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RESEARCH ARTICLE

QUANTITATIVE ANALYSIS OF DIGITOPALMAR DERMATOGLYPHICS IN THIRTY FEMALE CEREBRAL PALSY CHILDREN

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ABSTRACT

By the one of genetic method, quantitative analysis of digitopalmar dermatoglyphics, we have made research in thirty female have made research in thirty female cerebral palsy children, in the prevention purpose. We have found statistically signific. We have found statistically significant differences to control in totally fifteen variables, in nces to control in t significance the sense of increased number of epidermal ridges: on the right hand and finger: the fifth finger finger (FRD5), on all five fin finger (FRD), on all five finger together (TFRCD), between triradii c-d (c-d rcD), triradii ((TFRCD), between triradii c-d (c-d a-b, b-c and c-d, all together (TPR rcD), and in Atd angle increased in degrees. Then, in together (TPF rcD) and in Atd angle seven variables on the left hand and fingers: first (FRL1), second (FRL2), and five all to- fingersal gether (TPRCL), between triradii b-c (b-c rcL) and c-d (c-d rcL), between triradii a-b (a-brcL)and c-d all toge- rcL), b-c (b-c rcL) and c-d (c-d rcL) all together (TPR cL) and Atd angle in degrees (AtdL) And on the end, in threeon both han And on the end, in three on both hands an fingers: ten fingers all together (TFRC), be- tween triradii a-b (b rcL), b-c (b-c rcL) and c-d (c-drcL) all together (TPRC) and Atd an- gles in degree both palm (ATDDL).The obtained data indicate a hypothetical genetic impact that simultaneously damaged brain and changed dermatglphic drawing, which c seems quite possible due to common ectodermal origin of both system.

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INTRODUCTION

Cerebral palsy (CP), is well-recognized neurodevelopmental conditionbeginnig in early childhood and persisting through the life span. Originally reported by Little in 1861 (and originally called "cerebral paresis"), CP has been the subject of books and papers by some of the most eminent medical minds of the past one hundred years. At the end of the 19th century, Sigmund Freud and Sir William Osler both began contribute important perspectives on the condition. From the mid-1940s the founding fathers of the American Academy for Cerebral Palsy and Developmental Medicine (Carlson,Crothers, Deaver, Fay, Perlstein and Phelps) in the United States, and Mac Keith, Polani, Bax and Ingram of the Little Club in the United Kingdom, were among the leaders who moved the concepts, and descriptions of CP forward and causedthis condition to become the focus of treatment services, advocacy and research efforts (The whole passage has taken from the Rewiev Introduction reference (Rosenbaum 2007), because of an exelent and concise presentation). The interesting remarks about, has made (Armstrong 2007). CP definition, describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain. The motor disrorders of cerebral palsy are often accompanied by disturbances of sensa- tion, perception, congnition, communaction, and behavior, byepilepsy and by secondary musculosceletal problems.

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MATERIALS AND METHODS

Dermograms of thirty female cerebral palsy children were analysed in keeping with instructions provided by Miličić *et al*. Methods (1989). Results were compared with 200 dermograms of phenotypically normal females from the Zagreb area,

obtained from the Institute of Anthropology in Zagreb (Schmutzer, 1977). Palmar and finger prints were taken by HSW finely granulated, silver-gray powder used in criminalistics, onto transparent, adhesive tape by a brush made of squirrel tail (Cvjetičanin 1990). Dermatoglyphic analyisis should be strictly separated according to sex, because of the great impact of sex chromosome and sex hormones on dermatoglyphic traits (Bener 1979; Al Jumaili 2010). Even significant sex differences have been found within control gruoup (Schmutzer 1997).

