

TO: Dr. Norman Garnezy

FROM: P. E. Meehl

DATE: March 10, 1965

RE: Collecting MMPI profiles on parents of your schizophrenic sample

[This is by way of preparing for a luncheon conversation, as you suggested last week, regarding the desirability of getting MMPI profiles on the parents of the schizophrenic sample you are about to begin studying intensively from the standpoint of family dynamics and the patient's developmental history. Needless to say, I do not expect you to have any particular sympathy for the substantive views here propounded, but since the parents will be coming in for study anyway, the extraction of an MMPI is relatively painless and costs almost nothing to the research project budget, I would be interested in the question whether your psychometric expectations would be distinguishable from my own; or whether, as I fear, this is one of those typical situations where the constitutional and the social-learning emphases do not lead to differential predictions as to the facts. I am not clear as to what your expectations would be, and, as you will see, I am rather tentative at this point regarding my own.]

1. By now you are pretty well acquainted with my own speculative theory about the determiners of schizophrenia, so I can go over them in a hurry. Briefly, I assume that there is a specific mutation which provides the precondition for the development of true schizophrenia, although of course we have to recognize the likelihood of plausible phenocopies as we do in other branches of genetics. As the neurologist now says "No spirochete, no paresis," I am betting "No mutated gene, no [true] schizophrenia." On the other hand, I assume the influence of polygenic determiners with respect to a set of potentiating variables, chief among which I would mention basic "temperamental" parameters of anxiety-readiness, rage-readiness, sexual drive, energy level, and a kind of Sheldonian dimension connected with mesomorphy and general "toughness." I do not view these potentiators quite as modifiers in the geneticist's sense, since I take the first stage in the chain of causality to be the schizotaxic phenotype and this, as such, does not have any personality or social content plugged into it. The only sense in which these polygenic potentiators are contributors is that they alter the probability of the individual developing a clinical psychosis or a diagnosable pseudoneurotic schizophrenia. And that is, as I view it, a rather different thing from what a modifier gene is conceived to do by the geneticist. Another way of saying this is that I cannot view the ideas of expressivity and penetrance as directly applicable, at least in their usual genetic meanings, to the schizophrenia problem. Nevertheless, when we are dealing with concordance statistics of the usual sort, or when we are beginning with a sample selected by virtue of the presence of clinical schizophrenia, we have to think in terms of something that is statistically analogous (i.e., in its role, formally) to expressivity or penetrance, in the sense that only certain combinations and amounts of behavioral dimensions will lead to one person being formally diagnosed as schizophrenia. The closest analogy would be, say, a gene or set of genes determining a taste-parameter or Olds<sup>+</sup>-center parameter for "how good sugar tastes." This has no influence within the causal chain from the specific diabetes gene → Isles of Langerhans → deficient insulin manufacture → aberrated carbohydrate

metabolism → hyperglycemia → clinical diabetic coma. But it alters (“psychologically”) the probability of sugar ingestion, hence is a potentiator of diabetic coma. In the behavior field, I think we need a special term, other than ‘modifier’, for such situations.

The importance of aversive learning in schizophrenia renders it practically certain, so far as I can see, that individual differences in the polygenic loading of anxiety-readiness will be among the most important factors influencing whether or not a person who carries the specific genetic condition (and, hence, is schizotaxic) will develop schizophrenia. As you know, I assume that any ordinary regime of social learning, when imposed upon a schizotaxic individual, will result in the development of a schizotypic personality make-up. That non-schizotaxics can be made (e.g., by LSD, isolation procedures) to behave “schizophrenically” does not concern me much, for two reasons. First, production of some schizophrenic-like phenomena is not equivalent to production of a phenocopy. As I understand the semantics, a phenocopy cannot be distinguished, phenotypically, from the genuine article. Do you or I believe that, given a week’s time to observe, test, interview, get a history [!], we could not discriminate an LSD-intoxicated subject, or an isolation-experimental subject, from a case of schizophrenia? I certainly don’t believe this, and I daresay you don’t either. Secondly, I point out that even if an indistinguishable phenocopy could be produced “experimentally” in non-schizotaxics, this does not refute my theory that all actual schizophrenics are schizotaxics, i.e., no nonexperimental regime produces such phenocopies. This is why I emphasized in my APA speech the matter of “actual existing regimes.” If you could produce a perfect phenocopy of schizophrenia by rigging the social learning optimally (which I doubt), I would ask whether any actual regime, arising naturally rather than as an experiment, is this “bad.” Diabetic comparison: We inject massive glucose intravenously at such a rate that nobody can mobilize insulin adequate to handle it. Thus we show that “anyone can get a diabetic coma.” This does not, obviously prove much against a genetic theory of diabetes; because nobody except an “experimental subject” has such an “environment.”

The difference between schizotypy and schizotaxia is a difference in level of analysis or, if you prefer, in the amount of socially acquired behavior content involved. It is also, of course, in the matter of which condition “comes first” developmentally. But it is not a difference formulable statistically in terms of subpopulations, since I am for the moment hypothesizing that all schizotaxics inevitably learn to be schizotypes, and all individuals who are truly schizotypic become so on the basis of a schizotaxic defect. The terms differ in intension; they do not differ in extension. Therefore when considering the identification of sub-populations, the important distinction is not between schizotaxia and schizotypy, which are in one-to-one correspondence according to my hypothesis; the important distinction statistically is between schizotypy per se and its degree of compensation or decompensation. I believe that the majority of schizotypes remain compensated throughout life and that less than half—my present bet would be a considerably smaller fraction than half—of schizotypes ever decompensate to the point that they would be diagnosable as clinically schizophrenic even if studied psychiatrically, which most of them aren’t. I do not see how anyone with even moderate therapeutic experience with schizotypes could have any doubts about this question. One sees many individuals who develop short-term micropsychotic episodes, and who are psychometrically “schizophrenic” on the MMPI, who 95% of the time are non-psychotic, and whose gross actual or potential pathology is not known to anyone except the therapist. Last time we discussed this, you laughingly

commented that “everyone is schizotypic, according to Meehl.” No, sir. In my private practice sample 1950-60 I estimated about half schizotypes, half not. But the “huge national estimate” you alluded to needn’t distress you. What’s so implausible about it? Millions more Americans carry genes for gout or diabetes or otosclerosis than ever get these diseases at a clinical level. Why do you think it strange to postulate that millions carry the schizogene? After all, according to your views, we all carry schizogenes, right?

