Journal of Personality and Social Psychology
1975, Vol. 32, No. 1, 102–112

The Nature of Extraversion: A Genetical Analysis

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A biometrical–genetical analysis of twin data to elucidate the determinants of variation in extraversion and its components, sociability and impulsiveness, revealed that both genetical and environmental factors contributed to variation in extraversion, to the variation and covariation of its component scales, and to the interaction between subjects and scales. A large environmental correlation between the scales suggested that environmental factors may predominate in determining the unitary nature of extraversion. The interaction between subjects and scales depended more on genetical factors, which suggests that the dual nature of extraversion has a strong genetical basis. A model assuming random mating, additive gene action, and specific environmental effects adequately describes the observed variation and covariation of sociability and impulsiveness. Possible evolutionary implications are discussed.

One of the central problems in personality research has been the question of whether such higher order factors as extraversion can be regarded in any meaningful sense as *unitary* or whether there are several independent factors, such as "sociability" and "impulsiveness," which should not be thrown together artificially. Carrigan (1960) concluded her survey of the literature by saying that "the unidimensionality of extraversion/introversion has not been conclusively demonstrated" (p. 355); she further pointed out that several joint analyses of the Guildford and Cattell questionnaires show that at least *two* independent factors are required to account for the intercorrelations between the extraversion–impulsiveness variables. These two factors, she suggested, may correspond to the European conception of extraversion, with its emphasis on impulsiveness and weak superego controls, and the American conception, with its emphasis on sociability and ease in interpersonal relations. Eysenck and Eysenck (1963) have reported quite sizable correlations between sociability and impulsiveness, a conclusion replicated by Sparrow and Ross (1964); this would suggest that there is a close connection between the two conceptions (Eysenck & Eysenck, 1969). Furthermore, Eysenck and Eysenck (1967) have shown that the correlations of extraversion items (whether sociability or impulsiveness) with subjects' reactions on a physiological test devised on theoretical grounds were proportional to their loadings on the extraversion factor. The recognition that extraversion is a unitary factor in behavior is thus vindicated by prediction from a psychological theory as much as by a correlation between primary factors (Eysenck, 1967).

We now develop a model for the genetical and environmental determinants of extraversion and of its primary components, sociability and impulsiveness. Our intention is to analyze the phenotypic variation and covariation of sociability and impulsiveness into their genetical and environmental components in order to determine, as far as our data permit: (a) the simplest model for the genetical and environmental variation of extraversion considered as a unitary trait and (b) the simplest model for the genetical and environmental determination of the interaction between subjects and the component tests of extraversion, sociability and impulsiveness.

In fulfilling these aims, we are led to compare the unitary and dual models of extraversion with regard to their relative contri-

We are indebted to the Colonial Research Fund and the British Medical Research Council for their support of the investigation. We are grateful to J. Kasriel for the collection of data and to the twins for their continued cooperation.

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butions to the representation of both genotypic and environmental determinants of variation among the responses of subjects to a personality inventory.

Earlier research from the standpoint of the psychological theory underlying this work has mainly been concerned with the analysis of extraversion as a unitary trait (Shields, 1962). Claridge, Canter and Hume (1973) reported analyses of extraversion, sociability, and impulsiveness, but these authors themselves admitted that their samples were too small to justify the kind of analysis we attempt here. Our model will be derived from an analysis of twin data and will, therefore, inevitably reflect the limitations of twin studies as sources of genetical information (Jinks & Fulker, 1970). Even twin studies, however, have seldom been used to best advantage. We hope that our particular analysis will have the additional virtue of demonstrating how twin data in general may be manipulated to test simple hypotheses about the causes of variation. We have adopted the methods and notation of biometrical genetics (Mather & Jinks, 1971) because we believe them to be the most precise and general, while embodying a defined procedure for the analysis of continuous variation which may be extended readily to the analysis of human behavior (Jinks and Fulker, 1970).

DATA

The analysis is based on the responses of 837 pairs of adult volunteer twins to an 80-item personality inventory. Of these items, 13 formed a scale of sociability, and 9 items were scored to provide a measure of impulsiveness. The relevant items are given in Table 1. On the basis of a short questionnaire concerning similarity during childhood, the twins were classified as monozygotic or dizygotic.[1] Such a procedure is surprisingly reliable (Cederlöf, Friberg, Jonsonn, & Kaij, 1961). A sample of digzygotic twins of unlike sex has been included in our study because these provide a critical diagnostic test of sex limitation. The composition of the sample by sex and zygosity is given in Table 2.