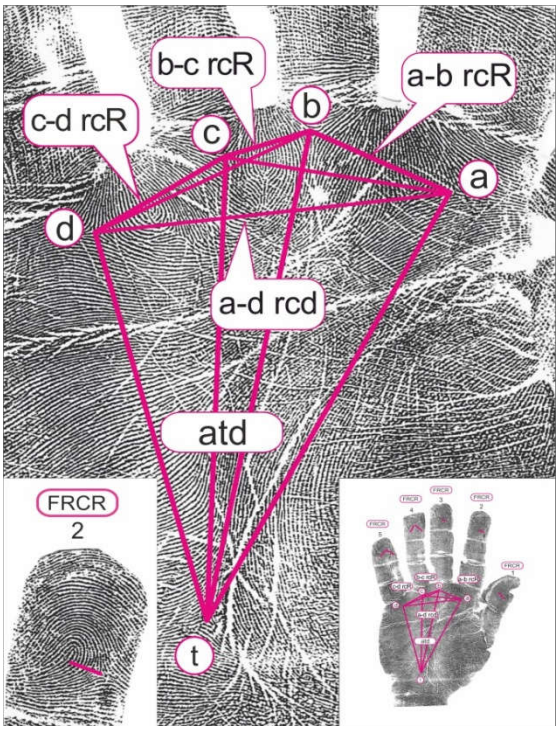
Later, it will be discussed in much more details about. Student's t-test was used to test statistically significant difference in the ridge count between the patient and control group. The following 25 traits were examined by the quantitative dermatoglyphic analysis, as it shown on Picture 2 and tables 1-3.

- FRD1 ridge count on the first finger of the right hand,
- FRD2 ridge count on the second finger of the right hand,
- FRD3 ridge count on the third finger of the right hand,
- FRD4 ridge count on the fourth finger of the right hand,
- FRD5 ridge count on the fifth finger of the right hand,
- TFRCD total ridge count on the all five fingers of the right hand,
- a-brcD ridge count between triradii a-b of the right hand,
- b-crcD ridge count between triradii b-c of the right hand,
- c-drcD ridge count between triradii c-d of the right hand.
- TPR rcD ridge count between all triradii, of the right hand, a-b, b-c and c-d all together.
- ATD angle on the right palm in degrees.
- FRL1 ridge count on the first finger of the left hand,
- FRL2 ridge count on the second finger of the left hand,
- FRL3 ridge count of the third finger of the left hand,
- FRL4 ridge count on the fourth finger of the left hand,
- FRL5 ridge count on the fifth finger of the left hand,
- TFRCL ridge count on all five fingers on the left hand,
- a-brcL ridge count between triradii a-b of the left hand,
- b-crcL ridge count between triradii b-c on the left hand,
- c-drcL ridge count between triradii c-d on the left hand,
- TPR rcL ridge count between triradii on the left palm (a-b, b-c and c-d all together),
- ATD L angle on the left palm in degrees,
- TFRC total ridge count on all ten fingers on hand,
- TPRC bilateral ridge count between all triradii a-b,c-d and c-d of the palms,
- ATDDL bilateral sum of atd angles in degrees.

RESULTS

The results are tabularly presented in Tables 1-3. Statistically significant difference to control by the Student's t-test was found in the fifteen variables in the sense of increasing number of epidermal ridges and Atd angles in degrees. In five variables on the right hand and fingers: the fifth finger (FRD5), on all five finger together (TFRCD), between triradii c-d (c-d rcD), triradii a-b, b-c and c-d, all together (TPR rcD), and in Atd angle increased in degrees. Then, in seven variables on the left hand and fingers: first (FRD1) and second (FRD2) finger, and five fingers all together (TFRCL), between triradii b-c (b-c rcL) and c-d (c-d rcL), between triradii a-b, b-c and c-d (TPR cL) and Atd (Atd L) in degrees. And on the end, in three on both hand and fingers, ten fingers all together (TFRC), between triradii a-b, b-c and c-d all together (TPRC) and Atd angles in degrees (ATDDL).

