OVIDIUS UNIVERSITY CONSTANȚA

DOCTORAL SCHOOL OF APPLIED SCIENCES

Fundamental Field: Biology / Biochemistry

PhD THESIS

SUMMARY

STUDY OF BIOCHEMICAL BLOOD MARKERS VARIATION IN PATIENTS DIAGNOSED WITH PSORIASIS AND TREATED WITH HERBAL EXTRACTS

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ABBREVIATIONS AND SYMBOLS

ACTH.....Adrenocorticotropic hormone
A/G.....Albumin / globulin ratio
ALB.....Albumin
Albumin.....Blood Albumin
ALP.....Alkaline phosphatase
Alpha 1.....α l globulins
Alpha 2.....α 2 globulins
ALT.....Alanine aminotransferase
AMY7.....Blood amylase
Area / cm².....The total area of skin lesions
approx. .....Approximate
AST.....Aspartate aminotransferase
ATS.....Atherosclerosis
Beta.....β globulins
BMI..... Body Mass Index
° C.....Celsius degrees
CA.....Calcemia
Ion.CA.....Ionized Calcium
CAVI.....Heart ankle vascular index
CHOL.....Cholesterol
CK.....Creatine chinase
cm.....Centimetre
Compl C3.....Serum complement C3
Compl C4.....Serum complement C4
CRP.....C reactive protein
CR-S.....Blood creatinine
div.....Division
dl.....Decilitre
DLQI.....Dermatology Life Quality Index
D-ROM.....The reactive oxygen metabolites
DZ.....Diabetes mellitus
ESR.....The erythrocyte sedimentation rate
Exp.....Experiment
Rheum. Fct.....Rheumatoid factor
FE.....Sideremia
FORM.....Free Oxygen Radicals Monitor
Free T4.....Free thyroxine
Gamma.....γ globulin
GGT..... Gamma glutamyl transferase
GLU.....Blood sugar (blood glucose)
Mol. wei.....Molecular weight
HDLD.....High density lipoprotein colesterol
HIV.....Human Immunodeficiency Virus
HLA.....Human leukocyte antigen
HLA-Cw6.....Human Leukocyte Antigen Cw6
IFN.....Interferon
IL.....Interleukin
I.U. .....Insulin Units
K.....Potasemia
LDL.C0I.....Low-density lipoprotein colesterol
LD-P.....Lactate dehydrogenase
mg.....Miligram
ml.....Mililitre
MG.....Magnesemia
min.....Minute
mm.....Milimetre
NA.....Natremia
nm.....Nanometre
Pt. Nr.....Patient number
NSAIDs.....Nonsteroidal anti-inflammatory drugs
PASI.....Psoriasis Area and Severity Index
PGA.....Prostaglandin A
PGB.....Prostaglandin B
PGE.....Prostaglandin E
PGF.....Prostaglandin F
PGE1.....Prostaglandin E1
PGE2.....Prostaglandin E2
PGF2.....Prostaglandin F2
PHS.....Phosphoremia
C reac. Prot.....C reactive protein
Tot. Prot.....Total proteins
PTH.....Parathyroid hormone
PUVA.....Psoralen Combined with Ultraviolet A
Free Rad.....Free Radicals
At. Risk.....Atherogenic Risk
Rx.....Radiography
Sc.....Stresclin derma
Sc+Kh.....Stresclin derma + Klinhaem
Serotonin.....Serum Serotonin
MS.....Metabolic syndrome
Tb.....Tablets
TBIL.....Total bilirubin
TG-B.....Triglycerides
THF.....Tetrahydrofuran
TNFα.....Tumor necrosis factor α
TP.....Total proteins
TSH.....Thyroid stimulating hormone
U.....Unit
UREA.....Blood urea
URIC.....Uric acid
UVA.....type A Ultraviolet
UVB.....type B Ultraviolet
VEGF.....Vascular endothelial growth factor
µm.....Micrometre
♂.....Male
♀.....Female

TABLE OF CONTENTS

Study of biochemical blood markers variation in patients diagnosed with psoriasis and treated with herbal extracts

OBJECTIVES AND PURPOSE OF THE STUDY

Existing data in the specialty literature points out that the complex pathology of psoriasis is not yet fully known.

The main objective of this research theme was to determine some biochemical parameters leading to the determination of new correlations between biochemical substrate and the occurrence of the disease, the severity of psoriasis symptoms, correlations to support and contribute to the key objectives of the therapeutic strategy of psoriasis.

The proposed objectives were subordinated to this main objective and consist of widening the current database of biochemical imbalances highlighted in psoriasis and the biochemical changes occurred after a period of treatment.

Approaching the biochemical study of diseases etiopathogenesis in general and chronic dermatological diseases especially, is the prerequisite of better understanding the mechanisms of diseases setting and support. Whatever the etiological factors incriminated (primordial and triggers), clinical changes and paraclinical changes of the disease still occur through biochemical mechanisms.

Equally important is the assessment of biochemical changes occurring during and due to treatments applied. Predictable findings of changes in biochemical parameters during and after treatments, must be supported by thorough studies which demonstrate adverse reactions predicting and intra- and extra-therapeutic complications.

The purpose of this paper is to highlight the beneficial therapeutic effects of administration of plant extracts in patients with psoriasis, in correlation with the change in blood biochemical markers. However, by analyzing these parameters, it is proposed to refer the therapeutic contraindications, the diet and the evolution of biological constants during and after the action of administered therapeutic products in conjunction with any diseases associated to treated disease.

This paper presents five experiments:

- an experiment of minimal sampling and skin biopsies technical processing;
- an experiment of electro microscopic observations on the lysosomes involvement in psoriasis pathogenesis;
- three therapy experiments using extracts of plants.

I tried to apply cheap laboratory techniques and commended cheap therapeutic products, with a certain reliability of the results, respectively minimal side effects.

I was fascinated with the details obtained by electron microscopy. I looked very carefully at the changes through these details, by comparison with similar healthy skin tissue images obtained from the electron microscopy examination. I had the revelation of highlighting lysosomal changes, of which I have selected suggestive images.

Encouraging results led me to look for correlations between these biochemical parameters, research which will complete with interesting findings, with practical applications to improve the prognosis and life of patients with psoriasis.

This work fits into the overall effort to investigate diseases with complex etiology, difficult to discern entirely, aimed to highlight effective therapies with minimized side effects.

PART II. PERSONAL CONTRIBUTIONS

INTRODUCTION

I studied the etiopathogenesis and the treatment of severe psoriasis since the early years of medical school, motivated by the fact that the family had such a case, but also by the challenge of the complexity of this entity in human dermatological pathology. The involvement of genetic mechanisms, with the possibility of hereditary transmitting and predisposition to develop one or more forms of psoriasis, leads me to understand the fears of patients and psychological associated changes.

Individual and social behavior constrains the lifestyle of patients with psoriasis who should avoid triggers: stress (mental, physical), streptococcal infections of the upper airways, skin trauma, smoking, consumption of central nervous system stimulants (alcohol , coffee, cocoa, tea, energy drinks and food), consumption of NSAIDs and steroids, use of propranolol, etc. That in assessing the development of psoriasis the term "cured" is not used, but "lesions were bleached", gives the patient an unpleasant prospect, affecting wishes and future plans.

As a dermatological specialist (primary) , I tried to recommend to psoriasis patients the most effective treatments, in relation to lowest adverse effects.

Following favorable therapeutic observations made after administration of two oral preparations, containing plant extracts, I decided to objective the changes in the values of biochemical constants after six weeks of treatment.

CHAPTER 4

4. MATERIALS AND METHODS

To conduct the 5 experiments, we used a variety of materials and methods, some common for at least two experiments, others for each experiment separately only. For this reason, I will describe them differentiated for each experiment.