2. Whether, and how soon, and how badly, a schizotype decompensates depends partly upon the genetic background within which the schizogene finds itself, but also upon the social learning regime to which the individual is exposed. I suppose that even here you and I would have some degree of disagreement since I would be inclined to think that the constitutional parameters of temperament, particularly the anxiety-parameter, play a more important role in producing individual differences than the reinforcement regime; whereas you, while readily admitting the importance of inherited differences in anxiety-readiness, would attach the major role in variance-generation to the social learning reinforcement schedule. Leaving these quantitative emphases aside, however, where we can agree is in our belief that the social learning regime has a material influence upon the probability of schizophrenic decompensation, and that the most important source of these individual differences is the primary provider of rewards and punishments in the early developmental period, i.e., the mother.

3. As you know, I consider 90% of studies on the parents of schizophrenics to be useless for purposes of deciding between the two theoretical emphases in etiology, for the reason that the findings can be predicted in advance on either theoretical ground. In the old days, when psychologists were hereditarian in their biases, the fact that stupid or crazy people tended to have stupid and crazy relatives was taken for granted as indicative of genetic determiners; today, when the bias of social scientists is strongly in the other direction, the presence of stupid or crazy relatives is taken as evidence of the influence of social learning. The methodological point is, of course, that such data are equally understandable, and would be equally predictable, on grounds of either theoretical position. Furthermore, until the theories are tightened up to the point that they permit the making of reasonably exact predictions, preferably point-predictions but at least range predictions, a “mere significant difference” in the direction predicted by theory is almost useless. As you know, I take a rather hard line on this methodologically, since I view most theory corroboration in social science today as based upon an inadequate understanding of the problem of corroboration in science. I hold that psychologists have fallen into a naive kind of procedure for theory-testing which is in some ways almost exactly the reverse of that which is employed in the more advanced sciences. (I can only refer you to the writings of Karl Popper in support of this thesis.)

The problem with regard to parents is, therefore, that once we have rejected “diagnosable, clinical, florid schizophrenia” as the appropriate concept—which I believe everybody does who has any sophistication in these matters—we are left without any good way to evaluate the pathological family constellation from the standpoint of choosing between, or of weighting, the two major theoretical emphases. Those kinds of behavior on the part of parents which can be assumed to be adverse as a social learning environment are also kinds of behavior which are evidential of a bad genetic constitution in those same parents.

The kind of causal model which I am betting on involves the notion that the schizo-

typic child has a greater chance of becoming clinically schizophrenic if he inherits heavier polygenic loadings from either/or both parents which render him more anxious, more rageful, extremely high (or extremely low) in sexual drive, low in energy level, low in “mesomorphic toughness,” and so forth. But given any fixed level of these polygenic determiners, the probability of his developing a clinical schizophrenia will be a function of his life experiences and particularly of the degree and kind of pathology found in the social environment of the primary group. The more pathological the mother or father, but particularly the mother, happens to be, the greater will be the statistical probability of his becoming diagnosed and therefore of his belonging to the sample of proband with which your present investigation commences.

However, it is not just a question of “general pathology,” which you would be the first to insist upon methodologically. Furthermore, the evidence is pretty clear that schizophrenia—and I assume therefore schizotypy—has not only a significant but a very pronounced effect upon fertility, so that in a sense one may view the present schizophrenic offspring as being, so to say, “exceptions,” i.e., they are produced by parents who on the average ought not—given a genetic view—to have produced offspring. The easiest approach to this statistically is to assume that a schizophrenic proband receives the specific gene from one parent and the potentiator genes from the other parent. That is to say, members of the preceding generation who carried both kinds of genes, i.e., both (1) the schizogene and (2) the potentiators for anxiety, rage, low energy level, and so on, are less likely to become parents than the members of that parental generation that carry the schizogene alone without the other potentiators, or the members of that generation who carry the potentiators alone without the schizogene. This seems to be an almost immediate consequence of the genetic model I am proposing. Putting it crudely, we might imagine that the “typical schizophrenic” (as distinguished from the typical schizotaxic who is not typically schizophrenic) has received the schizogene from one parent and the potentiating genes from the other parent. This means that one of the parents should be a schizotype, although usually well compensated (otherwise he would be much less likely to have offspring), and the other parent should be a non-schizotypic neurotic. Does this make sense to you?

4. Should we expect a symmetry between fathers and mothers in this respect? I have to admit here, in all frankness, that I am engaging in a bit of ad hoc theorizing. I have been very impressed as a psychotherapist with the same clinical observation that has led to such a strong current emphasis upon the social learning influence of the schizophrenogenic mother, namely, that schizophrenic patients uniformly—I am ready to say without any clear exceptions in my experience—have negative reactions to their mothers; and in most, although not quite all instances, are also able to report behavior on the part of the mother which (even when corrected for a considerable amount of distortion by the schizophrenic patient) indicate that mother was a pretty looney person.