Picture 1. Right hemiparesis cerebral palsy female child with her mother's permission for taking a picture



Picture 2. The areas of quantitative analysis on palm and fingers dermatoglyphics

Table 1. Quantitative properties of right handdigitopalmar dermatoglyphicsin patients and controls

Variable	Patient group	Control group	Risk
	n x SD	n x SD	p
FRD1	30 18,9 3,17	200 17,23 5,56	0,103
FRD2	30 13,6 5,44	200 11,62 6,55	0,116
FRD3	30 13,4 4,76	200 11,44 5,31	0,058
FRD4	30 17,5 4,64	200 15,78 5,72	0,125
FRD5	30 15,2 5,22	200 12,70 4,83	0,011
TFRD	30 78,6 16,76	200 68,77 21,65	0,018
a-b rcD	30 42,0 4,67	200 41,03 6,02	0,384
b-c rcD	30 28,8 5,71	200 27,31 6,00	0,204
c-d rcD	30 40,7 4,38	200 36,70 6,43	0,001
TPR cD	30 111,5 10,61	200 105,01 12,68	0,009
Atd D	30 51,1 12,24	200 46,87 8,67	0,019

Table 2. Quantitative properties of left hand digitopalmar dermatoglyphics in patients and controls

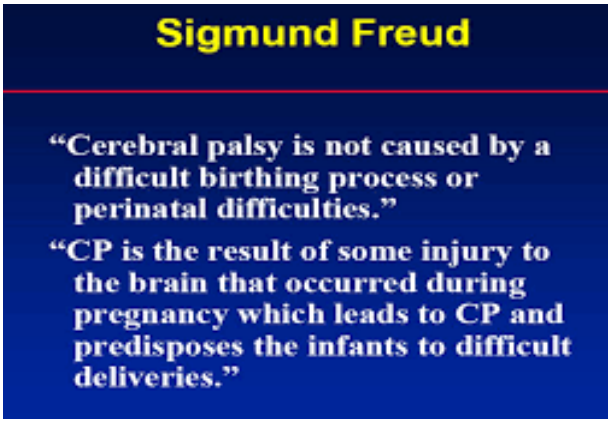
Variable	Patient group	Control group	Risk
	n x SD	n x SD	p
FRL1	30 17,5 3,66	200 14,805,76	0,015
FRL2	30 13,7 5,44	200 10,87 6,88	0,030
FRL3	30 13,3 5,78	200 11,585,72	0,440
FRL4	30 15,4 4,48	200 15,13 5,25	0,056
FRL5	30 13,9 4,80	200 12,26 4,80	0,015
TRCL	30 73,7 16,95	200 64,62 22,08	0,031
a-b rcL	30 42,3 5,97	200 41,82 5,90	0,803
b-c rcL	30 29,15,99	200 26,90 5,67	0,048
c-d rcL	30 40,0 5,42	200 36,34 6,86	0,005
TPR cL	30 111,5 13,55	200 105,20 13,28	0,018
Atd L	30 53,1 10,84	200 47,70 8,39	0,002

Table 3. Quantitative properties of digitopalmar complex both hands in patients and controls

Variable	Patient group	Control group	Risk
	n x SD	n x SD	p
TFRC	30 152,3 32.37	200 133,39 42,57	0,021
TPRC	30 223,0 22,57	200 211,08 24,46	0,013
ATDDL	30 104,2 21,66	200 94,56 15,88	0,003

DISCUSSION

Despite improved obstetric practice and better antenatal and perinatal care, several study indicate there has been little reduction in the incidence of CP over the last several decades. This data and other findings have led some investigators to suggest that, unknown pathophysiologic processes "must be at work to account for significant proportion of CP, which means that much of this unknown pathophysiology may be owing to genetic and epigenetic factors, then hundreds of CP genes likely await discovery (Fahey, 2017). From the very beginnig, Sigmund Freud (1897), for example, initialy a practicing – neurologist concerned with children, became greatly interested in relationship between non progressive neurological deficits and prematurity, and pleaced greater etiological emphasis on intrauterine developmental abnormality. Exactly the same, after hundredyear later, have mentioned (Stanley 1997) (Bakketeig 1999), (Jacobsson 2004), (MacLennan 2015: epidemiological studies have shown that origins of most CP, are prior to labor and clinical risk factors could act as triggers for CP where there is genetic susceptibility. These new findings should refocus research about the causes of these complex and varied neurodevelopmental disorder. Then, Blickstein: meticulous review of the scientific data suggest that overholming majority of CP cases has nothing to do with the process of labor (Mazaki-Tovi, 2017). The next important fact, because of that



