differential probabilities with a relatively small number of weak signs.

I take the schizophrenic psychodynamics from Bleuler, Rado, Arieti, Sullivan, Fromm-Reichmann, Rosen, Federn, Karon and VandenBos, and other clinicians who have studied schizophrenes intensively in long-term psychotherapy, buttressed by my own clinical experience. Space does not permit an exegesis of these writers indicating where I might deviate from their emphases, which would be a somewhat pointless exercise anyway. The important point in understanding my views is that unlike many clinicians of "biological orientation," I reject the position, which makes neither philosophical nor clinical sense, that when we find that the predisposition to a

mental disorder is genetic, and even learn something about the neurophysiology and brain chemistry of it, then what we thought we had learned from clinical experience, not to say psychometric and experimental study, of the schizophrenic mind should all go overboard as either false or as purely epiphenomenal without causal efficacy. Bleuler's neglected *Theory of Schizophrenic Negativism* provides a brief summary of the psychisms that produce the negativism so characteristic of these patients (especially for those of us old enough to have seen patients before the phenothiazines). After considering the predisposing causes of negativistic phenomena (ambitendency, ambivalence, the psychic splitting, and the imperfect logic) he focuses on the ordinary external negativism and speaks of the following causes as at work:

> (a) The autistic withdrawing of the patient into his fantasies, which makes every influence acting from without comparatively an intolerable interruption. This appears to be the most important factor. In severe cases it alone is sufficient to produce negativism.
> (b) The existence of a hurt (negative complex, unfulfilled wish) which must be protected from contacts.
> (c) The misunderstanding of the surroundings and their purpose.
> (d) Direct hostile relations to the surroundings.
> (e) The pathological irritability of the schizophrenic.
> (f) The pressure of thought and other difficulties of action and of thought, through which every reaction becomes painful.
> (g) The sexuality with its ambivalent feeling tones is also often one of the roots of negativistic reaction. (Bleuler, 1910–1911/1912, p. 2)

Eighty years later, I doubt that one could improve much on this account.

I incline to derive almost everything from two characteristics of the psychisms, which have in turn been quasi-derived above from hypokrisia, namely, the *associative loosening* and the *aversive drift*. The aversive drift leads to secondary hypohedonia, pain dependent pleasure, and ambivalence as described above. The impact of these psychisms is likely to be greater on highly stochastic reinforcement regimes and hence particularly powerful in the interpersonal domain. In fact, contemplating the previous list of parametric properties conducing to domain impairment, we see that the interpersonal sector is especially susceptible to all but D, "rare opportunities"; and for that one, the substitute adjustments are typically pathogenic ones (e.g., retreat to fantasy, overintellectualization, misinterpretation, passive–aggressive tactics, Rado's "coercive rage"). However, in our society with its technological aspects, its emphasis upon sociability and aggressiveness, and its demands on efficient performance, the usual work situation is sufficiently permeated with interpersonal components that a person with autistic, dereistic, socially aversive psychisms is likely to suffer a sequence of aversive conditionings and a deficiency of positive reinforcements educationally and vocationally. The chief exception to this tendency would be one so fortunate as to have special interests and talents, so that he can survive and be socially rewarded for artistic, literary, or scientific productions, becoming a successful Benedictine scholar, a poet, a professor of theoretical physics, or a maverick inventor. The autism in turn

conduces to a statistical reduction in consensual validation, both as to its success in reality testing and as to the amount of effort the patient is likely to apply to it. As time goes on, the autocatalytic and feedback effects of dereistic withdrawal lead to an increased feebleness of *effort* in most domains of life. It does not require an additional auxiliary theory to understand why such a person could be become increasingly occupied with the contents of fantasy; although as Beck (relying on the Rorshach) was one of the first to point out, one must distinguish between whether the fantasy is rich, which it frequently is not, or merely receives more cathexis than external reality, especially social object relations.

Should we consider Bleuler's accessory symptoms (delusions, hallucinations, catatonic phenomena) purposive? My theory does not give us help in that direction, and I doubt that anyone is in a position at the present time to say. At least my theory neither contradicts nor requires that all, or some, of the accessory symptoms be purposive. We should leave open the possibility that there are varying degrees of teleology involved, and I would prefer to substitute, for "purposive" or "motivated," simply "affectively–motivationally influenced," a weaker claim. It is the difference between Freud's insistence that every dream is at bottom the fulfillment of a wish, and the weaker statement, one that would probably be held by the majority of psychodynamic practitioners today, that the content of dreams merely reflects (as my first analyst, H. S. Lippmann, used to say) "what the unconscious is currently cooking." Some delusions and hallucinations do appear to fit the restitutional theory, but one must admit with the hard-nosed biological skeptics that others do not, except by the ad hockery that is always available in psychodynamic theory. Delusional and hallucinatory content that is not manifestly wish fulfilling, and in fact productive of affects of fear or rage, seem prima facie to speak against a restitutional view. But no thorough-going psychodynamicist would be bothered by that, anymore than by the aversive elements present in neurotic symptoms or dreams. Thus, the patient projects a homoerotic wish into the delusion of persecution with a homosexual theme, and we are not surprised that this is attended with manifest anxiety, at least in some patients and in the early phases. On the other hand, it is plausible to understand some delusional ideas as simply "explanatory." A patient claims that some malignant others use a machine that produces peculiar bodily sensations in him, which is the best he can do to explain them. Theory might attribute those sensations to first-order (unmotivated) phenomena directly attributable to the neurophysiological integrative defect. I doubt that the eight neurological items that appear on MMPI Scale 8 could reasonably be construed as consequences of some effort at reinvestment of cathexis in social objects, so I consider them as having the same origin as the aberrations in soft neurology and psychophysiology, such as SPEM, P50, and dysdiadochokinesia. If episodic dissociations of neurophysiological subsystems are rendered more probable in the sleepy state, as even Freud would admit in connection with the character of dreams, it seems reasonable for a person who experiences buzzings, tinglings, numbings, kinesthetic or genital sensations when half asleep—given general ambivalence and a hostile–fearful perception of the social world—to explain it by saying that "somebody is deliberately inducing these puzzling, unpleasant, and frightening experiences in me."

I cannot imagine any basis, experimental or psychometric, upon which we could, on present knowledge, come to a decision between these theories. And it is important, in thinking about that issue, to avoid the routine assumption (from the heyday of psychodynamics) that if we can understand the content of a delusion or hallucination as a direct or symbolic expression of a motive or an affect, *therefore* (prima facie or ipso facto) the reason why the patient hallucinates or has a delusion lies in the motive force of that drive or emotion. That, of course, does not follow, although it is consistent with a biological view while not derivable from it. Schizophrenics, like neurotics and normal people, have pretty much the same things on their minds as everybody else does, for example, love and hate, sex, power, status, dependency, closeness and the fear of closeness, success, disease and death, and the meaning of life. So we shouldn't be surprised if a schizophrene has delusions about seduction, persecution, politics, or religion rather than about more "neutral" topics, for example, the weather. I am not aware of statistics proving that the concentration of attention on what you could call minor or peripheral aspects of life, such as the weather, occur with greater or lesser frequency in schizophrenes than they do in the nonpsychotic conversations or preoccupations of normal persons. (I do recall a schizophrene I examined as a psychology intern who informed me that being God wasn't too bad a job except that sometimes it was a bit complicated managing the weather! Are we to assume that here that "weather" was a symbolic representation for some more significant content? I would be quite willing to entertain that hypothesis, but I would certainly not accept it as being a foregone conclusion.)

Like Bleuler, I view most of the phenomena of catatonia (which younger psychiatrists and psychologists don't get to see very often because in this country we start loading the patient up with antipsychotic medication as soon as we have a tentative diagnosis of schizophrenia) as part of the negativistic withdrawal psychisms. But this may be one of those cases in which we should invoke different levels of explanation, concurrently, as jointly needed for the effect to occur. I doubt that a patient with normal CNS, however strongly defended against intrusion from the external world or even concerning himself with having commerce with it, could retain urine to the point of rupturing his bladder, as occurred in the early days before it was realized that some catatonics must be catheterized. Even here, one must be careful about setting limits on the power of psychisms when we consider the different reactions to pain or the threat of pain in mystics and saints, or relatively "normal" people in different cultures as reported by anthropologists. One suspects that some catatonic phenomena, such as the waxy flexibility, come about by a combination of the negativistic and withdrawal psychisms with massive dissociative processes that *can be achieved* because of the fundamental neurophysiological slippage. That many aspects of catatonia have to be interpreted with reference to psychisms, rather than to some kind of first-level neurology, was clearly shown by Bleuler in his classic works, for example, superficially "opposite" social responses such as echolalia, echopraxia, and command automatism on the one side, and active negativism, where the nurses learn to get patients to do things by telling them to do the opposite, which one cannot explain directly in neurological terms. These apparently "opposite" symptoms are easily

understood as being different ways, sometimes both being manifested on different occasions by the same patient, of minimizing significant social interaction and inner concern about the demands of the social surround.

Bleuler included stereotypes of manner, posture, speech or even place (e.g., everyday after breakfast the patient goes and stands in the same corner of the room or under the same oak tree on the asylum grounds), although today most clinicians want something as dramatic as the waxy flexibility or mutism before they will speak of a symptom as "catatonic." I may be simplistic here, but I am not sure we need anything beyond common sense knowledge of normal people to explain, say, a stereotype of place. Most of us are creatures of habit, as anybody who thinks of himself as not at all habit-bound discovers in a hurry when the car won't start, or when even pleasant, easy to deal with houseguests visit for a few days! There are marked individual differences among normals, and among neurotic patients, with regard to how aversive a change in routine is or a disruption of expectations—what developmental psychologists and primate ethologists call "disconfirmation of a schema"; so we ought not to be surprised that among schizophrenes some are relatively more habit-bound than others. Perhaps no additional explanation is needed for something like a spatial stereotype than that the patient has a ubiquitous aversion to novelty, and does not want the activities of the inner life to be interfered with by unpredictability in either the social or inanimate surround. I am not a schizotype, but I like to take a long walk in the morning and think about clinical and theoretical matters before I begin to dictate about them. One reason—doubtless not the only one—why I always take the same path is that it has become so routinized and predictable that my scholarly ruminations are not being constantly interrupted by having to choose whether to turn left or right, which side of the street to walk on, or whatever. Here again, a commonsensical explanation by merely extrapolating from the behavior of nonpsychotic persons is compatible with certain places or objects having acquired a special meaning associated with the patient's complexes, as Bleuler would emphasize. We do not have a case of either/or that has to be settled, since mixtures of pure happenstance with katathymic choice could exist in all degrees from one patient to another.

Finally, the stereotyped behaviors that develop spontaneously in infrahuman mammals deprived of "ethologically normal" stimuli and behavior possibilities offers a suggestive analogy, as does the adult pattern of primates socially deprived as infants: "In sharp contrast to [the] wealth of opportunities for physical development and socialization in the wild, in a limited cage environment socially isolated chimpanzees showed both short- and long-term abnormalities…. These chimpanzees, deprived as infants, showed species typical facial grimacing, prolonged rocking, grooming movements out of social contexts, and gave screams and grunts while alone in an enclosure (called *in vacuo* by Lorenz…)" (Bauer, 1980, p. 103). The schizophrene's social withdrawal, while initially self-generated, makes his long-run psychological situation similar to that of animals isolated experimentally or in a zoo. Some simple, nondynamic CNS process (like sheer overflow of unchannelled arousal) could be enough to produce catatonic stereotypy, although here again we recognize that the *content*, the preference hierarchy, may be psychodynamically determined.

What about the unique, special, and in my view pathognomic (as an *inclusion* test only) aberrations in verbal behavior? I still like my 1962 example of the patient who explained his presence in the institution by saying, "Well, doctor, naturally I am growing my father's hair," which would not leave a first-year medical student or undergraduate psychology major in doubt as to the psychiatric diagnosis. I recall a patient I tested on my internship with the Healy Picture Completion II who had placed an alarm clock (instead of a book) in the picture where the boy is spilling his books on the way to school. Asked in the inquiry why he did that, he replied, "So he will have time to get to school." Nobody but a schizophrene would say such a thing. Or consider the patient who, when asked why his conversation was sometimes hard to follow by others, replied, "Oh, that's because I usually speak Echo Monster Head Affair Language." A fainter one, but which I find classified immediately by experienced clinicians, a patient (responding during the inquiry as to the aliveness of the usual lateral animals on Rorschach VIII) said, "Oh, yes, these images have the true resembling properties of live animals." One must distinguish between the rambling quality of some schizophrenic discourse (as in many of the examples in Bleuler's book), which can be understood primarily as a lack of a guiding, overarching goal, so that the patient speaks like somebody free associating on the couch or trying to write a "stream of consciousness novel," and the strange aberrations in syntax and semantics that can appear in discourse that preserves normal relevance, focusing, and goal-orientation. We may conceive of normal waking speech as controlled by a hierarchical CNS system (Miller, Galanter, & Pribram, 1960), which in molar language we describe as the regnancy of "mental sets" or the influence of "context" (Jenkins, 1974). Conjecturally this involves delicate balancing among quantitative values, the partial derivatives of the excitation level of a cell assembly or module with respect to the input levels from several others. There must be hierarchies of such subsystems operating in both the normal and the schizotaxic brain. At the top hierarchic level we are "keeping our mind on the subject at hand," such that a whole set of otherwise available module hierarchies are frozen out, dissociated, or somehow inhibited. It requires quite a complicated hierarchy to take care of even the elementary aspects of deep structure involved in getting the sheer grammar right, quite apart from customary semantics. The subtle deviations do not even violate the semantics. All they amount to, in my opinion, is a slight excess of low transitional probability word occurrences at various positions in a word sequence. I retain a belief that this is how it works, despite the imperfect performance of indicators like Cloze (Neale & Oltmanns, 1980; Taylor, 1953, 1956) and other linguistic tasks for diagnostic purposes.

This slight parametric aberration in hierarchical control of verbal dispositions would be conceived as springing directly from the synaptic slippage, with the usual contribution of normal-range individual differences in speech. But when that aberration is combined with a weakening of the overarching control, and then potentiated by katathymic intrusions (Bleuler), there will be severe degrees of schizophasic speech. So, here again, we move freely from one level of explanation to another as long as we don't have an inconsistency. We combine the primary loosening, a direct derivative of hypokrisia, with the higher-order failures of guiding inhibition attributable

to powerful complexes, the schizophrene's abandonment of interpersonal, task-oriented, and realistic problem-solving mentation.

As to "blunted affect," this standard language is pedagogically unfortunate, because it suggests to psychiatry residents and psychology students that there is normally an overall reduction in affectivity. Experienced clinicians know that the "blunting" is partly qualitative inappropriateness with respect to situation content and instrumental action, and, to the extent that it is a quantitative reduction in the intensity of affects, is mostly found in the positive domain. What Rado calls the "emergency affects" (fear and rage) are often of normal or even exaggerated intensity, although the element of inappropriateness is also evident there because of the sometimes puzzling array of elicitors. There is also a problem of the fine line between motives and affects, in the sense that what we ordinarily think of as a "normal affective tone" manifests itself as a sort of psychological pressure, tension, or "involvement" that is phenomenologically indistinguishable from the "arousal" that goes with a strong, regnant motive. Despite controversies and unsettled questions in the intracranial stimulation research, it seems generally agreed that stimulation of the appropriate regions tends almost always to have both a "priming" or "motivational" function and a reinforcing or "reward" function (Gallistel 1973, 1983; Gallistel & Shizgal, 1981). Aversive drift and hypohedonia (primary or secondary, usually both) leading to a decathexis of social objects in reality-oriented cognitive and instrumental processes, will appear subjectively (to the patient who can introspect and report) and behaviorally to the clinician, as "weakened affectivity." The cognitive and situational enfeeblement of *some* (not all) elicitors of the emergency affects in schizophrenia I conjecture arises partly from the fact that in normal and neurotic persons the emergency affects are often elicited by blocked strong positive strivings (the frustration–aggression hypothesis), so that an impairment or statistically unusual ranking among such reality-oriented strivings yields a distribution of fear or rage levels that strikes us as strange. Secondly, as I have suggested elsewhere in a context not directed specifically at schizophrenia theory (Meehl, 1974, 1974/75, 1987), if one way the emergency affects are normally inhibited, "buffered," or "softened" is by concurrent activity of Olds (+) centers, then one derivative of primary and secondary hypohedonia is an attenuation of that normal softening, hence disproportionate fear and rage, especially the latter, that we see in even chronic deteriorated schizotypes. Why more rage than fear? I conjecture, because a quasi-complete decathexis of social objects leaves less "to be afraid of," whereas motives of autonomy, hunger, depersonalized sex are still available as blocked initiators of the frustration–aggression sequence.

What about the "chaotic sexuality" emphasized by a number of clinicians and by one (Rosanoff, 1938) to the extent that he substituted that label, in the last edition of his famous *Manual*, for the chapter on schizophrenia? I am not satisfied with what my theory has to say about this mysterious feature of the syndrome, and must put this down as a debit against the theory, although I may say I have not come across any other theory of schizophrenia that does appreciably better. The simplest explanation, which goes a good way but somehow fails to satisfy me but is suggested by my colleague Garmezy (personal communication, 1989), is

that the general breakdown of normal social controls in florid schizophrenia permits overt or thinly disguised expression of erotic components that are present but aim inhibited in normal and neurotic subjects. (My clinical experience provides insufficient data on chaotic sexuality in psychotic manics. Do we know? This might suffice to accept Garmezy's answer). It seems possible that in addition to a straightforward explanation in terms of effective control over erotic components shared by all of us, we may get added explanatory help from the associative loosening, to which I *always* look for illumination in schizophrenia (as did Bleuler, although not as consistently as one might expect). The loosening may combine with the aversive drift, which is so heightened in the interpersonal domain, as a *positive* source of maintaining stronger polymorph-perverse components. One thinks of the establishment of classical psychodynamics' "genital primacy" as involving not merely the primacy of the genitals as erogeneous zone but, importantly in Freud, the affectional bonding with a sex partner (even if, in some persons, rather shortlived). If aversive drift, leading to weakened affectional bonding, and the associative loosening are both present, one might consider the Freudian "component instincts" as being, so to speak, imperfectly fused and marshalled, in the way they are when a mature person includes oral and exhibitionistic elements as part of normal courtship and sexual foreplay, the distinction there being one of the criteria by which Freud decided whether an adult was "perverse." I don't know precisely how to work the associative loosening into this picture, but I have in mind the fact, observed by others apart from Freud, that there is something a little strange about the sexual impulse in comparison with our other biological drives for food, water, shelter, and the like. There is an amusing passage in C.S. Lewis where he points out that if people would pay money to go to a restaurant and watch the waiter briefly expose a roast beef sandwich, that would be a very crazy way for the hunger drive to express itself! Freud, when trying to explain the specificity of the erotic pathogenesis, suggested that one way in which sexuality is special, perhaps unique among human motivational systems, is that connections form between sexual pleasure and various activities and persons, leading to a sometimes rich network of associations, and including some distorted cognitions due to the child's undeveloped ego and imperfect knowledge (the cloacal theory of birth, sadistic conceptions of coitus, etc.). Subsequently, in the teens when the sex hormones begin to circulate, this mixed up and unrealistic associative network is for the first time provided with a head of steam to drive the machinery. My thought, admittedly rather vague but I cannot do better, is that the tendency of the schizotaxic brain to form patterns of associative linkages functioning under a different distribution of parameters from the normal brain, and the extent to which the aversive drift in the interpersonal domain leads to autism, dereism, and excess cathexis upon fantasy, are partly responsible for some of the oddities of schizophrenic eroticism. I suspect that something along these lines, better worked out than I am able to do (and requiring more detailed knowledge of primate ethology than we now possess for the normal state), will play a role in understanding schizophrenic sexuality along with the powerful factors of inadequate consensual validation and weakened social controls.

GENETIC CONSIDERATIONS

Several writers (including, unfortunately, authors of general and abnormal psychology texts) attribute to me the view that schizotypy is inherited, which I have never said and, in fact, have specifically denied (e.g., Meehl, 1962a, p. 830f. [1973a, p. 140ff.]; 1972b, pp. 379, 396; 1972a, passim). Since schizotypy includes a set of *psychisms* having form and content based upon social learning, the development of various positive and negative cathexes in the life history, obviously one cannot, strictly speaking, inherit schizotypy, any more than one can inherit a schizophrenic delusion. I like to quote Bleuler here, "One cannot have a delusion about Jesuits if he has never learned about Jesuits." What I conjectured in 1962 was that *all schizotaxics develop schizotypy on all actually existing reinforcement regimes*. That was not surplus verbiage, and it was italicized there, but apparently that red flag to readers did not suffice. I was raising the possibility that a sufficiently prophylactic social reinforcement schedule might theoretically be good enough to prevent the development of the schizotypal personality organization. That seems somewhat optimistic, but one cannot exclude it. Since my theory of ambivalence, and the concentration of the aversive drift heavily in the interpersonal domain of life experiences, involves the stochastic character of the social reinforcement schedule, especially by the primary care giver, one can fantasize that an "ideal antischizophrenogenic mother," or perhaps some different mode of caregiving entirely, might minimize the aversive drift in the interpersonal domain such that a schizotaxic might end up being somewhat less socially fearful than, say, a nonschizoid subject with a heavy load of polygenes for garden variety social introversion. The point is not whether that is a realistic hope, but simply that it makes a theoretically important distinction. The extent to which a true phenocopy, or what I prefer to call a mixed "genophenocopy," of schizotypy is possible I shall consider below in connection with genetic heterogeneity.