5. Suppose one accepts Ian Gregory’s conclusion that none of the proposed genetic models fit, but then goes on to argue, as I do, that we shouldn’t expect them to fit because what is inherited is schizotaxia-schizotypy (all cases of one being cases of the other even though there are different levels of causal description involved) whereas the published statistics deal with formal diagnosis. Secondly, one argues that a model which does not take into account (a) the lowered fertility and (b) the sex difference in the impact of

schizophrenia upon fertility, will be inadequate; and, finally, perhaps most importantly (at least from your point of view), one emphasizes a marked potentiating effect, socially-mediated, on the likelihood of the schizotype developing clinical schizophrenia if mother—as compared to father—is the schizotypic parent; then we can start afresh and try to concoct a model which will do justice to these plausible relationships.

6. Adopting this more complicated causal scheme, which I feel is practically forced upon us even by our present knowledge (so that anything less complicated will almost certainly be incapable of fitting that data), I am still inclined to opt for the simple theory that we are dealing with an autosomal dominant of complete penetrance. I emphasize again that I am here referring to penetrance in the strict sense of a phenotypic consequence of the genotypic condition, which in this case does not mean clinical schizophrenia but schizotaxia. Schizotypy is the learned system of reactions, following as a result of a variety of reinforcement regimes, and if it is possible to develop a behavior pattern indistinguishable from schizotypy in the absence of schizotaxia we would properly conceive this as a phenocopy. As stated above, I am inclined to doubt that a genuine duplication of schizotypy can occur in the non-schizotaxic neurotic since, while the interpersonal aversiveness and other social aspects of behavior can occur there, my own clinical experience suggests that the characteristic forms of cognitive slippage and body image aberration are absent in the neurotic even when his interpersonal aversiveness is very strong. Thus I agree with Bleuler's original conception that there is a schizoid thought-disturbance which, however subtle at times (and therefore often missed on short-term psychiatric study), is nevertheless unique to schizophrenia. And, of course, I readily admit that the problem of a specific psychometric device for schizoid cognitive slippage has not as yet been solved.

My more "scientific" reason for sticking (until further notice) to the dominant gene guess is a single statistic which seems to me of very great importance even though it is based upon formal diagnosis. Let us assume that, at least in most instances, a schizotypic parent is as bad (or worse) than a "neurotic" or other form of "schizophrenogenic parent," and that a priori a diagnosably schizophrenic parent should be, in general, still worse. Suppose we assume further that a pile-up of the polygenic potentiators of schizophrenia mentioned above will be statistically more difficult to avoid in the offspring if both parents have a piling up of such in the adverse direction. That is, the zygote gets potentiators from both parents. Then both on genetic and on social learning grounds, the schizotaxic child of two clinically schizophrenic parents should be expected on the average to have just about the worst possible condition for being preserved from psychotic breakdown. That is, the offspring of two clinically schizophrenic parents, if he receives the schizotaxic gene, will not have the usual opportunity to get a "chance assortment" of potentiator genes for the other temperamental variables. Then, in the family circle the mother will present him with an adverse learning regime (I assume that the kind of counter-transference strain that most of us experience in dealing with the schizophrenic and even the compensated schizotype will be reflected in a similar kind of phenomenon, including the double-bind business, when such a person is in the parent role). Furthermore not only will the father fail to "protect" such a schizotaxic child from the schizophrenogenic regime imposed by a schizophrenic mother, but he will typically add to it by presenting ambivalence and double-binds and testing operations and the like arising from his own neurotic pathology. Setting for the moment the qualifications that

would have to be made on this generalization (such as the possibility that clinical psychosis in the parents would lead to the child being frequently nurtured by other figures free of psychopathology), I take it that having two schizophrenic parents with whom one lives a fair share of his childhood in adolescence involves a piling up of just about the worst circumstances, both in terms of the statistical probability of receiving a heavy loading of adverse polygenic determiners and also an adverse environmental regime. On the average, schizophrenic parents will be those who in addition to being schizotaxic also have adverse polygenic determiners in their own genotype. This being inferred from the fact they are clinically schizophrenic rather than compensated schizotypes, the latter being the usual state of affairs.

Now suppose we assume that this extremely adverse combination of polygenic potentiators and schizophrenogenic family milieu raises the probability of developing a clinical schizophrenia in the offspring near to its maximum, on the average. That is, we assume that if this combined polygenic-environmental adversity is present and the child is schizotaxic, he is practically certain to become clinically schizophrenic. That would mean that if the schizogene is recessive and completely penetrant, nearly 100% of the offspring of two clinically schizophrenic parents should be schizophrenic, which is false. If, on the other hand, we were to assume that the schizogene is a dominant of complete penetrance, and that the heterozygote is normal, we would expect the incidence of diagnosable schizophrenia in the offspring of two diagnosed schizophrenic parents to be a little less than 75%. Now, the observed incidence of schizophrenia in the offspring of two schizophrenic parents is .68 (Kallman data). Since this is close to, and slightly under, the theoretically presumed incidence of the dominant gene in the offspring, it is consistent with the theoretical model.