Genetics linked to nearly half of all cerebral palsy cases, research found

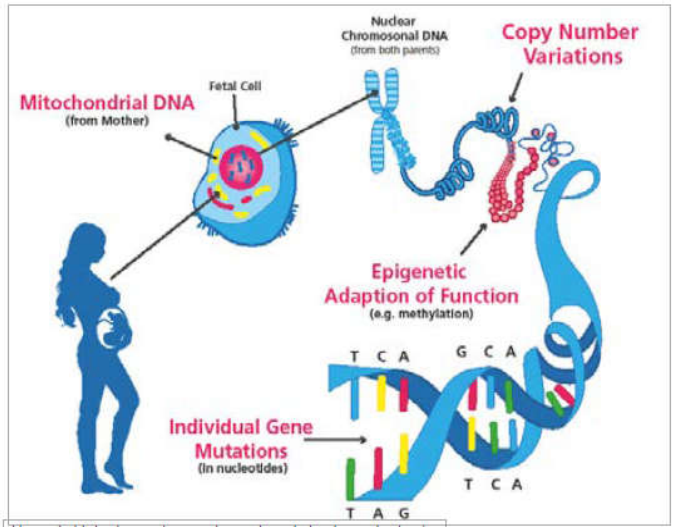
Genetics linked to nearly half of all cerebral palsy cases researchers find. https://www.abc.net.au/news/2015-06-03/ cerebral-palsy

It is quite a dramatic finding and we'll have to change the way we think about cerebral palsy and the way we go about trying to chastise the people who we are trying to help.

Professor Alastair MacLennan

That genetics in cerebral palsy it may be the main etiological factor, which we should looking for. The perinatal and postnatal or environmental, are the precipitation factors only, or a less of the importance. Until now genetics linked to nearly half of all cerebral palsy cases, research found, but it seems much more (Mac Lennan 2015). A gene is the basic physical and functional unit of heredity. Genes are makeup of DNA. Some genes act as instructions to make molecules called proteins. However, many genes do not code proteins. In humans, genes vary in size from a few hundred DNA bases to more than 2 million bases. The Human Genome Project estimated that humans have between 20.000 and 25.000 genes. Every person has two copies of each gene, one inherited from each parent. Most genes are the same in all people, but a small number (less than 1 percent of the total) are slightly different between people. Allels are forms of the same gene with difference in their sequence of DNA bases. These small differences contribute to each person's unique physical features. There are four main types of DNA variation, that contribute to CP pathogenesis (Picture 3), yet the ultimate effect of most mutations is a loss of the normal cellular function of the protein coded by the gene

1. Mitochondrialinheritance
2. Copy number variation
3.Epigeneticadaptation of function
(eg methylation-meaning a process that regulates genetic readout, working as ON/OFF signal that tells genes whether or not to produce proteins)and
4.Genomic variation/mutation (Mac Lennan, 2016).



Picture 3. Main sites and types of genetic variation in cerebral palsy (2016 AH MacLennon, used with permission).

