Indian Journal of Psychophysiology
1984, vol. 2, 12—20.

Genetic Sources of Variation in Electrodermal Measures : A Twin Study.

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Fifteen electrodermal variables were measured on 20 monozygotic and 20 dizygotic pairs of twins. A Simple habituation paradigm was used, subjects receiving 21 tones, each of 1 sec duration, at an inter-stimulas intervals of 33 secs. The tones were at an intensity of 95 dB (re 20 N/cm^2), sinosoidal and at a frequency of 1000 Hz. A computerised technique was used to generate latency, frequency, amplitude and basal scores for each subject, together with the corresponding change and habituation scores. The biometrical genetical technique was used to analyse the data. Large and significant genetic effects were found for basal conductance, spoteneous response rate and for response amplitude. No significant genetic effect was found for the change and habituation scores although it was felt that this might have been due to lack of reliability. Problems were encountered in the analysis of the latency scores.

It is possible to gain some information about the nature of psychophysiological variables by investigating the extent to which they are inherited. One method of investigating genetic influence is by the measurement of the relevant variables in twins, comparing the between and within family variances for monozygotic (MZ) and dizygotic (DZ) twins.

There are several twin studies in the literature which have looked at electrodermal variables. Rachman (1960) using 7 MZ twin pairs as subjects, presented them with 35 two-sec buzzer tones at an interstimulus interval of 30 secs. He found high intrapair correlations (0·947) for electrodermal response onset latency indicating genetic determination for this variable, but no significant correlation was found for habituation, measured as the number of responses occurring before a criterion level of 3 consecutive zero responses.

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Vandenberg (1961) analysed an experiment in which 34 MZ and 26 DZ twin pairs were tested with several types of stimuli (flashes, bells and hammering) He looked only at mean electrodermal response amplitude for which he calculated an F ratio of DZ within family variance divided by MZ within family variance. This F ratio gives a test of significance for any genetic contribution to the variance. He found no significant effect on this variable for any of the stimulus types.

Block (1967) used 21 MZ twin pairs and presented them with a conditioning paradigm involving a 1 sec shock to the leg as the US, a 1000 cps tone as the CS+ and a 400 cps tone as the CS-. Thirty tones were presented on a partial reinforcement schedule with an inter-stimulus interval of 40 secs. Only responses to the CS's were analysed. The electrodermal measures used were basal resistance at stimulus onset, variance of this basal level, mean resistance response amplitude, and habituation, measured as change in response magnitude over trials. He found high intrapair correlations for the base measure

(0·82) and for the variance of the base measure (0·67). For mean response magnitude no significant intrapair correlation was found, while for habituation the reliability of the measure was only 0·24 so that no significant effect could be expected.

Lader and Wing (1966) measured several electrodermal variables on 11 MZ and 11 DZ twin pairs. They found significant differences between the intrapair correlations for the two types of twin on two measures of habituation and on spontaneous activity. Such a significant difference indicates a genetic contribution independent of between family environmental effects. The habituation measures used were, however, highly correlated with spontaneous activity. Change in basal log conductance between the beginning and the end of the experiment was also measured as was the amplitude of the response to the first stimulus. Neither of these variables showed a significant effect.

For skin potential, Claridge, Canter and Hume (1973) measured 20 MZ and 23 DZ twin pairs. They calculated both the intrapair correlation coefficients and the Vandenberg F ratios on their data. A significant F ratio was found for skin potential habituation slope. The two intrapair correlation for this variable were, however, not significantly different. No significant F ratios were found for the other variables used (basal potential, spontaneous responding rate and amplitude of the response to the first stimulus), although the MZ intrapair correlation was significantly different from zero.

There are limitations on some of the above studies. Rachman and Block both used only MZ twins so that any genetic effects are confounded with the between families environmental variance (E_2). Also none of the statistics used in any of the studies make full use of the data. Neither Vandenberg's F ratio nor the intrapair correlation coefficients take differences between the between family variance for the two types of twin into account. In none of these studies were the effects of E_2 considered. Correlations between the variables are not always given so that we sometimes do not know the effects of the variables on each other. Variables are measured in different ways in the different studies.

