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Comparison of Digito-Palmar Dermatoglyphic Traits in Children with Cerebral Palsy and Their Close Family Members

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A B S T R A C T

Cerebral palsy is one of the main causes of severe disability in children. Sixty children (30 boys and 30 girls) were included in the study. Quantitative digito-palmar dermatoglyphic traits were analyzed. Prints of digito-palmar dermatoglyphs obtained from the children's parents (60 mothers and 60 fathers) and from 400 phenotypically healthy adults from the Zagreb ware used as control groups. Analysis of quantitative dermatoglyphic traits of the digito-palmar complex revealed statistically significant differences in a number of variables between the fathers and their children suffering from cerebral palsy (TRC 180.3>158.6), with a greater number of variables involved in male children with cerebral palsy. Some variables showed statistically significant differences in dermatoglyphic patterns between fathers and control group of healthy males as well as between boys with cerebral palsy and healthy control males. Differences in dermatoglyphic patterns were significantly lower between mothers and girls with cerebral palsy (TRC 152.1<152.3) as well as between mothers and healthy control females. Study results support the hypothesis on the possible role of genetic predisposition in the occurrence of central nervous system lesion, with a more pronounced paternal impact.

Key words: *quantitative dermatoglyphics, cerebral palsy, genetic predisposition*

Introduction

According to the International Cerebral Palsy Society, the incidence of cerebral palsy in Europe, North America and Australia has been estimated to 2.5% of live births[1]. This is a heterogeneous disease and the etiology has not yet been fully clarified. In many cases, cerebral palsy can be prevented by timely rehabilitation; however, once the clinical picture has fully developed, the patient's quality of life is significantly impaired and therapeutic options are relatively limited in spite of all advances in modern medicine. Therefore, all efforts should be invested in elucidating the etiology of the disease onset in order to improve treatment modalities[2]. According to the latest studies, only 10% to 15% of cerebral palsy cases can be ascribed to intrapartal problems. Perinatal asphyxia cannot be confirmed in the majority of children born after 36th week of gestation that developed cerebral palsy, while a potential cause is identified in only 40% of cases[3]. Other authors report on perinatal asphyxia as the cause of cerebral palsy in only 6% to 8% of cases, whereas prenatal risk factors account for approximately 75% of cerebral palsy cases[4].

Some studies suggest the possible role of genetic factors in the genesis of cerebral palsy. According to literature data, genetically determined causes are identified in as many as 40% of cerebral palsy patients[5]. During development, these genetic factors can influence development of the brain and perhaps of other organs developing at the same time and from the same germ layer. Central

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nervous system (CNS) develops from ectoderm at the beginning of the third week. It is a long-term process, taking approximately 30 years to achieve an adult person configuration[6].

Like brain, dermatoglyphics also develop from ectoderm, in the same period, thus the factors influencing development of the brain can be postulated to influence development of dermatoglyphics as well. Specific digito-palmar dermatoglyphic traits have been detected in many diseases[7–9], indicating the hereditary nature of these diseases like specific L shaped loops in Down syndrome, and high frequency of arches in most other chromosomal aberrations[10]. In our previous studies in diseases with unknown etiology, higher frequency of whorls in different type of cancers[9] or higher frequencies of arches in mental retardations[11] were found. The study by Cvjetičanin and Polovina revealed that clinically severe CNS lesions were probably associated with certain changes in digito-palmar dermatoglyphics[12]. Therefore, we intended in this present study to compare digito-palmar dermatoglyphics in children suffering from cerebral palsy, their parents and a healthy control group, in an attempt to identify the possible genetic predisposition for this debilitating disorder.

Material and Methods

The study included 60 children (30 boys and 30 girls) with cerebral palsy, aged 18 months to 14 years, treated at »Prof. Milena Stojčević-Polovina« Polyclinic for Physical Medicine and Rehabilitation in Zagreb, Croatia. Digito-palmar dermatoglyphic prints were obtained from their parents (60 mothers and 60 fathers) as a comparative group, and from 400 phenotypically healthy persons (200 males and 200 females) from the Zagreb area as a control healthy group[13]. Dermatoglyphic traits were analyzed on both palm prints according to Cummins and Midlo[10] and Miličić et al.[14].