To perform a detailed analysis of the issues noted in each experiment, we continued the study by grouping the results in tables and enroll the results in different charts. Finally, we performed a series of statistical tests, highlighting, or denying the existence of correlations on variation of analyzed parameters: before and after treatment, between them, or in connection with the development of skin lesions under treatment and possible side effects.

Statistical tests used were as follows:

1. For statistical processing of data on pre-treatment / post-treatment differences, we used the software IBM SPSS 19.

- I checked in the first instance whether the differences between the pre-treatment and post-treatment values shows a normal data distribution, using the Kolmogorov-Smirnov and Shapiro-Wilk statistical tests. Also, I checked and the possible existence of "outliers" (extreme individual values, very different from the others obtained in the group), which could then influence the choice of optimal method to further analysis of data.

- If after the test mentioned above, resulted in a normal distribution of data and lack of significant "outliers", I then applied the parametric test "Student Paired Samples T-test", to see if differences between the pre-test and post-test are statistically significant. Otherwise, when either data distribution was not normal, whether there were "outliers", I applied a non-parametric alternative test named "The Paired-sample Wilcoxon Signed Rank Test".

2. For statistical processing of data to identify significant correlations between obtained values for different markers, I used the software SOFA Statistics. The test was used to calculate the Spearman correlation coefficient.

4.1. Experiment 1.

Contributions to improve the technique of sampling and preparation of the skin biopsies

(Nelu-Doru Popescu, Emma Gheorghe, Natalia Roşoiu) (The experiment was published in **Archives of the Balkan Medical Union**, **50**, 1, 38-44, **(2015)**).

A skin fragment with the diameter of approx. 1 mm was harvested, then submitted to a rapid, adapted and improved processing technique.

An algorithm of working steps was elaborated, inspired by the technique used in histology and histo-pathology laboratories of the Faculty of Medicine Constanța, respectively Military Emergency Hospital Constanța:

Preparation techniques of histology slides (common and *quick*)

Prelevation of the biopsy (1cm³ minimum, *respectively 1mm³*)

Cover Sheet: Name, sex, age, personal identification number, dg., date.

FIXATION – room temp. formalin 37% one part to 4 parts of water. Fixation time = 24-72 hours.

- At 40 ° C with 37% formalin, 1 part to 3 parts of water with stirring. Fixation time = 5 min.

PARTS ORIENTATION

DEHYDRATION : ethyl alcohol 70 ° - **1-2 hours ...at 40 ° C.., with stirring, for 1 min.**
ethyl alcohol 80 ° - **1 hour .....at 40 ° C, with stirring, for 1 min**.
ethyl alcohol 90 ° - **1 hour ……at 40 ° C, with stirring, for 1 min**.
ethyl alcohol 96 ° - **1 hour .....at 40 ° C, with stirring, for 1 min.**
ethyl alcohol 100 ° - **1 hour ..... at 40 ° C, with stirring, for 3 minutes.**
ethyl alcohol 100 ° - 1 hour
ethyl alcohol 100 ° - 1 hour

- **CLARIFICATION :** ... .toluene (xylene) ... - ... 1 hour at 40 ° C, with stirring, for 3 minutes.
toluene (xylene) ... - …1 hour
toluene (xylene) ... -… 1 hour
- **PARAFFINING :**….. paraffin 56-58 ° C ..- 1.30 hours. ***At 57,5-58ºC, stirred for 5 min.***
…..paraffin 56-58 ° C ..- 2.50 hours
- **SLICING to 5µ**
- **SCOPE SECTIONS ON GLASS SLIDES**

Mayer albumen: egg albumen + anhydrous glycerol 1/1+ 1 thymol crystal.
The blades are coated with albumen Mayer.
Slides placed on the plate at 52 ° C oiled side up.
Spot 1 ml distilled water slide .....0,5 ml
Place the sectioned tape
Drain the water
Thermostatic 1 hour at 52°C ***……at 52ºC for 1 minute.***

- STAINING ……pipetting and blotting absorption

- **DEWAXING .....…….toluene (xylene) 5 min ... *52ºC for 1 min*.**
toluene (xylene) 5 min
toluene (xylene) 5 min
- **DEHYDRATION**. ethyl alcohol 100 ° for 5 minutes …..***at 52ºC for 15-20 sec.***
ethyl alcohol 100 ° for 5 minutes
ethyl alcohol 96° for 5 minutes …..***at 52ºC for 15-20 sec.***
ethyl alcohol 96° for 5 minutes
ethyl alcohol 90° **..** ***at 52ºC for 15-20 sec.***
ethyl alcohol 80 °**..** ***at 52ºC for 15-20 sec.***
ethyl alcohol 70 °**..** ***at 52ºC for 15-20 sec.***
- **WASH** …… tap water for 5 minutes …… ***at 52ºC for 1 min***
tap water for 5 minutes
- STAINING ……..... Hemalaun 5-10 min…..…. ***at 52ºC for 30 sec.***
- **WASH** …… tap water for 10 min..... ***at 52ºC for 1 min***.
- STAINING ……...….Eosin .....2-5 min……...***at 52ºC for 20 sec.***
- **WASH** …….. tap water ……..… ***at 52ºC for 1 min.***
- **DEHYDRATION**.. ***ethyl alcohol 70°***..... ***at 52ºC for 15-20 sec***
ethyl alcohol 80°… **.** ***at 52ºC for 15-20 sec***
ethyl alcohol 90°**…. .** ***at 52ºC for 15-20 sec***
ethyl alcohol 96 ° for 5 min …. ***at 52ºC for 15-20 sec***
ethyl alcohol 100°for 5 min.. . ***at 52ºC for 15-20 sec***
ethyl alcohol 100°for 5 min
- **CLARIFICATION** ……..... toluene (xylene) 5 min ..... ***at 52ºC for 1.5 min.***
Toluene (xylene) 5 min
TOTAL: approx. 17 hours…….**TOTAL: approx. 40 minutes**

Mounting between the blade and midsole with **entelan** (synthetic resin), as replacement of canada balsam.

REQUIRED MATERIALS:

Figure 8. Materials used are: graduated test tubes, pipettes and alcohol measuring instrument for preparation of various concentrations of alcohol, stand for test tubes, electronic thermometer, 1.2 diameter syringe needles, magnifiers, artisanal crafted microtome, heater container with thermostat, glass containers for solvents, blotting paper..

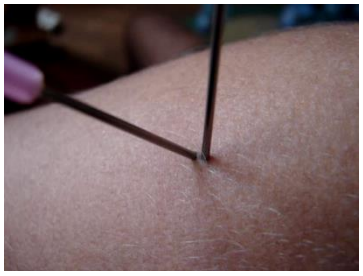


Figure 9. The sampling biopsy

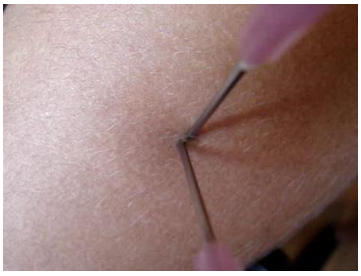
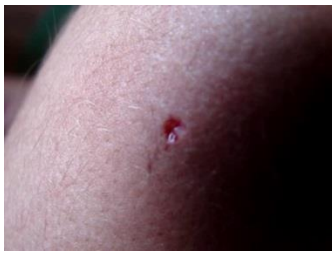


Figure 10. Tilting incision needles

Skin biopsy may be performed with two needles type 18 G 1 ½, with a thickness of 1.2 mm, a diameter of 1.2 mm, and a length of 40 mm. By puncturing the skin at an angle of 45 ° and close to the two bevels, there is a millimeter cube tissue section with minimal bleeding.

Figure 11



After sampling, the sample retracts approx. 40-50% and the remaining wound increases its diameter approximately the same proportion.