What is, strictly speaking, heritable is the schizotaxic brain. In delineating this CNS anomaly, one may adopt varying amounts of specificity or detail in one's conjectures. Another clinician theorist might agree with me about the most broadly defined meaning of it but not when it comes to filling in the neurophysiological details. Schizotaxia, like schizotypy and schizophrenia, is an *open concept* (see Meehl, 1972a, 1977, 1990b; Meehl & Golden, 1982; Siever & Gunderson, 1983). The broadest definition is that schizotaxia is a genetically determined integrative defect, predisposing to schizophrenia and a sine qua non for that disorder, although I hold (like Rado) that only a minority of persons with the CNS defect decompensate to the point of being diagnosable by DSM-III or other "florid" criteria. So in this respect most schizotypes are like men with the genome for gout who do not develop clinical gout throughout their entire life (I believe the accepted figure today is that only about 1 in 20 males who have the gout genome, and the endophenotype of elevated uric acid titer, develop clinical gout). Of course, "integrative neural defect" is a vague term, as befits an open concept in the context of discovery (Reichenbach, 1938); but that a concept is open does not mean that it is empty or empirically meaningless

(Pap, 1953, 1958, chap. 11, 1962, chap. 3). By saying that the defect is *integrative*, I mean that it is not a defect of input, storage, or retrieval, but it is analogous to (but different from) some other well recognized integrative defects: dyslexia, tone deafness, the specific spelling defect in some persons of normal intelligence and verbal ability, nonanatomic dysarticulation syndrome, attention deficit disorder, and perhaps some extreme degrees of poor motor control in persons free of neurological injury. Like many constructs in medicine, when we do not completely understand all the causal machinery at work, we define partly by negation or exclusion.

But a further positive feature that I did not mention in 1962 (although I was aware of it because of my interest in the soft neurology known by then) is that it is ubiquitous, something wrong throughout the CNS. If you have the normal number of cells, normal conduction rate, normal ramified connections and so on, what could this defect be? Since learning involves a modification of micro structure at the synapse (we have to believe in the engram whether we like the word or not!), one conceives schizotaxia as ubiquitous aberration in some functional parameter of synaptic control, as explained above. Finally, the very specific conjecture about hypokrisia speculates as to how this synaptic dyscontrol originates. So the relation between these three concepts—hypokrisia, schizotaxia, schizotypy—is one of *class inclusion* and *causality*. Obviously another clinician theorist might agree with Rado and Meehl about schizotypy but not share the view that it involves an inherited integrative defect of the brain. Or one might agree that it involves an inherited defect of the brain but is something about the wiring diagram (microstructure) rather than the functional control parameters at the synapse. Or one might agree that it is ubiquitous and involves synaptic control but deny the specific idea of hypokrisia in terms of signal selectivity, and the associated notion of a substance whose normal function is to stabilize the cell membrane and resist depolarization. I have been faulted for needless proliferation of language in distinguishing schizotaxia from schizotypy, since on my theory they are perfectly correlated in the population. Suffice it to point out that a correlated extension of two attributes does not collapse them into one attribute. As logician Quine likes to point out, the taxonomic fact that every animal with a heart has a kidney does not mean that the terms “heart” and “kidney” are synonyms to the biologist! Here we have different levels of analysis, one (schizotypy) in terms of psychisms, a second (schizotaxia) as an open concept about the integrative function of the brain, and a third (hypokrisia) as a specific conjecture about the neuron level process that underlies the schizotaxia. They are three distinguishable concepts, which is why a theorist could buy one of them and not the other two, or two of them and not the third. So we need a semantics adequate to discuss them.

It is not viciously “circular” to make the diagnosis of schizophrenia hinge theoretically upon the schizotaxic defect as etiological, as that is exactly the kind of causal model that has been so successful in medicine, genetics, and other areas. For one who holds such a theory, that is the rational way to proceed diagnostically, although one may be constrained by administrative considerations (e.g., third party payment) to apply the terminology in accordance with the accepted conventions of DSM-III. But surely we ought not to allow scientific theories to be subservient to the economics of health

insurance, especially operating in the context of discovery! If one thinks that a disease has a specific etiology, then it is incoherent to diagnose patients as having this disease if one does not think that they have that specific etiology. There is no need for disputes about this, because in clinical practice we adopt the accepted terminology that is specified by the rules of the game, but that ought not constrain us in the research or theorizing enterprise.

I have argued elsewhere (Meehl, 1972b, 1973b, 1989b, 1990b) that the geneticists' semantics are not optimal for theorizing about this kind of disorder. The technical terms "penetrance," "expressivity," "trait," and "modifier" are intimately linked in standard genetic usage, and it does not become a psychologist to pontificate to geneticists how they should employ their terms of art. However, within my kind of theory I need a different concept from that designated by the term "modifier," and I think of the relationship between "penetrance" and "expressivity" for something like schizophrenia in a slightly deviant way from the geneticist's standpoint. Since I have discussed these elsewhere I can be brief about it here. Genetics texts customarily introduce the concept of penetrance as primary, and then go on to say that if, in a given gene carrier, the gene manifests itself phenotypically (penetrates), then one goes on to quantify that manifestation. Expressivity is, so to speak, a secondary or derivative concept. I view this as, if not a conceptual mistake, at least an inconvenient convention in the use of these terms. For many of the phenoltypic indicators of a gene (or set of genes), the indicator, if examined closely, is present in various degrees. Even manifestations that one normally thinks of as a present-or-absent quality (such as having or not having an extra digit on the hand) are, in fact, present in varying degrees, although the distribution may be bimodal. It is convenient for a psychologist who comes from the psychometric tradition to look upon the quantitative aspect as the more general one and to formulate matters accordingly. This convention does not prevent those indicators that are intrinsically dichotomous, having a sharp cutting score, step-function, or present/absent qualitative attribute, from being so described. We do this when we consider the dependent variable in a linear discriminant function as a "numerical" variable that takes on only integral values zero and one. From this viewpoint the basic concept mathematically is expressivity, whereas penetrance is a more or less arbitrary percentage, based upon adopting a conventional cutting score for saying that the trait is "present" or "absent."

Suppose a dominant gene results in a shifted distribution of some phenotypic indicator variable x in a specified breeding-cum-environment population. We may not know the parameters of this indicator distribution, but we know that it must exist; that is, it is a fact of nature rather than a construction of the human investigator. The penetrance is the proportion of gene carriers above a specified cutting score, and as in psychometrics, this cutting score will have an element of arbitrariness about it. If I raise the cutting score, I will decrease the penetrance. So penetrance is mathematically a secondary variable, dependent on a conventional cut on the expressivity function. The exception would be the special case in which there is zero overlap between expressivity functions of the heterozygotes and the (normal) homozygotes, in which case the penetrance is 100%. So we have as

many penetrances as we have possible cutting scores on the expressivity function. A further complication arises in the case of a complicated disorder like schizophrenia, or any other psychopathologic taxon except a truly monosymptomatic disorder (are there any?). The phenotypic syndrome is a "loose syndrome," in that the pairwise correlations of the indicators that define it are not uniformly $\simeq 1$. One can of course set up a count-and-pattern convention, a disjunction and conjunction of signs and symptoms, as is done in the syndromes specified by the criteria of DSM-III. We specify that the patient must have at least three of the list S_1, S_2, … S_5, and at least one of the list S_6, S_7, provided he does not have S_8 or S_9. No one maintains that such stipulations are based upon an optimizing statistical procedure, such as Bayes's Formula.

Consider a schizotype who has many polygenes for social extroversion and few polygenes for the anxiety parameter, as a result of which he is the "friendly (and often socially inappropriate) schizotype" that one sometimes meets as a stranger on the street. If our concept of the schizotypal syndrome lays emphasis (as it would for most clinicians) on social withdrawal, this schizotype doesn't fit the syndrome very well. (Perhaps he would if we had a representative sampling of situations.) Does it have a clear mathematical or biological meaning to ask whether the dominant schizogene is "penetrant"? I don't think so.

Similar problems arise with respect to the geneticist's concept of a modifier. In a theory like mine, polygenic variables such as anxiety proneness or social introversion or hedonic capacity do not literally "modify" the expression of the schizogene which is, at the endophenotypic level, the neurointegrative anomaly in the brain, and which expresses itself directly in the exophenotype in the form of soft neurology and psychophysiology. Again, I do not conceive the characteristic psychisms of the developed schizotypal personality makeup as "modified" by something like the polygenic introversion variable, in the way that modifying genes influence gene expression in the fruitfly. The causal situation is quite different for psychopathology, especially because the manifestations that we focus on clinically have a socially learned *content* (e.g., suspicions about Jesuits). It's not as if the polygenes for introversion somehow "get into the causal chain" between the schizogene in DNA and the parameters of social reinforcement. These parameters are manifested in the exophenotype, as are intelligence, energy level, dominance, and the like. It is the interaction between these dispositions of the exophenotype and the social environment, with its stochastic reward/punishment schedule, that influences the course of the schizotypal personality development and determines whether a schizotype decompensates with schizophrenia. A high IQ enabling you to be a successful "mad scientist," so you can stay out of the mental hospital by living in the sheltered setting of academia despite your schizoid makeup, is a totally different kind of causal situation from an epistatic gene in the fruitfly, determining whether the first gene expresses itself in an altered eye color or wing shape. The causal chain leading to a schizophrenic delusion resulting in an attempt to assassinate the governor is extremely complicated, involving many feedback relations in learning processes both of cognitions and cathexes, maybe over many years, in which multiple genetic systems completely unrelated biochemically, not linked to the schizogene, produce

exophenotypic dispositions that have no "descriptive behavioral qualitative overlap" with the more specific consequences of the schizogene. Such a situation is more analogous to one in which a person unfortunate enough to have inherited the genes for diabetes, as well as the genes for alcoholism, traumatizes his foot while intoxicated, as a result of which he develops a diabetic gangrene and has to have an amputated leg, which mobilizes his infantile castration anxiety (Meehl, 1973b, p. 220). It would be a strange way of using the geneticists' technical language if we were to talk of the gangrenous leg, let alone the castration anxiety, as being an "indicator" and, as a percent of diabetic genomes, a statistical estimator of the penetrance of that genome. (Note that if we were to permit such a locution, we could with equal semantic justification consider the castration anxiety a measure of the alcoholism gene's "penetrance," a pretty silly way to talk.) The confusing character of such semantics arises chiefly because such complex causal situations require analysis in terms of J. L. Mackie's INUS condition (Mackie, 1965, 1974; Meehl, 1977). For this reason I require another neologism for which I have employed the term *potentiator*. A potentiator is *not* a genetic modifier of a particular causal chain from the DNA to an exophenotypic position. It does not have that intraorganismic specificity. A potentiator is any genetic factor which, given the presence of the schizogene *and therefore of the schizotypal personality organization*, raises the probability of clinical decompensation. It is conceivable that a certain potentiator may sometimes operate in the opposite direction—as a *de*potentiator—when certain other polygenic factors are present.

There are as many expressivity functions as there are facets of a loose syndrome, which is one reason I prefer to speak of "indicators" rather than of "traits," and of "clinical penetrance" only as a concession to the conventional usage. As regards traits, the psychologist is entitled to give the geneticist some advice, because "trait" is a term of art in both disciplines. A psychological trait, when carefully analyzed, almost invariably turns out to be a more or less loose cluster of first- or second-order dispositions (Broad, 1933; Carnap, 1936/37). To question the existence (reality) of a phenotypic trait is to question the occurrence of substantial pairwise correlations between the members of such a cluster of elementary dispositions. The other condition for the reality of a phenotypic trait is that the members of this correlated cluster of dispositions ought to have some discernible common property at the descriptive level, or close to it. For further discussion of the methodological problems arising in respect to the trait concept see Meehl (1986b). That a construct like schizophrenia or cyclothymia is not, psychologically, *a trait* but rather a loose statistical cluster of traits contributes further to the fuzziness of the penetrance concept, since in addition to there being as many penetrances as there are cutting scores on a given indicator variable, if those were somehow fixed we still have as many "penetrances" as facets of the syndrome. While I mainly want to use language capable of expressing the concepts with as much precision as the theory of open concepts permits, I admit to a slight missionary urge vis-à-vis the geneticists so that they will realize that in the area of psychopathology the character of the causal relations between the exophenotypic dispositions and the environment is such that a special terminology, such as "potentiator," would help us all around in thinking clearly.

Since only about 10% of schizophrenes have a schizophrenic parent, or, putting it another way, the risk to parents collectively is in the neighborhood of .05 (Gottesman and Schields, 1982, table 5.1, p. 85), on a dominant gene theory, the "clinical penetrance," if one talks that way, is only around .10. Since the lifetime risk for schizophrenia in the general population is approximately .01, it follows that the schizotaxia base rate in the general population must be around .10 (so the schizogene frequency must be around .05). I find that this high base rate bothers geneticists and some psychologists, but I don't understand quite why it bothers them. There seems to be a habit of thinking that any "mental disorder" should have a very low gene frequency, something like PKU or the other Mendelizing mental deficiencies. For me, schizophrenia is more analogous to diabetes, gout, hypertension, coronary disease, or rheumatoid diathesis. It seems to me that all we need to say about that alarmingly high base rate is that we should get used to it. After all, the prevalence of diagnosable schizophrenia is orders of magnitude larger than the Mendelizing mental deficiencies, so why should we be shocked at a conjecture that the mere predisposition, as a sine qua non (requiring concurrence of several genetic and environmental potentiators for illness), is as high as 10% in the population? Probably this comes partly from the malignant connotations of the *word* "schizophrenia" as a kind of bad outlook "cancer of the mind." When one thoroughly assimilates Rado's concept of schizotypy, and thinks about the fairly contented and highly achieving schizotypes like "Kepler and Newton, Calvin and Kant, Schiller and Rousseau, Erasmus and Spinoza, Whistler and Goldsmith, Wagner and Chopin, Robespierre and George Washington…" (Menninger, 1930, p. 76), matters appear differently. Naturally the schizotypes one knows as a clinician are more or less distressed, at least part of the time, or we wouldn't find them in our practice. For someone with my views, who finds many schizotypal nonpatients among colleagues, neighbors, students, it seems evident that there are a lot of schizotypes who lead relatively contented and fulfilling lives, especially if their polygenic situation is favorable, (e.g., not too much anxiety parameter or hypohedonia), and if they have good fortune in their occupational choices and their choices of a mate. Of course this is an epidemiological question that has to be answered by empirical data. All I mean to say here is that the armchair objection "Oh, my goodness, we couldn't possibly accept a schizogene frequency as high as .05!" does not bother me.

However, it does pose a problem because of the low fertility of schizophrenes. As is well known, both the polygenic and major locus theory have difficulties about this, but admittedly a major locus theory is in somewhat worse trouble. Why hasn't the adverse mutation been eliminated by now? I look upon this as a weighty objection to a major locus theory, and I have not seen anybody give a satisfactory reply to it. Methodologically speaking, how much work one should go to in attempting to concoct a reply depends upon how strong the evidence is (or becomes) corroborating the dominant gene theory (Meehl, 1978, 1990c). If taxometric research on families should eventuate in such support, we would be warranted in trying to explain away the evolutionary paradox in accordance with philosopher Clark Glymour's advice "Do not make a mockery of honest ad hockery" (personal communication, 1980). The extent to which one is well

advised to adopt a strategy of Lakatosian defense depends upon how much money the defended theory already has in the bank by having passed stiff tests or having predicted Wesley Salmon's "Damn Strange Coincidences" (Salmon, 1984, personal communication, 1980). Those convinced that a major locus theory already stands clearly refuted will of course have no reason to work on solving the microevolutionary paradox it presents. For the others, I offer some ad hockery for whatever it's worth:

Since schizotypy is not the same as schizophrenia, we cannot move directly from the impaired fertility of diagnosed schizophrenes to an equal impairment for schizotypes. The development of psychotic symptoms early in life is presumably a more severe impairer of reproductive fitness than merely having a schizoid personality makeup. It is well known that the fertility impairment is considerably lower in female schizophrenes than in males. I think a semicompensated female schizotype sometimes combines a certain carelessness about contraception with a special kind of erotic attractiveness (alluded to briefly in my 1962 paper), especially for neurotic males. In my 1962 paper I conjectured (although I have no quantitative support) that a plausible family constellation genetically for a schizophrene consists of a nonschizotypal father with anxiety or introverted polygenes, attracted to a compensated schizotypal mother, so that the proband gets the specific genetic loading from the mother's side and the adverse polygenic potentiators from the father's side.

If schizotypy had in past time some survival value for individuals, this could countervail a somewhat lower reproductive rate for those that survive. It has been suggested to me by a student that the narcissistic element of the schizotype could quite possibly have a survival value in the individual refusing to engage in group conflict such as war, raiding parties, etc., which are dangerous. Then there is a fairly good likelihood that in some preliterate cultures the shaman becomes so because of schizotypal characteristics such as hallucinations, dissociative tendencies, and the peculiar charisma of some bright, dominant schizotypes (Hitler, Wittgenstein). But I agree that this kind of speculation is dangerously ad hoc, perhaps beyond Glymour's boundary of honest ad hockery.

A more plausible line of thought is a culture–history one, that heterosexual aggressiveness was probably far less relevant to mating in the past than it has been since modern times. This is not entirely ad hocking. Historians and sociologists tell us something about this, without specific reference to psychopathology. In the 12th century mating often occurred by sheer propinquity; that is, you married the milk maid who lived next door, or married by family arrangement, especially in the upper classes and nobility. The active heterosexual seeking (e.g., "getting a date") which characterizes our society's courtship patterns played a smaller role in whether one married or not in past times. So perhaps until recent centuries the reproductive fitness of the schizotype did not differ as much from the normal as in our day.

There may be features of modern culture that enhance the decompensation likelihood—urbanization, ambiguous roles, competitiveness, mobility, anonymity, "mass society," weaker family bonding, individualism, anomie, decline of religious faith—on which I entertain no opinion. Some scholars believe these societal conditions are schizophrenogenic, so the

lesser percentage of decompensated cases in past times would have meant less reproductive effect.

Finally, I offer an unusual hypothesis which my colleagues tend to greet with disapproval or incomprehension. It is possible that what we consider the "normal" gene, as contrasted with the schizogene, is the gene that *has been slowly taking over*, and has now reached a gene frequency of .95 in the population. Population geneticists almost always make some kind of formal bow to the importance of evolutionary theory in their thinking, but in concrete instances they do not do that when behavior disorders are involved. (Perhaps not for some nonmental disorders, either? How about nutritional aberrations, such as lacking an enzyme for katabolizing a certain food substance?) Why is this? If human evolution is considered to be continually taking place, since we have evolved in quite important respects from our hominid ancestor (I presuppose here the Theory of Evolution, although I have doubts about its scientific adequacy; see Meehl, 1989, p. 378), presumably a number of genes are characteristic of modern man that arose as mutations in our remote ancestors. Why is it invariably assumed in the field of psychopathology that the undesirable gene is the mutation, never the "healthier" or "more advantageous" one? One might conjecture, as to hypokrisia, that a fairly recent primate mutation, within genus *Homo*, might have taken place involving a further stabilizing of the neuron cell membrane, with a resulting increase in signal selectivity which would—for an animal increasingly dependent upon its "smarts"—have an advantage. Putting it crudely, one may raise the question whether ancestral man perhaps "thought more schizy" than we do. Such a theory is hardly testable by any conceivable evidence, but one should keep in mind that when a fact such as low reproductive fitness is offered as a definitive *falsifier*, it is legitimate methodologically to look at the auxiliary hypotheses that are being considered nonproblematic, without which the falsification argument does not go through modus tollens.

HOW TO TEST THE DOMINANT GENE SCHIZOTAXIA THEORY

The basic strategy I favor to test a dominant gene theory of schizotaxia is somewhat different from the conventional approach familiar to geneticists. I think that the causal (and hence statistical) structure of a genetic–environmental model complicated enough to be even plausible is such that statistics of family member risks for a formal diagnosis of schizophrenia have taken us about as far as we can go unless some new analytic method is invented.

> I am aware that the major locus idea is an unpopular one among behavior geneticists and that some, such as my friend and former colleague Professor Gottesman, consider it to have been refuted (Gottesman & Shields, 1972, 1982; O'Rourke, Gottesman, Suerez, Rice, & Reich, 1982; McGue, Gottesman, & Rao, 1983; [but see] Holzman et al., 1988). This is not the place to go into the details of that, and I am not competent as a behavior geneticist. I content myself with saying that the alleged refutations do not deal with the type of complicated causal

> model that I postulate. In designing empirical tests of a theory one must be sure to "give the theory its head" without being circular, sometimes a thorny problem. I am skeptical as to how adequately we can test a major locus theory with polygenic potentiators by employing formal diagnosis analyzed by conventional methods. Consider the following factors, an incomplete list which surely underestimates the true causal complexity: In studying families, we must either exclude with near certainty, or somehow "correct for," genophenocopies like the SHAITU syndrome below, meanwhile keeping in mind that *its* components operate as polygenic and environmental potentiators for true schizophrenia. The analysis must take account of the sex difference in impact of schizotypy on fertility, and these numerical values cannot be safely extrapolated from the data on full blown schizophrenia. There may be positive or negative assortative mating for the schizogene taken over the whole range of clinical compensation, despite its negligible amount for formal diagnosis. There will almost certainly be assortative mating for the polygenic potentiators, and some of these correlations could be negative (e.g., dominance). The potentiators are not likely to be equally impactful on fertility for the sexes, partly on their own, but partly because of different potentiating effects for decompensation and, hence, fertility. The parental "side-concentration" of polygenes must be included in one's statistics, because a proband (who has decompensated or he would not have been ascertained) is more likely to have received his potentiating polygenes from both sides than from the schizotypal parent, since if the latter carried a heavy loading of them, he would have been less likely to mate. And this side-concentration effect is probably different for the sexes. Psychodynamically, one would expect differences in the potentiating effect of identification with the schizotypal parent depending on whether that parent is the same sex as the proband, and this complication will interact with the sex difference in fertility. Finally, we only locate families where some member has decompensated, and the diagnostic statistics suggest that there must be around 10 times as many schizotypes in the population as there are diagnosed schizophrenes. The family statistics for this path diagram, even if the small and unstable figures for second degree relatives are included, will involve fewer equations than unknowns, so no good test is available. (Meehl, 1989b, p. 939)

I therefore look upon formal diagnosis of schizophrenia primarily as a means of locating the families within which the expected schizotaxic incidence is high enough to be susceptible of satisfactory analysis. (Preferable to this high-risk family approach, and the only way to get an accurate value for the "clinical penetrance," that is, the joint influence of polygenic potentiators and environmental stressors, would be a large-*N* epidemiological survey employing the soft neurological, psychophysiological, and psychometric indicators shown valid for schizotypes in remission and studying their pattern statistics taxometrically.) The syndrome looseness and the complications resulting from that (e.g., What weight do we give various indicators in classifying a subject?) leads me to advocate a taxometric approach to the analysis of *indicator patterns* as an indirect test capable of

refuting a major gene model. Despite the statistical indirection involved in such an approach, I believe it to be capable of yielding a stronger Popperian test than further studies of the percentage of diagnosed schizophrenes among MZ and DZ twins, parents, and siblings. One does not demand a quasi-infallible marker trait, although the Super Bootstraps Theorem (Meehl, 1973b, 1989b—for the proof see 1990b; Meehl & Golden, 1982) will enable us to find such a marker should it exist. For a clarifying analysis of the loose concept *marker*, distinguishing susceptibility ("risk," vulnerability) markers as either (a) nongenetic, (b) merely gene pool correlated, (c) genetically linked, hence familial, or (d) pleiotropic indicators of the pathogene, see Iacono (1985). I always employ the term "indicator" to denote type (d), because we are concerned only with pleiotropic markers when testing the dominant schizotaxia theory.