The worth of mention is CNVs (Fahey *et al.,* 2017) mentioned already 10% to 20% versus 1% in comparation to general population Altough de novo CNVs (genomic variants not found in mother or father occuring in an affected child), CNVs can be inherited maternally or paternally from unaffected parents, this often leds to the conclusion that a given CNVs is not relevant to diseases process, well documented examples exist wherein varibale expressivity, incomplete penetrarnce, higer genetic robustness of females versus males, or the presence of genetic/epigenetic for proteins modifiers or second hits instead, explaine why the parent does not manifest symptoms but their childdoes (Fahey 2017). Epigenetic, then, means changes in gene function that can not beexplanied by changes in DNA sequnce. Prions and non-coding RNA are considered to be epigenetic (there are three basic epigenetic marks: (1. post-translational modifications that occur on the proteins around which the DNA is wrapped (histons), 2. methilation on the deoxyribonucleotide basis within DNA, and 3 hydroxy - methilation of DNA). Mitochondrial inheritance is the inheritance of a trait encoded in the mitochondrial genome, Because of the oddities of mitohchondria, mitochondrial inheritance does not obey the classic rules of genetics. Persons with a mitochondrial diseases, may be male orfemale but they always related in the maternal line and no male with disease can transmit it is children. The mitochondria are normal structures or organelles in cells.They are located in the cell's cytoplasm outside the nucleus. The mitochondria are responsible for energy production. They consist of two sets of membranes, a smoothcontinuos outer coat and inner membrane arranged in tubules or in folds that form plate-like double membranes (cristae). The mithohondria are in fact the principal energy source of the cell (thanks to the cytochrome enzymes of terminal electron transport and enzymes of the citric acyd cycle, fatty acid oxidation, and oxidative phosphorylation). The mitohondria convert nutritiens into energy aswell as doing many of the specialized tasks. Each mitohondrian has achromosome that is made of DNA (mitochondrial or mtDNA) but isotherwise quite different form the better known chromosomes in thenucleus. The mitochondrial chromosome is much smaller. It is round(whereas the chormosome in the nucleus are shaped like rods). Thereare many copies of the mitochondiral chromosome in every cell (whereas there is normal only one set of chormosomes in the nuclelus). Mitochondirial DNA contains 37 genes which all are essential for normal function of the mitochondria. Many genetic conditions are related to changes in particular mitochondrial genes,(Medical Definiton of Mitochondrial inhertence 2018).

The next interestingthing is, why are males affected more tnen females CP patients. Cerebral palsy is more common in males than females, but the reasons for this disparity are uncertain. Hormonal influlences on the fetus and neonates are substancially different from those in adults. Then other reports demosntrated major differences between male and female neurons grown separately in cell culture, suggesting that sex differences in fetal or neonatal period result fromintrising differences in cell deatlh pathways. This new information indicates that there are important neurobiological differences betweenmales and females with respect to their response to brain injuries.Cerebral electrical activity matures earlier in male than in female term neonates.Du et al 2004, grew neurons fromfemales (XX) and males (XY), separately in vitro and found that XY celles were more sensitive to toxic effects. Thordstein *et al.* 2006 reported that recessive X linked chromosome variants may contribute to this difference and males may be more vulnerable to genetic mutation variations then females. About one third of sex linked disorders with major motor abnormalities found in CP (spastic paraplegia and ataxia). Sex-linked conditions disproportionately affect males because males have only one X chromosome. If the X chromosome is abnormal, unlike the female, there is no potentially normal X chromosome to counterbalance it. The counterbalancing phenomenon relates to the fact, within a cell, only one of the two X chromosomes is activated during the life of that cell.. If it is the healthy one, the abnormal one is effectively silenced (copy number variations) (lionization phenomenon), but if is not, desease could came after. Stevenson at al 1990.Johnston 2007 has made a detailed analysis of this topic. Zarrei *et al.,* 2018, found de novo inherited genetic (CNVs) in the hemiplegic form of CP. Interestingly, after they divided patients in two categories, those with identified CNV risk and second without a CNV risk, there were significantly more females in CNV risk group (p=0,006). Abou-Jamra *et al.,* 2001, identified one mutation in each of three genes encoding adaptor protein complex 4 (AP4) subunits: nonsense mutation in AP4S1, frameshit mutation in AP4B1 and splice mutations in AP4E1 in progressive spastic paraplegia. Tuyusuz *et al.,* have found mutations in adaptor protein complex-4 (AP4) and in all four subunits AP4 (AP4M1, AP4E1, AP4S1 and AP4B1) in spastic tetraplegia, (a difference between hereditary and CP is the last is nonprogressive clinical outcome). Parolin-Schnekenberg *et al.* 2015, have found de novo mutations in three different genes KCNC3, ITPR1 and SPTBN2 in ataxic CP which were associated with increased paternal age. On the end, the last data, Corbett *et al.,* December 2108, have found the next in 184 patients: deletions of 2p25.3, Xp and 22q11. Then, two CP female from previous study in50 patients, with partial chromosome X monosomy and in this studyone new female with Xp monosomy and the terminal 1q43q44 trisomy due to translocation t(X;1), (P11.22;q43). De novo two CNVsa 16p11.2-p12.2 deletion and 1q21.1 duplication., de novo micro- deletion at 3p22.3 only one gene PDCD6IP (ALIX). There are variants of uncertain significance, five deletions and nine dupliactionsbut authors predict that six of them have potential to be involved inCP 6p22.3, 9p24.3-p24-2, 9q34.1, 10q22.1-q22.2, 19p13.3 and Xq22.1.

