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A genetical analysis of covariation between finger ridge counts

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Summary. 1. The genetical and environmental structure of covariation between finger ridge counts in twin and sibling data has been analysed using the method of Martin and Eaves (1977) adapted from Jöreskog.

2. The model for environmental covariance contains a single factor loading on all ten digits but most environmental variance is specific to each finger.

3. For additive genetic variance there is one common factor loading on all digits. There are also five other independent factors, one for each digit. The thumb factor loads only on the two thumbs but the four finger factors load on the finger in question and on the adjacent fingers.

4. A single common factor for non-additive genetic variance produces a considerable improvement in the model.

5. The pattern of genetic effects differs between left and right hands.

6. Although the same model is appropriate for males and females, different parameter estimates are required.

7. The fit of models is sensitive to the scale of measurement.

1. Introduction

Since the ridge count was introduced as the most convenient measure of pattern size on fingers by Galton (1892) and later modified to its present form by Henry (1900), there has been no consensus of opinion as to how this measurement should be used for comparative or genetical studies. The main controversy concerns use of the total ridge count (TRC) which is the sum of higher counts of all fingers as opposed to the higher counts on individual fingers considered separately.

Holt in her series of studies on inheritance of the TRC (summarized in Holt 1968) showed that the heritability of this character is high and postulated the existence of a single, predominantly genetic, factor determining the general magnitude of finger ridge counts on all ten fingers with the variability between fingers being of an accidental nature.

An opposite view has been expressed by Weninger (1964, 1965, Weninger *et al.* 1976) who claims that ridge counts on individual fingers are genetically independent traits, and consequently, TRC is merely a combination of different traits so that genetic parameters obtained for this combination are invalid. Evidence for an intermediate position assuming a certain degree of genetical independence of ridge counts on individual fingers has been presented by others (Roberts and Coope 1975, Mi and Rashad 1975, Reed *et al.* 1975, Loesch 1979).

The mean ridge counts vary greatly between individual fingers but the correlation between them is high, ranging from about 0·4 to 0·8. In most cases the pattern of correlations is generally consistent in different population samples and in different races (see, for instance: Mavalwala 1962, Brehme *et al.* 1966, Knussmann 1967, Roberts *et al.* 1974, Jantz 1977). However, lower correlations have been reported in the Waskia tribe of New Guinea (Harvey and Singh 1977) and higher correlations in some African samples (Jantz 1977) and these extremes suggest that real racial differences exist. Jantz

(1977) also claims that there is a tendency in some populations for correlations in males to be higher than those in females.

More specific information concerning the relationships between ridge counts on individual fingers has been provided by numerous studies using factor analytic and other techniques (Knussmann 1967, Roberts and Coope 1975, Crawford *et al.* 1976, Jantz and Owsley 1977, Reed *et al.* 1978, Lin *et al.* 1979, Siervogel *et al.* 1979). The results, in particular, show the relative independence of the thumb from other digits and a close relationship between fingers II and III and between IV and V. There is an apparent contrast between the three medial and the two lateral digits. At the same time, however, there is a strong common or 'general' factor determining the magnitude of ridge count on all ten digits.

It has been evident though, that the relationship between statistically identified factors and biological reality is obscure. Consequently, genetical analyses of the identified ridge count factors instead of individual characters have been applied based on variance analysis in MZ and DZ twins (Reed and Young 1979) in the offspring of MZ twins (Reed and Young 1979), or on regression analysis in other relatives (Chopra 1977, Rostron 1977). These merely disclosed a substantial genetic component in some of these factors, comparable to that in individual variables. A maternal effect has also been reported for some factors (Reed and Young 1979) as it has for individual variables (Reed *et al.* 1979). Iagolnitzer (1979) found a stronger genetic determination of the component representing general magnitude of ridge counts than of those representing its variability between individual fingers.

Univariate analyses of the digits with left and right counts added indicate considerable differences in heritability, some being as high as for TRC (Holt 1968, Reed *et al.* 1975, Ghindilis 1977, Loesch 1979). This suggests that there may be genetic factors specific to corresponding pairs of digits. None of the ten individual finger heritabilities is as high as the heritability of TRC (Martin *et al.* 1982a).

However, none of the methods in the papers cited above tests either the implicit genetic model concerning the *sources* of covariation or the structural model hypothesised to explain the *pattern* of covariation. In this respect they are all inadequate since there is no good criterion for saying that one model is better than another.

In this paper we adopt the approach of the genetical analysis of covariance structure which uses a maximum likelihood technique to allow the simultaneous testing of hypotheses about both the sources and structure of covariation.

2. Materials and methods

Finger ridge counts were available for 60 male and 50 female MZ twin pairs, 62 male and 49 female DZ pairs, and 80 pairs of opposite sex siblings. The data are described in detail elsewhere (Martin *et al.* 1982a) together with the results of univariate analyses of the characters under investigation here.

