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A Genetic Model of Anxiety

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STRESS, STRAIN, AND PERSONALITY

In looking at stress and anxiety, it may be useful to take seriously the physical model of stress and strain that has been so widely adapted to serve our psychological purposes. This physical model finds its expression in Hooke's classical law of elasticity: $\text{Stress} = k \times \text{Strain}$, where k is a constant (the modulus of elasticity) that depends upon the nature of the material and the type of stress used to produce the strain. This constant k , i.e., the ratio stress/strain, is called Young's modulus and is illustrated, with certain simplifications, in Fig. 5-1 (top). A and B are two metals differing in elasticity; they are stressed by increasing loads, and the elongation corresponding to each load is plotted on the abscissa. Identical loads θ give rise to quite divergent elongations, α and β , depending on k . Figure 5-1 (bottom) illustrates a similar analysis of human or rat behaviour in an experimental situation productive of emotion. Again the stress (independent variable) is plotted on the ordinate, and the strain (dependent variable) on the abscissa. A

This chapter was originally Chapter 5 in Volume 2 of this series, and tables and figures are numbered accordingly.

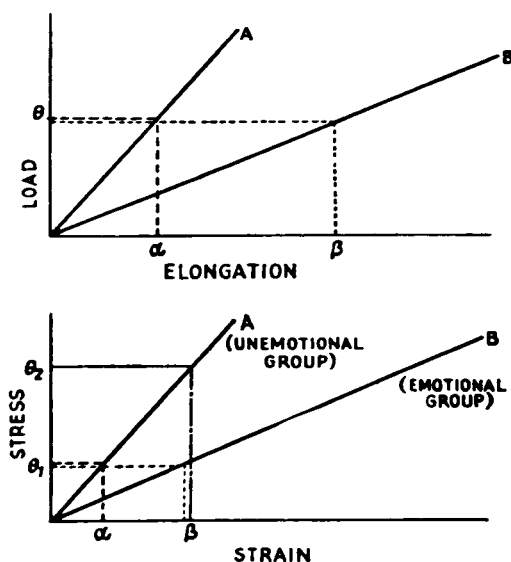


Fig. 5-1. Hooke's Law as applied to physical stress-strain relations (top) and to psychological stress-strain relations (bottom.) A and B refer to different materials, or to different genetic predispositions with respect to anxiety. θ represents two identical loads; α and β represent different elongations (top) and stress or strain (bottom).

and B represent stable and neurotic groups of human subjects, or perhaps the specially bred unemotional and emotional Maudsley strains of rats. Identical stress θ_1 gives rise to quite different strains α and β . It would require stress θ_2 to make the strain in A animals equal to that produced by θ_1 in B animals. Differences between θ_1 and θ_2 are the kind of differences traditionally studied by experimental psychologists; differences between A and B are the kinds of differences traditionally studied by personality psychologists and behavioural geneticists. Physicists have never attempted to make a choice between these two sets of variables, or to study them in isolation; it seems equally futile for psychologists to do so. Provided the modulus employed is even moderately correct, and more than a mere analogy, the experimental possibilities suggested by this method of approach seem promising (Savage & Eysenck, 1964).

In animal work this constant k (emotionality, fearfulness, anxiety) has been studied genetically in considerable detail, using the defecation score in the open field test as the selection measure. Figure 5-2 shows a selection experiment that has been going on for some three dozen generations in our laboratories; the figure shows the trend over the first 15 generations. The mean defecation scores of the animals in the emotionally reactive strain have risen to 4+, whereas those of the animals in the emotionally nonactive strain have fallen to 0; there is practically no overlap between descendants of parents in the two strains. There is ample evidence that this genetic selection has not simply been along the lines of high and low defecation; the two strains behave quite differently from each other in experimental situations where the evidence entitles us to expect different behaviour on the basis of high and low emotionality, respectively. Thus, reactives show greater conditional emotional responses (CER's), learn less efficiently in escape-avoidance conditioning, show greater response to shock, are more susceptible to frustration, develop conditioned inhibition more quickly, and so on. There are also physiological differences: emotionally reactive animals have more body fat, heavier thyroids, more cholesterol in the blood, heavier adrenals, more 17-Ketosteroid output, more thyrotrophic hormone content, and heavier pituitaries (Eysenck, H. J., & Broadhurst, 1964).

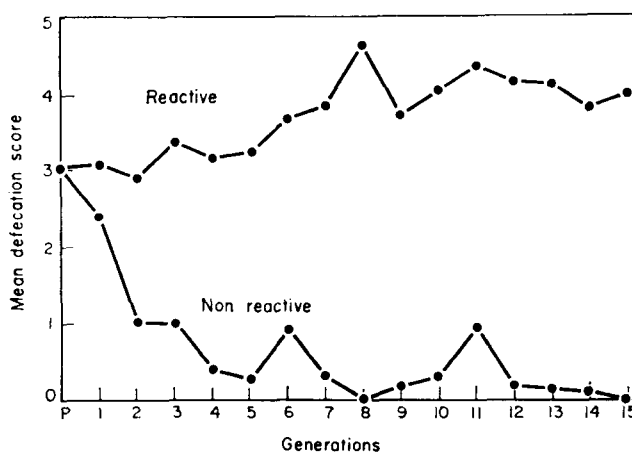


Fig. 5-2. Mean defecation scores (measures of emotional reactivity) in rats bred for high or low defecation over 15 generations.

We might say that we can trade organismic variables against experimental variables; a very elastic metal does not require much stress to produce a given strain, whereas a nonelastic metal might require a considerable stress. Thus we can obtain the same result by trading the modulus of elasticity of the metal against the stress required. In the same way, to produce the same degree of fear/anxiety, we can take an emotionally unreactive rat (or human) and subject the organism to a very frightening experience, or we can take an emotionally reactive rat (or human) and subject the organism to a much less frightening experience, using an appropriate scale of "frighteningness."

To illustrate this argument, consider the work of Rosenbaum (1953, 1956). He found that threat of a strong shock led to greater generalization of a voluntary response than did threat of a weak shock. He also discovered that anxious subjects showed greater generalization to identical stimuli than did nonanxious subjects. In other words, to attain any given degree of generalization, we can either manipulate the stress (strength of shock) or the organismic property of fearfulness or anxiety which is a characteristic of the person. This principle of *trading off* may be useful because we can use the one to measure the other; we could, in the Rosenbaum experiment, express differences in anxiety along a scale the units of which were marked out in terms of strength of shock. Or we could compare both strength of shock, in its psychological meaning, and trait anxiety in terms of degree of generalization. Last, threat (stress) produces certain effects that are identical to those produced by trait anxiety; thus, the measure used for trait anxiety is validated. A similar validation is seen in the case of our rats. Reactive rats, as compared with nonactive rats, behave in a typically different fashion in an experimental situation which is known to be productive of anxiety stress; thus, the measure of emotional reactivity used with the animals is validated.