Figure 12

In order to be manipulated, the skin fragment is perforated in hypodermic portion with an atraumatic surgical needle No.6(fig 13) .

Figure 13

Figure 14

The immobilized sample in the needle is inserted for "fixing" in a test tube with 10% formalin, and then maintained at 40 ° C for 5 minutes or kept at room temperature for a few hours until the next steps continue (fig 14).

Figure 15

The initial dehydration is performed by successive bathing and stirring for 1 minute in test tubes with increasing alcohol concentrations: 70 °, 80 °, 90 °, 96º and for 3 minutes alcohol 100 ° (absolute ethanol).

Figure 16

The next step is clarification in toluene for 3 minutes with stirring at 40 ° C

Figure 17

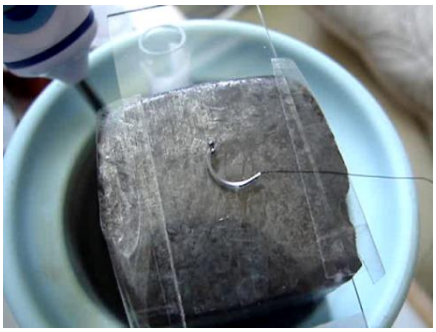


Figure 18

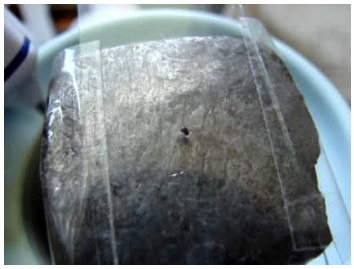


Figure 19

After maintaining the sample for 5 minutes in a test tube with liquid wax at 58ºC-57,5ºC temperature,it is deposited on a glass slide maintained at the same temperature.

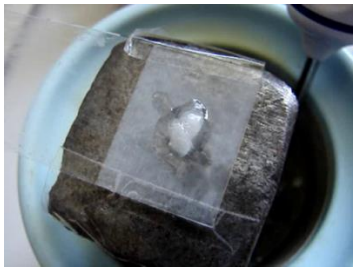


Figure 20

To create a direct paraffin small block on a glass slide, the sample is placed on the mat portion, oriented with the skin perpendicular to the slide surface and then coated with a wax shell, which is kept at the same temperature a few seconds to complete enclosing.

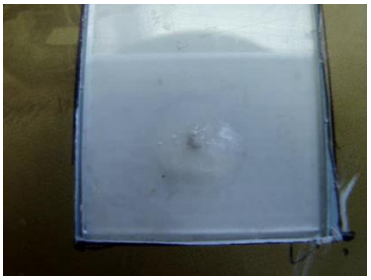


Figure 21

Then insert the blade into the slot with the same size and shape of the crafted microtome holder.

Figure 22 Paraffin small block is sectioned with the microtome blade held at an angle of approximately 45 °. Even though the wax grip of the mat glass portion provides an increased resistance of piece fixation, to facilitate severing, print forward movement along with light laterality movements.

5µ thick sections is provided by a number of 12 gauges made of tracing paper (which have this thickness), cut into rectangles with 7/10 cm size. Inner gauges are cut square the same size as the width of the glass slide.

A slide glass is prepared by lubrication with Mayer albumin and maintained at 52ºC throughout the staining. After dropping 0.5 ml of distilled water, resulted section are placed. Absorb water with blotting paper, and after two minutes it continues on the same slide staining stages:

- Dewaxing 0.5 ml of toluene for 1 minute
- Moisture with 0.5 ml of alcohol in decreasing concentrations: 100 °, 96º, 90 °, 80 °, 70 °, and then 1 ml of tap water for 15-20 seconds each time, the liquid is absorbed by the blotting paper.
- Hemalaun staining for 30 seconds.
- Wash with 1 ml tap water for 1 minute.
- Eosin staining 15 seconds.
- Wash with 1 ml tap water for 1 minute.
- Dehydration with 0.5 ml of alcohol with increasing concentrations: 70 °, 80 °, 90 °, 96º, 100 °for 15-20 seconds each..
- Clarification 1ml toluene for 1.5 minutes.

Mounting between slide and lamella with ENTELAN

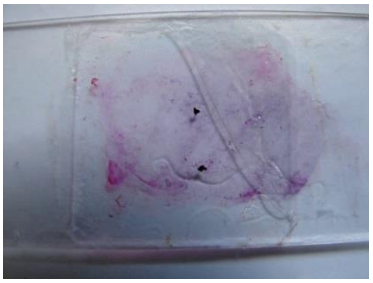


Figure 23 The final aspect of the blade with two skin biopsy sections

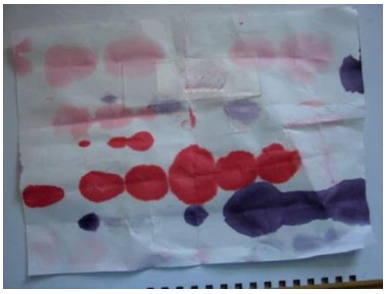


Figure 24 Appearance of blotting paper after successive absorption of fluids from the blade

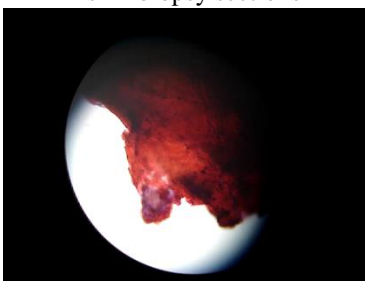


Figure 25

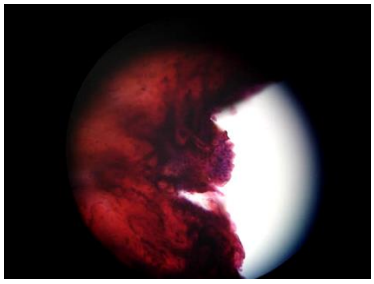


Figure 26

Microscopic aspects of the two prepared sections, photographed directly through the eyepiece, using a 40 X objective.

4.2. Experiment 2.

Lysosomes changes highlighted in psoriasis vulgaris, electron microscopy

(Nelu-Doru Popescu, Victor Ciupină, Gabriel Prodan, Stela Zamfirescu, Emma Gheorghe, Constanţa Ştefanov, Natalia Roşoiu)

(the paper was published in **Archives of the Balkan Medical Union, 50**, 4, 523-525, **(2015)**)

For the second experiment, I sampled biopsy using the same minimally invasive technique. Three fragments of approx.1 mm diameter of psoriasis lesions were taken from two patients before treatment.

SAMPLE PREPARATION.

Modified Jastrow method was used for sample preparation:

- Immersion of the samples in 2.5% glutaraldehyde solution buffered with 2% paraformaldehyde in 0.1 M sodiumphosphate buffer (Sörensenbuffer) pH 7.2 - 7.4;
- Keeping the samples overnight at 4 ° C;
- Washing 3x15 min. In 0.1 M sodium phosphate Sorensen +0.1M sucrose;
- Postfixation 90 minutes in 2% osmium in THF, pH 7.4, at + 4 ° C;
- Wash dec3x15 min. In sodium phosphate buffer 0.1M (Sorensen), pH 7.4;
- Dehydration 2x15 min. With 50% of acetone (distilled water);
- Contrasting overnight in 70% acetone + 0.5% uranyl acetate + 1% phosphotungstic acid at + 4 ° C.

DEHYDRATION

- 2 x 15 min. with 80%.acetone; - 2 x 15 min. with 90% acetone; - 2 x 15 min. with 96%.acetone;
- 3 x 20 min. with 100% acetone; - 2 x 15 min. with propylene;
- 30 min 2: 1 mixture of propylene oxide Epon; - 30 min 1: 1 mixture of propylene oxide Epon;
- 30 min 1: 2 propylene mixture Epon; - Impregnation with Epon overnight at 4 ° C;
- Taking fresh sample and placement in Epon; - Incubation for 48 hours at 60 ° C for polymerization;
- Slicing 50-100 nm; - Washing sections.