TABLE 1

PERSONALITY INVENTORY ITEMS INCLUDED IN THE ANALYSIS

Item	Key
18. Do you suddenly feel shy when you want to talk to an attractive stranger?	−S
23. Generally, do you prefer reading to meeting people?	−S
27. Do you like going out a lot?	+S
30. Do you prefer to have few but special friends?	−S
36. Can you usually let yourself go and enjoy yourself a lot at a gay party?	+S
40. Do other people think of you as being very lively?	+S
44. Are you mostly quiet when you are with other people?	−S
48. If there is something you want to know about, would you rather look it up in a book than talk to someone about it?	−S
56. Do you hate being with a crowd who play jokes on one another?	−S
66. Do you like talking to people so much that you never miss a chance of talking to a stranger?	+S
69. Would you be unhappy if you could not see lots of people most of the time?	+S
75. Do you find it hard to really enjoy yourself at a lively party?	−S
77. Can you easily get some life into a rather dull party?	+S
1. Do you often long for excitement?	+I
4. Are you usually carefree?	+I
8. Do you stop and think things over before doing anything?	−I
12. Do you generally say things quickly without stopping to think?	+I
16. Would you do almost anything for a dare?	+I
20. Do you often do things on the spur of the moment?	+I
33. When people shout at you, do you shout back?	+I
59. Do you like doing things in which you have to act quickly?	+I
62. Are you slow and unhurried in the way you move?	−I

Note. S denotes an item scored for sociability, I for impulsiveness; + indicates that "yes" scored 1, and − indicates that "no" scored 1 for scale under consideration.

The mean sociability and impulsiveness scores of the five groups are given in Table 3. An analysis of the variation between and within groups revealed highly significant (but substantively fairly small) differences between groups with respect to the sociability scores. The groups did not differ with respect to their mean impulsiveness scores. We shall regard

[1] The twins were asked: (a) "Do you differ markedly in physical appearance and coloring?" and (b) "In childhood were you frequently mistaken by people who knew you?" If consistent replies were not given, reference was made to previous questionnaires, twins' letters, and additional information in an attempt to assess zygosity. Many of the twins have been blood-typed subsequently, and the original diagnoses have generally been confirmed (Kasriel, J., personal communication, December 1974).

TABLE 2

STRUCTURE OF TWIN SAMPLE

Twin type	No. pairs
Monozygotic female	331
Monozygotic male	120
Dizygotic female	198
Dizygotic male	59
Unlike-sex dizygotic	129

the groups as representative of the same population as far as their means are concerned. The groups are homogeneous with respect to their dispersion, as will become clear from the subsequent genetical analysis. The pooled standard deviations within groups were 3.0015 and 1.7586 for sociability and impulsivenses, respectively.

Since we wished to minimize the possibility of spurious interaction between subjects and tests, we standardized the raw scores of the twins on both sociability and impulsiveness by dividing the scores by the corresponding average within-groups standard errors. For each group of twins separately, the mean squares within pairs and between pairs were calculated for each of the standardized scales. The analogous within-pairs and between-pairs mean products were also calculated. The mean squares and mean products form the basic statistical summary for the analysis to follow (see Table 4).

We studied the inheritance of extraversion by analyzing the mean squares derived from the subjects' total scores of the two standardized tests. The mean squares for the twins on the measure of extraversion (E) may be

TABLE 3

MEAN SOCIABILITY AND IMPULSIVENESS SCORES OF TWIN GROUPS

Twin type	N	M	
		Sociability	Impulsiveness
MZf	662	6.5045	3.7039
MZm	240	5.7875	3.8125
DZf	396	6.6869	3.7525
DZm	118	6.6441	4.0678
DZos	258	6.4884	3.7054

Note. Abbreviations are as follows: MZf = monozygotic female, MZm = monozygotic male, DZf = dizygotic female, DZm = dizygotic male, and DZos = unlike-sex dizygotic.

TABLE 4

MEAN SQUARES AND MEAN PRODUCTS WITHIN AND BETWEEN TWIN PAIRS FOR STANDARDIZED SOCIABILITY AND IMPULSIVENESS SCORES

Item	df	MS (S)	MS (Imp)	MP (S–I)
Between MZf pairs	330	1.5339	1.3777	.6517
Within MZf pairs	331	.5394	.6403	.1762
Between MZm pairs	119	1.5595	1.2904	.3126
Within MZm pairs	120	.4817	.6630	.1497
Between DZf pairs	197	1.0855	1.1804	.3069
Within DZf pairs	198	.8380	.8408	.2918
Between DZm pairs	58	1.3919	.9799	.6309
Within DZm pairs	59	.6693	.8441	.1516
Between DZos pairs	128	.9457	1.2839	.3638
Within DZos pairs	129	.9290	.7697	.3581

Note. Abbreviations are as follows: MZf = monozygotic female, MZm = monozygotic males, DZf = dizygotic female, DZm = dizygotic male, and DZos = unlike-sex dizygotic. MS = mean square, MP = mean product; S = Sociability and I = impulsiveness. No correction for the main effect of sex was necessary for the DZos.

derived directly from the mean squares and mean products (MP) of Table 4, since $MS_E = MS_{(S+I)} = MS_S + MS_I + 2MP_{S,I}$, where S and I refer to sociability and impulsiveness.

