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A Family Study of Dermatoglyphic Traits in India: A Search for Major Gene Effects on Palmar Pattern Ridge Counts

S.B. GILLIGAN, I.B. BORECKI, S. MATHEW, K.C. MALHOTRA, AND D.C. RAO

Department of Biology, Washington University, St. Louis, Missouri 63130 (S.B.G.), Division of Biostatistics, Departments of Preventive Medicine and Psychiatry (I.B.B., D.C.R.) and Department of Genetics (D.C.R.), Washington University School of Medicine, St. Louis, Missouri 63110, Department of Human Genetics and Anthropology, Albert Ludwigs University, Freiburg, West Germany (S.M.), and Anthropometry and Human Genetics Unit, Indian Statistical Institute, Calcutta, India (K.C.M.)

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ABSTRACT Palmar pattern ridge counts were subjected to segregation analysis in an attempt to identify possible major gene effects on these dermatoglyphic traits. The phenotypes considered were total palmar pattern ridge count, and ridge counts for the right interdigital III and IV and left interdigital IV individual palmar areas (sample sizes were too small for the other palmar areas). Evidence of familial resemblance was found for all of the phenotypes studied, and initial evidence for a major effect was found for all but the right palm interdigital III ridge count. However, this initial evidence could be attributed to nongenetic effects in each case, including skewness in the trait distribution. Tests for agreement with Mendelian transmission frequencies were found to be very useful in discriminating between a non-Mendelian major effect and a major gene. We concluded against a major gene effect for any of these traits, and multifactorial inheritance remains a plausible alternative explanation for the familial resemblance.

Malhotra et al. (1981, 1982) recently developed a technique of counting ridges on palmar patterns and defined a new measure called the *total palmar pattern ridge count* (TPPRC). This trait is simply the sum of ridge counts for all ten palmar configurational areas. In their initial investigation of the TPPRC, Malhotra et al. (1981) reported that total finger and palmar ridge counts were nonsignificantly correlated, thus suggesting that finger and palmar elements may be inherited independently. The genetics of the TPPRC has been investigated by Malhotra and Rao (1982) using the method of path analysis. They concluded that about one-third of the variation in this trait is accounted for by additive genetic factors ($h^2 = 0.37 \pm 0.06$).

In further investigations, Malhotra et al. (1985a,b) and Kamali et al. (1985) reported significant differentiation of several populations in India and Iran with respect to both size and frequency of patterns in individual palmar areas. This finding, coupled with the relatively small heritability reported by Malhotra and Rao (1982) for the TPPRC, led to the suggestion that the genetics of ridge counts for individual palmar areas should also be studied separately.

Borecki et al. (1985) investigated the multifactorial basis of ridge counts for individual palmar areas, combined distal areas, and the TPPRC in families belonging to two strictly endogamous Brahmin castes of peninsular India. The two populations were found to be different with respect to the heritability of ridge counts for individual areas, with different degrees of polygenic determination for individual palmar areas within each of the family series. Estimated heritabilities ranged from 0.28 to 0.66 in the first series, and from 0 to 0.52 in the second. The proportion of

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genetic variation for the TPPRC was on the order of 52% in both populations studied.

To date, no systematic investigation of major gene effects on such traits has been carried out. The purpose of the present investigation is to test hypotheses regarding possible major gene effects using complex segregation analysis. Several individual palmar pattern ridge counts as well as the TPPRC are considered. The analysis is based on one of the family series analyzed by Borecki et al. (1985).

THE DATA AND VARIABLES

The data were described in detail elsewhere (Borecki et al., 1985). Originally, dermatoglyphic prints were taken on related individuals from two endogamous populations. In the present paper, only one family study is examined, consisting of 125 nuclear families with 375 offspring, sampled from the Velanadu Brahmin caste residing in Waltair, Andhra Pradesh.

