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The connection between genetic anomalies and dermatoglyphics: insights from chromosomal, monogenic, and polygenic disorders

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Abstract

Background: Dermatoglyphics, the study of epidermal ridge patterns on fingers, palms, and soles, provides stable markers for genetic and developmental anomalies due to their formation during early fetal development. Genetic disorders, including chromosomal, monogenic, and polygenic conditions, are often linked to altered dermatoglyphic traits and increased fluctuating asymmetry (FA), indicating developmental instability.

Aims: This study investigates the relationship between genetic anomalies and dermatoglyphic traits, focusing on quantitative and discrete patterns, variability, and FA in individuals with chromosomal syndromes (Turner, Klinefelter, Down), cystic fibrosis (CF), cleft lip and/or palate (CL/P), and endometrial/cervical carcinoma, compared to healthy controls.

Methods: Dermatoglyphic data from approximately 2,450 Israeli individuals, including patients, their parents, and controls, were analyzed. Finger and palm prints were evaluated for 64 traits, including ridge counts, pattern frequencies, and FA indices. Statistical analyses included χ^2 tests, ANOVA, Kruskal-Wallis tests, Principal Component Analysis (PCA), and discriminant analysis to differentiate patient and control groups.

Results: Chromosomal syndromes exhibited the most significant dermatoglyphic deviations, with increased ulnar loops, altered ridge counts, and elevated FA (e.g., atd angle, a-b ridge breadth). CF and CL/P patients showed moderate deviations and higher FA, while carcinoma patients displayed distinct patterns and FA. Parents of affected individuals exhibited intermediate traits. PCA and discriminant analysis achieved 80–90% accuracy in distinguishing groups.

Conclusion: Genetic anomalies are associated with distinct dermatoglyphic alterations and elevated FA, reflecting disrupted developmental homeostasis. Dermatoglyphics offers potential as a non-invasive tool for early diagnosis and genetic counseling in clinical genetics.

Keywords: dermatoglyphics, genetic anomalies, fluctuating asymmetry, developmental homeostasis, chromosomal syndromes

Introduction

Dermatoglyphics, the study of epidermal ridge patterns on fingers, palms, and soles, is a valuable tool in medical and anthropological research due to its genetic basis and stability throughout life. Formed between the sixth and 24th weeks of fetal development, these patterns serve as markers of genetic and developmental anomalies.[1,2] Previous studies have identified associations between dermatoglyphic traits and specific genetic disorders, such as chromosomal syndromes (e.g., Turner, Klinefelter, Down), monogenic diseases like cystic fibrosis (CF), and polygenic conditions like cleft lip and/or palate (CL/P).[3–5] However, the systematic relationship between dermatoglyphic alterations, particularly fluctuating asymmetry (FA), and a broad spectrum of genetic anomalies, including conditions of uncertain etiology like endometrial/cervical carcinoma, remains underexplored.

The problem addressed in this study is the lack of comprehensive research integrating dermatoglyphic traits and FA across diverse genetic disorders to elucidate their role as non-invasive markers of developmental instability. While individual studies have reported dermatoglyphic deviations in specific conditions,[6–8] there is a gap in understanding how these traits and FA collectively reflect disrupted developmental homeostasis across chromosomal, monogenic, polygenic, and carcinoma-related conditions. Elevated FA, indicating random bilateral deviations, is a known measure of developmental instability,[9,10] yet its application across such a wide range of disorders is limited.

This study aims to address this gap by examining dermatoglyphic traits, variability, and FA in approximately 2,450 individuals with chromosomal syndromes, CF, CL/P, and endometrial/cervical carcinoma, compared to healthy controls. We hypothesize that these genetic anomalies are associated with distinct dermatoglyphic patterns and increased FA, reflecting underlying developmental disruptions, and that such traits can enhance non-invasive diagnostic and genetic counseling approaches.

Materials and methods

Study population

Dermatoglyphic data were collected from approximately 2,450 individuals in Israel during the 1960s–1990s, including patients with genetic disorders and healthy controls. The patient groups comprised individuals with:

Chromosomal syndromes: Turner syndrome (45,X), Klinefelter syndrome (47,XXY), Down syndrome (trisomy 21).

Monogenic disease: Cystic fibrosis (CF).

Polygenic defect: Cleft lip with or without cleft palate (CL/P) and cleft palate (CP).