Just as we obtained an E score for each subject by summing over tests, so we may obtain a difference (D) score for each subject by taking the difference between his scores on the standardized tests. The MS derived from these differences summarizes the variation arising because subjects do not perform consistently on the two tests. We may obtain the MS for the D scores directly from the raw MS and mean products of Table 4, since

$$MS_D = MS_{(S-I)} = MS_S + MS_I - 2MP_{(S,I)}.$$

The mean squares for E and D are found in Table 5. Clearly, since the mean products are all positive, the MS_E's are larger than the corresponding MS_D's. Since we are only concerned with these particular tests, the mean squares between subjects for E contain none of the interaction variation. Thus the fact that the MS_E's are approximately twice as large as the MS_D's is an indication that E accounts for more of the total variation of the two tests than D.

We analyze the MS_E to provide a genetical model for variation in extraversion, and we analyze the MS_D to determine the extent to

which genetical or environmental factors contribute to the resolution of E into sociability and impulsiveness. Finally, we show that the covariation of sociability and impulsiveness reflects both genetical and environmental factors by an analysis of the raw mean squares and mean products of Table 5.

TABLE 5

MEAN SQUARES FOR EXTRAVERSION AND INTERACTION OF SUBJECTS AND COMPONENT TESTS

Item	*df*	*MS*	
		E = S + I	D = S − I
Between MZf pairs	330	4.2150	1.6082
Within MZf pairs	331	1.5321	.8273
Between MZm pairs	119	3.4751	2.2247
Within MZm pairs	120	1.4441	.8453
Between DZf pairs	197	2.8797	1.6521
Within DZf pairs	198	2.2624	1.0952
Between DZm pairs	58	3.6336	1.1100
Within DZm pairs	59	1.8166	1.2102
Between DZos pairs	128	2.9572	1.5020
Within DZos pairs	129	2.4749	.9825

Note. Abbreviations are as follows: MZf = monozygotic female, MZm = monogyzotic male, DZf = dizygotic female, DZm = dizygotic male, and DZos = unlike-sex dizygotic. E refers to extraversion, and D refers to the interaction of subjects and component tests. S and I refer to sociability and impulsiveness, respectively.

METHODS

Formulation of the Model

Most analyses of classical twin studies have merely demonstrated the existence of a genetical component of variation by showing that monozygotic twins are more alike than are dizygotic twins. Such an intuitive approach is imprecise and does not lead to any exact predictions about the similarity between other degrees of relatives. For this reason the classical approach is not very helpful in guiding the design of future research. In adopting the methods of biometrical genetics we are able to specify, for a given set of assumptions about the kinds of gene action and environmental effects, precise expectations for the components of variance (and consequently for the mean squares) derived from the analysis of variance of any group of relatives. Furthermore, having specified our assumptions and the consequent expectations of mean squares, we are able to provide a statistical test of the agreement between observations and expectations and, consequently, to test the validity of the assumptions we made at the outset.

Clearly, twin data of the kind we have summarized in this article do not allow us to test any but the simplest set of assumptions about the causes of variation. We also recognize that failure of particular assumptions in principle may not lead to failure of the model in practice, either because sample sizes are too small for the test to be sufficiently powerful (see, e.g., Eaves & Jinks, 1972) or because failure of certain assumptions may contribute more to a bias in estimation than to departures of what is observed from what is expected.

We now consider individually the assumptions we make in the analyses which follow. Some of these are an undesirable necessity of the limited data we have available and may not be tested very powerfully by our analysis. Other assumptions are quite likely to be disproved in practice, even with the data available, if they are unjustified. We emphasize that these limitations apply not to the method, which is the most explicit and flexible available, but to the particular data upon which we seek to build our model. Other experimental designs would enable us to test with greater conviction the assumptions which we now make tentatively, (Eaves, 1972; Jinks & Fulker, 1970).

1: *Alleles and loci act additively and independently.* We assume that there is no dominance, epistasis, or linkage for the loci contributing to variation of the traits under consideration. If our other assumptions are justified, nonadditive variation may be detected in principle with our data, but in practice the necessary sample sizes are likely to be too large (Eaves, 1972). Failure of either of the two following assumptions may make the detection of nonadditive variation virtually impossible with the data of our study.

2: *Mating is random.* There is little evidence of assortative mating for extraversion. We might expect to detect the genetical consequences of assortative mating provided there is substantial genetical variation and a fairly high correlation between spouses (Eaves, 1973a). The design of the present study, however, since it involves twins reared together, makes it impossible to distinguish the effects of assortative mating from those of environmental influences shared by members of the same twin pair. Eysenck (1974) reports a nonsignificant correlation for the extraversion scores of husbands and wives. This suggests that assortative mating can safely be discounted as a factor contributing to the genetical variability of extraversion.

