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Comparative Study on Dermatoglyphics in Patients with PTSD

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A B S T R A C T

The factors situated at the bases of the genesis and development of PTSD are divided in: biological, psychological and social factors. Primary factor is a stressful event of extremely dangerous and threatening nature. The sort and the intensity of the stressful event too play an important role, followed by the personality structure, the relation with the environment and the genetic constitution. The study was thought to determine the quantitative dermatoglyphic properties of the digito-palmar complex in patients with PTSD aiming to establish whether there are biological, that is, genetic bases for PTSD, in what measure they determine the clinical manifestation of the disorder, and whether there is a dermatoglyphic marker, characteristic for people with PTSD. We analyzed the quantitative properties of the digito-palmer complex on a group of 100 male examinees over 18 years of age with PTSD, no psychiatric comorbidity, and who were two or more times cured at the Psychiatric Hospital »Sveti Ivan«, comparing them with the quantitative dermatoglyphic properties of a group of 100 phenotypically healthy male examinees over 18 years of age. Using the method of descriptive statistics, we found no statistically significant differences among the results of the examined groups. With the T-test we evaluated the heterogeneity of the groups, and the results showed the existence of statistically significant differences among the comparison group and the group of patients with PTSD on three variables. We calculated the Fluctuating Asymmetry (FA) measure, which illustrates the compatibility, that is, the symmetry of the observed property on the right and on the left side of the body, indicating a difference on one variable. The outcome did not confirm the existence of a connection between a particular dermatoglyphic result and the genesis of PTSD. This is, however, in line with the hypothesis that, in patients with PTSD and other psychiatric disorders, there is a multiple effect of several micro-abnormalities in different genes, along with the inevitable and essential influence of environmental and/or physical and/or psychosocial stressogenic factors.

***Key words**: PTSD, dermatoglyphics, genetic heritage, stress, trauma*

Introduction

The human skin on the palm of both hands and fingers and on the sole each foot and toes is ridged, so that you can see many ridges separated by grooves, whose courses create lines of different shapes. These lines were named dermatoglyphics, which comes from the Greek words *derma*=skin and *glyphe*=to carve. The coin was proposed by Cummins and Midlo in 1926, two scientists who created the basics for the modern science that studies dermatoglyphics[1].

Volar pads start defining themselves during the third month of the embryonic growth. A volar pad is a raised mesenchymal tissue situated in the proximity of the most distal metacarpal bone of each finger, in each inter-digital space and in the area of the thenar and hypothenar space, on the palms and the soles as well as on the heels. When volar pads are at the peak of their formation in the third month of embryogenesis, the border between the epidermis and the dermis, which is initially smooth, comes to a corrugation process of the epidermis' basal membranes, which leads to the formation of dermatoglyphics[2]. This corrugation process in the fetus takes place between the 12th and the 36th weeks of the embryonic growth. All factors that participate in the genesis of dermatoglyphics are primarily genetically determined, but we must not forget or ignore the effect of a whole series of non-genetic factors, which determine in what phe-

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notype the genotype expresses itself. The development of dermatoglyphics ends with the beginning of the seventh month of the embryogenesis, when the lumens of the sweat gland open on the surface of the papillary ridges (Duvančić, 1971, Schaumann et al, 1976)[3,4].

Throughout several analyses, it was noticed that dermatoglyphic changes are most expressed in chromosomal aberrations, both sexual chromosomes and autosomes. This knowledge was implemented with diagnostic purposes, therefore they described a defined sample for different chromosomopathies as it is Turner's or Klinefelter's syndrome (Penrose and Loesch, 1969 and 1970, Škrinjarić and collaborators, 1976)[5–7].

Studies on dermatoglyphics were carried in relation to different illnesses that appear in people with a normal number of chromosomes, assuming the genetic influence on the genesis of a certain disorder and the influence of external factors on the development of dermatoglyphics in the early prenatal period[8–11].

