GENETIC STUDY OF HYPOTHENAR PATTERNS ON THE PALM: ESTIMATION OF THE HERITABILITY OF LIABILITY

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Summary   Palm prints of 349 Japanese families, including 340 pairs of parents and 650 children, were studied with special reference to the inheritance of hypothenar true patterns.   It was suggested that the segregation data were more consistent with the hypothesis that hypothenar patternings were transmitted from parents to their children by a multifactorial rather than a simple genetic system.   It was demonstrated that individuals with true patterns on both hands had more genetic potential for the patterns than those with unilateral manifestation.   The detailed observation of pedigrees revealed that genetic bases corresponding to radial loops and to ulnar loops might be different from each other and that the genes corresponding to radial loops might be related also to the manifestation of carpal loops and whorls.   Based on Falconer's method, the heritability values were calculated at $63.9 \pm 9.0\%$ for the manifestation of radial loops, $75.8 \pm 19.8\%$ for that of ulnar loops, and $62.8 \pm 10.7\%$ for the bilateral manifestation of radial loops, indicating a considerably strong genetic determination in hypothenar patternings.

INTRODUCTION

The hypothenar configuration is of extremely variable patterns as compared with the other configurations on the palm.   Four primary types of whorls, loops, tented arches, and plain arches and a series of derived varieties have been distinguished there (Cummins and Midlo, 1961), being likely to be "quasi-continuous" variants as finger pattern types. There have been some studies to analyze their heredity and to indicate the genetic control of the presence or absence of the patterns (Weninger, 1947; Holt, 1975; Pons, 1979; Sognier *et al.*, 1979; Floris and Mameli, 1981), but little conclusive information has been obtained regarding the exact modes of inheritance and the degree of genetic determination.   This lack of conclusive information may be due to the scarcity of pedigree data to examine intensively.

This paper is concerned with an analysis of the segregation of hypothenar true

patterns in 349 Japanese families and an estimation of the heritability, for getting further towards an answer to the question of the relative importance of heredity and intrauterine conditions in palmar dermatoglyphic patternings.

MATERIALS AND METHODS

The present materials were derived from 349 Japanese families, which were a part of Furuhata's dermatoglyphic collection in the possession of Department of Forensic Medicine, Tokyo Medical and Dental University. There were 650 children falling into 340 sibships with both parents available and 9 sibships without one or both parents. The mean size of sibship was 1.9 and its distribution is given in Table 1. Since the general Japanese samples (Kasai, 1951; Matsui, 1978) showed no sex difference in true pattern frequencies, sex was disregarded for securing a sufficient sample size.

For each palm, the pattern type on the hypothenar area was formulated according to the description of Cummins and Midlo (1961). Materials available for only one hand were not used in the analysis.

The pattern type frequencies (per palm) used here are given in Table 2, where the combinations of true patterns and arches were assigned to the corresponding true pattern and vestiges were incorporated in open/arches. No significant heterogeneity in pattern distribution was found between parents and children. Further, an appreciable deviation was not noticed in pattern type frequencies when compared the present sample with the general Japanese samples (Kasai, 1951; Matsui, 1978).

The calculation of heritability was made by Method 2 in Falconer (1965), using both the regression of parents on their children and that of individuals on their sibs. The Falconer method estimates the heritability of liability to a multifactorial all-or-none trait based on converting the information contained in the incidences into a correlation estimate between relatives. In applying this method, it is assumed that

Table 1. Composition of families in sample.

No. of sibs	No. of families	No. of children	No. of sib pairs
1	175	175	
2	101	202	101
3	40	120	120
4	20	80	120
5	8	40	80
6	3	18	45
7	1	7	21
8	1	8	28
Total	349	650	515

Table 2. Frequencies of pattern types per hand in present sample.

Pattern type	Parents		Children		Parents + Children	
	n	%	n	%	n	%
Open/Arches (A)	1,014	75.4	933	76.6	1,947	76.0
Tented arches (T)	1	0.1	2	0.2	3	0.1
Single loops (Lʳ)	289	21.5	235	19.3	524	20.5
(Lᵘ)	25	1.9	31	2.6	56	2.2
(Lᶜ)	5	0.4	4	0.3	9	0.4
Double loops (Lʳ/Lᵘ)	4	0.3	4	0.3	8	0.3
Whorls (W+S)	6	0.5	9	0.7	15	0.6
Total number of palms	1,344		1,218		2,562	

there is an underlying variable, or liability, normally distributed, determined by multiple environmental and genetic factors with additive effects, and immediately related to the causation of hypothenar patterns, and that, on the scale of liability, there is a point above which the true patterns are present and below which they are absent, or threshold.