THE DIMENSIONALITY OF ANXIETY

Anxiety, unfortunately, is not a unidimensional variable; it is implicit in the construction of such devices as the Taylor Manifest Anxiety Scale (MAS) that what is being measured is in some sense univocal, but this is not so. Anxiety is conceived as conditioned

fear reactions (Eysenck, H. J., & Rachman, 1965); and the strength of such conditioned fear reactions depends on two independent variables, not one: (a) degree of emotionality or fearfulness, which determines the strength of the unconditioned stimulus (UCS) and (b) strength of conditioning, which determines the degree to which the conditioned stimulus (CS) and the UCS will be associated. In terms of personality theory, emotionality or fearfulness is measured as the personality dimension of neuroticism (N), and conditionability is closely associated with the personality dimension of extraversion-introversion (E-I) (Eysenck, H. J., 1967). Thus, people whose personality puts them in the dysthymic quadrant (high N, low E) are most predisposed to neurotic disorders and anxiety generally, both because of their strong fear reactions, and their ability to form strong conditioned responses. Scores on the MAS, as one would expect from this analysis, correlate both with N (positively) and with E (negatively); the former correlations are higher than the latter ones (Eysenck, H. J., & Eysenck, S. B. G., 1969). The experimental evidence is strongly in favour of a positive correlation between introversion and strength of conditioning, provided that certain parametric requirements of the theory are fulfilled. Figure 5-3

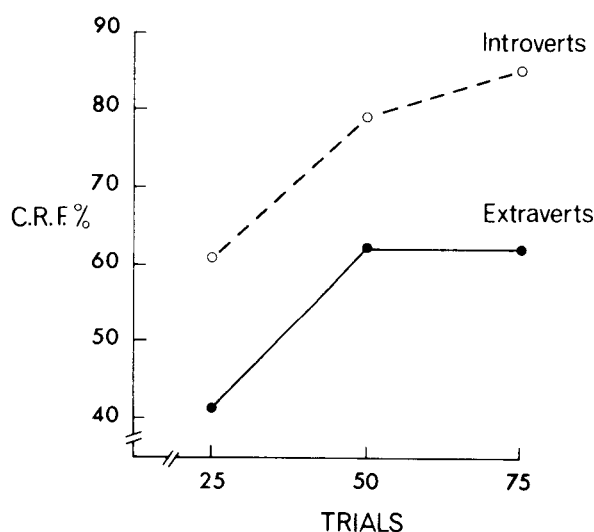


Fig. 5-3. Proportions of conditioned eyeblink responses given by extraverted and introverted subjects during three sessions of 25 trials each. CRF represents conditioned response frequency.

shows some recent results of work with the eyeblink conditioning task; data are shown for the frequency of conditioned responses (CR's) during three sets of 25 conditioning trials. Differences are highly significant for all three sets of data; it is noteworthy that extraverts reach an asymptote during the second set of trials, whereas introverts are still improving during the third set. Figure 5-4 shows, for the same subjects, differences in a work-rate measure; this is defined as the ratio of CR amplitude at unconditioned response (UCR) onset over peak UCR amplitude and is considered to be an estimate of the amount of "physiological work" taken over by the CR from the UCR (Martin & Levey, 1969). Clearly, conditioned responses are not only more frequent in introverts, but also more effective. This "conditionability" is not confined to one test; there is good evidence of a more general factor of "conditionability," which extends from aversive to appetitive conditioning (Barr & McConaghy, 1972).

This general view of anxiety as a conditioned response, determined in part by personality factors N and E, agrees well with the useful distinction between trait and state anxiety, a distinction first drawn by Cicero almost 2,000 years ago. He distinguished

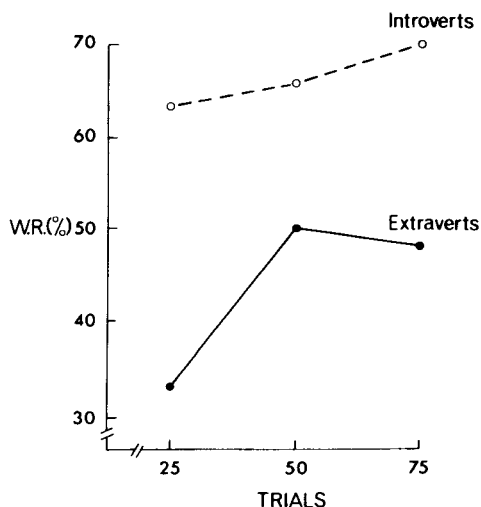


Fig. 5-4. Work rate (WR) (percent) of introverted and extraverted subjects on eyeblink conditioning task during three sessions of 25 trials each.

carefully between *angor* and *anxietas*; *angor* is a transitory attack, provoked by a specified stimulus, *anxietas* is an abiding predisposition. "*Anxium proprie dici qui pronus est ad aegritudinem animi, neque enim omnes anxii, qui anguntur aliquando; nec qui anxii, semper anguntur.*" (He is anxious who is prone to the sorrows of the soul, but they are not necessarily anxious who sometimes feel fear; nor do those who are anxious always feel fear.) *Anxietas*, on this account, would be similar in conception to N, i.e., general fearfulness or emotionality; *angor* would correspond with state anxiety, i.e., the evocation of specific conditioned fear responses.

Factor analytic work with such fear schedules as that of Geer (1965) has shown that, as expected on these grounds, fears for a variety of objects and concepts have both general and specific properties. The work of Rubin, Katkin, Weiss, and Efram (1968) and Landy and Gaupp (1971) has unearthed a number of factors that group such fear-producing stimuli into meaningful groups, such as interpersonal events, discrete objects, death and illness, animate nonhuman organisms, and fears of the unknown. In addition, each item, of course, possesses some unique variance; items differ markedly in this respect. The communality for "deep water" is .80, that for "untimely death" is .78, and that for "heights" is .94; this may be contrasted with "sharp objects," which has a communality of .46, "strange dogs" (.41), and "arguing with parents" (.42). These values, of course, are dependent to some extent on the particular sample of items and subjects chosen; but they are characteristic of the range of generality of fears in our civilisation.

Practically all factor analytic studies in the personality field agree on the overwhelming importance of two major factors resembling N and E, although the names used are often different (Eysenck, H. J., & Eysenck, S. B. G., 1969.) Some writers, in particular Cattell, prefer to work at the primary factor level, using a much larger number of factors; the evidence now seems conclusive that these alleged "primary" factors can no longer be posited. It has been shown that Cattell's 16 Primary Factor (PF) scales have unacceptably low reliability; that they are not replicable in independent researches; that when corrected for attenuation the correlations between them are so high as to approach unity, making them simply rather unreliable measures of E and N

(and possibly one or two additional higher order factors.) Reference may be made here to the work of Becker (1961); Borgatta (1962); H. J. Eysenck (1971, 1972); H. J. Eysenck and S. B. G. Eysenck (1969); Greif (1970); Howarth and Browne (1971); Levonian (1961 a, b); Peterson (1960); Sells, Demaree, and Will (1968); and Timm (1968). These works leave no doubt that work on "primary" factors in personality research is not yet advanced enough to make the use of such factor scores feasible.

It seems reasonable to assume that powerful human reactions, such as fear and conditioning, are mediated by well-demarcated physiological and neurological systems, anatomically identifiable; and there is good evidence to suggest that neuroticism is mediated by an especially labile autonomic system, coordinated in its activity through the visceral brain, while extraversion-introversion is mediated through the cortical arousal system, interacting with the ascending reticular activation system. Those systems are essentially independent most of the time, except when the sympathetic system is strongly aroused; this, like all types of external and internal stimulation, produces strong cortical arousal and washes out any differences that might exist in the resting state between introverts and extraverts (Eysenck, H. J., 1967).

ANXIETY AND HERITABILITY

Having thus, briefly, introduced our psychological and underlying physiological conception of anxiety, we must next turn to the question of heritability. At first sight it might seem unlikely that conditioned fear reactions could be determined to any large extent by hereditary causes, because by definition these reactions are learned, i.e., determined by environmental factors. But this is not the proper way to look at things. It is proposed that people differ systematically from each other in the degree to which they experience fear in the presence of pain and other stimuli which are perceived as threatening life or organic integrity; both the strength of fear experienced, and the length of time over which such fears persist, are determined by the autonomic system, coordinated in its activity by the visceral brain, and are strongly based on polygenic hereditary aspects of the individual's constitution. Similarly, such fears become attached to formerly neutral aspects of the situation in which these fears are experienced (conditioned stimuli), and the speed with which, and the degree to which such conditioning

proceeds are also determined genetically. Thus given random exposure to fear-producing stimuli, some people (high N, low E) are genetically predisposed to develop conditioned fear responses because of strong UCR's and strong CS-CR associations. Even if exposure is not random, these genetic predispositions will still powerfully influence a person's behaviour; it is the task of empirical study to determine the degree to which these various components of the anxiety response are heritable. Current orthodoxy in psychiatry and psychology attributes minimal (or even no) importance to hereditary predispositions, and stresses exclusively the role of environmental variables; we shall see that this stress is one sided and that heredity plays a very powerful part indeed in the causation of neurotic and other anxiety responses.