POSTCONTRAST

- 10 min. in the sol. 8% uranyl acetate;- 5 min. in the sol. 0.7% lead citrate + 0.9% sodium citrate.
- Drying of the grid 15 min. and examination.

The electron microscopy device used to obtain photographic images is a transmission electron microscope type CM 120 produced by Philips and FEI, Eindhoven, Netherlands, purchased by Univ. Ovidius in 1998.

Essential technical features: power resolution 210-10 m, beach increments: 35-1200 000, diffraction: 18-4300 mm, ultra-high vacuum and crimioscopie options. As an accessory, we used the device IMV UPS D-31model 15kV for protection in case of power failure in the electric network, acquired by the University Ovidius in 2000.

Using the prepared sections we made a number of 55 images of semi-fine sections and 34 images with a transmission electron microscope, with a magnification of x 610,000. Of these, to demonstrate observations, I selected a number of 6 photographs, which we will study in the next chapter.

4.3. Experiment 3

Clinical, biochemical and therapeutic outcome of a patient with psoriasis arthropathy, tumor history and insulin-dependent diabetes.

(Nelu-Doru Popescu, Mihaela Bașa, Emma Gheorghe, Natalia Roșoiu)

(paper was published in **Archives of the Balkan Medical Union, 49**, 4, 534-537, **(2014)**)

For the third experiment, biochemical, immunological analyzes were performed and total free radical testing, from a patient with arthropathic psoriasis associated with insulin-dependent diabetes and a history of malignant tumor, then began treatment with immuno-modulating and antioxidant preparations Tinefcon and Omega 3 cps., administered by general route, and locally Daivobet gel for 7 days. At the end of the set, repeated laboratory investigations differ from the reference values from the first sample. There were clinical and laboratory assessments of disease progression under treatment.

We determined the total amount of free radicals in the peripheral blood, with FORM Free Oxygen Radicals Monitor device (Full system for immediate determination of free radicals in whole blood).

After 7 days of treatment, determination was repeated, and we made comparison with initial values, in conjunction with clinical symptoms.

4.4. Experiment 4

Correlations between changes in biochemical values of constants and evolution of skin lesions, two cases of psoriasis vulgaris treated systemically with two herbals

(Nelu-Doru Popescu, Victor Ciupină, Emma Gheorghe, Mihaela Bașa, Natalia Roșoiu) (paper was published in **Archives of the Balkan Medical Union, 50**, 4, 518- 522, **(2015)**)

We randomly selected two cases of psoriasis vulgaris, both females, 48 years old and respectively 51 years old. Both cases have been blood and urine sampled before treatment and after 6 weeks of treatment, comparing biochemical values, hormones values, serotonin (neurotransmitter) and quantitative assay of free radicals (oxidative stress evaluation).

Figure 34 Psoriasis vulgaris scalp

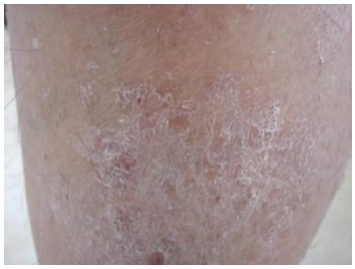


Figure 35 Psoriazis vulgaris knee

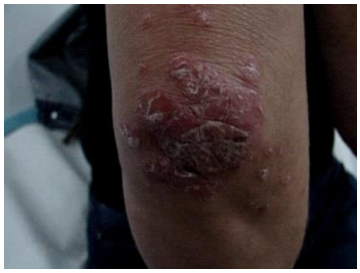


Figure 36 Psoriasis vulgaris elbow

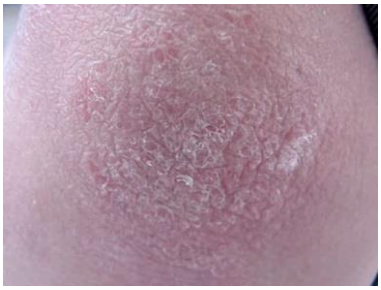


Figure 37 Psoriazis vulgaris knee

Patients received treatment with two pharmaceutical products including herbal extracts in the following amounts, with the main specified actions:

D-ROM test measures oxidizing ability in the plasma sample by spectrophotometry, this method mainly shows the free radicals derived from hydroperoxides.

In healthy subjects, D-ROM has a value between 250 and 300 units CARR (U CARR). Values above 300 U CARR indicate a state of oxidative stress.

Electron microscopy

Microbiopsy were collected from the skin lesions, which were processed according to the procedure preparations for electron microscopy. There were 34 images obtained, which were interpreted for appreciation of the cells ultrastructure changes.

4.5. Experiment 5.

Study of blood biochemical markers changes in patients diagnosed with psoriasis and treated with herbal extracts

(Nelu-Doru Popescu, Emma Gheorghe, Mihaela Bașa, Radu Dumitru Roșoiu, Natalia Roșoiu)

(The experiment is pending for publish in ISI rated journals)

The experiment targeted variation of a number of 43 biological parameters (biochemical and hormonal) from two groups consisting 7 patients each, with psoriasis vulgaris, treated with two natural therapeutic products specified in experiment 4:

- The first group with Stresclinderma capsules (two capsules/day) and Klinhaem syrup (two tablespoons/day) for 6 weeks.
- The second group, only with Stresclinderma capsules (two capsules a day) for 6 weeks.

Laboratory equipment was the same and it is specified at experiments 3 and 4.

Blood samples were taken before and after treatment, after which, we performed comparative studies. The method used was the calculation of the average reference intervals for individual investigations, calculating average values of analyzis for each studied group and then comparing them.

Reference ranges for normal laboratory test values were entered in a table, to which we added their average values, then used as the basis, to compare the values obtained from patients included in the study.

For the statistical processing of the data obtained after performing laboratory investigations, we have achieved the tables containing the test values before treatment and after 6 weeks of treatment.

CHAPTER 5 RESULTS AND DISCUSSION

5.1. Experiment 1.

Contributions to improve the technique of sampling and preparation of the skin biopsies

Results

The experiment succeeded in demonstrating that because of the shrinkage of skin biopsy specimens, the technique of preparation of histological slides can be improved, meaning shortening the working time by approx. 40 minutes.

Histopathological examination is a requirement for diagnosis of dermatological disorders with atypical or difficult to interpret manifestations.

Typically, the minimum time to process biological samples for histo-pathological diagnosis, is at least 7-10 days. Respecting known phases of preparation, but drastically shortening times of sample biopsy from fixing, dehydration, clarification and parafining we managed shortening the total time to approx. 40 minutes. This is possible because of the small size of the biopsy material of about 1 mm cube.

Discussions

Manual technic is valuable if the positive aspects are compared to negative aspects thus:

Negative aspects:

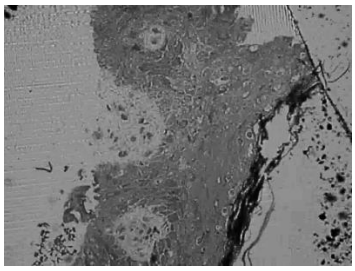
- In the working time can be processed only one histological sample.
- Risk of no eloquent sample prelevation for investigated lesion.
- Consideration should be given great attention and skill to both sampling and the processing, because the risk of leakage of small sample size, particularly in the cutting of the paraffin small block, and staining on the blade.
- From a sample can be made few sections, and you cannot keep the outstanding block for possible reanalysis.
- The cost of investigation may be greater than that of the standard.