Hereditary predisposition for the occurrence of CNS lesion in children was assessed by quantitative analysis of digito-palmar dermatoglyphic complex. The following quantitative traits were analyzed: finger ridge count right (FRR1-FRR5); finger ridge count left (FRL1-FRL5); and total finger ridge count on both hands (TRC); on palms, ridge counts right (R) and left (L) (a-b rcR and a-b rcL; b-c rcR and b-c rcL; and c-d rcR and c-d rcL); total palmar ridge count (TPRC); values of atd angle (atd R and atd L) for both hands.

The descriptive statistics of quantitative digito-palmar dermatoglyphic traits was performed. Differences between the children and their parents were assessed by dependent t-test, and differences between the parents and control group of healthy subjects by independent t-test. Discriminant analysis was performed to determine separating variables and the relationship between examined groups in discriminant area. All analysis ware performed using SPSS 7.5 for Windows package.

Results

Descriptive statistics (means and standard deviations) of studied variables in boys and girls suffering from cerebral palsy are presented in Table 1. Descriptive statistics of analyzed variables in fathers and mothers of children with cerebral palsy ware presented in Tables 2, and those obtained in the group of healthy male and female controls in Table 3. The fathers of boys with cerebral palsy had a very high total finger ridge count (TRC=180.3) that differ from TRC in boys with cerebral palsy (TRC=158.6) and this difference is statistically significant ($t=2.63$; $p<0.014$) (Table 4). The fathers also showed higher values of all finger variables, differing significantly from boys with cerebral palsy in FRR1 ($t=2.17$; $p<0.038$), FRR4 ($t=2.16$; $p<0.039$), FRL3 ($t=2.28$; $p<0.030$), FRL4 ($t=2.18$; $p<0.038$), FRL5 ($t=3.45$; $p<0.002$). The boys showed higher values of atd angle than their fathers, however, it may have been due to the palm size. There was no difference between the boys with cerebral palsy and their mothers according to finger variables,

TABLE 1
DESCRIPTIVE STATISTICS OF QUANTITATIVE DERMATOGLYPHIC TRAITS OF DIGITO-PALMAR COMPLEX IN BOYS AND GIRLS WITH CEREBRAL PALSY

	Boys with cerebral palsy		Girls with cerebral palsy	
	χ	SD	χ	SD
Right hand				
FRR1	20.0	3.02	18.9	3.17
FRR2	13.9	6.71	13.6	5.44
FRR3	13.6	6.21	13.4	4.76
FRR4	16.6	4.67	17.5	4.64
FRR5	16.6	12.76	15.2	5.22
TFRCR	80.7	23.24	78.6	16.76
a-b rcR	40.9	7.74	42.0	4.67
b-c rcR	27.3	5.89	28.8	5.71
c-d rcR	35.9	7.96	40.7	4.38
atd R	46.1	7.60	51.1	12.24
Left hand				
FRL1	19.1	3.80	17.5	3.66
FRL2	14.4	8.08	13.7	5.44
FRL3	13.1	6.09	13.3	5.78
FRL4	16.6	5.72	15.4	4.48
FRL5	14.7	4.72	13.9	4.80
TFRCL	77.9	22.25	73.7	16.95
a-b rcL	41.6	6.55	42.3	5.97
b-c rcL	27.4	6.69	29.1	5.99
c-d rcL	35.7	8.08	40.0	5.42
atd L	46.1	6.03	53.1	10.84
Total				
TRC	158.6	42.99	152.3	32.37
TPRC	208.8	32.97	223.0	22.57

TABLE 2
DESCRIPTIVE STATISTICS OF QUANTITATIVE DERMATOGLYPHIC TRAITS OF DIGITO-PALMAR COMPLEX IN PHENOTYPICALLY HEALTHY FATHERS AND MOTHERS OF THE CHILDREN WITH CEREBRAL PALSY