RESULTS

Detailed data on the segregation of hypothenar patterns in the 336 families available for the analysis are presented in Table 3. Further, the combined incidence of all true patterns in the children of different mating types is summarized from Table 3 in Table 4, where the families are arranged in decreasing order of the number of true patterns, *i.e.*, the degree of affectedness, observed in the parents.

It should be noted that a considerable number of children with true patterns on one or both hands were born from quite unaffected parents and that both parents with true patterns could produce children having none. This suggests that hypothenar true patterns are not inherited by a single autosomal dominant or recessive gene with a nearly complete penetrance.

It is obvious that the proportion of affected children increases progressively as their parents have more true patterns, indicating that there is a clear correlation between parents and their children regarding the occurrence of hypothenar true patterns. Only one parental combination disturbed the regularity of the increase in children; in the $-- \times$ PP mating type, the number of children with true patterns was unexpectedly high. This disturbance should be noted because of consistency with the results of Weninger (1965) and Morgan *et al.* (1978). The evaluation is open to examination in the future.

Further, the proportion of the PP children was highest in the PP × PP combination, lowest in the $-- \times --$ one, and midway in the other parental combinations,

Table 3. Incidences of hypothenar patterns in children from different mating types (Sex and laterality not distinguished).

Parents		Children						
Crosses	N	n	AA	AL^r	AL^u	L^rL^r	L^rW	Other patterns
$AA \times AA$	146	243	192	31	10	6	0	AW, AT^r, L^rL^u, L^uL^u
$AA \times AL^r$	62	114	87	13	2	11	0	AA^r
$AA \times L^rL^r$	69	136	69	32	4	24	3	AL^c, L^rT^r, L^rL^r/L^u, L^uW
$AA \times AL^u$	10	16	10	3	2	0	0	L^uW
$AA \times L^uL^u$	4	7	3	0	1	1	0	L^uL^u, L^uA^r
$AA \times L^rL^u$	2	2	2	0	0	0	0	
$AA \times AL^c$	4	5	5	0	0	0	0	
$AA \times AA^r$	1	3	1	1	0	1	0	
$AA \times AL^r/L^u$	1	1	0	0	0	1	0	
$AA \times L^rL^r/L^u$	1	1	0	1	0	0	0	
$AA \times WL^r$	3	3	1	1	0	1	0	
$AA \times WL^r/L^u$	1	4	1	1	0	2	0	
$AL^r \times AL^r$	6	13	7	2	0	3	0	L^uL^u
$AL^r \times L^rL^r$	8	12	6	4	0	2	0	
$AL^r \times AL^u$	1	3	1	0	1	0	0	L^rL^u
$AL^r \times AW$	1	1	1	0	0	0	0	
$AL^r \times AA^r$	2	8	5	1	0	1	0	AA^r
$L^rL^r \times L^rL^r$	7	21	5	5	0	8	1	AA^r, AL^r/L^u
$L^rL^r \times AL^u$	2	7	4	1	0	1	0	AL^c
$L^rL^r \times L^rL^u$	1	2	0	1	0	1	0	
$L^rL^r \times AL^r/L^u$	1	2	1	0	0	0	0	L^cL^r/L^u
$L^rL^r \times AL^c$	1	1	0	0	0	0	1	
$L^rL^r \times AT^r$	1	3	1	0	0	1	0	L^rL^c
$L^rL^r \times L^uW$	1	1	0	0	0	1	0	
Total	336	609	402	97	20	64	5	AL^c, 2; AW, 1; AT^r, 1; AA^r, 3; AL^r/L^u, 1; L^rL^u, 2; L^rL^c, 1; L^rT^r, 1; L^rL^r/L^u, 1; L^uL^u, 3; L^uW, 2; L^uA^r,1; L^cL^r/L^u, 1

suggesting that individuals with bilateral manifestation have more genetic potential for true patterns than those with unilateral manifestation.

These results may be more consistent with the hypothesis that hypothenar true patterns are caused by a multifactorial rather than a simple genetic system. Moreover, the model assuming two thresholds, *i.e.*, unilateral and bilateral ones, and three phenotypic classes, *i.e.*, absent, present on only one palm, and present on both palms, may be more appropriate to analyzing the inheritance of hypothenar patterns than

Table 4. Incidences of true patterns in children from different parental combinations (Sex and laterality were not distinguished).