The genetical analysis of covariance structure was adapted from the work of Jöreskog (e.g. 1973) on confirmatory factor analysis. Its development is discussed by Martin and Eaves (1977) and further illustrated by Eaves *et al.* (1977), Fulker (1978) and Martin *et al.* (1979). Briefly, it allows one to test hypotheses about the genetical and environmental *sources* of variation simultaneously with biological hypotheses about the contribution of theses sources to the *structure* of covariation between variables and the residual variation specific to particular variables.

A simple model for the sources of variation in our data is that only individual environmental experiences (E_1) and the additive gene action (D_R) need be invoked to explain variation in finger ridge counts. In the univariate case these sources contribute to the mean squares between (B) and within (W) MZ and DZ pairs with the following coefficients:

$$\mathrm{MS}_{\mathrm{BMZ}} = E_1 + D_{\mathrm{R}},$$
$$\mathrm{MS}_{\mathrm{WMZ}} = E_1,$$
$$\mathrm{MS}_{\mathrm{BDZ}} = E_1 + \tfrac{3}{4}D_{\mathrm{R}},$$
$$\mathrm{MS}_{\mathrm{WDZ}} = E_1 + \tfrac{1}{4}D_{\mathrm{R}},$$

Note that D_R is *twice* the additive genetic variance (V_A) in a randomly mating population (Mather and Jinks 1971). This simple model was found adequate to explain individual differences in each of the ten separate ridge counts (Martin *et al.* 1982 a).

It seems, therefore, that the multivariate extension of this simple $E_1 D_R$ model will be a good initial hypothesis for the sources of covariation, and this may be written:

$$\textstyle\sum_{\mathrm{BMZ}} = \mathrm{HH}' + E^2 + \Delta\Delta' + D^2,$$
$$\textstyle\sum_{\mathrm{WMZ}} = \mathrm{HH}' + E^2,$$
$$\textstyle\sum_{\mathrm{BDZ}} = \mathrm{HH}' + E^2 + \tfrac{3}{4}(\Delta\Delta' + D^2),$$
$$\textstyle\sum_{\mathrm{WDZ}} = \mathrm{HH}' + E^2 + \tfrac{1}{4}(\Delta\Delta' + D^2),$$

where $\sum_i$ is the i-th expected mean products matrix. Hence H and Δ are matrices of E_1 and D_R factor loadings respectively, and E^2 and D^2 are the corresponding diagonal matrices of specific variance components for those two sources.

In more general terms, we may write the expectation for a mean-products matrix:

$$\textstyle\sum_i = \sum_{j=1}^{P} c_{ij}[\mathrm{B}_j(\Lambda_j\Phi_j\Lambda_j')\mathrm{B}'_j + \Theta_j^2],$$

where there are p sources of variation and c_{ij} is the coefficient from the univariate model for the i-th mean square and j-th source. For the j-th source Δ_j is the matrix of the factor loadings and Θ_j^2 the diagonal matrix of specific variance components, as above. Note, however, that we may complicate the model by introducing correlations between the factors in Φ_j, or relate the factor structures of different sources by a simple scalar held in the diagonal matrix B.

Having specified the sources of variation and the factor structures of a model, how can it be tested? The approach is described fully by Martin and Eaves (1977). Generally, the data will consists of k matrices of mean products. We may write S_i for the ith matrix, having N_i degrees of freedom. Given some model for the S_i, we compute the expected values $\sum_i$, being positive definite, for particular values of the parameters of the model. When the observations are multivariate normal, the log likelihood of obtaining the k observed independent S_i is

$$\log L = -\tfrac{1}{2}\sum_{i=1}^{i=k} N_i[\log|\textstyle\sum_i| + \mathrm{tr}(\mathrm{S}_i\textstyle\sum_i^{-1})]$$

(omitting the constant term).

For a given model we require the parameter estimates that maximize log L. Given maximum-likelihood estimates of our parameters, the hypothesis that a less restricting model (i.e., one involving more parameters) does not significantly improve the fit can be tested by computing

$$\chi^2 = 2(L_0 - L_1),$$

where L_1 is the log likelihood obtained under the restricted hypothesis (H_1) and L_0 is the log likelihood obtained under the less demanding hypothesis (H_0). The H_0 we use is that which assumes that as many parameters are required to explain the data as there are independent mean squares and mean products in the first place, i.e., $\sum_i = S_i$ for every i. In this case we have simply

$$L_0 = -\tfrac{1}{2} \sum_{i=1}^{i-k} N_i[\log |S_i| + p].$$

When there are k matrices the χ^2 has $\frac{1}{2}kp(p+1) - m$ d.f., where m denotes the number of parameters estimated under H_1 and p is the number of variables.

