DOCUMENT RESUME

ED 113 642 95 CG 010 146

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TITLE Needed Research on the Genes and Environment in Human Psychological Development: Perspectives from Behavior Genetics. A Special Report of the USOE-Sponsored Grant Study: Critical Appraisal of Research in the Personality-Emotions-Motivation Domain.
INSTITUTION Texas Christian Univ., Fort Worth. Inst. of Behavioral Research.
SPONS AGENCY Office of Education (DHEW), Washington, D.C.
REPORT NO IBR-R-72-13
PUB DATE Jun 71
GRANT OEG-0-70-2665(508)
NOTE 64p.

EDRS PRICE MF-$0.76 HC-$3.32 Plus Postage
DESCRIPTORS Behavioral Science Research; *Environment; Environmental Influences; *Genetics; *Heredity; *Human Development; Individual Development; Psychological Design Needs; *Research Needs; State of the Art Reviews

ABSTRACT

The task group report presented in this publication is one of a series prepared by eminent psychologists who have served as consultants in the U.S.O.E.-sponsored grant study to conduct a Critical Appraisal of the Personality-Emotions-Motivation Domain. In order to attain the goal of identifying important problems and areas for new research and methodological issues related to them, an approach was followed in which leading investigators in specialized areas were enlisted as members of task groups and asked to reflect on their current knowledge of ongoing research and to identify the research needs in their respective areas. The articles in this publication are: (1) The Genes and Environment in Human Psychological Development; Perspectives from Behavior Genetics (Loehlin); (2) The Future of Human Behavior Genetics with Special Reference to the Genes and the Environment in Cognitive Development (Vandenberg); (3) Unanswered Questions on the Relationship of Genetics to Normal Personality Variation (Scarr); and (4) The Genes and Environment in the Development of Psychopathology (Horn). (Author)

NEEDED RESEARCH ON THE GENES AND ENVIRONMENT IN HUMAN PSYCHOLOGICAL DEVELOPMENT: PERSPECTIVES FROM BEHAVIOR GENETICS

A SPECIAL REPORT OF THE USOE-SPONSORED GRANT STUDY: CRITICAL APPRAISAL OF RESEARCH IN THE PERSONALITY-EMOTIONS-MOTIVATION DOMAIN

Prepared by Task Group 1404 -
The Genes and Environment in Human
Psychological Development:
Perspectives from Behavior Genetics
John C. Loehlin, Chairman, S. G. Vandenberg
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Under the Editorship of S. B. Sells and R. G. Demaree
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TEXAS CHRISTIAN UNIVERSITY

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FOREWORD

The task group report presented in the following pages is one of a series prepared by eminent psychologists who have served as consultants in the U. S. Office of Education sponsored grant study to conduct a Critical Appraisal of the Personality-Emotions-Motivation Domain. The study was planned with the advice of an advisory committee including Professors Raymond B. Cattell and J. McV. Hunt (University of Illinois), Donald W. MacKinnon (University of California, Berkeley), Warren T. Norman (University of Michigan), and Dr. Robert H. Beezer (USOE) and follows a topical outline included as an appendix to the present report. In order to achieve the goal of identifying important problems and areas for new research and methodological issues related to them, an approach was followed in which leading investigators in specialized areas were enlisted as members of task groups and asked to reflect on their current knowledge of ongoing research and to identify the research needs in their respective areas. The general plan is to publish these reports as a collection with integration contributed by the editors. It is hoped that these reports will prove to be valuable to research scientists and administrators.

S. B. Sells, Ph.D.
Robert G. Demaree, Ph.D.
Responsible Investigators

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1404 - The Genes and Environment in Human Psychological Development: Perspectives from Behavior Genetics

Task Group Chairman

John C. Loehlin
University of Texas

In the present section, three experts look at three aspects of psychological development from the general viewpoint of behavior genetics. It is not my purpose in this introduction to second-guess what they are about to say: they will speak for themselves in due course. Rather, I should like to attempt two things. The first is to set these essays in a general context of strategy in psychological research. And the second is to draw attention to several common themes which run through them.

Let me begin by stating three principles widely agreed upon by contemporary behavior geneticists (and by a good many other psychologists and biologists, for that matter):

Principle 1. In order to understand the characteristic responses of individuals to their environments, one is well advised to take their genes into account.

Principle 2. In order to understand the characteristic responses of individuals to their environments, one is well advised to take their prior history of environmental inputs into account.

Principle 3. Principles 1 and 2 are inherently more powerful taken jointly than taken separately.

Much of traditional research in psychology can be seen as an elaborate development of Principle 2. Much of the early effort of behavior genetics was a countervailing attempt to document the

validity of Principle 1. Work along the separate lines of Principles 1 and 2 continues to be pursued vigorously and productively--as well as it should: there is much to be said for the methodological dictum of divide and conquer. But quite broadly in word, and increasingly often in deed, the ultimate supremacy of Principle 3 is being acknowledged.

The three authors of the present section each inspects from a modern behavior-genetic viewpoint recent and prospective trends in a substantive psychological area. Vandenberg looks at cognitive development; Scarr at normal personality development; Horn at psychopathology.

Perhaps the most striking concordance in what they see is reflected in a unanimous cry for better measurement techniques. Vandenberg documents shocking blind spots in the comparatively well-studied realm of cognitive measurement; Scarr finds measurement limitations to be a major stumbling-block to progress in research in personality development; Horn sees the problem of diagnosis as a central issue in genetic research in psychopathology. The latter two authors note the potential for profitable feedback from behavior-genetic studies in sharpening the definition of the traits we are measuring.

A second common focus of the three essays is a stress on diversity of research methods. Gone is the day when a human behavior-genetic study was almost by definition the comparison of a small group of MZ and a small group of DZ twins. While all three authors feel twin studies continue to have an important place, they urge increased emphasis on the vigorous pursuit of a variety

of alternative designs as well: adoption studies, family studies, half-sibling studies, cross-racial or national studies, drug-response or biochemical studies, studies of early development, and others.

A third communality of the three writers is in a concern with environmental variables, particularly of a socio-cultural or interpersonal kind. Vandenberg discusses the problems of adequate and objective measures of the social and psychological environment; Scarr emphasizes intrafamily interactions; Horn stresses the importance of exploring the environmental factors determining why a given genotype in one of a pair of MZ twins may lead to schizophrenia while in the other twin it does not.

Finally, while the three authors differ in their degrees of optimism concerning the prospects for immediate large-scale progress in their areas, all agree that: (1) there are basic social and scientific problems crying for solution; (2) methods exist that offer promise of progress; and (3) the methods ought to be applied to the problems.

It is hard to argue with this verdict.

The Future of Human Behavior Genetics with Special Reference to the Genes and the Environment in Cognitive Development.

S. G. Vandenberg
University of Colorado

In trying to guess what the future of behavior genetics is going to be, it is well to keep in mind that the most convincing prophets are those who are in a position to influence the course of events. Since history does to some extent repeat itself, knowledge of the past also helps. Unpredictable serendipity has led to major breakthroughs but the ability to exploit such events has still depended on considerable knowledge of the existing science.

In what follows a distinction will be made first of all between what is likely to happen and secondly what should happen, in terms of research that is "central" to behavior genetics, and thirdly, more "peripheral" or "ancillary" research that is needed. In all of this the emphasis will be the typical pragmatic one of much of modern psychology on techniques and empirical facts. In the fourth and fifth parts, some more theoretical approaches will be suggested. Finally, in an appendix, a test battery will be suggested.

Part I. Most Likely Future Research:

It is rather a sure bet to predict that there will be more reports of twin studies using all kinds of variables. In general such studies will not add another iota to our fundamental understanding, unless by chance or exceptional brilliance the authors discover some variable controlled primarily by a single gene or

demonstrating some other truly insight-providing discontinuity. To be of any use at all such studies should at least include a sufficient number of ability, personality or perceptual variables to permit a meaningful multivariate analysis, so that a contribution can be made to the unresolved question whether or not there is an important general hereditary component or whether there are a number of equally important independent hereditary components in cognition. Such studies can also help to contribute to an understanding of the precise phenotypic nature of the cognitive processes to which these hereditary components may make important contributions, based on estimates of tenetic correlations.

It is a discouraging thought that, in a way, much of the work done by ability factor analyses will have to be repeated, with twins and in parent-offspring studies.

The second safe bet is that there will be new parent-offspring studies. Because there have been few of these which used measures of special or "primary mental" abilities, these will be of some use but again such studies would contribute a lot more if they had a multivariate character.