	Fathers of the boys with cerebral palsy		Fathers of the girls with cerebral palsy		Mothers of the boys with cerebral palsy		Mothers of the girls with cerebral palsy	
	χ	SD	χ	SD	χ	SD	χ	SD
Right hand								
FRR1	22.2	4.82	20.0	7.88	19.6	5.98	16.3	6.69
FRR2	16.4	6.30	14.1	7.46	11.7	7.13	14.8	8.49
FRR3	15.5	5.26	13.0	5.76	12.2	4.91	13.1	7.15
FRR4	18.9	4.46	17.2	5.97	15.7	6.53	16.1	7.31
FRR5	17.8	4.31	15.5	5.05	13.1	5.02	13.8	5.24
TFRCR	90.8	19.34	79.8	26.27	72.2	22.32	74.2	28.89
a-b rcR	41.4	7.52	42.4	7.08	43.4	5.37	41.6	5.27
b-c rcR	28.7	5.89	30.1	7.01	27.4	5.95	26.9	5.52
c-d rcR	36.5	9.05	39.0	6.89	38.5	6.74	37.9	5.93
atd R	43.7	6.08	45.1	7.84	46.7	9.43	44.6	6.10
Left hand								
FRL1	20.5	5.23	16.8	8.02	17.7	4.87	16.3	6.69
FRL2	16.7	6.57	12.0	6.97	13.8	7.62	14.8	8.49
FRL3	15.4	6.20	13.9	7.12	11.9	7.40	13.1	7.15
FRL4	19.0	5.12	16.8	5.33	14.5	6.87	16.1	7.31
FRL5	17.9	3.58	14.3	5.43	13.2	5.36	13.8	5.24
TFRCL	89.5	21.23	73.9	27.02	71.0	24.00	74.2	28.89
a-b rcL	40.8	5.54	44.9	7.63	44.6	5.30	41.6	5.27
b-c rcL	28.1	5.74	29.2	6.11	26.0	6.44	26.9	5.52
c-d rcL	37.5	9.31	38.0	6.05	38.1	7.54	37.9	5.93
atd L	41.9	4.94	45.9	8.64	45.3	7.05	44.6	6.10
Total								
TRC	180.3	39.61	153.7	52.40	143.2	45.26	152.1	52.58
TPRC	213.0	28.09	223.6	30.73	218.1	29.01	211.1	18.62

but differences were recorded in a-b rc right ($t=2.05$; $p<0.049$) and left ($t=2.38$; $p<0.024$) and ridge count in the interdigital area between digital triradii a and b. Arrieta et al.[15] reported that this variable show great differences between men and women, being for a significantly longer period influenced by environmental factors during development in men and more genetically determined in women. In contrast to their fathers, the boys suffering from cerebral palsy differed from the control group of healthy male population in a few variables. Total ridge count was also significantly greater in boys with cerebral palsy (TRC=158.6) when compared with the control group of healthy male controls (TRC=141.0) but difference is not statistically proved (Table 4).

The girls suffering from cerebral palsy showed no significant differences from their fathers except for atd angles on the right hand ($t=2.44$; $p<0.021$), and from their mothers except for a-b rcR ($t=2.90$; $p<0.007$), and atd angles right ($t=2.57$; $p<0.016$), and left ($t=4.07$; $p<0.001$). In comparison with the control group of healthy women, these girls had greater ridge count in all variables; statistically significant differences were recorded in FRR5 ($t=2.58$; $p<0.011$), FRL1 ($t=2.46$; $p<0.015$), FRL2, ($t=2.18$; $p<0.030$). Total finger ridge count was considerably greater in the girls suffering from cerebral palsy (TRC=152.3) as compared with the control group of healthy women (TRC=133.4; $t=2.33$; $p<0.021$) (Table 4).