An example will illustrate the present "orthodox" position of total environmentalism; this is what Redlich and Freedman (1966) (in a widely read textbook of psychiatry) have to say about the influence of heredity on neurotic and sociopathic disorders characterized by anxiety: "The importance of inherited characteristics in neuroses and sociopathies is no longer asserted except by Hans J. Eysenck and D. B. Prell"—referring to a paper in which Eysenck and Prell (1951) demonstrated a high degree of heritability for a factor of neuroticism derived from a battery of psychological tests, administered to monozygotic (MZ) and dizygotic (DZ) twins. Note that there was no discussion of the experiment in question, or criticism of it; neither was there any mention of the large number of authors who have reported empirical results demonstrating the importance of heredity in these types of disorder, or of those experts in the field of behavioural genetics who share this belief and might be quoted as "asserting" it. An equally widely read psychological text by Ullman and Krasner (1969) completely fails to mention genetics or heredity in the Index, and refers to twins only in connection with schizophrenia. Textbooks on personality almost universally refuse to discuss genetic factors; occasionally they mention the completely out-of-date Newman, Freeman, and Holzinger (1937) study as demonstrating the lack of heritability of personality variables.

THE DEFINITIONS OF HERITABILITY

Before looking at the evidence, we must define with some precision the notion of heritability, because psychologists have used this term in many divergent and often meaningless ways; even the

statistical definition given it by Newman et al. in the classic study mentioned previously and widely used by investigators ever since, has no assignable genetic meaning. Briefly, heritability (usually written h^2) is a population statistic which expresses the proportion of population variance in a given phenotypic characteristic attributable to genetic factors. It is usually estimated from a sample of the population and is therefore subject to sampling errors, the magnitude of which is inversely related to the square root of the sample size; representativeness of the sample is of course vitally important in drawing conclusions from the sample to the population. Being a population statistic, h^2 is clearly not a constant in the physical sense, nor does it apply to individuals. It may be defined by reference to the components of variance that enter into it:

$$h^2 = \frac{V_G}{V_P}$$

where V_G is the genetic variance and V_P is the phenotypic or total observed variance. The genetic variance can be divided into four main components:

$$V_G = V_A + V_D + V_{Ep} + V_{AM}$$

where V_A refers to the additive genetic variance; V_D to that portion of the nonadditive genetic variance due to dominance at the same gene loci; V_{Ep} to that portion of the nonadditive genetic variance due to interaction between different gene loci, called *epistasis*; and V_{AM} to genetic variance due to assortative mating, i.e., the increment in total variance attributable to degree of genetic resemblance between mates on the characteristic in question.

The phenotypic variance is made up of the following:

$$V_P = V_G + V_E + V_{GE} + \text{Cov. } GE + V_e$$

where V_G is the genetic variance already defined, V_E is the additive environmental variance that is independent of the genotype, V_{GE} is variance due to interaction of genotypes and

environments, $\text{Cov. } GE$ is covariance of genotypes and environments, and V_e is error variance due to unreliability of measurements.

There are three different ways in which environment and heredity can interact. First, if a particular change in the environment has a uniform effect in raising or lowering the score of every member of the sample on the test used, this effect would be indexed under V_E . Second, if the environmental change interacts with genotypes to produce different phenotypic effects in different genotypes, this source of variance would be indexed under $\text{Cov. } GE$; this covariance increases the total population variance in the trait. Roberts (1967) and other geneticists include $\text{Cov. } GE$ as part of the total genetic variance rather than as part of the environmental variance, and define V_E accordingly as those environmental effects that are independent of the genotype. Finally, V_{GE} denotes interaction of genotype and environment.

It will be clear that we have, not one definition of heritability, but several. Heritability in the narrow sense (h_N^2) is the proportion of additive genetic variance:

$$h_N^2 = \frac{V_A}{V_P}$$

while heritability in the broad sense (h^2) is V_G/V_P . If we follow Roberts and include $\text{Cov. } GE$ in the numerator, we obtain:

$$h^2 = \frac{V_G + \text{Cov. } GE}{V_P}$$

Generally this latter formula is in fact used, either on the assumption that the covariance is due to the genotype, or because the particular method of estimating h^2 used does not permit separation of V_G and $\text{Cov. } GE$. Doubts also arise about the inclusion of V_e in the phenotypic variance; it is not clear why errors in measurement due to unreliability of the measuring instruments should be attributed to phenotypic variance. It seems best to correct empirical results for unreliability before carrying out calculations that would attribute the remaining variance to G and E .

One further distinction is important in understanding the work that has been done on the heritability of personality variables. V_G

and V_E may both be partitioned into two components, one due to differences *between* families and the other due to differences *within* families. The terms used express the fact that, on the genetic side, there are differences between the average value of the trait measurements of different families in the population, and also genetic differences among the offsprings within each family: $V_G = V_{GB} + V_{GW}$. Similarly, there are systematic environmental differences *between* families that do not make for differences among offsprings reared together in the same family, and there are environmental influences *within* families which make for differences among offsprings reared together in the same family: $V_E = V_{EB} + V_{EW}$. The portions of G and E contributed by these different factors are sometimes referred to as G_1 and G_2 , on the genetic side, and E_1 and E_2 , on the environmental side.

MODELS OF GENE ACTION

Empirical work requires us to construct first of all a model to which we can then proceed to fit the data; we also require statistical tests of significance to tell us whether our model is adequate in fitting the data or not. Obviously the various components of V_P cannot be measured directly; they must be estimated indirectly, using estimates made from correlations (or preferably covariances) among persons of differing degrees of kinship (twins, siblings, cousins, etc.) and relationship (reared together or reared apart.) It would not be useful or possible here to go into the detailed formulae which make this possible. The reader is referred to Mather and Jinks (1971) or to Jinks and Fulker (1970); the latter reference gives detailed examples of the application of the formulae to data from studies of intelligence and personality.

Jinks and Fulker (1970) stated:

There are currently three alternative approaches to the genetical analysis of human twin and familial data. There is what might be termed the classical approach through correlations between relatives, culminating in the estimation of various ratios describing the relative importance of genetic and environmental influences on trait variation. This approach leads to ratios such as the H of Holzinger (1929), the E of

Neel and Schull (1954), and the HR of Nicholls (1965), each of which measures an aspect of the relative importance of heredity and environment. There is the more systematic and comprehensive approach of the Multiple Abstract Variance Analysis (MAVA) developed by Cattell (1960, 1965) leading to both the estimation of nature : nurture ratios, and an assessment of the importance of the correlation between genetic and environmental influences within the family as well as within the culture. . . . Finally, there is the biometrical genetical approach initiated by Fisher (1918), and extended and applied by Mather (1949), which includes the first two approaches as special cases, and attempts to go beyond them to an assessment of the kinds of gene action and mating systems operating in the population [p. 311].

Of these approaches, it is in practice largely the classical Holzinger-type of analysis which has been used in the genetic study of personality; unfortunately, this has such serious drawbacks that we can do no more than answer the question of whether the intraclass correlation for MZ twins is significantly larger than that for DZ twins. If it is, and provided that certain objections to the twin method can be satisfactorily discounted, we may conclude that heredity has played some part in causing variation in the trait under investigation; however, we can say nothing about its heritability in any quantitative fashion, nor can we answer any questions about the contribution of nonadditive sources of variance. We are further restricted to a consideration of within-family variance; in interpreting the results in terms of heritability we have to assume that between-family variance is nonexistent. Nor can we answer questions about interaction between *E* and *G* in any manner.

