THE ORIGINS OF SOME OF MY CONJECTURES CONCERNING SCHIZOPHRENIA

Paul E. Meehl

UNIVERSITY OF MINNESOTA

> *Editorial note:* This chapter came to be written under different circumstances than the other chapters, as it is a slightly modified version of a personal letter from Professor Meehl to myself. Meehl was writing in response to an invited commentary that I had written for the *Journal of Personality Disorders*, concerning Meehl's (1990c) important theory paper in that journal. My commentary expressed the wish that Meehl would spell out the origins of his conjectures concerning the nature of schizophrenia. In this letter, Meehl both gives the origins of several of the conjectures in his theory paper and also explains why he does not always give such information. Happily, Professor Meehl accepted my invitation to publish his letter.
>
> —Loren Chapman

Regarding your critique (Chapman, 1990) of my schizophrenia paper (Meehl, 1990c) in Millon's journal, of course I took no umbrage at your comments, nor those of the three other commentators. There is surely no point in asking four distinguished scholars to critique a paper unless one expects them to raise questions, objections, and requests for clarification. This is especially true for myself as a neo-Popperian. If we conceive of science as the history of mistakes and the advancement of science as the corrections of mistakes, any theorist who cannot tolerate tough objections to his conjectures should get out of the business. I threw out an awful lot of conjectures in that paper, and I would be surprised if more than half of them turned out to be substantially correct; and, given the field we are working in, it would be surprising if more than a few of them turned out to be wholly correct!

It did occur to me in reading your critique that I might have offended you by the strong position I took regarding the impossibility of settling a taxonomic question by conventional correlational, or what I call "high-versus-low," statistical analyses. I perhaps should have explicitly stated, at that locus in my paper, that the reader should hark back to previous material (such as that on pp. 45-46) on screening indicators of schizotaxia; the obvious necessary step in such screening of candidate indicators consists of conventional correlational, or "high-versus-low," analyses of the data. After all, every item on the scales of my favorite personality test, the Minnesota Multiphasic Personality Inventory (MMPI), was so identified by Hathaway and McKinley. Or for that matter, in the Golden-Meehl (1979) paper on schizotaxia and MMPI items, although the main point of that paper was bootstrap taxometrics using my methods, the *first step* was to carry out a traditional item analysis using diagnosed schizophrenes in the files as a criterion group. I find it hard to imagine any other way to get started, and I certainly would not complain against any of your important research on schizophrenia, including your high-quality efforts to objectify my checklist, on

the grounds that you proceeded in the usual high-versus-low method. That's the best way to get our foot in the door, and it's hard to think of any other way to go about it.

The point of my criticism of traditional (nontaxometric) studies was that *after* one has identified an indicator by the conventional high-versus-low method, one may (optionally) go on to inquire whether what is being discriminated by the identified indicator is a *factor* in the quantitative sense (such as general intelligence or introversion or mechanical ability) or a *taxon* (such as would be produced by a major locus mutation). Once that question has been raised, one must apply statistics appropriate for the detection of taxonic structure, and my argument was that conventional correlational or discriminating statistics are not adequate to *that* task. I do not have the impression from your critique that you disagree with this, but if I misunderstand you in this regard, I would be interested to know your objections.

Reflecting on your response to that argument in my paper, I also realized that I have to amend it a little bit to cover a certain possible although extreme case. Let us imagine that we have a quantitative indicator that has very high construct validity for a latent taxon, so high that when we study a mixed population in which the taxon base rate is nonnegligible in size, the indicator gives us a bimodal (or even a U-shaped) distribution of scores. And now suppose we are lucky or clever enough to find a second indicator that acts the same way, and then maybe a third indicator, which is not quite that good but which yields a distribution that shows bitangentiality, or perhaps just considerable skewness or platykurtosis. These different distribution features are only marginal as evidence of taxonicity, because we know they can be generated in various other ways, especially by bad luck in the distribution of item difficulty levels in relationship to interitem phi coefficients. But if we pick out the individual subjects who are high and low in the first bimodal or U-shaped curve, and we then discover that they are overwhelmingly the same ones who are high and low on the other two indicators, it would be pretty strong evidence of taxonicity, and it does not involve any such complicated taxometric methods as the ones that I have devised. One can think of it graphically in a situation where no statistics at all would be involved, and one could do it simply by inspection. Suppose I have an indicator 3-space and we find a big swarm of points representing the subjects like a star cluster at the origin, then an almost empty region in the space, and far out in the northeast corner a little clump of data points; it would be pretty safe to conclude that we were dealing with a taxon. However, over the past 70 years, going back, I believe, to the 1920s, psychologists interested in dichotomizing types of schizophrenes, either psychometrically or by experimental procedures or ratings on traits and life history, have tried to test the "two types" conjecture by the simple high-low method. My contention is that, lacking such gorgeous separations as in the above example (and nobody manages to achieve such separations), the high-low method is simply incapable of answering the taxonic question.