The importance of sexual frustration, sexual ruminations, and chaotic sexuality in the schizoprene leads me to suppose that among the polygenic contributors, along with a heightened anxiety parameter, a heightened rage parameter, low energy level, and relative weakness of the “mesomorphic toughness” as polygenic potentiators of clinical psychosis in the schizotype, one should probably include a somewhat heightened general sexual hunger at least for one subset of cases. (I do not believe this is incompatible with the historical datum that poor premorbid have a deficient heterosexual experience history, since I look upon this as primarily attributable to the interpersonal aversiveness of schizophrenic individuals.) Presumably also whatever genetic factors are responsible for what Freud viewed as the constitutional disposition to strong pre-genital fixations, rooted in a polymorph-perverse libidinal disposition, would also be part of the contributors; since it on the one side makes an adequate heterosexual adjustment even more difficult for the interpersonally aversive individual; and secondly, in our society, probably contributes to feelings of sexual guilt and “being different from others.” We know that many schizotypes, even relatively compensated, feel concerned about what they recognize to be aberrations in their sexual impulses and fantasies (whether acted out or not). Given the well established decrease in marriage and sexual fertility even within marriage among clinical schizophrenics, which I assume is also true (although presumably not to such great extent) among semi-compensated schizotypes, the population of offspring of two clinically schizophrenic parents would, on my theoretical picture of the situation, be a population arising from parent-pairs whose sexual drive was above average strength, since the sexual drive is presumably one of the determiners of marriage

and of parenthood within marriage. But since both of the parents are known to be clinically schizophrenic, we have to assume that for the sexual drive to be capable of overcoming the influence of the polygenic potentiators we infer must be there since the parents are clinically schizophrenic rather than compensated cases, this drive must be at least above average for all schizotypes and quite possibly above average for non-schizotypic individuals in the neurotic or normal population.

7. I come now to the clinical impression, which I am forced to share in spite of my theoretical bias being against it, and which so far as I know is practically universal among therapists who treat schizotypic individuals, that the psychopathology in the parent-pairs of our schizophrenic patients is not symmetrical, in so far as we can infer it from the patient's verbal productions in treatment. The dominant gene theory, without being further qualified as regards sex differences in marriage and fertility, would lead us to expect that about half the time the patient under study got the schizoid gene from the mother, and about half the time he got it from the father. I am forced, willy-nilly, to admit that if we take the patients' reports at anything like face value, the situation with regard to father versus mother is not anywhere near this symmetrical. At first glance, the nearly uniform finding of more severe pathology in mother and with, at most, a kind of "ancillary" pathology in father as inferred from the patient's reports in treatment, constitutes a very strong clinical objection to the kind of genetic model I am proposing. I should like, however, to permit myself a little ad hoc speculation, with the understanding that this must itself be checked out by more direct means. And I point out that it does not involve purely speculative notions, but can be somewhat defended on the basis of present statistical evidence; and, I think, quite strongly defended on the basis of the same kind of support, namely, clinical impressions, which we are for the moment agreeing to trust on the basis of therapist's impressions. As I pointed out in my paper on schizotaxia, there is some statistical evidence to show that the effect of diagnosed schizophrenia on the fertility of females is less than it is upon the fertility of males. Dr. Reed tells me there is no doubt in his mind that this is the case, and he cites some data suggesting that the schizophrenic female may produce almost twice as many children as does the schizophrenic male. This statistical evidence, which admittedly is not fully replicated as yet, is in accord with clinical experience in treatment of schizotypes. It is also something one could presumably predict from general psychological considerations, namely, that the characteristic demands of the heterosexual role in our society are such that the lowered social aggressiveness and the interpersonal aversiveness of schizoid individuals should be, on the average, expected to have more of an effect in reducing the mate-seeking, marrying, and even the Kinsey "outlet" rate within marriage for schizoid males than for schizoid females. It furthermore is in harmony with the clinical experience, which I think most therapists will agree with, that one of the phenomena of the schizotypic female (particularly when she is not psychotic but at least semi-compensated most of the time) is that she finds herself in sexual situations. As a matter of fact, in my schizotypic sign-list I include the strange phenomenon of the schizotypic woman actually entering into marriage with a man whom she does not particularly like, love, or even find sexually attractive. Whatever the relationship of these tendencies may be to the personality pattern, I think that (with the usual qualifications regarding purely clinical impressions) it is a safe empirical generalization to make. I would furthermore anticipate that the difference in production of offspring between schizoid males and females would be

enhanced when we move from the clinically psychotic population of parents into the compensated schizotypic population, on the grounds that diagnosable psychosis presumably exerts a rather sizeable negative pressure against mating and procreation for both sexes, but when we move into a semi-compensated range where the gross psychopathology is not so obvious to a potential marriage partner, the relative importance of social and heterosexual aggressiveness in relation to mate-seeking role behavior would be further enhanced in favor of the female. It is also a clinical impression, not confined to myself, that many schizotypic women exert a peculiar fascination for men partly because of their “mysteriousness” and partly because of certain features of their social and sexual behavior that many males (even normal ones) find interesting, such as the clinging dependence, a certain excitement produced in men by the woman’s ambivalence, and, very importantly in my clinical experience, the attractive features of chaotic sexuality in a woman. On the other hand, while the compensated schizotypic woman has this kind of multiple attraction for even normal males, I believe that men with a considerable loading of neuroticism are on the average more likely to find such a woman attractive; and I assume neurotic males are also likely to commit the “error of judgment” involved in marrying such a person without adequately assessing the total bookkeeping on such a woman as a wife and mother. That is, I am assuming here that one statistical result of neuroticism is an unrealistic and psychopathology-based pattern of sexual choice. On this kind of scheme, our genetic expectation would be that in the total marital population of mixed parent-pairs, i.e., pairs in which one partner is schizotypic and the other not, we would expect that the non-schizotypic partner is, on the average, considerably more neurotic than the average member of the general population; and, secondly, we would expect that in neurotic-schizotypic parent-pairs, the situation will not be statistically symmetrical but that the more frequent combination will be that of a neurotic father who has married a schizotypic mother. In terms of the parental genes available for transmission to the offspring, this would mean that when the offspring is selected because he has become clinically schizophrenic, the more usual situation will be that he has received the specific schizoid gene from mother but that he has received the preponderance of his adverse potentiators (such as the high anxiety parameter) from the father. The more polygenic potentiators are present in a woman who is also a schizotype, the less likely she is to have offspring; and consequently when we start with the diagnosed schizophrenic offspring as the index case, we do not find neurotic/schizotypic symmetry in going backward to the parent pairs.