The likelihood is maximized by attempting to minimize $-\log L$ for a given model. There are many numerical methods for doing this and a variety have been implemented by the Numerical Algorithms Group (Mark 6 1977). We employed their FORTRAN routine EO4JAF for minimization. Several models were also fitted using the LISREL IV computer program (Jöreskog and Sörbom 1978) as employed by Cantor *et al.* (1982) in their analysis discussed below. In each case identical results were obtained and this gives confidence in the models being uniquely specified.

3. Results

The phenotypic correlation matrices for males and females are shown in table 1. They are in close agreement with those found by Holt (1951, 1959) and others in that (i) the correlations are all appreciable ranging from about 0·8 to a 'base level' correlation of about 0·4; (ii) the highest correlations are between corresponding digits on the two hands; (iii) after that, correlations are highest with adjacent fingers and decline with more remote fingers; (iv) the exception appears to be the thumb which has only a base level of correlation with other fingers.

We now proceed to investigate the genetical and environmental basis underlying these correlations using the genetical analysis of covariance structure described above.

Table 1. Observed phenotypic correlations between finger ridge counts. Males, upper triangle, females lower triangle. Decimal points omitted.

	L1	L2	L3	L4	L5	R1	R2	R3	R4	R5
L1	—	54	49	40	42	79	45	50	34	40
L2	49	—	67	57	51	52	65	62	50	50
L3	62	69	—	67	51	48	64	75	59	48
L4	51	57	73	—	65	42	53	63	76	61
L5	51	63	70	71	—	46	47	51	64	80
R1	74	48	56	54	49	—	48	49	39	46
R2	60	72	74	60	68	54	—	70	53	51
R3	52	60	74	68	64	53	68	—	61	52
R4	45	55	69	82	72	45	59	69	—	66
R5	45	54	63	67	75	46	61	60	66	—

Just as the raw data for univariate model fitting are the between and within pairs mean squares of each twin or sibling group calculated from an analysis of variance, so the raw data for the analysis of covariance structure are the ten 10×10 mean products matrices shown in the Appendix. Note that one degree of freedom is subtracted from the opposite sex sibling within pairs matrix for the vector of differences in sex means by which the matrix has been corrected.

All the explanatory model-fitting has been to the male data, which are slightly more numerous, and only when we have developed a model for males shall we fit it to the female and opposite sex data.

Because nearly all the information about E_1 comes from variance within MZ pairs, we may obtain a good idea of the E_1 covariance structure by first fitting models to the MZW mean products matrix. The simplest model for the action of E_1 is that there is a single factor causing covariation between all fingers and that there is specific environmental variation for each finger. This model has 20 parameters. In the MZ within pairs matrix there are $n(n+1)/2=55$ unique statistics and so there are 35 d.f. to test the fit of the model. The residual $\chi^2_{35}=41{\cdot}4$ ($P \sim 0{\cdot}20$) so this model gives an adequate account of E_1 covariation. The estimates of the E_1 factor loadings ($\hat{\lambda}_{E_1}$'s) and specific standard deviations ($\hat{\theta}_{E_1}$'s) and their significance are shown in table 2. It can be seen that all but one of the factor loadings are smaller than their specific counterparts and that many of $\hat{\lambda}_{E_1}$'s are not significant. Only the thumb and little finger are consistent in showing any pattern of E_1 covariation. Thus any environmental influences on ridge counts are largely specific to the individual fingers. Nevertheless, attempts to eliminate factor loadings, in whole or in part, from the model result in its failure and for this reason we retain the first model of E_1 covariation in subsequent genetical and environmental models fitted to all four male mean products matrices.

In the first attempt to fit a genetical and environmental model to all the male data, we simply replicate the E_1 structural model for the D_R source of covariation; i.e. for both E_1 and D_R sources there is a single factor and specifics. This model has 40 parameters but there are now 4×55 or 220 unique statistics. The result of fitting this model is $\chi^2_{180}=508$, equivalent to a standard normal deviate, $c=12{\cdot}8$. Clearly this model is quite inadequate to explain covariation between finger ridge counts.

Rostron (1977) and others have carried out principal components analyses in an attempt to account for genetical covariation between fingers and this was the next approach tried. Rostron proposed two factors, a general factor loading on all ten

Table 2. Common factor loadings and specific standard deviations for environmental covariance in the MZ male within pairs mean-products matrix. Significance of estimates is indicated.

	L1	L2	L3	L4	L5
Loadings	0·83*	−0·07	0·14	−0·04	1·73***
Specifics	2·15***	2·55***	2·37***	2·07***	1·04**
	R1	**R2**	**R3**	**R4**	**R5**
Loadings	0·93*	0·76	1·24*	0·14	1·23***
Specifics	2·38***	3·35***	3·47***	1·91***	1·75***

$\chi^2_{35}=41{\cdot}4$.
* $0{\cdot}01<P<0{\cdot}05$.
** $0{\cdot}001<P<0{\cdot}01$.
*** $P<0{\cdot}001$.

fingers and an independent factor described as a contrast between the thumb and digits II–V in males and III–V in females. Preliminary principal components analysis of our data suggested that a contrast of thumb with digits III–V was more appropriate for the male data. This model was fitted and the results are shown in table 3. It includes six more D_R factor loadings than the first but the resulting $\chi^2_{174}=368$, while a great improvement on the first model, indicates a quite inadequate account of the covariation. A number of variations on the principal component model, including correlated D_R factors and extra loadings were tried but none gave any significant improvement in the chi square. This illustrates the weakness of the *post hoc* principal components approach which postulates structures of covariance without providing any test of the models proposed.