Posttraumatic Stress Disorder (PTSD), according to contemporary psychiatric classifications, belongs to the group of anxiety disorders. It appeared as a psychiatric nosologic entity for the first time in 1980, in the third edition of the Diagnostic and Statistic Manual of Mental Health (DSM-III) of the American Psychiatric Association[12]. The main etiological feature of PTSD, without which a disorder can not appear, is the exposure to an unusually intensive traumatic event. The event can be a situation of risk for the life of a person or for their physical integrity. Other situations could be the witnessing of the death, the injury or the threat to the physical integrity of another person, and also the cognition of the unexpected or violent death, the death danger or injury of an intimate person. Intensive fear and feeling of helplessness and terror appear as a response to such events. A specific symptomatic paradigm appears a consequence of being exposed to a trauma, which features: repeated re-experiencing the traumatic event, constant avoiding of stimuli associated with the trauma, dullness of general reactivity and constant symptoms of increased arousal. The period of disturbances including all symptoms has to be longer than a month and they have to cause clinically significant handicaps or damage in one's social, professional or some other functioning[12,13].

According to studies carried out on the general population, the prevalence of PTSD during one's lifetime goes from 1% to 14%, depending on the data gathering methods and on the kind of sample that were used. Studies formulated on specific risk samples, such as war veterans, victims of volcano eruptions and criminal violence, showed a prevalence of 3% to 58%[12,14].

The factors situated at the bases of the genesis and development of PTSD are divided in: biological, psychological and social factors. The primary factor is a stressful event of extremely dangerous and threatening nature. The sort and the intensity of the stressful event too play an important role, followed by personality structure, relation with the environment and by the genetic constitution[13].

In correlative studies carried out up until now, molecular genetic research has grouped eight main genotypes that could be correlated with PTSD[15]. The hypotheses involve genes in the serotonin (5-HTT, Lee et al., 2005)[16], dopamine (DRD2, Lawford et al.; 2003., Young et al., 2002.; Gelernter et al., 1999.; Comings et al., 1996.; DAT, Segman et al., 2002.)[17–21], glucocorticoid (GR, Bachmann et al., 2005.)[22] and GABA systems (GABR3, Feusner i sur., 2001)[23], and the genes in the apolipoprotein (APOE, Freeman i sur., 2005.)[24], brain-derived neurotrophic factors (BDNF, Zhang i sur., 2006. a,b; Lee et al., 2006.)[25–27] and neuropeptide Y (NPY, Lappalainen et al., 2002.)[28] systems. Although the studies produced inconsistent results, it was legitimate to conclude that the interaction of different genes and the interaction of these genes with the environment are likely to make certain subjects vulnerable and exposed to the developing of PTSD[15].

Material and Methods

The study sample was a group of 100 male examinees over 18 years of age with PTSD, without psychiatric comorbidity, who were cured at the Psychiatric Hospital »Sveti Ivan« two or more times. Diagnoses were given by psychiatrists according to the International Statistical Classification of Diseases and Health Related Problems – Tenth Revision (ICD–10), or confirmed, according to the same criteria, in patients who were already cured in other psychiatric hospitals with a diagnosis of PTSD (F43.1)[29]. The comparison group featured 100 phenotypically healthy male examinees, over 18 years of age.

We took dermatoglyphic prints of the patients' digito-palmar complex (the prints of all ten fingers and both palms) through the method described by Cummins and Midlo (1943–1961) and following the instructions from the books »Practicum of biological anthropology – genetic methods« (1977) and »Dermatoglyphics in antropological researches« (1989)[8]. We analyzed the following properties: number of ridges on the fingers (finger ridge count FRR1–FRR5), number of ridges on the interdigital areas of the palm (a-b rcR, b-c rcR, c-d rcR, a-b rcL, b-c rcL, c-d rcL), measuring of atd angles (atd R, atd L). We compared the quantitative dermatoglyphic properties in the group of patients with those in the group of healthy examinees. In the processing of the data, we implemented descriptive statistics methods, the multivariate (MANOVA) and univariate (ANOVA) analysis of the variation, discriminative analysis and factor analysis. We used the Statistical Package for Social Sciences Software (SPSS) in all mentioned statistical processing methods.

Results

Descriptive analysis shows the features of the quantitative dermatoglyphic properties of the digito-palmar complex in patients affected by PTSD and in the comparison group of phenotypically healthy examinees (Tables 1 and 2).

