Multivariate Analysis of Twin Differences

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It is useful in thinking about psychological contributions to human genetics to adopt a scheme of stages or degrees of knowledge about hereditary mechanisms in man as a way to place a lot of facts in relation to one another.

In the most advanced stage, we would have knowledge about a condition due to one gene located in a known region on a given chromosome; in general, human genetics has not reached this state yet, although we are approaching it for sex-linked traits. We have not reached this stage because there are very few firmly established linkages, so we have not been able to construct chromosome maps in man.

The next best stage of knowledge is one in which the gene can be assigned to a specific chromosome although not to a definite location on it. Once there are several, widely spaced, markers for each chromosome one could determine the chromosome on which other single gene traits are located. In human genetics, this stage also has been reached only for genes on the sex chromosomes. Tentative locations for such genes will probably soon be suggested by studies of chromosomal abnormalities.

A stage of yet less exact knowledge is where we can only distinguish between autosomal and sex-linked traits. Varying degrees of ignorance might further be distinguished within this stage; for instance, we may suspect that a single gene is responsible for a given trait, but, because the condition is a rare one, lack enough cases to work out a precise mechanism; or, one gene is thought to be responsible but other diseases may lead to conditions closely resembling this one, so called phenocopies, resulting in a diagnostic

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problem. I should emphasize that I am thinking especially about problems of neuropsychiatric or psychological conditions or phenomena.

Then we might distinguish situations where more than one gene is involved. If it turns out that there are only two or three genes, pedigree studies might still be useful, and we can try to build various models for combinations of genes and test how well such models fit actual data. We could do this right now, I think, on such things as height and handedness; perhaps even intelligence, if we are willing to regard intelligence as a general factor.

Finally we may have conditions controlled by many genes. Here we have to revert to the methods of biometrical genetics: analysis of variance, regression analysis, path coefficients, and heritability estimates and this is where twin studies come in. Regardless of the number of genes involved, we often have situations in which the effect of the gene can be so varied as a result of interaction with the environment that pedigree studies are, at the moment, not profitable. In such situations you can still do biometrical genetic studies, but conclusions from similar studies done in different locations may differ if the environmental conditions vary in the samples used. Here anything we can learn about environmental effects would make later studies of hereditary factors better, if not easier. As a matter of fact we do not have very good ideas yet about how theories derived from studies of environmental effect can be fitted into biometrical genetic studies. A demonstration with appropriate theoretical models could help clarify this whole problem and set the pace for future work. It would be good to take a particular phenomenon, such as height, which is understood fairly well, and work it through in as much detail as we can, to provide a model for studies of other variables. Such an approach can lead to more adequate models than the one in which heredity and environment are proposed as two independent sources of variance which together add up to 100%. The multiple abstract variance analysis, proposed by Cattell (1953), in which between-family and within-family hereditary and environmental variances are considered, as well as various interaction terms, is a good example along these lines. Finally it is well to keep in mind that complete knowledge of hereditary mechanisms is not necessary for doing useful applied work. In animal breeding, production of milk,
It is true, however, that rather drastic selection is necessary to achieve such improvement. The mere elimination of poor producers would, in subsequent generations, alter production little or not at all; for this reason such methods would not work as eugenic measures with man.

My own background and temperament has led me to choose problems near the bottom of this list rather than near the top and this is dictated in part by previous work I have done, some familiarity with statistical techniques, and other things in my personal history, but in no way reflects a value judgment about what is more important. Furthermore, for what are socially the most significant and most important psychological problems, we are more or less forced to work at the bottom of this list, if we are going to make a start right now. A few more general remarks may be in order before I go to the area of twin studies. For neuropsychiatric problems it is still useful to get more adequate surveys of the incidences of various diseases in various parts of the world, because unless there are marked geographical differences in gene distribution, the presence of similar incidence rates under widely differing cultural conditions would tend to point toward hereditary factors. These studies of incidence rates could be made more profitable if they were combined with the collection of biochemical information. We can also do family studies and studies of incidence rates in relatives of patients, and work of this kind should certainly be done more widely.