8. I next rely upon a fact which I believe is not in dispute, namely, that schizophrenic individuals have a markedly exaggerated tendency to perceive aversive social inputs when the latter are objectively lacking, and to exaggerate them when they are objectively present. I know that this is a problem which you have been concerned about methodologically in evaluating retrospective studies of the schizophrenogenetic parent, and you have no doubt thought more about this I have, so I will not elaborate the point.

9. Finally, I invoke a phenomenon about which we might disagree in evaluating its quantitative significance but I am sure that you will agree with me that it at least has qualitative truth to it, namely, that not all of the causal arrows in the mother-child interaction can be assumed to originate in mother and flow to child. To some extent they must also go in the child → mother direction. Now when we are examining the consequences of a postulated genetic model, it is imperative to give the model its due,

and that will include assuming that the genetically schizotaxic child, as soon as he begins to develop the schizotypic personality on the kind of mixed regime that all children experience even with a good family background, will begin to provide mother with aversive inputs, and not to provide her with adequate social reinforcements, meanwhile making demands upon her, in the same way as normal, non-schizotypic siblings do. Since schizoid patients are somewhat a pain in the neck even for the psychotherapist who doesn't spend many hours of the week with them, who gets paid to help them, and who has a variety of professional as well as personal motivations to work with them, it is utterly inconceivable to me that a schizoid child or adolescent could fail to be a pain in the neck even to a healthy mother, let alone to a neurotic or schizotypic one. I admit that this is reasoning from the armchair but it strikes me as pretty solid armchair in this instance. There are, of course, some data which provide statistical support for this conclusion, in the study by Lewis Klebanoff, "Parental attitudes of mothers of schizophrenic, brain injured, retarded, and normal children," American Journal of Orthopsychiatry, 1959, 29, 445-454. I realize that you have some reservations as to the discriminating power of the parental attitude measuring instrument Klebanoff employed, so I don't want to make a federal case on the basis of this one investigation; I merely want to include it along with the armchair considerations as a ground for saying that the hypothesis I am developing here is not completely ad hoc and devoid of factual support at present.

10. It is foolish to try to work out any very detailed mathematics, particularly the mathematics of the polygenic variables, in the present state of our ignorance. Therefore I shall treat the theory dichotomously for purposes of exposition. We have the following factors:

a. Starting with a schizophrenic patient as the index case, he will more often have received the schizogene from mother than from father; i.e., instead of the chances being 50:50 that mother is a schizotype, the chances are considerably greater than half that she is a schizotype.

b. Whether the mother is a schizotype or not, the schizotaxic patient will become schizotypic and will therefore provide inadequate positive reinforcement and excessive aversive inputs to mother, thereby increasing the probability that mother, whether schizotypic or not, will objectively behave ambivalently and "schizophrenogenically" to the schizotypic index case while he is a child. This will presumably include an objective tendency, on the average, for a mother to show discernible signs of preferring the ~~parent's~~ patient's siblings, half of whom are presumably not schizotypic.

c. Even if the mother is not herself schizotypic, and even if she avoids developing any "schizophrenogenic" behavior as a result of the schizotypic child's inputs to her, such a mother's "ordinary" (i.e., not systematically different from other mothers) behavior will invariably contain some garden variety aversive and ambivalent features, which the schizotypic child will perceive more readily, and will react to more aversively, than will his non-schizotypic siblings.

d. Finally, of course, the current theoretical orientation of most psychotherapists, particularly those interested in intensive treatment of schizotypes, will provide plenty of interview scheduling in which verbal behavior and dredging up of recollections about

how crumby mother was will be plenty sufficient to guarantee that the patient's material will include recitation of episodes along these lines.

11. I don't think you have to blow up the figures from plausible armchair values to show how such a causal model could add up to a practically 100% incidence of "bad mothers" as these mothers are experienced by the psychotherapist via his schizophrenic index case. If we assume that when mother is actually a schizotype she will behave in a schizophrenogenic manner, such that any schizotypic child will experience, record, and as an adult schizophrenic in therapy will report this behavior, I suppose it will be safe to say that for all practical purposes 100% of the schizophrenic index cases in therapy whose mothers were in fact schizotypes will report these mothers as what the therapist would classify as schizophrenogenic. That is, assuming there is no distortion on the part of the patient, and no theory-based brain-washing on the part of the therapist, we already start with a base tally which will be as large as the frequency of schizotypic mothers in the population of parent-pairs of schizophrenic patients. Suppose for example that the differential fertility of schizotypic for males and females in the previous generation were, say, in the ratio of 2 to 1. Then starting with schizophrenic index cases, 67% of them would have schizotypic mothers. Substantially all of these schizotypic mothers would be (correctly) perceived by the therapist as schizophrenogenic; i.e., if mother is in fact a schizotype, the probability that one of her children who develops schizophrenia and gets treated for it will fail to describe her to his therapist this way may be taken as essentially zero. But what about the other 33% of mothers of schizophrenic patients in therapy who were not schizotypic? Well, first of all, they will presumably be more neurotic than the average for the reasons advanced above, and they will therefore be likely to over-react in an "unhealthy" manner to the schizotypic behavior of the child. But even if they aren't neurotic, the schizotypic child will put the mother on a regime such that she will react aversively and ambivalently and with evidence of preference to other siblings. I don't know how to evaluate the percentage in this case, since it is purely speculative, but presumably we can say that at least "on the average" the non-schizotypic mother of a schizotypic child will provide a more than average amount of aversive, ambivalent, rejective, and sibling-preferential regimes. I could really be closer to 100% here, but suppose that we assume that it is merely "on the average," so that, speaking dichotomously, we assume that approximately half of these non-schizotypic mothers will, again quite objectively, react in what will be seen by the patient's therapist as schizophrenogenic. That would account for another 16–17% of the mothers (i.e., half of the remaining 33%). We have 16–17% left to go. This 16–17% consists of mothers who are not schizotypic, not particularly neurotic, and who in fact are healthy and nurturant enough so that they are able to restrain themselves from reacting schizophrenogenically to the behavior of the schizotypic child who is our index case. I am sure that you are willing to assume with me that the majority of schizotypes will have aversive experiences on a completely normal or average regime such as we are imagining for this remaining one-sixth of mothers; therefore what they report to the therapist will be that "mother didn't like me," "mother preferred my siblings," "mother never wanted children anyway," and meanwhile report many bad-mother episodes in concreto. Again, dealing with the statistics dichotomously, if the majority of schizotypes react this way to average or neutral inputs, provided by a normally mixed maternal regime, we can again take a rough figure of half of the remaining group of mothers, that is to say 7–8%, from the 13% who

