

**A family study of dermatoglyphic traits in India.  
Resolution of genetic and environmental effects for manus, pes and  
total pattern ridge counts in man**

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**Abstract.** Various constructed ridge count phenotypes were studied in two endogamous populations from peninsular India. Heritabilities were estimated for five summed pattern ridge count traits: fingers and toes together; palms and soles together; fingers and palms together (manus); toes and soles together (pes); and fingers, palms, toes and soles together, defined as the total ridge count in man. In general, these phenotypes were found to be highly heritable, with the summed ridge counts for fingers and toes, and total ridge count showing almost complete determination by additive polygenes. Total manus and pes pattern counts are less heritable. Little or no uterine environmental effects were detected for any of these phenotypes.

**Keywords.** Total pattern ridge counts; dermatoglyphics; heritability; path analysis.

## 1. Introduction

Extensive genetic investigations (see Holt 1968, and Loesch 1983, for reviews) have established beyond a doubt that the total finger ridge count (TFRC) is determined by additive polygenes with independent effects, without dominance and without environmental, in this case uterine, influence. Mathew (1980) reported a similar mechanism for total toe ridge counts (TTRC). Recently, analogous ridge count techniques have been applied to true palmar patterns (Malhotra *et al* 1981a, 1982a) and true plantar patterns (Malhotra *et al* 1981c, 1984c). Subsequently, the genetics of total palmar pattern ridge counts (TPPRC) were investigated (Malhotra *et al* 1981a, b, 1982a; Malhotra and Rao 1982; Karmarkar and Malhotra 1981; Borecki *et al* 1985). Borecki *et al* (1985) additionally studied palmar pattern ridge counts on individual configurational areas and combined counts on interdigital areas II, III and IV. In summary, it appears that the TPPRC is moderately heritable ( $h^2 = 0.52 \pm 0.07$ ) with minimal uterine effects. However, individual palmar areas seem to have different heritabilities, and there is evidence for interpopulation variability in heritability estimates. There is no evidence to suggest that ridge counts for the distal palmar areas (interdigital II, III and IV) are determined any differently than the TPPRC. These same phenotypes were submitted to segregation analysis by Gilligan *et al* (1985) in order to identify any possible major gene effects, and no such evidence was found.

The genetics of the total sole pattern ridge counts (TSPRC) as well as counts on individual plantar areas and combined counts on distal sole areas were studied by Malhotra *et al* (1984b, 1987b). The main findings of these studies are: (1) the heritability of the distal pattern ridge count does not differ from the TSPRC and is moderate, (2) there are indications of uterine effects, and (3) there are differences in the heritability of counts for individual areas within as well as between populations.

Thus far, counts on each of the four dermal areas, i.e., fingers, palms, toes and soles have been investigated separately in order to determine if there are varying degrees of genetic control (Borecki *et al* 1985; Malhotra *et al* 1987). However, we also recognize that individual areas may well share common determinants. The evidence from embryological studies shows that "the ridge patterns of the hand must be conceived as one total system in which the fingers are subdivided afterwards" (DeWilde 1980). This is also true in the case of the foot (Cummins 1929).

Therefore, it may be useful to entertain the hypothesis that the pattern counts of fingers and toes together (FTFPRC and AFTPRC), as the most distal portions of the extremities, may share some common determinants, and that determinants of pattern counts on palms and soles (TPSPRC and APSPRC) may also be similar.

On the other hand, Malhotra *et al* (1982b, 1984c) suggested that it is most likely that the ridge counts on fingers and palmar areas, and on toes and plantar areas are controlled by separate genetic factors. Accordingly they proposed two new quantitative dermatoglyphic measures, namely, total and absolute manus pattern ridge count (TMPRC and AMPRC) and total and absolute pes pattern ridge count (TpPRC and ApPRC). Malhotra *et al* (1984a) showed that the distribution of the trait TMPRC was almost normal with slight negative skewness, and that this trait was more strongly correlated with TFRC than TPPRC. It may be noted here that Karmakar and Malhotra (1981) found a non-significant correlation between the measures TFRC and TPPRC; Malhotra *et al* (1982b) confirmed these results in three other population samples. These later results, therefore, do not strictly correspond with the embryological evidence detailed above.

Malhotra *et al* (1981c) also proposed that counts on fingers, palms, toes and soles of an individual should be pooled together to reflect the total and absolute pattern ridge count in an individual (TRC and ARC). In fact, Cummins and Midlo (1943) did postulate that "all areas (fingers, palms, toes and soles) are to some extent subject to a common control - rigid enough to determine not only the existence of patterns, as distinguished from open fields, but also to regulate pattern type."