The quantitative dermatoglyphic properties of the digito-palmar complex in patients affected by PTSD sta-

TABLE 1
RESULTS OF THE DESCRIPTIVE STATISTIC OF THE QUANTITATIVE DERMATOGLYPHIC PROPERTIES OF THE DIGITO-PALMAR COMPLEX IN THE GROUP OF PATIENTS AFFECTED WITH PTSD (N=100)

	$\overline{X}$	Standard deviation (SD)	Min	Max	N
RIGHT HAND					
FRR1	16.38	4.917	2	29	100
FRR2	10.64	5.904	0	26	100
FRR3	10.11	5.141	0	23	99
FRR4	12.76	5.425	0	22	99
FRR5	11.09	4.356	1	20	100
a-b rcR	35.68	7.260	18	56	100
b-c rcR	25.63	6.907	7	58	100
c-d rcR	30.91	5.756	14	43	100
atd R	44.54	7.067	32	72	100
LEFT HAND					
FRL1	14.38	5.071	0	26	100
FRL2	10.53	5.208	0	21	100
FRL3	10.71	5.309	0	23	100
FRL4	13.07	5.377	0	24	100
FRL5	11.44	4.611	1	21	100
a-b rcL	36.71	6.274	20	54	100
b-c rcL	25.39	5.451	10	42	100
c-d rcL	33.53	6.666	10	45	100
atd L	44.76	7.332	33	73	100

FRR1 – FRR5 – finger ridge count right,
FRL1 – FRL5 – finger ridge count left
a-b rcR, b-c rcR, c-d rcR – ridge count between digital triradii a-b, b-c and c-d on the right palm
a-b rcL, b-c rcL, c-d rcL – ridge count between digital triradii a-b, b-c and c-d on the left palm
atd R – atd L – values of atd angle of right and left hand

TABLE 2
RESULTS OF THE DESCRIPTIVE STATISTIC OF THE QUANTITATIVE DERMATOGLYPHIC PROPERTIES OF THE DIGITO-PALMAR IN THE COMPARISON GROUP (N=100)

	$\overline{X}$	Standard deviation (SD)	Min	Max	N
RIGHT HAND					
FRR1	16.09	5.143	0	33	100
FRR2	9.97	5.723	0	24	100
FRR3	10.20	4.851	0	23	100
FRR4	13.21	4.619	0	31	100
FRR5	10.38	3.969	2	21	100
a-b rcR	33.03	7.323	13	49	100
b-c rcR	24.03	6.211	11	46	100
c-d rcR	28.81	8.187	5	45	100
atd R	43.39	8.252	30	85	100
LEFT HAND					
FRL1	14.46	4.633	0	24	100
FRL2	9.71	5.767	0	23	100
FRL3	11.14	5.162	0	25	100
FRL4	13.75	4.680	3	30	100
FRL5	11.31	4.182	3	20	100
a-b rcL	35.47	6.614	16	55	100
b-c rcL	24.65	6.379	9	44	100
c-d rcL	30.03	8.100	8	47	100
atd L	43.73	7.092	32	72	100

FRR1 – FRR5 – finger ridge count right,
FRL1 – FRL5 – finger ridge count left
a-b rcR, b-c rcR, c-d rcR – ridge count between digital triradii a-b, b-c and c-d on the right palm
a-b rcL, b-c rcL, c-d rcL – ridge count between digital triradii a-b, b-c and c-d on the left palm
atd R – atd L – values of atd angle of right and left hand

tistically significantly differ from those in the group of phenotypically healthy examinees as follows:

- The number of ridges between the triradii a and b on the right palm (p=0.011): patients affected by PTSD have in average a higher number of ridges on the same anatomic location of the right palm than the examinees from the comparison group;
- The number of ridges between the triradii c and d on the right palm (p=0.037): patients affected by PTSD have in average a higher number of ridges between the triradii c and d on the right palm than the examinees from the comparison group;
- The number of ridges between the triradii c and d on the left palm (p=0.001): patients affected by PTSD have in average a higher number of ridges on the same anatomic location of the left palm than the examinees from the comparison group (Table 3).