One chooses indicators from *different levels of molarity* and *qualitatively diverse domains* for several reasons. *First*, the domain content is relevant to theory. For instance, indicators at a more molar level are links to psychopathology, whereas the less molar (e.g., soft neurology) are links to the schizotaxic CNS defect. Suppose the taxometric analysis reveals that a psychometric indicator such as an MMPI score, or an interviewer rating on hypohedonia, behaves as a relatively strong indicator of a statistically identified taxon among the nonschizophrenic relatives of our probands, whereas soft neurology signs such as the P50 evoked potential anomaly or the SPEM sign do not; those two facts would constitute strong discorroborators that what is transmitted genetically is a general schizotaxic integrative defect, rather than something more specific to social bonding. *Second*, one must permit a certain amount of conceptual drift on the basis of taxometric bootstrapsing procedures, *but not too much*. We must be able to answer affirmatively the question, "Are we now studying something basic, not schizophrenia but a predisposer condition?" and yet be able to answer negatively the dangerous question, "Are we now studying something not causally related to schizophrenia at all?" The latter, for example, can easily happen if an investigator latches on to a powerful polygenic potentiator, especially when the clinical penetrance of disease is low as the dominant model requires. While the presence of the specific schizogene is the sine qua non, the decompensation variance is heavily contributed to by such a potentiator, so it could statistically swamp the influence of the schizogene, due to the looseness of the syndrome and our lack of clear guidelines as to how diagnostic indicators change their relative importance when we move from the decompensated through the semicompensated to the compensated range. *Third*, a less molar, less "psychological" or "social" kind of indicator is connected to the DNA by a shorter causal chain, hence fewer attenuating factors are involved, and there is a better likelihood of its being a taxometrically strong indicator of the schizogene. *Fourth*, diversifying our levels and domains tends to hold down nuisance correlations within the latent classes, which improves the taxometric situation, being closer to the idealized model in the taxometric statistics. Also the diagnostic probability values computed by Bayes's Theorem go up more steeply with more signs present if they are relatively independent within the taxon and the complement class, so that one can be more confident in classifying

individuals. *Fifth*, if a quasi-infallible indicator should exist, it will almost certainly be one at a less molar, nonsociopsychological level.

My overall approach to a statistical treatment of this question could be described as neo-Popperian. It involves several somewhat deviant methodological guidelines. I do not consider it useful merely to refute null hypotheses in testing a substantive theory (Meehl, 1967, 1978, 1990a, 1990c). Point predictions are crucial, as usual in testing any strong genetic model. But one should avoid strict falsificationism (hence the label "neo"-Popperian) and should follow a "good enough" rule of thumb (Dar, 1987; Meehl, 1990c; Serlin & Lapsley, 1985), since the idealizations, both genetic and taxometric, are surely not fulfilled precisely by the real situation. One should rely on multiple avenues of inference to the theoretical parameters, rather than focus upon optimizing statistics such as *MLE*s or exact confidence belts (Meehl, 1978). We average multiple estimates of the taxon rate *P* of our sample. Who can specify "the population" from which a sample is allegedly randomly drawn? Obviously there are all sorts of schizotype-carrying families out there, the great majority of which do not come to our attention because of the low rate of decompensation. The role of random sampling error here is not the conventional one. Rather it is random error as a disturbing source of local irregularities, discontinuities, "holes in the Swiss cheese," "coarseness of grain in the graphs we draw." Finally, coherency tests play a critical role in taxometric bootstrapping. We want to show that we get the same taxon rate using different indicators, or using different taxometric algorithms on the same indicators, or a mixture of the two.

One can set up guidelines for the selection of candidate indicators. Candidate indicators should discriminate between groups as follows:

a. Schizophrenes from affective disorders and from normal controls (strongly).
b. Schizophrenes in remission from major affective disorders in remission and from normal controls (strongly).
c. MZ twins of schizophrenes from normal controls (strongly).
d. Siblings of schizophrenes from siblings of affectives (moderately) and from siblings of normal controls (moderately).
e. Preschizophrenes from normal controls (strongly).

The list of groups that a candidate indicator should *not* discriminate appreciably is as important as the positive requirements. A good candidate indicator should *not* discriminate:

a. Schizophrenes from schizophrenes in remission (or only very weakly).
b. Affective disorders from normal controls (or only very weakly).
c. Siblings of affectives from normal controls.
d. Schizophrenes as to clinical severity or as to subtype.

In evaluating these ideally null differences one must take into account whatever reasonable overlap bounds can be set on the basis of how the probands and others were diagnosed (e.g., structured interview? multiple examiners?

psychometrics?), since diagnostic error generates a spurious overlap that does not invalidate the schizospecificity of an indicator.

The reader may be surprised that I have said nothing about discrimination between schizophrenes and nonaffective "miscellaneous psychiatric" or "neurotic" controls. That is because the schizotypy rate among these patients is controversial. Expert opinion varies widely as to what proportion of miscellaneous psychiatric patients, diagnosed neither schizophrenia, major affective disorder, nor organic brain syndrome are schizotypal. The estimated base rate of schizotypy ranges from .10 to nearly .90 (I know a skilled clinician who estimates that nearly 90% of general psychiatric outpatients are schizotypes, and Rado held the base rate to be at least a preponderance). The point is not whether the reader (or I) would agree with any such extreme high or low estimates, but that finding a low, middle, or high discrimination *would not falsify anything at this preliminary stage*. Since my own estimate, partly from my private practice and partly from a research study (Golden & Meehl, 1979) is around 35–40%, I could put only weak constraints on the size of statistical separation between miscellaneous psychiatric patients and schizotypes as a preliminary screen for an otherwise good looking indicator. *Extreme case:* A false-positive rate of 10% in diagnosing schizophrenia (optimistic, even using SADS plus psychometrics) with a schizotypal rate in mixed psychiatric patients of 80% could easily yield a small (and statistically nonsignificant) difference on a highly valid sign.

Medication must either be eliminated or matched. For example, we know that bipolars in remission can show the schizophrenic SPEM aberration if they are on lithium.

I must emphasize that these criteria should be relied on only to *choose candidate indicators* and to *maintain the connection with the diagnostic entity of interest*. They should not be used to estimate relative sign validities, or to assign indicator cuts, which latter is done later as a result of coherent taxometric bootstrapsing. It could easily happen that the merit of two indicators as judged by size of the listed separations would differ from their true merit for discriminating schizotaxia, and therefore we want to infer this latter almost wholly from the taxometric results. The preliminary indicator screen is qualitative, not quantitative.

One prefers quantitative indicators to qualitative (yes/no or trichotomous) ones wherever possible. Some that are conventionally dichotomous should be quantified. *Example*: A favorite of mine, a (±) dysdiadochokinesia (which has shown up in a couple of reviews of the soft neurology but is not strongly emphasized by most practitioners or researchers), should be studied by instrumentation and mathematization. Instead of the neurologist simply looking at the patient when he tries to pronate and supinate hands or rotate clenched fists in the usual neurological exam, one could have the patient hold a small rod in each hand which would have a light source at each end (aiming away from the patient). We photograph the light spot movements and analyze the resulting pattern with respect to phase difference, amplitude difference, and drift of the centroids. I predict that this combination of instrumentation and quantitative analysis would reveal dysdiadochokinesia as a fairly powerful indicator of the integrative defect, perhaps as good as physiological signs like P50 or SPEM. A (±) Romberg

should also be instrumentated and quantified, as was done many years ago by the Worcester group (Angyal & Sherman, 1942; Freeman & Rodnick, 1942) and later by Eysenck (Eysenck & Eysenck, 1976). I list here some plausible indicator candidates by domain, each of which should be screened on the preliminary list of criteria for discrimination and lack of discrimination above. I cannot cite published research for some of these, either because they come from my own clinical experience, or lore passed on by other perceptive clinicians (Starke Hathaway, George S. Welsh, Harold Gilberstadt, Bernard C. Glueck), or because I cannot locate a source.

1. *Anatomical* (some of these may be behavior-residuals):
   a. The nailfold capillary bed anomaly (Buchanan & Jones, 1969; Maricq, 1963; Maricq & Weinrich, 1980).
   b. Facial asymmetry (Wolff, 1943). Here I would employ the foldover photo method and present the concocted full-face pairs to judges who would be told to rate on degree of similarity, or perhaps to guess whether they are MZ or DZ twins.
   c. There is a kind of spatular-spidery hand that I think I can recognize when I see it, but I do not know how to describe it verbally, and I am unaware that anybody has ever researched it. This could presumably be done by photographs, although that could lose some information contained in subtle features of hand movements.
   d. Skull size and shape. Here I think of the clinical impression, which has not been consistently supported by quantitative research, of the "pinhead" sign, and also of the "dome" sign, in which there is something about the skull shape that makes one think of the monster in the first Frankenstein movie. Whether this impression is an artifact of state hospital haircuts or idiosyncratic hairstyles in schizophrenes I do not know, but I think it should be investigated.
   e. I have the impression that there is a kind of asymmetry in the facial wrinkles of older schizophrenes, especially the lines in the forehead. Here again I don't know exactly how to characterize it, but it makes me think of a slightly bungled spider web. I am unaware of any quantitative research on this one either.
   f. I vaguely recall a 1950s study showing that the spatial distribution ("clumping") of color spots in the iris of schizophrenes is abnormal, but have failed to find it, so perhaps it's my own idea. The eye being embryologically a CNS outgrowth, the suggestion is not completely fanciful, especially if the hypokrisic defect is microanatomical (e.g., synaptic knob or dendritic twig distribution) rather than the biochemical conjecture I prefer.
2. *Soft neurology*: Some physicians have come to disapprove of references to soft neurology, and I think I know why. Partly it's a matter of abuse of "soft neurology" in the diagnosis of minimal brain dysfunction by pediatricians and child psychiatrists. But, after all, there *are* soft signs, which in the original usage meant "nonlocalizing" but now has come to connote also (a) marginal intensity, a (±) sign descriptively, (b) not indicative of organic CNS

disease in the conventional sense of "organic," whether localized or diffuse. The abuse of the phrase "soft signs" by some careless diagnosticians does not warrant rejecting the concept, which still has validity and is particularly important when the postulated defect is one of subtle CNS integrative functions. In general, I favor instrumenting and quantifying the soft neurology whenever a sign appears promising in the ordinary type of neurological examination, especially if it is mentioned by master clinicians such as Bleuler, Kraepelin, Schilder, and Lewis. I would include dysdiadochokinesia as described above; postrotatory nystagmus and postrotatory past pointing, both the quantitative deviance and the paradoxical response reported almost a half century ago by the Worcester group; Romberg sign; discoordination and fine tremor as easured by the Whipple apparatus.

3. *Psychophysiological*: One would certainly include what appear to be two of the strongest indicators, ones that persist in remitted schizophrenes and show up in probands, they are the SPEM (Clementz & Sweeney, 1990; Diefendorf & Dodge, 1908; Holzman et al., 1988; Holzman, Levy, & Proctor, 1976; Holzman, Proctor, & Hughes, 1973; Icaono, 1988; Iacono, Tuason, & Johnson, 1981) and the P50 auditory anomaly (Freedman et al., 1987).
4. *Perceptual–cognitive*: One should screen the signal-to-noise ratio; attentional deficit; a difference score between kinesthetic (hefted) and touch alone (nonhefted) weight discrimination (Erwin & Rosenbaum, 1979)—I have here in mind Rado's proprioceptive diathesis; loss of orientation with passive transport; the Witkin stick test; the autokinetic effect; size estimation of body parts in comparison with other inanimate objects (Arnhoff & Damianopoulos, 1964; Cleveland, 1960; Cleveland, Fisher, Reitman, & Rothaus, 1962); impairments of spatial perception, especially three dimensional; the oscillation parameters of the Necker cube; and the stabilized retinal image parameters ("Ditchburn–Riggs phenomenon," Pritchard, Heron, & Hebb, 1960). This last is particularly interesting to me, because it gets directly at the fading in and out (refractory phase) of the elements in cell assemblies, and hence would be very close to the basic hypokrisia aberration. I was struck by an anecdote recounted by Donald Hebb (personal communication, 1962) when I asked him about the plausibility of using the stabilized retinal image phenomenon for such a purpose. He told me that in the original work on this phenomenon, with a dozen or so presumably normal college students, one of the subjects showed only the faintest amount of image disappearance under the experimental conditions. Hebb did not study him intensively, but said that "he appeared normal in other respects." Some months later, he was hospitalized in the Student Health Service with an attack of florid schizophrenia. I recount that $N = 1$ evidence for what it's worth.
5. *Language*: I used to advocate studying the statistics of intraverbal linkages on the theory that, extrapolating from the striking syntactical and semantical aberrations that are almost pathognomonic, one might

conjecture that in the compensated range this oddity would be reflected in lesser degrees that did not involve violations of syntax and semantics but simply low probability occurrences of a word in a certain position as based upon statistical studies of the information theoretical kind. The Cloze method has always seemed to me a good way to study this, although those who have tried it have concluded that it is not as useful for subtle schizoid thought disorder as had been hoped (T. F. Oltmanns, personal communication, May 19, 1987). The older literature on the Kent–Rosanoff community of response measure of one-word associations (see Rosanoff, 1938) perhaps warrants reviving it. But I would suggest improving the metric to make it more sensitive, as follows: We are not interested in the psychodynamic signify-cance of delays or editings associated with emotionally charged content, but rather in deviant frequencies. One would sharpen up the time measure by use of a voice key, distribute the reaction times for each individual subject to detect multimodality, and delete from consideration all responses whose RT occurred in the second or third mode, on the grounds that they were the result of editing. Next, the crude way of numerifying "community" employed by Kent and Rosanoff and subsequent workers can hardly be an optimal metric, which is one reason I am encouraged, because even so they achieved some fairly good separations. It would be more appropriate to assign a transformed value of deviance to responses, based upon the distribution of percentages on a large normal sample, and then pay scoring attention only to the more extreme ones, those that the probability figures put extremely far out. What we want to avoid is moderate amounts of departure from community, easily attributable to nonschizotypal factors (e.g., differences in culture, education, and family background, or differences in values and interests).

6. *Psychometrics*: I would of course advocate using the MMPI, not only the clinical keys but others, such as Wiggin's (1966) psychoticism, Adams and Horn's (1965) "purified *Sc*," Welsh's (1952) "pure schizophrenia," the Harris and Lingoes (1955/1968) content-based subsets of Scale 8, and schizotypal scales (Chapman & Chapman, 1985, 1987; Morey, Waugh, & Blashfield, 1985), a couple of which I have developed but not published. Whether the seven-item set by Golden and Meehl (1979) cross-validates is unclear at present, but there's no harm in trying it, using Bayes's Formula on item patterns (as we did) rather than employing it as a short additive key. Whether the standard Rorschach is sufficiently sensitive in the compensated domain I have doubts, but it probably should be tried. I would lean somewhat more to the Holtzman inkblots (1958). It might be worthwhile to construct a self-report inventory of soft neurology aimed at the awareness of neurological phenomena that show up in eight MMPI Scale *Sc* items. If given encouragement, many schizotypal patients are able to report being clumsy, spilling when pouring, bumping into doors, and having been looked upon as the "klutz" of the family when they were younger (Heinrichs & Buchanan, 1988; Manschreck, Maher, Rucklos, & Vereen, 1982; Meehl, 1964).

HETEROGENEITY AND GENOPHENOCOPIES

Since the death of Eliot Slater the local lore is, "Of the dozen people in the world still interested in a major gene theory of schizophrenia, four are in Minneapolis (Grove, Heston, Lykken, and Meehl)." As one of the holdouts for what most behavior geneticists look upon as a refuted theory (although the theory as refuted does not have the kind of complexity I advocate), I should say something here about genetic heterogeneity and phenocopies. In standard genetic usage, "heterogeneity" does not refer to the existence of polygenic modifiers nor even, if I understand it, to a single locus epistasis. We do not consider that Huntington disease has genetic heterogeneity, despite the fact that different patients do not manifest the same initial predominance of the irritability, paranoid ideation, dementia, and choreic movements. That there must be powerful modifiers involved is shown by high sibling correlations as to age of onset, but that does not prevent us from considering a single mutation at a certain locus as the specific etiology (Meehl, 1972a, 1977). On the other hand, we know that many diseases in nonpsychiatric medicine can arise from mutations at different loci, which leads some theorists to assign a high prior probability (by analogy) to multiple loci for predisposition to schizophrenia. In my hypokrisia conjecture, where deficiency of a neuron membrane stabilizer, "Substance *S*," impairs signal selectivity, a dominant mutation at a locus for either the enzyme controlling synthesis of *S*, or for a different enzyme controlling synthesis of any of its precursors, would produce the hypokrisic defect. Any of such a set might be dominant, but not the same over all schizotaxic pedigrees. Taxometrics applied to indicators arising from the synaptic slippage cannot tell these apart.

Given the MZ concordance of only a little over 50% (lifetime risk), environmental factors are assumed to play an important role. There is an alternative to this, namely, essentially "random" turning on and off of the schizogene, such as sometimes appears to occur in the major affective disorders, when it is impossible to discern any plausible environmental precipitator of a depression or manic episode, even stretching the latent time back for weeks or months. While we cannot exclude this possibility, persistence of soft neurology and psychophysiology (SPEM or P50) among schizophrenes in remission, and among their first-degree relatives who have never fallen ill, renders it unplausible. Setting that possibility aside, I will argue from the armchair that environmental factors *must* be potent determiners of which schizotypes decompensate. One can divide environmental influences into those that were operative during the formative period of the psyche and current stressors on the adult. The decline of the schizophrenogenic mother theory has led to dismissal of her theoretical importance, so I will go out on a limb and forecast that when we have better data, requiring high-validity personality measures, she will be found to play a causal role, and one larger than father. The most malignant parental pattern for male schizotypes is a dominant, controlling, hypohedonic, ambivalent, character armored, compensated schizotypal mother (who transmits the schizogene and provides an aversive reinforcement schedule) and an anxious, insecure, passive, introverted, ineffectual father (who transmits the potentiating polygenes and provides a poor role model and no protective

buffering from mother). This combination is bad genetically and environmentally, and maximizes the probability of adult decompensation. As a colleague put it, harshly but conveying the family flavor, "The worst setup is a schizy battleaxe mother and a Caspar Milquetoast father." But I admit that the present statistics on formal diagnosis do not support such a conjecture. We must be careful to distinguish between the pathogenic influence of having a mother who is a schizophrene and having a mother who is a compensated schizotype. It is quite possible that the latter could be more schizophrenogenic, as Arieti (1955) and Karon and VandenBos (1981) point out.

With some stretching of language, I subsume under the heading of "trauma" both major single traumata (e.g., childhood rape, witnessing violent death of a parent) and accumulative small traumata such as double bind rejections, unfavorable comparisons with preferred siblings, rejection by the peer group in school, effects of grinding poverty, and the like. I believe that cumulative small traumata or one major trauma raise the odds of adult decompensation. More broadly, I conjecture that *almost any insult organic or social, suffices to raise the odds*. It is no threat to a theory such as mine to find that difficult birth or maternal exposure to influenza alters the probability significantly. Further, I conjecture that *total adverse load* (genetic and environmental) is what counts, except for the schizogene. The other factors are numerous enough so that relying on Wilks' theorem (Dawes & Corrigan, 1974; Wainer, 1976; Wilks, 1938), we can treat them as quasi-fungible.

With regard to the precipitating stressors of adult life, I am inclined to lay great weight upon the "luck factor." I agree with Sir Karl Popper that sheer luck, happenings that befall us that are not attributable even to some unconscious selection of our friends or occupations and which have nothing to do with the repetition compulsion, play a crucial role in human happiness, health, and achievement. Social scientists underestimate the sheer luck factor because it does not lend itself very readily to inclusion in our predictive and explanatory equations (Meehl, 1978, p. 811 and references cited thereat). The difference between a schizotype who wins the Pulitizer Prize for poetry, and his MZ twin who ends up as a chronic deteriorated schizophrene in the state hospital may not always be understandable by some kind of systematic factor of the sort that psychologists and sociologists are accustomed to putting into their equations, such as a strong parental preference for one twin, or one twin having been dropped on its head as a baby. The difference may be simply the outcome of a random walk (Meehl, 1972c). It is not difficult to think of plausible examples such as the following:

> 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 relation being idiographic content features that alter the subsequent psychological 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 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…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, 1972b, pp. 404–405)

Suppose someone lacking the schizogene has a heavy loading of polygenes for submissiveness, hypohedonia, anxiety and introversion, is traumatized as a child, and has a run of bad luck as an adult. I think such a person has a good chance to present a clinical syndrome sufficiently close to true schizophrenia (as defined etiologically) to be so diagnosed. Since polygenes cooperate here with developmental trauma and adult stress, I call such a case a *genophenocopy*, and the putative syndrome I label SHAITU (*s*ubmissive, *h*yphohedonic, *a*nxietous, *i*ntroverted, *t*raumatized, *u*nlucky). Whether the accessory symptoms of delusions and hallucinations could present the same quality and duration in the SHAITU syndrome as in schizotaxic schizophrenia I have no confident prediction, but I am inclined to doubt it. More important, the lack of consensual validation that might result in secondary cognitive slippage in the SHAITU syndrome should not suffice to produce the oddities of schizophrenic speech, and I can think of no plausible basis on which SHAITU etiology could produce the soft neurology and psychophysiology of the schizotaxic brain. I therefore expect the latter to be powerful indicators for separating the SHAITU syndrome, or other genophenocopies not here considered, from schizotaxic schizophrenia.