Accordingly, the first author in his Master Thesis 1990, by the quantitative analysis of digito palamar dermatoglyphics has found in 208 (122 males and 86 females cerebral palsy patients and 400 controls (200 males and 200 females), 13 statistically significant differences to control in males and only 2 in females, in triple analysis according to sex, heavines of injury, and clinical form of injury. For females between triradius b-c on right palm in decreased number of epidermal ridges in group medium heavy and heavy injury, but not to control, and in paraparetic female CP children between triradius b-c right palm in decreasing number of epidermal ridges too. That is in harmony with above mentioned, greater risk contracted to CP males children. Detailed presentation of digito palmar dermatoglyphic traits in CP female children, by the quantitative analysis, is listed in our previous paper (Cvjetičanin *et al.,* 2016). The new one, which is not metioned until now in our analyses, is the Simsek's *et al.,* article 1998, in which in 28 boys and 17 girls, they have found the decreasing number of epidermal total ridge counts (on all ten fingers), in girls (0,001) and increased atd angles (0,001) in girls too, which is not comparable to the first author findigns and in this research also.

Consequently, in this analysis there are to many positive variables, fifteen in CP girls, in the sense of increasing number of epidermal ridges, just opposed to previous all researches to date. Why the dermatoglyphic analysis, as a one of genetic method, is useful in genetic research? The first of all, it is cheap and nonagressive. Then, identical twins have the identical DNA, but their dermatoglyphic traits are never the same. Because of that, there is a room to find out environmental factors, The CP-study MOBAND aims to chart several of them, which might increase the risk of CP, e.g. parent's weight, diet during pregnancy, smoking and caffeine-intake, infections during pregnancy and premature birth, racial affiliation (Durkin 2015) (Maenner *et al.,* 2012), (Wu *et al.,* 2011) and it seems important factor is education status of parents.

Conclusion

Why so many genetic information has put it in this article? Because sooner or later it will come true that, CP has genetic etiology, others factor are only precipitating one. Second, the main goal is prevention. Namely, there is a strong correlation between brain and fingerprints. Stillborn babies who had no brains did not have fingerprints either, or damaged brain, have changes in dermatoglyphic traits, because of the common ectoder- mal origin of both system (Relation between Human Brain and fingerprints, 2014). The most poverful gene in the fourth chromosome, SMARCAD1, who deletes the dermatoglyphic drawing is the next chalange (Cvjetičanin 2014). The inheritance of dermatoglyphics include activity of several major with multitude of modifying genes plus outside factors. It seems quite likely, that the polygenic system with a small additive action of each of the genes in the development of dermatoglyphic pattern, is identical to the polygenic system or loci for CP.

Of particular interest are de novo mutatinos, copy number variants which were inherited from a healthy parent suggesting another genetic or environmental contribution which maid be discovered by dermatoglyphic research, namely as we mentioned above, that identical twins have the identical DNA, but never the same dermatoglyphic drawing (the question of environmental contribution factors). However, prevention starts by family data, and genetic characteristics of CP, not on the end when it came. Thus, it should be point out again, that earliest possible postnatal detection of risk group by use of dermatoglyphics pattern in CP should be immediately followed by medical exercises. These exercises should be iniated not later than ninemonth of life, as this period of growth is characterized by brainplatsticity, when the damage of brain can still be corrected andrendered clinically unperceivable, (M. Stojčević-Polovina 1978, Disertation, Zagreb).

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Ethics

There is not any danger for the patient from this kind of research. Dermatoglyphic analysis, which is one of genetic method, is without any harmful conscequence for sick persons. The procedure are in accordance with ethical standards inthe scientific research at Croatia Medical Association's Codex of Medical Ethic and Deontology, and Helsinky Declaration of World Medical Association, Edinburg, 2000.

Conflicts of interest: There is no conflicts of interes among the authors at all.

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