Diseases of uncertain etiology: Endometrial and cervical carcinoma. Control groups consisted of healthy Israeli Jewish males and females, matched for age and ethnicity. Parents of patients with Down syndrome, CF, and CL/P were also included to assess familial dermatoglyphic patterns.

Dermatoglyphic Data Collection (see Table 1 for fill list of collected triats)

Table 1 List of the utilized traits and indices.

22 quantitative traits	**13 directional asymmetry (DAs) traits**
Finger RC, I r	DA I = Div II - Div I
Finger RC, II r	DA II = PII, rh - lh
Finger RC, III r	DA III = a-b RC, r - l
Finger RC, IV r	DA IV = hRC, rh - lh
Finger RC,V r	DA V = S^2, rh - lh
Finger RC, I l	DA VI = Div VIII - Div VII
Finger RC, II l	DA VII = atd angle, r - l
Finger RC, III l	DA VIII = ab dist., r-l
Finger RC, IV l	DA IX = ridge breadth, r-l
Finger RC,V l	DA X = fRC, Vr – Vl
Total RC (TRC)	DA XI = fRC, IVr – IVl
AbsRC	DA XII = fRC, IIIr - IIIl
PII, lh	DA XIII = fRC, IIr - IIl
PII, rh	DA XIV = fRC, Ir - Il
PII, both h	DA XV = MLI, rh - lh
a-b RC, rh	**14 Fluctuating asymmetry (flas) traits**
a-b RC, lh	FA I = [Div I - Div II]
A-line exit, l	FA II = PII, [rh - lh]
A-line exit, r	FA III = a-b, RC, [rh - lh]
D-line exit, l	FA IV = hRC, [rh - lh]
D-line exit, r	FA V = [Div V - Div IV]
MLI	FA VI = [Div VIII - Div VII]
38 traits (diversity and asymmetry):	FA VII = atd angle, [r - l]
11 Diversity traits (Div)	FA VIII = Div II – Div I
Div I = max - min fRC (lh)	FA IX = PII, rh-lh
Div II = max - min fRC (rh)	FA X = fRC, [Vr - Vl]
Div III = max - min fRC (both h)	FA XI = fRC, [IVr - IVl]
Div IV = S^2 for lh, (or S^2L)	FA XII = fRC, [IIIr - IIIl]
Div V = S2 for rh, (or S2R)	FA XIII = fRC, [IIr - IIl]
Div VI = S2 (both h)	FA XIV = fRC, [Ir - Il]
Div VII = IIDL (for lh)	FA XV = MLI, [rh - lh]
Div VIII = IIDR (for rh)	FA XVI = A1, asymmetry index
Div IX = $S\sqrt{10}$, (both h)	**Numbering of the traits remains as in our other publications, for simplification of comparison with our previous data**
Div X = $S\sqrt{5}$, (both h)	
Div XI = Shannon's index	

Abbreviations: RC, ridge count; r, right; l, left; h, hand; PII, Pattern Intensity Index, MLI, main line index, Div I to Div XI, indices of intra-individual diversity of finger ridge counts; DA I to DA XV, indices of directional asymmetryFA I to FA XVI, indices of fluctuating asymmetry.

Finger and palm prints were obtained using ink pads (Lamedco Inc., Knoxville, Tennessee) and paper (Promedica Co., Tel Aviv). Prints were analyzed following established protocols (Cummins and Midlo, 1961; Penrose, 1968), identifying patterns (e.g., loops, whorls, arches), ridge counts, and palmar measurements (e.g., a-b distance, atd angle). A total of 64 dermatoglyphic traits were examined, including:

Quantitative traits: Finger ridge counts (FRC), total ridge count (TRC), absolute ridge count (AbsRC), a-b distance, ridge breadth, maximum atd angle.

Discrete traits: Frequencies of finger pattern types, pattern combinations, palmar patterns, hypothenar triradius patterns, Sydney and Simian lines, and axial triradius positions.

Variability and asymmetry indices: Shannon index for pattern diversity, directional asymmetry (DA), and fluctuating asymmetry (FA) indices, calculated as per Holt (1968)[11] and Jantz (1975)..[12]

Statistical analysis

Differences in discrete traits were assessed using χ^2 tests or t-tests (Sokal and Rohlf, 1981). Quantitative traits and DA were analyzed using one-way ANOVA, with significance set at $P<0.05$. FA and variability indices were evaluated using the Kruskal-Wallis test with Bonferroni correction ($P<0.001$). Multivariate analyses included:

Principal Component Analysis (PCA): Conducted on 22 quantitative traits and asymmetry indices using BMDP software[13].