These (and other) defects of the classical method are responsible for the frequently heard remark that we cannot apportion genetic and environmental variance quantitatively, or that it is impossible to assign any meaningful value to the heritability of a given trait or ability. This is true provided we are restricted to the classical method of analysis and are unwilling to make certain simplifying assumptions that would enable us to make at least an estimate of the heritability of the traits or abilities involved. It is not true, however, of the new biometrical genetical methods; these enable us

to construct a proper genetic model, to estimate the various parameters of that model, and to make quantitative judgments regarding the closeness of fit of the data to the model. These are important advances, and they enable us to make certain quantitative estimates in the genetic field which take us a great deal nearer to the proper understanding of the dynamics of heredity.

RESEARCHES USING THE CLASSICAL METHOD: THE BEGINNING

Before turning to studies which have used these modern methods, we must briefly look at the large number of investigations using the classical methods of analysis. A detailed survey has been published by H. J. Eysenck (1975), and there would be little point in undertaking another review here. We shall selectively mention some of the more important studies and summarize the remainder. We shall then turn to concordance studies, which share some of the defects of investigations using the classical twin method, and finally close with a discussion of the studies that have used the methods of biometrical general analysis. The first study is the now classical book by Newman et al. (1937), the results of which are still widely quoted as representative of the literature.

These writers compared 50 pairs of MZ and 50 pairs of DZ twins with respect to a number of physical measurements and mental and educational tests; they concluded:

the physical characteristics are least affected by the environment; that intelligence is affected more; educational achievement still more; and personality or temperament, if our tests can be relied upon, the most. This finding is significant, regardless of the absolute amount of the environmental influence.

The lack of hereditary influence on personality here suggested is still assumed to be a true statement of the fact in the accounts given by most textbooks of psychology and psychiatry; but there are reasons for regarding it with suspicion, not only in the light of more recent work, but also in view of the many criticisms to be made of this early work. These have been summarized by H. J. Eysenck (1967) as follows:

There are two main criticisms. In the first place, the measures used would not now be regarded as either reliable or valid. They included the Woodworth-Mathews Personality Inventory, the Kent-Rosanoff Scale, the Pressey Cross-Out Test, and the Downey Will-Temperament Test. It is doubtful whether any psychologist would nowadays wish to make very strong claims for these measures; even if they could be regarded as reliable and valid, the question would still have to be asked; valid for what? The Woodworth-Mathews Inventory is the only one for which detailed statistics are presented, and we discover that for identical twins the intraclass correlation is .562, for fraternal twins it is .371, and for identical twins brought up in separation it is .583. If we regard, as the original authors certainly did, this questionnaire as an inventory of neurotic tendency, then we would here seem to have some mild indication of the importance of heredity, seeing that identical twins are distinctly superior in point of intraclass correlation to fraternal twins. Moreover, and this is a particularly interesting feature of this table, identical twins brought up in separation are more alike than are identical twins brought up together; this is the only test used, including physical measurements, where this is true. The authors comment that "the Woodworth-Mathews Test appears to show no very definite trend in correlations, possibly because of the nature of the trait and also because of the unreliability of the measure." It is not quite clear to the present writer why there is this denial of a definite trend; it seems fairly clear that identical twins, whether brought up in separation or together, are more alike than are fraternal twins, and we shall see, later modern work has amply justified such a conclusion.

We must now turn to our second criticism, which has curiously enough not to our knowledge been made before. The personality tests used by Newman, Freeman, and Holzinger were essentially tests for adults; the Woodworth-Mathews Inventory for instance was constructed specifically for selection purposes in the army and in hospitals. It is quite inadmissible to use tests of this kind on children, and as is made clear on page 106 of the twin study, the average age of the whole group of identical and fraternal twins is only about

thirteen years. No details are given, but it is clear that there must have been children as young as eight or even younger in this group, and it is doubtful whether a large proportion of the children were in a position to understand the terms used in the tests or to give meaningful replies to them. Our own work in questionnaire construction (S. B. G. Eysenck, 1965) has clearly shown the difficulties attending the construction of personality inventories for children, and the difficulties which children may encounter in answering questions even when these are specifically constructed for them. Taking together these two criticisms of the Newman, Freeman, and Holzinger study, it is perhaps justifiable to say that the data do not support their conclusions. Identical twins whether brought up together or in separation are clearly more alike than are fraternal twins, thus suggesting the importance of heredity in contributing to the temperamental differences studied; the poor reliability and inappropriate nature of the test used suggest that the differences found would have been larger and possibly much larger had more suitable tests been employed. We shall indeed find in our examination of the evidence that later studies using more appropriate methods of examination have resulted in a much better discrimination between identical and fraternal twins. It must of course be remembered that the Newman, Freeman and Holzinger study was a pioneering venture and that at the time few if any personality tests existed, particularly as far as children were concerned. More to blame perhaps are later writers who have cited their work as support for the proposition that heredity is a relatively unimportant factor in the causation of individual differences in temperament and personality, without a closer look at the details of the evidence offered.

The Newman et al. study thus emerges with a heritability of about 30%, which is not negligible when we consider the inappropriateness of the tests; more impressive perhaps is the fact that MZ twins brought up separately are, if anything, more alike than MZ twins brought up together. It is possible that this odd result caused the authors to be hypercautious in their conclusions; we will see that later studies have found similar results, so that we may consider the finding as probably nonartefactual. (It should be added here that our strictures apply only to that portion of the

Newman et al. work concerned with personality; for the rest, their book fully deserves its high standing in the literature. They themselves obviously did not feel happy with the section on personality, as their caveat, already quoted, makes clear.)

RESEARCHES USING THE CLASSICAL
METHOD: LATER WORK

Since the work of Newman et al., quite a number of studies have appeared using MZ and DZ twins as subjects and questionnaires as personality measures; Table 5-1 lists the more important ones, as well as the inventories employed. The outcome of all this work can be stated quite simply; for practically all measures used, MZ twins show higher intraclass correlations than do DZ twins, and the differences are mostly statistically significant, although for some subscales significance may not be reached, particularly when number used are small. Lindzey, Loehlin, Manosevitz, and Thiessen (1971) surveyed some of the more recent of these studies,

Table 5-1
Twin Studies Using Personality Inventories To Discover Genetic
Determination of Individual Differences in Personality

Author and date	Scale used
Carter, 1935	Bernreuter
Cattell, Blewett, & Beloff, 1955	Cattell High School Personality Questionnaire
Vandenberg, 1962	Cattell High School Personality Questionnaire
Gottesman, 1963	Thurstone Temperament Schedule Minnesota Multiphasic Personality Inventory (MMPI) Cattell High School Personality Questionnaire
Wilde, 1964	Maudsley Personality Inventory (Adaptation) MMPI
Gottesman, 1965	Sociability Inventory
Partanen, Bruun, & Markkanen, 1966	California Personality Inventory
Nicholls, 1966	California Personality Inventory
Gottesman, 1966	Myers-Briggs Type Indicator
Vandenberg, 1966	MMPI
Reznikoff & Honeyman, 1967	Comrey Personality and Attitude Factor Scales
Vandenberg, Comrey, & Stafford, 1967	California Personality Inventory
Schoenfeldt, 1968	Middlesex Hospital Questionnaire
Young, Fenton, & Lader, 1971	

and have derived certain "fairly clear conclusions" that also apply to the earlier reports.

Lindzey et al. concluded the following:

1. There is ample evidence that MZ twins are more alike with respect to personality than are DZ like-sexed twins.
2. Correlations are lower, in both groups, for personality traits than they are for IQ test scores; even when corrected for reliability (personality tests are usually less reliable) correlations only come up to .61 and .37, respectively, assuming reliabilities of .75.
3. Heritability is considerably lower than in the case of IQ tests; even when corrected for attenuation, coefficients would be below 50%.
4. The fourth point is perhaps the most interesting. It appears from some calculations made by Thompson and Wilde (1971) that when different studies are compared for the order of heritability coefficients for component scales, no similarity whatever appears; in other words, scale A may have a higher heritability than scale B in one study, but a lower one in another. Lindzey et al. (1971) concluded that "for the present, the most economical interpretation of the data would seem to be that while the genotype may have an appreciable effect on personality, the network of causal pathways between genotype and phenotype is so complex in this realm that the effect of the genotype is spread almost evenly across the broad phenotypic measures that personality and interest questionnaires provide."