The dermatoglyphic traits considered in the present study include palmar pattern ridge counts for several individual configurational areas and total palmar pattern ridge counts (sum of the counts for all ten individual areas on both palms). The method for obtaining ridge counts is found in Malhotra et al. (1981). For each of these traits both maximum and absolute measures were considered. The maximum measure is defined as the highest of multiple ridge counts when there is more than one triradius; the absolute measure for such an area is the sum of the multiple ridge counts.

Individuals with missing values and with open field patterns, who are traditionally assigned a count of zero, were excluded from analysis. In these cases, a ridge count of zero implies no pattern rather than no ridges; thus the value of zero does not constitute a proper measurement in the quasi-continuous distribution of ridge counts. For further discussion on the exclusion of zero values see Borecki et al. (1985). Not all individual palmar areas were analyzed here because exclusion of individuals with no triradii led to inadequate sample sizes for some areas. Three areas were analyzed: right interdigital III (RPIII) and right and left interdigital IV (RPIV, LPIV). Descriptive statistics of the distributions for each of the areas studied are given in Table 1. Coefficients of skewness (β_1) and kurtosis (β_2-3) are given where both are zero for a normal distribution. Each variable was standardized with the respective sample mean and sample variance of Table 1. These standardized counts are subjected to further analysis.

METHODS OF GENETIC ANALYSIS

Commingling analysis

Commingling analysis is used to distinguish between spurious skewness and mixtures of distributions. Since spurious skewness may be interpreted as a mixture of distributions due to major gene, MacLean et al. (1976) suggested the use of a power transformation to remove the effects of skewness, thus providing a more conservative test of the major gene hypothesis.

For a standardized score x, the power transformed score y is given by

$$y = \begin{cases} \frac{r}{p}\left[\left(\frac{x}{r}+1\right)^p - 1\right], \text{ if } p \neq 0 \\ r \ln\left(\frac{x}{r}+1\right), \text{ if } p = 0 \end{cases} \quad (1)$$

where the constant r is chosen such that every $(\frac{x}{r} + 1)$ is positive. The computer program SKUMIX fits one, or a mixture of two or three distributions, described by up to six parameters. The six parameters are (1) e, the common variance in each component, (2) u, the overall mean, (3) q, which determines the relative proportion (q^2) of the component distribution with the highest mean, (4) t, the

TABLE 1. Descriptive statistics for variables analyzed in the family study[1]

Variable	N	Mean	SD	$\gamma_1 \pm$ SE	$\gamma_2 \pm$ SE
Total palmar pattern ridge count (max)	617	30.366	20.878	1.474 ± 0.099	2.758 ± 0.099
Total palmar pattern ridge count (abs)	617	33.877	24.048	1.648 ± 0.099	3.895 ± 0.099
Right palmar III (max)	411	8.180	3.862	0.499 ± 0.121	0.439 ± 0.121
Right palmar III (abs)	411	8.255	3.901	0.469 ± 0.121	0.340 ± 0.121
Right palmar IV (max)	291	10.141	5.733	0.489 ± 0.144	−0.589 ± 0.144
Right palmar IV (abs)	291	10.703	6.360	1.000 ± 0.144	1.656 ± 0.144
Left palmar IV (max)	424	8.462	5.286	1.127 ± 0.119	2.758 ± 0.119
Left palmar IV (abs)	424	9.475	5.766	1.131 ± 0.119	2.504 ± 0.119

[1] $\gamma_1 = \beta_1$ and $\gamma_2 = \beta_2$-3, and therefore, $\gamma_1 = \gamma_2 = 0$ for a normal distribution.

displacement between the two extreme components, (5) d, the amount of displacement for the middle component, and (6) p, the power transformation parameter (Morton et al. 1983).

Parameters are estimated by the method of maximum likelihood, and tests of hypotheses are carried out using the likelihood ratio test. The test criterion, given by twice the difference between the log-likelihoods under two appropriate models, is distributed asymptotically as a chi-square whose degrees of freedom are equal to the difference in the numbers of parameters in the two models.

Parameters are estimated under each of four hypotheses: (I) a single normal distribution (E, u), (II) a single skewed distribution (E, u, p), (III) a mixture of two normal distributions (E, u, t, q), and (IV) a mixture of two skewed distributions (E, u, t, q, p). For the latter two hypotheses, d is set to zero. Relevant contrasts include hypothesis I vs. II, I vs. III, III vs. IV, and II vs. IV.