Discriminant Analysis: Performed using SPSS to classify individuals into patient or control groups based on dermatoglyphic traits.[14] Data were processed at Tel Aviv University's central computer.

Formulae for some indices of dermatoglyphic diversity and asymmetry: The directional asymmetry (DA) was computed using the following equation: $DAij = X_{iR} - X_{iL}$.

The fluctuating asymmetry (FA) was computed by using the absolute differences between the bilateral measurements. The distributions of the non-absolute differences for each individual were corrected (Livshits et al., 1988)[15] to avoid additional influences (scaling effects) like size of the trait or directional asymmetry, yielding the following equation for computing FA:

$$FA_{ij} = \left| \left(X_{iR} - X_{iL} \right) - 1/n \sum_{i=1}^{n} \left[\left(X_{iR} - X_{iL} \right) \right] \right|$$

Where, xi = trait (x) of individual (i); R, L = right and left, n = size of the sample, and FA_{ij} is the value of FA of trait (j) in the I th individual.

Citation: Kobyliansky E, Kalichman L. The connection between genetic anomalies and dermatoglyphics: insights from chromosomal, monogenic, and polygenic disorders. *J His Arch & Anthropol Sci.* 2025;10(2):49–56. DOI: 10.15406/jhaas.2025.10.00324

Div I, Div II, Div III. Maximal minus minimal finger ridge counts in the five left (Div I), five right (Div II), or in the ten finger ridge counts (Div III). Div IV, Div V = $\sum_{i=1}^{5} q_i^2 - Q^2 / 5$, for the left (Div IV, S^2L), or right fingers (Div V, S^2R); Div VI, $S^2 = \sum_{i=1}^{10} q_i^2 - Q^2 / 10$; Div VII, Div VIII = $\sqrt{\sum_{i=1}^{5} q_i^2 - Q^2 / 5}$, for the left (Div VII, IIDL), or right finger (Div VIII, IIDR); Div IX, $S\sqrt{10} = \sqrt{\sum_{i=1}^{10} (q_i^2 - Q^2 / 10) / 10}$; Div X, X, $S\sqrt{5} = \sqrt{\sum_{i=1}^{5} (k_i^2 - Q^2 / 5) / 5}$;

In these formulae, q_i is the ridge count for the i[th] finger, Q is the sum of the five finger ridge counts of a hand (Div IV, V, VII, VIII) or of all the ten fingers (Div VI, IX, X), and k is the sum of ridge counts of the i[th] pairs of homologous right and left fingers.

Div.XI. Shannon's index, $D = -\sum_{i=1}^{4} P_i \log P_i$ where P_i is the frequency of each of the four basic finger pattern types on the ten fingers; Abs XVI, AI = AI $= \sqrt{\sum_{i=1}^{5} (R_i - L_i)^2}$, where R_i and L_i are the ridge counts for the i[th] finger of the right and left hand.

Computation of the directional asymmetry (DA) was affected by the following equation:

$$DA_{IJ}=(X_{IR}-X_{IL})/[0.5*(X_{IR}+X_{IL}]$$

The fluctuating asymmetry (FA) was computed using the absolute difference between the bilateral measurements.

$$FA_{IJ} = 100 \mid (X_{IR} - X_{IL}) / [0.5 * (X_{IR} + X_{IL})] - 1/n \sum_{i=1}^{n} [(X_{IR} - X_{IL}) / 0.5 (X_{IR} + X_{IL})] \mid$$

Where Xi = trait (x) of individual (i); R,L = right and left, n = size of the sample, and FAij is the value of FA_{IJ} of trait(j) in the i-th individual.