An alternative explanation, and one which I would prefer, is that the "spreading" is due to the failure to use univocal trait measures, by failing to employ factor analysis in the purification of the scales in question. Where scales are based on relatively arbitrary collocation of items, nothing else really can be expected.

In addition to studies using personality questionnaires, investigations have also been carried out using psychophysiological measures (Block, 1967; Eysenck, H. J., 1956; Goodman, Luke, Rosen, & Hackel, 1959; Jost & Sontag, 1944; Kryshova, Beliaeva, Dmitrieva, & Zhilinskaia, 1963; Lader & Wing, 1966; Vandenberg, Clark, & Samuels, 1965.) These are so diversified that it is difficult

to give any sort of interpretation without going into tedious detail. The results reported leave no doubt that autonomic functioning under a variety of different types of stimulation is to a large extent under genetic control, but beyond that it would not be wise to go at the present moment. It is unfortunate that investigators have seldom if ever linked their psychophysiological studies with personality investigations; hence our gain in knowledge has not been commensurate with the amount of work done.

STUDIES USING CHILDREN

The studies reviewed so far were carried out on adults and adolescents; do young children give results dissimilar to those reported on older groups? Scarr (1966 a, b, 1969) studied 24 MZ and 28 DZ pairs of girl twins of elementary school age, using ratings and observations in standardized situations; mothers' ratings on the Adjective Check List (ACL) were also used. Median intraclass correlations for the former type of observation were .39 and .23, and for the mothers' ACL ratings they were .40 and .11; again there is overall evidence for greater personality similarity of MZ twins, as compared with DZ twins. The work of Koch (1966) lends some mild support to this conclusion. For even younger twins, Brown, Stafford, and Vandenberg (1967) assessed eight variables, ratings being based on interviews with the mother. MZ twins were more alike on seven of these variables, impressively so on feeding and sleeping problems. Freedman (1965) had films of a small group of twins rated for behaviour in standard situations; smiling and fear of strangers showed the most marked MZ similarities. Lindzey et al. (1971) pointed out:

These data on younger twins, taken together, tend to support the common observation that the greater resemblance of identical twins has early roots, but the data are insufficient to cast much light on the differential influence of heredity and environment on different traits, and thus to assist much in the interpretation of findings at later ages. A really large study of twins followed through the early years of life would be most welcome.

This conclusion is supported by the work of Juel-Nielsen (1965),

who presented detailed case-history-type information of 12 Danish pairs of MZ twins reared apart from early infancy, or early childhood in some cases. Unfortunately, he relied on projective tests, notoriously unreliable and lacking in validity (Zubin, Eron, & Schumer, 1965) and subjective interviews that are difficult to quantify; a proper study of this kind would be extremely interesting and important.

Using factor analysis of parental ratings, Buss, Plomin, and Willerman (1973) found that "four inherited tendencies are suggested for humans: emotionality, activity, sociability, and impulsivity." Activity, sociability, and impulsivity are three of the main components of extraversion; emotionality corresponds closely to neuroticism. The actual *H* values calculated for twins under and over 55 months of age are given in Table 5-2; the numbers in the cells are not very large, and it would not be wise to read too much into the results other than that heredity plays a powerful part in producing individual differences in these four components of personality.

The work of H. J. Eysenck & Prell (1951) differs in two important directions from most of the studies so far reviewed. In the first place, they argued that objective tests of behaviour are superior to personality questionnaires, particularly when used on children, and are in any case less liable to faking. In the second place, they argued that conceptions such as neuroticism are essentially based on the notion of intercorrelated traits and measurements, and that twin studies carried out on single measures

Table 5-2
Holzinger *H* Values for 4 Components of Personality

Component	Boys		Girls	
	Under 55 months	Over 55 months	Under 55 months	Over 55 months
Emotionality	.55	.76*	.71*	.69*
Activity	.83*	.73*	.24	.70*
Sociability	.72*	.42	.36	.22
Impulsivity	.87	.86*	—	.66*

* $p < .01$.

Source: Buss, A. H., Plomin, R., & Willerman, L. The inheritance of temperaments. *Journal of Personality*, 1973, 41, 513-524.

confound the issue by mixing up variance due to the trait under investigation and specific variance relative to the test in question. They suggested, therefore, that a whole battery of tests should be given and factor analysed and that a score based on a combination of tests having the highest saturations with the factor in question should be used. In addition, they suggested that these factor scores should be validated against some form of external control; in their own work they did so by comparing experimental groups of children under treatment at a child guidance clinic with normal children in school, demonstrating significant differences on "neuroticism" between the two groups of children (Eysenck, H. J., & Prell, 1952).

Using this approach, H. J. Eysenck and Prell found that the factor score derived from all the tests gave an intraclass correlation of .851 for identical twins and of .217 for fraternal twins; this factor score showed a greater difference between identical and fraternal twins than any single constituent test, thus suggesting that it was indeed a general factor of neuroticism which was inherited rather than specific variance for any single test. The Holzinger h^2 coefficient showed a hereditary determination of .810 if we are willing to assume that this coefficient can indeed be used to measure hereditary determination in this manner.

In another, similar study, H. J. Eysenck (1956) reported results on a battery of tests of intelligence, extraversion, and autonomic activity. Again, intraclass correlations for identical and fraternal twins were calculated for three factors corresponding to these concepts rather than individual tests. For extraversion, the correlation for identical twins was .50; for fraternal ones, $-.33$; for the autonomic factor, the two correlations were, respectively, .93 and .72. For intelligence, the values were .82 and .38. Holzinger's h^2 statistic was calculated for all three factors, giving very similar results in the neighbourhood of .7 for all three. The appearance of a negative intraclass correlation for the fraternal twins is unusual, and H. J. Eysenck (1956) concluded:

[It seems] likely that this value represents a chance deviation from a true correlation of zero, or of some slight positive value, an assumption strengthened by the fact that a correlation of the observed size is not statistically significant. Under the circumstances, however, we cannot regard the h^2

statistic derived for the factor of extraversion as having very much meaning. . . . Much more reliance fortunately can be placed on the significance of the differences between identical and fraternal twins for this factor which. . . is fully significant.

The results of the Eysenck studies give values indicating a greater influence of hereditary causes than would be true of the other studies quoted so far. Apart from the possibility of chance deviations, it may be suggested that the following causes have possibly been operative: (a) behavioural tests are more likely than questionnaires to reveal deep-seated constitutional features of the personality; (b) factor scores are more reliable and valid than single tests; and (c) the measures selected have been chosen on the basis of a theory of personality that perhaps has more experimental and theoretical backing than the theory that gave rise to the measures used by the earlier workers. It is possible that any or all of these causes may have been operative, and it must be left to future investigation to discover to what extent these hypotheses can be upheld.

CLINICAL AND CONCORDANCE STUDIES

Heritability estimates, using questionnaires or objective laboratory tests, give an unequivocal indication of the importance of genetic factors in determining a person's neuroticism and extraversion; similar results are produced when we turn to concordance studies of criminals and neurotics. These are relevant when we consider that theoretically (and in actual fact) neurotics tend to fall into the N+E⁻ quadrant, while criminals fall into the N+E⁺ quadrant (Eysenck, H. J., 1970; Eysenck, H. J., & Eysenck, S. B. G., 1969). If these relationships are truly causal, then we would expect greater concordance for MZ twins than for DZ twins; this is of course what has been the general finding. Table 5-3 shows concordance figures for criminality, Table 5-4 for neurosis. It will be seen that of almost 800 pairs of twins, concordance is shown for criminality by MZ twins in 55% of all cases, by DZ twins in only 13% of all cases, i.e., in a ratio of over 4 to 1. For neurosis, the ratio is only 2 to 1, out of over 300 pairs of twins.