Results presented in Table 5 show that fathers differed significantly from the control group in all finger variables with a significantly higher ridge count: FRR1 ($t=2.61$; $p<0.010$), FRR2 ($t=3.54$; $p<0.001$), FRR3 ($t=2.79$; $p<0.006$), FRR4 ($t=2.35$; $p<0.020$), FRR5 ($t=4.24$; $p<0.001$), FRL1 ($t=3.65$; $p<0.001$), FRL2 ($t=4.47$; $p<0.001$), FRL3 ($t=2.89$; $p<0.004$), FRL4 ($t=2.36$; $p<0.019$), FRL5 ($t=5.02$; $p<0.001$). Statistically significant differences ware found for palmar variables a-b rcL ($t=2.06$; $p<0.040$), and atd angle right ($t=2.39$; $p<0.018$) and left ($t=4.08$; $p<0.001$). The fathers of children suffering from cerebral palsy had a statistically significantly higher total ridge count as compared with the control group of healthy men (TRC=167.0

TABLE 3
DESCRIPTIVE STATISTICS OF QUANTITATIVE DERMATOGLYPHIC TRAITS OF DIGITO-PALMAR COMPLEX IN GROUP OF PHENOTYPICALLY HEALTHY CONTROLS

	Control males		Control females	
	χ	SD	χ	SD
Right hand				
FRD1	19.4	5.63	17.2	5.56
FRD2	11.4	7.27	11.6	6.55
FRD3	12.0	6.58	11.4	5.31
FRD4	16.2	6.15	15.8	5.72
FRD5	13.6	5.16	12.7	4.83
TFRCD	72.6	24.65	68.8	21.65
a-b rcD	41.9	6.86	41.0	6.02
b-c rcD	28.6	5.87	27.3	6.00
c-d rcD	37.9	6.07	36.7	6.43
atd D	47.4	8.27	46.9	8.67
Left hand				
FRL1	16.2	6.14	14.8	5.76
FRL2	10.8	6.78	10.9	6.88
FRL3	11.8	6.37	11.6	5.72
FRL4	16.2	6.17	15.1	5.25
FRL5	13.5	4.60	12.3	4.80
TFRCL	68.5	23.88	64.6	22.08
a-b rcL	43.6	7.05	41.8	5.90
b-c rcL	28.7	5.85	26.9	5.67
c-d rcL	36.6	7.00	36.3	6.86
atd L	47.9	7.70	47.7	8.39
Total				
TRC	141.0	47.44	133.4	42.57
TPRC	217.9	27.19	211.1	24.46

vs. TRC=141.0; t=4.31; p<0.001). Although there ware few differences between the mothers of boys suffering from cerebral palsy and control group of healthy women. Differences ware found for FRR1 (t=2.13; p<0.035), FRL1 (t=2.62; p<0.009) and FRL2 (t=2.12; p<0.035).

Fathers of the girls do not differ statistically from the healthy control males, while mothers of the girls differ statistically in FRR2 (t=2.23; p<0.027), FRR3 (t=2.85; p<0.005), and FRL2 (t=2.85; p<0.005) and TRC (t=2.18; p<0.030). Palmar variables do not show any differences between fathers and mothers of girls with cerebral palsy when compared with the healthy control group.

Discriminant analysis has shown statistically significant first two functions Wilks' Lambda for the first function was 0.634 (χ^2=245.413; p<0.001) and for the second function was 0.730 (χ^2=155.394; p<0.001), and the function values are presented on Table 6. Graphical presentation is on Figure 1. From the Figure 1 there is clear difference between males and females as was expected because dermatoglyphic variables show sex difference. Only the boys suffering from cerebral palsy are positioned very close to their mothers and also to the mothers of the affected girls. Both mothers differ from healthy control females. Fathers of the girls suffering from cerebral palsy are similar to the healthy control males, but fathers of the boys differ from all other groups.