Now let me say something about your basic question "Why does Meehl think that…?" about the list of conjectures that you mention in your critique. Let me explain my expository dilemma in writing that paper. Despite Millon's kindness in being willing to devote an entire issue to me and the four critics, I was conscious of the fact that I was really pushing the limits of space, and I rather expected him to say I would have to cut it down some, which pleasantly did not happen. I do not know whether the commentators were held down to fewer pages than you might have liked, but it wouldn't surprise me to learn that. As you say, my Popperian approach to scientific conjectures means that I do not require

affirmative "inductive support" as a sort of justification (we neo-Popperians don't believe in justification!), but that doesn't mean that the reader is satisfied with a bunch of conjectures with little or no hint of what gave rise to them in the theorist's mind. My neo-Popperian orientation in philosophy of science does not, of course, forbid me to sympathize with that curiosity in a reader (as in your p. 111, third paragraph).

But consider my situation. With limited space, I had a choice between two approaches. I could have confined myself to a much smaller list of conjectures and suggested research projects, each explained in detail as to its context of discovery. What I have learned is that psychologists, including those who are somewhat familiar with the Popperian line, nevertheless find it difficult to adopt a Popperian stance in the concrete. As David Faust pointed out in a recent conversation, the intellectual tradition of psychology is so strongly inductivist that even if one introduces a set of conjectures by a Popperian preface, as soon as you give a smidgeon of plausibility considerations for a conjecture, the reader's mental set switches away from Popper, so the reasons you give, however stoutly you emphasize that they are merely plausibility considerations, *tend to be treated as if you thought they were knock-down arguments, which they are not*. Thus the pedagogical effect of giving a couple of mere plausibility reasons for a conjecture is counterproductive because the reader tends to treat them as if the theorist thought they were *proofs* that his conjectures are correct, which of course he didn't claim in the first place. I have learned by experience that a *brief summary* of plausibility considerations is almost invariably dealt with in this way, even by persons who are bright and charitable, so it puts one in the position of maintaining, as if proved, something that one certainly did not prove or attempt to prove. The brevity of such a short list of mere plausibility considerations has, in my experience, the effect of looking either thin or dogmatic, or both. So I make a practice of saying loud and clear that they are conjectures and nothing but conjectures, and they are now candidates for theoretical criticism and empirical test. Since I had resolved that this was my last longish paper on schizophrenia theory, I thought it preferable to record as many conjectures as I have come to over the years, either from purely theoretical speculation, or clinical experience, or quantitative research, on the theory that all kinds of researchers would be able to pick them up and test them. It seemed to me a more worthwhile contribution to science, given the premise that I am not going to write any more about this until we have more empirical evidence (as we are now collecting here at Minnesota in the framework of my theory), to suggest lots of things to study than to present only, say, one-tenth as many but those presented being "defended." The resulting set, while understandably frustrating to a critical reader, was not carelessness or dogmatism on my part but sprang from a conscious decision concerning which of two expository policies would be more fruitful in the long run.

To give you an idea of how much longer the paper would have become had I tried to explain the rationale of each conjecture *even somewhat dogmatically*, let me take a couple of those conjectures that you found baffling because I didn't explain their origin. I should preface this by saying that I do not accept Popper's view that the scientist should prefer low-probability theories for efforts to falsify. In this respect I am a Bayesian rather than a Popperian, and I have never met any scientist in any field who thought it was a good thing to start with highly improbable theories. But Popper's meaning of antecedently improbable is a logician's meaning; he certainly did not mean that you should prefer theories that are antecedently improbable *on the basis of being inconsistent with received background*

knowledge. I think it is universal among scientists to prefer theories that are antecedently probable on the basis of background knowledge. Where they look for the improbability is in the empirical facts derivable from the theory, which facts would be antecedently improbable if one did not have the theory in mind. Two recent papers (Meehl, 1990a, 1990b) deal with that subject in a way I think is closer to the practice of working scientists than what some philosophers of science have taken Popper to be saying.