A very worthwhile contribution can be made by combining the twin study method with the parent-offspring method. Elston & Gottesman (1968) have provided a method for obtaining refined heritability estimates from such data.

Without additional effort such studies can also provide data for a study of assortative mating. There is no information.

about assortative mating for more modern, narrower and precise conceptions of special or "primary" abilities. Incidental to such work it would be interesting to know how several of these abilities are distributed in both sexes at various (middle) ages. Other than in one study from Holland, there is no such data. Even socioeconomic distributions of these abilities are poorly studied (Verhage, 1964).

In all the preceding and following remarks it should be noted that two parallel studies in rather different settings (or even different countries) would provide much more than twice the information. Perhaps UNESCO could coordinate multinational projects of this type.

The third and final safe bet is that there will be more studies of the psychological concommitants of diagnosed genetic anomalies, both, single gene substitions and aneuploidies. While these will be interesting in themselves, useoof a common set of psychological variables that will permit comparisons across studies is to be recommended. In an appendix at the end of this report an effort will be made to suggest some of these reference variables.

Part II. Research That Is Somewhat Less Likely But Which Should Be Encouraged:

The multiplicative value of multivariate methods has already been indicated. Other models will now be outlined, but before doing so, a plea will be made for cooperative research. In some studies of rare genetic diseases it has

become a fairly common practice to combine data on patients seen in a number of locations or even for investigators to adopt a common set of diagnostic procedures in order to obtain a sufficient number of probands for a meaningful analysis. Behavior geneticists will have to find a way to do the same thing: either a number of them will have to do cooperative studies or they will have to find ways of reporting on a small number of cases in sufficient detail to permit future integration of a number of such reports.

A good example of what can be accomplished in this way is the summary by Moor(1967) of the effect on the global IQ of various types of sex aneuploides from which I have constructed the graph shown in Figure 1.

If the individual investigators from which these cases were collected had in addition used a common battery of short tests of different abilities and had also obtained data on the performance of parents and sibs on these measures, an even more informative analysis could have been made.

In a recent paper, Berman, J. L. & Robin Ford, (1970) performed a study in which they predicted by a multiple regression equation the intelligence of children affected with PKU from IQ measures of parents and sibs. Then they related the difference between the predicted and observed IQ to blood phenylalanine levels.

Practical application of Ray Cattell's ingenious MAVA method (1953, 1960, see also Loehlin, 1965) which calls for information about unrelated children raised in the same home,

twins reared apart and other unusual situations, or the more conventional method of family studies involving more than two generations of interrelated nuclear families will also require such cooperation. Still other examples are furnished by studies of the rarer types of aneuploidies such as XYY and XXY.

We needed more studies of adoped children and those in more detail than the one of Skodak and Skeels (1949), further analyzed by Honzik (1957). While social agencies may be resistant to a single investigator mounting a frontal attack, perhaps a more personalized search for single cases by a number of individual behavior geneticists will encounter less organized resistance. Similarly we need studies of children born to parents who were married more than once. Again, an accumulation of cases by a number of separate investigators may be feasible. Perhaps a central organization could be set up to facilitate and coordinate such research.

Because there are after all only 23 chromosomes in man, the time has come to start routine searching for linkage between continuous variables and bloodgroups or other single gene markers. To make this more practical there may also have to be a central facility which would provide serology laboratory services by airmail and computer facilities. The basic principles have been worked out and several computer

programs for this purpose are now available from Bock; Elston & Haseman; Mi; Renwick and others.[1]

The method of co-twin control studies, which permits study of the influence of specific environment on a constant genotype, seems to have been completely abandoned. Even relatively small efforts, say with 10 to 15 pairs of identical twins, would be very informative. At best the twins would attend a special nursery or kindergarten in which for example, one of each pair was given number games and the other pre-reading games. Vandenberg explored this approach during one summer in Louisville and found it quite feasible.

Part III. Needed Ancillary Research:

We now come to the less central research needed to avoid much inconclusive research with poor methods. As in all sciences, improvement in techniques should not be seen as merely tedious "development rather than research" oriented efforts. Human behavior genetics has no monopoly in having to rely on the available tests. Unfortunately we seem to be going through a period in which work on such problems is regarded as second rate, hardly worth the efforts of ambitious scientists. It is time to call a halt to the furthering of research depending on poorly developed, ad hoc measuring techniques. The hope for quick solutions by instruments created

[1]Information about these programs can be obtained from the authors: D. R. Bock, School of Education, University of Chicago; R. C. Elston, Department of Statistics, University of North Carolina; P. M. Mi, Department of Genetics, University of Hawaii

for a single study is sometimes fostered by a contemptuous attitude toward the somewhat less glamorous efforts of improving existing tests. Factor analysis has been one very potent technique in such efforts. Unfortunately it has rarely used outside criteria. While conventional factor analysis continues to clarify the relationship between the many ability measures, there remain many unresolved questions, partly because of its reliance on group administered tests. A few examples will suffice to demonstrate this.

1. We still do not understand well the processes required in the performance on the subtests of the three Wechsler batteries, although the studies by Cohen (1957, 1959), and by Saunders (1959) have given us some broad outlines. Larger studies are needed in which the Wechsler subtests as well as a carefully chosen set of factorially relatively "pure" tests are administered.

2. We have only glimmerings of understanding about the relationship of success on the Piagetian tasks and their associated stages to conventional psychometric measures. Again, some beginnings have been made but much more work is needed, if possible on substantially larger samples without sacrificing the "clinical" quality of such investigations.

3. Research on the development of language will someday have to be integrated with the measurement of intelligence in young children.

4. The relation between individual performance on various types of learning tasks and psychometric ability measures has received little attention although a few promising studies exist.

The flip side, as disk jockeys say, of genetics, is environmental influences. The assessment of environmental factors influencing cognitive factors is a very difficult task that has perhaps too often been left to sociologists, because it is not easily brought under experimental control. The result is that there are many vague general statements but little hard knowledge. Perhaps the broad outlines of how such an assessment should proceed can be indicated, but little progress in refining these ideas has been made since Barbara Burks' 1928 paper, except for the very fine-grained analyses by scientists studying infantile perception of patterned versus non-patterned stimuli, or the more "impressionistic" formulations by cultural anthropologists.

Considering past efforts, some requirements can be specified. Environmental assessment needs to take into account several levels or types of information:

1. Socioeconomic status. Warner's triplet: occupation, education and type of home still provides a good measure and up to date revisions are available (Reiss, 1961).

2. Size and composition of the family, plus ordinal position of a given child. These easily obtained data may not add much over and above that obtained from the first category except for within family variance.

3. Psychological atmosphere in home:

   a. As indicated by more objective items such as number of books, types of magazines, membership of parents in clubs or other organizations, hobbies of child and parents.

b. More "psychological" attributes that are more difficult to assess: Parental attitudes, expectations for the child's career and type of disciplinary control. Parent questionnaires may give mainly their perception of the currently fashionable child rearing practice. Some shrewd interviewers can do fairly well in getting below this surface impression. Some teachers may also be able to provide useful data..

Part IV. Within and Between Ethnic Group Comparisons:

For theoretical reasons we need to study cognition cross-culturally, if we are to arrive at biologically relevant generalizations about the species.

It is my considered opinion that attempts to estimate heritabilities in American Negroes and/or Chicanos will be quite informative about heredity-environment interactions and will tend to show that heritability estimates on whites cannot serve as the basis for inferences about racial differences in ability. While this point should be obvious, it apparently is not widely understood and may need many more experimental demonstrations than the one small study of Vandenberg (1970).

If at this time it seems more expedient for political reasons not to do such studies on Negroes in the continental United States, they could be done, perhaps also at less expense, on other ethnic groups in Hawaii; or in Puerto Rico or Alaska; or even in Brazil.

There has been some talk about assignment of an index of white gene admixture to each of a number of Negroes in a study, using

gene frequencies of ancestral African and white groups to arrive at the probability that a given allele is of white ancestry and weighting a number of these alleles to obtain for each person a total value (in the nature of a proportion of white genes in the total genome). This value can then be correlated with ability test scores.

Again I would not expect such a study to provide simple results which would given comfort to either racists or over-eager equalitarians. If skin color and socioeconomic status were also measured, I would predict large interaction and covariance effects that may well outweigh additive genetic variance.