Finally we come to twin studies. Twin studies are, perhaps, a more economical approach to human behavior genetics than family studies, as long as we are completely ignorant of hereditary factors. First of all, the twin-study method eliminates the age factor which we cannot deal with in many of our variables. Even in studying height, age is a serious problem; if you want to do pedigree studies you can start with adults which limits the accessible range of relatives somewhat, or if you start with children you must wait until they reach adult status before you can compare their measures with those of their relatives. In twin studies you do not have to worry too much about the sampling problems at this stage of the game. For the study of normal variations, twins can furnish quite a bit of information. We will not be able to work out precise mecha-
anisms, but we can do a lot of screening and look for promising variables or areas in which variables might be found that can subsequently be studied by other techniques. I think that we need a lot more work on normal psychological variation before we are ready to do pedigree studies with existing measures of cognitive or personality variables.

Now I would like to review what can be done with twins. First of all, of course, there is the conventional comparison of the concordance rates of identical and fraternal twins, either for qualitative traits when you are looking for all-or-none phenomena, or for quantitative traits when you compare fraternal and identical within-pair variances and evaluate the statistical significance of their ratio with the usual F test. There must be replications of these studies because there can be marked fluctuation in the results of twin studies by different investigators. There have been a fair number of twin studies, but we are only now reaching some uniform conditions, such as the determination of zygosity by standardized methods. It is difficult to compare results that were based on other methods of determining zygosity. Furthermore, the samples are usually rather small, a fact for which no investigator can be blamed, but one which will lead to fluctuation of results; therefore replication is almost essential before we can base firm conclusions on twin studies. I hope that we will find a good deal of agreement when we do the replications. The second point I want to make is that it will be very interesting to try to understand why there are discrepancies between different studies and why a seemingly small change in a psychological test can lead to quite different results in heritability estimates. This may give us a new kind of experimentation in which we try to change heritability estimates by maximizing hereditary factors in a particular psychological variable, or maximizing environmental aspects, sometimes perhaps by a rather small change in the measure used. Still, within the framework of the conventional twin studies, we should be alert to the fact that when we collect such data, which, although economical in comparison with family studies, still are rather expensive, we have the opportunity to perform several other analyses on the same data. First of all, since we do collect the information on the blood types, it might be worthwhile to check routinely for linkage of any of the variables with the blood types
using the fraternal twins as sibships of two cases. One of these studies alone will not give any firm information because the samples will be too small, but if there are consistent results between studies (and everybody is going to have to collect the information on blood types, anyway), it is possible that we would find something. I am not hopeful that this is going to lead to many discoveries, but it is not going to be very difficult to do. Secondly, when we have identical twins we can ask why some identical pairs are more divergent than others. While this is not going to tell us much about heredity, it will tell us a lot about environment. It may be a valuable complement to the study of hereditary factors particularly if this is in work with very young twins, where we can still secure accurate childhood histories. Next we can do conventional correlational studies. Finally, I would like to summarize a few reports to show further methods of getting more information from traditional twin studies than we get by the usual passive collection of twin data. We can introduce at least some experimental dimension. We normally consider variables either as all-or-none phenomena, where we look for the presence or absence of concordance, or as continuous and then we do analysis of variance. Dr. Stafford points out that it may sometimes be fruitful to break this set. For instance, PTC (ability to taste phenylthiocarbamide) was formerly considered an all-or-none phenomenon (this is the ability to taste a substance that tastes bitter to most people), and yet it is actually a continuous variable. Conversely, we might take some psychological variable known to be continuous, but just set an arbitrary cutoff point and classify people above a certain score as having this attribute and those below as devoid of the trait. This may be more economical and it may lead to clearer results. It is not impossible, for instance, that such a crude classification might be useful in studying the inheritance of pitch perception. We can also go the other way—color vision is traditionally considered an all-or-none phenomenon, but Pickford’s work (1951) seems to indicate that it might be more accurate to call it a continuous variable. Most people are at one end of the distribution, but there is still enough spread within the normal range to give the usual genetic models interesting complications. Another approach has been tried by Loehlin (p. 163 of this volume). He takes information obtained on identical and fraternal twins, looks for items
which give a high concordance in identicals and relatively low concordance in fraternals, then constructs a new test from items which will give you high heritability to see what sort of an animal you have. One can analyze the collection of items one has obtained to see if one dimension underlies the collection or whether there are more. Gottesman in his chapter will have something to say about that too. In this way an experimental approach is added to the traditional twin-study method. This is particularly emphasized because both the analysis of my data and comparison with other data have failed to yield a short easy generalization of my results. When I thought about this, an idea which is certainly not new struck me with great force; namely, some psychological traits may have a simple genetic mechanism controlling them, but these traits with high heritability are not necessarily variables which we are currently looking for, or even aware of. Most of the variables which are of interest to genetics today were discovered accidently. Even the "obvious" trait of color blindness is a rather recent discovery, yet people must have had this defect for centuries before science became aware of this polymorphism. We may have missed many differences because they have no applied usefulness at this time. In general, most of our psychological tests have been constructed for their usefulness in predicting success in graduate school, or in elementary school, or whether or not one is going to have problems when he enters the armed forces, etc. We have to start with these tests, but we shall need new instruments for studies of the genetic bases of psychological traits.