The purpose of this paper is to investigate the genetics of 8 traits: 1FTFPRC, AFTFPRC, TMPRC, AMPRC, TpPRC, ApPRC, TRC and ARC among a series of family data drawn from two strictly endogamous Brahmin castes of peninsular India.

## 2. Materials and methods

### 2.1 *The data and variables*

The data have been described in detail in Borecki *et al* (1985). Briefly, dermatoglyphic prints were taken on related individuals from two different

endogamous populations. In Family Series I (FS-I), 125 nuclear families were sampled from the Velanadu Brahmin caste residing in Waltair, including 375 offspring. A twin series consisting of 35 MZ pairs and 62 DZ pairs was also drawn from the population of individuals in this geographic region. A substantial number of these belong to the Velanadu Brahmin caste, although twins from other caste groups were also included. Family Series II (FS-II) consists of 90 families with 235 offspring sampled from the Havik Brahmin caste residing in the western state of Karnataka. Not all of the sampled individuals were included for analysis.

Descriptive statistics for each variable were reported in Borecki *et al* (1985, table 1). Due to technical difficulties, it was not possible to obtain toe prints for the individuals in FS-II, thus the variables involving toe pattern ridge counts were not analyzed for this population. All variables were standardized within father, mother and child groups prior to analysis.

## 2.2 Path analysis

First, we reduce the nuclear family data in terms of estimates of familial correlations. For each phenotype studied, let us define the following variables on the members of a nuclear family with  $s$  children:  $P_F$  = phenotype of father,  $P_M$  = phenotype of mother, and  $P_i$  = phenotype of the  $i$ th child ( $i = 1, \dots, s$ ). Assuming that  $(P_F, P_M, P_1, \dots, P_s)$  follow a suitable multivariate normal distribution, we can write the log-likelihood for the  $j$ th of  $n$  families, denoted by  $\ln L_j$ . Thus, the overall log-likelihood for a random sample of  $n$  families is  $\ln L = \sum_j \ln L_j$ . By assuming that all children are identically distributed, that a parent is equally correlated with any child, and that sib-pairs are equicorrelated,  $\ln L$  is a function of 3 means, 3 variances, and 4 correlations (spouse-spouse, father-child, mother-child, and sib-sib). As discussed by Rao *et al* (1984), the 3 variances and 4 correlations are estimated for each variable, fixing the means at sample values. The correlation estimates are denoted by  $r_i$  ( $i = 1, 4$ ). At the end of estimation, an empirical variance-covariance matrix among the 7 estimates is calculated, denoted by  $S$ . The  $4 \times 4$  sub-matrix in  $S$  corresponding to the 4 correlation estimates is then inverted. This inverse, denoted by  $K$ , is the empirical information matrix among the 4 correlation estimates. This  $K$  matrix is used in the statistical method of analysis described later.

MZ and DZ correlations were estimated from twin data in FS-I by analysis of variance (denoted by  $r_s$  for MZ and  $r_b$  for DZ). The number of twin pairs varies from phenotype to phenotype.

For path analysis of the correlation estimates thus obtained, we consider a special case of a more general model (Rao *et al* 1984), which is similar to the one used earlier for the analysis of ridge counts (Malhotra and Rao 1982). The basic model postulates an additive genotype ( $G$ ), and a uterine environment ( $U$ ). Their effects are assumed to be additive. The two basic parameters are:

- $h$  = effect of genotype on phenotype (square root of genetic heritability).
- $u$  = effect of uterine environment on phenotype.

While the polygenic component is the sole determinant of vertical transmission (parent-offspring), the uterine environment contributes  $u^2$  to sibling and twin cor-

relations. Additional parameters are required to explain the familial correlations for certain phenotypes. Such cases include significant marital correlations, or twin correlations that cannot be explained by  $h$  and  $u$  alone. To accommodate such observations, we introduce the following secondary parameters:

$p$  = correlation between marital phenotypes treated as a copath (see Cloninger, 1980 and Rao *et al.*, 1984 for details).

$t$  = a correlational path between the phenotypes of twins, perhaps induced by simultaneous sharing of the uterine environment, i.e. intrauterine effect.

Although intrauterine effect seems biologically plausible, phenotypic assortative mating does not appear to arise out of biological considerations. Nonetheless, the parameter  $p$  is required to explain the observed pattern of correlations for some phenotypes. In fitting path models, we first fit only the significant components of the basic model (i.e., one or both of  $h$  and  $u$ ). Only when such a model yields a significant misfit, we include one or the other of the secondary parameters. In this procedure, we are guided by the principle of parsimony.