Part V. Need for "Basic" Theoretical Formulization:

On a much more theoretical level, we are lacking well worked out approaches to the structure of populations with respect to ability measures. While there are some large bodies of data that are relevant, most of these were collected without benefit of modern ideas about gene pools with restrictions on gene flow between these pools, except for social mobility tied to highly visible, uniquely human attributes signalled by outstanding school grades; great beauty or social charm and exceptional athletic or artistic gifts. Purely theoretical work and computer modeling may help to advance our understanding of the very complex multidimensional processes governing the changing distributions of genes influencing psychological variables. It should be understood that few individuals have adequate backgrounds for undertaking worthwhile

work in this area. An evolutionary perspective would have to be formulated in which the personal motives of many individuals who mate and reproduce and the often unintentional but sometimes serious ecological consequences of industrialization and continued expansion of human populations are interacting in subtle ways.

Such theories may soon be needed to justify decisions related to curtailment of reproduction or economic penalties for producing retarded children, since various organizations are urging legal measures to curb the population explosion.

APPENDIX 1. Suggestions for "core" data to be collected in cooperative studies.

Karyotypes, repeated several times or if this is not possible, determination of Barr bodies.

Birthweight and data on subsequent physical growth to be compared to standards.

Height of father, mother and sibs.

Parental ages at birth of proband.

Fingerprints and palm prints.

General intelligence: IQ (Stanford Binet or Wechsler) plus similar measures of both parents.

Social competence: Vineland social maturity scale.

Patterning of abilities: Wechsler subtests, better yet, scores on special tests of separate abilities such as PMA, Pacific Multifactor tests, (Meyers, et al. 1962, 1964) or some European test battery.

Photos of proband repeated at following visits. (perhaps somatotype).

Sexual identity or gender role questionnaire and when techniques become available quantitative sex hormone assay.

EEG, especially Kappa-waves.

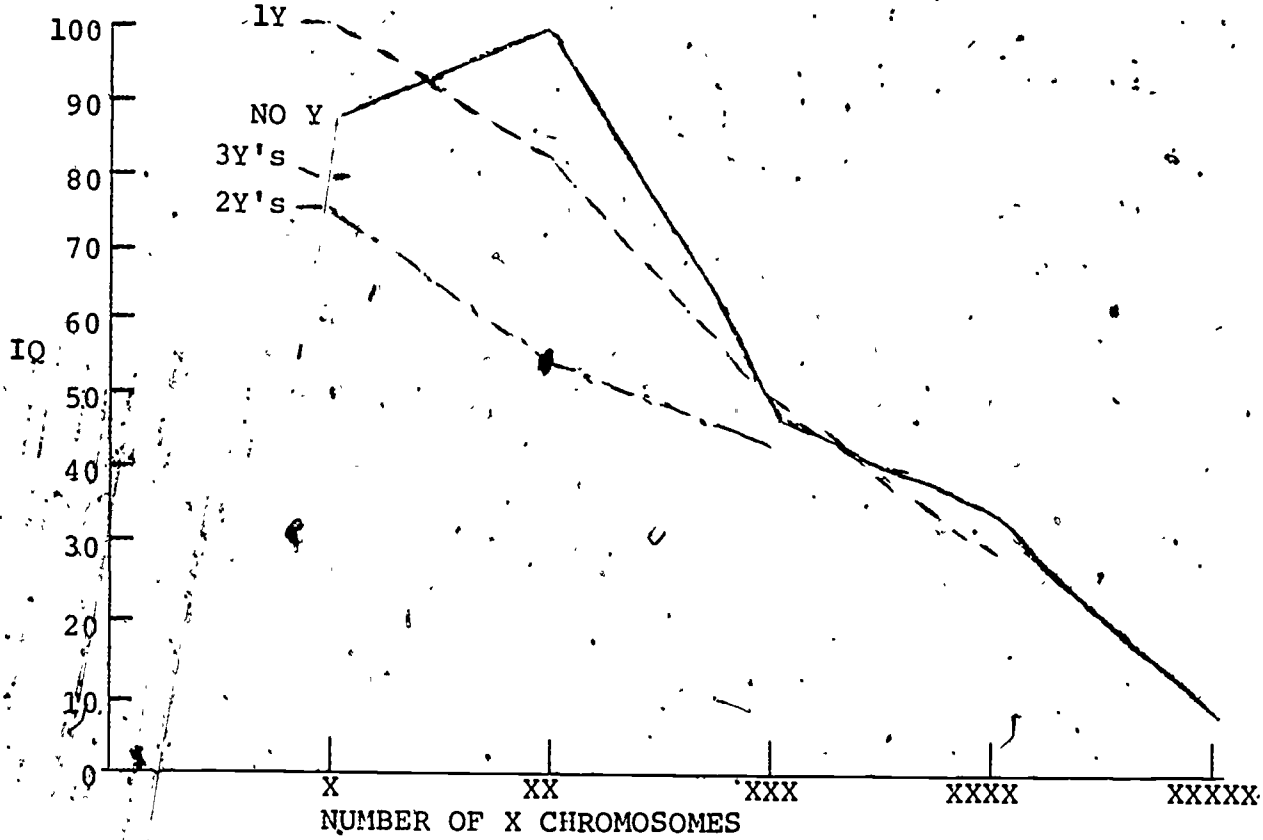
Teacher ratings of aggressiveness, popularity, outgoingness or sociability ("compared to all the youngsters you have known, how do you think x rates?").

If proband is capable of it, a personality questionnaire such as the one by Porter and Cattell (1968).

If more extensive ability testing is desired, the E.T.S. kit of reference tests should be consulted (French, 1951, French, et al. 1963).

Figure 1

MEAN IQ OF INDIVIDUALS WITH ABNORMAL NUMBERS OF SEXCHROMOSOMES (MOOR 1967).



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Unanswered Questions on the Relationship of Genetics to Normal Personality Variation

Sandra Scarr
University of Minnesota

In their review of the recent literature in behavior genetics, Lindzey and his colleagures (Lindzey et al. , 1971) noted the mediocre results of studies in the area of normal personality development.

> During the past five years the total volume of available data on the inheritance of normal human personality, interests, and social behavior has probably at least tripled. It would be pleasant to say that this influx of new data has settled conclusively most of the outstanding questions in the field. It would also be untrue. (p. 59)

The collective research efforts of twenty or so major investigators has resulted in the establishment of the fact that MZ twins are somewhat more similar than DZ twins at all ages on standard tests of personality. In the face of persistent ocial reinforcement theories to account for all of human personality development (e. g., Mischel, 1968) the establishment of that fact is probably worthwhile. But where do we go from there?

Several different directions have been identified by Freedman (1968), Hirsch (1967), McClearn (1967), Mischel (1968) and others. Their advice is often contradictory and bears critical evaluation.

An ethological approach

An ethological approach to human development has been suggested to provide guidelines to important variables in human behavior. Behaviors typical of many primates, such as infant-mother attachments, social signallying, and pair bonding, have species-specific patterns of development. Individual differences in genotypes may produce individual variations on the basic hominid themes (Freeman, Loring and Martin, 1967). Selection of ethologically-important variables can assure the investigator that nearly all normal members of the species will demonstrate some version of the behavior and that natural selection has had a chance to favor some phenotypes over others and to build genotypic variances into the behavior (Emlen, 1966).

Attempts to look at patterning of development in social attachment and related variables have been limited to three studies in my knowledge: Freedman and Keller (1963) with a small N; Wilson et al., as yet unreported from the Louisville twin study, and my own efforts with Philip Salapatek in studying another small number of twins monthly for a year. For the moment I guess that the genetic results will be modest for our as-yet-unanalyzed data. For some variables such as fear of strangers and contact-seeking behaviors, MZ twins seem to be more similar than DZ's over the first 18 months of life. Much of the variance, however, seems to arise between families since both DZ's and MZ's are quite similar and the ratio of $\sigma^2_{WDZ}/\sigma^2_{WMZ}$ is not statistically significant.

The kind of analysis usually attempted in twin studies may have limited payoff when applied to ethological studies. The portioning of total variance into genetic and environmental, within- and between-family components does not take all of the transactional history of the child and his family into account. To some (unknown) extent the child shapes his parents' behavior, which in turn alters his range of responses to similar situations. We as investigators have no control over genotype-environmental covariation. At least in the normal range of family environments, parents tend to adjust their behaviors to match the child's behavior. Most of this covariance ends up on the environmental side of the equation when much of it seems to be genotype-dependent.

