DERMATOGLYPHIC STUDIES IN PATIENTS WITH COOLEY'S ANEMIA AND COOLEY'S TRAIT

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The dermatoglyphic analysis of a group of Italian patients with Cooley's anemia and Cooley's trait shows Cooley's anemia patients to undergo a significant increase of loops on fingers and a corresponding decrease of whorls. The total loops frequency correlates negatively with the ridge distribution per loop and the total ridge count is significantly decreased. The atd angles are larger. Minor changes in the distribution of patterns in the thenar and hypothenar areas are also observed. In the Cooley's trait patients, loops on fingers are more frequent than in controls, but no difference with Cooley's anemia series is observed. The above findings suggest that the gene(s) controlling loops formation in linkage disequilibrium with thalassemia mutant may have been favourably selected on the "thalassemia chromosome" in the malaria environment.

The epidermal ridge configurations are of value in diagnosing a variety of abnormalities, particularly those associated with chromosome aberrations.

Abnormal dermatoglyphic patterns have also been reported in other genetically determined disorders, in patients with certain congenital malformations (review in Dallapiccola 1968, Holt 1968), and in subjects exposed to enviromental factors in the fetus, such as rubella virus (Achs et al. 1966, Alter and Schulenberg 1966, Purvis-Smith and Menser 1968), or thalidomide poisoning during the first trimester of pregnancy (Pfeiffer and Berghe 1964).

Also single gene disorders have been studied and this approach has been rewarding in some cases (Anonymous 1972). However, further investigations are needed. The studies carried out in phenylketonuria (Alter 1967), Wilson's disease (David 1971, David and Ajdukiewicz 1972), and tuberous sclerosis (David 1972), have failed to reveal any significant dermatoglyphic changes. The changes observed in anonychia (Penrose 1968) and in Holt-Oram syndrome (Rosner and Aberfeld 1970) are clearly secondary results of the genes causing the nail abnormality and the hand deformity, respectively. The claim that patients with Huntington's chorea have minor dermatoglyphic differences from control groups (Barbeau et al. 1965) requires confirmation.

The preliminary results of an investigation carried out in a group of patients with Cooley's anemia have been reported by one of us (Dallapiccola 1968, 1972). It was concluded that no changes of diagnostic value were observable. However, in Cooley's patients the total ridge count (TRC) was significantly decreased, maximal atd angles were increased, and so were the frequency of accessory triradii in the fourth interdigital areas.

Rosner and Spriggs (1968) in another study on a group of patients with Cooley's anemia

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and with Cooley's trait found significant changes both on fingertips and palm patterns. They observed an increased frequency of whorls and a reduction of ulnar loops on fingertips, resulting in an increase of TRC. The mean atd angles were significantly larger only in Cooley's group. It was suggested that " the genetic defect in Cooley's anemia... may well be responsible either directly or indirectly for the dermatoglyphic abnormalities found in patients with this disease. "

In two subsequent investigations on the palmar dermatoglyphic patterns of individuals with Cooley's anemia and Cooley's trait, Maxia and Floris (1970, 1972) found larger atd angles and a decrease of the index for interdigital patterns in Cooley's patients.

The present dermatoglyphic investigation, which concerns a larger group of subjects with thalassemia was performed for three reasons. First, the discrepancies in the results obtained in previous studies require clarification and the assumed dermatoglyphic differences need confirmation. Second, significant dermatoglyphic abnormalities, if found, might be useful diagnostically. Third, thalassemia is a single gene disorder and fulfils some important requirement for this type of study. It is polymorphic in some populations, so that it is relatively easy to obtain a large number of test cases, together with control specimens, belonging to the same populations. In addition, the healthy carriers (Cooley's trait) can be identified, and the study of heterozygotes becomes possible.

MATERIALS AND METHODS

All the subjects studied lived in the area of the Po River delta, in the province of Ferrara, Italy. The sample consists of 127 patients with Cooley's anemia, 116 persons with Cooley's trait and 205 unrelated normal controls of the same age group. Both sexes are represented in similar proportions. All the individuals studied during this investigation belong to indigenous families who are natives of the Ferrara county.

The diagnostic criteria for every case were both clinical an haemathological, including RBC and MCV studies, fragility tests, hemoglobin electrophoresis.