are left. This leaves us with a mere 7–8% of mothers who are in fact (a) not schizotypic, (b) not particularly neurotic, (c) refuse to respond schizophrenogenically to the feedback of the schizotypic child, and (d) who are not falsely perceived as having done so due to the patient's childhood aversiveness nor by his retrospective distortions in therapy. Then all you need to assume by way of therapist bias and brainwashing as a factor in producing the prevailing clinical impression of the powerful etiological role of mother is a little 7–8% slippage-factor for therapist, engendered by the current theoretical orientation. This, I am sure is a considerable underestimate. Consequently we get, without making any outlandish assumptions, the derivation that close to 100% of mothers of schizophrenic patients will be reported as having been schizophrenogenic by therapists.

12. I want to make very clear what I view as the methodological status of the above derivations. I hope you do not interpret me as arguing, from the mere possibility of constructing such a model as the preceding, that the existing clinical data differentially corroborate a genetic view of schizophrenia, or weaken the corroboration provided by these data to the social-learning view of etiology. My point is partly substantive but mainly methodological. My point is that what at first glance appears to be strongly discorroborative of a genetic view, namely, the approximately 100% incidence of bad mothers in the seemingly uniform experience of psychotherapists who work with schizophrenia, is not in fact discourroborative because it can be plausibly shown, on the basis of existing theory and existing statistics, to flow as a consequence of a model which attaches critical importance to the dominant gene. My point is that it is essentially neutral with regard to the two major competing interpretations. What I object to is the almost universal tendency of psychotherapists to argue, on the basis of what they hear in the interview, for a social learning specific etiology when a genetic etiology, placing only minor emphasis upon social learning factors, would lead to the same expectations. I further want to emphasize the point that aside from the quantitative details, the previous line of thought indicates what I take to be an absolute minimum complexity for any genetic interpretation of schizophrenia and therefore shows that it is not feasible to subject a genetic theory to statistical tests unless (a) the polygenic potentiators, (b) the differential fertility of the sexes and, finally, (c) the social learning factors which make mother (as primary nurturant figure) more important in the life history than father, are included in the mathematics of the genetic model. If one already knows in advance that even granted the genetic theory certain complications will necessarily arise, it is rather pointless to subject the genetic theory to family concordance statistical tests which the model tells us, in advance, should not work out anyway!

13. I believe the preceding analysis will also indicate why I would not place any particular faith in the value of retrospective studies utilizing the “normal” siblings of known schizophrenics as mother-raters for purposes of evaluating a social learning or a genetic etiological theory. Consider this situation adopting the genetic model above. If we are dealing with a dominant gene of complete penetrance for schizotaxia (and therefore one which always leads to the development of schizotypy, but leads less than half the time to the development of clinical schizophrenia), we know to start with that approximately half of the siblings of schizophrenic index cases will be schizotypes, whether they are clinically schizophrenic or not. Most of the preceding analysis of the situation with the schizophrenic proband applies mutatis mutandis to that of the non-schizophrenic but schizotypic sibs. Entering the parent and sib population via the known

schizophrenic index case but concentrating our attention now upon how the non-schizophrenic siblings, taken statistically as a group so located, will perceive mother, we start out with the same basic figure from the fertility statistics so we assume that 2/3 of the mothers are in fact schizotypes. We assume that the schizotypic siblings will perceive mother as being the way she is, and then some. Furthermore they will, like the index cases who went on subsequently to become overtly schizophrenic, provide unsatisfactory reinforcement schedules for the remaining 1/3 of mothers who are not in fact schizotypic but who, as in the above analysis, will be frequently neurotic because they will be the contributors of the potentiator genes to the index case. The only important difference in the progressive accumulation of percentages by halves is, of course, the fact that half of the siblings will not be schizotypes and therefore will not tend to elicit bad mother behavior from the 1/3 of mothers who are themselves not schizotypic. Of course they will still be eliciting some bad-mother behavior from that unknown (but presumably sizeable) fraction of this 1/3 who are neurotic. It follows that the incidence of "bad mother" perceptions by the nonschizophrenic sibs will be greater than would be the case for the perceptions of control cases, i.e., cases chosen on the basis that there is no schizophrenia in the sibs yet, and in fact will be very sizeably larger than this because, on the genetic model, we do not expect any appreciable incidence of schizotypy among the control mothers to begin with; and, secondly, we do not expect the siblings in the control group to be schizotypic and therefore we do not expect them to elicit rejective or ambivalent behavior from the normal mothers; and, finally, we do not expect nearly as great an amount of distortion (either current or retrospective) on the part of the control sample. On all three counts, therefore, the perceptions of mother by the non-schizophrenic siblings of schizophrenic probands should be shifted in the bad mother direction when contrasted with controls. On the other hand, the incidence of 50% of non-schizotypic siblings will mean that they ought not, as a group, show such a strong trend in this direction as is true of the schizophrenic index cases. Therefore the "bad-mother perception" shown by sibs of schizophrenics may be expected to be somewhere between the perceptions of mothers in a control sample and the perceptions held by the schizophrenic patients themselves; and, on my view, somewhat closer to the latter than to the controls' perceptions. But since neither theory has sufficient precision at present to make quantitative predictions restricted to a narrow range of values, this finding is also neutral, and does not shed any light upon the merits of the two interpretations. Your theory would imply that the mothers of known schizophrenic index cases should be perceived as schizophrenogenic even by the non-schizophrenic siblings of these index cases; so does my theory. Your theory does not enable you to predict the magnitude of these differences, neither does my theory. Both of us would be surprised, and would have a job of explanation on our hands, if it turned out that the siblings perceive the mothers no differently from the way people in general perceive their mothers. Both of us would be surprised, and would have an explanation job on our hands, if it turned out that the non-schizophrenic siblings on the average perceived the mothers exactly as do the diagnosed schizophrenic patients.