Although I accept Reichenbach's distinction between the context of discovery and the context of justification, that doesn't mean that there is no rationality involved in the discovery phase. On rereading Reichenbach, I don't know whether he meant to imply such a thing, but if he did he was obviously wrong. But despite the occurrence of "reasonable considerations," theoretical and empirical, in the context of discovery, one must be careful not to make the same demands of such considerations in the context of discovery as would be appropriate—setting higher standards of proof—in the context of justification.

Now for a couple of examples. I made the conjecture that among carefully diagnosed schizophrenes, accurately diagnosed by present criteria (e.g., DSM-III), around 85% would be schizotaxic as predicted by my theory, and the other one-sixth or one-seventh or so would be nonschizotaxic. You wonder where that number came from, and I dare say so did every other reader. Context of discovery reasoning: Because the smooth pursuit eye movement (SPEM) anomaly looks pretty good on present evidence and because it fits very nicely my list of good armchair properties of a candidate indicator (Meehl, 1990c, pp. 17–18), suppose I conjecture that—absent drug effects, advanced age, organic brain damage, or psychosis when tested (as in manic depressives apparently)—it is a quasi-pathognomonic indicator of the schizotaxic brain. Relying partly on the research literature but admittedly more heavily on some of my local experts (Clementz, Grove, Iacono), I took as a rough figure that by the best methods and the best cutting score one could detect around 75% of schizophrenes, including schizophrenes in remission, at the expense of around 10% false positives in the control population. As argued elsewhere in the paper, on the dominant-gene conjecture every schizophrene must have a schizotaxic parent, and since only 10% have a schizophrenic parent (putting it another way, only 5% of the parents are diagnosable schizophrenes), the clinical penetrance of the schizogene *for diagnosable disease* is only around 10%. The lifetime morbidity risk for schizophrenia in the general population is 1%; therefore, it would follow that the incidence of schizotaxics in the general population is 10%.

That's a rather nice coincidence; so suppose I further conjecture that the 10% false positives among controls are pseudo-false positives, that is, they are schizotaxics who have remained compensated. Continuing with the daring conjecture that the indicator is quasi-pathognomonic (with suitable exclusions), what about the 25% of carefully diagnosed schizophrenes who do not show the SPEM anomaly? Nobody claims that even Endicott and Spitzer (1978) using SADs can have better than a 90% confidence in the diagnosis of schizophrenia, so I conjecture that 10% of the schizophrenes in the research studies are wrongly diagnosed *by extant phenotypic criteria*. But that doesn't explain why we get only 75% SPEM positives among them instead of 90% SPEM positives. The obvious explanation would be that, distinguishing between schizotaxic schizophrenes and nonschizotaxic schizophrenes, we have to knock that 90% down to 75%, and the resulting number for schizotaxic schizophrenes is around 85%. Therefore, the remaining 15% are *clinical schizophrenes, correctly diagnosed but not of schizotaxic etiology*. Keeping in mind that

we are operating with conjectures and in the context of discovery, the reasoning seems quite straightforward, but of course all of the assumptions might be false.

Then you ask how I arrived at the non-specific components of the SHAITU (Submissive, Hypohedonic, Anxietous, Introverted, Traumatized, Unlucky) syndrome. That also seems to me fairly straightforward given other information about the disorder. We know from looking at the monozygotic (MZ) and dizygotic (DZ) risks that on my theory, there have to be some other genes than the dominant schizogene, which it is plausible to conjecture are polygenic. Then we ask what temperamental traits are known to be highly heritable and would plausibly be potentiators of schizophrenia in a schizotype. Now obviously, one is not going to pick out something like mechanical ability or manual dexterity or tone deafness as a likely potentiator. The two big facts that lead us to identify a clinical schizophrene are the cognitive slippage and the breakdown of normal interpersonal relations. If persons with a schizotaxic brain decompensated only in the direction of exacerbated soft neurology (e.g., if the episodes of "illness" consisted of tendencies to lose one's balance or fall down stairs instead of merely having a subclinical Romberg as detected by Eysenck and the old Worcester group), we would not label schizophrenia a *mental* illness, because it would not have any "mental" symptoms.

When we contemplate the expressed emotion (EE) research on relapse caused by the return of patient in remission to a hostile, critical family environment, or when we ask what is it that laypeople usually mention as descriptive of a schizophrene who commits a senseless murder (they say things like "a loner," "sort of shy," "kind of strange with people"), and we consider the phenomenology and inferred psychodynamics of the schizophrene, it seems rather obvious that polygenes for social introversion, polygenes for the anxiety experience, polygenes for pleasure deficiency, and polygenes for low social dominance— all traits that we have evidence are heritable in humans and some other mammals—are obvious potentiating candidates. If you can think of other basic temperamental dimensions of personality that (a) are known to be heritable and (b) whose psychological nature would seem highly relevant to whether a schizotype falls ill, I would be interested to know which ones they are. You will recall that in the text of the monograph I did consider adding something about rage, and I could just as well put it in as leave it out. So that's the basis of the inherited components of the SHAITU syndrome. Each of those is researchable by sufficiently complicated family studies given adequate measures of these temperamental traits.