A few more methods will be mentioned that should be used to get needed information. First, I cannot make a strong enough plea for new co-twin control studies with samples larger than two or three pairs of twins and with clear aims. For instance, one could test the prediction that systematic training of one twin in concept formation at age 3 would give differences between the twins at elementary school age which would be relatively stable, or would not be stable depending on the type of training. Or, one could take a small number of identical twins and give one of each pair early training in reading to see if there were persistent, or even permanent twin differences. This would be a very valuable complement to studies of the role of heredity. Again, sometimes we can capitalize on accidents. In Sweden, a psychiatrist, Dencker (1958),
studied the effect of brain damage, relatively independent of hereditary and early childhood factors, by comparing brain-damaged twins with their unaffected identical twins. We must be more original. There are probably many studies of this kind that could be done with various diseases if one could find enough identical pairs in which only one twin has the disease. A Swedish psychologist, Naeslund (1956), a former student of Husen, I believe, used a very ingenious way of studying the effectiveness of competing methods of teaching. He took 18 pairs of twins, including 10 identical pairs, and separated them into two classrooms. In one room, reading was taught by the phonics method and in the other by the sight method. Two teachers rotated between the two classes to control for the influence of the teachers. The phonics method was superior for average children, but for gifted children there was no real difference; so there is no clear-cut answer for all children as you would expect. I think this is a wonderful way to do research on teaching methods and one which should be used more. I would also like to make a plea for the study of identical twins discordant for mental illness. A start has been made; I was told that the National Institutes of Health is organizing a registry for such pairs all over the country.

I would like to conclude by presenting an idea for multivariate analysis of twin data. A formal presentation of this usually causes difficulties in understanding on the part of the audience, so I am going to retrace my own thinking instead. While not as elegant, it may be a more psychological presentation since in following my attempts to deal with the problem you will, I hope, arrive at an intuitive understanding of my solution.