Segregation analysis

Segregation analysis was performed using the unified mixed model as implemented in the computer program POINTER (Lalouel et al., 1983; Lalouel and Morton, 1981; Morton and MacLean, 1974). This model assumes that a trait y results from one or more of three sources: (1) a major transmissible effect, g, (which may or may not be a Mendelian gene), (2) a multifactorial transmissible component, c, and (3) a random, nontransmitted environment, e, with $y = g + c + e$. These three factors are assumed to be independently distributed. Factors c and e are normally distributed, N(0, C) and N(0, E), respectively. The major effect is modeled as a single locus with two alleles A and a, leading to three genotypic classes with prior probabilities expressed in terms of a binomial parameters, q, the prior probability of allele a in the reference population. Genotypic effects can be expressed either as three means μ_{AA}, μ_{Aa}, μ_{aa}, or alternatively, in terms of the following three alternate indices. The expected value of g is u, the overall mean, the distance between the two homozygous means on the scale of x is called displacement, $t = \mu_{aa} - \mu_{AA}$, and the position of the heterozygous mean relative to both homozygous classes is expressed by the dominance parameter, $d = (\mu_{Aa} - \mu_{AA})/(\mu_{aa} - \mu_{AA})$. It follows that $E(y) = E(g) = u$; the variance of y, denoted by V, is such the $V = G + C + E$, where G is the variance due to the major effect. Multifactorial heritability is defined by $H = C/V$. Thus, in the mixed model as used here, there are six parameters under the assumption of Mendelian transmission at the major effect: V, u, d, t, q, and H. However, specification of the unified model also requires the definition of transmission rules from parent to offspring. Random mating is assumed. The transmission frequencies τ_1, τ_2, τ_3 denote, respectively, the probabilities of genotypes of AA, Aa, and aa transmitting allele A, with $\tau_1 = 1$, $\tau_2 = 1/2$, $\tau_3 = 0$ for Mendelian transmission. These transmission frequencies are also estimable in addition to the other six parameters.

Parameters are estimated under each of a variety of hypotheses by the method of maximum likelihood, and null hypotheses are tested using the likelihood ratio criterion. Setting all parameters but mean and variance to zero provides a test of no familial transmission. No major effect corresponds to $d = t = q = 0$, and no polygenic heritability corresponds to $H = 0$. If a major effect is inferred by rejecting the null hypothesis $d = t = q = 0$, it is important to test for Mendelian transmission before concluding that the major effect is a major gene; this can be done by iterating τ_2, or more generally, by iterating all three transmission probabilities, in addition to the other relevant parameters of the mixed model.

RESULTS

Total palmar pattern ridge counts

The results of commingling analysis for both the maximum and absolute measures of the total palmar pattern ridge count (TPPRCM and TPPRCA, respectively) are presented in Table 2. Residual skewness under the assumption of a single distribution (hypothesis I vs. II) or a mixture of two distributions (III vs. IV) was highly significant for both measures of the trait (TPPRCM: I vs. II : $\chi^2_1 = 225.35$; III vs. IV : $\chi^2_1 = 108.63$; TPPRCA: I vs. II : $\chi^2_1 = 252.45$; III vs. IV : $\chi^2_1 = 192.63$). Given that the skewness is substantial, the relevant test for commingled distributions involves the contrast of hypothesis II vs. IV: a mixture of two distributions fits significantly better than one, for both the TPPRCM ($\chi^2_2 = 22.20$) and the TPPRCA ($\chi^2_2 = 89.45$).