3: *All environmental effects are specific to individuals within families.* Most of the twins in our study lived together, especially when they were young. The fact that both individuals in a pair have the same biological mother and grew up in the same family may make both monogyzotic and dizygotic twins more alike than we would expect on the basis of our simple genetical and environmental model. For twins reared together, such effects are formally indistinguishable from those of assortative mating and, if they are substantial, may contribute to failure of our simple model, which assumes that assortative mating and common environmental influences make a negligible contribution to the observed variation. We have some independent test of the contribution of postnatal shared environmental influences for extraversion because we would expect the intrapair differences of twins to increase with the period of separation. Although age and duration of separation are, of course, highly correlated, we can detect no relationship between the intrapair difference for extraversion scores

TABLE 6

A GENETICAL AND ENVIRONMENTAL MODEL FOR A SET OF OBSERVED MEAN SQUARES

MS	Coefficient of parameter	
	D_R	E_1
Between MZf pairs	1	1
Within MZf pairs	·	1
Between MZm pairs	1	1
Within MZm pairs	·	1
Between DZf pairs	$\frac{3}{4}$	1
Within DZf pairs	$\frac{1}{4}$	1
Between DZm pairs	$\frac{3}{4}$	1
Within DZm pairs	$\frac{1}{4}$	1
Between DZos pairs	$\frac{3}{4}$	1
Within DZos pairs	$\frac{1}{4}$	1

Note. Abbreviations are as follows: MZf = monozygotic female, MZm = monozygotic male, DZf = dizygotic female, DZm = dizygotic male, and DZos = unlike-sex dizygotic. D_R refers to an additive genetical component, and E_1 refers to a within-family environmental component.

and the age or duration of adult and juvenile separation of these twins. We hope to make a detailed consideration of this issue the subject of a future publication. We assume, until we have evidence to the contrary, that any environmental variation for the traits in question is the result of influences which are unique to particular individuals rather than shared by members of the same family. If such an assumption were clearly unjustified, then we would find our observations quite obviously did not coincide with our expectations and we would be forced to reject our simple model.

Jinks and Fulker (1970) showed in their biometrical genetical reanalysis of Shields's (1962) extraversion data that common environmental influences must be fairly unimportant. This adds some weight to our assumption that common environmental influences can be ignored, but we should first indicate the likely sensitivity of our experiment for detecting such effects if they still contribute to the variation between pairs.

Power calculations, familiar in the context of biometrical genetics, (e.g., Eaves, 1972; Eaves & Jinks, 1972; Kearsey, 1970) reveal that a sample of approximately 220 pairs of monozygotic and the same number of dizygotic pairs would allow us to be roughly 50% certain of detecting common environmental influences which accounted for 20% of the total variation against the background of an additive genetical component which accounted for about 40% of the total variation. This gives some indication of the power of the present study to detect common environmental influences (and the confounded consequences of assortative mating), provided there is no nonadditive genetical variation. To be 95% certain of detecting a common environmental component of this magnitude, sample sizes would have to be about four times as great.

4. *Sex linkage and sex limitation are absent.* If we were to adopt the usual practice of analyzing correlations rather than mean squares, we would have only poor tests of sex linkage and sex limitation. We would, however, expect the numerical values of comparable mean squares to vary significantly between sexes in the presence of sex linkage or sex limitation except under very restrictive assumptions about the magnitudes and types of gene effects (see Mather & Jinks, 1971). In applying our model to the mean squares rather than to the correlations, we may expect any gross distortion due to either of these causes to result in significant failure of the model.

5. *Genotypic and environmental deviations are uncorrelated.* Under some circumstances we might expect genotype–environmental covariation to contribute to failure of our simple genetical model for monozygotic and dizygotic twins. These circumstances, however, are rather restricting for a study of this type, and we should be cautious about assuming that the adequacy of the model for twin data means that we can ignore this source of variation. As a consequence of the covariation of genotype and environmental effects we could, in principle, find that the environmental components for dizygotic and monozygotic twins are no longer comparable. In practice we are unlikely to detect such differences with these data.

6. *Any variation due to the interaction of genotype and environment is confounded with the environmental variation within families.* It is inevitable that any interaction between genotypic effects and those environmental influences specific to individuals will be confounded with variation due to specific environmental factors in human studies (Jinks & Fulker 1970). We hope that an analysis of such interactions for personality variables will be the subject of a future article. If we are justified in our assumption that there are no common environmental influences, then we are also justified in our assumption that these do not interact with the genotype. Should our model fail because of common environments, we would find that our estimate of the additive genetical component was biased by the variation due to any interaction of these influences with genotypic factors.

We represent the six assumptions by writing a model for the *mean squares* for pairs of monozygotic and dizygotic twins in terms of an additive genetical component (D_R) and a within-family environmental component (E_1). Mather and Jinks (1971) showed how D_R may be defined in terms of the frequenices and effects of many loci. The coefficient chosen for D_R will depend on the mating system. Since we are assuming mating to be random, the coefficients involve no further unknown parameter and may be written for monozygotic and dizygotic twins as they are shown in Table 6. The expectation for a *within-pair mean square* is simply the expectation for the corresponding *within-family component of variance*, σ_w^2, and the expectation for a *between-pair mean square* is σ_w^2 plus twice the expectation for the corresponding *between-families component of variance*, σ_b^2. Full tables of expectations of mean squares and variance components for different kinds of relatives may be found elsewhere (e.g., Eaves, 1973a; Jinks & Fulker, 1970; Mather & Jinks, 1971).