TAXOMETRIC ANALYSIS OF DATA

That there is a potent hereditary influence in the predisposition to schizophrenia can no longer be doubted by a rational informed mind. There is little point in collecting a large batch of data, whether neurological, psychophysiological, psychometric, or interview and life history, that merely bears on this settled question, without tending appreciably to corroborate or refute a more specific genetic model. Furthermore, one would like to test both the *mode of genetic transmission* and the *substantive conjectures as to just what it is that is inherited*. So what we want to test with respect to my theory is that there are persons (initially identified by being biologically related to diagnosed schizophrenes) who carry a dominant gene whose immediate, and only strictly "inherited," endophenotypic consequence is a diffuse integrative defect in the CNS, which in turn is linked, although perhaps only stochastically, to exophenotypic indicators of a psychophysiological and neurological character. This much is asserted by the theory, so that if this conjunction of conjectures is falsified by the facts, then the theory is falsified. It is left open, for good learning theoretical and clinical reasons, to what extent the primary aberration in CNS function, manifested with high indicator-expressivity in the less molar behavior domains, will also be manifested in the loose cluster of acquired, socially learned molar-level dispositions that make up the schizotypal syndrome as a *psychological* pattern.

One must be clear about the distinction between two methodological difficulties in appraising such a causal and statistical structure, which are sometimes confused. There is first the question of how strong a statistical linkage is expected to obtain between the schizotaxic (basic, primary) defect and a psychological indicator; and, secondly, what are the reliabilities and validities of available methods for measuring or detecting the psychological indicator. Furthermore, one should keep in mind that there can be a tradeoff between reliability and validity. Reliability (more precisely, its square root) sets an upper bound on validity, but the intrinsic construct validity of some measures may be much lower than that of others, so that ordering indicators as to their reliability and then ordering them as to their *net attenuated construct validity* will, in general, not result in a perfectly correlated ranking. The physician takes blood pressure as a part of a routine physical examination rather than regularly apply a micrometer to measure wrist width, despite the fact that the former has disturbingly poor reliability in the .60s, whereas the latter has reliability better than .98. Simply put, it is (in clinical work as well as in theoretical inference) better to measure an important thing somewhat inaccurately than an unimportant one with high precision. Some of the disagreements concerning DSM-III stem from differences in the importance clinicians and theorists attach to the preceding point (Faust & Miner, 1986; Meehl, 1986a, 1989b).

There is a methodological tension between a sophisticated recognition of the unavoidability of open concepts in psychopathology, and a broadly Popperian view of theory corroboration. If you accept open concepts (by which I do not mean joyfully wallowing in fuzziness) and yet desire strong potential falsifiers as prescribed by Popper and his followers, you have to permit a little compromise and tradeoff between these two metaprinciples. It is simplistic to require an "operational definition" of something like schizotypy or schizotaxia, if by "operational" one means a literal logical conjunction of attributes, each of which is judgeable with high reliability. Neither the state of current theory nor the accuracy of our instruments allows this as a realistic requirement. But if we relax the requirement, accepting a loose cluster of fallibly measured indicators, we have fuzzed up the sharp distinction between a falsifying and a corroborating empirical finding. This methodological problem is nicely illustrated by the subject matter of genetics. The ideal situation is one in which the phenotypic syndrome is clearly defined, both in the sense that we can write down a conjunction or disjunction of indicators which are, as so conjoined and disjoined, necessary and sufficient conditions for the presence of the gene, and then can examine in Mendelian fashion a set of family pedigrees. Almost as good is availability of a marker trait which, while not part of the pathological entity of interest, turns out to be almost perfectly correlated with the genotype. The essential epistemic step in this ideal state of affairs is our ability, by one means or another, to identify *individuals* who carry the gene; and to be able to do that is an appropriate goal for the genetic investigator who conjectures a major locus. If we can find such markers, or define such tight syndromes of the entity itself (as we do in disorders like Huntington Disease or PKU), we should of course do so. In discussing the rather different taxometric approach that follows, I do not wish to be

misunderstood as in any way criticizing the usual approach of the geneticist, which is surely the right one when it is possible.

However, when we deal with a loose syndrome that statistically defines an open concept, and when the substantive theory itself leads us to expect that the aspects of the syndrome that lead to our clinical interest in the first place are remote in the causal chain originating in the DNA, subject to complicated and highly variable social learning schedules and "accidents" of the environment, then we have the complicated problem of deciding what weight to give to different domains and levels of indicators. The usual notion of straightforward "validation" of a strong indicator does not work in such situations, for the simple reason that the theory itself specifies that there should be stronger indicators than those that lead the psychoclinician to notice such a syndrome as schizophrenia in the first place. Even setting aside the problem of genetic heterogeneity, this is obvious when one reflects upon the great variation among schizophrenes in what kinds of manifest psychopathology predominate in the clinical picture. Hence the difficulty of perceiving a unity here, which awaited the clinical genius of Kraepelin, and the further extension provided by Bleuler. Hippocrates could recognize mania and hypochondria, but it took a Kraepelin to fuse the subtypes into the concept of *dementia praecox*. When one goes beyond this to identify the compensated or pseudoneurotic schizotype as a member of the class (that does *not* mean "broadening the concept 'schizophrenia' unduly"—I hope I have made that conceptually clear), the problem of specifying attributes, or of "validating" a nonbehavioral indicator such as a soft neurological, psychophysiological, or anatomical sign, is one of great conceptual and mathematical difficulty.

While we may hope to find, sooner or later, a two-way pathognomic sign of the schizogene if such exists (in the language of the epidemiologist, a sign having perfect sensitivity and specificity, functioning perfectly as both inclusion and exclusion test, with zero false positives and zero false negatives), we would like to test competing genetic models before that perfect sign is found. Furthermore, if such a perfect sign exists, we still would have the methodological problem of how we *find* such a perfect sign, that is, how do we know when we have it? At first glance this seems like an inherently insoluble problem, because one is asking for a method of proving that something is infallible when the criterion is fallible. Psychologists educated in conventional concepts of predictive validity and who have never read Cronbach and Meehl (1955) sometimes reject the very idea of doing such a thing. But they shouldn't be troubled, since the history of all sciences is one of successful "bootstrapping," in which one starts with common-sensical ways of slicing up the pie, with crude measures and partly mistaken clusterings of attributes, and by a complicated process of refinement ends up with indicators so powerful that they are sometimes taken as theoretically definitive. All sciences including physics present numerous examples of such successful bootstrapping, although I do not know that any statistician or logician has shown that the methodological machinery, experimental or statistical, is highly similar from one subject matter to another. (I conjecture that there must be a deep-lying statistical similarity in all varieties of successful bootstrapping, but that is not a position I need to defend here.)

In psychology I suppose the clearest example is the intelligence test. One begins with the everyday observation that some people appear to be a good deal "brighter" than others. One of the important places where this difference in brightness manifests itself fairly clearly, and in a socially significant way, is in school learning. When asked by the French school system to devise some short but accurate way of determining which school children were doing poorly because they were not very bright (as contrasted, say, with those who were poorly motivated, or needed glasses and sat too far from the blackboard, or whatever), Binet and Simon tried a number of different indicators, including ones that today seem to us a bit outlandish, such as two-point threshold, graphology, and palmistry. These avenues were not pursued because none of them seemed appreciably correlated with teachers' judgements as crystallized in age/grade location, or in the form of ratings. The subsequent development of intelligence tests and the clarification of the concept of *g* is too well known to be detailed here; suffice it to say that the development of factor analysis made it possible to do a more formal kind of bootstrapping than had been possible earlier. If the history had been different, not involving the appraisal of school children for a practical purpose, it could have gone like this: Ordinary observation shows that some grownups are smarter than others. However, it seems that smarts do not always manifest themselves in quite the same way in different people. Having Thurstone's mathematics available as an analytic tool, let us prepare a longish set of tasks of a sort that would usually be described as broadly "cognitive" in nature, and then let the statistics do the rest. Result: An omnibus intelligence test.

Another example would be personality measures such as MMPI Scale *Pd* (= psychopathic deviate). An item analysis of a hodgepodge of 550 items ranging over many different subject matters, including items not having any apparent reference to psychopathology, was conducted by comparing normal people in the Minnesota population with subjects (referred to a psychiatric service by the court) who had engaged in antisocial acts and who fit the old criteria of "constitutional psychopathic inferior" or "psychopathic personality, asocial amoral type." Here the manifest traits that commence the bootstrapping are social impact traits, such as adjudicated delinquency, marked malperformance in school, or inability to adjust in a foster home. Then the psychiatrist made his assessment from interview, life history, and ward behavior, that the subject was characterized by poor judgement, inability to learn by experience, lack of normal conscience, and egocentricity. One could say, in the terminology used for schizophrenia, that these cases were identified by "positive symptoms" of a broadly acting out, impulsive sort. Having constructed the *Pd* scale by item analysis against this criterion group, one can then study persons who have not been in trouble with the law or the school system; and one finds, in a variety of settings, that they differ from "people in general" in a number of interesting ways, some of which would be classifiable as "negative symptoms" by analogy with schizophrenia, notably, lack of normal social fear. A certain ability to charm, which facilitates their hoodwinking others (although not necessarily as law violators) becomes part of the syndrome. Today, sophisticated MMPI users speak of the *Pd* syndrome in terms of the Hathaway code as "49s." When we use that code language we do not imply that the odds are

good that the person has been convicted of a criminal defense, adjudicated a juvenile delinquent, or expelled from school. Rather we are designating a *personality type*. MMPI users who have had the opportunity to study 49′ subjects in a nominally "normal" population, such as college students, and who are familiar with Cleckley's (1976) and Lykken's (1957, 1984) classic works on the psychopath, regularly conceive of the entity in terms of the *psychological* traits of abnormally low social fear, a pseudoextroversion (which probably stems from that negative trait), and hence a lack of true warmth or solid affectional bonding, a tendency to be easily bored, resistance to rules and demands, superficial charm, and ability to "con" other people—none of which traits is equivalent to, or necessarily productive of, adjudicated crime, delinquency, or expulsion from school. Similar examples could be adduced for almost any test of personality.

Any statistical bootstrapping procedure is likely to result in "psychometric drift," because an adequate statistical bootstrapping method will, when it functions successfully, alter the relative weights provided by our initial commonsensical or clinical experience indicator set (Meehl & Golden, 1982). This psychometric bootstrapping results, as in the psychopath example just given, in a certain amount of *conceptual drift*. How one conceives of the trait, whether the phenotype or the latent, inferred source trait, will properly be influenced by new quantitative information as to the factor loadings or taxonic validity of the several indicators. Such conceptual drift, consequent upon psychometric drift, is normally desirable, since it constitutes a theoretical advance, both in delineating the manifest syndrome and correcting theoretical inferences as to its underlying causation (pathology and etiology). However, one must be constantly alert to the danger of an undesirable psychometric–conceptual drift which may happen under adverse circumstances (bad algorithm, poor thinking, or just plain "bad luck" in the research program or the sample). A research program relying heavily on bootstrapping, whether factorial or taxometric, must make provision for keeping in touch with the clinical entity that motivates the investigation. No simple rule can be given for how you do this. Safety procedures will vary over behavior domains, and will differ as between studying genetics or social learning, as to whether one conjectures the source trait is a taxon or a dimension (factor), and as to reliability and construct validity of the measures.

The essential feature in taxometric bootstrapping of a loose syndrome for the purpose of appraising competing genetic models is that one does not begin by requiring the availability of a high-validity sign, let alone a pathognomic one. Absent such a sign (which, I repeat, we will be happy to utilize if we should stumble across one in the course of our taxometric bootstrapping), how can one combine the unavoidable fact of syndrome looseness, and, hence, indicators that are fallible singly and collectively, to generate a strong Popperian test, one capable of refuting the theoretical conjecture? If we cannot realistically hope for a clear-cut set of family pedigrees, given the openness of the concept (looking at it another way, the very low "clinical penetrance" required on the dominant gene conjecture), a strong falsifier must be achieved by predicting a subset of observable numerical values from latent numerical values that are in turn inferred, via the taxometric bootstraps process, from another subset of observable values.

Briefly put, *one can falsify a major locus theory by predicting proportions of signs and sign patterns among relatives of probands, although to identify which individual relatives are schizotypic may be impossible to do with high confidence*. That is the essence of my taxometric analysis proposal.

The first question one puts to a set of taxometric search procedures (ideally, computer algorithms) is, "Given a set of subjects having a specified biological relationship to a schizophrene, is the latent statistical structure underlying the relations among their fallible indicators taxonic or not?" What this amounts to in practice is different ways of ascertaining whether the single and joint distributions of the fallible indicators can be considered composites of overlapping unimodal distributions associated with two latent classes, the schizotaxon and the complement class (= everybody else, sick or well). The alternative intepretation is that the various patterns of pairwise correlations, and higher order patternings or configurations, among the relatives should be viewed as being generated by factor loadings on underlying dimensions, in which there is one latent distribution rather than two overlapping ones. We do not require anything stronger about the latent distributions of the taxon and complement than that each should be unimodal since we know from research in psychopathology (and especially schizophrenia) that Gaussian form or approximate equality of variances are not safe assumptions, and in fact are almost certain to be grossly off.

A polygenic theory of schizotaxia might be adjustable given suitable assumptions about assortative mating, thresholds, etc., to fit the statistics generated by a latently taxonic situation, although I am inclined to doubt this, given sufficiently large samples. But even if that were the case, it would be an ad hoc adjustment, and hence provide little or no corroboration for the polygenic theory, since if it is thus adjustable it cannot be falsified by the taxometric results. If you could so rig the statistics, there is no *affirmative reason* for predicting a latent taxonic structure on the polygenic theory that I have ever heard proposed by its advocates. For example, suppose one taxometrically analyzes parent pairs of schizophrenic probands employing as fallible indicators a quantified dysdiadochokinesia, the SPEM test, and the P50 anomaly quantified. If a polygenic theory were true, a showing of clear taxonicity would require us to suppose that people tend to marry with a high negative assortative mating for these three neurological and psychophysiological traits, which is hard to imagine.

Suppose the dominant gene conjecture were correct. Assuming true schizophrenia in all of the probands (discussed below), each proband must have received the schizogene from one of the two parents. Given our available statistics on clinical penetrance and gene frequency (presupposing the dominant model, only rarely a dose from both parents), we don't expect more than 2% such double-schizotype marriages among the parents. So one expects the base rate of the schizotaxon among parent pairs to be not less than $P = .5$, (in a "pure" sample, with SHAITU cases removed), and perhaps a couple of points in the second decimal place higher than that allowing for doublets. The model requires not merely (a) that we have a taxon, and (b) that it have a base rate at or slightly above one half, but (c) the schizotypes must not be randomly distributed over parents but must be allotted (almost always) as schizotype–normal matings. From

this it follows that if p_s = valid positive rate and p_n = false positive rate for an optimal cut on an indicator variable, and we set up a fourfold table for each variable in which the axes are not values of the variable but the dichotomy father/mother (so the table tallies represent parental concordances and discordances for the indicator), then the covariance (= numerator of the phi-coefficient) for such a table should be approximately $-\frac{1}{4}(p_s - p_n)^2$. Alternatively, in terms of the latent means, we expect for fallible indicator x that the numerical covariance between the parents should be $\text{cov}(x_m x_f) = -\frac{1}{4}(\bar{x}_f - \bar{x}_m)^2$ (Golden & Meehl, 1978). In order to deal with this kind of quantitative finding, a polygenic theorist must ad hoc the data by conjecturing that there is a pronounced negative assortative mating for soft neurology (which most of the subjects will never have had elicited until they were in our experiment), that the indicator has a quasi-step function with respect to the latent polygenic system determining it, and that the step function occurs at the median value .5 predicted on the dominant theory. Whether this is mathematically possible assuming an underlying polygenic system I am not sure, but even if it is, the combination of the statistical adjustment ad hockery with the implausibility of the psychological and social conjectures, and the conjecture about the step function location being the same for each quantitative indicator, would exclude it from serious consideration.

On the other side, no matter how great the overlap of the indicator distributions between the taxon and complement class may be (assuming that the initial validity screening and the taxometric bootstrapping have corroborated that there is good validity for each indicator), a dominant gene model requires that the statistical structure be taxonic, and it further requires that the taxon rate among parents be close to .5. There is no plausible way for the major locus theorist to get out of this consequence. So if the statistical structure is not taxonic, or if the taxon rate is markedly different from .5, or if there if is no negative covariance in the parent-pair fourfold tables, the dominant gene theory would stand refuted.

Since this is a paper on schizophrenia theory and not on taxometrics, it is appropriate only to summarize briefly the procedures applicable in my approach. About some of these we have a good deal of information from both real data and Monte Carlo studies, and about others only the basic mathematical derivation with little or no empirical validation so far. Suppose we have a set of four quantitative indicators x, y, z, v which have been shown in previous research to discriminate (and not discriminate!) between various diagnosed patient and control groups. We would hope to find indicators that have at least a 75% hit rate in an equal group situation, and I would hold out for that in screening. This is not unduly optimistic, although I gather some think it is. Unless there are artifacts of social class or other nuisance variables, we can usually conjecture that a hit rate achieved with concurrent validation as the mode (e.g., criterion schizophrenes diagnosed by the usual methods, not employing SADS but relying on DSM-III criteria, versus "normal controls") will be an underestimate of the true taxonic validity. This is especially true on the dominant gene hypothesis, where we must suppose that 10% "false positives" in an unselected general population sample will be *pseudo-false positives*, schizotypes who either have not been subjected to psychiatric scrutiny or who have remained compensated to the point of not being diagnosable. (This may

seem arguing in a circle for the theory, but it is not, since in evaluating any theory one must give the theory its head, build into the testing procedure those auxiliaries that the theory itself requires, including adjusted estimations of the reliability and validity of instruments.) Suppose we locate a cutting score on indicator *x* (e.g., SPEM) that identifies 90% of diagnosed schizophrenes, operating on the view that even with careful application of DSM-III criteria (perhaps even using SADS?) there will be at least a 10% diagnostic error among the patients. If that cutting score yielded 15% false positives in the so-called normal controls, since the dominant theory implies that 10% of "normal" controls are in fact schizotypes, what we have then is a net "*true* false positive rate" of only $5/90 = 5.6\%$ for the indicator when cut at that place. If thinking along these lines strikes superoperationists as being too dangerous, all I can say is that they will be unable to investigate competing genetic models for schizophrenia unless and until a pathognomonic marker trait is found.

If the four indicators *x*, *y*, *z*, *v* have passed the multiple screenings listed above for good candidates, we then proceed to test the relatives of schizophrenic probands who have been diagnosed with extreme care employing SADS administered by two skilled examiners, perhaps some strong life history and ward behavior diagnostic criteria, plus a clearly schizophrenic MMPI profile, so we are superconfident in the descriptive diagnosis. This does not mean that we are superconfident that we have a single diagnostic taxon, because that begs the question of genetic heterogeneity. All we are saying is that *as schizophrenia is currently clinically and psychometrically recognized*, if anybody has it, assuming there is an 'it' to have, these probands do. That is an auxiliary conjecture as part of the research program, and susceptible of amendment as the data are analyzed. We have to conjecture this, even knowing it is an idealization, to get our foot in the door. But we are prepared to abandon the conjecture if we can falsify it by the taxometric bootstrapping results.

It may be objected that this carries with it a methodological danger of failing to include some schizophrenes who don't meet the strict criteria of DSM-III. I have in mind particularly the fact that DSM-III emphasizes the positive rather than the negative symptoms, whereas Bleuler would put at least equal weight (if not more) on the negative ones. I will return to that objection later, but for now, suffice it to say that the taxometric approach is not hampered by the epidemiological problem of incomplete ascertainment that usually dogs the steps of the geneticist. One might put it this way: *If* we find a clear taxon that is inherited as a dominant of high expressivity for certain indicators, *then* we can return to samples of patients diagnosed more loosely to see what they look like. The main point in this phase is that we do not want any carelessly diagnosed psychopaths, manic–depressives, anxiety states, or patients with organic brain damage contaminating the sample. For taxometric purposes we do not worry about the true schizophrenes left out of our sample, so long as we can thereby prevent an appreciable number of nonschizophrenes from sneaking in.

I do not consider the 75% hit rate unduly optimistic, because even psychometrics with pre-SADS and pre-DSM-III unreliable criteria can do better than that. The 75th percentile of the normal population and the 25th percentile of a schizophrenic population would be one *PE* from the mean of

both groups (= 2/3 σ), corresponding to a mean separation of 1.33 σ. For that to be true, the mean *T* score on the *Sc* scale of the MMPI, in the careful validation studies, would be only 63, whereas good studies place that upper mean in the region 70–80. The only MMPI scale that does as badly as *T* = 63 is Scale 6 (= *Pa*), a short scale which (despite the subtle items) some intact paranoids cleverly manage to outwit. When old (1942) MMPI *Sc* does as well as it does against less reliable diagnoses, it should be possible to find at least four psychometric, neurological, or psychophysiological indicators that do as well as the weak *Pa* scale. Present evidence would warrant us in saying that at least two of them, SPEM and P50, achieve better than 75% hits.