Perhaps we need a new methodological approach to family studies, again borrowed from etology. The study of parent behavior with MZ twins, DZ twins, sibs (at the same age), and adopted children (at the same age) could be most instructive. Measures of differences in parental responses to children in related and unrelated pairs over time might produce some useful data on feedback loops between the child organism and his environment. What characteristics of child behavior evoke differential responses from parents, are there changes in parent perception and parent behavior over time that are related to changes in child behavior, and so forth? The developmental process as a function of both genotypic and environmental contingencies has hardly been touched.

Within the usual framework of family studies, normal personality variation due to genetic variation seems lost in the tangle of

behavioral plasticity, environmental modification through feedback loops, and differences in parental behavior within- and between-families. I do not think that selection of other variables within the personality domain will improve the mediocre results already obtained. Only a more thorough understanding of organism-environment, stimulus-response sequences will clarify the role of genotype in personality development.

Toward this goal, studies of personality resemblance and transaction between parents and their natural children (sibs and half-sibs) and parents and adopted children could be useful.

Sex differences and sexuality

Behavior genetic studies of sex differences is another promising area of investigation. It obviously makes a major difference in morphology to have an X rather than a Y chromosome, and to have only one or more than two sex chromosomes. Differences in behavior between the sexes, and between normal and anomalous persons, are well-documented from an early age. But do we know what differences in the degree of "masculinity" and "femininity" are genetically determined? I think not.

From an evolutionary view, males have probably been selected over generations for assertiveness, hypersexuality, and courage in the face of physical threat. Females have probably been selected primarily for nurturance and receptivity to males' sexuality. To what degree are manifestations of these characteristics inherited? If males in a family are highly masculine on measures of assertiveness, etc., are their sisters more, or less, feminine? And vice

versa? Masculinity and femininity as sexual identity, as outlined above, should be carefully separated from degree of sexual drive and choice of sex object, which seem to me to be orthogonal variables.

Sex-object choice, as evidenced in homosexuality, has a small behavior genetic literature to support claims for inheritance. Kallman (1952) and Heston and Shields (1968) reported provocative results for male homosexuals. Can we extrapolate to the normal range of behavior and account for individual differences in sex object choice? Studies of the similarity in sibs' and adopted children's sex-object choices could be helpful. It would be enlightening to chart sexual preferences of males and females reared in the same home environment but differing in genetic relatedness.

Degree of sexual drive is also a variable amenable to behavior genetic study. According to evolutionary theory, differences in sexual drive should have consequences for reproduction and selection. The frequency and intensity of sexual behaviors would seem to be valuable measures to collect on related and unrelated pairs reared in the same home environments. The selective advantages of high sexual drive, if any, could also be studied by differential reproductive performance in various populations. An ethological approach to variable-selection could identify more "natural" units of behavior that have evolutionary consequences.

A molecular approach

Another approach, suggested by McClearn (1967) is the selection of more molecular behaviors for behavior-genetic analysis. Reaction time, psychophysiological responses, and the like, are more

accurately measured and likely to have more direct gene-behavior pathways (Hirsch, 1967). The wave of this future would be reductionism, the selection of variables more amenable to study than broad personality patterns.

The demonstration of genetic contributions to molecular responses is a good bet for genetic research. Individual differences in CNS organization seem as likely as differences in thyroids, livers, and phenylalanine metabolism. The consequence of choosing molecular variables, however, is often a loss of explanatory breadth for human behavior. The contrast between studying individual differences in IQ scores and differences in seratonin production is mostly at the level of inferences that can be made from the findings. A study of seratonin production leads most likely to inferences about other sorts of brain chemistry and their genetic pathways. A study of IQ differences leads most likely to inferences about more molar behavioral variables, like achievement motivation and self-esteem. It is the latter level of explanation that personality studies seek ultimately to achieve. The shortest path to explaining molar behavior may not be a molecular one although there is undoubted value to a clear understanding of the effects of genotype and environmental variables on molecular behaviors per se. But, if personality is the issue, molecular variables are unlikely to be the best choice.

Situational variables and inconsistency

Mischel (1968) and others of the social learning persuasion have come to an almost nihilist position regarding personality variables. The amount of variance in behavior accounted for by

consistent individual differences over time and across situations has been found to be insignificant, and personality inferences are therefore judged to be useless. Correlations for the same measures across time and situations are generally in the .3 to .4 range, accounting for ten to twenty percent of the total variance. Only self- and other-ratings have higher test-retest correlations, and here the consistency is said to be in the eye of the beholder. Situational determinants of individual consistency and reinforcement history effects are chosen as explanations of the small amount of consistency that does exist. Measurement unreliability is rejected as an explanation of inconsistency in favor of inconsistency as a principle of behavior.

There are three problems with Mischel's approach to personality studies as they pertain to behavior genetics. First, for situational measures of personality the test-retest reliability for individuals is of approximately the same magnitude as the MZ co-twin correlation. Observer ratings of situational behavior (e.g., Scarr, 1966, 1969) and experimentally manipulated behavior (Scarr and Salapatek, in preparation) of MZ co-twins shows about as much similarity across co-twins as across time for the same individual. If the same individual on two occasions achieves the same correlation as two MZ co-twins observed separately on the same occasion, and if DZ correlations are lower, then significant genetic effects are surely present.

Second, the relatively low test-retest and co-twin correlations for experimental measures of personality variation can be accounted

for by alternative responses which the same person can give on two occasions and which two MZ co-twins can give independently. Alternative responses that are functionally equivalent greatly reduce the seeming consistency of individual behavior over time.

Samples of behavior obtained in a limited experimental or paper-and-pencil test are probably minute examples of individuals' possible responses in that same situation and to that same test. Inventories of response hierarchies (sounds like Hull revisited) could vary greatly from person to person, so that five or six likely responses could be treated as equivalent for one person while five or six others could be treated as equivalent for another. A well-known principle of test reliability is that the length of the test is directly correlated with its reliability; an extension of this principle would say that the discrimination of individual differences on personality measures is a direct function of the number of equivalent responses that can be used to differentiate one person from another.

Observer ratings by parents, teachers, and longitudinal investigators may produce more representative samples of behavior from which to infer personal consistency than one-shot paper and pencil tests or experimental studies. Observers tend to make inferences from a large number of potentially equivalent behaviors, which are thought to be examples of the same underlying personality dimension. The actual set of behaviors suggesting consistency to the observer may vary considerably from one subject to the next. If independent ratings of individuals who vary in genetic and environmental

similarity yield a systematic set of correlations, and if MZ twins' correlations are as high test-retest reliability, then genetic factors can certainly be inferred.

Third, more consistent test-retest results for standard personality tests cannot a priori be dismissed as observer bias. Studies of MZ twins show fairly high correlations for some measures of social introversion-extraversion, activity, and a few other variables. Although the average correlation of MZ co-twins on all personality measures is only .4, their average correlation on selected measures such as social behavior approach the test-retest reliability.

A major job for behavior genetic studies may be to clarify and purify personality measures by increasing their heritability (Jones, 1971). At least for the population studied, an increase in heritability should be accompanied by an increase in reliability. Studies of twins, siblings, half-sibs, parents and children, adopted children with their natural and adoptive parents, and other persons living together and apart, can aid in the process of improving measures of persoanlity and in estimating genetic and environmental variances in personality.

Developmental strategy

A final strategy of behavioral genetic studies in personality is a developmental one, similar to an ethological approach but using standard measures of personality. With young children the typical measures are observations and ratings by others from

which personality inferences are made. The developmental strategy requires observations on family patterns of correlation over time. To save developmental studies from the pitfalls of unreliable measures may require a return to observational and rating techniques, made by blind raters. If children within several families were rated individually or observed in several situations without knowledge of their family membership, it is possible that similarity in behavior could be separated from observer biases toward similarity in sibs (the eye of the beholder problem). Consistency in ratings for individuals across situations and over time could be used as a baseline for correlations of family members at the same point in time and over time.

Recommendations

None of the cited approaches to studies of genetics and personality will solve all of the thorny problems in the area. Since personality variables are always inferences from behavior, their measurement depends upon remote operations from which investigators are willing to generalize. The following suggestions for behavior genetic approaches to personality will hopefully improve both the measures and our knowledge of the sources of individual personality variation.

1. Studies of adopted children and their natural and adoptive parents would be extremely helpful in estimating genetic and environmental components of personality differences.

2. Studies of siblings and half-siblings, living together and apart, could clarify personality variation under different conditions of rearing and different degrees of relatedness.

3. Studies of personality variation within and between families of different populations (social class, racial, cultural groups) would be interesting. Heritabilities for personality measures are almost sure to vary across populations. H^2's may be related to current socialization practices and/or selective pressures over time within various cultures.