Discussion and Conclusion

In the last 30 years, genetics has proved to be a significant component in almost all aspects of research and clinical science[16]. A number of clinical reports point to the role of genetic factors in the etiology of cerebral palsy. In 1999, Al-Rajeh et al., studied a group of children with cerebral palsy and demonstrated a 2.5-fold incidence of the disorder in families where parents were consanguineous relatives. The authors conclude that hereditary factors have a significant role in the pathogenesis of cerebral palsy[17]. Morton et al.[18] assessed the prevalence of severe neurologic disorders in children of various ethnic groups (Pakistan and India) and found genetic diseases associated with such disorders to be ten times more com-

TABLE 4
DIFFERENCES IN QUANTITATIVE DERMATOGLYPHIC TRAITS OF DIGITO-PALMAR COMPLEX AMONG MALE AND FEMALE CHILDREN WITH CEREBRAL PALSY, THEIR PARENTS AND HEALTHY CONTROL GROUPS

	Boys with cerebral palsy			Girls with cerebral palsy		
	Fathers[a] t p<	Mothers[a] t p<	Control males[b] t p<	Fathers[a] t p<	Mothers[a] t p<	Control females[b] t p<
Right hand						
FRR1	2.17; 0.038	0.38; 0.710	0.63; 0.532	0.69; 0.496	0.17; 0.867	1.64; 0.103
FRR2	1.83; 0.078	1.40; 0.171	1.76; 0.079	0.35; 0.733	0.74; 0.463	1.58; 0.116
FRR3	1.89; 0.069	1.12; 0.273	1.29; 0.199	0.31; 0.759	1.00; 0.327	1.91; 0.058
FRR4	2.16; 0.039	0.61; 0.546	0.35; 0.726	0.17; 0.868	0.86; 0.397	1.54; 0.125
FRR5	0.55; 0.585	1.55; 0.132	2.26; 0.025	0.33; 0.741	1.33; 0.194	2.58; 0.011
a-b rcR	0.33; 0.743	2.05; 0.049	0.67; 0.503	0.29; 0.778	2.90; 0.007	0.87; 0.384
b-c rcR	0.88; 0.385	0.13; 0.899	1.14; 0.255	0.87; 0.393	1.39; 0.176	1.27; 0.204
c-d rcR	0.27; 0.792	1.78; 0.086	1.64; 0.103	1.14; 0.264	1.65; 0.111	3.27; 0.001
atd R	2.24; 0.033	0.32; 0.750	0.85; 0.398	2.44; 0.021	2.57; 0.016	2.37; 0.019
Left hand						
FRL1	1.41; 0.169	1.38; 0.179	2.49; 0.014	0.47; 0.644	0.81; 0.423	2.46; 0.015
FRL2	1.14; 0.264	0.33; 0.742	2.70; 0.007	1.28; 0.211	0.73; 0.470	2.18; 0.030
FRL3	2.28; 0.030	0.85; 0.400	1.04; 0.299	0.56; 0.579	0.09; 0.927	1.51; 1.33
FRL4	2.18; 0.038	1.70; 0.100	0.27; 0.788	1.17; 0.253	0.49; 0.628	0.27; 0.786
FRL5	3.45; 0.002	1.54; 0.133	1.37; 0.172	0.34; 0.733	0.09; 0.929	1.71; 0.088
a-b rcL	0.61; 0.550	2.38; 0.024	1.42; 0.156	1.66; 0.108	0.52; 0.604	0.42; 0.679
b-c rcL	0.44; 0.661	0.89; 0.380	1.12; 0.264	0.03; 0.980	1.68; 0.104	1.99; 0.048
c-d rcL	1.06; 0.299	1.84; 0.076	0.67; 0.506	1.61; 0.119	1.37; 0.180	2.81; 0.005
atd L	4.37; 0.001	0.62; 0.542	1.17; 0.243	0.14; 0.890	4.07; 0.001	3.16; 0.002
Total						
TRC	2.63; 0.014	1.72; 0.095	1.91; 0.057	0.12; 0.905	0.02; 0.986	2.33; 0.021
TPRC	0.66; 0.513	1.99; 0.056	1.66; 0.099	3.14; 0.004	2.41; 0.023	2.50; 0.013