Estimating Parameters and Testing the Model

If we consider only one trait we have, in this study, 10 observed mean squares. Let these be written as the column vector **x**. Our model (see Table 6) involves two parameters whose coefficients in the expectations of **x** may be represented by the 10 × 2 design matrix **A**. We may obtain our estimates of the two parameters, denoted by the two-element vector $\hat{\boldsymbol{\theta}}$, by solving the simultaneous equations:

$$\hat{\boldsymbol{\theta}} = (\mathbf{A}'\mathbf{W}\mathbf{A})^{-1}\mathbf{A}'\mathbf{W}\mathbf{x},$$

where **W** is the (10 × 10) matrix of information about the observed statistics. When the **x** are mean squares, the amount of information about mean square $\mathbf{x}_i$ is $n_i/2(\varepsilon\mathbf{x}_i)^2$, where n_i is the degrees of freedom corresponding to $\mathbf{x}_i$. Clearly we do not know $\varepsilon\mathbf{x}$ until the model has been fitted so we have to use the observed **x** to provide trial values for the amounts of information and proceed iteratively until our $\varepsilon\mathbf{x}$ are stable. In practice, however, it is often unnecessary to go beyond the first cycle provided the model is adequate, since **x** will then be a close approximation to $\varepsilon\mathbf{x}$. For the case in which we are considering a single trait and our mean-squares are all independent, **W** is diagonal and the computations for simple models are not tedious. Providing that our observed statistics are normally distributed, our estimates of $\boldsymbol{\theta}$ are the maximum-likelihood estimates and the scalar

$$S = (\mathbf{x} - \varepsilon\mathbf{x})'\mathbf{W}(\mathbf{x} - \varepsilon\mathbf{x})$$

is distributed as a chi-square with degrees of freedom equal to the number of statistics less the number of parameters estimated from the data. The assumption of normality is probably not far from the truth with the sample sizes available. Should this chi-square be significant, we would be compelled to reject our model as inappropriate for the description of the variation for the trait under consideration.

Although the preceding statistical considerations are not new, they have not been generally applied to the genetical analysis of human behavior, with the result that data have been used inefficiently, standard errors of estimates rarely quoted, and assumptions rarely tested. The usual analyses of twin data either concentrate on the variation within pairs or on a comparison of monozygotic and dizygotic correlations. The method we employ combines both approaches in a single test of a simple model. In effect, our test of the D_R, E_1 model is not merely testing whether the within-pair variances differ for the two types of twins but whether the estimates derived from within-pair comparisons can be used to predict the variation between pairs. We expect the prediction to be poor if certain of our assumptions fail. These and other considerations, such as that of sex limitation, are all combined in our weighted least-squares analysis of the full set of raw mean squares.

RESULTS

Genetical Analysis of Extraversion

The estimates of the parameters and the elements of their covariance matrix, $(\mathbf{A}'\mathbf{W}\mathbf{A})^{-1}$, are given for extraversion in Table 7. We see from the nonsignificant residual χ^2 that our model is clearly adequate so that the data give no reason to suppose that our assumptions are unjustified. We divide the square of each estimate by the corresponding variance term to give, for each estimate, a $\chi^2(1)$ which tests the significance of that parameter. Clearly both D_R and E_1 are highly significant components of the variation in extraversion.

TABLE 7

GENETICAL ANALYSIS OF EXTRAVERSION

Parameter	Estimate	χ^2	*df*	*P*
D_R	2.2487	98.50	1	<.001
E_1	1.5280	276.04	1	<.001
Residual		7.05	8	.50
V D_R	.051336	—	—	
V E_1	.008458	—	—	
Cov D_RE_1	−.011814	—	—	

Note. D_R refers to an additive genetical component, and E_1 refers to a within-family environmental component. *V* and *Cov* refer to variance and covariance, respectively.

On the basis of our tests of the model we tentatively adopt the view that most of the genetical variation is additive and most of the environmental variation can be attributed to E_1. We may use our estimates to estimate the proportion of the population variance for extraversion which can be attributed to genetical causes. Since all of the variation is additive, we have no need of the distinction between "broad" and "narrow" heritability in the present context; we just estimate:

$$\hat{h}^2 = \tfrac{1}{2}\hat{D}_R/(\tfrac{1}{2}\hat{D}_R + \hat{E}_1)$$
$$= .424 \text{ for extraversion.}$$

This means that 42% of the variation in extraversion may be attributed to genetical causes. In the present case E_1 includes variation due both to "unreliability" and "real" specific environmental influences. There seems little point in correcting for unreliability if all predictions are to be made on the basis of one administration of a test such as that analyzed here. If our genetical model is in fact appropriate, we may predict the correlations between other degrees of relatives for extraversion as measured by this test. For parents and offspring, for example, we would expect a correlation of $\frac{1}{2}h^2 = .21$. Such data as we have suggest that the observed correlation is