In the taxometric methods (Golden, 1982; Golden, Campbell, & Perry, 1987; Golden, Gollub, & Watt, 1983; Golden & Meehl, 1974, 1978, 1979, 1980; Golden, Tyan, & Meehl, 1974; Golden, Vaughan, Kurtzberg, & McCarton, 1988; Meehl, 1968, 1973b, 1979; Meehl & Golden, 1982; Meehl, Lykken, Burdick, & Schoener, 1969) we deal with single indicators, pairs, triads, and tetrads. *With a single indicator* we do make the idealizing assumption that the latent distribution is approximately Gaussian. I do not know how robust my method is under departures from that idealization, although in the one empirical trial in which we used MMPI Scale 5 to distinguish the biological sexes (Meehl, Lykken, Burdick, & Schoener, 1969), that scale being quite skew to the right, the Gaussian idealization sufficed to give a satisfactorily accurate answer. One conjectures an arbitrary base rate *P*, which determines the complement rate *Q*; then one assigns an arbitrary taxon mean M_t, which, taken with the conjectured base rate and complement rate determines the complement mean M_c; finally one assigns an arbitrary standard deviation SD_t which, given the previous assignments and the total sum of squares, determines the complement SD_c. All of this is based upon algebraic identities and is distribution free. But in the next step we idealize the situation as two overlapping Gaussian distributions, and obtaining the ordinates (or areas corresponding to small abscissa intervals) of the latent distributions, we predict the manifest frequency in each class interval. We then compute a chi-square between the predicted and observed values. We repeat this process for various assigned *P*, M_t, SD_t combinations, and we examine the graphs of the chi-squares obtained for the branching trees of arbitrarily assigned base rates, means, and sigmas. For a given base rate, one gets a set of curves of chi-square: each curve corresponds to an arbitrary mean (given that base rate); each point on the curve corresponds to a particular sigma for that combination of base rate and mean. We find the minimum of the minima of the minima. If (as found in our sex study) that is the only chi-square that fails to depart significantly from the theory, we infer the six latent parameters to be those corresponding to that minimum. Obviously this will not yield anything like the right answer for the manifest frequency distribution if there is no latent taxon/complement situation but only *one* underlying frequency distribution. Since we have four indicators, they yield four independent taxon detectors, and they give us four independent estimates of the taxon base rate *P*, despite the fact that the four indicators will differ greatly in their respective taxonic validities, in their sigmas, etc. Two valid taxonic separators may differ in overlap and in the ratio of the two standard deviations, but they should give the same estimate

of *P*. Agreement of these four independent *P* estimates within tolerances (I am not here interested in doing a significance test, but rather Monte Carlo runs on the relation of tolerances to accuracy) constitutes a simple *consistency test*. The role of consistency tests is of great importance in my taxometric approach, since good agreement of independent estimates of a numerical value is far more important than optimizing statistics (such as *MLE*s), their mathematical precision being illusory anyway, an idealization of the formalism.

If one considers *two indicator variables* (*x*, *y*) at a time, the method used relies on the idea that the mean of a mixed group increases monotonically with the ratio of the base rates *P*, *Q*. Hence the difference between the means of two groups to which we apportion taxon and complement members will tend to rise as we improve the "purity" of the two groups by optimizing a sliding cut on another indicator. Calling indicator *x* the "input variable" and indicator *y* the "output variable," we define the quantity $\overline{d}_y(x) = \overline{y}_a(x) - \overline{y}_b(x)$, the mean of *y* for cases lying above the *x* cut minus the mean of *y* for cases lying below the *x* cut. This "mean above minus below a cut" index christens the method MAMBAC [see Meehl & Yonce, 1994]. In a nontaxonic latent situation, where the observed correlation between the two indicators is attributable to their both having factor loadings on an underlying quantitative dimension (factor), the characteristic shape of the graph of this index is concave, the concavity depending upon the size of the factor loadings. Whereas if the latent situation is taxonic, and the correlation is being generated by the latent taxonic separation on both *x* and *y*, then the characteristic shape is convex. We are developing pattern indexes for mechanically distinguishing between the two, but suffice it to say here that a statistically naive person, after being shown a couple of samples, has no trouble sorting the resulting graphs into two piles with better than 95% accuracy when the base rate is one half (the case we are interested in when testing the dominant gene theory). Several analytical developments of how to estimate the base rate and the latent means once one has concluded for taxonic structure are still under investigation, but for empirical examples see Golden and Meehl (1979) and Meehl and Golden (1982). With four indicators we have six indicator pairs, and since for any pair the MAMBAC program can be run in either direction, this gives us twelve graphs for inferring taxonicity versus not, and twelve only slightly redundant ways of estimating the taxon rate *P*.

Considering *three indicator variables* (*x*, *y*, *z*), we have one procedure for taxon detection and parameter estimation, and a second one which has so far been studied only with respect to estimating the base rate but not to detecting taxonicity of structure. In the method called MAXCOV–HITMAX (Meehl, 1973b) one relies on the fact that the covariance of a mixed group is a function of the taxonic validity of the two indicators and the taxon mixture. Let the indicators (*y*, *z*) be initially selected for their negligible correlation within a group of schizotypes and within a group of controls. Then the main source of an observed *yz* covariance lies in the taxonic validities of *y* and *z* and the proportions *P*, *Q* of a group of cases. Since $P = Q$ in the subset of cases lying in the *x* interval corresponding to the HITMAX cut on *x*, when we plot the graph of the observed *yz* covariance over *x* intervals it starts from zero, rises to a maximum (corresponding to the latent HITMAX cut on *x*), and then declines to zero again. This convexity of

the *yz* covariance graph as a function of *x* is a strong taxon detector, because the nontaxonic (factorial) case gives us a flat curve. Here the presence of nuisance covariance between *y* and *z* constitutes an advantage, because it reduces the sampling dispersion of points around that flat graph and makes its nontaxonicity easier to detect on inspection. Further computational steps are explained elsewhere (Meehl, 1973b; Meehl & Golden, 1982; [Meehl & Yonce, 1996]), but, briefly, one goes from solving for a certain constant that corresponds to the crude taxonic separations knowing that $P = Q =$ one half in the HITMAX interval, to solving a quadratic in the proportion of schizotypes in an *x* interval relying on that constant, then solving for the number of schizotypes per *x* interval, and summing these gives the total number of schizotypes in the sample, from which we get the base rate *P*. In this procedure one in effect has the computer draw the latent frequency distribution on each variable used as input, and hence gets an estimate of the latent means which can be cross-checked with the validity mixture term arrived at in the first step of MAXCOV. We have developed a half dozen consistency tests to be applied when using this method. With four indicators one has four ways to choose which will be used as input, and then three ways to choose the output pair, for a total of twelve graphs detecting the taxonicity of structure and twelve fairly nonredundant estimates of the base rate and the latent means. The method is quite robust under small departures from the idealization of zero nuisance covariance within latent classes (Meehl & Golden, 1982), and a generalization free of that constraint has been derived but not yet studied empirically (Meehl, 1995).

The other way of using three variables has as yet been incompletely studied although so far the results are most encouraging. In this method we assign an arbitrary base rate *P*, write an equation for the proportion of pairwise positive signs determined by arbitrary cuts in terms of the latent valid and false positive rates, and solve for the latter. The three equations in pairs of latent variables p_{sx}, p_{sy}, p_{sz} (the false positive rates being simple functions of these, given the base rate assignment) are hyperbolas, so in the three space they define hyperbolic cylinders. In a three space, the *xy* cylinder is cut by the *xz* cylinder, leaving trace lines; and then the *yz* cylinder intersects these trace lines to determine two points, chosen physically. We now use the latent values to reconstitute the eight observed frequencies of three-indicator sign-patterns $(x^+ y^+ z^+)$, $(x^+ y^+ z^-)$, … $(x^- y^- z^-)$. Summing the discrepancies between predicted and observed frequencies, we have a Discrepancy Index (DI) for the arbitrary taxon rate *P*. Repeating this process for other assignments of *P*, we plot the DI graph and its minimum is our estimate of *P*. The hyperbolic cylinder equations motivate acronym HYPCYL ("hipp-sill") for this method.

Two additional nonredundant methods (for a total of seven) have been analytically derived, but the empirical runs on them are so scanty as of this writing that I shall refrain from expounding them here. But the big point is, *the more inferential paths and consistency tests*, *the better*.

Suppose most schizophrenes carry a dominant schizogene. (If we knew that, we would pack this specific etiology into the revised *explicit* definition of true schizophrenia, and it would cease to be a pure open concept.) But suppose a sizable minority of patients diagnosed schizophrenia suffer from the SHAITU syndrome which might with some frequency be diagnosed as

schizophrenia. Within the whole class of cases diagnosed schizophrenia on adequate observation by careful clinicians employing the currently accepted criteria, my prediction is that 85–90% are schizotaxic and 10–15% are genophenocopies. What will these pseudoschizophrenic cases do to a taxometric analysis? Confining our attention to parent-pairs (the strongest test of a dominant model, and one not harmed by matters of ascertainment, cooperation, etc.), how will the taxometric inferences be influenced by the presence of these nonschizotaxic cases? Will they mislead the taxometric investigator? I believe they will not.

The SHAITU probands belong not to the taxon but to the complement class. So will most, not all, of their parents. (Why not all? Because of the .10 schizotaxic general population base rate, and there being no reason to assume that SHAITU polygenes and environmental factors are *negatively* correlated with the schizogene. A SHAITU case and a true schizophrene can occur in the same family, and not with negligible frequency. An interesting combination, likely to puzzle genetic researchers, would be sibships consisting of a sick SHAITU diagnosed as schizophrenia and a compensated schizotype. Imagine what *that* pattern does to our family statistics on psychometrics and soft neurology!) Hence, instead of the dominant model's parental taxon rate $.50 \leq P \leq .55$, we obtain a lower one, conceivably much lower if a sizable fraction of "schizophrenic" probands are, in fact, SHAITU syndromes.

However, the latent structure is still taxonic, and easily detectable as such. It is the taxon rate P that will be in error, and the dominant model requirement that each parent-pair include a taxon member (and around 95% of pairs *only* one taxon member) will not be satisfied by the data. We will have corroborated a one- or two-major-locus model, but we lack a strong numerical test between them. For example, a two-locus model with dominant epistasis could yield $P \simeq .25$; alternatively, a sizable minority of proband SHAITU could easily push the parental taxon rate down to around .25. *Conclusion*: Convergence of taxometrics could corroborate a major locus model and discorroborate a polygenic one, despite the SHAITU contamination of purported schizophrenic probands; but it will neither corroborate or refute a pure dominant conjecture without further "internal" analysis.

Note that the reasoning neither requires nor forbids that the fallible indicators of parental schizotaxia be measures of the polygenic components S, H, A, or I. They may measure all, some, or none. They need not, using DSM-III of course *will not*, be the same indicators used to identify probands. Consider an extreme (and interesting) case: Probands having been identified by DSM-III criteria, taxometrics are run on soft neurology and psychophysiology indicators *only* (i.e., no indicators aimed at, *or appreciably influenced by*, any of the polygenic factors S, H, A, I). If the neurology–physiology indicators are high valid for schizotaxia, they will yield a taxon among the parents, with taxon rate $P < .50$; and from this taxon rate the neurology–physiology taxon rate among the probands is computable. The taxon rate among parents also permits derivation of parent-pair pattern frequencies, since among the parent-pairs of SHAITU probands, double "normals" should strongly preponderate ($P > .95$) instead of the (+ –) pattern usually found for parents of truly schizophrenic probands.

Since each of the taxometric methods independently draws the latent distributions among parents, the dichotomous validity of a sign can be inferred by

studying changes in its (+) frequency as we move from one interval of an indicator to another. Numerous other internal checks have been developed and are being studied Monte Carlo as of this writing.

The second impure case is more troublesome, as it involves a phenocopy taxon in the complement class. Suppose the "trauma" factor T is a specific kind of life-history event which has a "present-or-absent" character, as in Freud's early theory of the etiological difference between hysteria and the obsessional neurosis. Here it might be, say, a terrible shaming event at school, or early head injury, or gross sexual abuse. It is improbable, theoretically, that such trauma-induced phenol-copies should precisely mimic the syndrome of schizophrenia in its cognitive, social, and neurological aspects. In simpler, nonbehavioral genetics, strict phenocopies are very hard to come by (Vogel & Motulsky, 1986). One reason for employing multiple indicators, minimally correlated and phenomenologically diverse, is to capitalize on this rarity. (Do we seriously envisage the possibility that a slow virus or head trauma can mimic the peculiar schizotypal cognitive slippage, or that a narcissistic wound suffered in Sunday school can induce aberrant eyetracking and (±) dysdiadochokinesia?) The other two reasons for an extensive, qualitatively diverse indicator list are: (a) more signs, better confidence; and (b) qualitative diversity, less nuisance correlation, hence a cleaner taxometric model. However, many—possibly all—of the polygenic potentiators may be involved, given the usual basis (psychopathology, emphasizing the "social") on which diagnoses are made. So if indicators are in the list partly because they tap the relevant polygenic dispositions, the phenocopies could be fairly similar. It is inconceivable that the quantitative *distributions* on all of them should be the same for traumatic phenocopies and major-locus schizotaxics, but small differences in the two polygenic profiles will not help us much at this stage.

It is crucial to keep in mind that the parents of "T-phenocopies" will not be schizotypal except for the general population base rate of .10, so their latent distribution will be only weakly taxonic, hardly detectable unless N is large. Nor will they show the "at least one parent per pair" pattern required by the dominant model for parents of true schizophrenic probands. The phenocopy proband is more likely to have received his adverse dose of introverted, submissive, anxious, etc., polygenes relatively equally from both sides than heavily piled up from either, especially if (as is probable) these schizoid-imitating components reduce the probability of parenthood. We would like to pick out the phenocopies, which we could do with indicators that are not positive in the phenocopy (e.g., SPEM, P50, soft neurology, nailfold capillary anomaly). But while this is the ideal situation, lacking that but given a set of strong and independent indicators (although nonschizospecific), it is still possible to segregate the complement class, proceeding as follows:

Consider a situation bad with respect to proband diagnoses but good as regards indicator taxon validity. Say 25% of the "schizophrenic" probands are in reality genophenocopies (SHAITU). (Here I assume 75% of SADS-diagnosed probands to be "true" schizophrenes in my etiological sense, which I realize is a good deal higher than conjectured by most heterogeneity advocates.) Suppose we have four indicators, independent within latent classes, all with 2-σ separations, yielding hit rates of 85% symmetrically

when taxon rate $P = ½$. Sign-pattern frequencies computed from terms of $(p_s + q_s)^4$ and $(p_n + q_n)^4$ are, among $N = 200$ parents,

$Sc(4^+)$	= 0.52	$n = 52$	D_x confidence $\simeq$ 1.00
$-Sc(4^+)$	= 0.00	$n = 00$	
$Sc(3^+)$	= 0.37	$n = 37$	D_x confidence = 0.97
$-Sc(3^+)$	= 0.01	$n = 1$	
$Sc(2^+)$	= 0.10	$n = 10$	No D_x bets, even odds
$-Sc(2^+)$	= 0.10	$n = 10$	
$Sc(1^+)$	= 0.01	$n = 1$	D_x confidence = 0.97
$-Sc(1^+)$	= 0.37	$n = 37$	
$Sc(0^+)$	= 0.00	$n = 0$	D_x confidence $\simeq$ 1.00
$-Sc(0^+)$	= 0.52	$n = 52$	

There are some 20 parents at even odds, and the other 180 parents are diagnosable with [2(52) + 2(37)]/180 = .99 confidence. Now we examine individually the parent-*pairs* (who is married to whom having played no part in computations). We can say nothing about the (rare) pairs where both parents show the ambiguous middle pattern (2^+ signs each). But any pair in which both parents show a *non*schizotaxic pattern (0^+ or 1^+ sign) is quasi-certain ($p = .99^2 > .98$) not to have produced a schizotaxic proband. So we delete these probands from the "true *Sc*" group, and recompute the taxon rate among the parents who remain. There will be around 25 such families, and the new taxon rate among the 75 remaining pairs (150 parents) should have moved up close to the theoretical ½.

Suppose that even carefully diagnosed probands fall into 3 classes as to etiology:

1. Dominant schizogene (the schizotaxic schizophrene).
2. Bad mix of polygenes (for, e.g., introversion, anxiety, hypohedonia).
3. Phenocopy (trauma, slow virus, whatever).

There exist indicators (all fallible but strong, say, yielding 80% hits in a $P = Q$ situation) for the schizogene and for the trauma phenocopy, against normal controls. There exist moderate-to-high-valid indicators of each polygenic variable (no "hit rate" definable for these as nontaxonic). There is a double barrelled indicator which can be elevated by either the schizogene or by environmental trauma. Unfortunately the polygenic factors that produce genetic heterogeneity are also potentiators of psychosis for schizotaxics and for the effect of trauma. Thus, if I inherit a big dose of anxious, introvert, or hypohedonic polygenes, as well as the schizogene, I am more likely to decompensate than is a schizotaxic who is temperamentally nonanxious, extrovert, and has good primary pleasure capacity. Similarly the effect of trauma will be worse for the person who inherits the polygenic potentiators. And if these polygenic factors are large enough, a

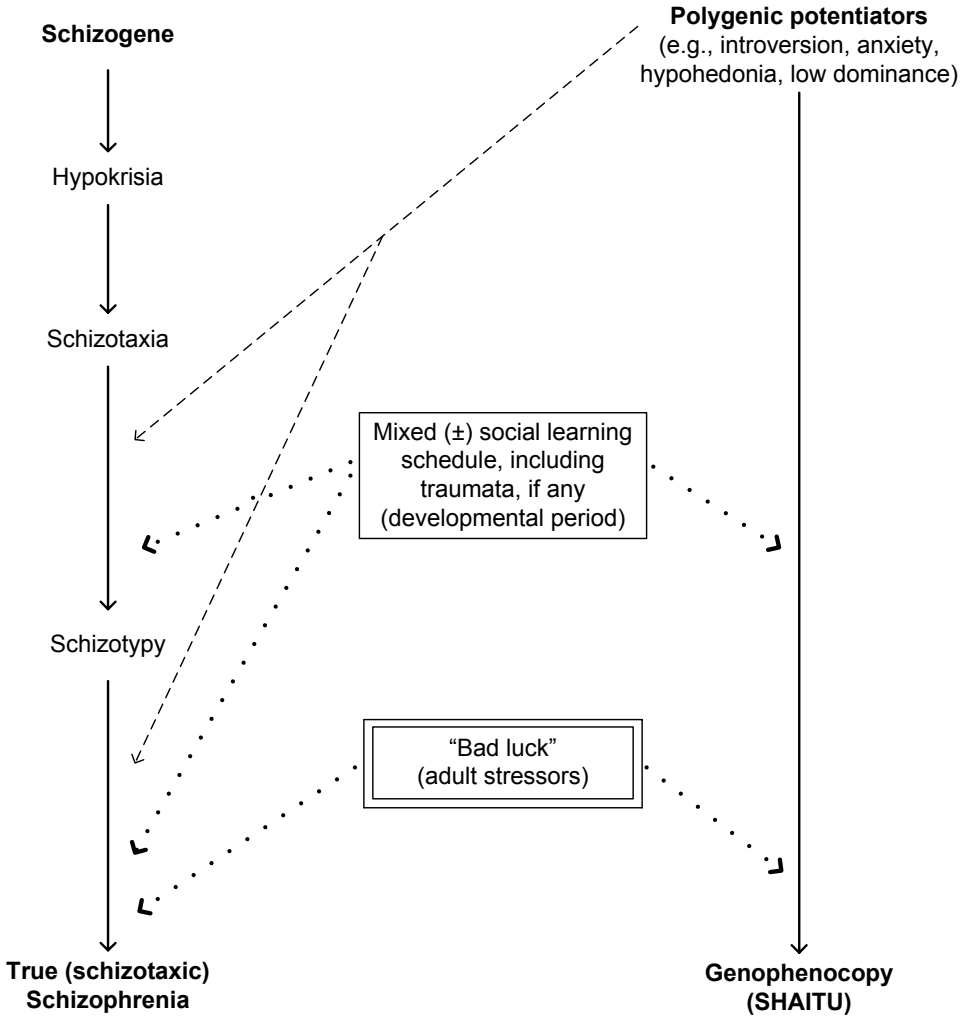

Figure 2. Conjectured causal paths determining schizotaxic schizophrenia and nonschizotaxic genophenocopies.

*non*schizotaxic *non*traumatized person may fall ill in a schizophrenic-appearing way, especially given "bad luck" (as distinguished from a specific trauma) in adulthood (see Figure 2).

One may doubt whether close phenocopies of such a complex syndrome can exist (cf. Vogel and Motulsky's [1986] view that, even for lower organisms and simpler traits, the phenocopy concept has been overused as an explanation). One may also doubt that polygenes for personality traits can fully mimic true schizophrenia, *if we had better taxometric weights for the latter*. For instance, given what normal-range social introversion is, do we really think being up 3 σ on such a trait could produce defective eyetracking and semantic slippage? Do we believe 3-σ hypohedonia could produce the massive blunting of chronic schizophrenia? But for purposes of argument, I will assume these are possible, at least with present diagnostic methods. (High diagnostic reliability does not, of course, prove high construct validity of DSM-III for the latent taxon we're after.)

Let $s_1, s_2, \ldots s_m$ be schizogene indicators.
Let $t_1, t_2, \ldots t_m$ be trauma indicators.
Let $v_1, v_2, \ldots v_m$ be polygenic variable indicators.

What is the probable latent structure's influence on trends among these? The *s*s behave taxonically in a sample of schizophrenics and their families, with or without "control" families. The *t*s also behave taxonically if the sample of probands includes an appreciable fraction of phenocopies. The *v*s would not behave taxonically in a sample devoid of both traumatic and schizotaxic cases, but that doesn't help here. Since they are schizophrenia potentiators, they will be weakly taxonic when mixed with normal controls (or affective controls). The polygenic pseudoschizophrenes will tend to have higher *v*s than the true schizophrenes, because to appear sufficiently schizophrenic to be so diagnosed requires more extreme anxiety, introversion, etc., *in a person lacking the schizogene* than is required to potentiate a schizotype into diagnosable schizophrenia. This is not a strong or detailed prediction useable for testing theory, but it is plausible enough to worry about when examining *other* proposed tests of theory, e.g., segregation among families at risk. In this mix of etiologies, the taxon rate (for schizotaxia) will be lower among parents than the dominant model ($P = ½$) predicts.