In the Michigan twin study, we found statistically significant results for so many variables that people keep asking me: “Do you really think that all these variables are different with independent hereditary control for each?” Well, I did think that this was true for many of the variables, but there was no way I could prove it. If the conventional correlation coefficient obtained in some study of unrelated people shows a very low correlation between the scores on two tests, it does not say anything about independence of genetic mechanisms or genetic control over these two traits. Even if each of the variables had a high heritability and was the result of, or was influenced by the same hereditary sources of variance
they could still have a low correlation if different environmental factors acted on each trait and vice versa. First of all I asked whether the twin that was taller was also the brighter twin, and I did this for identical and fraternal twins separately. For clarity I will consider height and intelligence as two very simple traits. Let me give you just two correlations now. I will go to psychological variables to make the discussion more concrete. If we intercorrelate the differences on the Information and the Vocabulary subtests of the WISC for elementary school age identical twins we get a correlation of .06; in other words, whatever it is in the environment within the family which produces differences or is associated with differences on the Information subtest is not related to whatever produces differences in the Vocabulary subtest. However, the intercorrelation for the differences on these same tests for fraternal twins gives a somewhat different picture—the correlation is .56. To keep the argument fairly simple, we have here the same within-family differences operating to produce (or associated with) differences on these tests, but in addition we have hereditary twin differences which can contribute to (or lower) the correlation. Here I would be tempted to conclude that this hereditary component has led to the greater correlation between the twin differences. I think this is essentially correct reasoning, but it is very awkward and we have lost some very significant information. In calculating correlations, we really lose sight of the size of the twin differences; each of the two correlations is calculated around its own mean with its own standard deviation, so we have really thrown away our most significant information, which is that differences tend to be greater for fraternal than for identical pairs. So we should compare covariances and we should do this simultaneously for a number of variables, because it is very awkward to treat successive pairs of variables and later reconcile findings on variables A and B, and variables B and C, and on A and C, etc. What we want is a method for treating a set of variables simultaneously, to determine whether or not they contain a number of relatively independent hereditary components. Well, given two tables of covariances of twin differences, one for identical and one fraternal twins, could an investigator subtract the identical twin difference covariance matrix from the fraternal twin difference covariance matrix? The covariance matrix for the fraternals is due
to hereditary and environmental factors while the one for identical twins would only be due to environmental factors. Thus, if subtracted, one would have hypothetical hereditary covariances left. This is, of course, not permissible for a number of reasons. First of all, mathematically, if you subtract two matrices you may get a matrix that is no longer Gramian, that is, it no longer has properties such that you can obtain roots and vectors. Intuitively one might see that the implied relations between the variables obtained by the subtraction may be incompatible. Furthermore, this subtraction is incorrect genetically, because heredity and environment are not merely additive components; there is a strong interaction component that may actually be larger or more significant than either of the two separately. Yet we would like to do something of this kind, and it turns out that there is something we can do. Let me remind you what we do with just one variable: We calculate the ratio between the within pair variance for fraternals and for identicals and then test the significance of this ratio by the F test. Now we must generalize the F test to a multivariate test. The mathematical equivalent of getting a ratio between two covariance matrices is to find a matrix such that multiplying one of the original ones by it yields the second. Mathematically this is quite straightforward, although not easily calculated without a computer. Then we can ask how many roots are significant in the resultant matrix, which is the multiplier or transformation matrix that produces the fraternal from the identical covariance matrix, and this will tell us how many independent sources of hereditary variance there were in our data. Here I have to insert warnings about the size of the sample, about the fact that you cannot interpret mathematical models in terms of real phenomena without reservations, and so on. We can also ask whether certain variables have independent hereditary components, and I suspect that we may even, to some extent, be able to identify certain hereditary components of variance with particular variables, somewhat as you would in factor analysis. What we have been talking about is solving an equation of the form \[ \begin{vmatrix} D - \lambda M \end{vmatrix} = 0, \] where \( D \) is the covariance matrix of fraternal or dizygous twin differences and \( M \) the covariance matrix of the identical or monozygous twin differences. We are obtaining a multiple discriminant function on the differences of the scores of the two types of twins. We get a composite score from
our differences that will discriminate identical and fraternal twins as well as possible, then we remove the effect of this first composite and find another composite that will again give us the best possible discrimination and continue until we have exhausted the variance or until we cannot make any further discrimination. This is really a very conventional technique but one has to grow accustomed to thinking in this way, at least it took me a long time to see the hereditary implication.

This method has been applied to the six scores of the Primary Mental Ability Test (PMA) (Thurstone, 1941) that was administered to 45 pairs of identical and 37 pairs of fraternal twins as part of the Michigan Twin Study.

The over-all design of this study has been described by Sutton, Vandenberg, and Clark (1962). The zygosity diagnosis was based on a decision function derived from the results of tests of blood group systems (see Sutton, Clark, & Schull, 1955 for details about this procedure).

The equation \( D - \lambda M = 0 \) was solved for the six PMA difference scores and the six roots shown in Table I were obtained.

| Roots | \( | D - \lambda M | = 0 \) |
|-------|-----------------|
| 1     | 3.99972         |
| 2     | 2.23172         |
| 3     | 1.58655         |
| 4     | 1.00141         |
| 5     | .64598          |
| 6     | .38206          |

To test the significance of these roots Bartlett's chi squared test of the homogeneity of the remaining roots after extraction of \( 1, 2 \ldots k \) roots was applied (Bartlett, 1950). The results are shown in Table II and indicate that at least four roots are significant, or in other words, that in this sample there are at least four independently significant hereditary components.

It is of interest to compare these roots with the F-ratios for the DZ
over the MZ within pair variances for the six original scores. Table III shows these F-ratios; four of them are significant beyond the 1% level.

We concluded that there are at least four independent components in the six PMA scores of the subjects in this sample. Since the analysis of the single scores led to the conclusion that number,

<table>
<thead>
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<th>k</th>
<th>Chi square</th>
<th>d.f.</th>
<th>p</th>
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<tbody>
<tr>
<td>1</td>
<td>30.698</td>
<td>20</td>
<td>between .10 and .05</td>
</tr>
<tr>
<td>2</td>
<td>17.483</td>
<td>6</td>
<td>less than .01</td>
</tr>
<tr>
<td>3</td>
<td>7.250</td>
<td>2</td>
<td>less than .05</td>
</tr>
<tr>
<td>4</td>
<td>2.147</td>
<td>.5</td>
<td>n.s.</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0</td>
<td></td>
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</tbody>
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verbal, space, and word fluency were under a statistically significant degree of genetic control, it may be warranted to conclude that the four independent components represented by the significant roots of \(|D - \lambda M| = 0\) are rather similar to the number, verbal, space, and word fluency abilities. More details about this analysis are given in another place; Vandenberg (1965).