TABLE 8

GENETICAL ANALYSIS OF INTERACTION BETWEEN SUBJECTS AND TESTS OF SOCIABILITY AND IMPULSIVENESS

Parameter	Estimate	χ^2	*df*	*P*
D_R	.8591	62.94	1	<.001
E_I	.8359	288.95	1	<.001
Residual	—	8.76	8	>.30
V D_R	.011727	—	—	—
V E_I	.002418	—	—	—
Cov $D_R E_I$	−.003277	—	—	—

Note. D_R refers to an additive genetical component, and E_I refers to a within-family environmental component. V and Cov refer to variance and covariance, respectively.

somewhat lower but not significantly so. Such a difference, if it turned out to be significant, might be attributed to the interaction of the genotypic difference between individuals with an overall differences between the environments of parents and offspring or to the fact that our estimate of the heritability is somewhat biased by undetected common environmental effects. A common environmental effect which accounted for about 10%–15% of the total variance might explain the disparity and is more likely than not be to undetected in our study.

We obtained estimates of the internal consistency of the scales. For sociability the reliability was about .75 and for impulsiveness, .60. We may correct our heritability estimate for unreliability provided we can assume the Subjects × Items interactions estimate experimental error only. Using the estimates of genetical and environmental variance and covariance obtained below, we found the heritability of extraversion, after correction, to be .57. By correcting for unreliability, we have attempted to partition the environmental variation for extraversion into that part which may reflect stable environmental influences on the development of the trait and that part due to experimental error. If subjects and items interact, the contribution of experimental error to E_I will be overestimated. Such interactions may have a genetical component which we could analyze using the methods adopted in this article. Confounded with our "true" environmental variation will remain variation reflecting day-to-day changes in behavior whose contribution can only be assessed by repeated measurement.

Genetical Analysis of Subject × Tests Interaction

The results of the analysis of the mean squares for the D scores appear in Table 8. Broadly speaking the results for the interaction are very similar to those for E. The main difference is the reduction by half, in this case, of the estimates of D_R and E_I. This reflects the greater discriminating power of E resulting from the positive covariation of sociability and impulsiveness. However, the simple model is again adequate, since the residual $\chi^2(8)$ is not significant. D_R and E_I are, once more, highly significant. This means that the discrimination between sociability and impulsiveness is justified in genetical terms. We have to conclude that not all the genetical factors contributing to variation in sociability and impulsiveness contribute equally and consistently to both. We estimate the heritability of the interaction to be .339. Although this value is somewhat lower than that for E, the difference is not large and we must notice that the *relative* contribution of unreliability variation will be greater for the interaction than for E. Using the reliabilities given above, and the estimates of the genetical and environmental variance and covariance components from a later analysis (see below), we estimate the heritability of the interaction of subjects and tests to be .72. The marked change reflects the relatively large positive environmental correlation between sociability and impulsiveness, particularly when the environmental variances are corrected for unreliability.

Table 9 summarizes the results of both analyses in terms of the proportions of the total variation of sociability and impulsiveness

TABLE 9

THE RELATIVE CONTRIBUTIONS OF GENOTYPIC AND ENVIRONMENTAL FACTORS OF EXTRAVERSION AND SUBJECTS × TESTS INTERACTION TO THE VARIATION BETWEEN SUBJECTS FOR SOCIABILITY AND IMPULSIVENESS

Causal factor	Psychological factor		Total
	Extraversion	Interaction	
Genetical	.2870(.3402)	.1096(.2631)	.3966(.6035)
Environmental	.3900(.2156)	.2134(.1807)	.6034(.3963)
Total	.6770(.5558)	.3230(.4440)	1.0000(.9998)

Note. Proportions of estimated reliable variation are given in parenthesis.

TABLE 10

RESULT OF FITTING SIMPLE MODEL TO VARIATION AND COVARIATION OF SOCIABILITY AND IMPULSIVENESS

Parameter	Estimate	Covariance ($\times 10^4$) of estimate with estimate of						$\chi^2(1)$
		D_{RS}	D_{RI}	D_{RSI}	E_{IS}	E_{II}	E_{ISI}	
D_{RS}	.9214	67.05	8.54	23.89	−14.80	−1.61	−4.92	126.63*
D_{RI}	.7132		68.38	24.01	− 1.62	−19.34	−1.93	74.33*
D_{RSI}	.3419			38.08	− 4.92	− 5.64	−9.30	30.70*
E_{IS}	.5410				10.42	1.11	3.41	280.77*
E_{II}	.6441					14.32	9.92	290.45*
E_{ISI}	.1758						6.68	46.27*

Note. The parameters D_{RS}, D_{RI}, D_{RSI}, E_{IS}, E_{II}, and E_{ISI} correspond to the components of the mean squares of sociability, impulsiveness, and the mean products of the two traits, respectively.
* $P < .001$.