Thus there is significant commingling for both TPPRCM and TPPRCA even when skewness is incorporated. In order to demonstrate the effects of skewness on the inference regarding major gene effects, segregation analysis is performed on the untransformed data as well as on the power-

TABLE 2. Commingling analysis of total palmar pattern ridge counts (d = 0)

Measure	Hypothesis	−2 lnL+c	df	E	u	q	t	p
Maximum	I. One normal	1,750.02	2	0.978	0.000	0	0	1
	II. One skewed	1,524.67	3	0.757	−0.243	0	0	−2.515
	III. Mix 2 normal	1,611.10	4	0.467	0.000	0.328	2.349	1
	IV. Mix 2 skewed	1,502.47	5	0.341	−0.254	0.694	1.297	−2.634
Absolute	I. One normal	1,750.44	2	0.999	0.000	0	0	1
	II. One skewed	1,497.99	3	0.728	−0.243	0	0	−2.622
	III. Mix 2 normal	1,601.17	4	0.435	0.000	0.304	2.474	1
	IV. Mix 2 skewed	1,408.54	5	0.360	−0.014	−0.530	1.273	−2.139

transformed data. The estimate of p under the assumption of noncommingling is generally used for such purposes.

The results of segregation analysis of the total palmar pattern ridge count—maximum (TPPRCM) are shown in Table 3. Rejection of the null hypothesis of no familial transmission ($\chi^2_4 = 116.02$) for the untransformed data demonstrates that the trait is heritable. Both multifactorial transmission ($\chi^2_1 = 4.93$) and a major effect ($\chi^2_3 = 72.08$) are significant. To explore if this major effect is attributable to a major gene, Mendelian transmission was then tested. τ_2 alone significantly differed from its Mendelian value of 1/2 ($\chi^2_1 = 4.10$), and also all three τ values differed from their Mendelian values ($\chi^2_3 = 15.80$). Therefore, although there is substantial evidence of a major effect, it appears that Mendelian transmission is untenable. Most if not all of the evidence for the major effect must have resulted from the spurious skewness in the untransformed data ($\beta_1 = 1.474 \pm 0.099$). This suggests that the transmission frequencies play an important role in safeguarding against falsely inferring a major gene based on skewed data.

Analysis of the same data after the effects of skewness were removed (p = −2.515) still showed good evidence for familial transmission ($\chi^2_4 = 69.63$). However, the test of no major effect is no longer significant at the conventional df (3) ($\chi^2 = 6.45$). Since the number of degrees of freedom for this test is actually between 2 and 3, one may not consider it as conclusively nonsignificant. To further investigate this situation, we also estimated all three transmission frequencies. This shows that even if a major effect is entertained, it cannot be due to a Mendelian gene ($\chi^2_3 = 18.63$). The inference would have been different if one estimated τ_2 only. In this case, the hypothesis that $\tau_2 = 1/2$ is not rejected ($\chi^2_1 = 2.1$). We can conclude that the evidence for a major effect in the untransformed data can be attributted to the skewness in the distribution as that evidence is lost after power transformation. Even without transformation, the rejection of Mendelian transmission makes a major gene for TPPRCM highly unlikely.

A similar segregation analysis was performed on the total palmar pattern ridge count—absolute (TPPRCA), and the results are presented in Table 4. The untransformed data give significant evidence for familial resemblance ($\chi^2_4 = 128.92$), multifactorial transmission ($\chi^2_1 = 15.93$), and also for a major effect ($\chi^2_3 = 76.85$). Testing for Mendelian transmission with τ_2 alone showed no significant deviation ($\chi^2_1 = 2.60$). However, when all three τ's were iterated, the Mendelian hypothesis became untenable ($\chi^2_3 = 10.45$). This demonstrates the need to test on all three transmission frequencies simultaneously. Evidence for familial resemblance is maintained in the transformed data ($\chi^2_4 = 54.31$), however, again evidence for a major effect is lost ($\chi^2_3 = 2.68$). Since the chi-square test is obviously nonsignificant on any d.f., further tests of Mendelian transmission are not necessary. Again, we conclude against a major gene. It is interesting to note that the test of τ_2 alone was not sufficient to reject the Mendelian hypothesis in the analysis of the untransformed data as the abberant segregation is evident in the probability of obtaining A from the aa genotype represented by τ_3. Once again, only significant skewness in the untransformed data appears to have resulted in the significant evidence for a major effect, since power transformation removed that evidence.