Other data, e.g., from foster children, support the conclusion that crime and neurosis are in part produced by a genetic

Table 5-3

Concordance Rates for Criminality: Identical and Fraternal Twins

Author and date	Identical		Fraternal	
	Proportion	Percent	Proportion	Percent
Lange, 1929	10/3	77	2/15	12
Legras, 1932	4/0	100	0/5	0
Rosanoff, Handy, & Plesset, 1934	25/12	68	6/54	10
Krantz, 1936	20/11	65	23/20	53
Stumpfl, 1936	11/7	61	7/12	37
Borgström, 1939	3/1	75	2/3	40
Yoshimasu, 1961	17/11	61	2/16	11
Hayashi, 1967	11/4	73	3/2	60
Christiansen, 1968, 1970	27/54	33	23/340	6
Total	128/103	55	68/467	13

Source: Eysenck, H. J. *The inequality of man*. London: Temple Smith, 1973.

predisposition (Eysenck, H. J., 1973.) A review of clinical work on the genetic aspects of anxiety, specifically, is given by Slater and Shields (1969). They concluded that "genetic factors play a part in determining the predisposition to anxiety [p. 70]."

All the studies cited so far depend on three hypotheses that may be of doubtful standing. First, it is assumed that MZ twins are in fact 100% identical with respect to heredity; this is almost certainly untrue (Eysenck, H. J., 1967), and calculations based on

Table 5-4

Concordance Rates for Neurosis: Identical and Fraternal Twins

Author and date	Identical		Fraternal	
	Proportion	Percent	Proportion	Percent
Slater, 1953	2/6	25	8/35	19
Ihda, 1960	10/10	50	2/3	40
Braconi, 1961	18/2	90	13/17	43
Tienari, 1963	12/9	57	[not included]	
Parker, 1964, 1965	7/3	70	4/7	30
Shields & Slater, 1966	25/37	40	13/71	15
Total	76/67	59	40/133	30

Source: Eysenck, H. J. *The inequality of man*. London: Temple Smith, 1973.

this hypothesis seriously *underestimate* the contribution of heredity to an unknown extent. Second, it is assumed that the fact that twins are MZ or DZ does not by itself cause parents and others to treat them more or less alike. The evidence suggests that this is not so, and that MZ twins are treated more alike; however, Scarr (1966c) presented evidence to show that this factor is of no great importance in this connection. She examined the child-rearing practices of mothers who were wrong about the zygosity of their twins, and found that actual zygosity had more effect on child-rearing practices than did the (incorrect) zygosity assumed by the mother. Insofar as this difference is important, it would lead us to *overestimate* the contribution of heredity to an unknown extent. Third, it is assumed that conditions in the womb are alike for MZ and DZ twins; this is not so. MZ twins show much more evidence of serious interference of one twin with the other; the effects of this would show up as environmental effects, although it would be stretching the usual meaning of that word to include intrauterine conditions of this kind. On the whole, these three departures from the model are likely to lead to an underestimation of hereditary contributions; and conclusions are therefore likely to be on the conservative side (Eysenck, H. J., 1975).

APPLICATIONS OF THE GENETIC MODEL

We must now turn to a consideration of the most recent work, using more adequate models and methods of analysis. A suitable start is a study by Shields (1962), in which he used a self-rating questionnaire devised by the writer which is very similar to the MPI, sharing a number of items in common with it; this questionnaire provides scores for extraversion and neuroticism. Shields applied it and two tests of intelligence to a collection of pairs of twins who had been separated from one another in childhood; he also had a control group of twins who had been brought up together. There were 44 MZ, 44 nonseparated MZ control pairs, and 32 pairs of DZ twins, 11 of which had been brought up apart. His findings with the questionnaire are given in Table 5-5. It will be seen that identical twins are much more alike than fraternal twins, regardless of whether they are brought up together or in separation; in each case the twins brought up separately are more

Table 5-5
 Intraclass Correlation Coefficients for Monozygotic (MZ)
 Twins Brought Up Together (C), MZ Twins
 Brought Up Apart (S), and Dizygotic
 (DZ) Twins Brought Up Together

Item	MZ		DZ
	C	S	
Height	+0.94	+0.82	+0.44
Weight	+0.81	+0.37	+0.56
Dominoes	+0.71	+0.76	-0.05
Mill Hill	+0.74	+0.74	+0.38
Combined Intelligence	+0.76	+0.77	+0.51
Extraversion	+0.42	+0.61	-0.17
Neuroticism	+0.38	+0.53	+0.11

Source: Shields, J. *Monozygotic twins*. London: Oxford University Press, 1962.

alike than twins brought up together. These results are therefore in good accord with those originally reported by Newman, Freeman, and Holzinger as well as with those of later writers. They are rather more clear-cut perhaps, due to two possible causes: (a) the subjects of the experiment were adults rather than children, and consequently the questionnaires applied to them much more readily than they would apply to children; and (b) the questionnaires used had been elaborated for many years on the basis of factor analytic studies of personality and were consequently perhaps more reliable and valid than those used earlier.

Table 5-5 shows that the fraternal twins have a negative intraclass correlation for extraversion, very much as in the study of H. J. Eysenck (1956). Again this intercorrelation is not significantly different from zero, but the coincidence is certainly striking, although I cannot present any reasonable hypothesis which would account for such a negative correlation.

Results similar to those of Shields were reported by J. S. Price (personal communication, 1969), who tested 102 pairs of MZ twins, of whom 57 pairs had been living apart, and 45 pairs living together. Using the EPI, he found that for N the former pairs showed an intra-pair correlation of .69, as compared with one of .57 for the latter pairs; for all pairs $r = .65$. For E, correlations

were .45 and .29, averaging .38 over all pairs. Thus, here, too, twins living apart were more alike than twins living together.

So far we are still in the field of classic "heritability"; we now turn to the reanalysis of Shields' data by Jinks and Fulker (1970). They were concerned with the construction of genotype-environment models, including interaction variables, and in doing so they went far beyond the simplistic classical models. As they pointed out, even in the absence of genotype-environment interaction, partitioning the total variation between two components, the genetic G and the environmental E , must lead on to a partitioning of these two components into within-family and between-family (G_1 and G_2 , E_1 and E_2 .) Classical formulae ignore explicitly important sources of variation (Holzinger's H ignores G_2 and E_2 , for example.) Nor do classical formulae enable us to test for interaction, or for dominance, or for assortative mating. Jinks and Fulker developed a model that does enable us to do these things, and then apply this model to the data collected by Shields and briefly described previously. What is the outcome of their analysis?

The model requires first of all that the four groups used (male and female MZ and DZ twins) should be homogeneous with respect to N (the first variable to be discussed); the data pass this test. The second assumption to test is the possible importance of genotype-environment interaction. There is no evidence of GE_1 or GE_2 , nor are correlated environments found to be a complication in these data. "Thus on the basis of these tests we are justified in fitting the simple G and E model to the data."

When the calculations are done (they are given in detail in the original paper), G and E_1 are clearly significantly greater than 0, while E_2 is not. There is some slight indication of assortative mating, but the figures are not significant.

At the same time, the absence of dominant gene action is clearly indicated by the fact that $G_1 > G_2$. The absence of dominant gene action strongly suggests that an intermediate level of neuroticism has been favoured by natural selection, and constitutes the population optimum for this personality trait [Jinks & Fulker, 1970].

Further tests confirm the adequacy of the simple model, and "this

simplified model, which fits the data extremely well, . . . may now be used to calculate heritabilities with some degree of confidence." The broad heritability = narrow heritability = .54, which is fully significant. Cattell's nurture : nature ratios were also calculated and indicated that although environment is more important than genotype in producing differences among siblings, the differences in neuroticism observed between families is entirely genotypic in origin. "Evidently cultural and class differences have no effect on this major personality dimension."

We now turn to the analysis of extraversion scores. Here too "we conclude that the types of family represent reasonably adequate samples from the same population." Testing for interaction, it is found:

there is evidence of a certain amount of GE_1 , but not GE_2 . . . Introvert genotypes are more susceptible to environmental influences than extravert genotypes, the latter being relatively impervious. This finding is, of course, fully consistent with Eysenck's (1967) theory that the introvert is more conditionable than the extravert [Jinks & Fulker, 1970].