The prints were recorded on paper, using black fingerprint ink. Plain and rolled impressions were obtained from all fingers, together with at least three sets of palm prints. The following features were analyzed: fingerprint patterns, total finger ridge count (TRC), palmar flexion creases, a-b ridge count, patterns in the hypothenar, thenar and interdigital areas and atd angles.

RESULTS

Fingerprint Patterns. The percentage frequencies of fingerprint pattern types found in patients with Cooley's trait and controls are shown in Table 1 for males and in Table 2 for females.

The overall incidence of ulnar loops was significantly increased both in Cooley's anemia males and females as compared with controls ($P < 0.01$ and $P < 0.05$, respectively). A significant increase in the percentage of loops was also observed in Cooley's trait females ($P < < 0.0001$). No significant difference was detected in Cooley's trait males ($P < 0.5$), but this result may be misrepresented by the smallness of the sample examined. The difference observed between Cooley's anemia patients and Cooley's trait persons are not significant.

Considering the fingers separately, ulnar loops are more frequent than in controls only on the fourth finger of the right hand in Cooley's trait females. The difference is significant ($0.01 < P < 0.025$).

When the data concerning the frequency of loops are considered for total fingers, adding

TABLE 1
PERCENTAGE FREQUENCIES OF FINGERPRINT PATTERNS (Males)

Subjects	Left Hand					Right Hand					Frequency on all fingers
	V	IV	III	II	I	I	II	III	IV	V	
Ulnar Loops											
Cooley's anemia (N = 63)	85.7	66.6	66.6	41.2	58.7	57.1	26.9	73.0	55.6	82.6	61.4
Cooley's trait (N = 16)	81.2	68.7	62.5	50.0	56.2	18.7	25.0	62.5	62.5	81.2	56.9
Controls (N = 100)	77.0	53.0	74.0	31.0	55.0	48.0	31.0	77.0	45.0	73.0	56.5
Radial Loops											
Cooley's anemia (N = 63)	0.0	0.0	0.0	15.0	0.0	0.0	25.4	0.0	1.6	0.0	4.6
Cooley's trait (N = 16)	0.0	0.0	6.2	6.2	0.0	0.0	37.5	0.0	0.0	0.0	5.0
Controls (N = 100)	0.0	0.0	1.0	14.0	0.0	0.0	15.0	0.0	0.0	0.0	3.0
Whorls											
Cooley's anemia (N = 63)	14.2	30.1	20.6	30.2	38.1	41.3	33.3	20.6	41.3	15.9	28.6
Cooley's trait (N = 16)	18.8	31.3	12.5	25.0	37.5	81.2	37.5	31.2	37.5	18.8	33.1
Controls (N = 100)	23.0	42.0	19.0	43.0	39.0	50.0	44.0	18.0	51.0	21.0	35.0
Arches											
Cooley's anemia (N = 63)	0.0	3.2	12.7	9.5	3.2	1.6	14.3	6.3	1.6	1.6	5.4
Cooley's trait (N = 16)	0.0	0.0	18.8	18.7	6.3	0.0	0.0	6.3	0.0	0.0	5.0
Controls (N = 100)	3.0	4.0	8.0	11.0	3.0	3.0	9.0	6.0	3.0	5.0	5.0

males and females together, a significant difference is observed between patients with thalassemia (Cooley's anemia and Cooley's trait: 64.31% and 65.39%, respectively) and normal controls (57.86%, $P < 0.0001$).

There are fewer whorls (25.04% and 28.45% in Cooley's anemia patients and in Cooley's trait individuals, respectively, as compared with 34.68% in controls), and, contrary to the usual tendency for the frequency of arches to increase as whorls diminish, fewer arches (6.77% and 5.54% in Cooley's patients and in Cooley's trait subjects as compared with 7.32% in controls).

Total Finger Ridge Count. Mean values and standard errors in patients and in controls are given in Table 3. Mean TRC is lower in Cooley's anemia individuals of both sexes (males $P < 0.001$; females $P < 0.01$) than the corresponding means for the control population. The mean ridge count of ulnar loops is 10.75 in Cooley's anemia males, 12.39 in Cooley's trait males, and 12.78 in control population. The mean number of ridges per loop is 10.05 in Cooley's anemia females, 10.60 in Cooley's trait females and 10.18 in control group.