The variable with the highest discrimination, that is, the variable that has the highest share in the explained variance of outcomes between the group of patients affected by PTSD and the comparison group, is the number of ridges between the triradii c and d on the left palm (Table 4).

Considering the extracted discriminative function, 59% of the examinees classified correctly, 65% of which were from the PTSD group and 53% from the comparison group (Table 5).

During the analysis of the so called latent dimension, the number of extracted factors, on which each variable is extracted through the highest projections, was five. We noticed the following differences: in the group of PTSD patients, the quantitative dermatoglyphic properties of all fingers on both hands are grouped together apart from the thumbs, which are separated as a factor *per se*. In the group of phenotypically healthy examinees, the quantitative dermatoglyphic properties of the thumbs are grouped together with the fourth and the fifth finger of both hands in one factor, while the quantitative properties of the dermatoglyphics of the second and third finger of both hands are separated as a factor *per se*. In addition, in the PTSD group, the areas between the triradii c,

TABLE 3
TEST OF THE DIFFERENCES IN THE AVERAGE NUMBER OF FINGER RIDGES

	Levene's test of homogeneity of variance		t-test of average equity		
	F	p	t	df	p
RIGHT HAND					
FRR1	0.106	0.745	–0.408	198	0.684
FRR2	0.148	0.701	–0.815	198	0.416
FRR3	1.103	0.295	0.125	197	0.900
FRR4	3.302	0.071	0.634	197	0.527
FRR5	0.475	0.492	–1.205	198	0.230
a-b rcR	0.033	0.856	–2.570	198	0.011
b-c rcR	0.000	0.987	–1.722	198	0.087
c-d rcR	8.180	0.005	–2.098	178	0.037
atd R	1.267	0.262	–1.058	198	0.291
LEFT HAND					
FRL1	0.478	0.490	0.116	198	0.907
FRL2	1.156	0.284	–1.055	198	0.293
FRL3	0.000	0.984	0.581	198	0.562
FRL4	3.481	0.064	0.954	198	0.341
FRL5	0.369	0.544	–0.209	198	0.835
a-b rcL	0.004	0.951	–1.360	198	0.175
b-c rcL	2.669	0.104	–0.882	198	0.379
c-d rcL	1.323	0.251	–3.337	198	0.001
atd L	0.022	0.881	–1.010	198	0.314

FRR1 – FRR5 – finger ridge count right,
FRL1 – FRL5 – finger ridge count left
a-b rcR, b-c rcR, c-d rcR – ridge count between digital triradii a-b, b-c and c-d on the right palm
a-b rcL, b-c rcL, c-d rcL – ridge count between digital triradii a-b, b-c and c-d on the left palm
atd R – atd L – values of atd angle of right and left hand

d and b, c on both palms are grouped together, while the area between the triradii a, b form factor *per se*. In the group of phenotypically healthy examinees, the areas between the triradii b, c and a, b on both palms are grouped together, while the area between the triradii c, d form a factor *per se* (Table 6 and 7).

We calculated the Fluctuating Asymmetry (FA) measures. Between the comparison group and the PTSD group there is a statistically significant difference in the fluctuating asymmetry only in the number of ridges between the triradii a and b (p=0.046). The comparison group has a higher fluctuating asymmetry than the PTSD group. On other variables there are no statistically significant differences in the correlation of values of the right and left hand between the comparison group and the PTSD group.

Discussion

In the Classification of Mental and Behavioral Disorders – Clinical Descriptions and Diagnostic Guidelines (Tenth revision) of the World Health Organization, Posttraumatic Stress Disorder (PTSD) is classified as a nosological unit in the group of Neurotic Disorders – stress--related and somatoform disorders (F40–F48). This implies that, in its etiology, environmental factors, or stressors, and stressful events, play a significant role, which means that they almost take a dominant position in relation to other potential etiological factors[29]. On the other hand, neuroscientific studies, which have been mainly carried out in the past two decades, pointed out the sig-

TABLE 4
CORRELATION OF DISCRIMINATIVE VARIABLES AND CANONICAL DISCRIMINATIVE FUNCTION AMONG THE GROUP OF PATIENTS AFFECTED WITH PTSD AND THE COMPARISON GROUP OF EXAMINEES, SHOWN IN ORDER OF SIZE OF THE CORRELATION INSIDE THE FUNCTION.