<table>
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<tr>
<th>F-RATIOS OF FRATERNAL OVER IDENTICAL WITHIN-PAIR VARIANCES OF SIX PMA SCORES FOR 37 AND 45 DEGREES OF FREEDOM</th>
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<tbody>
<tr>
<td>Number</td>
</tr>
<tr>
<td>Verbal</td>
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<tr>
<td>Space</td>
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<td>Word fluency</td>
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<td>Reasoning</td>
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<td>Memory</td>
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I shall close with two more remarks. I overlooked, for quite a while, the fact that before one compares results between identical and fraternal twins there is an opportunity to look at identical twin differences only and their correlations. This provides a method
for studying the extent to which within-family differences between twins on different variables are correlated. This is a way of studying in great detail at least a limited aspect of the environment, and with information on early childhood experiences, one has a really powerful way of studying how these experiences affect cognitive variables, personality variables, etc.

References


Discussion

Gardner: I’d like to say, Bravol for this method of treating twin difference data to get at underlying relations. In practice this should give us answers we have long wanted.

Vandenberg: I think it does. I would prefer to work with larger matrices because I am worried about sample size. This is one reason why I have been holding up publication till I have repeated this with a larger sample.

Gardner: When you get down to the point of counting the number of significant latent roots you really have to worry about sample size. When you are concerned with replications you are on the right track.

Vandenberg: I think so. Next we should take a variable that has repeatedly shown evidence of a hereditary factor and subject it to a procedure of “refining” for the purpose of increasing heritability. Then you could finally go to family studies.
Freedman: I should like to stress that each identical twin pair has a different inheritance, so to speak, from every other identical twin pair. When we lump these different identical twin pairs together to extract what is inherited in common from among them, we lose so-called genetic individuality. We come up with something which is necessarily less than what is actually inherited, or may be inherited.

Vandenberg: I couldn’t agree more. I have just two comments on that. First, I think there is a great deal of uniformity in the way we acquire the primary or most important cognitive abilities, so there may be less difference here than with personality variables. Second, this is merely a link in a chain that will, maybe 10 years from now, lead to some fairly precise knowledge about hereditary mechanisms in human behavior. This method allows us to take two variables that we have considered independent and test for a relation in the genetic component. If we find such a relation we can look for a way of constructing a new test, a measure of this new construct which has some characteristics of both variables. We would then be one step closer to the underlying hereditary component. In the personality area, everybody may be somewhat unique and it may be difficult to find measures which give a high heritability index, let alone a neat genetic mechanism. We may always be limited somewhat by this uniqueness. But I’m not pessimistic about the cognitive area and prospects may not be poor in personality studies either, particularly with certain gross characteristics as the schizoid personality.

Freedman: Yes, there are certain maturational events that are certainly common to the whole human race, such as smiling and sitting up.

Stafford: Your report about the Swedish psychologist studying learning is very apropos. We often try to find the way of teaching reading. In fact, the best way is undoubtedly going to differ depending upon the child. It is probably different at different maturational levels. If there are two discrete types, phonics and sight readers, these differences may be hereditary. One other question, did you mention linkage?

Vandenberg: Yes, I mentioned linkage with blood types as a by-product of twin studies. Eventually we are going to have a fair amount of information on the blood types of fraternal twins, their height, their shoulder widths, their scores on some tests, etc. and it seems a shame to have this unused. Other people might be going to great expense to collect these data.

Stafford: I wonder if it would expedite our research and possibly make communication a little easier if we took the chromosomes and, applying the knowledge that we have to date of linkage, assigned letters to the known linkage groups, hoping they did not exceed 23. Later, with the help of some chromosomal aberrations we might be able to tie C to chromosome 17, or A to 9, etc. I do not know if this would be helpful, and it might never come to pass, but certainly it would make communication easier.