The question now arises whether I can make any psychometric predictions on the genetic model with sufficient confidence so that if they were refuted they would constitute strong evidence against the genetic theory; and, if so, whether you can make the same predictions on all matters that some other theory can make. The question is how the two stack up with regard to how many detailed predictions they can make, and

whether they succeed in passing the hurdles which these detailed predictions impose. As Popper says, that a scientific theory “fits the facts” gives it very little support, since it is easy to concoct theories which fit the facts, and the problem is to concoct theories which will not fit the facts unless the theories have a high degree of verisimilitude. My objection to most of the schizophrenia research is that it does not provide any difficult hurdles for the theories to pass, and that it is almost entirely neutral with regard to the theories currently competing. It should be the business of an empirical test of a theory to provide a hurdle which the theory is very unlikely to pass if it is largely false; and which, at the same time, either provides a refutation of the alternative theories or, at least, involves a prediction which the alternative theories do not enable you to make one way or another. We must distinguish therefore between theories which are highly corroborable and theories which are highly corroborated. One objection I have to most theories about schizophrenia is that they are not highly corroborable because they do not make predictions which generate sufficiently difficult hurdles so that the successful negotiation of those hurdles can be viewed as contributing much to their credibility.

The difficulty with my own theory in this respect is that it does not enable us to make a prediction of MMPI results without at least assuming something as a lower bound for the validity of one or more MMPI keys. Since the items of MMPI Scale 8 were derived on frankly schizophrenic patients and since the items have a considerable amount of “gross pathology” in their verbal content (as can be discerned by reading through them), there is a problem as to whether the number of subclinical or subtle items on Scale 8, which are at the same time possessing of sizeable validity, is sufficient to use as a discriminating device within the compensated range. Unfortunately neither I nor anyone else has the answer to this question at the present time. I will therefore have to begin by saying that the first thing to find out is whether the distribution of Scale 8 scores for the parents of the schizophrenic sample differs from that of the parents of a control sample. If it doesn't, this is distressing as to the state of psychometric technology but it does not argue very forcibly against a genetic theory because of course we don't know whether that finding arises because the scale was not capable of extrapolation into the compensated range for which it was not originally built, or because there is nothing particularly schizoid about the parents of schizophrenics as a group. I would like to be able to predict something about this but I honestly do not believe it possible to do so.

However, let us suppose that preliminary analysis shows that there is a considerable difference between the distribution of scores on Scale 8 for the parents of known schizophrenic patients and the parents of controls. This is what I would expect to happen but I wouldn't bet you odds of more than around, say, seven to three. If it does occur, I would next raise the question whether the difference is of sufficient magnitude to indicate a respectable psychometric validity at work. You understand that when I talk about a respectably psychometric validity I do not mean the mere establishment of “statistically significant difference,” which I view as a necessary but far from sufficient condition to find anything exciting when it comes to either theory corroboration or clinical practice. Roughly, I am not interested in differences which are not close to one standard deviation in size. A separation of this magnitude will lead to around five hits out of six with symmetrical base rates, and is a representative value for the discriminating power of individual MMPI scales on cross validation using diagnosed criterion groups. You can see therefore why I am somewhat skeptical as to the power of Scale 8 for the present

purpose since this would require that it function about as well in identifying subclinical cases or compensated schizotypes as it functions in identifying clinically diagnosable ones. However, I am currently working on some subclinical keys, partly by item analysis in my own small sample of psychotherapy cases but partly (don't tell Starke!) on an armchair basis by identifying items which are from their content indicative of anhedonia, cognitive slippage, and interpersonal aversiveness. If I can get an anhedonia key which is counter-balanced for masculinity-femininity content, and a cognitive slippage key which is sufficiently subtle, it might be possible to do a decent job in the compensated range. Anyhow, let us suppose that we first find that Scale 8, or one of these other experimental scales, shows a sizeable separation between the parents of schizophrenic patients and the parents of controls. (For reasons which will become apparent below, the way I would actually do this would be to pick the higher T-score from each parent-pair and study the distributions of these scores for parent-pairs of schizophrenics and ditto for controls.)