Variable	FUNCTION
c-d rcL	1.000
c-d rcR (a)	0.442
atd L (a)	0.282
a-b rcR (a)	0.266
a-b rcL (a)	0.265
b-c rcR (a)	0.210
atd R (a)	0.197
b-c rcL (a)	0.166
FRR3 (a)	0.155
FRL4 (a)	0.108
FRL2 (a)	0.105
FRR4 (a)	0.098
FRL3 (a)	0.095
FRL1 (a)	0.076
FRL5 (a)	0.066
FRR5 (a)	0.064
FRR2 (a)	0.060
FRR1 (a)	–0.005

(a) – variable is not used in the analysis
FRR1 – FRR5 – finger ridge count right,
FRL1 – FRL5 – finger ridge count left
a-b rcR, b-c rcR, c-d rcR – ridge count between digital triradii a-b, b-c and c-d on the right palm
a-b rcL, b-c rcL, c-d rcL – ridge count between digital triradii a-b, b-c and c-d on the left palm
atd R – atd L – values of atd angle of right and left hand

TABLE 5
OVERVIEW OF THE RESULTS OF THE EXAMINEES' CLASSIFICATION CONSIDERING THE DISCRIMINATIVE FUNCTION

	N	Classified			
		Correctly		Incorrectly	
		n	(%)	n	(%)
PTSD	100	65	(65%)	35	(35%)
Comparison group	100	53	(53%)	47	(47%)
Total of correctly classified:		59%			

TABLE 6
FACTOR ANALYSIS OF VARIABLES IN THE GROUP OF PATIENTS AFFECTED BY PTSD (VARIMAX ROTATION WITH KAISER'S NORMALISATION)

	Factors				
	1	2	3	4	5
FRL3	0.856	0.133			
FRL4	0.826		–0.209	0.150	0.138
FRR3	0.806	0.169	0.187		
FRR2	0.795		0.163		0.134
FRL2	0.787		0.185	–0.109	0.172
FRR4	0.767			0.108	0.213
FRL5	0.754		–0.247	0.174	0.312
FRR5	0.709		–0.203	0.221	0.265
c-d rcR	0.163	0.814	–0.169	0.217	
b-c rcR		0.723	0.372		0.173
c-d rcL	0.218	0.714	0.152	0.162	
b-c rcL		0.657	0.399		0.141
a-b rcR		0.166	0.833	0.194	
a-b rcL		0.228	0.821	0.279	
atd L			0.173	0.846	
atd R	0.155		0.199	0.809	
FRR1	0.301				0.838
FRL1	0.397	0.156			0.765
Variance percentage	34.043	17.752	8.414	6.414	5.910

FRR1 – FRR5 – finger ridge count right,
FRL1 – FRL5 – finger ridge count left
a-b rcR, b-c rcR, c-d rcR – ridge count between digital triradii a-b, b-c and c-d on the right palm
a-b rcL, b-c rcL, c-d rcL – ridge count between digital triradii a-b, b-c and c-d on the left palm
atd R – atd L – values of atd angle of right and left hand

TABLE 7
FACTOR ANALYSIS OF VARIABLES IN THE COMPARISON GROUP (VARIMAX ROTATION WITH KAISER'S NORMALISATION)

	Factors				
	1	2	3	4	5
FRR1	0.756	0.216		–0.224	0.113
FRL1	0.738	0.145	0.104		
FRR5	0.733	0.224	–0.109	0.217	
FRL5	0.726	0.160		0.246	
FRL4	0.693	0.449			0.103
FRR4	0.640	0.456	0.160	–0.133	
FRL2	0.244	0.856			
FRR2	0.268	0.799	–0.182		
FRR3	0.497	0.687			
FRL3	0.587	0.671			
b-c rcR	–0.139		0.823		–0.145
b-c rcL			0.803	0.134	–0.151
a-b rcR			0.755		0.372
a-b rcL	0.191	–0.148	0.617	0.241	0.343
atd L			0.122	0.862	0.153
atd R		0.136	0.336	0.749	0.165
c-d rcR	0.206				0.807
c-d rcL	–0.117			0.245	0.762
Variance percentage	32.166	17.050	8.652	6.668	5.560