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The Genes and Environment In the Development of Psychopathology

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Recently there has been a dramatic development in the behavior genetic study of schizophrenia. We have had our equivalent of the Michelson-Morley experiment (the adoption study) and the ether (schizophrenogenic mother) has vanished. Researchers emphasizing genetic factors in the etiology of schizophrenia have always had to contend with the plausible competing theories of the researchers emphasizing environmental factors, and one of the most prominent environmentalist notions was that schizophrenia is primarily a learned disorder -- that the parents of schizophrenics create the conditions for the learning of pathological responses. Arieti (1959) has described the mothers of schizophrenics as hostile but at the same time overprotective, overanxious, cold, and rejecting. Lidz et al. (1957) have emphasized the marital schism and marital skew present in the families of schizophrenics. When one parent tries to align himself with the child in a conspiracy against the other parent Lidz speaks of marital schism whereas marital skew refers to one parent adopting the pathological characteristics of the spouse. Implicit in both of these theories is the idea that the most schizophrenogenic of parents would be a schizophrenic one and that children reared by a schizophrenic parent should be at a risk for schizophrenia very much above the population risk of one percent. Various family studies have indeed shown that between 7 and 17% of the children of schizophrenics develop schizophrenia themselves, as opposed to

about 1% in the general population, but the point is that the recent adoption studies show these children to be at the same high risk even when brought up entirely by foster or adoptive parents.

Heston (1966) was the first to report on the incidence of schizophrenia among a group of children born to schizophrenic mothers but separated from them at birth. Forty-seven such children were identified and followed-up when the children were 35.8 years old on the average. Five of the 47 children had developed schizophrenia by the time of follow-up for an empirical risk of 10.6% -- corrected for age, 16.6%. The adoption can't be blamed: Heston also studied a control group of 50 subjects who were born to non-schizophrenic mothers but who received the same kind of foster home and adoptive rearing as the experimental group. None of the subjects in this control group were schizophrenic at the time of follow-up. Heston's figures argue persuasively that the familial clustering in schizophrenia is genetic and not a result of schizophrenic modeling or deviant child rearing practices.

It was only two years before the Heston finding was substantially confirmed. Kety, et al. (1968) succeeded in identifying in the City and County of Copenhagen, Denmark, 33 adopted individuals who had developed schizophrenia. They also selected a matched control group of 33 adoptees who had not developed schizophrenia. Their method was to compare, for both groups of adoptees, the incidence of schizophrenia among the biological relatives with the incidence of schizophrenia among the adoptive relatives. Their results are reported in Table 1. These results clearly show a genetic as opposed to an

TABLE 1

DISTRIBUTION OF SCHIZOPHRENIA SPECTRUM DISORDERS AMONG THE BIOLOGICAL AND ADOPTIVE RELATIVES OF SCHIZOPHRENIC INDEX CASES AND CONTROLS

	Biological relatives	Adoptive relatives
Total sample of 33 index cases and 33 controls		
Index cases	$\frac{13}{150}$	$\frac{2}{74}$
Controls	$\frac{3}{156}$	$\frac{3}{83}$
p(one-sided, from exact distribution)	0·0072	N.S.
Subsample of 19 index cases and 20 controls separated from biological family within 1 month of birth		
Index cases	$\frac{9}{93}$	$\frac{2}{45}$
Controls	$\frac{0}{92}$	$\frac{1}{51}$
p	0·0018	N.S.

Numerators = number with schizophrenia, uncertain schizophrenia or inadequate personality.

Denominators = number of identified relatives.

environmental pattern for the transmission of schizophrenia. The authors conclude "that the roughly 10% prevalence of schizophrenia found in the families of naturally reared schizophrenics is a manifestation of genetically transmitted factors."

Another report agrees quite substantially with the findings of Kety et al. In his genealogic studies of schizophrenia on Iceland, Karlsson (1966) found 29 offspring of schizophrenics that had been adopted into other homes. Six of these individuals were schizophrenic while none of the 28 foster sibs who were reared in the same homes were schizophrenic. Thus the adoption studies are unanimous in their failure to support the idea that schizophrenia is learned from the parents or results from the stresses of being reared by deviant parents.

Although it appears quite unlikely, in the light of the adoption studies, that the schizophrenogenic mother hypothesis can be salvaged, it should not be concluded that the environment is of no consequence in the causation of schizophrenia. As a matter of fact, the best evidence we have on this point comes from the twin studies which also provide strong evidence that genetic factors are important. Gottesman and Shields (1966b) have summarized most of the existing twin studies on schizophrenia, and the interested reader is encouraged to consult this excellent paper. A representative study is Gottesman and Shields' (1966a) own. They found that 42% of their identical twin index cases had co-twins who were also schizophrenic (42% concordance) whereas only 9% of fraternal twin index cases had schizophrenic co-twins. The difference between

42% and 9% is testimony to the importance of genotype while the difference between 100% and 42% indicates that some environmental differences exists or has existed between the identical twins to precipitate schizophrenia in one twin or to protect one twin from breakdown. One of the urgent tasks before psychiatric researchers is to try to identify the sources of these environmental differences. Mednick's (1968) longitudinal study of the premorbid psychological and psychophysiological characteristics of children born to schizophrenic mothers should be able to establish some meaningful differences between the children who develop schizophrenia and those who don't. Another promising strategy is to study identical twins discordant for schizophrenia. Pollin and Stabenau (1968) have identified 16 pre-illness factors that differentiate the ill members of identical twin pairs from the well members. The ill twins have the following behavior characteristics: neurotic as children, submissive, sensitive, worriers, obedient, dependent, well behaved, shy, and stubborn. In addition, the physical characteristics of the ill twins included such things as having had a central nervous system illness as a child, birth complications, weaker, shorter, and being lighter at birth.

Another significant development in the behavior genetic study of schizophrenia has been the refinement of the twin study as it applies to psychopathology. The earlier twin studies were plagued by the problems of sampling and diagnosis. Rosenthal (1961) has an excellent review of the difficulties, and although the intervening decade has not produced twin studies free of difficulties (the problems of diagnosis are still largely unsolved) there has been considerable improvement.

Rosenthal noted that the earlier twin studies sampled almost exclusively from resident hospital populations thereby introducting a bias toward severe chronic cases. Gottesman and Shields (1966) corrected this deficiency by collecting their twin index cases from 45,000 psychiatric patients seen as outpatients consecutively over a 16-year period at the Maudsley and Bethlem Royal Joint Hospital in Great Britain. This procedure was sufficient for the gathering of two groups of schizophrenic twins -- those with mild cases of schizophrenia and those with severe cases. The criterion for severe disorder was hospitalization for a year or more. The concordance in the co-twins of the severe index cases was 67% while the concordance in the co-twins of the mild cases fell to a figure of 20%. A similar trend appears in Rosenthal's (1961) reanalysis of Kallmann's (1946) data, showing 100% concordance when the proband's illness was moderately or extremely severe but only 26% when the illness was mild.

Generally the trend in recent twin research is to find lower concordances than reported in the earlier twin studies. Kringlen (1969) and Tienari (1968), report identical twin concordances anywhere from 6% to 38%, depending on the way the data are analyzed, and Pollin et al (1969) report a 15% concordance. Kringlen and Tienari gathered their index cases from twin registers and thereby insured the inclusion of mild and atypical cases of schizophrenia in their samples, while the Pollin et al sample actually excluded many severe cases by

virtue of the fact that both members of a twin pair had to be well enough to have served in the Armed Forces in order to get on the twin register from which the index cases were selected. No doubt there were other differences between the populations studied as well.

It should be emphasized that the later twin studies do not prove that the earlier concordance estimates, for the samples studied, were artificially inflated. The later twin studies do suggest that a co-twin's risk is much less if the proband has a mild case of schizophrenia that if the disorder is severe.

The problems of diagnosis have proved much more refractory than have the sampling difficulties. Indeed, the biological unity of the schizophrenias is seriously in question today. Rosenthal (1959) identified in Slater's twins a group of paranoid schizophrenics where the co-twin was well and there was no schizophrenia among the relatives. There, then, doesn't seem to be much of a genetic base for this kind of schizophrenia. Kallmann (1938) also has reported lower familial incidence figures for paranoid and simple schizophrenics than for the hebephrenic or catatonic types. In addition, Kety et al (1968) report, "...in the 30 biological relatives of 7 probands diagnosed as acute schizophrenic reaction, no instance of schizophrenia spectrum disorder was found. This raises a serious question regarding the validity of classifying that syndrome as a type of schizophrenia..."