Lasker: I am not sure that an arbitrary numbering system would be better than what we have. We have letters now for the three known linkages on autosomal chromosomes, one is with ABO, one is with Rh and one is with
MN, if I am not mistaken. An arbitrary system might only confuse matters as it has with numbering the chromosomes by their length and shape, and perhaps as Patau has claimed, the Denver system has already gone too far in trying to number even the morphology of the human chromosomes. One cannot always tell the difference between different chromosomes. For instance, in a preparation one cannot always distinguish the X chromosome from an autosome.

Sutton: The possibility of exact identification of particular chromosomes varies with both the preparation and the investigator. Some investigators claim that they can identify every chromosome, and others can as convincingly point out where they cannot. I think the hope still exists that one can by use of various accessory techniques, but there is danger in falsely precise labeling.

Vandenberg: I think some people may not be familiar with this. I wonder if Dr. Sutton is willing to say a little bit about how the technique works and so on.

Sutton: The technique for studying human chromosomes involves taking about 10cc of blood and culturing it through several divisions. Colchicine is added which halts cell division at the metaphase stage. There are then various techniques for spreading the white cells on a slide and examining metaphase figures. It is a fairly simple matter to count 46 chromosomes if all of them are there and perfectly spread. It is also a fairly simple matter to assign individual chromosomes to one of 6 or 7 groups, and within these groups several of the chromosomes can be identified quite clearly because of their total length and the position of the centromere. But within other groups identification is far from clear. If you take a whole series of objects of slightly different lengths, it is possible to array them so that the two longest match up, and the next two, and so forth giving a very convincing array going from long to short, which is the primary basis for arranging chromosomes. But, because there are slight differences in contraction, such arraying is subject to tremendous errors. A little bit of progress has been made in assigning linkage groups to specific chromosomes, and some suggestions are now in the literature, but we are very far from knowing any good associations.

Lindzey: In the chromosomal aberrations associated with mongolism, where multiple chromosomes are mentioned, would the fact that more than one is mentioned ordinarily be attributed to a measurement error, or is there some suspicion that there are multiple chromosomes involved?

Sutton: Well the similarity among persons with mongolism suggests that it is always the same chromosome. Now, if you get a variation in the chromosomal pattern you next try to interpret this variation in terms of a translocation which maintains the original extra chromosome 21. So far this has not always been possible, but I am not sure that the restrictions are very severe. I think this is a reasonable procedure, although there is some circular reasoning. The fact that so few viable trisomic conditions have been found, and the fairly uniform manifestations of mongolism rather convincingly suggest that the same supernumerary element is involved in all cases.
Fuller: The other trisomics have been identified with a different chromosome, and, though very rare, they also show a uniformity. This strengthens the argument, although only two other trisomics are known.

Sutton: Well, a whole variety of aberrations are known. There is the so-called isochromosome, where the division is apparently at right angles to the normal division, so that the daughter chromosomes consist of two non-identical chromosomes, one of which has both the long arms, the other which has both the short arms. Each daughter cell is thus a partial trisomic and partial monosomic. This has been observed on several occasions and the fact that a coherent picture can be made of all this suggests that there is some reasonable basis for it.

Gottesman: I am wondering if I might comment on the concept of heritability in twin studies, since people in animal genetics use the same term. They are using it in a different way than we are. Heritability in a twin study refers to within family variance and it underestimates by an unknown amount. If there were random mating we could multiply by two and get a better estimate, but we do not know how much assortative mating there is for each of the various behavioral characteristics. I would like Dr. Fuller to clarify the relationship between “heritability” in twin studies and the “heritability” that the animal geneticists talk about.

Fuller: Well, I think at this stage it is perfectly true that in a family, on an over-all family population basis, with the assumptions of random mating, half of your genetic variance will appear between, and half within the family, and if you make your comparison of the identical twins solely against their co-twins it is one-half the total. However, remember that only with the genetic variance do you get half the estimate. You are really measuring phenotypic variances, and here, of course, we do not really know the ratio of the within-family phenotypic components to the between-family phenotypic components. All we can say is that you always tend to underestimate heritability from the twin study.

Loehlin: It is also true that you underestimate the environmental variation in a twin study. So it is a question of which are you underestimating more, the environmental or the hereditary variance, when you confine yourself to within family analyses.

Fuller: That is something of course that we really do not know. We would like to say that there is more variation between families, but I think it is quite conceivable that some characteristics, for instance, being the older sib in a family, may really have more effect upon a specific area of psychological development, than does growing up in different families. This is why we speak very loosely when we try to say that growing up in different families is an important factor in the psychological development.