Results

Dermatoglyphic traits in chromosomal syndromes

Individuals with Turner, Klinefelter, and Down syndromes exhibited significant dermatoglyphic deviations compared to controls (P<0.05) (Tables 2-5). Key findings include:

Table 2 Summary data on significant differences between patients with Down syndrome, their parents, and healthy control groups. Finger and palm patterns

Variables	Fathers /Ctrl.M	Down M. /Ctrl.M	Down M. /Fathers	Mothers /CtrLF	Down F. /Ctrl.F	DownF. /Mothers
Ulnar Loops (UL)	-	*	*	-	*	*
Whorls (W)	-	*	*	-	*	*
RL on finger IV	-	*	*	-	*	*
RL on finger V	-	*	*	-	*	*
U-U on fing. II-II	-	*	*	-	*	*
Same pat. on 10 fing.	-	*	*	-	*	*
UL+RL+W on 10 fing.	-	*	*	-	*	-
PII lh	-	***	***	-	***	***
PII rh	*	***	***	-	***	***
PII both hands	*	***	***	-	***	***
RCs of Radial loops	*	-	*	-	*	*
RCs of Whorls	*	*	*	-	*	*
FRC I-r	-	*	-	-	-	-
FRC IV-r	-	***	***	-	***	***
FRC V-r	-	***	***	-	***	***
FRC I-l	-	*	*	-	*	-
FRC IV-l	-	***	***	-	***	***
FRC V-l	-	***	***	-	***	***
TRC	-	***	*	-	*	*
Absolute RC (ARC)	-	***	***	-	***	***
Patterns Hypoth.	-	*	*	-	*	*
Patterns Thenar	-	*	-	*	*	-
Patterns ID. III	-	*	*	-	*	*
Patterns ID. IV	-	*	*	-	*	*
Indiv. with d'	*	*	-	-	*	*
a-b RC rh	-_	***	***	-	***	***

Citation: Kobyliansky E, Kalichman L. The connection between genetic anomalies and dermatoglyphics: insights from chromosomal, monogenic, and polygenic disorders. *J His Arch & Anthropol Sci.* 2025;10(2):49–56. DOI: 10.15406/jhaas.2025.10.00324

Table 2 Continued....

a-b RC lh	-	***	***	-	***	***
atd angle	-	*	*	-	*	*
t	-_	*	*	-	*	*
t	-_	*	*	-	*	*
t"+t	*	*	*	-	*	*
Simian line	*	*	*	-	*	*
A line exit lh	***	***	***	***	***	***
A line exit rh	***	***	***	***	***	***
D line exit lh	-	***	***	-	***	***
D line exit rh	-	***	***	*	**	***
MLI	*	***	***	*	***	***

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001. *** -MP – interdigital zones.

Table 3 Summary data on significant differences between patients with Down syndrome, their parents, and healthy control groups. Diversity, fluctuating, and directional asymmetry.

Variables	Fathers /Ctrl.M	Down M. /Ctrl.M	Down M. /Fathers	Mothers /Ctrl F	Down F. /Ctrl F	Down F /Mothers
DivII	-	*	-	-	-	--
Div III	-	*	-	-	-	-.
DivV	-	*	-	-	-	-
DivVI	-	*	-	-	-	-
DivVIII	-	*	-	-	-	-
Div IX	-	*	-	-	-	-
Div XI	-	**	*	-	**	*
FLAs II	-	*	*	-	-	-
FLAs III	-	**	*	-	**	-
FLAs IV	-	**	-	*	*	-
FLAsVI	-	**	-	-	-	-
FLAsVII	-	**	*	-	**	*
FLAsVIII	-	**	*	-	. *	-
FLAsX	*	**	-	**	**	
FLAs XI	* '	**	-	-	**	-
FLAs XIV	-	**	-	-	*	-
FLAs XV	-	*	-	-	**	*
FLAs XVI	*	*	-	**	**	-
DAsI	-	*	-	-	-	-
DAsVII	-	_	-	-	*	-
DAsIX	-	-	-	*	*	***
DAsXII	-	- -	-	-	*	-
DAsXIV	-	-	-	-	*	-
DAsXV	-	***	***	*	*	***

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001.

Table 4 Summary data on significant differences between male and female individuals from the control group and male individuals with Klinefelter syndrome.