### 2.3 Statistical analysis

Fitting path models to the sample correlations from nuclear family data follows the method of analysis from Rao *et al.* (1984), which is called Method 2. Assuming the joint distribution of the 4 nuclear family correlations to be approximately multivariate normal, and assuming univariate normality for the 2 transforms of the twin correlations, the log-likelihood may be approximated by (Borecki *et al.* 1985)

$$\ln L = -\frac{1}{2} \chi^2 + \text{constant},$$

where,

$$\chi^2 = \begin{cases} \sum_{i=1}^4 \sum_{j=1}^4 (r_i - \rho_i) K_{ij} (r_j - \rho_j), & \text{without twins.} \\ \sum_{i=1}^4 \sum_{j=1}^4 (r_i - \rho_i) K_{ij} (r_j - \rho_j) + N_5 (z_5 - \xi_5)^2 + N_6 (z_6 - \xi_6)^2, & \text{with twins.} \end{cases}$$

where the  $\rho$ 's are predicted correlations derived as functions of certain path coefficients,  $K_{ij}$  are elements of the  $K$  matrix obtained earlier, and  $z_i$  is Fisher's  $z$ -transformation of a sample twin correlation with sample size  $N_i$ , and  $\xi_i$  is the  $z$ -transformation of the corresponding predicted correlation ( $i = 5$  for MZ and  $i = 6$  for DZ). Goodness-of-fit of a model may be measured by the residual  $\chi^2$  with 4- $w$  d.f. (degrees of freedom) for FS-II and 6- $w$  d.f. (degrees of freedom) for FS-I, where  $w$  is the number of parameters that are estimated. Likelihood ratio tests of specific null hypotheses may be carried out by subtracting the residual  $\chi^2$  under the general model from that under the null hypothesis. Degrees of freedom are obtained by a similar subtraction. All these methods are implemented in PATHMIX, a FORTRAN program on a HARRIS computer. The program can be obtained from the authors.

Table 1. Summary correlations for Family Series I with twins.

Variable number	Variable name	Family data						Twin data					
		$r_{FM}$	$\bar{N}_{FM}$	$r_{FC}$	$\bar{N}_{FC}$	$r_{Mm}$	$\bar{N}_{Mm}$	$r_{Mz}$	$\bar{N}_{Mz}$	$r_{CC}$	$\bar{N}_{CC}$	$r_{Cz}$	$\bar{N}_{Cz}$
1	Total fingers + toes (max) (TF+PTC)	0.094	103	0.333	153	0.232	149	0.355	155	0.744	33	0.535	57
2	Total fingers + toes (abs)	0.005	102	0.446	149	0.444	146	0.516	150	0.966	32	0.654	53
3	Total palm + sole (max) (TPSPRC)	0.185	114	0.273	225	0.225	192	0.336	227	0.620	33	0.565	57
4	Total palm + sole (abs) (APSPRC)	0.169	112	0.347	214	0.312	188	0.382	221	0.686	33	0.550	57
5	Manus (max) (TMPRC)	-0.130	127	0.420	196	0.401	180	0.478	205	0.892	34	0.586	61
6	Manus (abs) (AMPRC)	-0.089	127	0.444	187	0.421	177	0.521	198	0.943	34	0.606	61
7	Pes (max)	0.185	96	0.430	158	0.443	149	0.481	148	0.862	33	0.686	54
8	Pes (abs) (APPRC)	0.121	97	0.401	165	0.461	160	0.439	152	0.919	33	0.669	54
9	Total pattern count (max) (TRC)	0.056	96	0.469	153	0.448	141	0.513	152	0.900	32	0.743	53
10	Total pattern count (abs) (TRC)	0.025	98	0.450	150	0.461	141	0.530	151	0.938	32	0.720	53

\* Within family correlations, and sample sizes for relatives taken pairwise, where F = father, M = mother, and C = child.

Table 2. Summary correlations for Family Series II.

Variable number	$\hat{r}_{FM}$	$\hat{N}_{FM}$	$\hat{r}_{FC}$	$\hat{N}_{FC}$	$\hat{r}_{MC}$	$\hat{N}_{MC}$	$\hat{r}_{CV}$	$\hat{N}_{CV}$
3	-0.271	56	0.238	100	0.167	107	0.370	113
4	-0.298	57	0.252	105	0.067	106	0.345	126
5	-0.155	77	0.206	99	0.454	107	0.491	99
6	-0.144	78	0.261	107	0.486	111	0.482	109

### 3 Results

Estimates of the sample correlations for both family series are shown in tables 1 and 2. The estimated sample sizes are also shown, corresponding to the effective number of independent pairs of observations leading to these correlations with their associated standard errors. There are significant positive and negative marital correlations. Twin correlations with respective sample sizes are also shown.