Broad heritability was calculated as accounting for 67% of variation, but it should be added that this figure depends on certain assumptions that must be made because the simple model does not fit the data as well as it does in the case of neuroticism. Jinks and Fulker raise the possibility that failure may be due to "competition" (intrauterine or later), particularly between DZ twins; Eysenck's (1956) data, giving a negative intraclass correlation on E for DZ twins, support this view. Jinks and Fulker discuss the reasons for the failure of the simple model in some detail; it would take us too far to follow them in this.

An interesting further analysis of some of these data was undertaken by Fulker and Eaves (Unpublished manuscript). Using only MZ twins pairs, they plotted mean scores for each pair against differences, arguing that mean scores were the best available estimate of the degree of extraversion (or neuroticism) of the pair while the difference would indicate the action of environment (and errors of measurement.) These plots are shown in Figs. 5-5 and 5-6; they are clearly bowed, rather than linear, and indicate very

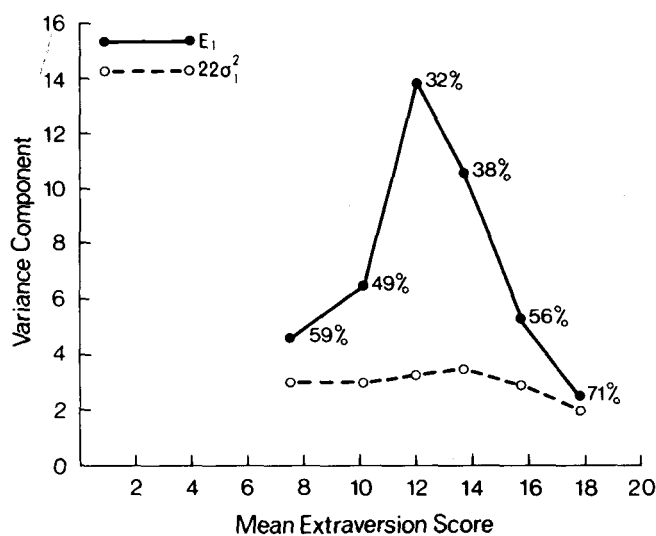


Fig. 5-5. Plot of means and differences between monozygotic twins on extraversion questionnaire. Percentage figures indicate heritability at different points of the extraversion continuum.

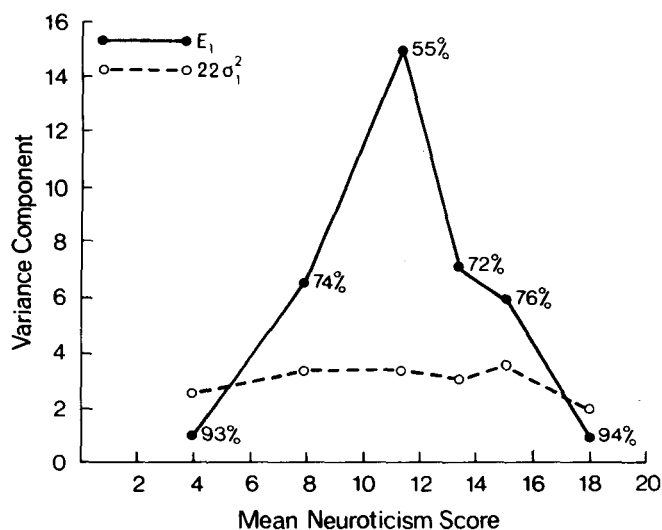


Fig. 5-6. Plot of means and differences between monozygotic twins on neuroticism questionnaire. Percentage figures indicate heritability at different points of the neuroticism continuum.

clearly that environmental factors are more important in the middle range of scores than at the extremes. In part this must be a statistical artefact; very high or low mean scores are incompatible with large within-pair differences. However, this is clearly not the whole story; when the same analysis was done on IQ data, the plotted data were almost entirely linear, with a very weak quadratic component. The data suggest, then, that hereditary influences are stronger for E and N when these personality traits are either very strong or very weak; ambiverts are more determined in their conduct by environmental determinants. This finding may be worthy of being followed up in future work.

FURTHER STUDIES OF THE MODEL

So far, we have assumed that factors like neuroticism and extraversion are unitary; this has been disputed, and Eaves (1973) published a genetic analysis of the PEN scale in which, while N appeared as a unitary factor, E items were found "not to form a single genotypic factor, possible because the observed phenotypic unity of E depends on the correlated environmental modification of more than one underlying genotypic factor [p. 281]." An exhaustive genetical analysis of this problem was undertaken by Eaves and Eysenck (in press); using a much larger number of twins than any previous study, they subjected separately measured traits of "sociability" and "impulsivity" (the two major components of E) to a test of model fitting. They found that between 60% and 70% of the "reliable" variation was genetically determined, and that both genetic and environmental factors contributed to the covariation of sociability and impulsiveness. "Combining Soc and Imp 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 Soc and Imp items of the questionnaire." The data were consistent with the view that the genetical variation was mainly additive, and there was no evidence for a large effect of the family environment on any of the traits studied. Mating was effectively random for the traits in question, and the genetical and environmental determinants of variation were homogeneous over sexes, suggesting that the effects of sex linkage and sex limitation are negligible. More detailed analyses of this kind, using variation and covariation of primary traits in the

genetical analysis of the higher order factors like extraversion and neuroticism, will undoubtedly provide us with much clearer evidence for the precise action of genetic factors in the causation of individual differences in personality.

It will be obvious that certain aspects of the genetic model can be investigated directly, as well as in terms of the biometrical genetical formulae. One example is assortative mating (V_{AM}), which can to some extent be studied by correlating the relevant personality inventory scores for married couples. (This is only partly relevant, because it is possible that married couples change after marriage; ideally one ought to have the personality test scores of men and women before marriage, but this is rather difficult.) A review of the literature reveals that the degree of assortative mating for N and E is slight or nonexistent; this is in good agreement with the results of the biometrical work reviewed (Eysenck, H. J., 1975.) In a recent study, which did not form part of that review, H. J. Eysenck (1974) tested 241 couples with a personality questionnaire; correlations between spouses were .06 for E and .22 for N. The figure for N is significant, but not high enough to affect the issue; this is in accord with the Jinks and Fulker analysis which suggested some slight degree of assortative mating, for N, but not for E. We thus have external support for the accuracy of the biometrical genetical analysis.

Several recent large-scale reviews, in several different languages, have surveyed the field of genetic influences on personality and anxiety (Bracken, 1969; Roubertoux & Carlier, 1973; Shields, 1973); all are agreed on the important role heredity has to play in this field. In particular, the major personality dimensions of N and E, and their constituent factors, emerge again and again in the various studies surveyed as being determined to an important degree (usually in excess of 50%) by heredity. This leaves little doubt that it is not feasible to study anxiety without taking these genetic components into account; research designs that attempt to assess only environmental variables inevitably lead to ambiguous conclusions that leave the door open to alternative, genetic interpretations. There are good reasons for regarding the estimates of heritability for both E and N reviewed in these pages as underestimates; few authors have corrected their figures for V_e , for instance. Failure of the tests employed to have perfect validity also detracts from the "true" heritability of the traits in question. H. J.

Eysenck and Prell, using a whole battery of tests for the measurement of N, obtained higher heritability estimates than other authors; their results indicated that the use of single tests is less likely to optimize findings. But even when we take up the minimum estimates found in the literature, we must conclude that something like one-half of the total variance in the personality variables associated with anxiety is accounted for by heredity. This is an important finding, and it is to be hoped that future work will allow us a more detailed breakdown of this figure and permit us to construct a more detailed model than is currently available. The methods of analysis are now available for carrying out this task, and we know enough about the demands of the experimental design to be able to carry out these improvements (Eaves, 1969, 1970, 1972 a, b); it will not be long before our knowledge in this field will be adequate to the demands we would like to make on it.