Positive aspects:

- ice extemporaneous histological examination can be replaced with an examination of biological product hematoxylin-eosin stained.
- Biopsy of a skin lesion reduces the risk of possible tumor cell dissemination during short time (40 minutes) to the histo-pathologically diagnosis.
- There may be a rapid therapeutic decision to the amplitude of surgery decisions.
- The risk of developing infectious or aesthetic complications is minimal, because of the very small size sample taken.
- Sampling does not require local anesthesia, reducing the risk of allergic reactions to anesthesia.
- Due to reduced discomfort felt by the patient, more biopsy samples may be performed from various injuries eventually to follow the development of diseases in time.
- Investment cost for such a laboratory is much smaller than standard laboratory.
- All materials are easily transportable in a simple bag or briefcase.

5.2. Experiment 2.

Lysosomes changes highlighted in psoriasis vulgaris, electron microscopy



Results:

6 photos that I believe are representative:

Figure 48 Epidermal hyperplasia with parakeratosis and orthokeratosis

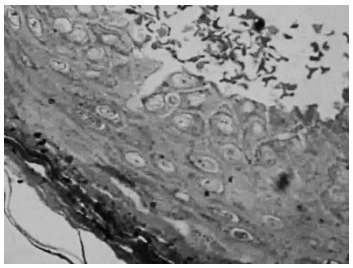


Figure 49 - Epidermal hyperplasia with parakeratosis and orthokeratosis

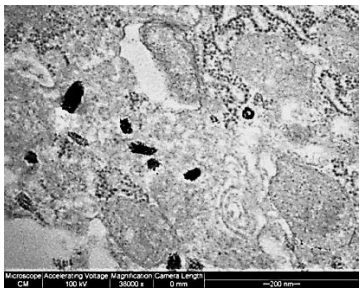


Figure 50 - Many lysosomes disseminated, some grouped into formations with irregular borders

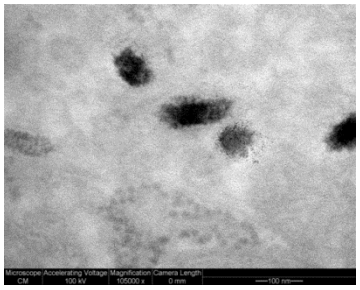


Figure 51 Edges of the lysosomal groups appear softer at a magnitude of x105.000.

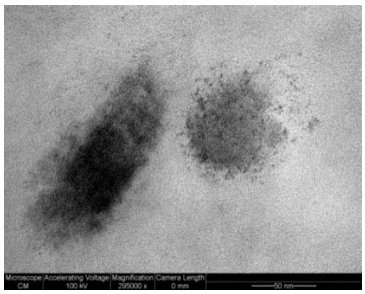


Figure 52 Blurring the lysosomal groups edges is evident at a magnitude of x 295,000.

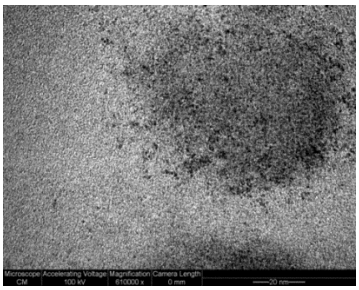


Figure 53 Magnitude of x 610.000. Lysosomal groups have the appearance of granular clouds, with diffuse edges, which are lost in the cell cytoplasm, proof of their dissolution.

Discussions

Lysosomes are organelles having size of 0.1-1.2 μm, the appearance of spherical droplets that contain more than 50 hydrolytic enzymes to lyse the biomolecules like lipids, proteins, nucleic acids, carbohydrates and cell debris. Lysosomes are responsible for cellular homeostasis, repair the plasma membrane, and cell energy metabolism.

By studying electron microscopy images obtained from processed biopsies of psoriasis lesions, we can obtain important information on the pathogenesis of this chronic disease.

We highlighted epithelial cells harvested from squamous plaques of the skin overloaded with lysosomes, and the association of lysosomes in groups in different stages of involution.

Continuing electron microscopic study and all other cellular organelles, we will bring concrete arguments to understanding the pathogenesis of psoriasis and histo-chemical changes that occur during the application of therapeutic procedures.

The experiment reveals lysosomal changes highlighted by electron microscopy in biopsy samples taken from two cases of psoriasis vulgaris.

Through detailed analysis of microscopic images, from semi-fine sections with order of size up to 320 x 610,000 images, stands apart, from the specific histo-pathological aspects of psoriasis vulgaris, the abundance of lysosomes in various stages of development.

5.3. Experiment 3

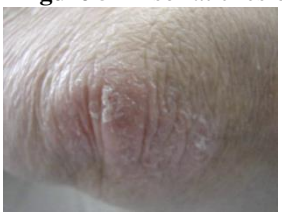
Clinical, biochemical and therapeutic outcome of a patient with psoriasis arthropathy, tumor history and insulin-dependent diabetes

Case study

Patient L.M., female, age 61, from Constanta, was diagnosed with psoriasis vulgaris in 1977, at the age of 25 years. Follows a regular dermatological treatment with favorable therapeutic results, but frequent relapses.

Psoriatic skin lesions, typically located in the projections limb joints (elbows, hands, knees) were photographed :

Figure 54 Psoriatic lesions of the elbow



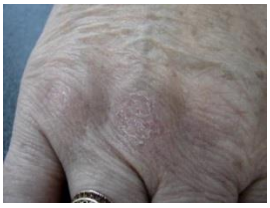


Figure 55 Psoriatic lesions of the hand

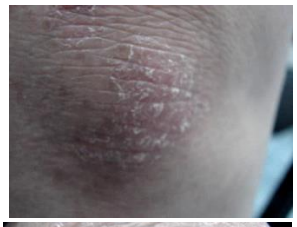


Figure 56 Psoriatic lesions of the knee

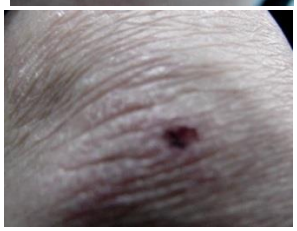
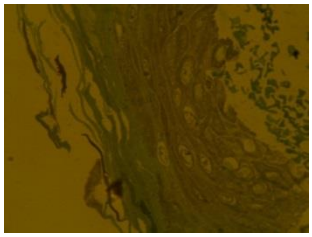


Figure 57 A small biopsy fragment (1.5 / 2 mm) was taken from the left knee. There was no need for sutures, due to the small size of the excised fragment, as seen in the picture above.



The skin biopsy fragment was prepared histopathological, sectioned and stained.

Figure 58 We can observe some main typical histological changes in psoriasis: acanthosis, parakeratosis, granular layer disappearance.

Medical history. In 1993, radical right mastectomy, subtotal hysterectomy, bilateral oophorectomy and sector left breast excision for: invasive ductal carcinoma, muciparous and glanduliform of the right breast (G3, pT2, pN2), 9 lymph nodes metastatic invaded fibroids, uterine fibromastosis and left cystic mastosis.

After surgery, the patient followed repeated courses of telecobalt therapy and chemotherapy.

In 2001 she was diagnosed with insulin-dependent diabetes.

In 2008 she was diagnosed with psoriatic arthropathy:- C5 - C6 discarthrosis, right sacroiliitis, and ankylosing spondylitis stage 3, for which he received treatment with methotrexate and anti-inflammatory drugs;

- Motor sensory diabetic polyneuropathy.

The biochemical tests revealed hypercholesterolemia (total serum cholesterol 272 mg / dl and LDL cholesterol 190 mg / dl) and triglycerides (137 mg / dl).

In 2009, bilaterally femoral neck osteogenic bone densitometry exam revealed a moderate risk of fracture.

In 2011 emphasizes symptoms and laboratory changes of psoriatic arthropathy (ESR 55 div / h, C-reactive protein positive +++. During the same year cervicectomy was performed (vaginal subtotal hysterectomy).