The fitted solutions are shown in table 3. It is interesting to note that the estimated heritabilities for these traits are significantly higher than the moderate heritabilities found for the individual component areas. The exception is the combined palm and sole ridge count phenotype (TPSPRC and APSPRC; variables 3 and 4) which is moderately heritable. This finding is particularly interesting in contrast to the high heritability found for ridge counts combined over fingers and toes (TFTPRC and AFTPRC; variables 1 and 2).

Twin intrauterine effects were significant for several variables in FS-1, and simple uterine environmental effects were significant for two of the variables. The parameter  $p$  was invoked twice to improve the goodness-of-fit.

In only one case was the estimated heritability different for the maximum and absolute measures of a phenotype, and this was for the combined palmar and sole ridge counts (TPSPRC and APSPRC; variables 3 and 4 in FS-1). Otherwise, maximum and absolute measures of ridge counts for other variables appear to be equally heritable.

Table 3. Best fit solutions to the path model.

Variable numbers	Family Series I with twins					Family Series II			
	$\chi^2$	d.f.	$h^2 \pm s.e.$	$u^2 \pm s.e.$	Other $\pm s.e.$	$\chi^2$	d.f.	$h^2 \pm s.e.$	$p \pm s.e.$
1	5.33	5	0.90 ± 0.06	0	—	—	—	—	—
2	0.32	4	0.91 ± 0.06	0.09 ± 0.04	—	—	—	—	—
3	4.10	3	0.47 ± 0.07	0	$t = 0.25 \pm 0.12$ $p = 0.20 \pm 0.09$	7.25	3	0.59 ± 0.10	0
4	9.38	5	0.65 ± 0.07	0	—	3.88	2	0.57 ± 0.13	4
5	5.21	5	0.91 ± 0.04	0	—	7.05	3	0.79 ± 0.08	0
6	1.50	4	0.93 ± 0.05	0.07 ± 0.03	—	5.09	3	0.85 ± 0.07	0
7	6.55	4	0.74 ± 0.07	0	$t = 0.25 \pm 0.12$	—	—	—	—
8	2.73	4	0.77 ± 0.07	0	$t = 0.23 \pm 0.12$	—	—	—	—
9	7.50	5	0.89 ± 0.06	0	—	—	—	—	—
10	7.54	5	0.91 ± 0.06	0	—	—	—	—	—

\* $p = 0.26 \pm 0.12$

#### 4. Discussion

Ridge counts in man appear to be highly heritable phenotypes. Recognizing that individual areas in all dermal surfaces have differing degrees of genetic determination (Borecki *et al* 1985; Malhotra *et al* 1987), some general conclusions can be drawn from these analyses pooling ridge counts over various combinations of areas. Combined finger and toe ridge counts (FTFPRC and AFTPRC) are significantly more heritable ( $h^2 \approx 0.9$ ) than combined palm and sole pattern ridge counts ( $h^2 \approx 0.6$ ). The data from FS-1 further suggests that the manus ridge counts (TMPRC and AMPRC) have a higher heritability than pes ridge counts (TpPRC and ApPRC), approximately 90% vs. 75%. Overall, the total ridge count in man (TRC and ARC) is highly heritable ( $h^2 \approx 90\%$ ) showing no significant uterine effects.

Considering the results of the present analysis, as well as the previous analyses of the individual areas (Borecki *et al* 1985; Malhotra *et al* 1987), it appears that although simple intrauterine effects were occasionally significant, the simultaneous sharing of the uterine environment more often caused additional similarity between members of a twin pair. On the whole, it does not appear that uterine environment plays an important role in the determination of ridge counts.

Estimated heritabilities of pattern ridge count phenotypes summed over various individual areas must be interpreted with caution as they appear to reflect an average figure over the component ridge counts and obscure variations in  $h^2$  for different individual areas. Developmental events can suggest ways of combining ridge counts into potentially meaningful phenotypes, as we have done. Now that we have shown these phenotypes to be highly heritable, perhaps a better way to approach this problem is to consider individual area pattern ridge counts and attempt to distinguish common and unique genetic determinants taking the intercorrelations between these into account.

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