In the present study a large number of electrodermal variables were measured. The biometrical genetical technique (Mather & Jinks, 1971) was used to analyse the data. This method obtains maximal information from twin samples.

METHOD

Subjects

The 40 pairs of twins used were all male and were taken from a twin register compiled at the Institute of Psychiatry. Twenty of the twin pairs were MZ while the rest were DZ. Zygosity was tested by a battery of blood tests. The mean age was 24·2 years, ranging between 17 and 44 years. EEG evoked potentials were also measured on the twins. The results for this measure appear in Rust (1975).

Apparatus

Skin resistance was measured with an apparatus built in the department and described elsewhere (Venables & Martin, 1967, model b!). One modification was that the constancy of the current flow was maintained by the use of a transister in place of RI. Electrodes were Ag/AgCl and of a diameter of 9.04 mm. Electrode placement was bipolar to the first and the second fingers of the left hand. The electrolyte was a saline lubricating jelly (Johnson and Johnson Ltd.). The skin

resistance was recorded on a Mingograf EEG polygraph (which has channels for DC recording), and on magnetic tape for subsequent computer analysis. Tonal stimuli were generated by an Audio Oscillator (SG65A Advance) and were presented binaurally through stereophonic headphones.

Procedure

All subjects received 21 stimuli, each of one sec duration, with a constant interstimulus interval of 33 secs. All stimuli were sinosoidal, at a frequency of 1000 Hz and at an intensity of 95 dB (re 20 N/cm^2). The subjects were tested in a soundproofed room seated in a comfortable chair in the dark. While the electrodes were being fixed on the subject the procedure was explained to him. The subject was completely instructed about the stimulus paradigm. He was asked to keep his eyes closed during the experiment, but otherwise to sit back, relax and do nothing.

Analysis

The skin resistance record on magnetic tape was analysed on a Linc-8 computer using a system of programs developed in the department in collaboration with L. Law (Martin, Levy & Siubicka, 1975). Fifteen electrodermal variables were generated for each subject. This is discussed more fully in Rust (1974) and in Martin and Rust (1976). These variables were :—

(1) Basal conductance calculated from the mean absolute resistance at the point of stimulus onset. Units were square root micromhos

(3) Number of responses to stimuli (maximum = 21).

(4, 6 and 8) Response onset, peak and half-recovery latencies. The criterion for a response was that a response onset should occur within 5 secs of stimulus onset. Response latencies were measured in seconds.

(10) Rate of spontaneous responding in responses per minute, estimated from the frequency of non stimulus-tied responses in the interstimulus intervals.

(12 and 14) Response amplitude and magnitude. Response size was calculated from the difference between conductance at onset and conductance at peak of the stimulus-tied response. Where no response occurred the value was assumed to be zero. Response amplitude was the mean response size excluding zero responses, while response magnitude was the mean including zero responses. Units were square root conductance, square roots being taken after calculation of differences but before averaging.

(2, 11 and 15) Change in base, change in spontaneous responding and change in response magnitude. These three variables were the linear regression coefficients over trials of variables 1, 10 and 14 respectively.

(5, 7, 9 and 13) Change in onset, peak and half-recovery latencies, and change in response magnitude. These four change scores were also regression coefficients, but were calculated with allowance made for missing data (see Rust, 1974).

RESULTS

The means and standard deviations for the fifteen electrodermal variables appear in table 1. It will be noticed that the use of a 95 dB stimulus, together with computer scoring, is effective in producing a reasonable number of responses on which to base measurement. The average number of responses to stimuli is 17·9 out of a possible 21. These statistics, together with the intercorrelations between the variables and factor analyses are discussed more fully in Martin and Rust (1976). Similar data from a larger

sample of non-twins was also included in this study. The second order promax factor analysis for the twin data is reproduced here in table 2 as this gives a useful framework in which to interpret the genetic data.