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ADDENDUM

In the roughly 10 years that elapsed since writing the preceding chapter, the author has found a great deal of new material which broadly supports the general conclusions arrived at in that chapter. In particular, there has been an enormous increase in the amount of information available on the influence of genetic factors on personality. An excellent review of the most recent work has been given by Fulker (1981), who also discusses in detail the most modern methods used by behaviour geneticists. His general conclusions are broadly in line with those given in my chapter.

Two large-scale studies of genetics and personality which have appeared recently are the books by Buss and Plomin (1975), and by Loehlin and Nichols (1976). Both present very clear-cut evidence for the importance of the genetic contribution to personality differences, and both add considerably to our knowledge by also looking at certain criticisms that have been made of the methods used, and concluding that these criticisms are unjustified.

A third group of researchers which has contributed empirical material and theoretical considerations in this field is represented by the work of Eaves (1978), Eaves and Eysenck (1976a,b; 1977), Eaves et al. (1977, 1978), and Martin and Eysenck (1976). These studies extend the work of Eaves and Eysenck (1976) referred to in the main article.

As an example of unjustified criticism, consider the often repeated assertion that the greater similarity of identical twins, as

compared with fraternal twins, can be explained by the fact that identical twins tend to be treated more alike than fraternal twins. Now it is true that identical pairs are exposed to more similar environments than are fraternal pairs, but the important question of course is that of how much difference does this make in their later personalities? As Loehlin and Nichols point out: "One way of assessing this is to see whether *within* zygosity groups differences in these experiences are associated with personality differences." (p. 51.) As an example they give the possibility that identical twins who are dressed alike may turn out to be more similar in personality than identical twins who are not, identical twins in general being dressed alike more frequently than fraternal twins. "If they do not, then it is perhaps a bit gratuitous to invoke dressing alike as a reason why identical twins are more alike than fraternal twins." (p. 51.)

Loehlin and Nichols go into this question in very great detail, using of course many other areas of similarity apart from dressing alike, and come to the conclusion "that the greater similarity of our identical twins' experience in terms of dress, playing together, and so forth can not plausibly account for more than a very small fraction of their greater observed similarity on the personality and ability variables of our study." In view of the large number of twins involved (there were 850 sets of twins in this study) this conclusion must be regarded as settling this question fairly definitively.

Another criticism which has often been made, particularly of concordance studies among criminals, is the argument that these were usually highly selected, and that the process of selection might have been responsible for the greater concordance among identical as compared with fraternal twins. The work of Christiansen (1970, 1974) and Cloninger et al. (1978) firmly contradicts this hypothesis, as they worked with an unselected sample of Danish twins. The last named study is also interesting because the authors translated concordance values into correlations, and calculated a proper heritability index from their very large sample. They found that the correlation for criminality among MZ twins ($.70 \pm .05$) is significantly higher than that among DZ twins ($.41 \pm .12$). They estimated the heritability to be .59, a value which of course considerably underestimates the true heritability because it is not corrected for unreliability, which in the case of criminal conviction is probably quite high. Any reasonable correction would elevate the heritability estimate to above .70. The authors discuss the various possible

criticisms of their work in detail, and conclude that "the data demonstrate little net effect of these complications." (p. 949.)

One criticism which is frequently made of genetic studies of intelligence, personality, criminality, etc. is that different authors arrive at different values for the heritability of the trait in question. There are two points that require to be borne in mind. In comparing one study with another, one must compare like with like. There are four different estimates of heritability which should be carefully distinguished. We have already mentioned the difference between *narrow* and *broad* heritability in the main text of the article; we should in addition distinguish between heritability estimates *corrected* or *not corrected* for attenuation. Inevitably all the phenotypic data we collect are subject to errors of measurement, and inevitably the formula will assign these errors to the environmental side, i.e., inflating the estimate of environmental influence, and lowering the estimate of heritability. What we really need, of course, is an estimate of the heritability of the "true" phenotypic variance, which can be done by suitable statistical formulae given by Eysenck (1979). Thus we might find that three different authors estimate the heritability of intelligence as 60%, 70%, and 80%, and critics may point to these different values and say that there is clearly little agreement. Yet these three values may refer to the uncorrected narrow heritability (.60), the uncorrected broad heritability (.70), and the corrected broad heritability (.80), all emerging from the same study, and referring to somewhat different entities. There is not necessarily any essential difference between different values as long as these refer to different types of heritability!

In addition we must always bear in mind errors of sampling, which are particularly relevant when we consider the fact that usually the number of pairs of twins involved in a study is not very large, and that these may come from different countries, different cultures, and different social environments. Thus no geneticist would expect a study carried out in Switzerland to give identical results with a study carried out in the U.S.A., as far as the heritability of intelligence is concerned, simply because the range of environmental differences is very much greater in the U.S.A. than in Switzerland, thus making it likely that the influence of genetic factors was stronger in Switzerland than in the U.S.A. When all these considerations are borne in mind, then it is truly remarkable that the pub-

lished figures are as similar as in fact they are, and indeed all of them point to a very strong determination of behaviour by genetic factors.

This conclusion is often misunderstood, because few psychologists are properly trained in behavioural genetics. In particular, it is often suggested that to say that there is a strong genetic factor in criminality is the same as saying that a person is preordained to become a criminal, and this notion of a "fixed" behavioural pattern for a given person is not only unappealing but seems contradicted by experience. But of course the conclusion thus drawn from the genetic data is quite wrong, and does not represent proper genetic thinking. In the first place, of course, heritability estimates are population estimates, and do not apply to a particular person. A good example of the misunderstandings that arise so frequently is the argument often heard that it is as impossible to sort out genetic and environmental influences as it is to say which is more important for determining the size of a field, its length or its width. Now clearly the determination of heritabilities is a question of analysing the *variances*, and a single field does not produce any variance; hence it is entirely the wrong example! If we had a thousand fields, varying in length and width, we could say very quickly which of the two was more important in determining the area of these fields. The fact that this entirely false analogy of the single field has entered into many textbooks illustrates the lack of knowledge which bedevils discussions in this general area.

Another point that must be considered when we talk about heritabilities as population estimates is the fact that new discoveries and new practices may entirely change the influence of heritability. We have already mentioned the likelihood that heritabilities for criminality would be different in Switzerland and in the U.S.A.; we may perhaps illustrate the point by what is purposely a rather artificial example. Consider the female bosom. The three major parameters (size, shape, and consistency) are largely determined by heredity, and if we exclude extreme environmental factors, such as starvation, we might say that hereditary determination is almost complete. Now consider recent advances, such as silicone injections, plastic surgery, etc. These make it possible to change the various parameters governing the female bosom in its phenotypic aspects so profoundly that we can imagine a group of people (say women living in California) where in the next 50 years or so environmental

influences will be all-important, and heredity will play a very small part. Thus to say that because heredity plays an important part in determining the shape of the female bosom at the moment, does not mean that this will invariably be so for all eternity; new discoveries, new developments of a technological kind, and new practices may very well change the situation completely.

The fact is that personality and intelligence are largely determined by heredity at the moment because we have failed so far to find any ways of altering them to any great degree. This does not mean that we would never be able to do so, and indeed once we recognise the importance of genetics in this field we can ask the crucial question: "What precisely is it that is inherited?" Once we know that, we may be able to find ways around it. I have suggested ways of how this could be done in relation to criminality (Eysenck, 1977) and will not do so again here. The situation is constantly changing, and heritability of any particular characteristic of human behaviour may change rapidly with the changing social climate, with the advent of new discoveries and improvements, etc. The notion that everything is fixed forever and a day is not one which represents genetic thinking; it is a caricature imported by people who have little understanding of just what it is that behavioural genetics implies.

All in all, then the outlines of the genetic model of anxiety given in my chapter have been strengthened considerably by recent work on behavioural genetics, both theoretical and practical, and we may now take it for granted that there are strong genetic determinants which give rise to marked individual differences between persons in this respect.

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