[a] dependent t-test; [b] independent t-test

mon in Pakistani children, which was attributed to the customary consanguineous marriage in this country. Monreal et al. [19] reports on the significant genetic impact in a high percentage of cerebral palsy patients; neurologic disorder underlain by a familial factor and a genetic source indicating the condition were identified in 60%–70% of patients diagnosed with cerebral palsy. Costeff et al.[5] performed mathematical analysis of prenatal and perinatal factors in 681 cases of idiopathic cerebral palsy and found that the disease could not have been avoided by preventing preterm delivery or improving intensive care of the preterm infant in the group of patients with genetically determined disorder.

Kalil Pessoa de Barros et al.[20] found significant association between cerebral palsy and apolipoprotein e4 allele mutation, considering it a risk factor for the development of cerebral palsy as a consequence of trauma of the central nervous system before its final stage of maturation. Kuroda et al.[21] also found apolipoprotein e4 and e2 genotypes as susceptibility factors in determining neurologic outcomes after perinatal brain injury.

McHale et al.[22] investigated the gene for ataxic cerebral palsy in four families and identified the gene locus responsible for cerebral palsy on 9p12-q12 chromosome. In the study performed by Nelson et al., genotyping of 96 children with cerebral palsy and 119 control children revealed a different gene distribution in the former[23]. Gibson and colleagues have shown that if fetuses with halotypes that produce low levels of lectins are exposed to viruses antenatally and that some forms of CP are due to the combination of environmental viral infections and abnormal genetic halotypes[24].

Quantitative analysis of digito-palmar dermatoglyphic traits supported our hypothesis on the genetic predisposition involved in the etiology of cerebral palsy. As we found no comparable reports in the available literature, we have to rely on our own observations. Differences recorded on all ten fingers in both boys and girls suffering from cerebral palsy pointed to statistically significant changes in quantitative finger traits in children. The finger ridge count that was considerably higher in children with cerebral palsy when compared to controls, and in fathers particular, ware associated with the subse-

TABLE 5
DIFFERENCES IN QUANTITATIVE DERMATOGLYPHIC TRAITS OF DIGITOPALMAR COMPLEX BETWEEN PHENOTYPICALLY HEALTHY PARENTS AND HEALTHY CONTROL GROUPS

	Fathers of boys CP/Control males[a]	Mothers of boys CP/Control females[a]	Boys CP/Girls CP[a]	Fathers of girls CP/Control males[a]	Mothers of girls CP/Control females[a]
	t p<	t p<	t p<	t p<	t p<
Right hand					
FRR1	2.61; 0.010	2.13; 0.035	1.38; 0.174	0.51; 0.613	1.74; 0.083
FRR2	3.54; 0.001	0.04; 0.968	0.19; 0.850	1.88; 0.061	2.23; 0.027
FRR3	2.79; 0.006	0.71; 0.481	0.16; 0.871	0.80; 0.425	2.85; 0.005
FRR4	2.35; 0.020	0.04; 0.967	0.75; 0.457	0.90; 0.370	0.40; 0.691
FRR5	4.24; 0.001	0.39; 0.700	0.56; 0.580	1.89; 0.061	0.99; 0.324
a-b rcR	0.36; 0.723	2.04; 0.043	0.67; 0.508	0.41; 0.684	1.68; 0.095
b-c rcR	0.10; 0.919	0.08; 0.939	1.02; 0.310	1.31; 0.191	0.13; 0.901
c-d rcR	1.12; 0.264	1.40; 0.164	2.87; 0.006	0.88; 0.383	1.39; 0.165
atd D	2.39; 0.018	0.08; 0.939	1.93; 0.059	1.42; 0.156	1.52; 0.131
Left hand					
FRL1	3.65; 0.001	2.62; 0.009	0.166; 0.102	0.46; 0.649	1.31; 0.193
FRL2	4.47; 0.001	2.12; 0.035	0.39; 0.695	0.96; 0.338	2.85; 0.005
FRL3	2.89; 0.004	0.25; 0.803	0.13; 0.897	1.68; 0.095	1.34; 0.180
FRL4	2.36; 0.019	0.61; 0.540	0.88; 0.383	0.50; 0.621	0.93; 0.355
FRL5	5.02; 0.001	0.95; 0.341	0.71; 0.483	0.91; 0.365	1.59; 0.114
a-b rcL	2.06; 0.040	2.47; 0.014	0.41; 0.682	0.92; 0.358	0.19; 0.847
b-c rcL	0.53; 0.594	0.76; 0.448	1.06; 0.295	0.39; 0.695	0.03; 0.975
c-d rcL	0.65; 0.519	1.31; 0.190	2.46; 0.017	1.06; 0.290	1.18; 0.240
atd L	4.08; 0.001	1.49; 0.139	3.08; 0.003	1.25; 0.211	1.94; 0.053
Total					
TRC	4.31; 0.001	1.16; 0.246	0.64; 0.526	1.35; 0.180	2.18; 0.030
TPRC	0.92; 0.0359	1.41; 0.159	1.94; 0.057	1.04; 0.299	0.01; 0.996