TABLE 1

Means, standard deviations and narrow hereditabilities of electrodermal variables

Variable	Mean	SD	Narrow hereditability	Accepted non-E_1
1) Basal conductance	4.051	1.312	62.28**	D_R + + +
2) Change in (1)	—.0003	.068	64.50***	D_R + + +
3) Number of responses	17.89	3.520	44.71*	E_2 +
4) Response onset latency	1.870	.39	56.58*	E_2 + +
5) Change in (4)	—.015	.028	4.91*	
6) Response peak latency	3.840	.84	45.38*	D_R + +
7) Change in (6)	—.008	.042	21.61	
8) Response half-recovery latency	6.480	1.86	50.76**	D_R + +
9) Change in (8)	—.033	.179	20.39	
10) Spontaneous response frequency	1.153	.70	75.04***	D_R + + +
11) Change in (10)	—.016	.038	22.04	
12) Response amplitude	.813	.409	65.60***	D_R + + +
13) Change in (12)	—.0344	.0261	18.92	
14) Response magnitude	.724	.437	77.88***	D_R + +
15) Change in (14)	—.0346	.0216	22.57	

Units for variables 1, 2, 12, 13, 14 and 15 :- square root micromhos. Units for variables 4 to 9 :- secs. Variables 10 and 11 are in frequency per minute. Variables 12 to 15 were transformed into log conductance before calculation of the narrow hereditability. Significance levels for the narrow hereditability are for the test for difference from zero. The symbols following the letters in the last column indicate the confidence which can be placed in deciding between D_R and E_2.

* $p < .02$; ** $p < .01$; *** $p < .001$.

TABLE 2

Factor Analysis of electrodermal data

Variable	Factor				
	I	II	III	IV	V
1) Basal conductance	*.90*	.10	—.18	.22	.05
2) Change in (1)	·24	*—.73*	—.05	.01	.32
3) Number of responses	.36	—.26	.09	—.28	—.34
4) Response onset latency	—.03	—.13	—.12	*.92*	.04
5) Change in (4)	—.10	.22	*—.81*	—.23	.11
6) Response peak latency	—.02	—.09	.11	*.91*	.01
7) Change in (6)	.10	—.07	*—.87*	.05	—.29
8) Response half-recovery latency	.05	.11	.19	*.88*	—.03
9) Change in (8)	.25	—.23	—.15	.22	*—.77*
10) Spontaneous response frequency	.07	*—.56*	—.07	*—.65*	.17
11) Change in (10)	.31	—.26	.01	.30	*.61*
12) Response amplitude	*.86*	.19	.11	— 22	—.02
13) Change in (12)	—.12	*—.94*	.03	.05	—.16
14) Response magnitude	*.81*	.09	.15	—.27	—.09
15) Change in (14)	— 37	*—.92*	.13	—.03	—.11

An oblique rotation second order promax solution is given. Units for variables 1, 2, 12, 13, 14 and 15 are square root micromhos. The higher factor loadings have been put in italics for clarity.

Genotype-environment interaction (GE) was tested for each variable by checking the correlation between family sums and absolute differences for the MZ twins (Jinks & Fulker, 1970). These were significant for the mean latency scores, for mean amplitude and magnitude and for the number of responses. If we wish the within family environmental variance (E_1) to be unbiased by GE then it is necessary to eliminate the latter by transforming the scale. Where significant correlations were found several transformations were attempted and the one which reduced the correlation to a minimum was adopted, as is standard practice in the genetics of continuous variation. For the amplitude and magnitude mean scores this was achieved by transforming from square root to log conductance. Amplitude and magnitude change scores were also transformed to a log scale to make them compatible with their means. Although the GE correlation had not previously been significant this transformation did make the correlations closer to zero.

For variable 3 (number of responses) the sum-difference correlation was -0.56 ($P < ·01$). This probably results from a ceiling effect as the maximum possible score on this variable is 21, while the mean was 17·89. It

is likely that the correlation represents a correlation between mean and variance within subjects rather than GE (Rust, 1975). The use of a transformation to eliminate this effect would not be advisable as its effect would be to inflate minor differences at the maximum points of the scale at the expense of more interesting differences lower down. The validity of the scale would thus be reduced.