In 2012, C-reactive protein remains positive and ESR reached 73 div / h.

In 2013 she was hospitalized 3 times in the rheumatology department of Constanta County Emergency Hospital.

During the experiment, the patients were undergoing treatment with: Thiogamma 600mg-1 cp. / day, methotrexate 2.5 mg-5 cp. / week single dose taken, Gabaram 300mg-1-2 cp. / day, Plaquenil 200 mg-1-2 cp / day, Simvacard 20mg-1 cp / day, 30 by 22 I.U. insulin Mixtard in the morning and 24 I.U. in the evening, 10 I.U. of Actrapid Insulin in the noon (lunchtime).

Results

Before starting the treatment, a series of laboratory investigations was performed, which revealed that biochemical tests and immunological tests were normal, besides inflammatory tests (positive rheumatoid factor, ESR 20 div / h) and free radicals 361 U Carr.

After current combination therapy with Tinefcon 700 mg x 2 /day and omega 3 cps. 1000 mg x 2 / day for 7 days, we saw a slight accentuation of pain joint symptoms, which is consistent with increased ESR, the concentration of free radicals, glucose and lipid balance sheet values. Skin lesions showed no significant changes.

Biochemical tests have shown:

- Blood glucose levels increased from 103 mg / dl to 116 mg / dl.
- Persisting positive rheumatoid factor, C-reactive protein remains negative;
- ESR increases from 20 div/h to 26 div/h;
- Free radicals increase from 361 U Carr to 445 U Carr;

Even if it is within normal range, changes in lipid balance values are observed:

Total cholesterol increased from 172 to 188 mg / dl;

Triglycerides increased from 88 to 128 mg / dl;

LDL-cholesterol increased from 94 to 103mg / dl;

Total lipids increased from 565 to 641 mg / dl;

Atherogenic risk increases from 2.9 to 3.2.

Over the next five weeks, clinical symptoms and laboratory parameters values were gradually improved, demonstrating the favorable treatment.

DISCUSSION

The study focused on the association, at the basic treatment of a patient with psoriasis arthropathy and metabolic disorders, of two preparations in the category of dietary supplements: Tinefcon aimed at inhibiting TNF-α, associated with Omega 3, as an antioxidant.

After 7 days of treatment combination was a slight worsening of subjective symptoms (complaints joint pain) and certain laboratory parameters (blood glucose, ESR, free radicals, total cholesterol, triglycerides, LDL-cholesterol, total lipids, atherogenic).

In terms of the evolution of skin lesions, we can say that administered the combined treatment was successful. Side changes of raised blood parameters are temporary and do not significantly affect the overall condition of patients.

For this reason, it may be appropriate to continue treatment until complete healing.

5.4. Experiment 4

Correlations between changes in biochemical values of constants and evolution of skin lesions, two cases of psoriasis vulgaris treated systemically with two herbals

Results

Following the evaluation of the psoriasis etiopathology, we summarized that the onset and the maintenance of the disease are determined mainly by immunological factors, modulated by neurotransmitters released in stressful situations.

After 6 weeks of treatment, the specific lesions of psoriasis disappeared by 100%.

To try a correlation of the excellent therapeutic outcome with biochemical investigations values before and after treatment, we calculated the arithmetic mean of the reference range for each parameter analyzed. The differences between test results and the arithmetic mean calculated were assessed a percentage of the average values and then entered in graphic columns.

Analysis name	Reference Range	patient **I.C**.03.04.15	patient **I.C**.16.05.15	patient **R.S**.03.04.15	patient **R.S**.16.05.15
Free Rad.	250-300 U.Carr	229 U.Carr	310 U.Carr	550 U.Carr	501 U.Carr
A.C.T.H.	< 46 pg/ml 7.2-63.3 pg/ml	14.0 pg/ml -	- 17.41 pg/ml	8.83 pg/ml -	17.4 pg/ml -
PTH	11 – 67 pg/ml	24,8 pg/ml	25,9 pg/ml	11,8 pg/ml	16.8 pg/ml
Serotonin	40-200ug/l	167.20 ug/l	144.50 ug/l	64.95 ug/l	113.57 ug/l
Albumins	57.00-65.00 %	51.66 %	47 %	54.05 %	54.22 %
ALFA 1	1.0-4.0 %	3.5 %	2.2 %	3.6 %	3.8 %
ALFA 2	6.00-13.00 %	9.48 %	12.5 %	10 %	10.40 %
BETA	8.00-13.00 %	12.78 %	13.1 %	13.27 %	13.10 %
Gamma	12.50-21.00 %	22.62 %	25.2 %	13.06 %	18.47 %
Alb./glob.	1.2-1.5	1.1	0.89	1.2	1.2
Total protein	6.70-8.70 g %	7.13 g/dl	7.4 g/dl	7.01 g/dl	7.01 g/dl
Compl.C3	83-183 mg/dl	102 mg/dl	106 mg/dl	130 mg/dl	134 mg/dl
Compl.C4	15-57 mg/dl	30,5 mg/dl	30 mg/dl	37.6 mg/dl	37.5 mg/dl
Estradiol	<144 pg/ml 18.4-201pmol/l	86 pg/ml -	- 126.1 pmol/ml	<10 pg/ml -	<10 pg/ml -
Progesterone	<0.2 ng/ml 0.3-2.5 ng/ml	0.1 ng/ml -	- 3.02 ng/ml	0.1 ng/ml -	0.1 ng/ml -
S .Cortisol	3.7-19.4 ug/dl 171-536 nmol/l	7.4 ug/dl -	- 508.1nmol/l	11.4 ug/dl -	14.2 ug/dl -
T. cholesterol	100-200 mg/dl	191 mg/dl	175 mg/dl	237 mg/dl	232 mg/dl
C reactive protein	0.10-6.00 mg/l <0.5	1.44 mg/l -	- 0.20 mg/dl	1.60 mg/l -	2.01mg/dl -
Blood glucose	60-115 mg/dl	63.00 mg/dl	84 mg/dl	83 mg/dl	95.00 mg/dl
Triglycerides	50-150 mg/dl	94.00 mg/dl	127 mg/dl	104 mg/dl	80 mg/dl
Rheumatoid factor	<10 UI/ml	<8UI/ml	9UI/ml	<8 UI/ml	<8 UI/ml
Free T4	0.89-1.74ng/dl 10.6-22.7pmol/l	1.44 ng/dl -	- 15.1pmol/l	1.43 ng/dl -	1.47 ng/dl -
TSH	0.4 – 4 uUI/ml	0.988uUI/ml	1.46 uUI/ml	2.57uUI/ml	3.06 uUI/ml
Urine density	1015-1025 g/l	1025 g/l	1025 g/l	1020 g/l	1020 g/l
Urine pH.	5.0 - 8.0	5	5	6	6
Urinary sediment.	Rare cel.epitel.	1-15/hpf	1-15/hpf	Rare cel.epit.	Rare cel.epit.