	Significant differences*	
Variables	K/C.Mal/	K/C.Fem
Arches (A)	*	-
Patterns U-U on III-III fingers	*	*
only whorls on the ten fingers	*	*
same pattern on the ten fingers	*	*
Ulnar loops (mean ridge count)	*	-
Radial loops (mean ridge count)	-	*
FRC I-r	**	-
FRC II-r	*	-
FRC V-r	**	-
FRC 1-1	*	-
FRC II-l	*	-
FRC IV-1	-	*
FRC V-l	**	-
TRC	**	-
Patt.(thenar), both hands	*	-
Patt.(interdig.III), both hands	*	*
accessory trirad. d', both hands	*	*
indiv. with absen.of c triradius	*	*
absence of c trirad., both hands	*	*
a-b ridge count, right hand	**	**

Citation: Kobyliansky E, Kalichman L. The connection between genetic anomalies and dermatoglyphics: insights from chromosomal, monogenic, and polygenic disorders. *J His Arch & Anthropol Sci.* 2025;10(2):49–56. DOI: 10.15406/jhaas.2025.10.00324

Table 4 Continued....

a-b ridge count, left hand	**	**
a-b distance (mm)	*	*
a-b ridge breadth (mm)	*	*
Sydney line	*	*
A line, exit 1	**	**
A line, exit r	**	**
D line, exit r	-	*
MLI	-	*
atd angle	*	*
axial triradius t	-	*
FLAs II	-	**
FLAs VII	*	**
FLAs VIII	-	*
FLAs IX	*	-
FLAsX	-	*
DAsI	-	*
DAsIV	-	*
DAsV	-	*
DAsVI	-	*
DAs XII	-	*
DAsXV	*	**

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001.

Table 5 Summary data on significant differences between men and women from the control group and women with Turner syndrome.

Variables	Significant differences* C. Males /Turner	C. Females /Turner
UL on the ten fingers	*	-
RCs of Ulnar Loops	*	*
RCs of Radial Loops	*	*
RCs of Whorls	-	*
FRC I-r	-	*
FRC II-r	**	**
FRC III-r	**	**
FRC IV-r	-	**
FRC V-r	**	**
FRC 1-l	-	*
FRC II-l	**	**
FRC III-l	*	**
FRC IV-l	-	**
FRC V-l	-	*
TRC	**	**
ARC	-	*

Table 5 Continued....

PII (Right hand)	**	-
Hypot. Patterns (both hands)	*	*
Indiv. with absen. of c trir.	*	-
Main line index (mean)	**	**
a-b ridge count (both hands)	**	**
atd angle (both hands)	*	*
axial triradius t	*	*
DivV	*	-
Div VIII	*	-
FLAsI	-	*
FLAs III	**	*
FLAs V	*	**
FLAs VI	*	**
FLAs VII	**	**
FLAs IX	**	**
FLAs XII	*	-
FLAs XIII	**	**
FLAs XIV	-	*
FLAs XV	*	**
FLAs XVI	*	**
DAsII	**	*
DAsIX	**	*
DAsX	**	*
DAs XIV	*	-

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001.

Turner syndrome: Decreased whorl frequency, increased ulnar loops, and elevated atd angles. TRC and a-b ridge counts were reduced by 8–15% compared to controls.

Klinefelter syndrome: Increased whorl frequency, decreased radial loops, and reduced U-U pattern combinations on finger III (40.9% vs. 54.0% in male controls). TRC was 10–12% lower than controls.

Down syndrome: High ulnar loop frequency (especially on fingers II and III), increased radial loops on fingers IV and V, and a 10-fold higher Simian line frequency. The atd angle was 72% higher in males and 65% higher in females compared to controls. TRC and a-b ridge counts deviated by 8–20%. Parents of Down syndrome patients showed intermediate ulnar and radial loop frequencies, significantly different from both affected children and controls (Tables 2-5) [7]

Dermatoglyphic traits in monogenic and polygenic disorders (Tables 6-7)

Cystic fibrosis (CF): Patients exhibited altered ridge counts and pattern frequencies, with increased FA in a-b ridge breadth and atd angle.[16] Parents showed subtle dermatoglyphic deviations, suggesting carrier status influences traits.

Table 6 Summary data on significant differences found between patients with cystic fibrosis, their parents, and healthy control groups.

Variables	Fathers /C.males	C.F.mal. /C.males	C.F.mal. /Father	Mothers /C.femal.	C.F.fem. /C.femal.	C.F.fem /Mothers
R-U on II-II fing.	-	*	*	_	_	-
Same pat.-on 10 fing	-	*	-	-.	-	-
RC of UL	-	-_	-_	-_	*	-
RC of W	-.	*	-_	-_	*	-
FRC I-r	***	-	*	-_	-	-
FRC IV-r	*	-	-	-_	-	-
FRC V-r	-.	-_	-_	*	-	-

Table 6 Continued....