Besides the attempt at identifying current "schizophrenia"

that perhaps do not deserve the label, there is a corresponding attempt to identify other disorders that are perhaps genetically related to schizophrenia or are alternate manifestations of the schizophrenic genotype. Perhaps the most promising development in this area is the current emphasis on the schizoid personality.

Psychiatric geneticists have always tried to go beyond the mere demonstration that schizophrenia is inherited to the identification of the particular mode of inheritance. The distribution of cases of strict schizophrenia among the relatives of schizophrenics has never fit any simple Mendelian pattern however. If people with schizoid personalities can legitimately be counted as carriers of the schizophrenic genotype then a much closer approximation to a single autosomal dominant pattern of inheritance is obtained. Heston's (1970) survey of data on this point is presented in Table 2 and Figure 1.

TABLE 2

Percentages of first-degree relatives found to be schizophrenic or schizoid

Relationship	Number of individuals	Schizophrenia* (%)	Schizoid (%)	Total: schizoid plus schizophrenics (%)
Children	1000	16.4	32.6	49.0
Siblings	1191	14.3	31.5	45.8
Parents	2741	9.2	34.8	44.0
Children of two schizophrenics	171	33.9	32.2	66.1

*Age corrected rates.

Figure 1

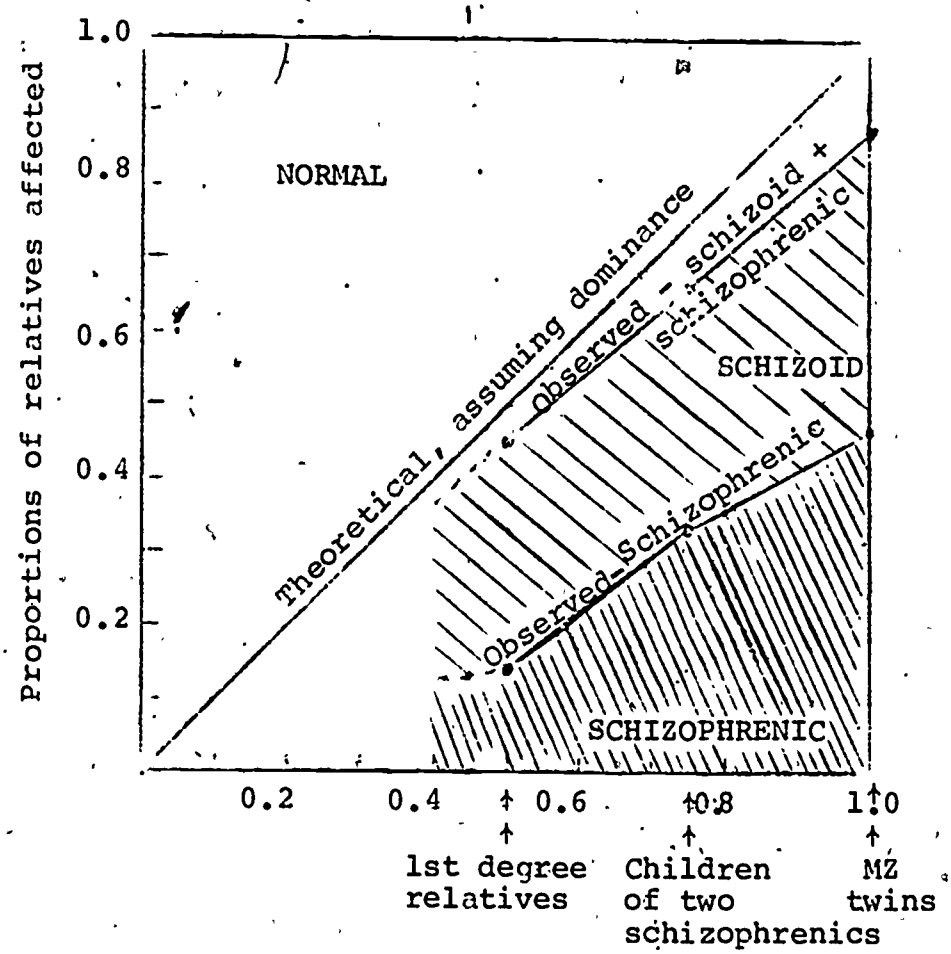

Fig. 1. Observed and expected proportion of schizoids and schizophrenics.

Heston's description of schizoids is contained in the following statement. "Rigidity of thinking, blunting of affect, anhedonia, exquisite sensitivity, suspiciousness, and a relative poverty of ideas - in variable combinations and intensities - characterize both the schizoid and the schizophrenic, though such characteristics are less prominent in the former. Though schizoids do not show a well marked thought disorder, delusions, and hallucinations, descriptions of some of the behavioral lapses of schizoids, especially

the schizoid psychopath, are bizarre enough to suggest micropsychotic episodes." This description clearly suggests a possible relationship between schizophrenia and certain of the neuroses and character disorders. Mitsuda (1967) reports that schizophrenia is found in over 21% of the families of anxiety and obsessional neurotics. Patients suffering from oversensitivity neurosis have schizophrenia in 1/3 of their families. These figures are to be compared to the less than one percent occurrence of schizophrenia in the families of hysterics, hypochondriacs, and neurotic depressives. Reviewing Mitsuda's data Rosenthal (1970) concludes that "some forms of neurotic disturbance are genetically linked to schizophrenia, whereas others are not, and that these forms should not be called neuroses but should carry another nosological classification."

A genetic association between schizophrenia and the character disorders is suggested by the results of the adoption studies mentioned earlier. Heston found 9 cases of sociopathic personality among the 49 offspring of schizophrenic mothers. Only two sociopaths were found among the 50 control group children who were foster reared also but not born to schizophrenic mothers. Kallman's (1938) data also support the idea of a genetic relationship between psychopathy and schizophrenia in that he found as much psychopathy among the first degree relatives of his schizophrenic probands as he found schizophrenia. As Rosenthal (1970) has noted, however, the genetic association between these two disorders was not found in the Rosenthal et al. (1968) and Karlsson (1966) investigations.

Given the current confusion over the proper identification of the group of schizophrenias, if indeed there is more than one, perhaps it is time to begin thinking of dropping the old nosological categories. A novel approach would be to reclassify patients solely on the basis of their response to the administration of certain drugs. The drugs could be administered singly or in sequences with an eye to both the behavioral and the physiological effects on the patients. I am encouraged in this suggestion by the very dramatic success of lithium therapy in the treatment of manias. Johnson et al. (1968) have compared the effectiveness of lithium and chloropromazine in the treatment of (1) manics and (2) schizo-affectives with psychotic excitement. The investigation was double-blind and the results were unequivocal. Fourteen of the 18 manic patients treated with lithium showed full recovery while only 4 of 11 manics on chloropromazine recovered. As far as the schizo-affectives were concerned 6 of the 7 on lithium got much worse while all 10 on chloropromazine improved. It seems that lithium is effective in the treatment of manias but aggravates the schizo-affective condition while chloropromazine is most effective in the treatment of schizo-affective disorder. Perhaps it is time to look at the various subtypes of schizophrenia and the various neuroses from the standpoint of drug response and regroup these disorders accordingly. Behavior genetic investigations of these new nosological entities might then be very instructive.

As mentioned previously one of the primary concerns in behavior genetic investigations of psychopathology has been to identify a

mode of inheritance. This concern is motivated primarily by the desire to be able to place exact probabilities on the likelihood of a disorder in a particular relative of an affected individual. With respect to schizophrenia however, genetic counselors have had to depend largely on empirical probabilities since the family distributions have not yielded simple Mendelian ratios. There is one genetic theory for the inheritance of schizophrenia that does not require Mendelian distributions however and that is the polygenic theory. The details of this theory will not be discussed here; the interested reader is referred to the article by Gottesman and Shields (1967). Briefly, though, the theory postulates a number of genes operating to predispose individuals to the development of schizophrenia.

For us at this point, the polygenic theory is important for reasons. First, it accounts for the fact that heredity is much more decisive in the severe cases (a large number of schizophrenic genes) than in the mild cases where there are, according to the theory, only a few negative genes. This fact has already been discussed. Secondly, the polygenic theory provides a way of dealing with the following paradox: schizophrenia has a sizeable genetic component; schizophrenics have only about 60% as many children as normals; but schizophrenia is as prevalent today as it was some eighty years ago. The polygenic theory can theoretically resolve the paradox by postulating that most schizophrenic genes are carried by "normals" or "schizoids" who have only a few of them, and who, because they are not clinically schizophrenic, protect these genes

from negative selection. If these individuals tend to have just slightly more children than normals, this could serve to keep a large constant supply of schizophrenic genes in the population.