TABLE 2
PERCENTAGE FREQUENCIES OF FINGERPRINT PATTERNS (Females)

Subjects	Left Hand					Right Hand					Frequency on all fingers
	V	IV	III	II	I	I	II	III	IV	V	
Ulnar Loops											
Cooley's anemia (N = 53)	84.9	54.7	66.0	34.0	60.4	58.5	30.2	60.4	60.0	79.2	58.9
Cooley's trait (N = 97)	86.6	51.5	72.2	39.2	61.8	56.7	38.1	83.5	59.8	79.4	63.0
Controls (N = 105)	71.4	47.6	60.9	31.4	50.4	48.6	35.2	76.2	41.9	77.1	54.1
Radial Loops											
Cooley's anemia (N = 53)	0.0	1.9	5.6	9.4	0.0	0.0	13.2	1.9	1.9	0.0	3.4
Cooley's trait (N = 97)	0.0	1.0	0.0	13.4	1.0	0.0	14.4	0.0	1.0	0.0	3.1
Controls (N = 105)	0.9	0.0	1.0	13.3	1.0	0.0	16.9	0.0	1.0	0.0	3.3
Whorls											
Cooley's anemia (N = 53)	13.2	32.0	11.3	33.9	32.1	35.8	43.4	24.5	33.9	18.9	27.9
Cooley's trait (N = 97)	11.3	42.3	17.5	35.1	34.0	40.2	37.1	12.4	38.2	17.5	28.5
Controls (N = 105)	23.9	47.7	23.9	28.1	43.8	48.6	38.1	16.2	53.3	22.0	35.5
Arches											
Cooley's anemia (N = 53)	1.9	11.3	17.0	22.7	7.5	5.6	13.2	13.2	3.8	1.9	9.8
Cooley's trait (N = 97)	1.1	5.2	10.3	12.4	3.1	3.1	10.3	4.1	1.0	3.1	5.4
Controls (N = 105)	3.9	4.8	14.3	17.1	4.1	2.9	10.5	7.6	3.9	0.9	7.1

TABLE 3
TOTAL FINGER RIDGE COUNT (TRC)

Subjects	N	Mean TRC	*SE*
Males			
Cooley's anemia	63	110.03	4.83
Cooley's trait	16	134.68	8.20
Controls	100	130.00	7.82
Females			
Cooley's anemia	53	102.07	5.86
Cooley's trait	97	116.89	4.44
Controls	108	120.19	4.34

TABLE 4
a-b RIDGE COUNT (Sum for both hands)

Subjects	N	Mean a-b ridge count	*SE*
Males			
Cooley's anemia	69	75.46	1.18
Cooley's trait	16	77.25	2.78
Controls	100	77.32	1.57
Females			
Cooley's anemia	57	73.15	1.38
Cooley's trait	100	74.18	1.24
Controls	107	73.19	3.76

TABLE 5

PERCENTAGE FREQUENCIES OF PATTERNS IN THE PALM INTERDIGITAL AREAS

Subjects	II interdigital area			III interdigital area				IV interdigital area		
	None	Loop	Loop with accessory triradius	None	Loop	Whorl	Loop with accessory triradius	None	Loop	Loop with accessory triradius
RIGHT PALM										
Males										
Cooley's anemia (N = 69)	89.8	0.0	10.2	39.1	58.0	0.0	2.9	52.2	31.9	16.0
Cooley's trait (N = 16)	87.5	0.0	12.5	25.0	75.0	0.0	0.0	62.5	25.0	12.5
Controls (N = 16)	97.7	0.0	2.3	44.2	51.2	0.0	4.6	58.1	37.2	4.7
Females										
Cooley's anemia (N = 57)	96.5	0.0	3.5	50.9	49.1	0.0	0.0	43.9	45.6	10.5
Cooley's trait (N = 100)	98.0	0.0	2.0	51.0	48.0	0.0	1.0	46.0	46.0	8.0
Controls (N = 107)	97.2	0.9	1.9	46.8	50.5	0.9	1.9	59.8	32.7	7.5
LEFT PALM										
Males										
Cooley's anemia (N = 69)	97.1	0.0	2.9	65.2	34.8	0.0	0.0	37.7	37.7	24.6
Cooley's trait (N = 16)	100.0	0.0	0.0	62.5	37.5	0.0	0.0	43.8	43.8	12.5
Controls (N = 43)	93.0	0.0	7.0	65.1	34.9	0.0	0.0	60.5	30.2	9.3
Females										
Cooley's anemia (N = 57)	100.0	0.0	0.0	75.4	24.6	0.0	0.0	42.1	43.9	14.0
Cooley's trait (N = 100)	100.0	0.0	0.0	67.0	33.0	0.0	0.0	39.0	51.0	10.0
Controls (N = 107)	97.2	0.9	1.9	75.7	23.3	0.0	0.9	40.2	52.3	17.4