We proceed from the initial observations that the largest correlations are between corresponding fingers and then between adjacent fingers. Our next model retains the general D_R factor loading on all fingers but adds five independent factors, one for each digit loading on the two corresponding fingers. This adds ten more factor loadings to the model but causes a drop in chi square of 202 to $\chi^2_{170}=306$. This is already a great improvement over the various principal components models tried but is still an inadequate account of genetical covariation.

The next step is based on the observation in our previous paper (Martin *et al.* 1982 a) that, contrary to earlier beliefs, there appears to be non-additive genetical variation for finger ridge counts. This may be either dominance or epistatic interaction between additive genetic effects. With only twin data the expectations of the two are completely confounded (Mather 1974). However, genetical analysis of a full set of twin, sibling and parent-offspring correlations for the trait of finger pattern intensity (which correlates 0·76 with TRC) suggests that the non-additive genetic component for finger pattern intensity is additive × additive epistatic variance (Loesch *et al.* 1982). For the sake of simplicity, therefore, we shall refer to this source of variance as epistasis, while remembering that we should remain agnostic about its true nature.

Addition of a single epistatic (I_R) factor loading on all fingers caused a significant reduction of $\chi^2_{10}=71$ to $\chi^2_{160}=235$. Regardless of the order in which the D_R model was elaborated, addition of a epistatic factor always caused this same significant reduction in residual chi-square. However, attempts to elaborate the factor structure or add

Table 3. Estimates for a model with two independent factors of additive genetic variation for males.

	Environmental		Additive genetic		
	Specific	Factor	Specific	Factors	
				I	II
L1	1·61	4·52	3·18	3·83	0·05
L2	2·77	1·62	3·83	6·46	—
L3	2·22	0·91	2·55	6·87	—
L4	2·25	0·17	2·43	5·69	2·59
L5	2·03	0·91	0·70	3·69	4·28
R1	2·40	4·00	2·03	3·45	0·73
R2	3·61	1·43	3·73	7·58	—
R3	3·10	0·98	0·05	7·19	—
R4	1·94	0·12	3·31	5·60	3·12
R5	1·89	0·98	1·84	3·80	4·30

$\chi^2_{174}=368$.

specific deviations for epistasis did not result in any further significant reduction. Clearly, fitting models to the full mean products matrices provides a more powerful test for the presence of epistatic variance than can be found in the univariate analysis of each finger separately.

We have yet to take account of the higher correlations observed between adjacent fingers. This is done by adding loadings to the five existing D_R individual finger factors. As previously observed, the thumb does not appear to be correlated with any other fingers above base level so this factor remains loading on the two thumbs only. However, the second finger factor is also allowed to load on the third finger, the third finger factor on the second and fourth and so on. This is a modification of the circumplex model described by Jöreskog (1973). It adds twelve more parameters to the model but results in a significant reduction of $\chi^2_{12}=35$ to $\chi^2_{148}=200$. We now find that this rather elaborate model has removed nearly all the specific additive genetic variation and if we remove the ten specific D_R deviations, the fit is only worsened by $\chi^2_{10}=6$ to $\chi^2_{158}=206$. Other attempts to simplify the model by omitting small D_R loadings, however, all resulted in a significant worsening of the model.

Our final model, therefore, contains 62 parameters and while it still fails ($P=0{\cdot}006$), is better than any other models tested. Further attempts to improve the model by relating all the I_R loadings to the D_R common factor loadings by a constant multiplier did not improve the model. To test whether there was any further additive genetic covariation not accounted for, we estimated a positive-definite matrix of additive genetic covariances without any constraint on structure. Removing all the constraints of the factor model for genetic differences, however, only reduced the residual chi-square very slightly, for the addition of many more parameters, to $\chi^2_{135}=194$.

It may be asked whether it is redundant to fit separate genetical loadings for corresponding fingers. If we demand that the D_R and H_R loadings for corresponding fingers are the same this reduces the number of parameters but significantly worsens the residual chi-square by $\chi^2_{21}=40$, a highly significant deterioration. This implies that although genetic factors affect corresponding fingers, their action is not identical so it appears that there is instability in the action of these genes in left and right hands. Estimates for this model are shown in table 4.

It is possible that a major contributor to model failure is that MZ and DZ total variances and covariances are not equal, although there was not much evidence of this

Table 4. Estimates for model with left and right genetic loadings constrained to be equal.