FRR1 – FRR5 – finger ridge count right,
FRL1 – FRL5 – finger ridge count left
a-b rcR, b-c rcR, c-d rcR – ridge count between digital triradii a-b, b-c and c-d on the right palm
a-b rcL, b-c rcL, c-d rcL – ridge count between digital triradii a-b, b-c and c-d on the left palm
atd R – atd L – values of atd angle of right and left hand

nificance of genetic components in the etiology of different psychiatric disorders, including PTSD[30–32].

It is important to point out a new paradigm that appeared in psychiatric genetics, in which genes still play an important role, but their possible influence is observed in a new way[33,34]. Now they are no longer seen as the direct cause of psychiatric disorder as they attempted to prove, but the cause of micro-molecular abnormalities that generate the risk of appearance of psychiatric disorders. Genes, or better, proteins, »lead« each neural network in the brain towards an inefficient processing of information and to a consequent loss of cerebral functions in certain environmental conditions, which then show themselves as specific psychiatric symptoms[33].

In other words, it is thought that each gene creates genetically modified proteins, which lead to micro-molecular abnormalities. These proteins play a part in neuro-development processes such as neural selection, migration, differentiation and synaptogenesis. Here are also included different protein enzymes, different kinds of bearers, receptors and many other molecules[33,35–37]. The appearance of a certain disorder is the result of a multiple micro-contribution of several genes interacting with the environmental factors. This is also called complex genetics, as it is not a matter of a simple dominating or recessive heritage, but a whole series of risk factors that favor the genesis of a certain disorder, but are not its direct cause[38]. In other words, this means that an individual does not inherit the illness, but the risk, which merges with the accelerating influence from the environment and eventually fully develops in the disorder's clinical manifestation[33,37].

These influences, as for instance in PTSD, can be all sorts of extremely unfortunate and troublesome life events and traumas, but also different kinds of viruses and toxins. Healthy people have a genome that can compensate the unfortunate influences of stressors and maintain a normal activity of their neural networks. However, people with multiple genetic risk factors, under unfortunate environmental influences, will not be able to adequately compensate the stressor's activity. This leads to an inefficient processing of information and consequently to a dysfunction of certain neural networks, which will eventually manifest in specific psychiatric symptoms. It is im-

portant to add that, in some cases, environmental stressors can be so intense as to cause a disorder even without the presence of a genetic risk or, as it was formerly commonly known, predisposition[35,38].

Hence, in the context of the mentioned classification-etiological and neuroscientific views, we thought and carried out this study with the intent to examine the quantitative dermatoglyphic properties of the digito-palmar complex in patients affected by PTSD. The aim was to find out whether there are biological and genetic bases for PTSD, and, if they exist, in what measure they determine the clinical manifestation of this psychic disorder. The study was inspired by already existing research on dermatoglyphic properties in patients affected by different diseases and disorders of unclear etiology, in whom heritage and genetic bases were supposed to play a significant role.