How about the T (trauma) and U (unlucky) of SHAITU? Well, I start with the fact that there is only 55% concordance for MZ twins. The 45% discordance shows that something "environmental" must be at work to make the difference between the twin who falls ill and the twin who survives the morbidity risk period without decompensating. It seems natural to divide the whole list of environmental factors into two broad classes, the first being events occurring in the developmental period, and the second being current environmental stressors. Now here I rely more on theory than you would perhaps consider legitimate, but I remind you again that we are in the context of discovery. Because clinical schizophrenia is—whatever else it is—a grave malfunction of interpersonal relations, I find it almost impossible to conceive that the social reinforcement schedule, positive and negative, provided by the primary caregiver in the early developmental period of a schizotaxic organism could be *utterly devoid of effects*. Therefore, I am still betting on the old schizophrenogenic mother theory having a little bit of truth in it, provided we get adequate

measures and have statistics suitable for unscrambling the components of causality. It would seem strange if the probability that a remitted schizophrene will suffer a relapse is higher when he returns home to a battle-ax mother but that same battle-ax mother's child-rearing practices and subtle attitudes toward the child were without influence. I admit that it is conceivable, but I think it extremely improbable that this should be so.

Under the heading of T, I specifically indicated that either a major trauma or a long series of minitraumas would do the job. There I admit I am relying on my therapeutic experience and that of other clinicians: one has the impression that such things as sexual abuse by a stepfather are conducive to adult neurosis or psychosis, and it makes theoretical sense if you have a schizotypal makeup to begin with. Whether a long series of mini-traumas by a battle-ax mother is more potent than one big trauma, such as rape or seeing a beloved sibling killed or whatever, I don't believe I offered any speculation and I don't have an opinion. But the big point is that *from the discordance data we have to put some causes in the environment*, and if we don't believe that it's the effect of witchcraft or sunspots or some virus or a diet deficient in zinc or whatever, doesn't it seem plausible to look to the things that happen to one as a child in the family and then in the peer groups in school, and so forth? Where else in the causal field would one look if not to these kinds of happenings?

As to the last letter in SHAITU, the U for "unlucky," see the discussion of luck in my "two knights" paper (Meehl, 1978). I admit that there I am relying mainly upon theory and clinical experience. As an extern at one of the first case conferences I witnessed at the University of Minnesota Hospitals when Drs. Hathaway and McKinley were running them, I recall a schizotype who, although known by his relatives and the people in his small town community as "a somewhat odd person," "a loner," and the usual set of adjectives, had nevertheless been free of discernible mental illness until late in the morbidity risk period, I think around age 43 or so. The story was that he and his older brother, the latter apparently a normal person, inherited the farm from their parents, who had died rather early. The older brother made all of the decisions and all of the major interpersonal contacts required, and the patient did routine chores as per instruction or habit, although at times he did go into town to make various simple purchases, and then he would occasionally have a beer with one or two people he had come to know and like. But then brother died suddenly of a coronary attack, and our schizotype was left to "run everything," including both the work planning and the various human interactions demanded. Within a couple of months, he fell ill with a florid paranoid schizophrenia. I remember Dr. McKinley pointing out the dif-ficulty of forecasting in psychiatry, in the sense that the brother's death of coronary disease was an event that you could not predict by any kind of psychological study of the patient, it being one of what Paul Horst in the classic (and currently neglected) *Prediction of Personal Adjustment* (1941) calls "contingency factors," happenings in the environment that it is not possible to anticipate (even with probability) by any study of the patient and in most cases by any study of the environment. McKinley pointed out that, having functioned at least marginally and without the development of a clinically diagnosable illness until the late age of 43, the chances were very good that had the brother not had a coronary attack this man would never have developed schizophrenia.