What can be done with these indicators? First we must clarify the concept of indicator specificity. "Schizospecific" cannot (usefully) denote the epidemiological ideal of high sensitivity with specificity = 1, in our present knowledge state. Even the Wasserman wasn't that good. Working with fallible quantitative indicators, I take "schizospecific" to mean that the indicator discriminates only the schizotaxon, any other real taxon being discriminated negligibly. This says nothing about how well it does it. The complement overlap may be small, medium, or large. But it is taxonic for this indicator, and the indicator is not taxonic within the complement class. One way to say that is, the complement class is not composed of taxa which *this* indicator discriminates. Of course the complement class may be intrinsically ("causally") taxonic, and in psychopathology it usually will be. Thus, the complement class for schizotypy in a psychiatric population includes the major affective taxa (manic, bipolar depressed, unipolar depressed), panic disorder, psychopathic deviate, and possibly obsessive–compulsive disorder taxa. (All the remaining "functional" mental disorders I conjecture are nontaxonic, being given category-style rubrics for administrative convenience and the medical tradition of disease entity semantics.) These within-complement taxa do not produce different latent distributions on the indicator we're considering. So specificity alleges appreciable (not necessarily high) taxonic validity for the taxon of interest and denies it for any within-complement taxa.

Suppose there are one or more *very* rare taxa in the complement class (say $p < .001$, like most Mendelizing mental deficiencies) that the indicator s_i discriminates strongly; or there are some sizable taxa therein that s_i discriminates *very* weakly. I call s_i *quasi-specific*, meaning specific enough to yield essentially correct results as to the taxon of interest. If a mutation with population frequency $P = 10^{-4}$ pushed s_i up a sigma, since this rate is three orders of magnitude less

than that for schizophrenia (and two orders less than for schizotaxia), that rare taxon will not foul up our taxometric results.

Suppose we have two schizospecific indicators s_1, s_2. This is pretty slim pickings, but the MAMBAC procedure can be run (both directions), and we can infer taxonicity and estimate the taxon rate among the probands. On what groups will it show taxonicity? Taxonicity should appear in these kinds of samples:

1. Mixture of probands with normal controls.
2. Mixture of probands with psychiatric controls.
3. *Within* proband group, due to a subset of traumatic genophenocopies. (We will not see three taxa in the total sample, because if s_1, s_2 are schizospecific, the traumatic genophenocopy—while a taxon—does not generate s_1, s_2 taxonicity within the complement class.) Taxonicity detected in this subsample would corroborate our conjecture that the probands are genetically heterogeneous, there being genophenocopies present among them, despite careful SADS diagnoses of schizophrenia (which nobody claims is over 90% "accurate" even accepting the DSM-III phenomenological concept of the entity).
4. Parents of probands. $P < ½$ because of proband heterogeneity (genophenocopies).

So on the dominant gene theory the taxon rate among parents is $½P_{dx:Sc} < ½$ when SHAITU cases have not been identified and excluded. It can be a little higher due to a few *Sc* doublet matings. We do not know whether there is assortative mating for compensated or Hoch–Polatin schizotypy, but it is probably small, extrapolating from the negligible assortative mating for schizophrenia, and the spouse $r = .08$ on MMPI Scale 8 found in the recent restandardization sample (W. G. Dahlstrom, personal communication, April 1, 1988). Checks of this kind do not require indicator-patterns valid enough to reclassify individual probands and thereby segregate schizotypal families from those ascertained via SHAITU probands. Only a valid taxon rate is needed for this kind of quantitative check on the model. However, deleting some probands as too doubtful, we can examine the high-confidence ones separately. (While indicators s_1, s_2 are quantitative, the taxometric methods locate a hitmax cut on each, whereby a score falling above the cut is treated as a dichotomous sign, denoted s^+ in what follows.) If s_1^+, s_2^+ have latent hit rates $p_s = q_n = .85$, we infer (conjecturing no nuisance correlation) the two-sign latent probabilities to be

$p(s_1^+ \quad s_2^+) = .7225$ in the schizotaxic class,

$p(s_1^+ \quad s_2^+) = .0225$ in the complement class.

The likelihood ratio is 32:1, and if only half of probands are schizotaxic, our diagnostic confidence in labeling them "schizotaxic" and "complement" = 0.97, safe enough for most research purposes, including ordinary pedigree diagrams for a Mendelizing entity.

Consider the parents of these two subgroups. Parents of the schizotaxics should show s_1, s_2 taxonic, with taxon rate $P = ½$, but parents of the nonschizotaxic probands should not. Further, the means of s_1, s_2 for parents

of nonschizotaxic probands should be down very near that of the normal and psychiatric controls, and near the means of the nonschizotaxic probands.

But there are only (.50) (.7225) (100) = 36 families with this 97% confident rediagnosis, thus only 72 total parents in each group, a bit small for some purposes. It appears that a taxometric approach to testing competing genetic models requires either large samples, high-valid indicators, or numerous indicators of moderate validity.

My view of strong inference from stochastic data differs from the conventional one. Sampling error matters to me chiefly because local fluctuations produce irregularities where the mathematics is smooth, discontinuities, jumps where the taxometric mathematics uses derivatives to minimize or maximize, multiple local extrema when the model has one only, "coarseness of grain," "holes in the Swiss cheese." These departures from idealization produce erroneous latent values of the sample which can fuzz up falsifications and corroborations. In the MAXCOV–HITMAX procedure, the latent value *K*, defined as the product of two taxonic separations $(\bar{y}_s - \bar{y}_n)$ $(\bar{z}_s - \bar{z}_n)$, is estimated from the observed (*yz*)-covariance in the hitmax interval of indicator *x*. Sampling error may place us in the wrong interval; and in the correct interval, the four latent means $\bar{y}_s$, $\bar{y}_n$, $\bar{z}_s$, $\bar{z}_n$ are subject to random fluctuations. It is this aspect of sampling error that troubles, rather than deviation of a *P* or $\bar{x}$ from that of a "population" (that we cannot even specify, or from which we cannot claim to have drawn our sample by a random number table). Reliance on coherence among nonredundant estimators of the same *sample* latent value, and multiple consistency tests with tolerances based on Monte Carlo runs, are therefore central features of my taxometric approach (Meehl, 1978; Meehl & Golden, 1982).

Assume the several taxometric analyses have shown that the proband group is taxonic, and have drawn the latent distributions on schizospecific indicators *x* and *y*. Setting aside diagnosis of individuals as schizotaxic or not (and, hence, of families as schizogenic or not), some useful quantitative relations obtain among the statistics. We now have estimates of the schizotaxon means $\bar{x}_s$, $\bar{y}_s$ and the complement means $\bar{x}_n$, $\bar{y}_n$. The latter include the proband phenocopies and polygenic pseudoschizophrenes. The latent complement distributions should agree tolerably with those of the parental latent complement as to means and sigmas, although not, of course, as to base rate. These means and sigmas (and distribution shapes) should agree tolerably with the manifest distribution of normal controls, but always allowing for the unascertained schizotypes of *P* = .10 in the general population. If our general population sample is large enough, it should have a taxonic structure with taxon base rate *P* = .10, and the means, sigmas, and shapes of the latent class distributions should agree tolerably with the corresponding latent distributions of the ascertained families.

Then the proband taxon rate *P* gives the proportion of schizogenic families (= parents who had a schizotaxic proband) among all parents who had a proband, true- or pseudoschizotaxic. Neglecting the small number of double-schizotaxic matings, assume each schizogenic parent-pair consists of one schizotaxic and one normal parent, according to the dominant model. Then the expected *x* mean over all parents is given by

$$\bar{x}_{\text{par}} = \tfrac{1}{2}\left(P\bar{x}_{\text{s}}\right) + \tfrac{1}{2}\left(P\bar{x}_{\text{n}}\right) + Q\bar{x}_{\text{n}}$$

(One could perhaps correct for doublet matings by comparing this predicted mean with the observed mean, an excess being attributable to the schiz-concordant pairs. It would be small but perhaps worth doing if the sample is large.) If the observed x_{par} is in tolerable agreement with this predicted value, the model is corroborated. I am not clear as to what the polygenic theory *predicts* about these means, although presumably it can be adjusted ad hoc to *fit* them.

AN INTRAPAIR TEST

There is a way to test whether the schizogenic parents are distributed over parent pairs as my theory predicts they should be. The mean absolute intrapair difference $|\overline{x_{\text{f}} - x_{\text{m}}}|$ for the schizogenic parents is $|\overline{\bar{x}_{\text{s}} - \bar{x}_{\text{n}}}|$, neglecting doublets. Among nonschizogenic parents, we conjecture negligible assortative mating for a schizotaxic indicator. (This can be tested directly in a general population sample of couples. Since assortative mating for diagnosable schizophrenia is negligible, it seems improbable that it would be appreciable for subtle schizotaxic indicators. As pointed out above, people do not marry on the basis of (±) dysdiadochokinesia or poor visual tracking.) Among nonschizogenic parents, the pairs are drawn randomly from the complement distribution of x. If the complement distribution is quasi-normal (testable taxometrically), the expected value of the absolute intrapair differences is given by

$$\left|\bar{\text{d}}\right| = 1.13 = 2/\pi^{1/2}$$

Combining these values from the two categories, one can predict the mean absolute difference over all parent pairs from

$$\left|\bar{\text{d}}\right| = P\left|\overline{\bar{x}_{\text{s}} - \bar{x}_{\text{n}}}\right| + Q\ 1.3\sigma_{\text{n}}$$

so tolerable agreement between this prediction and the observed $|\bar{\text{d}}|$ among all parent-pairs corroborates the model.

If the distribution of x in the complement class is distinctly non-normal, and one avoids fitting one of Pearson's twelve curve types (for which an analytical solution for $|\bar{\text{d}}|$ will exist), we can nevertheless estimate $|\bar{\text{d}}|$ by Monte Carlo runs on a distribution concocted in accordance with the latent complement distribution as inferred taxometrically.

If sample size and number of indicators permit, one can segregate the high-confidence schizotaxic probands (and, hence, the high-confidence schizogenic parent-pairs) and test the dominant model by several theorems in Golden and Meehl (1978). For example, among schizogenic parent-pairs (no doublets) the covariance of spouse scores is predictable from latent means $\bar{x}_{\text{f}}$, $\bar{x}_{\text{m}}$ by the relation $\text{cov}_{\text{fm}} = -\tfrac{1}{4}\left(\bar{x}_{\text{f}} - \bar{x}_{\text{m}}\right)^2$. No plausible polygenic model can explain that, let alone predict it in advance of data.

The intrapair method can be elaborated whenever one or more other indicators are available and taxonically valid. One can employ a sign or set of signs to sort

probands into k categories of taxon–member–probability. The "mix" of these gives the "mix" of the corresponding sets of parent pairs (again, we do not here try to diagnose the individual families, we only say, "There are more schizogenic families in parent Set I than in Set II; more in Set II than in Set III; …; and least in Set VIII.") The observed $|\bar{d}|$ of an unused indicator should show a trend over these groups.

If one or more other indicators yield diagnostic validities we can trust (not necessarily *high*, but *accurately estimated*, as judged by taxometric consistency tests), we can improve on the preceding by making point-predictions instead of ranking groups. Let the taxon probability associated with four values (intervals) of indicator x_i be p_1, p_2, p_3, p_4. Then we predict the mean intrapair absolute values of y for the parents in each x group by the relation

$$\left|\bar{d}\right|_i = P_i\left|\overline{\bar{y}_s - \bar{y}_n}\right| + (1 - P_i)\left|\bar{d}_n\right|, \quad \text{etc.}$$

The same can be done with pairs and triads of indicators other than x, and as the number of indicators increases this provides an accelerated number of such predictions to test. Thus, if there are three other indicators available, using the hitmax cut on each indicator when used jointly, four p levels on each of three indicators generates twelve cells. At the hitmax cut on each, for a pair of indicators, $p_s(++)$, $p_s(+-)$, $p_s(-+)$, $p_s(--)$, for the three possible pairs we get twelve cells to look at. The indicator triads yield $p_s(+++)$, $p_s(++-)$, $p_s(+-+)$, … $p_s(---) \rightarrow$ eight cells. So all told there are constructible 12 + 12 + 8 = 32 cells defined by the x, y, z scores, each of which can be tested empirically by using it to predict mean intrapair absolute values. As usual in my approach, we are not interested in testing whether the data are "significantly different" from the theoretical values, since we know they are (Meehl, 1990c). A "good enough" set of values is the point. One cannot meaningfully employ conventional significance tests when what is being estimated throughout are the sample values, "estimated" because they are *latent* and *structurally inferred*, not because they are sampled from some population. So long as the cells have widely dispersed p_s values, which they will (for single indicators one can choose cuts to make them disperse), a plot of the observed $|\bar{d}|$ values against those predicted from the equation should suffice to refute or corroborate the model.

NUISANCE COVARIANCE

Examining a major locus theory taxometrically is easier done when the indicators are negligibly correlated within the taxon and complement classes. In the first printed publication on the MAXCOV–HITMAX method (Meehl, 1973b) I explained this too briefly, and I find that even statistically sophisticated psychologists have trouble with it, probably because of our usual search for significant correlations in factorial (nontaxonic) situations. In the original presentation I wrote:

> Consider a provisional indicator set of not less than three phenotypic variables, deliberately chosen on the basis of the criteria above, plus

plausible grounds for hoping that they will be pair-wise uncorrelated (or approximately so) within the postulated latent taxa. So I am making the same assumption as in Lazarsfeld's latent structure analysis, that the observed correlation between the indicators is almost wholly attributable to the influence of the latent taxa. This assumption need not remain an "assumption" in the technical sense of the statistician, i.e., something we postulate without having any means of testing it on the data (as, for instance, the psychometric assumption underlying an arbitrary normalized transformation of test scores). The assumption itself can be a statistical hypothesis subject to refutation, and I have developed a group of "consistency" tests which should help us decide whether the intrataxon independence assumption is being grossly violated (Meehl, 1965, section 9 [pp. 24–34]; Meehl, 1968, section g [pp. 41–43]). Further, we can raise the odds that this independence condition will be approximately fulfilled, at least close enough for the use of the proposed bootstraps method. First, we may rely on theoretical considerations, such as that indicators sampling different behavioral domains or different neurological systems—having, so to speak, very little "qualitative phenotypic similarity or overlap"—ought to be relatively independent. Second, we can ascertain the empirical correlation between the indicators within a group of normals (where the base rate of schizotypy can be safely taken as so low as not to be capable of generating a correlation) as well as among diagnosed schizophrenics, and then extrapolate to the working hypothesis that if a pair of indicators is uncorrelated *within* the schizophrenic group and *within* the normal group, it will probably not be *markedly* correlated among nonpsychotic schizotypes found in the "normal" population or in a mixed psychiatric population with an erroneous (nonschizoid) diagnosis. Third, in the case of psychometric indicators such as scores on a personality inventory or a "mental status" checklist (Meehl, 1964) or rating scale, we can employ item-analytic procedures to reduce the intrataxon correlation, which we will be willing to do at the expense of sacrificing some amount of validity. I cannot yet make any general statement about the robustness of my method with respect to this assumption of zero intrataxon correlation, although I have some numerical examples (e.g., Meehl, 1965, section 13 [pp. 49–54]) as well as some empirical data on one taxonomic problem, suggesting that a Pearson *r* running up to .30 or .40 may not be too damaging.

An illustrative example of such a provisional indicator set would be (a) a psychometric measure of subclinical cognitive slippage based upon intransitive ("irrational") choice behavior (Braatz, 1970); (b) a measure of the paradoxical effect of alcohol ingestion upon post-rotatory nystagmus (Angyal and Blackman, 1941); (c) a patient's score on a structured personality inventory measuring (by self-report) the phenomenology of pleasure deficit (Rado's *anhedonia*; see Rado, 1956, 1960; Rado and Daniels, 1956). These three kinds of behavior are sufficiently different in the kind and level of dysfunction tapped that one would be surprised to find them appreciably correlated either in a normal population or in a clinical population of non-schizoid psychiatric patients from which organic brain disease, mental deficiency, and grossly psychotic cases

> had been excluded. So we have here three tentative indicators of schizotypy which we have plausible reasons to hope are relatively independent except as they are influenced by the hypothesized dominant schizogene. (pp. 205–206)

Since it appears that this passage is often ignored or misunderstood (some colleagues have said, "That independence requirement doesn't make causal or statistical sense to me"), let me try here to explain the rationale more fully. The taxometric task has two components, (1) corroborating the conjecture that a latent taxon exists, and (2) sorting of individuals into the categories thus identified (Meehl & Golden, 1982). The term "classification" in the broad sense covers both the process of constructuring or inferring classes (narrow sense) and individual "assignment," "identification," "diagnosis." Epistemologically, one considers first the *cumulative evidence for a taxon's existence*, and then the *evidence for an individual's membership in it*, to be stronger when the separate pieces of evidence, the various facts, would not be expected to agree unless the conjecture were true. A philosopher of science might speak here of "Popperian risk" or "Salmonian coincidence" (Meehl, 1978, 1990c). Corresponding to this epistemological preference is the statistical structure of Bayes's Theorem, where the diagnostic likelihood ratio is attenuated by sizable pairwise correlations of indicators within classes, as the joint conditional probabilities of indicator copresence do not follow the multiplication theorem for independent events. Further, most of the theorems relied on in Meehl's and Golden's taxometric methods assume independence, and the statistics becomes a good deal messier when that assumption is violated (cf. Meehl, 1995). So for both epistemological and statistical reasons, it is desirable to use indicators having low nuisance (within-category) correlations.

Having become accustomed over the years to "thinking taxometrically," I am puzzled when colleagues find this puzzling! There is nothing either causally or statistically odd about it. Even in nontaxonic situations (e.g., multivariate prediction of college grades) we know that we want the predictor variables to be as little correlated as possible. We do better using high school rank plus study habits or an academic achievement interest measure than we can using high school rank plus an intelligence test, much of the latter's valid variance being tapped by high school rank. Without such antecedent theorizing, a stepwise regression procedure would probably yield the same result.

My impression from conversations and correspondence (I know of no published objections) is that the unfamiliarity of taxonic thinking—American psychologists having been educated to presume that there are no real types, there are only deviations on dimensions—combines with difficulties about plausible causality to produce the incomprehension. How can indicators x and y have good taxonic validity if they don't, so to speak, "have anything in common (= share statistical variance, have overlap in statistical elements)?" Something along those lines seems to produce the conceptual hangup. To alleviate it, one must keep in mind that a theory with "deep structure," postulating a latent dichotomous causal factor that operates as a sine qua non (Meehl, 1972a, 1977), may give rise to a set of causal chains whose termini are qualitatively unalike, and some of whose

links are attenuated by other nonspecific causal influences that render the cause–effect linkage stochastic rather than nomological (Meehl, 1978). There is nothing mysterious or unusual about that, it happens all the time in the life sciences. *Example*: When geneticists invoke pleiotropy, we are not surprised or mystified. *Example*: In the general population of Caucasians, IQ is not appreciably correlated with fair complexion. But the PKU taxon has low IQ and fair complexion copresent as indicators (although note, *how low* IQ and *how light* complexion are not nuisance-covariant within the taxon either, just as they are not in the *normal* population). Nor does taxon mixture generate a significant Pearson *r* in the "general" population, which includes PKU cases, because the taxon base rate is so low. The causal situation underlying these statistical relations is well understood. One product of the normal katabolism of phenylalanine is tyrosine, which is a precursor of melanin. When the usual katabolic sequence is interrupted in the PKU case, associated with a loading up of phenylpyruvic acid (which impairs CNS function and hence lowers IQ) is a deficiency of tyrosine, hence less melanin, hence pale skin.

"Deep structure" causality produces qualitatively diverse signs and symptoms in nonpsychiatric medicine which would be utterly mysterious without details of the pathophysiology. A patient presents with complaints of weakness, fatiguability, malaise, night sweats, intermittent fever, joint pains, anorexia. He *looks* sick. Hearing a pronounced heart murmur, the physician asks, "What childhood diseases did you have?" and learns one was rheumatic fever. Whereupon he exerts special care in the abdominal exam, finding a barely palpable spleen. So he examines the inner eyelid (not part of standard physical) and, behold, sees a pale centered petechial haemmorhage. *Diagnosis*: Subacute bacterial endocarditis, $p > .95$. This set of "unrelated" signs and symptoms is analogous to the heterogenous collection of schizophrenia indicators (e.g., odd verbalization, soft neurology, interpersonal aversiveness, dereism, inappropriate affect, SPEM, and P50) that impels one to postulate a ubiquitous "deep structure" aberration as the specific etiology.

If an intrinsically dichotomous causal agent (e.g, Huntington mutation, head injury, spirochete infection, childhood seduction as in Freud's 1896/1962 theory) underlies a phenotypic taxon, why are taxonically valid indicators imperfect? Why is there distribution overlap on MMPI scales or psychophysiological measures between the taxon and complement classes? More generally, why are there so few signs and symptoms in general medicine that are two-way pathognomonic? Obviously, because other causal factors, over and above the taxon-specific cause, and largely independent of it (causally and, hence, statistically), generate individual differences variation among organisms *within* the taxon and *within* the complement. For instance, the schizotaxic integrative defect produces "clumsiness" (Heinrichs & Buchanan, 1988; Manschreck, Maher, Rucklos, & Vereen, 1982; Meehl, 1964). But numerous polygenic and environmental factors having nothing to do with schizophrenia, neither genetically linked nor genetically correlated by assortative mating with schizotypy, give rise to *normal-range individual differences variance* in the skill–grace–clumsiness domain; and there is no reason for expecting these factors to lose influence merely because the schizogene is present. What the latter does is shift this

multiple-factor individual differences distribution toward the "clumsy" end of the dimension. The taxonic validity of a quantitative indicator depends on the size of that schizospecific shift in ratio to the within-group sigmas generated by the nonschizotaxic polygenic and environmental factors. Observed correlation between two indicators either among schizophrenes or controls is therefore undesirable for *both* theoretical and psychometric reasons. When a correlation of .77 is obtained between the nailfold capillary bed anomaly and my Checklist of Schizotypic Signs (Buchanan & Jones, 1969), I like that because the sample studied was first-degree relatives of schizophrenes, among which my theory expects a base rate $P = .50$ of schizotypy, a high risk. But if a sizable Pearson r between these two indicators were found among "people in general," that would not please me at all, since the estimated schizotaxic base rate of $P = .10$ leads to a predicted correlation of only .26, assuming a taxonic separation of 2 σ. The more two candidate indicators correlate among schizophrenes or among controls, the less schizospecific they are; and, hence, the less useful they can be either for testing a taxonic theory, or for diagnosing individuals if the theory is accepted.