There is one more important topic in current schizophrenia research that must be discussed, and that involves the specter of increasing incidence figures for schizophrenia in the future. Erlenmeyer-Kimling et al (1969) have documented the fact that schizophrenics today in New York State are reproducing at a greater rate than schizophrenics in New York State thirty years ago. They allude to the fact that part of this increase may be due to different treatment regimes today than at the earlier period. It certainly seems likely that community treatment of schizophrenics will enhance their reproductive capacities over hospital treatment, and given the evidence of a genetic predisposition to schizophrenia it follows that more potential schizophrenics will be produced. Certainly, more evidence is required on this issue, but the possibility of significantly elevated future rates of schizophrenia is serious enough to warrent keeping this possibility in mind in planning research and treatment in this area.

There have also been some recent dramatic developments in research on manic-depressive psychosis. What may turn out to be a big breakthrough was the identification of two genetically distinct disorders masquerading under the one heading of manic-depressive psychosis. Following Leonhard's lead, Perris (1966)

divided the psychosis into two kinds: bipolar, where both manic and depressive episodes occurred in the same individual, and unipolar, where there were only recurrent episodes of the same phase (depressions only, in the data to be reported from Perris's study). Perris then looked at the risk for both bipolar and unipolar psychosis among the first degree relatives of both his bipolar and unipolar probands, The results are given in Table 3.

TABLE 3

Risks among relatives of bipolar probands

Diagnosis of relative	Parents	Sibs	Children	Total
Bipolar	6.4%	13.4%	4.6%	9.8%
Unspecified affective	1.8%	2.8%	3.1%	2.3%
Suicide	6.4%	4.8%	--	4.9%
Unipolar	0.9%	0.3%	--	0.3%

Risks among relatives of unipolar probands

Diagnosis of relative	Parents	Sibs	Children	Total
Bipolar	0.4%	0.1%	--	0.3%
Unspecified affective	2.7%	2.3%	1.8%	2.3%
Suicide	5.4%	2.9%	--	3.2%
Unipolar	3.6%	7.2%	0.9%	5.0%

These results are important for two reasons. First, they confirm earlier results showing a greater effect of heredity in bipolar psychosis than in unipolar psychosis. There is an

almost 10% risk for bipolar psychosis in first degree relatives as opposed to a 5% risk for unipolar psychosis. Secondly, they show relatives of bipolar patients to have an increased probability of being a bipolar psychotic but having a risk no greater than the general population risk of being a unipolar psychotic. Liekwise the relatives of unipolar patients have an increased risk for unipolar but not bipolar psychosis. It therefore seems that we have two genetically distinct forms of manic-depressive illness. Winokur and Clayton (1967) came to the same conclusion by an entirely different route. They divided a heterogenous group of manic-depressives into two groups on a basis of family history. There was a group with a clear family history of affective disorder (at least a two generation history) and a group without any history of affective disorder. There was a significantly greater number of manics in the positive family history group than in the negative family history group, the latter of the two being equated to the unipolar depressive type of psychosis.

In 1951, Merrell summarized the evidence then available on manic-depressive psychosis and concluded that genetic factors were definitely implicated in the etiology of the disorder and that a single autosomal dominant gene was the most likely mode of inheritance, after one made allowance for the fact that not everyone with the gene showed the trait. Fuller and Thompson (1960) came to essentially the same conclusion. Recent work has taken advantage of the unipolar-bipolar distinction to give a different picture of the mode of inheritance. Winokur and Reich

(1970) have defined manic-depressive psychosis as an affective disorder where at least one episode of mania is seen. These investigators note that when this definition is applied in the selection of manic-depressive fathers there are decidedly fewer manic-depressive sons observed than expected; a pattern that tends to implicate the sex-chromosome. In addition, linkage studies have shown an association between the Xg blood group (an X chromosome trait) and manic-depressive psychosis. These investigators also hypothesize a second genetic factor in the inheritance of manic-depressive psychosis. "In sibships where one manic exists and in the children of manic parents, 43% of the affectively ill members of these sibling groups show mania. This is not very far from what would be predicted (50%) if a second gene, an autosomal dominant, were involved in the transmission of the disease...Alcoholism may be one way in which such a second factor may manifest itself in the absence of the primary X-linked dominant contribution."

Of all this new information, perhaps the most important from the standpoint of the genetic counselor or clinical researcher is the possibility of identifying closely linked marker genes for the manic-depressive trait. With this information all one would have to do is see if a presently well child matched marker genes with an ill parent in order to judge the child's susceptibility to developing the disorder. With early detection of susceptible individuals research into prophylatic measures should be much facilitated.

Numerous behavior genetic investigations have been carried out in the areas of neurosis, criminality, homosexuality and alcoholism. There are very few firm conclusions that can be drawn from the research in these areas. In all likelihood the major difficulties stem from the heterogeneity of the individuals categorized together for the purposes of the investigation. Similar difficulties have been reviewed for schizophrenia and manic-depressive psychosis but the problems in the areas mentioned above are greatly magnified. In general it seems safe to say that the evidence from the family, twin, and adoption studies performed to date do indicate the presence of a perceptible genetic factor in the etiology of these disorders. The interested reader is referred to Rosenthal (1970) for an excellent survey of the research; perhaps a quotation from the section on alcoholism will serve to give the flavor of his conclusions.

> "In a study of alcoholics admitted to the Payne Whitney Clinic in New York, Sherfey (1955) found that 8.7% had schizophrenia (mostly paranoid), 6.8% had manic-depressive psychosis, 6.8% had poorly organized, asocial psychopathic personalities, 4.3% had epilepsy or epileptoid reactions, 3% had brain damage, 13.6% were males with obsessive-compulsive personalities, 10% were females with rigidly organized neurotic personalities with paranoid features, 18.6% were males with poorly organized inadequate psychoneurotic personalities, 7.4% were females with dependent psychoneurotic personalities with depression and tension, and 6.8% had depressions of middle and late life. Other studies have consistently revealed a wide variety of disorders among alcoholics. Kaij has questioned whether a number of neurological disorders thought to be the consequences of alcoholism may not indeed have antedated and perhaps even precipitated the alcoholism. We must indeed assume multiple motivations and etiologies in the persistent resort to alcohol of many individuals and among them perhaps an inherited factor which may be simple or heterogeneous, primary or secondary."

In summary, what can be said of the behavior genetic study of psychopathology? With respect to schizophrenia and manic-depressive psychoses we know beyond a reasonable doubt that genetic factors are implicated in their etiology. Unfortunately we are completely ignorant of the biochemical mechanisms underlying these dysfunctions. For these psychoses we also know that environmental factors are of some importance, but we cannot say just what environmental factors. Obviously, if we are going to be successful in treating or preventing these disorders we have to know more precisely just what it is that causes them. A comprehensive longitudinal study of monozygotic twins born to a psychotic parent might go a long way towards determining the potent environmental variables. On the biological side more effort must certainly go into the discovery of the underlying physiological mechanisms. Leads in this direction might come from the biochemical study of certain single gene disorders that have associated with them a significant amount of psychiatric disability. Dewhurst, Oliver, and McKnight (1970) found that 37 of 102 Huntington's disease patients were initially diagnosed as psychoneurosis, personality disorder, affective state, or schizophrenia. Still other leads may be found in the study of dopaminergic, norodrenergic, and serotonergic pathways in the brain (McGeer, 1971). Stein and Wise (1971) have made a very interesting beginning in this area.

With respect to the neuroses, alcoholism, suicide, and criminality it can only be said that more and better behavior genetic work is required before we can have much confidence in the importance of genetic factors.

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APPENDIX

Outline for PEM Study Adopted for Planning Purposes

(Detailed changes have been made by Task Groups at the discretion of group members.)