scores which may be attributed to the genetical and environmental components of extraversion and the interaction of subjects and tests. Approximately three fifths of the total variation is environmental (from the row totals of Table 9) and two thirds of the total variation is attributable to the extraversion factor (from the column totals of Table 9). The proportion of environmental variation is fairly consistent over columns, and the proportion of variation accounted for by extraversion is fairly consistent over rows. In Table 9 we also present a summary for the scales after the environmental components have been corrected for unreliability. So far we have shown that genetical factors probably contribute to individual differences in both E and D scores. A qualitative consideration of the conclusion suggests that E is more discriminating than D genetically and environmentally and leads us to the view that the positive covariation of sociability and impulsiveness has a basis which is both genetical and environmental. We could verify this directly by a statistical comparison of our estimates to test whether the estimates of D_R and E_I are significantly greater than the corresponding estimates for D. We prefer, however, to estimate separately the genetical and environmental components of the variation and covariation of sociability and impulsiveness scores, since this will allow us to estimate the genetical and environmental correlations between the traits.

Analysis of Variation and Covariation of Sociability and Impulsiveness

The weighted least-squares procedure described above may be extended without undue complication to the simultaneous analysis of the variances and covariances of multiple variables (Eaves & Gale, 1974). In this case, however, separate D_R's and E_I's are fitted for the variance and covariance terms. The information matrix, **W**, is no longer diagonal, since the model is fitted to mean squares and mean products which are no longer independent because each subject yields measurements on every trait.

In this instance the model is fitted to the 30 statistics of Table 5. Now six parameters are specified, D_{RS}, D_{RI}, $D_{RS,I}$, E_{IS}, E_{II}, and $E_{IS,I}$. These correspond to the components of the mean squares of sociability, impulsiveness, and the mean products of the two traits, respectively. The method and the definition of the parameters is discussed in more detail by Eaves and Gale (1974).

There are thus 30 statistics. Six parameters are estimated from the data, so the residual chi-square for testing the goodness of fit of the model has 24 degrees of freedom. This chi-square changed by less than .2% between the first and second cycle of the weighted least-squares analysis, when $\chi^2(24) = 29.41$, $p \simeq .20$. Thus the adequacy of the D_R, E_I model for the variation and covariation of sociability and impulsiveness was confirmed.

The estimates of the six parameters and their covariance matrix are given in Table 10. Clearly all the estimates differ significantly from zero. We estimate the heritability of sociability to be .460 and that of impulsiveness to be .356. Using, once more, our estimates of reliability, we infer that about 54% of the environmental variation for sociability is "reliable" variation, assuming that we have

accounted for all the unreliability. For impulsiveness the comparable figure is 38%. We may now obtain estimates of the proportion of *reliable* variance which is due to genetical causes. Our estimates are .61 for sociability and .60 for impulsiveness. There is, therefore, convincing evidence that both traits are under some degree of genetical control. This finding is not new. Claridge, Canter, and Hume (1973) reported an apparent genetical component of variation for both scales. Our model-fitting approach, however, leads us to suggest that there is no evidence of nonadditive genetical variation and no evidence of common environmental effects. Furthermore, we have demonstrated that the covariance of sociability and impulsiveness probably has a genetical basis but that environmental factors also contribute significantly to the covariation of the two scales. The extent to which the two traits may be regarded as sharing common genetical and environmental factors is represented by the genetic and environmental correlations r_{D_R} and r_{E_I}, respectively:

$$r_{D_R} = D_{RS,I}/(D_{RS} \cdot D_{RI})^{\frac{1}{2}} = .42$$

$$r_{E_I} = E_{IS,I}/(E_{IS} \cdot E_{II})^{\frac{1}{2}} = .32.$$

Variation due to unreliability contributes to E_I but not to D_R so we might expect the observed environmental correlation to be less than the genetic correlation. These correlations are a little less, though not considerably less, than the phenotypic correlation of .468 reported for sociability and impulsiveness by Eysenck and Eysenck (1969).

Providing we are justified in assuming the unreliability components of sociability and impulsiveness to be uncorrelated, we may correct our estimate of r_{E_I} for unreliability using the estimates of reliability given above. We now find that r_{E_I} is .66. This indicates that the unitary nature of extraversion is clearly evident in the environmental determinants of the trait, even though the genetical correlation between sociability and impulsiveness is rather less. Eaves (1973b) suggested, on the basis of a multivariate genetical analysis of monozygotic twins, that "the apparently unitary nature of extraversion at the phenotypic level could be due to environmental rather than to genetical influences." The different analysis we have presented here confirms this conclusion.

We should perhaps clarify what this finding means. It does not necessarily support the view that extraversion is an "environmental mold" trait, to use the conception of Cattell, that is, a trait which reflects the structure of environmental influences inherent in the environment itself. We may obtain exactly the same picture because the organism, by virtue of the integration of its nervous system, *imposes* a unitary structure on externally unstructured environmental influences contributing to the development of behavior.

CONCLUSIONS

The analyses presented above suggest the following principal conclusions.