FRC I-I	*	-	-_	-	-	-
FRCV-I	-.	-	-_	-	*	-
TRC	*	-	-_	-_	-	-
Then.pat.on both h.	-	*	*	-	-	-
ID.IV pat.on both h.	-	*	-	-	-	-
Absent of pat.ID.IV	-	-	-	-	-	*
Interdig.II b.s	-	-	-	-	*	-
Interdig.IV b.s	-	-	-	*	-	-
Indiv.With d'	-	*	.	-	-	-
Sydney line, lh	-	*	*	-	*	-
Sydney line, rh	-	*	*	-	-	-
A line, lh	-	-	-	**	*	-
A line, rh	-	**	-_	-	-	.
a-b RC, rh	***	*	-	*	*	-
a-b RC, lh	-	-	-	*	-	-
atd angle	-	*	*	-	-	-
DivI	-	*	-	-	-	-
DivIV	.	*	-	-	-	-
DivVII	-	*	-	-	-	-
FLAsI	-	-	-	*	-	-
FLAs IV	-	-	.	*	-	-
FLAsV	-	-	-	*	-	-
FLAsVI	.	-_	-_	*	-	-
FLAsVII	-	-	-	-	*	*
FLAsVIII	-	*	-	*	-	-
FLAsX	-	*	-	*	-	-
FLAs XIV	-	-	-_	*	-	-
FLAs XV	-	*	-	*	-	-
FLAs XVI	.	-_	-	*	-	-
DAs III	*	-_	-.	-	*	-
DAsVIII	**	*	-	-	*	-
DAsX	*	-_	-_	-	-	-
DAs XV	-	*	-	*	-	-

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001. - bs – bilateral symmetry.

- RU – radial and ulnar loops.

Table 7 Summary data on significant differences between patients with cleft lip (and cleft palate) or cleft palate, their parents, and healthy control groups.

Variables	**Fathers /Control males**	**CL(P)& CP males /C.males**	**CL(P), CP males /Fathers**	**Mothers /Control females**	**CL(P), CP females /C.females**	**CL(P), CP females /Mothers**
RCs of Whorls	-_	*	*	-	*	*
FRC IV-r	-	-	-	-	**	**
FRCV-r	-	-	-	-	*	*
FRC IV-I	-	-	-	-	*	*
TRC	-	-	-	-	*	*
absent of Hyp.patt.	-	-	-	*	-	-
absent of Then.patt.	-	*	-	-	-	-
absent ID.III part.	*	-	-	*	*	-
absent ID.IV patt.	-	-	-	-	*	-
Hyp.patt.on both h.	*	-	-	-	-	-
a' on rh. only	-	-	-	-	*	-
d' on rh. only	*	-	-	-	-	-
indiv. with c	*	-	-	*	-	-
Sydney line	-	-	-	-	*	-
A line, rh	-	-	-	-	-	*
atd angle	*	*	-	-	-	-

Table 7 Continued....

axial triradius t	*	-	-	-	-	-
a-b RC, rh	-	-	-	-	**	-
a-b RC, lh	*	-	-	*	-	-
Rid.breadth	*	-	-	*	-	-
FLAs II	-_	-	-	-	-	*
FLAs IV	-	-	-	-	**	*
FLAs VII	-	*	*	-	-	-
FLAs IX	-	**	**	-	-	-
FLAs XII	-	*	**	-	-	-
FLAs XIII	-	*	*	-	-	-
FLAs XIV	-	-	-	*	*	-
FLAs XVI	*	-	*	-	-	-
DAs III	-	-	*	-	*	-
BAs IX	-	-	-	-	***	-
DAsX	-	-	-	-	*	*

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001.

Cleft lip and/or palate (CL/P): Increased FA in finger ridge counts and palmar traits, with higher frequencies of rare hypothenar patterns. Parents displayed intermediate trait values, indicating a genetic component (Tables 6 & 7).[16]

Dermatoglyphic traits in endometrial and cervical carcinoma (Table 8)

Women with endometrial and cervical carcinoma showed significant dermatoglyphic deviations, including increased FA in a-b ridge breadth and altered pattern frequencies.[16] These changes suggest a potential genetic predisposition, though the etiology remains unclear (Table 8).