Individual palmar pattern ridge counts

The variables analyzed were ridge counts for right palmar interdigital III (RPIII) and right and left interdigital IV (RPIV, LPIV). For each trait both maximum and absolute counts were examined. The power transformation parameter (p) was estimated under the assumption of a single distribution. The

TABLE 3. Segregation analysis of total palmar pattern ridge count (maximum)

Hypothesis	−2 lnL+c	V	u	d	t	q	H	τ_1	τ_2	τ_3
Untransformed data										
Mixed model	875.78	0.763	−0.144	0.141	2.544	0.226	0.270	1	.5	0
No familial transmission	991.80	0.353	−0.066	0	0	0	0	—	—	—
No major effect	947.86	0.323	−0.113	0	0	0	0.514	—	—	—
No multifactorial component	880.71	0.708	−0.186	0.250	3.522	0.108	0	1	.5	0
$\tau_2 = \frac{1}{2}$	871.68	0.994	0.033	0.140	2.494	0.528	0.217	1	0.651	0
$\tau_1 = 1, \tau_2 = \frac{1}{2}$, $\tau_3 = 0$	859.98	0.488	−0.353	0.118	0.223	0.120	0.521	0.812	0.554	0.191
Transformed data (p = −2.515)										
Mixed model	850.17	0.694	−0.401	0.536	1.274	0.294	−0.141	1	.5	0
No familial transmission	919.81	0.703	−0.276	0	0	0	0	—	—	—
No major effect	856.62	0.687	−0.599	0	0	0	0.480	—	—	—
$\tau_2 = \frac{1}{2}$	848.07	0.683	−0.232	0.888	1.271	0.382	0.145	1	0.615	0
$\tau_1 = 1, \tau_2 = \frac{1}{2}$, $\tau_3 = 0$	831.54	0.385	−1.012	0.514	1.976	0.121	0.145	0.722	0.592	0.203

TABLE 4. Segregation analysis of total palmar pattern ridge count (absolute)

Hypothesis	−2lnL+c	V	u	d	t	q	H	τ_1	τ_2	τ_3
Untransformed data										
Mixed model	841.80	0.732	−0.163	0.088	2.508	0.229	0.220	1	.5	0
No familial transmission	970.72	0.812	−0.064	0	0	0	0	—	—	—
No major effect	918.65	0.779	−0.198	0	0	0	0.876	—	—	—
No multifactorial component	857.73	0.708	−0.177	0.209	2.896	0.179	0	1	.5	0
$\tau_2 = \frac{1}{2}$	839.20	0.910	−0.034	0.090	2.516	0.298	0.183	1	0.028	0
$\tau_1 = 1, \tau_2 = \frac{1}{2}$, $\tau_3 = 0$	831.35	0.725	−0.155	0.086	2.256	0.268	0.274	0.918	0.533	0.433
Power-transformed (p = −2.682)										
Mixed model	845.14	0.652	−0.394	0.624	1.416	0.267	0.062	1	.5	0
No familial transmission	899.45	0.670	−0.285	0	0	0	0	—	—	—
No major effect	847.82	0.658	−0.381	0	0	0	0.425	—	—	—

estimated p values with their associated chi-square values are presented in Table 5. For all areas studied, the distributions of both maximum and absolute individual palmar pattern ridge counts exhibited significant skewness.

Both the untransformed and power-transformed variables were subjected to segregation analysis. The test of the no-major-gene hypothesis for RPIIIM and RPIIIA in untransformed or power-transformed data was not significant. Since this initial evidence is lacking, it is not necessary to perform further analyses regarding transmission on these variables.