	Environmental		Additive genetic							Epistasis
	Specific	Factor	Specific	Factors						Factor
				I	II	III	IV	V	General	
L1	2·13	0·80	3·10	5·02	—	—	—	—	3·02	5·54
L2	2·60	0·03	3·03	—	0·32	1·51	—	—	4·17	8·72
L3	2·18	0·44	2·38	—	1·47	2·93	0·04	—	4·36	6·33
L4	2·21	−0·15	0·00	—	—	3·24	1·72	2·89	4·12	3·48
L5	1·71	1·13	1·48	—	—	—	2·83	2·68	3·37	3·28
R1	2·41	0·84	0·00	5·02	—	—	—	—	3·02	5·54
R2	3·47	1·19	3·38	—	0·32	1·51	—	—	4·17	8·72
R3	2·91	1·36	0·00	—	1·47	2·93	0·04	—	4·36	6·33
R4	1·98	0·51	2·60	—	—	3·24	1·72	2·89	4·12	3·48
R5	1·55	1·42	1·95	—	—	—	2·83	2·68	3·37	3·28

$\chi^2_{169}=242$

in the univariate analyses. We therefore tested the equality of dispersion in the MZ and DZ matrices using the multivariate equivalent of a variance ratio test (Morrison 1967, p.152). The value of $\chi^2_{55} = 71$ ($0{\cdot}05 < P < 0{\cdot}10$) suggests that this is a contributing factor to model failure but by no means an overwhelming one.

A further possibility is that the analysis of covariance structure is sensitive to departures from multivariate normality. In the previous paper (Martin *et al.* 1982 a) we showed that minimizing non-normality by raising the raw observations to the power 1·5 made little or no difference to the results of univariate model fitting. Although the normalization of individual variables is no guarantee of the multivariate normality of their covariances, it is not unreasonable to suppose that it improves it. We therefore fitted the final 62 parameter model to mean products matrices calculated using the same transformation of the raw observations. This yields a value of $\chi^2_{158} = 195$ ($P = 0{\cdot}025$) considerably less than the value of $\chi^2_{158} = 206$ of the same model fitted to the raw data. It appears that the method is sensitive to departures from normality and if the data were truly multivariate normal it is possible that even more considerable improvements to the fit could be made.

Does transformation change the relative contributions to the variance of different factors or indeed sources of covariation? Table 5 shows the relative contributions to variance of factors and specifics in the final model for both the raw and the transformed data. It can be seen that transformation does alter the pattern of contributions of different factors somewhat within a source but does not much change the total contribution for each source.

The fact that the final model fails is a common experience in covariance structures analysis and maximum likelihood factor analysis and has been discussed elsewhere (e.g.

Table 5. Contributions to variance (per cent) for each character in males according to 62 parameter model.

	Environmental			Additive Genetic Factors							Epistasis
	Specific	Factor	Total	I	II	III	IV	V	General	Total	Factor
(a) Model fitted to raw data											
L1	14	1	15	48	—	—	—	—	10	58	27
L2	17	0	17	—	1	3	—	—	21	25	58
L3	15	1	16	—	3	0	22	—	23	48	36
L4	21	0	21	—	—	7	16	13	29	65	14
L5	9	11	20	—	—	—	1	41	25	67	13
R1	24	2	26	40	—	—	—	—	16	56	18
R2	23	1	24	—	13	7	—	—	24	44	32
R3	25	3	28	—	7	2	7	—	28	44	28
R4	13	0	13	—	—	15	12	11	45	83	4
R5	12	6	18	—	—	—	0	19	58	77	5
(b) Model fitted to data raised to power 1·5											
L1	15	1	16	41	—	—	—	—	13	54	30
L2	19	0	19	—	3	1	—	—	24	28	53
L3	13	0	13	—	5	2	12	—	33	52	35
L4	22	0	22	—	—	2	18	10	38	68	10
L5	6	17	23	—	—	—	2	43	20	65	12
R1	27	4	31	31	—	—	—	—	22	53	16
R2	22	2	24	—	10	9	—	—	28	47	29
R3	24	2	26	—	9	0	3	—	39	51	23
R4	19	0	19	—	—	6	8	8	58	80	1
R5	14	6	20	—	—	—	2	27	48	77	3

Jöreskog 1969, Martin *et al.* 1979). Although the model fails, we can get some indication of its adequacy by using it to predict the phenotypic correlation matrix. This can then be subtracted from the observed correlation matrix (table 1) to give a residual correlation matrix and this is shown in table 6. It can be seen that the model predicts the phenotypic correlations very well and that most of the residuals are trivial, the largest being 0·05.

Having developed a model to explain the male covariance data, is the same model appropriate for the female data and, if so, can we fit a single model to explain covariation in the same-sex twin data and the opposite sex sibling data? The final 62-parameter model fitted to the female data yields $\chi^2_{158} = 192$ ($P = 0{\cdot}035$). This is a better fit than to the male data but chi-square is only smaller in the same proportion as females are less numerous than males. The residual correlation matrix for females is also shown in table 6 and the fit of the model is evidently as good as for the males.