Whorls are also smaller in Cooley's anemia patients than in controls, the mean number of ridges being 14.8 and 15.4, respectively. Therefore, in Cooley's anemia males, both the relative increase in the frequency of loops and the prevalence of small sized patterns are responsible for the lowering of the total ridge count. In Cooley's anemia females the lower count is mainly due to the presence of small loops.

Females with Cooley's trait have a mean TRC intermediate between the values obtained in Cooley's anemia patients and normal controls (116.89 ± 4.44). However, this difference is not significant. The mean TRC determined from 16 Cooley's trait males is similar to that obtained in controls.

Data from males and females with Cooley's anemia, Cooley's trait and from controls

TABLE 6

PERCENTAGE FREQUENCIES OF PATTERNS IN THE THENAR AREA

Subjects	Patterns	Left hand only	Right hand only	Both hands	Neither hand
Males					
Cooley's anemia	Loops	2.9	1.4	1.4	57.1
(N = 69)	Whorls	4.3	1.4	—	95.6
	Vestiges	2.9	3.8	—	89.8
Cooley's trait	Loops	12.5	—	—	87.5
(N=16)	Whorls	—	—	—	100.0
	Vestiges	18.7	—	—	81.2
Controls	Loops	13.9	4.6	4.6	86.0
(N=43)	Whorls	—	—	—	100.0
	Vestiges	—	4.6	—	95.3
Females					
Cooley's anemia	Loops	5.8	17.5	8.7	82.4
(N = 57)	Whorls	—	—	—	100.0
	Vestiges	5.2	1.7	—	94.7
Cooley's trait	Loops	11	7	5	89.0
(N = 100)	Whorls	—	—	—	100.0
	Vestiges	3	3	2	97.0
Controls	Loops	10.3	4.6	4.6	89.7
(N = 107)	Whorls	—	—	—	100.0
	Vestiges	3.7	2.8	0.9	96.2

were pooled in each of the groups. Correlations were calculated between the increased frequency of loops and the size of loops. The correlations were negative ($r = -0.68$) in the Cooley's anemia group, vs. $r = -0.04$ in the Cooley's trait group, and $r = 0.023$ in controls.

Palmar Flexion Creases. A single transverse palmar crease was noticed on one hand in 3 patients with Cooley's anemia, in 3 subjects with Cooley's trait, and in 2 control individuals.

a-b Ridge Count. The means and standard errors for the sum of the counts on both hand are shown in Table 4. No significant differences were found in patients of either sex.

Patterns in the Interdigital Areas of Palm. Patterns in the second, third and fourth interdigital areas were classified into the following groups: (1) loops, (2) whorls, and (3) loops accompanied by an accessory triradius. The results are given in Table 5. Comparisons show no significant differences between patients and controls.

Thenar/First Interdigital Patterns. The following groups were considered: (1) loops, (2) whorls, and (3) vestiges. The percentage frequencies of patterns observed are summarized in Table 6.