The qualitative results of tests of the three relevant hyotheses (no major gene, $\tau_2 = 1/2$, and $\tau_1 = 1, \tau_2 = 1/2, \tau_3 = 0$) for the remaining variables are presented in Table 6. Evidence

TABLE 5. Estimation of the power parameter p from SKUMIX for individual palmar pattern ridge counts

Variable	p	χ^2_1
RPIIIM	−0.096	18.17
RPIIIA	−0.053	16.59
RPIVM	−0.745	21.96
RPIVA	−1.323	49.67
LPIVM	−1.469	81.26
LPIVA	−1.282	77.75

for a major effect persisted for RPIVM even after transformation. Although τ_2 alone did not differ significantly from 1/2, Mendelian transmission could be rejected when all three τ's were estimated in either case. No major effect was detected for the untransformed RPIVA; however, analysis of the power-transformed data does give evidence for such an

TABLE 6. Tests regarding a major gene for individual palmar pattern ridge counts[1]

Trait	RPIVM		RPIVA		LPIVM		LPIVA	
Power-transformed?	No	Yes	No	Yes	No	Yes	No	Yes
No major effect	•	•	NS	•	•	•	•	?
$\tau_2 = \frac{1}{4}$	NS	NS	–	NS	NS	NS	NS	–
$\tau_1 = 1, \tau_2 = \frac{1}{4}, \tau_3 = 0$	•	•	–	•	?	•	NS	–

[1] • Likelihood ratio test significant at the $P = 0.05$ level or less.
NS, estimated parameters are not significantly different from the proposed hypothesis; –, test not performed; ?, converged solution not obtained, no conclusion.

effect. Here is an instance where perhaps the transformation actually produces the evidence we seek, rather than providing a conservative approach to major gene inference. If this is true, then we would not expect to find Mendelian transmission, which is indeed rejected.

Similar to the corresponding area on the right palm, the no-major-gene hypothesis was rejected for both untransformed and transformed LPIVM, with no significant deviations of τ_2 alone from its Mendelian value. It was not possible to obtain a converged model including all three τ's for the untransformed data; however, Mendelian transmission was rejected for the transformed data. Initial evidence for a major effect in the untransformed LPIVA was not testable in the transformed data owing to the fact that a converged solution could not be obtained. Further tests of Mendelian transmission in the untransformed data showed good agreement with the Mendelian hypothesis. However, recognizing that these data are significantly skewed, we were compelled to attempt an additional test corresponding to no transmission of a major effect. In this case, all the τ's are set equal, implying that the transmission of the putative gene is not dependent on the parental genotype; clearly, this is a valid representation of a non-Mendelian hypothesis. The no-transmission hypothesis also could not be rejected for these data. Thus, the transmission of the detected major effect is not clear.

Therefore, we conclude that there is no evidence for major gene effects on any of these variables. For those that gave initial indications of a major effect in untransformed data, either the evidence was lost upon removal of skewness in the distribution, or it was shown to be not due to a gene. In several instances, the test of τ_2 alone proved to be inadequate to detect non-Mendelian segregation, and in one instance, a specific test of no-transmission-of-major-effect proved useful.

Significant familial resemblance was found for each of these traits, and since a major gene cannot be inferred to account for this, multifactorial transmission is implicated. In fact, tests of the null hypothesis of no multifactorial component ($H = 0$) were consistently rejected. Thus, the value of H obtained under the no major gene hypothesis is a good estimate of the heritability. The values obtained are presented in Table 7. They compare favorably with the estimates from Borecki et al. (1985) using path analysis, which are also presented in Table 7.

DISCUSSION

Since the technique for obtaining ridge counts on palmar configurational areas was presented in 1981, the notion that ridge counts in these areas are controlled by heritable factors has been supported by several studies (Malhotra et al. 1981; Malhotra and Rao, 1982; Borecki et al. 1985). These studies have suggested polygenic heritability and have emphasized the need to explore and define biologically meaningful phenotypes. To this end, ridge counts for individual configurational areas and summed over several areas have been studied. In addition, both maximum and absolute counts have been considered. In general, all of these various phenotypes have been shown to be heritable and it appears useful to consider individual areas separately. Thus far, there in no indication as to whether it is more appropriate to study maximum or absolute counts.

In the present investigation, the query was posed: is there a detectable major heritable factor that imposes discontinuous variation on the distribution of ridge count phenotypes? Detection of such a factor could aid in the definition of meaningful phenotypes as well as clarify the genetic etiology of these traits.