Table 8 Summary data on significant differences between women with carcinoma and control groups.

Variables	Cancer/Control Females	Males
W-W on IV-IV fingers	*	*
Ridge counts of Radial Loops	*	*
Ridge counts of Whorls	*	-
FRC I-r	-	*
FRC I-l	-	*
FRC IV-l	*	-
PII, rh	-	**
PII, both hands	-	*
Absent of Thenar patterns	-	*
Individuals with c' triradius	*	*
A line, lh	**	-
a-b distance (mm)	-	*
Ridge breadth (mm)	-	*
atd angle (degrees)	-	*
Axial triradius t	-	*
Axial triradius t'	-	*
Div III	*	-
FLAs III	*	*
FLAs IV	*	*
FLAs VII	*	*
FLAs IX	*	**
FLAsX	**	*
FLAs XI	-	*
FLAs XII	-	*
FLAs XV	*	-
FLAs XVI	**	**
DAsII	-	**
DAsIII	*	*
DAs VIII	*	**
DAsXV	**	-

* - Differences are statistically significant at P<0.05. -**According to the Bonferroni correction for multiple comparisons, P<0.001.

Asymmetry and variability indices

FA indices, particularly FLA VII (atd angle deviation) and FLA IX (a-b ridge breadth deviation), were significantly elevated in all patient groups compared to controls (P<0.001). Chromosomal syndromes showed the highest FA, followed by CF and CL/P. Variability indices like the Shannon index indicated greater pattern diversity in affected individuals. PCA revealed distinct clustering of patient groups based on quantitative traits and FA, with discriminant analysis correctly classifying 80–90% of individuals into patient or control groups.

Discussion

This study confirms a strong connection between genetic anomalies and dermatoglyphic traits, with chromosomal syndromes exhibiting the most pronounced deviations, followed by monogenic and polygenic disorders. The observed alterations in ridge counts, pattern frequencies, and FA align with previous research.[3,4,7] The high atd angle in Down syndrome, for instance, reflects its environmental sensitivity (heritability 0.33–0.48; Barnicot et al., 1972),[17] while stable polygenic traits like TRC (heritability 0.92–0.95; Holt, 1968) show consistent deviations across syndromes.

FA emerged as a critical marker of developmental instability, with elevated levels in all patient groups. This supports the hypothesis that genetic anomalies disrupt developmental homeostasis, leading to random bilateral deviations.[9,15,18] The higher FA in chromosomal syndromes likely reflects their severe genetic disruptions, such as aneuploidy, which impair developmental canalization. CF and CL/P, while less severe, also showed increased FA, suggesting that even single-gene or multifactorial defects compromise developmental stability.[7,16,19-25]

The dermatoglyphic deviations in parents of affected individuals, particularly in Down syndrome and CF, suggest a genetic basis for these traits. Intermediate trait values in parents may indicate carrier status or subclinical effects, supporting the use of dermatoglyphics in genetic counseling.[18] The findings in endometrial and cervical carcinoma patients are novel, as few studies have explored dermatoglyphics in cancer.[16] The increased FA and altered patterns in these women may reflect genetic predispositions or early developmental stressors, warranting further investigation.

The multivariate analyses (PCA and discriminant analysis) underscore the diagnostic potential of dermatoglyphics. The ability to accurately classify individuals into patient or control groups suggests that dermatoglyphic traits, particularly FA, could serve as non-invasive markers for genetic anomalies. However, limitations include the retrospective nature of the data and the lack of longitudinal studies to assess predictive value. Future research should explore the molecular mechanisms linking genetic anomalies to dermatoglyphic changes and validate these findings in diverse populations.[26–30,31]

Conclusion

Dermatoglyphics provides a valuable tool for studying genetic anomalies, with significant deviations in ridge counts, pattern frequencies, and FA observed in chromosomal, monogenic, and polygenic disorders. Elevated FA reflects disrupted developmental homeostasis, with chromosomal syndromes showing the most pronounced effects. The dermatoglyphic traits of parents and the novel findings in carcinoma patients highlight the potential for dermatoglyphics in genetic counseling and early detection. These results advocate for integrating dermatoglyphic analysis into clinical genetics, offering a cost-effective, non-invasive approach to assessing developmental stability and genetic risk.

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None.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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