TABLE 7

PERCENTAGE FREQUENCIES OF PATTERNS IN THE HYPOTHENAR AREA

Subjects	Patterns	Left hand only	Right hand only	Both hands	Neither hand
Males					
Cooley's anemia	Ulnar loops	14.5	13.0	8.7	85.5
(N = 69)	Radial loops	21.7	21.7	15.9	78.2
	Whorls	—	1.4	—	98.5
	S-shaped patterns	—	7.2	—	92.7
Cooley's trait	Ulnar loops	12.5	—	—	87.5
(N = 16)	Radial loops	25.0	18.7	6.2	75.0
	Whorls	—	—	—	100.0
	S-shaped patterns	—	—	—	100.0
Controls	Ulnar loops	16.2	9.3	9.3	83.7
(N = 43)	Radial loops	25.6	20.9	18.6	74.4
	Whorls	—	—	—	100.0
	S-shaped patterns	4.6	—	—	95.3
Females					
Cooley's anemia	Ulnar loops	10.5	8.7	8.7	89.4
(N = 57)	Radial loops	33.3	19.3	7.0	84.2
	Whorls	1.7	1.7	—	98.2
	S-shaped patterns	1.7	8.7	1.7	91.2
Cooley's trait	Ulnar loops	7.0	3.0	1.0	93.0
(N = 100)	Radial loops	13.0	20.0	11.0	80.0
	Whorls	1.0	4.0	1.0	96.0
	S-shaped patterns	4.0	4.0	1.0	96.0
Controls	Ulnar loops	9.3	9.3	5.6	90.6
(N = 107)	Radial loops	21.5	20.5	12.1	78.5
	Whorls	0.9	5.6	0.9	94.3
	S-shaped patterns	1.8	2.8	1.8	97.2

Comparisons show only minor differences between patients and controls (decreased frequency of loops on the left hand of male patients).

Hypothenar Patterns. The following groups were studied separately: (1) ulnar loops, (2) radial loops, (3) whorls, and (4) S-shaped patterns. The percentage frequencies of patterns observed are reported in Table 7. Comparisons show slight differences in Cooley's anemia patients as compared with Cooley's trait individuals and controls. In particular, there is an increase in the frequency of radial loops on the left hand of female patients and an increase on the right hand of S-shaped patterns in patients of both sexes.

Sum (righ + left) Maximal atd Angles. The means and standard errors are shown in Table 8. The atd angles are significantly larger in Cooley's anemia patients as compared with the controls of either sex ($P < 0.05$ in males; $P < 0.02$ in females).

TABLE 8

MAXIMAL atd ANGLES (Sum both hands)

Subjects	N	Mean atd angles in degrees	*SE*
Males			
Cooley's anemia	66	96.25	1.80*
Cooley's trait	16	82.20	3.16
Controls	41	85.41	2.76
Females			
Cooley's anemia	59	101.23	2.21**
Cooley's trait	95	90.05	2.62
Controls	104	93.09	1.97

* $P < 0.05$; ** $P < 0.02$

Our results indicate variations of dermatoglyphic constitution in individuals carrying in single or double dose the gene for thalassemia.

The results can be summarized as follows:

Cooley's Anemia Patients

Fingers:

Pattern-type: loops (ulnar + radial) are more frequent when compared to normal controls. A corresponding decrease of whorls is demonstrable.

The total loops' frequency correlates negatively with the ridge count distribution for loops. The total ridge count is significantly decreased when compared to either the Cooley's trait series or the controls.

Palmar Dermatoglyphics:

atd angles are larger either than the control series or the Cooley's trait series, both in males and females.

Minor changes in the distribution of patterns in thenar and hypothenar areas are observed.

Cooley's Trait Patients

Fingers:

Pattern-type: loops (ulnar + radial) are more frequent when compared to normal controls. No difference with Cooley's anemia series is observed.

Ulnar loops are more frequent than in Cooley's anemia series and in controls on the fourth finger of the right hand of females.

TRC is not significantly modified when compared to the control series.

No palmar differences are observed.

No dermatoglyphic variation in Cooley's anemia and in Cooley's trait series is " unique ". No rearrangements are observed, but only changes in the frequency of normal pattern-types or size. Therefore, due to the large overlapping with the distribution in " normal subjects ", they are of no diagnostic value.

DISCUSSION

These results are in partial agreement with those obtained in similar investigations (Dallapiccola 1968 and 1972, Maxia and Floris 1970 and 1972), mainly with regard to the atd angles, and palmar dermatoglyphic abnormalities.