For the latency variables great difficulty was encountered in eliminating sum-difference correlations by transformation. This was particularly so for the peak latency where taking triple logarithms only reduced the correlation from 0·65 to 0·46. This is probably due to a lack of continuity in the distribution of these scores. Where subjects show little or no responding there is an increased probability of picking up a spontaneous response within five sec criterion interval. As the genuine responses will have a rather smaller variance than these spurious responses, the latter will make a somewhat disproportionate contribution to our statistic In view of this the full genetic analysis used the untransformed scores. The effect of this will be considered later.

The mean scores for the MZ and DZ twins on the variables were compared using a test. No significant differences were found, indicating freedom from sample bias on this point.

Analysis of variance was then carried out on the data to find the mean square estimates between families, and within families for both MZ and DZ twin pairs. The between family estimates were divided by two to make them equivalent to the V_f and V_f of Cattell (1960). These four observational parameters were then fitted to various models using the biometrical genetical technique (Mather & Jinks, 1971). This is a generalized technique developed in genetics, of which Cattell's MAVA is a special case. The F ratio of Vandenberg (1961) and the HR statistic of Nichols (1965) can also be deduced from the general model, but all these alternatives give less information than the full analysis.

The number of possible parameters which can be fitted to twin data is large. Many esoteric but possible influences have been suggested by the various parties in the dispute over genetic influences on intelligence. Many of these effects are confounded or highly correlated on twin samples, so that sample sizes of 1000 or more are often needed to separate them. In an uncontroversial area such as the consideration of genetic influences on GSR, where little evidence on the subject is available at all, it is essential to fit very simple models as a starting point.

The most basic genetical model would allow for a single additive genetic component (D_R) and simple environmental effects with no interactions. If we fit such a model we are in effect assuming random breeding, no linkage, no non-allelic interactions, no dominance and no genotype-environment interactions. The effects of deviation from this model can be estimated, but with a small sample it would not anyway be possible to discriminate such effects.

For the environmental variables it is necessary to assume both a between families environmental effect (E_2) and a within families environmental effect (E_1) as these two parameters behave differently within the model. With an experimental design containing only twins reared together Eaves (1972) has shown that under normal circumstances it will take a sample size of about 500 to discriminate D_R from E_2. However, the bio-

metrical genetical approach does give us a chi-square test of goodness of fit of our models, so that we are able to test for D_R and E_2 separately (assuming the absence of the other effect) and find out which model gives the better fit.

TABLE 3

The full model matrix.

D_R is the additive genetic variance, E_2 the between families environmental variance and E_1 the within families environmental variance.

Source	Parameter		
	D_R	E_2	E_1
Between MZ	1/2	1	1/2
Within MZ	0	0	1
Between DZ	3 8	1	1/2
Within DZ	1/4	0	1

The model given in table 3 was fitted using the approximate maximum likelihood technique (Hayman, 1960 ; Nelder, 1960). The model equation is given by

$$g=(M'V^{-1}M)^{-1}M'V^{-1}v$$

where g is the vector of estimated parameters, v is the vector of observed statistics, derived in this case from the mean square estimates, M is the model matrix given in Table 3 and V is a diagonal matrix of weights obtained from the expected variances of our observed statistics. It is possible to obtain such weights as our observed statistics, are in fact variances, and therefore have a chi-square distribution, the variance of which is given by $2V^2/N$. The maximum likelihood solution is approximated by an iterative process in which, following each solution to the equation, the expected value of the observed statistic under the model is recalculated ($E(v)=M'g$). E(v) is then used to give a new weight matrix (V). The iteration converges to a minimum chi-square solution.

The chi-square test of goodness of fit of the model is given by

$$X^2_{k-j}=(v-E(v))'\,V^{-1}(v-E(v))$$

where k is the number of observed statistics, j is the number of estimates and $k-j$ are the degrees of freedom.

The matrix $(M'V^{-1}M)^{-1}$ is a covariance matrix for the estimates. We can obtain the variance of the estimates from the diagonals of this matrix and can therefore test whether our estimates differ significantly from zero. With a maximum likelihood solution the estimates will have a chi-square distribution. This increasingly approximates a normal distribution as the sample size (N) increases, so that if N is large enough a standard normal deviate test can be used. With smaller N's this will give an approximate significance level. The correlations between the estimates can also be calculated from this matrix.