Once we have reason to believe in a sizeable psychometric validity for the scale, then I am prepared to make some psychometric predictions on theoretical grounds. Even if the coefficient of assortative mating for schizotypes is not zero, it would hardly be large enough to produce any appreciable numbers of parent-pairs both of whom carries the gene. So I assume that, with the possibility of a small number of exceptions, one of the members of each parent-pair is schizotypic and the other one is not. Further, my theory predicts that the schizotypic parent should be the mother rather than the father over half the time; and I will be willing to guess it would be as much as 2/3 of the time. Hence if we distribute the scores of the mothers we should find a distribution which is either bimodal (if the validity is sufficiently good) with the primary mode on the right and a secondary mode on the left and with the areas of the two component distributions being in a roughly 2:1 ratio or higher. Of course we don't know what the component distributions are but if bimodality exists we can draw some plausible inferences from the combined envelope. If the psychometric validity is not sufficient to generate a bimodality—but it should do so if the separation is as large as a standard deviation within sexes—then at least we can expect clear evidence of skewness to the left, the “tail” being the strongest available indicator of the presence of the smaller latent distribution, i.e., of non-schizotypic mothers. The situation for the fathers should be the reverse of this, i.e., we expect a distribution skewed to the right or if validity is pretty good a bimodal distribution with the primary mode low and the secondary mode high and again with areas of the two inferred latent distributions being in the ratio 2 to 1. If we take the difference score between the members of each parent-pair, and distribute these difference scores, the above effects should be blown up and a pretty clear bimodality should be in evidence, again with the one mode being higher than the other and the area of the curve under that mode being twice as large as the other one.

Suppose we draw a cutting line at the center of the “valley” for the male and female distributions separately. We can then classify each father as to whether he is above or below that cutting line, and we assume on the genetic model that the fathers below are the ones who do not carry it. Ditto for the mothers' distribution. But on the assumption that there are relatively few (if any) cases in a small sample where both parents carry the gene, the fathers who score above the fathers' cut should be married to mothers who score below the mothers' cut; and that implies, contrary to the expectations from an ordinary assortative mating correlation, that when we set up a four-fold table based upon

parent-pairs with the cells determined by these cuts on the distributions taken separately, we expect strong negative association within that table. You follow me?

It would be nice if one could make some stronger inferences concerning the actual underlining (underlying?) parameters of this system, and I am currently working on that with methods similar to those discussed in my recent memorandum on latent taxa. I am afraid I need to make a few additional assumptions beyond what I would be happy to make, but I am not sure about this. What I have in mind is that even without making assumptions about normality or equality of variance, one can write some algebraic identities for the means and variances for the mixed populations. I think perhaps that if we are willing to assume the coefficient of assortative mating within the nonschizotypic and within the schizotypic subsamples to be approximately zero (i.e., the “invalid systematic variance” of Scale 8 reflects components which do not tend to be correlated between spouses), it may be possible to set down enough equations for the relations of the latent means and standard deviations to the manifest values to actually solve for them. Thus, for instance, suppose we plot the means of Scale 8 moving from high to low over the entire range. If respectable psychometric validity obtains, the very high intervals on Scale 8 should consist of all males all of whom are schizotypes because by going out far enough on the continuum we will have passed beyond the limits of the upper tail of the latent nonschizotypic distribution. Similarly at the low end of the overall Scale 8 distribution for males we will be dealing with entirely nonschizotypes for the first sequence of intervals moving upward. Therefore the means for the wives of the sub-samples of husbands in these successive intervals should show no systematic rise or decline except that due to the coefficient of assortative mating for the invalid variance of Scale 8. However, in the intermediate region a different situation obtains, because in that region as we take successively higher or lower intervals on Scale 8 as defining sub-samples of husbands, we are steadily changing the proportion of schizotypic husbands in each interval as we move along, which means on the genetic theory that we are changing the proportions of schizotypic wives in the inverse direction. Consequently the curve for the mean Scale 8 scores of wives obtained by plotting these means against the values of Scale 8 for husbands from low to high should be ogival. Now if we find that there is a flat region of wife means for high-scoring husbands, this supports the model and at the same time indicates the correctness of the approximating assumption that the coefficient of assortative mating for the invalid components of Scale 8 variance is close to zero. That being so, the mean of the wives in the flat region should be equal to the mean of all the nonschizotypic wives in the sample. You can see that if this general line of thought were sufficiently developed (which will take a little more mathematical work than I have put in as yet) it might be possible to actually estimate the latent means and standard deviations for the sub-groups. But even short of that, I think the prediction of an inverse relationship in the four-fold table is a sufficiently striking and counter-intuitive prediction so that if it is supported it would constitute a good piece of corroboration for my theory. On the other hand, if preliminary analysis shows a respectable validity in Scale 8 for this purpose (I must insist up this precondition!), and it was strong enough to generate a bimodality of “higher parent” distributions or of within-sex distributions between parents of schizophrenics and parents of control; then a failure to get bimodal distributions with the positions of the modes reversed, or a failure to get an inverse relationship in the four-fold table, would constitute a rather strong discreditation of my theory.

Regardless of whether your interest is mobilized with regard to the immediate question of getting MMPIs on the parents from your sample, I hope that the discussion has communicated to you more adequately than I have in the past some of my methodological obsessions concerning schizophrenia research. I think you can see why I view the “psychometrics instrumentation” problem as critical, because the mere establishment of significant differences in a theoretically predicted direction cannot tell us enough about the construct validity of any instrument to permit the making of point predictions or range predictions tight enough so that the success or failure of such predictions can constitute a respectable scientific test of anything. I believe you don’t take this objection very seriously because the current psychological research culture is uniformly predicated on the assumption that the significance test is an adequate answer to the problem of theory-corroboration. Somehow or other I am determined to persuade you that in this respect the current psychological climate of opinion is just plain mistaken and it doesn’t make any difference to me whether 90 or 95 or 99% of APA members share this mistake, it is a mistake nevertheless. Furthermore it is not a minor mistake but one which exerts a malignant and pervasive influence upon psychological research. I am writing a paper on this subject for the Bulletin and will send you a draft as soon as it is complete.

cc: Dr. David Lykken, Dr. Sheldon Reed, Mr. Ralph Miller, Mr. David Wright