1000. PEM Aspects of Child Development

1100. Special Problems in Infancy and Early Childhood (birth to 5 years)

1101. Group care
- 1. Effects of orphanage rearing, multiple mothering vs one-to-one mother-child (or surrogate mother) relations
- 2. Related effects of environmental complexity

1102. Separation anxiety; fear of the strange

1103. Readiness
- 1. General concept
- 2. Special application to disadvantaged children

1104. Forced training ("pushing")
- 1. In relation to "natural" intellectual limits
- 2. In relation to readiness

1105. Sequential organization of learning
- 1. In infancy
- 2. In early childhood

1106. Parental involvement and influence on early development
- 1. Effects of home environment, of implicit theories and practices of parents
- 2. Manipulation of parental beliefs and practices, in enrichment programs

1107. Modes of learning and experience that affect early behavioral development
- 1. Differential effects on anatomical maturation and behavioral development
- 2. Correspondence between rates of anatomical and behavioral development
- 3. Effects of environmental (experiential) enrichment and impoverishment, and cumulative effects with increasingly complex circumstances
- 4. Hierarchical conceptions of intellectual development (Piaget)
- 5. Development of learning sets and their implications for intellectual, motivational, and personality development; resistance of resultant behaviors to extinction
- 6. Critical periods

1200. Child Socialization

1201. Conceptualization of the socialization process
- 1. Socialization pressures
- 2. Learning paradigms: e.g., dependency relations and adult control of "effects" (reinforcement), reference group formation

1202. Internalization of beliefs and values
  1. Conceptualization of attitude, belief, and value systems
  2. Identification processes
  3. Impulse control (self control)
  4. Effects of environmental resources
1203. Cognitive socialization
  1. Psycholinguistic structures, language development: effects on thought, beliefs, attitudes, interests; patterns of expression, values
  2. Uncertainty and information-seeking
  3. Development of expectancies; category accessibility; assimilation; effects on perception, cognition, action
  4. Symbolism, symbolic behavior

1300. Personality Development
1301. Developmental theories (Freud, Erikson, Piaget, Sears)
1302. Developmental sequences, stages
  1. Critical periods
  2. Fluid and crystallized patterns of intelligence (Cattell)
1303. Development of self-identity
  1. Self concept, ego theories, self theories
  2. Relations to social class, racial-ethnic factors, region, sex, family characteristics
1304. Effects of age, sex, culture, and other environmental factors
1305. Development of mechanisms of coping and adaptation

1400. Behavior Change
1401. Personality, learning
1402. Susceptibility to change of personality traits, attitudes, interests, beliefs, values
1403. Measurement of change
1404. Genetic, maturation, and learning factors in physical and psychological growth

2000. Personality

2100. Conceptual and Theoretical Approaches
2101. Criteria for a viable theory
2102. Development of unified, integrated theoretical formulations
  1. Cross-level comparisons and correlations
  2. Developmental histories of stable traits
  3. Relations among trait patterns at various developmental levels
  4. Relations of traits to perceptual responses in person perception and interpersonal interaction

2200. Cognitive Conceptions

2201. Cognitive style, complexity
2202. Balance theories
2203. Cybernetic formulations
  1. Computer simulation of personality
  2. Mathematical models

2300. Developmental Approaches (see 1300)

2400. Dynamic Approaches (see 1303, 4000)

2500. Morphologic Approaches

2600. Physiologic, Psychophysiological, and Biochemical Approaches (see 2102.1)

2700. Trait Structure, Multivariate Approach - Taxonomy of Trait-Explanatory Concepts of Stylistic and Temperament Aspects of Personality
2701. Methodological problems: definition of universes of behaviors for self-report, observation-rating, and objective test studies, cross-media matching of stable structures, design paradigms, including multi-modality designs and trait x treatment designs; construct validation of traits; effects of age, sex, sample, culture, and other environmental effects, and relations of these to resulting trait patterns; the range of roles and sets in relation to diversity of response patterns obtained (social desirability, acquiescence, and other specific sets), their similarities in terms of effects on self-description, and the relations of traits to moderator variables representing such sets
2702. Observational, rating methods: rater and "ratee" sources of effects in peer and "other" ratings, in observational trait assessment, and in interpersonal interaction; explicit concern with task, stimulus presentation, response format, socio-environmental setting, and demographic characteristics of participants; conceptual and empirical relationships among similar and related trait descriptors within observational-rating subdomain and in other subdomains (self-report)
2703. Self-report methods: item pools; format; item vs cluster factorization; measurement of and correction for response bias or distortion; development of a unified, consistent conceptual framework for concepts of personality style and temperament
2704. Objective test, misperceptive, indirect assessment, and development of fresh, new approaches to personality measurement and description

2800. Creativity
2801. Conceptualization of creativity; relations to intelligence, personality factors

2802. Characteristics of the creative person
2803. Analysis of the creative process
2804. Characteristics of the creative product
2805. Characteristics of the creative situation, short- and long-term; situational factors contributing to creative performance
2806. Measurement of creativity

3000. Emotions

3100. State Patterns: Physiological, Cognitive, Behavioral
3101. Arousal stimuli
3102. Response dimensions
3103. Uniqueness
3104. Learned-unlearned dimensions
3105. Affective learning; autonomic and physiological learning

3200. Relations to Traits, Roles

3300. Moderation of Expression by Learning
1. Culture patterns
2. Age, sex, group norms

3400. Drug Effects on Emotional Patterns

3500. Differentiation of States, Reflecting Situational, Organismic, and Stimulus Variations, from Traits, Represented as Long-Term Individual Dispositions

3600. Arousal States: Adrenergic Response, Stress

3700. Dysphoric States: Anxiety, Depression, Guilt, Shame, Remorse (see 4300)

3800. Duphoric States: Happiness, Elation, Joy, Hope, Confidence

4000. Motivation

4100. Conceptualization and Theory (human motivation)
4101. Homeostatic systems, physiological need
4102. Need-press system (Murray), subsystems (n Ach)
4103. Dynamic systems (Freud, Cattell)
4104. Cognitive and cybernetic approaches: motivation inherent in information-processing functions (Hunt), cognitive dissonance theory, incongruity, collative variables (Berlyne), balance theories, exchange theory
4105. Motivation inherent in individual performance, competence motivation (White)
4106. Trait systems and patterns (Guilford, Cattell)
4107. Values systems, moral character
4108. Conceptualization of interest, attitude, need, belief, value, ideal

4200. Process and Trait Formulations
4201. Relations and differences in conception and approach
4202. Process theories and formulations
1. Balance theories
2. Exchange theory
4203. Trait formulations: motives, values, character traits
1. Methodology of measurement: Strong paradigm, Thurstone scales, Likert scales, Cattell's and Campbell's indirect approaches: self-report, objective, misperception, observation, rating, content analysis, unobtrusive measures
2. Analytic approaches: factor analysis, multidimensional scaling, profile clustering
3. Factored patterns of sentiments, attitudes, interests, beliefs, values
4. Variations related to age, sex, sample, culture, and other environmental factors

4300. Frustration, Stress, and Anxiety
4301. Frustration theory and research evidence
4302. Conceptualization of stress
1. Relation to frustration (Selye)
2. Utility of stress concept in interpretation of behavior
3. Relationships among physiological and psychological aspects
4. Stress and coping, adaptation
4303. Adaptation-Level Theory (Helson) (see 5100)

4400. Conflict
4401. Conceptualization of conflict (Miller, Murphy, Cattell)
1. Types of conflict: role, value, internal
2. Approach and avoidance relations
4402. Conflict measurement and calculus
4403. Conflict in relation to interpretation and prediction of action

4500. Interests and Vocational Guidance
4501. Incremental value of interest measurement over ability and aptitude measures in predictions of various criteria on various populations (Thorndike, 10,000 Occupations; Clark, Minnesota study)

5000. Environmental Variables

5100. Conceptualization of Environmental Variables and Their Effects on Behavior; Human Ecology

5200. Methodologies for Encoding Environmental Factors

5300. Taxonomic Systems of Environmental Variables

5400. Normative Studies of Selected Behaviors in Relation to Defined Patterns of Environmental Setting: Sampling Problems in Relation to Populations, Behaviors, Macro- and Micro-Environmental Settings

6000. Interpersonal Behavior Processes

6100. Group Theory, Role Theory, Interpersonal Settings

6200. Interpersonal Perception, Attraction, Influence; Social Acuity, Empathy

7000. Variations in Psychological Processes

7100. Paradigms for such Research, Taking Account of Persons, Tasks, Environmental Settings, and Occasions (Cattell covariation chart, Campbell-Fiske model, longitudinal replication)

7200. Paradigmatic Studies of Selected Learning, Motivation, Perception, and Other Psychological Processes to Investigate Variations Attributable to Shifts in Subject, Task, Setting, and Occasion Dimensions

7201. Analyses to estimate magnitudes of variance components in standard dependent variables accounted for by trait, treatment, and trait by treatment sources and their specific constituents

7202. Analysis of total interaction parameter estimates into principal components or other dimensions in order to compare results by such methods with conventional R, P, Q analysis, both with single dependent variables and vectors (multiple dependent variables)