If the same model is appropriate for males and females is this true for the parameter estimates themselves? If we fit the same model jointly to all eight covariance matrices this yields $\chi^2_{378} = 538$, corresponding to a heterogeneity of $\chi^2_{62} = 140$ for the fit of the model in males and females. Evidently, there are significant differences in the size of the parameter estimates in the two sexes.

It is possible that most of this heterogeneity can be explained by a scalar difference in the size of parameter estimates in males and females. We thus restricted the model to the same corresponding parameters in males and females but related them (using Jöreskog's B matrix) by a constant. This constant factor, by which all female parameter estimates are greater than all male ones, was estimated as 1·056 and caused a significant but trivial improvement in the log likelihood equivalent only to $\chi^2_1 = 6{\cdot}15$. Evidently the heterogeneity of fit between males and females is caused by some, perhaps many, small discrepancies rather than a single scalar factor of size of variance components. This could have been predicted from the rather haphazard groups of sex heterogeneities observed in the univariate analysis of the data (Martin *et al.* 1982 a). One model attempting to account for some of these produced a small but not very substantial improvement of $\chi^2_{15} = 26$. It was concluded in the previous paper that these heterogenieties were of no great biological interest so perhaps we are justified in proceeding to fit the 62 parameter model to all ten covariance matrices of the male and female twin data and the opposite sex sibling data. There are now 550 unique statistics and the fit of the model is $\chi^2_{488} = 685$ ($P \sim 10^{-8}$), an increase of $\chi^2_{110} = 147$ over the fit to the male and female data jointly.

Table 6. Residual phenotypic correlations for 62 parameter model. Males top triangle, females lower triangle; coefficients multiplied by 100.

	Residual phenotypic correlations									
	L1	L2	L3	L4	L5	R1	R2	R3	R4	R5
L1	—	0	2	4	4	0	−1	4	3	3
L2	−2	—	1	0	2	0	0	−2	−1	0
L3	0	−1	—	0	−2	0	0	0	−1	−1
L4	−2	0	0	—	−2	3	−1	0	−3	−2
L5	0	1	2	0	—	5	0	−2	−2	0
R1	1	2	−1	1	4	—	1	1	2	2
R2	0	−2	−2	−1	0	1	—	0	−1	−1
R3	−4	−3	0	−1	1	1	−2	—	0	−2
R4	−2	0	0	0	0	1	−1	−1	—	−2
R5	−3	0	1	0	−1	2	2	−1	0	—

4. Discussion

The difficulty of the approach used above is to know when to stop elaborating the model. The method is so powerful that even trivial departures from the model will cause it to fail. We have discussed this problem in an earlier paper (Martin *et al.* 1979). Nevertheless, it is possible to recognize major improvements in the goodness-of-fit although there may be a certain arbitrariness in the details of the final model proposed.

Several features are clear. Our model represents a considerable improvement on the various principal components models proposed by Rostron (1977) and others. It is necessary to take account of the high covariance between corresponding fingers and also between adjacent fingers (excepting the thumb) although we are not claiming that improvements cannot be made to the structure we propose of one general additive genetic factor and an independent genetic factor for each of the five fingers. It is also apparent that a significant improvement is made if non-additive genetic parameters are included in the model. This is further evidence for our contention (Martin *et al.* 1982 a) that there is either epistatic or dominance variation (or both) for finger ridge counts. Better evidence might be obtained on this point if relatives other than twins and siblings were available.

Simultaneously with this analysis, Cantor *et al.* (1982) have explored the sources and structure of covariation between finger ridge counts in the relatives of MZ twins. They arrived at the same structure for E_1 and non-additive genetic covariance, but in their design the coefficients for dominance and epistasis are so similar that it is impossible to distinguish between them. Their structure for additive genetic covariance is somewhat different, however. They postulate eight independent factors comprising a general factor, two hand factors and five finger factors. Their loadings for the general and finger factors were equal for left and right hands so that, in all, 20 additive genetic parameters were estimated against our 32. When this model was fitted to our male twin data a fit of $\chi^2_{170}=234$ was obtained, significantly worse by $\chi^2_{12}=28$ ($P\sim0{\cdot}001$) than our 62-parameter model. Firstly their model takes no account of the high correlation between adjacent fingers. Secondly any differences between loading of fingers between left and right hands are confined to the two hand factors. Cantor *et al.* state that elaborating their model to take account of these effects produces no significant improvements. However, given the assumption about genetic and environmental effects under which both analyses were conducted this is not surprising. A design consisting of MZ and DZ twins provides more precise estimates of genetic covariances than a design consisting of comparable numbers of half and full siblings.