However, they are in contrast with other previous report (Dallapiccola 1968 and 1972, Rosner and Spriggs 1968) as far as pattern-type and TRC are concerned. The latter authors report in Cooley's anemia and in Cooley's trait, whose origin is not stated, both an increase of either whorls or TRC.

The evaluation of the mechanism which ultimately leads to the abnormal distribution of fingertip patterns and TRC is handicapped by the lack of clear understanding either of physiology, or of the genetic control of pattern-types and ridge count determination.

The differentiation of ridges begins early in the third month of intrauterine life and is completed at the end of the fourth. Thereafter, neither the ridges nor the patterns formed by them change, as far as can be ascertained. Certainly, from birth until death, there are no changes, except in size. Ridge alignement, however, is not considered to be due to "self-limited mechanisms within the skin... the epidermis possesses the inherent capacity to develop ridges, but the dispositions of the ridges are passively determined" (Cummins and Midlo 1961). It is known that pattern types (whorls, loops, arches) are in part genetically determined. This is shown by a study of pedigrees and from work on twins. With regard to ridge counts, the extensive work by S. B. Holt (1961) has shown that the dermination of total ridge count on fingertips is genetically controlled. The total ridge count is determined by a number of genes, whose effects are additive.

One of the most interesting aspects of our results is the relationship between pattern type, mean count for individual pattern types, and total ridge count. The Cooley's trait series (single dose of gene for thalassemia) shows a significant increase of the total frequency of loops (65.39%). However, the distribution of ridge counts for loop does not significantly deviate from the normal one and the TRC is not significantly affected.

On the contrary, in Cooley's anemia series (double dose of gene for thalassemia) we find the same total loop frequency (64.31%) but the distribution of loop size is modified, with an increase of smaller loops, and the TRC is significantly affected. The frequency of loops' increase in this series appears to be mainly represented by smaller loops. No such relationship exists in the Cooley's trait series.

Two thalassemia genes have apparently lowered the loops ridge count, while when only one thalassemia gene is present an increase of loops becomes apparent. This suggests that the determination of number of ridges and pattern type are related.

Assuming that our controls have been correctly selected and therefore represent a suitable specimen of the same population where Cooley's anemia and Cooley's trait series are derived from, we must discuss the possible mechanisms by which the frequency of dermatoglyphic patterns changes in the thalassemic population.

Three possible mechanisms could be envisaged:

(1) *A pleiotropic effect of the thalassemia mutant.* Such an effect should, in our opinion, be considered very unlikely because it is known that thalassemia is a single gene mutation affecting the synthesis of hemoglobin β chains (Conconi et al. 1972). No physiological correlations between the two phenotypes are apparent.

(2) The possibility must be considered that *the dermatoglyphic abnormalities in Cooley's anemia are secondary to disturbances produced by the primary genetic condition.* However, no reports are available indicating that in Cooley's and in thalassemia patients the embryological development is affected at the moment when dermatoglyphics are fixed. However, more recently, the suggestion was forwarded that β/γ chain synthesis is correlated to crown-rump length in the fetus of 5-16 weeks of gestation (Cividalli et al. 1974).

(3) Parisi and Di Bacco (1968) have reported that digital dermatoglyphics show high degree of correlation in twins, suggesting a " practically complete genetic conditioning " of type of patterns and size. Assuming genetic linkage between thalassemia and " gene/s " important for dermatoglyphic patterns, crossing-over disequilibrium could, in fact, be responsible for the observed deviations in the thalassemia and Cooley's populations from the " loops " distribution in the nonthalassemic population of the Ferrara county. As it is known, a situation of this type leads to an increased association of the two phenotypes in the population. It could be due either to the effects of natural selection or to the population having not yet reached equilibrium. Both mechanisms could have been effective in the Ferrara region because of the indication by historical data that a limited number of generations (about 30) have been available to a favourable selection of the thalassemia chromosomes in the malaria environment. Other data are consistent with this type of mechanism.

A very similar model has been described by Siniscalco (1963) in Sardinia for two sex-linked mutants: the mediterranean variant of G6PD deficiency and the mutant for deuteranopia. Both gene frequencies correlate positively with past incidence of malaria endemicity in Sardinia. No physiological clue to a protection from malaric infection of the deuteranopia mutant is however available. When the " coupling " combination (G6PD deficiency and mutant for deuteranopia) is studied in males, it is observed that it is significantly increased over expected frequency of random association, suggesting an increased fitness to that association.