I alerted the reader that my approach to testing a dominant gene model was unconventional, and in closing this taxometric discussion I should perhaps motivate that preference. More knowledgeable colleagues inform me that, given the data on prevalence, risk to relative, clinical penetrance, and diagnostic accuracy, it is difficult or impossible to distinguish a major locus situation from a polygenic one, unless we had thousands instead of hundreds of subjects. Further, I have argued above that the complexity of my causal model is such that, assuming infallible diagnoses and no sampling error, the system of equations based on MZ, DZ, parent, and child risk figures would be mathematically indeterminate. I detect a tendency for behavior geneticists to slip from epistemology to ontology here, "You can't tell them apart, so…." Since a dominant gene with polygenic potentiators is clearly different theoretically from a straight polygenic model, my answer to such methodological pessimism is to say, "The two theories are different, so it must be possible to tell them apart, somehow or other. If the standard methods of pedigree analysis, path diagrams, linked marker, etc., can't do it, then one looks for *other methods of statistical analysis* to do the job." I conjecture that proper taxometrics will do it, and their being novel and not "mainline statistics" does not trouble me. For a different and seemingly powerful taxometric approach (applied to the soft neurology of proband offspring), see Marcus, Hans, Mednick, Schlusinger, and Michelsen (1985) and earlier papers by Marcus and colleagues therein cited.

TWO TYPES OF SCHIZOPHRENIA

Taxometric analysis of probands and families, with emphasis on soft neurologic and psychophysiologic indicators of the conjectured dominant schizogene, has the aim of distinguishing a latent taxon defined theoretically by its specific etiology, the latter being "intrinsically dichotomous" rather than a quantitative influence (e.g., polygenic factor with a threshold for clinical illness). That conceptualization, if corroborated by coherent taxometric statistics,

would warrant a semantic convention distinguishing "true (= schizotaxic) schizophrenia" from genophenocopies that imitate it clinically, and even psychometrically, in various respects and degrees. The SHAITU syndrome is only one plausible possibility, being specified by five conjectured causal factors; other combinations would do as well, being collectively the alternative nonschizotaxic ways in which the currently accepted clinical picture of "schizophrenia," DSM-III or other, may originate. How does this theoretical division between schizotaxic and nonschizotaxic schizophrenia relate to some of the traditional divisions?

I hope I am not reinventing the wheel, merely concocting a novel terminology for something already well delineated. I think not, for this reason: The psychiatric literature presents such clinical dichotomies as "nuclear/non-nuclear," "process/reactive," and "schizophrenic/schizophreniform." Psychologists have constructed quantitative scales that can be treated dichotomously, e.g., Elgin "prognosis good/poor," Phillips "good/poor pre-morbid." Then we have the Schneiderian "first-rank/second-rank" symptoms, and the distinction between "positive" and "negative" symptoms. These various dichotomies, while importantly different, also have some degree of overlap, partly as to symptomatology, partly as to malignancy, and (sometimes) with a hint of different etiology. Even the original Kraepelinian subtypes, retained by Bleuler in his extension of the dementia praecox construct, are somewhat correlated with these divisions (e.g., Bleuler presents different percentages of clinical recovery for the four subtypes hebephrenic, catatonic, paranoid, and simple). Going back to Babcock's work on psychometric deficit in the early 1920s, psychometricians have noted the grosser cognitive impairment typically found in hebephrenics as compared with the "relatively intact" paranoids. Even somatotype has been associated, "mesomorphic toughness" predisposing to aggressive paranoid projection and ecto- or endomorphy to "hebephrenic jettisoning," the former also having stronger egos that enable them to remain compensated until a later age.

Despite the hundreds of studies conducted with these divisions serving as independent variable, the typological question remains unanswered. Why is this? A horde of bright researchers expended thousands of hours of scientific time and millions of taxpayer dollars, exercising considerable ingenuity in design and tasks, most of which showed "statistically significant differences," without solving the problem. This seems so strange that one should be prepared to accept a strange explanation. My explanation may seem strange, given the research tradition, but it is quite simple: (1) The researchers have not bothered to analyze the question being asked, and as a result, (2) they have failed to see that the statistical methods employed cannot answer it.

The question, "Are the subtypes (Kraepelin, paranoid/nonparanoid, process/reactive, or whatever) differences of *degree* or of *kind*?" is simply too vague as it stands. If explicated in terms of a latent taxon (produced by a specific etiology) versus its complement class of "clinical imitators," one sees immediately why *none of the investigations could possibly answer this question*. They all proceed by initial division into the conjectured categories (never mind how, by what criterion, or how reliably—it doesn't matter!). Then the groups are compared on some psychometric or experimental

measure (Rorschach, MMPI, pursuit rotor, nonsense syllable learning, reaction time, etc.). Suppose they differ, as they do more often than not, although the frequency of failures to replicate is disturbing. What has been proved about types or taxa? Nothing—literally *nothing*. If a collection of quantitative variables is correlated, *for whatever reason*, dichotomizing on any of them will, given adequate statistical power, result in significant differences on those variables that are treated as dependent. Such a design cannot reveal anything interesting about categories, types, taxa, differences in kind rather than degree, let alone specific etiology. How could it?

Analogy: Faced with the problem of children who do poorly in school, we conjecture that they belong to two "types," the mentally deficient and those who, say, "just dislike school—desks, books, pencils, teachers, rules, the whole scene." We construct an intelligence test, and divide the original group of low achievers ("clinically" identifiable) into "dulls" and "brights," cutting the IQ distribution at the median. Then we contrast the dulls versus brights on various other measures, for example, spelling achievement, study skills, days absent, lateness of assignment completion, liking for teachers, hours spent on homework, peer sociometrics. On most of these measures, we find differences; on others, not. So what? Suppose the brights are found to have greater teacher dislike than the dulls. So what? We will find that almost every variable increments the predictability of whatever we treat as "dependent." So what? No set of differences can inform us as to the reality of the two conjectured types, for the simple reason that if any two quantitative variables x and y are correlated, dichotomizing on x will show mean differences on y. The whole business amounts to nothing but exemplifying truisms of the statistician; for example, a Pearson r will, if variables are dichotomized, yield a nonzero phi-coefficient. And it's not merely that these trends do not conclusively *prove* there are two categories; they do not even *tend* to prove it. The sad fact is that the results of such a design and analysis *shed no light whatever* on the taxonic problem, assuming that is what was being intelligibly asked.

It is hard to understand why knowledgeable psychologists could have supposed such research capable of answering a significant question about "two kinds." *Moral: He who asks a taxonomic question must employ taxometrics in order to answer it*. It may be that some other ways of looking at such data can illuminate the taxonomic issue; I am not sure about that. Some seem to believe that bimodality on a composite is the test, but this is incorrect if taken simpliciter, as shown by Murphy (1964) and others. It is easy to generate a unimodal distribution from two overlapping ones, for example, if they are less than 2 σ in mean separation. Bitangentiality is perhaps suggestive of latent taxonicity, but not safe as a litmus test. If the factor structure underlying the correlation matrix of dependent variables is markedly different between the two groups, that is suggestive, although it can come about in several ways. For example, some tests may have weak loadings in some region of a factor, as digit span discriminates g better in its lower region than higher up; so psychometricians would not have retained it as a subtest in omnibus intelligence tests except for dull and borderline discrimination purposes.

In arguing this negative case against the conventional approach, I do not presuppose that taxometrics—my methods or others—can always or easily

settle the question. My point is that taxometrics is a *necessary* (but perhaps not *sufficient*) condition for testing an interesting taxonomic conjecture. Whether and how often multiple taxometric methods (including strong consistency tests) can mislead by identifying a "pseudotaxon" is presently an open question, involving not only analytical and Monte Carlo study of the mathematics but, equally importantly, *conceptual* clarification of the several meanings of "taxonicity" in the life sciences (Golden, 1990; Golden & Meehl, 1978, 1980; Meehl, 1972a, 1973b, 1977, 1979, 1986a, 1990b; Meehl & Golden, 1982).

The preceding shows why I consider the available data (as conventionally analyzed) insufficient to answer the "two-types" question, and suggests research directions that can, in principle, answer it. Given that skepticism for now, the whole of this paper (taken together with Meehl, 1989b, 1990b) delineates my personal conjectures about the matter. Since my metaconcept *taxon* is etiological and dichotomous, the theory here proposed postulates one real taxon, the schizotaxic schizophrene, and a complement consisting of "everyone else labeled 'schizophrenia,' by whatever criteria." I must stress again that this is an *ontological* dichotomy, and does not involve the epistemological concept of "misdiagnosis" *on the received criteria*. The latter is a result of the diagnosing clinician being careless in applying the nosological criteria she accepts (or that the profession "officially" stipulates); or, if careful, being unlucky in the behavior sample presented by the patient (cf. Peppard, 1949, his sizable error category "symptoms or signs *not found*"). By this way of reasoning, the complement class among "schizophrenic" probands is composed of (1) patients who should not have been labeled "schizophrenic" by presently accepted standards, and (2) patients correctly diagnosed "schizophrenic" by accepted standards who are etiologically not schizotaxic. I of course do not label subset (2) "diagnostic error," which would be high-handedly imposing my semantics upon those careful clinicians who do not share my etiological conjectures. However, *on* those conjectures, the complement class would be composed of SHAITU syndrome cases, plus miscellaneous "clinical mistakes," some more careless than others, and quite a few arising from noncareless "sampling error" (the proband as a statistical urn from which we draw a batch of varicolored interview and psychometric marbles).

I do not anticipate a clean mapping of any of the dichotomies mentioned at the beginning of this section onto my etiological dichotomy, should the latter prove correct. But there should perhaps be some correlation. For example, I expect the "negative" symptoms to be tied closer to schizotaxia than the "positive" ones, partly because my strong Bleulerian leanings classify the positive symptoms (delusions, hallucinations, catatonia) as "accessory"—their absence not adequate to rule out a Bleuler diagnosis of schizophrenia, and their presence not sufficient to rule it in. I think it fair to say that DSM-III, partly because of its composers' preoccupation with reliability (cf. Meehl, 1986a), has constituted a shift away from Bleuler's 1911 delineation of the schizophrenia concept. I conceive that to have been a mistake, but only taxometric (or "lucky biochemical") research will decide.

As to delusions and hallucinations, which I called "secondary cognitive slippage" (to distinguish from the "primary cognitive slippage" of Bleuler's *loosening*), everyone agrees that neither is schizospecific, except when a

content (e.g., bizarre) and a *context* (e.g., inappropriate affect) are specified. It is not even clear to what extent primary slippage contributes to secondary, as witness the negative correlation between psychometric and experimental measures of the loosening or "abstraction" defect and paranoid delusions (although the commonly stated relation is perhaps overdone). Consider delusions of persecution, surely a gross form of cognitive slippage, although one must not conflate *social malignancy* (via the atheoretical concept *severity*) with *degree of pathology* (cf. Meehl, 1972b). Readers who have been fortunate (as scientists, unfortunate as healers) in observing the development of a paranoid delusion during psychotherapy, and daring enough (like me) to pursue a cognitive correction approach (relying on a strong positive transference), will agree that the cognitive errors the patient commits need not hinge, even slightly, on subtle semantic slippage, Bleulerian loosening, or the "formal" aberrations emphasized by von Domarus, Kasanin, and others. Recently I kept logician's track of "how the patient did it" (I was concurrently reading some works by philosophers on the subjective element of empirical inference in science) and came up with the following list of inferential mistakes:

1. Selective attention (biased "data-collection")
2. Selective recall (biased information retrieval)
3. Restricted range of hypotheses (bias in not considering alternative explanations of data from (1)-(2)
4. Bayes's Theorem mistakes
   a. Maldistribution of priors (antecedent probabilities), inflating paranoid, deflating "normal" others
   b. Maldistribution of conditionals, inflating paranoid, deflating others
   c. Informal miscomputation of posteriors
5. Considering evidence strong for paranoid H_2 when it follows as well from H_1, where H_2 is a subcase of H_1, which covers numerous nonparanoid alternatives
6. Neglected Aspect Fallacy (violating Total Evidence Rule)

These are "autonomous knower" cognitive errors, setting aside the weakened push to consensual validation, which in healthy persons sometimes functions as a potent corrective, discussed below. None of these errors in evaluating evidence presupposes formal thought disorder, and in my patient the mild and rare episodes of schizophasia did not appear until several weeks *after* the occurrence of clearly delusional ideation. The other striking fact about the above list of meta-mistakes is that none of them is qualitatively pathological; they are all found in the mistaken empirical reasoning of ordinary life, and even of scholarly life, *including science*. More distressing, I would have a hard time showing that the patient's abuse of empirical inference is even *quantitatively* worse than that of thousands of "normal" persons who hold a variety of irrational views about politics, economics, religion, ethics, not to say the conduct of their personal affairs. One thinks, for instance, of the high-IQ New York literati who continued to defend Stalinism for two decades after the clearly fake Moscow trials; the contemporary right-wingers who think Nixon was framed; the psychotherapists who insist there is "no good evidence" that genes have anything

to do with schizophrenia; the 20,000 dues-paying members of the Flat Earth Society (who can "explain away" the falsifying evidence from the moonshot!). At a practical level, consider the 60% of Americans who don't fasten seatbelts, or who ruin their health by a variety of life-style practices; or the acres of "used" (hardly driven, smelling fresh from the factory) cars that were reposessed because the purchaser failed to make the most obvious inference from his checkbook record, that is, that he could not possibly keep up the payments. My (perhaps jaundiced) view is that the great preponderance of "normal," literate high school graduates can barely think rationally about anything, theoretical or practical. *Point*: It does not require anything wrong with one's neurochemistry or cerebral wiring diagram to hold beliefs that an inductive logic machine, if there were one, would classify as "empirically refuted with probability > .99." The extent to which basic CNS malfunction, describable at the biochemical or microstructural level, *facilitates* crazy ideation is, I suggest, simply unknown at present. I do not conjecture that the influence is zero, but we cannot say today, even roughly, how controlling it is.

One still hears the biotrope argument that, unless we are metaphysical dualists, *something* must be "wrong with the brain" if it thinks delusionally. This is an elementary mistake about levels of causal analysis, as if to argue that since speech is surely a brain process, therefore habitual bad grammar indicates a lesion in Broca's area, or that proletarian table manners must be due to something like dietary deficiency harming nerve cells, because "knowing which salad fork to use" is a cerebral process! One would hope that such category mistakes were cured in freshman logic class, but it appears otherwise. Skinner (1938) showed that rats can be trained to press a lever 192 times per pellet of food reward, if the reinforcement schedule is changed slowly enough. But shifting directly from crf to FR192 will starve the animal to death, despite adequate caloric payoff (cf. Meehl, 1962b). A dogmatic biotrope might insist that such a rat "must have something wrong with its brain." (I suppose a dogmatic sociotrope could invoke a rodential death wish.)

All such muddled thinking comes from failure to analyze the situation conceptually and mathematically. Conceptually, we deal with dispositions of various orders. Mathematically, we deal with the parameters of functions describing how dispositions are learned and, once learned, activated (acquisition and control functions). I must repeat that there is nothing deep, esoteric, or difficult about all this. To understand it one needs only to grasp the idea of a *disposition* (tendency, potency, capacity) and the idea of a *parameter* (numerical constant in an equation). I shall say more about this in the next section.

Clinicians have tried to distinguish varieties of secondary cognitive slippage as to their diagnostic weight. I see merit in some of those distinctions (e.g., Schneider's "first rank" symptoms, 1959), while insisting that the relative weights of candidate indicators should be based on strong taxometrics. Where in Schneider's perceptive and suggestive book does one find a metacriterion for assessing the candidate signs? There is none. He *thinks* in terms of an open concept of schizophrenia, but he does not state the open concept weight criteria. I cannot fault him for that, no adequate taxometric methods being available to him. I merely point out that"sign S_1 is first rank" and

"sign S_2 is not first rank" are statements that, absent an external pathological criterion (he says we have none), are *intrinsically* taxometric claims (cf. Meehl & Golden, 1982).

I agree with Albert Ellis and Bertrand Russell that irrationality (illogical deductions plus faulty collection and evaluation of data) is not a rare aberration but pretty much the normal human condition. Cognitive, clinical, and social psychologists tell us that cognitive mistakes do not require that powerful motivational–affective distorting influences be at work (Dawes, Faust, & Meehl, 1989; Faust, 1984; Nisbet & Ross, 1980; and see references cited by Meehl, 1990c). Of course such counterrational forces, whether psychodynamic or ideological, often act to potentiate the garden-variety slippage to which our cerebral computers are prone. Further, even hyperrationalist Bertrand Russell makes the point that most of what we believe is believed on authority, since no one has the time or competence to investigate all the subjects on which he entertains opinions. Reliance on others is not *irrational*, so long as one picks the right (?) others and gives them the proper (?) weights. The relevance of this consideration for psychopathology is that attenuation of social bonds may be expected to reduce the search for consensual validation which normally serves as a check on cognitive slippage. The schizophrene (including SHAITU) who has withdrawn cathexis from others cares less" what they think. (His "Asch effect" cut has shifted from $N = 1$ to $N = 0$). To this decathexis we add the predominance of ambivalence and mistrust in such object-relations as remain, as well as the sheer malcommunication problems, often of long standing. We know from the major affective disorders that sufficiently powerful motivations and affects can produce secondary cognitive slippage—to the extent of delusions—in the nonschizotaxic brain. I find it helpful to conceptualize the general ("normal" and "sick") cognitive slippage problem in terms of a basically inefficient, stochastic, approximate "inference machine," rather badly constructed, then poorly adjusted and honed by culturally normal education (precept, modeling, reward, and punishment). The constraints of rational thought and reality testing are quite loose, even for the "healthy" machine. Motives, affects, and social compliance largely determine our beliefs, except for specialized contexts (science, "hard-headed" business rationality, chess playing) in which the "rational rules" are more strictly drawn, the consequences of error severe and quasi-certain, and the social reference group idealizes rationality. When motivational–affective forces are powerful, and the psychic need and communicative efficacy for consensual validation are weakened, the degree of cognitive malfunction can exceed even the tolerant constraints of "normal" thought.

I conclude that delusions, in general, do not require an abnormal brain. This is of course compatible with conjecturing that an abnormal brain is sometimes the source, and that some kinds of delusions may be hard to come by if the brain's chemistry and microstructure are normal. One can analogize to a crazy computer printout (say, where the Pearson r is 1.83, or the partitioned sums of squares exceed the total SS_T). The transistors may be defective (like having schizotaxic neurons). Or a saboteur may have pounded it or poured acid into it (like a concussion or lead poisoning). Or the program may have bugs in it (like being raised to think oddly by a malcommunicative pathological

family). Or the mechanism and program may be all right, but the data fed in are incorrect (like news propaganda or a trusted friend deceiving one). All of these are "physical" in the metaphysician's sense, but they are obviously not all "organic disease" in the conventional sense of the neurologist.

This is the best I can do to defuse the biotrope/sociotrope conflict as concerns delusions. What about hallucinations? (I omit the hypnagogic and hypnopompic, and the "expectancy" kind, where one eagerly awaiting a visitor hears a faint car door slam, these being found in perfectly normal subjects.) Here I'm on the fence, but incline to doubt that clear, persistent auditory hallucinations can be produced by the set of psychological factors described above for delusions, given a CNS functioning "normally" at the cellular level. I also incline to think that vivid, persistent visual hallucinations are more pathological than auditory, and I would bet odds that a SHAITU cannot produce them. I tend to order secondary thought disorder on a scale running from least to severest pathology (with increasing probability of primary slippage due to *cellular level* malfunction involved) thus: Depressive delusions < Persecutory delusions < Grandiose delusions < Auditory hallucinations < Visual hallucinations. But all of this is highly speculative, and based more on clinical intuition and theory than on evidence. How these kinds and degrees of cognitive slippage relate to etiological "kinds" of schizophrenia is a taxometric question.

BIOTROPES AND SOCIOTROPES

The division between biotropes and sociotropes (Hollingshead & Redlich, 1958) is still with us, despite clarifications by myself (Meehl, 1962a, 1972a) and others, and I sometimes despair of explaining the conceptual and methodological situation so that practitioners and theorists will understand it. I am heartened by the impression that most graduate students are clearer in the head than psychologists and psychiatrists of my generation, so perhaps science does advance after all! Relying upon general theory, clinical experience, common sense, and the quantitative research on schizophrenia, we can identify three positions that are surely unsound. The dogmatic biotrope believes that the whole thing is a matter of aberrated neurochemistry or CNS microstructure, so that any description or causal analysis in social or psychological terms is an irrelevancy, having nothing important to say about causation, and therefore of no use in treatment. The dogmatic sociotrope persists in denying that there is anything either genetic or physiological about schizophrenia, and is impelled (although not always) to disapprove of pharmacological or other "organic" interventions such as EST. Then there is a third approach that appears superficially to be preferable because of its "tolerant" undogmatic character, but in the long run would be equally bad if not worse, namely, to say that there is no problem here because, after all, everybody has a heredity and an environment, so what can the argument be about? I say this one could even be worse in the long run, because it forestalls conceptual and mathematical analysis at the level of theory; and, if consistent, would deprecate empirical studies of heritability, and the interaction of genetic and experiential

factors, classifying the whole problem as an illusory one, or as a matter of "semantics." Disagreements about the way in which different classes of causal influences jointly determine an outcome, and the associated statistical question of parsing their net quantitative influence (e.g., analyzing the variance into its statistical components—but that's not the only way to mathematicize it) is *not* simply a matter of how somebody prefers to use words. "It's just a matter of semantics" has become an easy copout in the social sciences, of which we should strongly disapprove, because (as Popper quotes Immanuel Kant), "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" (Popper, 1935/1959, p. 13). It would be quite wrong, for instance, to criticize the labors of the constructors and revisers of DSM-III for wasting time on "verbal conventions" or "mere labeling of people," since if they are doing their job properly—a question I do not discuss here—they are attempting to arrive at semantic conventions that will, in the words of Plato, "carve nature at its joints." So this third approach, which sounds so melioristic and fair-minded, is in effect scientifically malignant, because it sweeps what are real problems of conceptualization, fact, and statistical analysis under the rug. It is meaningful and important to ask whether we are all born with an equal talent for developing schizophrenia, and if we are not, then what is is that the people born with that talent have dispositionally that the rest of us lack; and, given that talent, what are the factors in the developmental period, and in adult life, that alter the probabilities of schizophrenic illness? This will be my sixth (Meehl, 1962a, 1972a, 1972b, 1989b, 1990b) and, I trust, *last* attempt to clarify the question so that we can get on with the theoretical modeling and empirical testing unencumbered by conceptual confusions.