Most environmental variation is specific to each finger but many of the smaller E_1 covariance terms are negative (see Appendix) and together with positive additive genetic correlations between fingers this explains why the heritability of total ridge count is always found to be higher than the heritability of any of the ten individual finger ridge counts (Martin *et al.* 1982 a).

The failure of the attempt to fit the same genetical parameters for both left and right hands indicates that there are asymmetries in the action of genes and we explore the nature of these more extensively elsewhere (Martin *et al.* 1982 b).

The improvement of fit of the model when the raw data are transformed by raising them to the power 1·5 confirms that the method is sensitive to scale, probably because of departures from multivariate normality. However, we have chosen to work with the character on the original scale and must suffer the consequences of a worse fit of the model.

It is clear that there is significant heterogeneity of fit of the model to the male and

female data, but as with the case of significant left–right assymmetry, it is difficult to judge the substantive importance of these discrepancies. If the same discrepancies are found in other samples then one might begin to regard then as biologically, as well as statistically, significant.

We do not regard the model presented here as definitive but as a first attempt to provide a systematic hypothesis-testing approach to the study of the sources and structures of covariation between finger ridge counts. The methods and models applied may prove useful in the explanation of covariation in other biomedical and behavioural variables.

Appendix

Mean products matrices for raw data

MZ males between pairs 59 d.f.

76·61	41·54	31·45	27·36	23·94	58·27	43·98	36·33	23·24	22·81
5·33	65·62	40·63	31·85	22·20	37·90	57·65	44·13	27·46	22·44
−0·42	6·51	55·85	31·41	17·38	28·43	50·01	48·54	27·88	17·78
−0·05	1·83	5·64	38·82	20·77	25·03	42·30	34·48	35·87	21·38
1·19	−0·10	0·37	4·33	28·60	22·61	28·71	19·99	22·51	27·95
1·28	−0·04	−0·07	−0·28	4·08	60·21	39·69	35·48	25·05	24·11
1·49	−0·38	0·23	0·33	1·65	6·55	91·16	60·39	38·89	31·66
−0·08	0·75	0·17	−0·41	1·58	0·67	11·82	61·49	31·36	23·95
1·90	−0·93	−0·39	1·01	2·22	0·14	0·01	13·54	45·85	27·42
0·22	−0·03	1·15	−0·07	0·22	−0·51	0·03	1·89	3·68	35·77
1·26	−0·17	1·51	−0·18	2·13	1·00	0·49	1·25	0·03	4·59

MZ males within pairs 60 d.f.

DZ males between pairs 61 d.f.

42·00	24·18	24·56	19·80	16·50	32·55	18·30	23·68	15·63	16·98
25·39	42·84	31·08	31·60	25·18	24·91	32·95	29·98	26·13	26·50
16·65	35·04	47·17	37·34	25·42	25·48	37·11	37·32	31·44	24·34
12·71	18·93	20·19	46·01	30·53	21·11	32·32	34·35	37·29	29·27
2·81	9·79	9·80	17·93	32·60	19·90	22·18	22·81	28·14	27·88
4·50	9·65	9·35	9·97	15·82	39·29	25·99	23·44	18·22	19·74
14·73	9·23	5·90	1·50	2·27	17·11	60·73	40·31	30·58	27·44
16·88	23·01	16·42	4·69	8·38	9·40	38·17	46·37	31·26	25·64
11·65	18·19	16·50	8·20	9·75	6·08	18·93	21·42	43·63	28·19
6·11	14·53	12·02	11·95	10·44	4·10	11·19	13·08	20·70	36·13
5·05	9·52	7·38	8·31	9·96	3·43	7·81	6·85	10·84	11·31

DZ males within pairs 62 d.f.

MZ females between pairs 49 d.f.

58·92	40·03	44·77	41·29	37·34	49·42	55·76	33·90	30·94	36·67
9·03	74·48	56·68	44·87	49·31	35·89	71·68	44·47	40·32	47·05
−0·13	17·91	68·33	55·14	49·66	38·49	67·20	49·11	49·07	50·42
0·27	0·84	6·35	72·29	51·40	40·99	53·71	50·19	59·66	48·16
−0·98	0·53	3·31	9·17	52·00	34·53	53·65	41·07	45·62	47·94
0·89	0·01	0·40	−0·23	2·16	55·86	45·97	31·67	31·59	33·85
0·67	0·66	−0·22	−0·51	1·71	7·73	100·63	56·36	46·45	52·28
0·34	−3·26	−0·72	1·67	0·40	1·52	11·57	56·22	45·79	45·41
−0·56	−4·27	0·83	−1·47	0·25	0·03	−0·21	6·70	61·96	45·06
−0·12	−0·41	1·92	2·99	0·26	−0·62	−0·72	0·22	5·84	61·94
−2·01	−0·37	−0·13	−0·56	−0·95	0·03	0·96	−0·62	−0·45	5·24

MZ females within pairs 50 d.f.