The above mentioned results, suggest that there are not significant differences in the quantitative properties of patients affected by PTSD in relation to healthy subjects, which shows that the genes that influence on the formation and shape of dermatoglyphics are not involved. This is congruous with the empirical and etiological cognition that the influence of environmental factors on PTSD is extremely important. It is also part of the definition of PTSD, according to which the genesis of PTSD can not take place without an extremely intense stressogenic, traumatic event. However, this would bring a superficial and faulty conclusion. The analyses that were carried out are an attempted to identify, through data processing, the possible, assumed polygenic sets that are responsible for their conformation. The differences resulted in the extracted factors, that is, in the variance of each factor over different dermatoglyphic properties, can point out potential different polygenic sets in the studied groups (PTSD and comparison group of phenotypically healthy subjects). The outcomes may lead to the conclusion that a genetic predisposition can actually exist and indirectly manifest in the phenotype of the analyzed properties. In the context of the formerly mentioned paradigm on complex genetics in the background of psychiatric disorders, according to which the appearance of a disorder is the result and product of a joint micro-action of several different genes in cooperation with environmental factors, we can conclude that genes are not directly and independently responsible for the appearance and manifestation of PTSD. However, it does not mean that they have no influence on it at all. If we consider the numerous transgenetic studies and studies on twins, which pointed out the higher rate of PTSD in singular families and a higher concordance for the development of PTSD in monozygotic than in heterozygotic twins, then we definitely must not ignore the factor of genetic influence[39,40]. It is important to stress that the subject of this study was the analysis of the quantitative dermatoglyphic properties in the digito-palmar complex, which are highly defined by genetic heritage. As Rudan showed in 1975, their characteristics are: larger selective inertia, smaller variability, larger genetic stability and poorer subjection to genetic drift in relation to the qualitative dermatoglyphic properties[41]. They are a good indicator of the initial characteristics of the population (Rudan and Szirovicza, 1983)[42,43]. If we consider these facts, along with fact that we did not find significant differences between the examinees and the comparison group and the fact that dermatoglyphics' formation takes place between the 12^{th} and 20^{th} week of intrauterine development receiving their ultimate shape, we can conclude that the main influence of a certain environmental event most likely did not occur in that period of early gestation. This is also congruous with the formerly mentioned empirical cognition and with the generally accepted definition.

If we were once again to reinforce and confirm what was mentioned and possibly get to new cognitions in relation to the genetic bases of PTSD, we would recommend to carry out this study on a wider sample of examinees and definitely to carry out new studies on the qualitative properties of the digito-palmar complex in patients affected by PTSD in relation to healthy examinees.

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POREDBENO ISTRAŽIVANJE DERMATOGLIFA OBOLJELIH OD PTSP-a

S A Ž E T A K

Faktori koji se nalaze u podlozi nastanka i razvoja PTSP-a dijele se na biološke, psihološke i socijalne. Osnovni je pak faktor stresni događaj izrazito opasnog i ugrožavajućeg karaktera. Važnu ulogu imaju i vrsta te intenzitet stresnog događaja, zatim struktura ličnosti same osobe, odnos okoline prema osobi kao i genetska konstitucija osobe. Predmet su istraživanja bila kvantitativna svojstva dermatoglifa digitopalmarnog kompleksa kod osoba oboljelih od PTSP-a s ciljem ispitivanja postoji li biološka odnosno genetska etiološka podloga PTSP-a i u kojoj mjeri ona determinira kliničko očitovanje poremećaja, te postoji li dermatoglifski biljeg karakterističan za osobe oboljele od PTSP-a. Analizirana su kvantitativna svojstva digitopalmarnog kompleksa skupine od 100 ispitanika muškog spola starijih od 18 godina oboljelih od PTSP-a koji nemaju psihijatrijski komorbiditet te su dva ili više puta bolnički liječeni u Psihijatrijskoj bolnici »Sveti Ivan«, uz odgovarajuću usporedbu s kvantitativnim svojstvima dermatoglifa skupine od 100 fenotipski zdravih ispitanika muškog spola također starijih od 18 godina. Metodom deskriptivne statistike nisu nađene statistički značajne razlike u rezultatima između ispitivanih skupina. T–testom procjenjivana heterogenost ispitivanih skupina pokazala je da postoji statistički značajna razlika između komparativne skupine i skupine oboljelih od PTSP-a u tri ispitivane varijable. Provedeno je i izračunavanje mjere fluktuacijske asimetrije (FA) koja oslikava podudarnost odnosno simetričnost promatranog svojstva na desnoj i lijevoj strani tijela, a koja je pokazala razliku u jednoj promatranoj varijabli. Dobiveni rezultati nisu potvrdili povezanost određenog dermatoglifskog nalaza s pojavom PTSP-a, odnosno postojanje karakterističnog dermatoglifskog biljega kod osoba oboljelih od PTSP-a. To je pak u skladu s hipotezom da se kod PTSP-a kao i kod drugih psihijatrijskih poremećaja radi o mnogostrukom učinku više mikroabnormalnosti kod nekoliko različitih gena, uz neizostavan i ključan utjecaj okolišnih i/ili okolinskih fizikalnih i/ili psihosocijalnih stresogenih čimbenika.