Analyses of the total palmar pattern ridge counts (TPPRCM and TPPRCA) suggest that

TABLE 7. Comparison of estimates of heritability for palmar pattern ridge counts

Variable	Heritability ± SE segregation analysis (present study)	Heritability ± SE path analysis[1] (Borecki et al., 1985)
TPPRCM	0.51 ± 0.09	0.52 ± 0.07
TPPRCA	0.38 ± 0.06	0.45 ± 0.07
RPIIIM	0.56 ± 0.13	0.53 ± 0.09
RPIIIA	0.56 ± 0.12	0.51 ± 0.09
RPIVM	0.59 ± 0.13	0.61 ± 0.10
RPIVA	0.46 ± 0.10	0.49 ± 0.09
LPIVM	0.46 ± 0.10	0.49 ± 0.08
LPIVA	0.23 ± 0.09	0.28 ± 0.08

[1]The analysis utilized additional data on twins.

there is significant commingling in the distributions of the ridge counts, which could not be attributed to a major gene. Simulation studies have shown that mixed-model segregation analysis is sensitive to skewness and will tend to interpret it as a major effect (MacLean et al. 1975). Thus, transforming skewed data is essential prior to segregation analysis. Even when one fails to acount for skewness, the addition of transmission probabilities in the unified mixed model appears to provide a good safeguard against false conclusion of a major gene. When transmission was tested for these traits, significant deviations from the expected Mendelian ratios were found, making a major gene hypothesis *unlikely*.

Analysis of the LPIVA trait provides an instance in which the appropriate hypotheses were not testable in the power-transformed data. Further analysis of the skewed data underscores the importance of exploring the transmission properties of the major effect. It may be tempting to tentatively postulate a major gene on the basis of the tests of Mendelian transmission; however, it was shown that non-Mendelian transmission was also compatible with the data. This finding is just as important as compatibility with Mendelian expectations, and casts serious doubt on the tenability of a major gene hypothesis.

For two of the individual pattern ridge counts, there was no evidence for a major effect (RPIIIM and RPIIIA). Analysis of RPIVM and LPIVM provides consistent evidence for a major effect. However, it was shown that the major effect does not segregate in a Mendelian fashion. Thus, the interpretation of a major gene for these traits is not tenable. However, there is commingling which may be attributable to an environmental effect, perhaps mediated through the uterine environment when ridges were being formed.

Analysis of the RPIVA trait presents a very interesting case. The power transformation traditionally has been applied to data to decrease the probability of falsely asserting a major effect. For this case, it appears that the power transformation emphasized those aspects of the data that evidence commingling. We were able to show that the transmission was not compatible with the Mendelian gene hypothesis, suggesting that the transformation itself was responsible for the initial evidence of a major effect. However, it is conceivable that tests of transmission properties may be uninformative in other instances, warranting extreme caution in interpreting such results.

The addition of transmission frequencies in the unified mixed model has been very valuable in our search for major gene effects. However, r_2 alone has been found to be sometimes insufficient for the purpose of rejecting Mendelian inheritance. It may be that abberant segregation is more pronounced from the "homozygous" genotypes. For example, the analysis of the untransformed TPPRCA gave a nonsignificant chi-square for the test on r_2, yet when all three r's were iterated, it was found that the homozygous genotype aa segregates an A allele over 40% of the time. This is clearly inconsistent with known biological mechanisms. Therefore, just as it is important to adjust for skewness prior to segregation analysis, it seems equally important to attempt to test the full Mendelian hypothesis even on power-transformed data.

We conclude that the etiology of palmar dermatoglyphic traits are influenced by additive polygenes to a moderate extent with possible uterine environmental factors affecting the distribution of some ridge count phenotypes (see also Borecki et al., 1985). It does not appear that major genes play a significant role in the genetic etiology of these traits. While we are not certain of the extent to which our results were influenced by the continuous treatment of discrete ridge count data, given the range of variation we feel confident that such an effect is minimal.

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