It is puzzling why so many are still confused about the biotrope and sociotrope issue when from one big fact, combined with general theoretical knowledge about human behavior, one could predict a great deal of what is empirically known about schizophrenia with high confidence, and could assign high prior probabilities to theoretical conjectures that should properly influence any balanced interpretation of conflicting empirical studies. It is absurd to translate R. A. Fisher's rule that experiments in agronomy should be self-contained—a statistician's principle about where you should get your estimate of the residual sum of squares!—to mean that reflection on the import of schizophrenia research must take place in vacuo, as if the theorist trying to interpret data specific to schizophrenia should pretend that he knows nothing about conditioning, psychometrics, trait theory, motivation, ethology, developmental psychology, or mammalian learning. Psychologists do know quite a bit about how the mind works for people in general, and there is no good reason for assuming that because somebody develops a mental illness, all of those principles immediately go by the board and can play no role in interpreting research data.

The "one big fact" I allude to is the monozygotic concordance for schizophrenia of 50–60%. This being 50 times as great as the general population lifetime morbidity risk of 1%, four or five times greater than the like-sex DZ concordance, and yet far below perfect concordance, tells us that schizophrenia involves both powerful genetic factors, and powerful environment factors.

Any theory purporting to explain why some persons fall ill and others do not, that considers only one of these two classes of factors, is radically defective. Some humans have a talent for schizophrenia, just as some have a talent for playing the violin. But not everybody with violin talent becomes a violinist, and not everybody with a talent for schizophrenia becomes a schizophrene. In this respect we deal with a genetic predisposition that is only a necessary but not sufficient condition for illness, is a sine qua non, unlike Huntington Disease, or most of the 150+ Mendelizing mental deficiencies. As with PKU, something from the environment must be provided to produce the disease.

It doesn't take any quantitative research to know that the *content* of clinically diagnosable schizophrenia is *social*, and hence, *learned*. As in the Bleuler example, no matter what is going on in your brain, or what kind of genes you have, it is impossible to have a delusion of persecution concerning Jesuits if you have never heard of Jesuits. Simple commonsensical examples like that should have sufficed to resolved some parts of the biotrope/sociotrope controversy 80 years ago! (I leave open the possibility of some *very general* and "fuzzy" psychic contents to be in the wiring diagram, rather like Jungian archetypes. When my cat dreams, making the teeth chattering sound and whisker twitching that he does in looking at a bird, I don't exclude the possibility that he may be dreaming of breaking a vole's neck, although, being an "in house" cat, he has never had that experience. Such rare qualifications on Bleuler's truism do not affect the rest of my argument here). A schizophrene is recognized clinically on the basis of a collection of signs, symptoms, and traits all of which involve learned behaviors—gestures, words, postures, interpersonal emotional responses, acculturated motives, self concepts, expectations, perceptions, beliefs, and so on.

What do we know outside of psychopathology about what human beings learn? The list includes learned perceptions, expectancies, habits, cathexes, motives, cognitions, and affects. (It doesn't matter for our purposes here whether some of these are reducible to others, although I myself do not believe they are.) Now anything which is learned is learned on the basis of a series of experiences, that is, episodes in the life history. If you are a behaviorist you will talk about conditionings, reinforcements, shapings, chainings, and the like. But you don't have to be a behaviorist to say that learning the above kinds of things takes place as a result of doing various actions (or having various thoughts) in various contexts, coexperiencing various stimulus components in context, and the like. When these behaviors, or psychisms, have been acquired by the learning process, there then exist various *dispositions* of the individual, which can be *activated*. So we say that behavior can be characterized mathematically in terms of *acquisition functions* (how the disposition is acquired) and *control functions* (how, once acquired, it is activated). All of these acquisition and control functions must have some mathematical form, whether psychologists have yet identified them or not. One learns in ninth-grade algebra that mathematical functions contain variables and constants, the latter being called "parameters." As I once tried to argue succinctly when expounding trait theory to an audience of operant behaviorists:

> Unlike the messy problem of relating trait language to behaviorese, that of genetics is, in principle, quite simple. The essential ideas can be stated in three short sentences, thus: A mathematical expression of the functional relations holding among behavioral variables always contains parameters. These parameters exhibit individual differences over organisms. Such individual differences have a heritable component. There never should have been a problem about this. Of course, in psychopathology the aberrant behavior and experience has a content, and that content is *socially learned* …. One cannot, strictly speaking, "inherit" a mental disease, any more than one can inherit speaking English. The genes provide what the logicians call *dispositions*, of various orders; and the dispositions of any order exist in varying *amounts*. Once we are clear about these concepts (from undergraduate philosophy), the rest is math and facts. The math and facts may be complicated and hard to unscramble, but there is no methodological difficulty involved. (Meehl, 1986b, p. 330)

I do not pretend to make life easy for us by suggesting we can solve the problem of schizophrenia by these general methodological principles. Given them, we have to spell out in the theoretical system just *what* disposition is genetically determined and what role it plays as a parameter in acquisition and control functions; and then we can subject these conjectures to empirical tests. But we cannot even begin to theorize correctly, design studies, or interpret data properly as they bear on our conjectures, if we are as muddled metatheoretically and methodologically as some psychologists and psychiatrists continue to be. It is a truism among historians and philosophers of science that frequently the most important part of answering a scientific question is being careful about asking it properly. It's still true that neither biotropes, sociotropes, nor the "compromisers" know how to ask the question properly.

Continuing to reason from the theoretical armchair, relying on our general knowledge of the human mind, would it not be amazing that a pathological way of experiencing and behaving, the main earmarks of which are not thinking straight about oneself and one's relations to other people, and having aberrated ways of feeling about interpersonal matters, that involve impulses, defenses against impulses, fears, and resentments—that these kinds of psychisms should have nothing to do with how one's words, actions, and feelings were dealt with by the social environment in the developmental period, or would have nothing to do with illness? Or if we allow ourselves to take some generalizations from outside the field of psychiatry, the massive and quite consistent body of evidence showing that stress, anxiety, anger, character armor, habitual modes of defense, etc., can have a pronounced influence on organic disease, on proneness to minor infectious diseases like influenza or the common cold, on asthma, high blood pressure, arthritis, gout, and apparently even cancer (e.g., Justice, 1987)—but that social pressures and psychisms have no influence on the development, course, and content of a *mental* disorder? I must confess I am baffled by clinicians who are aware that the symptomatology of even a chronic back ward deteriorated schizophrene can be influenced by relatives' visits, or by an abnormal psychology class walking through the hospital, but who are confident that social

influences have nothing to do with whether the patient fell ill in the first place. I don't mean to say that this is a strictly logical contradiction, which of course it is not; I simply express my puzzlement as to why anybody would assign the prior probabilities in that direction.

If we recognize that what is inherited is a set of dispositions, presumably mathematicizable if we knew enough by stating altered values in the parameters in the various acquisition and control functions that make up the whole range of behavior and psychisms, we can go on to say some other highly probable things from the theoretical armchair. On a dominant gene conjecture such as developed in this paper, one can infer from the MZ and DZ concordances that if there is a major locus that is schizospecific, there must be other genes that potentiate the development of schizophrenia. Because when we divide the MZ risk by two in accordance with the dominant gene theory, we get a value which is several times larger than the observed DZ concordance. Given that other genes are at work to potentiate illness in the predisposed individual, what genes would they plausibly be? Would we conjecture that they are genes for tone deafness, mechanical ability, manual dexterity, or visual perceptual speed? Obviously not. So we are confident on theoretical grounds that the genes involved as potentiators are genes dealing with motivation and affectivity and primate ethology (e.g., as found in my diagram, Figure 2, anxiety proneness, rage, sex drive, dominance, social introversion, hedonic capacity). Testing that conjecture with schizophrenia research of course requires that we have adequate measures of these polygenic traits, but we already know from animal and human research that the above list has a sizable heritability.

The most impressive recent finding on the environmental side is research on expressed emotion (EE) in the family of schizophrenes as a powerful factor influencing whether a patient in remission will suffer a relapse. I have not read all of that literature but enough to convince me that this line of work should be classified along with the soft neurology, psychophysiology, and genetic research of the last 25 or 30 years as among the most important contributions to our understanding of schizophrenia. (See, for instance, Goldstein, 1987; Goldstein & Doane, 1985; Goldstein & Tuma, 1987; Kuipers & Bebbington, 1988; Leff & Vaughn, 1985; Miklowitz, Goldstein, Falloon, & Doane, 1984; Miklowitz et al., 1986; Mintz, Nuechterlein, Goldstein, Mintz, & Snyder, 1989; Valone, Goldstein, & Norton, 1984.) The replication in several countries, despite two or three failures to replicate, and especially the remarkable size of the differences in relapse rate as a function of the families' behavior (running from ratios of 2:1 to 4:1 between high- and low-EE families) is very encouraging. While the theoretical antecedent probabilities predispose me to accept these results as powerful ammunition against the dogmatic biotrope, methodological honesty requires that I express a couple of caveats. The authors (e.g., Leff and Vaughn) are not dogmatic sociotropes and recognize the role of genetic factors in who develops schizophrenia in the first place. Nor do they assert that the influence of familial EE on the remitted schizophrene's relapse probability permits an immediate inference as to the pathogenic influence in the early developmental period, although presumably it has evidential value in that respect. (Is it reasonable a priori to believe that although 45% of MZ twins are discordant, the child-rearing attitudes and practices of the primary

caregiver in the early developmental period are not among the potent environmental determiners?) But in interpreting their data they do not consider one possibility that introduces a genetic contaminating factor. Given the highly probable statement that multiple polygenic influences are at work to potentiate the influence of the schizogene, it would be plausible that the course of the disease in a decompensated schizotype, including short-term relapse rate, would also be affected by these polygenic factors (cf. Gottesman & Shields, 1972, 1982, on severity and prognosis). Since the patient shares polygenes with the parents, there would be a correlation between polygenic influences in the patient pushing toward relapse, and polygenic factors in the parental family that could contribute to their unhealthy attitudes and behaviors. This possibility also infects an important internal analysis of the EE data, namely, that the compliant families, on intervention aiming at reducing EE, had fewer relapses than the noncompliant ones or dropouts. Obviously dropping out, not cooperating, or not being appreciably influenced by the intervention could also depend upon the adverse polygenes shared with the parents. (A biotrope colleague of mine explains the whole EE effect thus.) This line of reasoning does not presumably apply when the family involved is spouse, although one might have to worry a little bit even there about the problem of assortative mating for some of these polygenic factors. This line of criticism, which I don't believe suffices to explain the remarkable size of the relapse differences, is nevertheless troublesome. It suggests that since internal analyses cannot be conducted to test these conjectures with presently available instruments, we have to put heavy weight on the intervention studies rather than on the first round correlational studies, emphasizing the efficacy of the intervention as compared with the control group without parsing the experimental group as to compliance or measured shift in EE achieved. A final minor caveat arises from some difficulty one has in fitting the marked trends found in dichotomous fourfold table data with the very low correlations published in Leff and Vaughn's table 7.3 (1985, pp. 106–107). This troubles me, because I know from my work in taxometrics that optimizing cutting scores with small or moderate size samples can have a marked influence on the "hit rate," and it is my belief that cross validation of optimizing cuts is probably as important in any dichotomizing procedure as it is in retaining test items, or assigning beta weights in multiple regression or discriminant analysis. I repeat that my overall response to the EE research is strongly positive and confirmatory of theoretical conjectures about social influences. But I think it important, given the longstanding biotrope/sociotrope controversy, that the possible influence of polygenic overlap in the parental family as a partial contributor to the trends should be considered.

PREDICTIONS

As a good neo-Popperian I should conclude the theoretical exposition with a set of strong, risky predictions which, if not found, would constitute falsifiers of my theory; and which, being risky, would constitute strong corroborators should they turn out as predicted (Meehl, 1967, 1978, 1990a, 1990c). The most

direct and strongest set of predictions is that concerning the taxometric findings on the psychophysiology and soft neurology of the probands, and their parents, in the section above. If the neurological indicators fail to behave taxonically, the schizotaxic conjecture is refuted, because the only reason for formulating the concepts schizotaxia, hypokrisia, and Fish's pandysmaturation (see, e.g., Fish, 1961, 1975, 1977, 1984) is the presence of such "soft" neurological aberrations. If there were nothing characteristic of schizophrenes, schizophrenes in remission, preschizophrenes, or the relatives of schizophrenes except the molar-level signs and symptoms of emotional, motivational, and social experience and behavior, the schizotaxia concept would be without warrant. So when we have succeeded in identifying high-validity indicators such as SPEM, P50 and some of the other less thoroughly investigated soft neurology signs (e.g., fine tremor, Romberg, disturbance in fine motor coordination, dysdiadochokinesia), if these do not behave taxonically, we would have to conclude that they were simply phenotypic indicators having high factor loadings on some such polygenic system as conjectured by Gottesman and others.

But one must be mindful that the theory does have two components which are partly separable. Existence of a taxon, as indicated by the soft neurology, would be a necessary condition for schizotaxia to be a dominant gene, but not sufficient. It is possible that the taxometric analysis could corroborate the existence of a schizotaxic taxon as a neurologic predisposer to schizophrenia, but not its being transmitted as a Mendelian dominant of high penetrance. In what follows I presuppose that the taxometric analysis described in the section above will corroborate both prongs of this conjecture.

A neo-Popperian is not required (even by Popper) to possess instruments that would be adequate for a falsifying test, but only to say what they *would* have to be; that is, we do not want the "scientifically admissible" character of a conjecture to hinge upon the precision of existing measurement technology. (Even the Vienna Circle positivists circa 1930, in formulating the [now abandoned] "meaning criterion," distinguished between technical verifiability and verifiability *in principle*.) Unfortunately when we get past the taxometric test of the dominant gene schizotaxia part of my theory, and ask what predictions we can make regarding the SHAITU syndrome and the polygenic potentiators of schizotaxic schizophrenia, we can only say what the results would have to be like, if the theory were correct, provided adequate measuring instruments were available. At the present time, they are largely lacking. But this is not a conceptual defect of the theory, it reflects the shabby state of psychopathology as regards high-precision assessment devices. We cannot, for instance, test the polygenic potentiators of low dominance, hypohedonia, anxiety, and social introversion as potentiators, or as components of the nonschizotaxic geno-phenocopy SHAITU syndrome, without good measures of each of these as a *primary disposition*. It will not do, for example, to claim that we have "validated" the hypohedonia scale of the MMPI and then proceed to apply this in family studies, absent showing that the scale is measuring primary hypohedonia and not the secondary consequences of aversive drift (in the schizotype) or an analogue to aversive drift that presumably can occur in the SHAITU case as well.

Assuming that we had good measures of primary hypohedonia, etc., what predictions can we make on the theory as presented?

I shall only list a few of the main ones here, and refer the reader to additional predictions made in the two companion papers (Meehl, 1989b, 1990b).

Identifying SHAITU probands by the *absence* of neurologically defined patterns that identify the schizotaxon, one expects at the symptomatic level that the "true" (schizotaxic) schizophrenes will have a higher incidence of the so-called negative symptoms than would be found in the SHAITU genophenocopies. Due to the critical role of Bleulerian associative loosening in my conception, if more satisfactory psychometrics or experimental procedures for detecting subtle semantic slippage (short of clear clinical schizophasia) were available, I expect this to be present in much higher incidence among the schizotaxics than among the SHAITUs. We would anticipate that SHAITU cases have more clearly identifiable precipitating events, or a greater run of "adult bad luck" prior to decompensation, than found in the schizotaxic patients. Relatives of nonschizotaxic probands should average more polygenic factors than the relatives of schizotypes, because in order for the nonschizotype to decompensate to the point of receiving a schizophrenia diagnosis by reasonably tight standards, the required piling up of the nonspecific factors should be on the average greater than in a person strongly disposed by the presence of the major schizogene. On genetic grounds (the markedly reduced fertility of schizophrenes), we should expect a fairly common pattern to be one parent as a compensated schizotype—more frequently the mother because of the sex difference in reduced fertility of schizophrenic illness—with the other parent providing the potentiating polygenes. I do not attempt to assign differential weights to the polygenic potentiators and the present paper in any case has considered only the four that I view as most malignant, whereas the diagram in Figure 1 lists several others, such as a lack of Sheldon's mesomorphic toughness, components of polymorph perverse eroticism, lower general intelligence, and even such factors as physical attractiveness. There is no point in trying to theorize about the relative potency of the dozen I have listed, and at this stage of our knowledge it is reassuring to remember Wilks' Theorem (1938) and related psychometric truths about quasi-fungibility of additive factors (e.g., Dawes & Corrigan, 1974; Einhorn & Hogarth, 1975; Wainer, 1976, 1978).

While in the long run such empirical predictions of this sort are the ultimate aribitrators of the theory's verisimilitude, I (like the Bayesians) differ from Popper in wanting the theory to have a high prior probability since I believe that it is the empirical predictions of the theory that we want to have a low prior probability, as emphasized by Salmon (1984). Meanwhile, my personal "prior probability" on the main schizotypal potentiators being pretty much the same as the polygenic sources of the SHAITU syndrome is admittedly based upon considerations of general psychology and the psychopathology in other kinds of patients rather than specific evidence regarding these potentiators, at present largely unavailable. As I have said above, there is no reason we should forget what we as psychologists know about the mind in general just because we happen to be studying the etiology of schizophrenia. The antecedent probability is surely

high that the polygenic potentiators (which the genetic statistics show *must* be involved in who falls ill) are going to be in the "temperamental" domain of primary dispositions to the occurrence and the conditioning of such state variables, affective and motivational, as anxiety, rage, sex drive, and affiliative hunger.

Schizotaxia theory is testable via taxometric analysis of molar-level abnormalities (soft neurology, psychophysiology, perceptual, psychomotor, pandysmaturation); and such evidence is presently strong and rapidly growing, although not being optimally analyzed from the taxonomic standpoint. Whether the hypokrisic interpretation of schizotaxia is presently testable I do not know. Neuroscientist colleagues inform me that the technology of single unit stimulation and single unit response recording sometimes permits direct study of spike probabilities on different synaptic scale input patterns for simple functional systems whose wiring diagram is well worked out. The essential feature of a definitive experiment would be the graph of optional spike probabilities associated with selected input patterns (times of arrival at specified loci), the hypokrisia conjecture predicting that this graph should be (a) elevated, and (b) less peaked in the schizotaxic brain. Since ethics precludes such an *in vivo* study, one would either have to proceed *in vitro* (organ donor, brain from abortus at genetic risk), or substitute primate brain perfused with schizotaxic CFS. But as I have argued (Meehl, 1989b) this is a non-Popperian "test" which informs only when it succeeds, because of the possibility that the schizogenic locus is a recent advance in primate evolution, the "normal" orthotaxic human brain being the result of a favorable mutation.

Being uninformed, I have not touched on psychopharmacology. Guessing from ignorance, I should suppose the hypokrisia theory has weak implications in that domain, for example, schizomimetic drugs such as LSD, benzedrine, mescal, psilocybin, might be expected to heighten the excitability and reduce the signal selectivity of the neuron. Another weak prediction would be that the phenothiazines tend to stabilize the cell membrane, resisting the spread of depolarization area, or demanding a larger depolarized area to reach spike threshold. The size of this effect ought perhaps to be correlated with therapeutic potency over the set of phenothiazines chemists have concocted. In the context of discovery, it is conceivable that if such a quantitative relation obtains, reflection on how the phenothiazines' side chains differ might suggest what kind of molecule the neurochemist should be looking for that is in half-normal supply among schizotypes. Even these weak predictions are countervailed by the persistence of SPEM anomaly in pharmacological remission, a strong indication that phenothiazines are "antischizophrenic" or "antipsychotic" in their action (as many clinicians now believe) rather than "antischizotaxic."

SUMMARY

I find it impossible to summarize properly the diverse lines of argument and evidence presented above. Research advice is to liquidate the biotrope/sociotrope controversy, recognizing that what genes determine in the psychological domain is sets of dispositions—quantitative parameters of a

variety of acquisition and control functions. The genome–phenotype causal model is complex, and conventional concepts like penetrance and modifier are inadequate here. We should get away from the view of schizotypy as a kind of watered down "light case" of schizophrenia, the proper analogue being more like gout, violin talent, or poor spatial perception than like subclinical diabetes or early Alzheimer. Genetic research should focus on indicators closer in the causal chain to the DNA and less subject to social learning. Some schizotypal phenomena are direct consequences of the CNS integrative defect, while others are remote derivatives of this and have a rich psychodynamic and social content. Whatever the indicators used, the taxonomic question can only be answered by taxometric statistics. Considerations of interrater reliability and administrative necessity (e.g., third-party payment) should not deceive theoretical researchers into the naive notion that consensus diagnostic criteria, as in DSM-III, SADS, and the like, constitute "operational *definitions*" in the logician's sense, or that they are metatheoretically consonant with the powerful etiologic model of nonpsychiatric medicine, which they most certainly are not. Finally, to quote from another recent summary (Meehl, 1989b), we should never forget that the brain is at least as complicated as the kidney.

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