[a] independent t-test

quent development of cerebral palsy. The mother effect is also suggestive of the genetic predisposition for the disorder, but there is a greater paternal impact in the development of CNS lesion.

TABLE 6
VALUES OF THE FIRST TWO STATISTICALLY SIGNIFICANT FUNCTIONS

Analyzed groups	Function I	Function I
Boys with cerebral palsy	0.654	–0.242
Girls with cerebral palsy	–0.236	–0.608
Fathers of boys with CP	1.351	–0.114
Fathers of girls with CP	0.111	0.360
Mothers of boys with CP	0.395	–0.196
Mothers of girls with CP	0.401	–0.185
Control males	–0.132	0.388
Control females	–0.300	–0.251

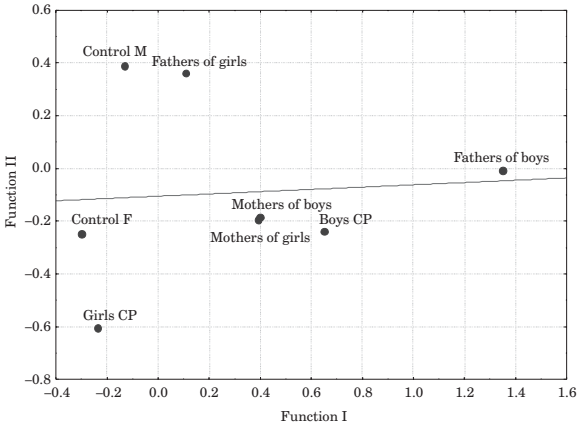

Fig. 1. Graphic presentation of the first two statistically significant functions for all examined groups.

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KVANTITATIVNA ANALIZA DIGITO-PALMARNIH DERMATOGLIFA DJECE OBOLJELE OD CEREBRALNE PARALIZE I NJIHOVIH RODITELJA

SAŽETAK

Cerebralna paraliza je jedan od glavnih uzroka teških nesposobnosti u djece. Naše ispitivanje je obuhvatilo 60 djece (30 djevojčica i 30 dječaka) s cerebralnom paralizom i njihovih roditelja. Kod ispitivane djece provedena je analiza kvantitativnih obilježja digito-palmarnih dermatoglifa. Kao zdrava komparativna skupina u analizi uzeti su otisci njihovih roditelja (60 majki i 60 očeva) te 400 odraslih i fenotipski zdravih osoba iz zagrebačke regije. Analiza kvantitativnih svojstava digito-palmarnih dermatoglifa pokazala je statistički značajne razlike u više promatranih varijabla između očeva i djece oboljele od cerebralne paralize, što je u većem broju varijabla zastupljeno u dječaka. Takvi rezultati govore u prilog moguće genetske predispozicije u nastanku oštećenja središnjeg živčanog sustava pri čemu je značajniji utjecaj očinske linije.