Parents			Children				PP/(-P+PP)
Crosses	N	n	---	-P	PP	-P and PP	
--×--	147	246	193 (78.5%)	44 (17.8%)	9 (3.7%)	53 (21.5%)	0.170
--×-P	79	144	109 (75.7%)	21 (14.6%)	14 (9.7%)	35 (24.3%)	0.400
--×PP	80	153	76 (49.7%)	42 (27.5%)	35 (22.9%)	77 (50.3%)	0.455
-P×-P	8	17	9 (52.9%)	3 (17.6%)	5 (29.4%)	8 (47.1%)	0.625
-P×PP	13	25	12 (48.0%)	6 (24.0%)	7 (28.0%)	13 (52.0%)	0.538
PP×PP	9	24	6 (25.0%)	7 (29.2%)	11 (45.8%)	18 (75.0%)	0.611
Total	336	609	405 (66.5%)	123 (20.4%)	81 (13.1%)	204 (33.5%)	0.397

Note, P represents the presence of some ture pattern on the hand and – the absence.

Table 5. Genetic interrelationship among pattern types.

Parents	Radial loop (L^r)		Ulnar loop (L^u)		Whorl and double loop	
	n	Incidence among their children	n	Incidence among their children	n	Incidence among their children
1. With the pattern type on at least one hand	194	160/385=41.82%	25	9/46 =19.56%	8	1/12 =8.33%
2. With the other true patterns but without the pattern type	25	11/44 =25.00%	194	15/383= 3.92%	211	13/417=3.12%
3. Without any true pattern	453	177/789=22.56%	453	38/789= 4.81%	453	8/789=1.01%
Total	672	348/1,218=28.57%	672	62/1,218= 5.09%	672	22/1,218=1.81%

a simple multifactorial model which assumes one threshold and two phenotypic classes, *i.e.*, absent and present on at least one palm.

According to the two threshold model, it is expected from the property of the normal distribution of the underlying variable that, as the overall true pattern incidence (−P+PP) increases, the proportion of bilateral occurrence among the affected individuals will also increase, as pointed out by Ossenberg (1981). In actuality, the ratio of the PP children to the −P and PP ones gradually increases from the −−×−− combination to the PP×PP one, although the −P×−P combination displays a disturbance the cause of which is probably ascribed to a sampling variance due to the small number of children in that combination.

Next, the interrelationship of pattern types was examined with regard to pattern inheritance. The 672 parents were divided into three groups according to the absence or presence of a given pattern type: 1) Those with the pattern type on one or both hands; 2) those with the other pattern types but without the pattern type; and 3) those without any true pattern on both hands. In each group, the incidence of children with the same pattern type on at least one hand was examined, then all children were counted twice. This analysis was impossible for carpal loops (L^c) and tented arches (T) because of their extremely low incidences. As regards L^c, however, it should be noted that all children with L^c came from the crosses at least one partner of which had bilaterally L^r (Table 3). Here, W (including S) and L^r/L^u were treated together for the same reason as for the finger pattern classification.

The results are presented in Table 5, where it is shown that, for example, among 385 children coming from 194 parents with L^r, 160 (41.82%) manifested L^r on one or both hands. Children with L^r occurred at a significantly higher rate from parents with L^r than from parents without any true pattern ($\chi^2=24.42$, p<0.001), whereas there was no significant increase in the occurrence of children with L^r among parents with the other true patterns. The situation was also the same for L^u; children with L^u occurred at a significantly higher rate from parents with L^u ($\chi^2=14.44$, p<0.001), whereas they occurred at, though not significantly, a rather lower rate from parents with the other true patterns (most of which were L^r) than from parents without any true pattern. For W and L^r/L^u, however, a significantly increased occurrence of children with W or L^r/L^u was found among parents with the other true patterns as compared with those among parents without any true pattern ($\chi^2=6.66$, p<0.05), although a significance test was not impossible between the children of parents with W or L^r/L^u and those of parents without any true pattern.

These results suggest that L^r and L^u are inherited independently, that is, the corresponding genetic bases are different from each other. For W and L^r/L^u and perhaps also for L^c, however, there remains a possibility of expressivity, that is, the genes corresponding to L^r may be related also to their manifestation. Actually, as in the case of L^c, most of the children with W or L^r/L^u (9 out of 11 children) had at least one parent with bilateral L^r (Table 3).

In calculating the heritability of liability to a given trait, Method 2 in Falconer

Table 6. Heritability of liability to hypothenar pattern variations.

A. Based on parent-child data

Affectibility	Incidence among the children of affected parents	Incidence among the children of unaffected parents	Significance of difference (χ^2)	Heritability of liability (%)
1. Unilateral and bilateral manifestation of radial loop	160/385=41.56%	188/833 =22.57%	22.78 (p<0.001)	63.6± 9.0
2. Unilateral and bilateral manifestation of ulnar loop	9/46 =19.57%	53/1,172= 4.52%	16.16 (p<0.001)	75.8±19.8
3. Bilateral manifestation of radial loop	47/207=22.71%	85/1,011= 8.41%	27.12 (p<0.001)	62.8±10.7