The results of this analysis were converted into estimates of the hereditability $(\frac{1}{2}D_R/\frac{1}{2}D_R+E_1)$ as a percentage which are given in Table 1, together with the significance of their deviation from zero. Also given is the more likely source of non-E_1 variation (D_R or E_2) and an indication of the amount of confidence which can be placed in this decision.

DISCUSSION

The results are probably best discussed in terms of the factor analysis (table 2) of the fifteen electrodermal variables. The variables with high loadings on factor I (basal and response size measures) all have large genetic components accounting for about 70% of the variance and all significant at the 0.001 level. The high genetic component found for basal

conductance is in agreement with Block's (1967) result. Response size is the variable most frequently measured in previous work and here our result disagrees with both Vandenberg (1961) and Block (1967) who found no genetic basis for these variables. In the present study both response amplitude and response magnitude are highly correlated with base (.62 and .54 respectively), but it is interesting that both also have a larger estimated genetic component than does base. This makes it unlikely that the effect is just an artifact of the base result. The size of the base-amplitude correlations for the other studies is not known.

The extent of any genetic effect found is limited by the reliability of the measure as within subject variance is confounded with E_1 (Rust, 1975). There is some evidence for the significant reliability of most electrodermal variables (Bull & Gale, 1973). The unreliability will however come from several sources of variation such as that between days, between sessions, between electrode sites, and from the within subject sampling variance. This latter source will depend on the nature of the stimuli used, the number of samples taken and the duration of the experiment, among other things, and will vary in size from one experiment to another. For most electrodermal variables it is probably the largest source of unreliability and it may be that the failure by Vandenberg and Block to find significant genetic effects for response amplitude was due to this. There is no such contamination in the opposite direction so that the positive results of the present experiment should carry more weight.

For factor II (habituation) there is no evidence of any genetic effect for amplitude or magnitude habituation, while basal change has a 60% genetic component. There is no evidence for a genetic effect either in the other two habituation factors (III and V) so that base change is in fact the only change variable showing a hereditable component. The negative finding for habituation of response size is supported by Block (1967) and Rachman (1960). Block also found however that his measure of habituation had a reliability of only 0.24, so that his failure to find a genetic effect is not surprising. Unreliability is also a possible explanation for the present results on all habituation scores, so that the position with respect to these variables remains uncertain. Significant genetic effects for habituation have been found by Lader and Wing (1966) and by Claridge, Canter and Hume (1973). It seems likely however that Lader and Wing's result was due to the large correlation of habituation with spontaneous activity in their study. Claridge *et al* looked at skin potential so that his result is not directly applicable to the present situation. It can thus be concluded that to date there is no clear evidence for a genetic component in electrodermal habituation.

Unless ways can be found for improving the reliability of the habituation measure it will not be possible to obtain reasonable estimates of any genetic contribution to habituation without a very large increase in sample size. It could be argued that some of the unreliability in the present experiment resulted from having a stimulus intensity at a point which is usually considered to be the threshold between orienting and defensive responses, and that consequently some of the responses were orienting while others were defensive. However, this distinction is of more theoretical than empirical significance, and there is no reason to assume that responses at this stimulus intensity should be any less reliable than those from other intensities. Indeed it seems much more likely that they are in fact more reliable. With quieter tones less

responses would occur so that inevitably there would be more measurement unreliability. With louder stimuli there would be more cases where no habituation occurred and consequently slope scores would be more affected by the occasional large spontaneous response

Factor IV (spontaneous activity and latency) has a probable genetic component of about 50%. This may be E_2 for the latency measures, but spontaneous activity on its own shows a 75% genetic component significant at the 0.001 level. This result supports that of Block (1967).

Interpretation of the latency results is rather difficult. E_3 is indicated as being more important for onset latency, there being more support for a genetic interpretation for peak and half-recovery latency. The only result in the literature on this is that of Rachman (1960) who only used MZ twins so that we have no way of knowing if the high correlation he found for onset latency was due to D_R or E_2. The situation therefore remains uncertain. The effect of the GE found for this variable together with the sampling problems mentioned previously also confuse interpretation. It may well be that with a smaller stimulus 'window' that the 5 sec one used, less problems would have been encountered with these latency variables.

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Received September, 1984