But I am not relying solely upon theory and clinical impressions in speaking of bad luck episodes. There is a book, almost completely ignored by psychologists and psychia-trists: *Mental Illness and the Economy* by the Johns Hopkins sociologist M. Harvey

Brenner (1973), who analyzed employment statistics in private and public New York hospital admissions in a much more sophisticated way than anybody ever has before or, I believe, since. Proceeding by the methods of economists, taking out secular trend and doing a Fourier fit to data from before World War I to the late 1960s, he shows very large correlations, from −.60 to nearly −1.00, between industrial employment (as the single best indicator of macroeconomic performance) and mental illness. I don't discern anything wrong with his material, and I have not heard of any criticisms of it, and so I incline to think that he proved the point, a point that has been in dispute among us for a long time. There are the usual alternative hypotheses available, which I believe he does a satisfactory job of answering. Thus, for instance, if it is said that people who are "always a little crazy" go into hospitals in bad times because somebody gives them three square meals, it doesn't explain why Brenner gets the same strong results when he analyzes the private mental hospital data separately, that being more expensive for the family than going to a high class hotel! From the theoretical standpoint, keeping in mind our background knowledge of powerful environmental effects to explain the concordance data, how could a psychologist who *knows* that there are strong environmental effects and who *knows* from the EE studies that psychological stress in the home is potent, plausibly believe that losing one's job (or leaving one's job or living in chronic fear of losing one's job) would be without influence? I favor using a little common sense in these matters. If we already know there are environmental factors from the genetic data, and if we know specifically one kind of social vector is powerful—the EE study showing that relapse rate can be boosted by as much as 4 or 5 to 1 depending on how the family treats you—how can a psychologist suppose that economic stress would be without effect upon the probability of decompensation?

Of course, there is another kind of "bad luck" that cannot emerge in studies such as Brenner's, an idiographic sequence of events that have a peculiar meaning that would not be the same for another schizotype under the circumstances, such as I set out in my Afterword to Gottesman and Shields' first book (Meehl, 1972, pp. 404–405). See also the idiographic causal sequence in the diabetic/psychodynamic example (Meehl, 1973, pp. 220–221). As I have pointed out in several papers, we ought not to assume that the only potent environmental factors are those found in our standard social science list of variables, such as social class, broken home, race, rural/urban, education, and the like. But I hope this explanation is sufficient to show you where I got the components of the SHAITU syndrome. It is conjectural, based upon a mix of theory and clinical impressions and quantitative studies, certainly not whimsical or arbitrary on my part.

If we take the long list of conjectures that I offered and that you asked about in your critique—"Where did this one come from in Meehl's thinking?"—imagine how many additional pages if we multiply these pages it has taken for me to deal with three of them by a factor of 10 or more (you mention a half-dozen). The whole thing would have gotten completely out of hand; it would be a book rather than an issue in Millon's journal. As I say, I might have adopted a different policy and explained half a dozen of the conjectures and omitted the others, but I still think it makes a better contribution to science to put them all down so that people interested in researching the theory can choose cafeteria-style from the ones that strike them as most plausible and most fun to investigate empirically.

I hope this gives you some cognitive satisfaction, and I trust you won't mind if I circulate it among some locals who regard both you and me highly and who will find it of scholarly interest.

REFERENCES

Brenner, M. H. (1973). *Mental illness and the economy*. Cambridge, MA: Harvard Univ. Press.

Chapman, L. J. (1990). Meehl's theory of schizotaxia, schizotypy, and schizoprenia. *Journal of Personality Disorders*, *4*, 111–115.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The schedule for affective disorders and schizophrenia. *Archives of General Psychiatry*, *35*, 837–844.

Golden, R., & Meehl, P. E. (1979). Detection of the schizoid taxon with MMPI indicators. *Journal of Abnormal Psychology*, *88*, 217–233.

Horst, P. (Ed.) (1941). *The prediction of personal adjustment*. New York: Social Science Research Council, Bulletin 48.

Meehl, P. E. (1972). A critical afterword. In I. I. Gottesman & J. Shields, *Schizophrenia and genetics: A twin study vantage point* (pp. 367–416). New York: Academic Press.

Meehl, P. E. (1973). *Psychodiagnosis: Selected papers*. Minneapolis: Univ. of Minnesota Press.

Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, *46*, 806–834.

Meehl, P. E. (1990a). Appraising and amending theories: The strategy of Lakatosian defense and two principles that warrant using it. *Psychological Inquiry*, *1*, 108–141. Meehl's Reply to the commentators. *Psychological Inquiry*, *1*, 173–180.

Meehl, P. E. (1990b). *Corroboration and verisimilitude: Against Lakatos' "sheer leap of faith"* (Working Paper, MCPS-90-01). Minneapolis: Univ. of Minnesota, Center for Philosophy of Science.

Meehl, P. E. (1990c). Toward an integrated theory of schizotaxia, schizotypy and schizophrenia. *Journal of Personality Disorders*, *4*, 1–99.