1. Genetical factors contribute both to the variation and covariation of sociability and impulsiveness.
2. Environmental factors also contribute to the covariation of sociability and impulsiveness.
3. The genetical correlation between the two factors is estimated to be .42, the environmental correlation to be .66 after correction for unreliability.
4. Combining sociability and impulsiveness scores by addition to provide a measure of extraversion provides the most powerful single means of discriminating between individuals with respect to the genetical and environmental determinants of their responses to the sociability and impulsiveness items of the questionnaire.
5. The interaction between subjects and tests has a significant genetical component, so there is some justification for regarding sociability and impulsiveness as distinguishable genetically.

Furthermore we conclude:

6. About 40% of the variation in sociability, impulsiveness, and their combinations, as measured by this questionnaire, can be attributed to genetical factors.
7. Our data are consistent with the view that the genetical variation is mainly additive.
8. We find no evidence for a large effect of the family environment on any of the traits studied, but the environmental influences

specific to individuals (including unreliability of measurement) account for about 60% of the variation.

9. Mating is effectively random for the traits in question.

10. The genetical and environmental determinants of variation are homogeneous over sexes, suggesting that the effects of sex linkage and sex limitation are negligible.

DISCUSSION

Our analysis is necessarily tentative because it is based only on monozygotic and dizygotic twins. We would be particularly cautious about discounting genotype–environment correlations as an additional source of variation. It must also be emphasized that genotype–environment interaction may well be confounded with E_1 so that variation which we have ascribed to environmental factors may itself have a genetical component. We hope to clarify this matter in the future.

Between 30% and 40% of the variation in components of extraversion may be due to environmental factors that cannot be attributed to the inconsistency of the test. All of the detectable environmental variation is specific to individuals rather than common to families. This suggests that attempts to relate extraversion to aspects of the individual's "family background" are unlikely to be productive unless the family background has a direct genetical association with extraversion. Even though we might be able to measure social and domestic factors shared by members of the same sibship, we would not expect these to be very highly correlated (say, not more than $r = .2$) with the mean extraversion score of the sibship. Consistently larger correlations between such shared environmental factors and the mean raw E scores of sibship would lead us to suspect our simple model.

Since a considerable proportion of the variation in extraversion and its components is clearly due to environmental influences specific to individuals, we could expect, in principle, to relate the intrapair differences of monozygotic twins to differences in their environmental experiences. That this is feasible in principle, however, does not aid our efforts to specify or detect such likely influences. Attempts to predict the variation in extraversion for a random sample of individuals by measuring concomitant social or other variables, however, may be misleading, because any association we find could reflect either genetical or environmental communality of the traits in question. Merely attaching the label "social" to a trait does not constitute a prior case for environmental causation. Analyses of the kind we have conducted for the covariation of components of extraversion would have to be employed for the other variables if we were to discriminate between environmental and genetical association between extraversion and other variables in the social "environment."

The fact that between 60% and 70% of the "reliable" variation is genetically determined does not, of course, suggest which genes are involved nor what may or may not be done to modify the trait. It does, however, suggest that the segregation and recombination of alleles may be a primary cause of variation in the dimension of personality we have studied here. At the level of population biology it means that extraversion, like most other traits, reflects genetical polymorphism and as such is exposed to the directional, stabilizing or disruptive influence of natural selection. As far as we can judge from studies on other organisms (Kearsey & Kojima, 1967; Mather, 1966; 1967), we find that directional selection has been characteristically associated with the evolution of a genetical system demonstrating a large amount of directional dominance and duplicate gene interactions. When natural selection has favored intermediate phenotypes, the genetical system involves predominately additive effects, and dominance, if any, is ambidirectional. It would be too early to say whether our failure to detect nonadditive variation merely reflects the design of our study, the (relatively) low heritability, or the small amount of dominance relative to the additive variation. It may be difficult to obtain a definite answer to such questions for this trait because of the large samples required and because of the formal inability to disentangle completely the additive, dominance, and epistatic components of gene action for natural populations even when these can be raised in strictly experimental situations (Mather, 1974). We suggest very tentatively that the polymorphism we detect for extraversion and its components may be

subject to stabilizing selection because, as far as we can tell at the moment, the genetical variation is additive. That is, we should conclude that neither extreme introversion nor extraversion has been favored systematically during human evolution. It is possible that without either extreme the fitness of a human population would have suffered at sometime or other. We can at least conceive of situations in which individuals of more impulsive or sociable temperament may well have promoted the survival of themselves and their close relatives. Similarly, we can imagine that there are times or situations in which it would be advantageous to have the persistent, attentive behavior characteristic of introverts. In contrast to this, we may consider a trait such as high intelligence for which there is a suggestion of directional dominance (Jinks & Eaves, 1974; Jinks & Fulker, 1970) and for which, therefore, we suspect a history of directional selection. In the case of intelligence, it is difficult to conceive of as many plausible situations in which relatively high intelligence could not confer upon an individual greater reproductive fitness than average. Such speculations about the evolutionary significance of personality are less well founded at this stage because we cannot infer the genetical system with any great degree of confidence. We believe, however, that such speculations are legitimate if they engender a more systematic and thoughtful approach to the collection and analysis of data on human behavioral traits.

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(Received March 11, 1974)