Appendix (continued)

Mean products matrices for raw data

DZ females between pairs 48 d.f.

50·54	26·50	34·97	25·88	19·38	29·63	37·32	25·74	21·00	19·32
22·38	49·01	34·60	26·00	26·82	20·97	45·31	30·72	25·00	21·43
10·23	32·83	52·24	35·81	29·23	25·77	47·67	39·11	32·24	24·49
10·98	20·89	25·86	44·20	29·20	22·60	37·75	32·15	36·15	29·57
10·23	21·47	20·41	33·55	38·31	15·53	35·65	26·47	28·64	30·19
4·70	9·95	9·95	12·81	13·32	32·42	30·36	23·86	16·98	15·55
15·50	10·10	10·87	11·73	3·35	20·49	67·73	41·54	34·97	31·07
13.08	29·59	24·13	20·87	15·86	10·58	47·42	46·49	29·29	24·16
11·37	20·16	16·64	17·84	8·16	10·78	21·01	22·51	40·22	27·52
9·97	18·18	14·74	20·14	10·50	8·01	21·02	15·86	24·28	35·22
6·78	11·87	13·24	19·37	10·30	6·67	19·82	9·42	13·21	25·73

DZ females within pairs 49 d.f.

Opposite sex siblings between pairs 79 d.f.

37·95	17·47	18·98	16·33	19·75	28·34	21·11	12·81	13·88	13·33
22·90	53·78	34·99	23·44	19·15	18·12	36·99	28·62	24·44	19·02
8·08	34·67	61·04	35·65	20·89	21·77	43·04	42·79	32·38	20·30
6·14	14·09	23·89	46·00	27·58	20·28	37·51	28·12	34·42	23·91
5·48	6·98	9·22	21·15	34·87	21·34	27·59	17·02	24·28	23·54
8·20	11·82	6·83	8·70	18·47	47·11	19·32	15·60	17·09	17·84
13·24	7·13	6·90	4·70	6·90	18·54	66·14	36·27	34·29	26·76
12·30	20·62	18·03	10·13	13·58	11·97	39·77	44·79	28·95	13·98
11·14	14·88	16·49	9·36	9·24	7·59	17·69	28·66	38·77	20·39
5·59	9·11	8·25	10·32	9·45	4·88	10·97	9·20	16·20	28·48
6·30	8·83	4·82	6·66	10·26	5·33	7·44	7·26	6·90	13·84

Opposite sex siblings within pairs 79 d.f.

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Zusammenfassung. (1) Die genetische und Umweltstruktur der Kovarianz zwischen Fingerleistenzahlen bei Zwillings- und Geschwisterdaten wurde unter Verwendung der Methode von Martin und Eaves (1977), angepaßt nach Jöreskog, analysiert.

(2) Das Modell der Umwelt-Kovarianz enthält eine einzelne Faktorenladung für alle zehn Fingerbeeren, aber die meiste Umweltvarianz ist für jeden Finger spezifisch.

(3) Für die additive genetische Varianz gibt es eine gemeinsame Faktorenladung für alle Fingerbeeren. Es finden sich außerdem fünf weitere unabhängige Faktoren, jeder für eine Fingerbeere. Der Daumenfaktor betrifft nur die beiden Dauben, aber die vier Fingerfaktoren betreffen den jeweiligen Finger und die benachbarten Finger.

(4) Ein einzelner gemeinsamer Faktor für die nicht-additive genetische Varianz bewirkt eine beträchtliche Verbesserung des Modells.

(5) Das Muster der genetischen Wirkungen unterscheidet sich zwischen linker und rechter Hand.

(6) Obwohl dasselbe Modell für Männer und Frauen zutrifft, werden unterschiedliche Parameterschätzungen benötigt.

(7) Das Passen des Modells ist empfindlich gegenüber der Skala des Maßes.

Résumé. (1) La structure génétique et mésologique de la covariation entre les comptes de crêtes digitales chez des jumeaux et des germains a été analysée selon la méthode de Martin et Eaves (1977) adaptée de Jöreskog.

(2) Le modèle pour la covariance mésologique contient un seul facteur portant sur tous les dix doigts mais la majeure partie de la variance mésologique est spécifique pour chaque doigt.

(3) Pour la variance génétique additive, il y a un facteur commun portant sur tous les doigts. Il y a aussi cinq autres facteurs indépendants, un pour chaque doigt. Le facteur du pouce porte seulement sur les deux pouces mais les facteurs des quatre doigts portent sur le doigt en question et sur les doigts adjacents.

(4) Un facteur commun unique pour la variance génétique non additive produit une amélioration considérable du modèle.

(5) La configuration des effets génétiques diffère entre les mains gauche et droite.

(6) Bien que le même modèle soit approprié pour les hommes et les femmes, des estimations différentes des paramètres sont nécessaires.

(7) Le bon ajustement des modèles est sensible à l'échelle de mesure.