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RIASSUNTO

I Dermatoglifi in Pazienti con Morbo di Cooley e con Trait Talassemico

L'analisi dermatoglifica in un gruppo di pazienti italiani con morbo di Cooley e con trait talassemico indica, nei pazienti con morbo di Cooley, un significativo aumento delle configurazioni ad ansa sui polpastrelli ed una riduzione dei vortici. La frequenza complessiva delle anse correla negativamente con la distribuzione del numero delle creste per ansa. Il conteggio totale delle creste è ridotto in maniera significativa. Gli angoli atd sono significativamente piú ampi rispetto ai controlli ed ai soggetti con trait talassemico, in entrambi i sessi. Sono state evidenziate anche alcune differenze nella distribuzione dei disegni sul tenar e sull'ipotenar. Nei pazienti con trait talassemico la frequenza delle anse appare significativamente aumentata rispetto alla popolazione di controllo, ma non ai pazienti con morbo di Cooley. Queste osservazioni suggeriscono che il gene o i geni che controllano la formazione delle anse, in linkage disequilibrium con il mutante talassemico, siano stati selezionati sul cromosoma che porta il mutante per la talassemia nell'area malarica dalla quale provengono i pazienti esaminati.

RÉSUMÉ

Les Dermatoglyphes chez des Sujets avec Maladie de Cooley ou Trait Thalassémique

L'analyse dermatoglyphique chez des sujets italiens atteints de maladie de Cooley ou de trait thalassémique indique chez les patients de maladie de Cooley une augmentation significative de boucles et une réduction de tourbillons sur les doigts. La fréquence complexive des boucles est négativement corrélée avec la distribution du nombre de crêtes par boucle. Le nombre total des crêtes est significativement réduit. Les angles atd sont significativement plus larges par rapport aux contrôles et aux sujets avec trait thalassémique chez les deux sexes. Quelques différences dans la distribution des dessins sur les aires thénar et hypothénar ont aussi bien été remarquées. Chez les patients avec trait thalassémique la fréquence des boucles paraît significativement augmentée par rapport à la population de contrôle, mais pas par rapport aux sujets avec maladie de Cooley. Ces observations suggèrent que le gène ou les gènes qui contrôlent la formation des boucles en linkage mais pas en équilibre avec le mutant thalassémique, aient été selectionnés sur le chromosome qui porte le mutant pour la thalassémie dans la région malarique de laquelle provient le sujet.

ZUSAMMENFASSUNG

Hautleisten bei Patienten mit Cooley-Anämie oder mit Thalassämie-Trait

Bei einer Reihe italienischer Patienten mit Cooley-Anämie oder mit Thalassämie-Trait wurden Hautleistenuntersuchungen vorgenommen. Bei den Pat. mit Cooley-Anämie wurden folgende Beobachtungen gemacht: an den Fingerspitzen erheblicher Anstieg der Schleifenbildung und Verminderung der Wirbel; das Gesamtvorkommen der Schleifen steht in negativem Verhältnis zur Verteilung der Hautleistenzahl pro Schleife; die Zahl der Hautleisten insgesamt ist bedeutend vermindert; die *atd*-Winkel sind bei beiden Geschlechtern breiter als bei den Kontrollpersonen oder den Pat. mit Thalassämie-Trait; auch die Verteilung der Hautzeichnungen am Thenar und Hypothenar weisen wesentliche Unterschiede auf. Bei den Pat. mit Thalassämie-Trait scheinen die Schleifen — im Vergleich zu den Kontrollpersonen, aber nicht zu den Pat. mit Cooley-Anämie — merklich häufiger zu sein. Diese Beobachtungen lassen vermuten, dass in der Malariagegend, aus der die untersuchten Pat. stammen, das Gen oder die Gene, die in linkage aber nicht im Gleichgewicht mit der Thalassämie-Mutante die Schleifenbildung kontrollieren, eine Selektion auf dem die Thalassämie tragenden Chromosom ausüben.

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