Table 7 Values of biochemical analysis results

Analysis name	Reference Range	p.**I.C**.03.04.15		p. **I.C**.16.05.15		p.**R.S**.03.04.15		p.**R.S**.16.05.15	
		Dif.	%	Dif.	%	Dif.	%	Dif.	%
Free Rad.	*275*	*-46*	*-16,73*	*+35*	*+12,73*	*275*	*100*	*+226*	82,18
A.C.T.H.	23	-9	-39,13	-	-	-14,27	-62,4	-5,6	-24,35
	35,25	-	-	-17,84	-50,61	-	-	-	-
PTH	39	-14,20	-36,41	-13,10	-33,59	-27,20	-69,74	-22,2	-56,97
Serotonin	120	+47,2	39,33	+24,5	20,42	-55,05	-45,88	-6,43	-5,36
Albumins	61	-9,34	-15,31	-4	-6,56	-6,95	-11,39	-6,78	-11,11
ALFA 1	2,5	+1	40	-0,30	-12	+1,10	44	+1,40	56
ALFA 2	9,5	-0,02	-0,21	+3	31,58	+0,5	5,26	+0,90	9,47
BETA	10,5	+2,28	21,71	+2,60	24,76	+2,77	26,38	+2,60	24,76
Gamma	16,75	+5,87	35,04	+8,45	50,44	-3,69	-22,02	+1,72	10,27
Alb./glob.	1,35	-0,25	-18,52	-0,46	-34,07	-0,15	-11,11	-0,15	-11,11
T. protein	7,7	-0,57	-7,40	-0,3	-3,90	-0,69	-8,06	-0,69	-8,96
Compl.C3	133	-31	-23,31	-27	-20,30	-3	-2,26	+1	0,75
Compl.C4	36	-5,5	-15,28	-6	-16,67	+1,6	4,44	+1,5	4,17
Estradiol	72	+14	19,44	-	-	-62	-86,11	-62	-86,11
	109,7	-	-	16,4	14,95	-	-	-	-
Progesterone	0,1	0		-	-	0		0	
	1,4	-	-	+1,62	+1,62	115,71	-	-	-
S. cortisol	11,55	-4,15	-35,93	-	-	-0,15	-1,30	+2,65	22,94
	353,5	-	-	+154,6	43,73	-	-	-	-
Cholesterol	150	+41	27,33	+25	16,67	+87	58	+82	55
C rect. protein	3,09	-1,61	52,10	-	-	-1,45	-46,93	-1,04	-33,66
	0,25	-	-	-0,05	-20	-	-	-	-
Blood glucose	87,5	-24,5	-28	-3,5	-4	-4,5	-5,14	+7,5	8,57
Triglycerides	100	-6	-6	+27	27	+4	4	-20	-20
Rheumatoid fct.	5	-2	-40	-1	-20	-2	-40	-2	-40
Free T4	1,31	+0,13	9,92	-	-	+0,12	9,16	+0,16	12,21
	16,65	-	-	-1,55	9,31	-	-	-	-
TSH	2,2	-1,21	-55	-0,74	33,64	+0,37	16,82	+0,86	39,09
Urine density	1010	+15	1,49	+15	1,49	+10	0,99	+10	0,99
Urine pH.	6,5	-1,5	-23,08	-1,5	-23,08	-0,5	-7,69	-0,5	-7,69

Table 8 Weighted average results of biochemical tests

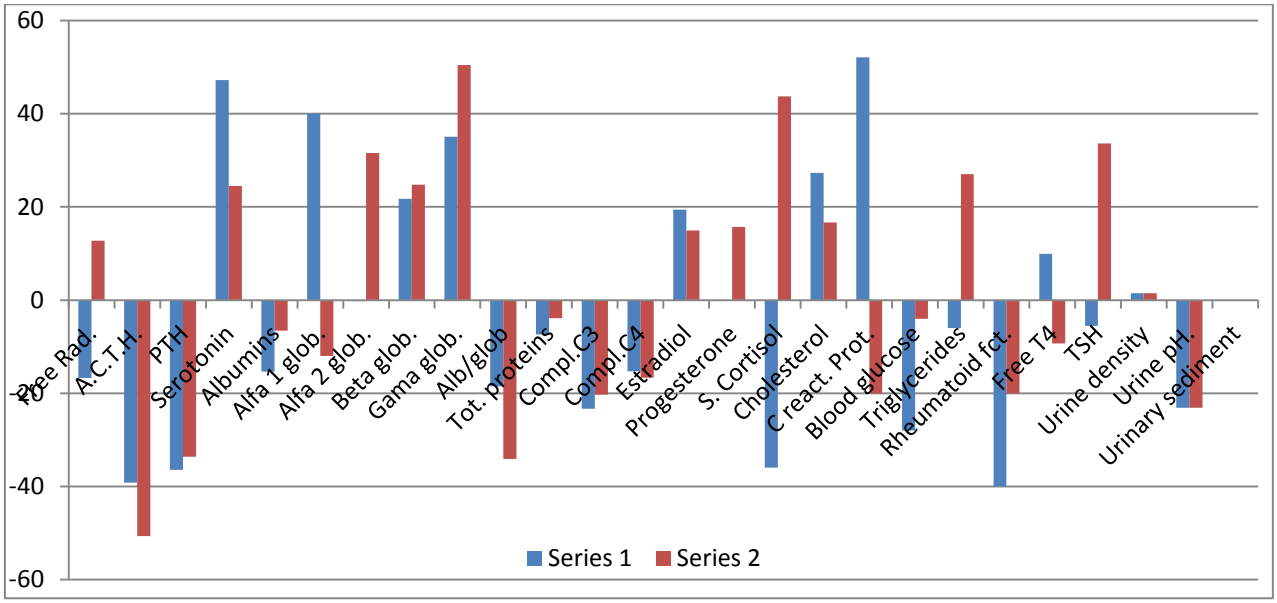


Figure 59 Variation of 26 biological parameters in patient **I.C.** after treatment

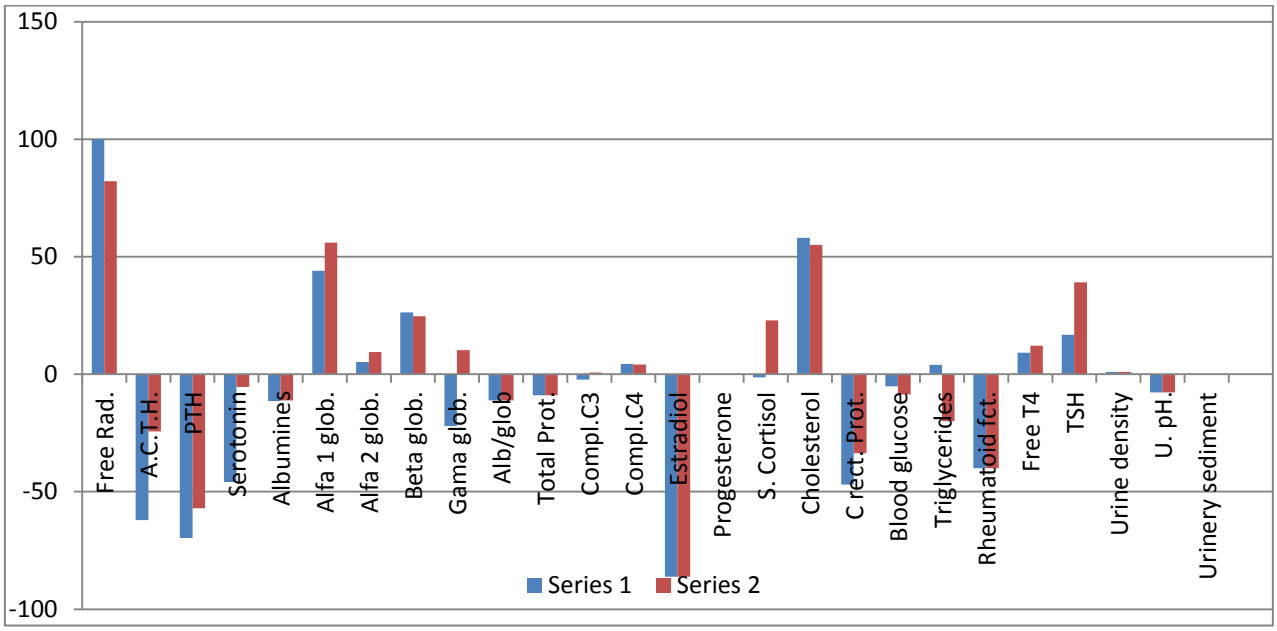


Figure 60 Variation of 26 biological parameters in patient **R.S**. after treatment

Discussions

Patient I.C.: the amount of free radicals was 21 U Carr below the minimum reference value before treatment (229 U Carr compared to 250 U Carr), , then values increased with 10 U Carr (310 U Carr compared to 300 U Carr) above the maximum reference after treatment.