B. Based on sib-sib data

Affectibility	Incidence among the sibs of affected individuals	Incidence among the sibs of unaffected individuals	Significance of difference (χ^2)	Heritability of liability (%)
1. Unilateral and bilateral manifestation of radial loop	126/284=44.37%	158/728=21.70%	27.33 (p<0.001)	74.0±10.9
2. Unilateral and bilateral manifestation of ulnar loop	8/42 =19.05%	34/970= 3.51%	19.69 (p<0.001)	81.8±20.0
3. Bilateral manifestation of radial loop	42/129=32.56%	87/883= 9.85%	35.39 (p<0.001)	85.7±12.6

(1965) needs both trait incidences among the relatives of controls and among those of the individuals affected by the trait. These incidences for hypothenar pattern types (obtained from Table 3) and the resultant heritability values are shown in Table 6A and B for parent-child data and for sib-sib data, respectively. In both series of data, the heritability was calculated for three kinds of liabilities to; 1) the manifestation of radial loops on at least one hand, 2) that of ulnar loops on at least one hand, and 3) only bilateral manifestation of radial loops. It was impossible to calculate the heritability of liability to bilateral manifestation of ulnar loops and to the other true patterns, since the incidences were extremely low in both parents and children.

For all categories analyzed here, the affected individuals occurred in a significantly increased frequency among the children of the affected parents (p <0.001) and among the sibs of the affected ones (p <0.001). All the six estimates of heritability were similar to one another and considerably large values, between 85.7% and and 56.6%, indicating that the major part of hypothenar variations depends on a genetic component.

Further, for all the categories, the estimate of heritability was somewhat larger

from the sib-sib data than from the parent-child data. This increment in the estimate from the sib-sib data may be mainly due to a sibship correlation caused by more common intrauterine factors between sibs than between parent and child. Therefore, the heritability estimate based on the sib-sib data is possibly overestimated and that based on the parent-child data is more appropriate to assessing the amount of genetic influence.

Accordingly, it is concluded that hypothenar patterns, especially radial loops and ulnar loops, are controlled by genetic factors in nearly 60–70% of their genesis.

DISCUSSION

Various investigators have produced evidence clearly indicating the presence of genetic factors in hypothenar patternings (summarized in Vrydagh-Laoureux, 1971). With regard to their mode of inheritance, however, there have been some varying suggestions; for example, Weninger (1947) and Pons (1979) assume that hypothenar pattern variations are too complex to be explained by a single-factor inheritance, while Loesch (1971, 1974) assumes radial loops to be determined mainly by a single recessive gene and Holt (1975) and Floris and Mameli (1981) assume radial arches to be determined probably by a recessive gene as well. The results obtained here may be interpreted as agreeing with Weninger's assumption and as giving a counter basis for the monogenetic hypothesis of hypothenar patternings.

When compared with the heritability estimate of hypothenar patterns obtained by Loesch (1971, 1974), the present one is nearly equivalent to her estimate for radial loops (50–78%) but much higher than her estimate for ulnar loops (28–30%). It is impossible to determine the cause for this partial discrepancy. Further, when compared with the heritability estimate of a missing c triradius obtained by Katayama (1980), the present estimate of hypothenar patterns is smaller, suggesting that the degree of genetic influence differs from one area to another on the palm.

Recently, Ossenberg (1981) has suggested that, in such bilateral nonmetric skeletal traits as mylohyoid bridging and third molar suppression, bilaterally affected individuals have more genetic potential for the traits than unilaterally affected ones, thus more weight should be assigned to bilateral than to unilateral cases in population studies. The present study suggests from the segregation data on hypothenar patterns that the situation may be the same also in bilateral nonmetric dermatoglyphic traits. If so, Kloepfer and Cummins's method which assesses the amount of genetic influence using the "penetrance" concept (Kloepfer and Cummins, 1963) is clearly meaningless. They obtained the "penetrance" values by dividing the number of individuals with the same variants on both hands by the number of individuals with the variants on either one palm or both, on the basis of assuming that bilaterally affected individuals and unilaterally affected ones have the same genetic potential for the variants and that the differentiation is brought about merely by reduced penetrance. Therefore, the "penetrance" values for the missing c triradius (about 30%)

by Kloepfer (1977) and for the hypothenar whorls (33.3%) by Sognier *et al.* (1979), which were based on a monogenetic hypothesis, are likely to be not appropriate at all.

Some population studies have shown a striking racial variation not only in the frequency of the combined true patterns but also in the relative frequency of radial loops to ulnar loops on the hypothenar area; in Negroids and Caucasoids, hypothenar loops are for the most part of the radial type (Plato and Wereterecki, 1972), whereas, in New Guinea and other Oceanic groups, ulnar loops preponderate (Plato *et al.*, 1972). Such evidence supports the present result that a different series of genes correspond to the manifestation of radial loops and ulnar loops on the hypothenar area.

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