Serum electrophoresis:

The amount of albumin was below the reference value in both patients, initially with 5.34 percent (51,66% compared to normal minimum level of 57%), then after treatment the value decreased by 20 percent (47% compared to normal minimum level of 57%).

Beta globulins increased above the reference value with an insignificant percentage of 0.1% (13,1% compared to 13% normal maximum level), in contrast, gamma globulins, which were originally 1.62% higher (22,62% compared to normal maximum level of 21%), increased with 4.2% after treatment (to the value of 25,2%).

The albumin / globulin ratio, initially lower by 0.1% (1,1 compared to 1,2 minimum level), decreased further, to 0.31% after treatment (to 0,89).

Patient R. S. : Impressive large amounts of free radicals, 250 U Carr before treatment (compared to 300 U Carr normal maximum level), decreased by only 49 U Carr after treatment (to 501 U Carr).

Variations of pathological beta globulins were relatively small of 0.27%. (13,27% compared to 13% normal maximum level), then decrease to 13,10 % after treatment.

Total cholesterol initially increased by 37mg /dl (237 mg/dl compared to normal maximum of 200 mg/dl), decreased by 5mg/dl (to 232 mg/dl).

From the analysis of 26 biological parameters of the two patients, we observed mainly the following:

Patient I.C. - Increased values: TSH by 88.64% (from 0,988 uUI/ml to 1,46 uUI/ml), but normal values (0,4 - 4 uUI/ml), serum cortisol by 79.66% (from 7,4µg/dl to 18,3 µg/dl), alpha 2 globulin by 31,79% (from 9,48% to 12,5%), by 15,4% free radicals (from 229 U.Carr to 310 U.Carr), by 29,46% blood glucose (from 63 mg/dl to 84 mg/dl);

- decreased values: C reactive protein by 72,10% (from 1,44 mg/l to 0,64 mg/l), alpha 1-globulin by 42% (from 3,5% to 2,2%) , rheumatoid factor by 11,25% (from 8 to 9 UI/ml), serotonin by 18,91% (from 167,20 µg/l to 144,50 µg/l), A.C.T.H. by 11,48% (from 14,0 pg/ml to 12,39 pg/ml).

Patient R. S. - Increased values: serotonin by 40.52% (from 64,95 µg/l to 113.57 µg/l), gamma globulins by 32,29% (from 13,06% to 18,47%), blood cortisol by 24,24% (from 11,4 µg/dl to 14.2 µg/dl), TSH by 22,27% (from 2,57 uUI/ml to 3,06 uUi/ml), blood glucose by 13,71% (from 83 mg/dl to 95 mg/dl)..

- decreased values : free radicals by 17,82% (from 550 U Carr to 501 U Carr), triglycerides by 16%, (from 104 mg/dl to 80 mg/dl), cholesterol by 3% (from 237mg/dl to 232 mg/dl).

5.5. EXPERIMENT 5

Study of blood biochemical markers changes in patients diagnosed with psoriasis and treated with herbal extracts

Results :

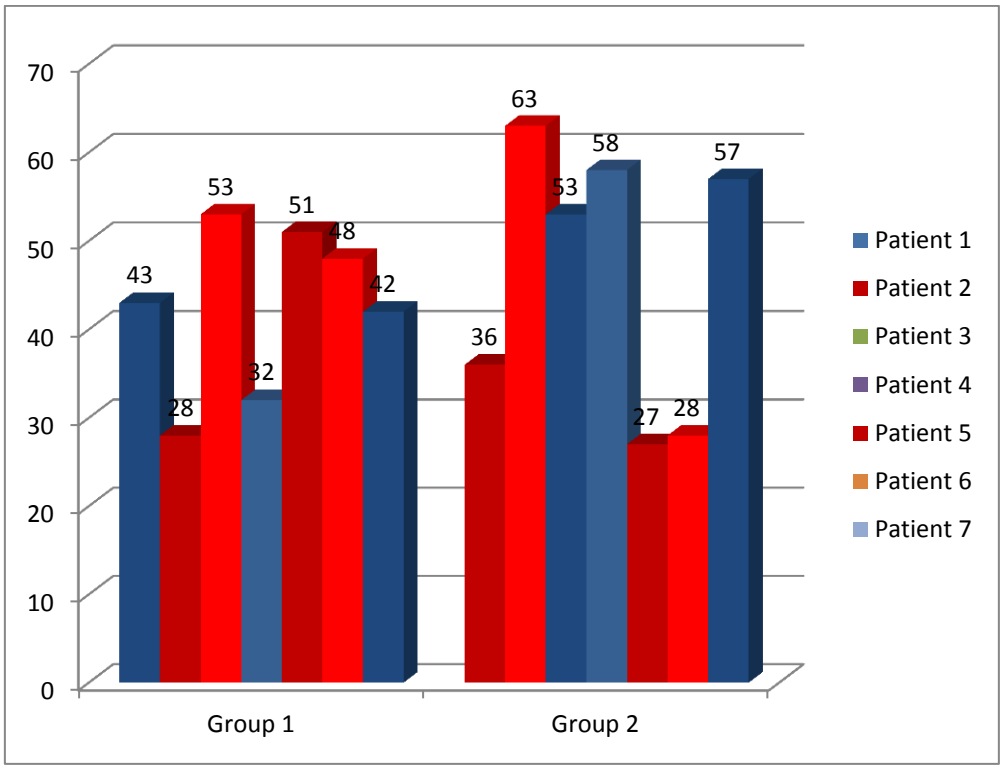


Figure 61 The gender structure of the two studied groups

The two groups under study are composed of seven patients, of whom 4 female patients and 3 male patients. Total 14 patients, 8 women and 6 men.

The age of patients is between 27 and 58 years with an average of 44.21 years; for the first group 44.43 years and 46 years for the second group.

Nr.	Analyze		Reference range	Average value
1	**GLU**	Blood sugar	65-115 mg/dl	90
2	**ALT**	Alanine transferase	5-40 IU/L	22,5
3	**AST**	Aspartate transaminase	5-42 IU/L	23,5
4	**LD-P**	lactate dehydrogenase	266-500 IU/L	383
5	**CK**	Creatinchinase	38-174 IU/L	106
6	**UREA**	Urea	15-50 mg/dL	32,5
7	**CR-S**	Creatinine	0,60-1,30 mg/dL	0,95
8	**URIC**	Uric acid	2,6-7,2 mg/dL	4,9
9	**CHOL**	Cholesterol	100-250 mg/dl	175
10	**TG-B**	Triglycerides	50-165 mg/dl	107,5
11	**HDLD**	High density lipoprotein cholesterol	29,0-71,0 mg/dl	50
12	**AMY7**	Blood amylase	25-125 U/L	75
13	**ALP**	alkaline phosphatase	32-92 IU/L	62
14	**GGT**	Gamma-glutamyl transpeptidase	7-64 IU/L	35,5
15	**TBIL**	Total bilirubin	0,2-1,2 mg/dl	0,7
16	**DBIL**	Direct bilirubin	0,0-0,02 mg/dl	0,1
17	**ALB**	Albumin	35/50 g/L	42,5
18	**TP**	Total protein	6,4-8.3 g/dl	7,55
19	**CA**	Calcemia	8,4-10,5 mg/dl	9,45
20	**MG**	Magnesemia	1,7-2,8 mg/dl	2,25
21	**FE**	Serum iron	65-165 ug/dl	115
22	**PHS**	Phosphoremia	2,5-4,6 mg/dl	3,55
23	**NA**	Natremia	136-145 mmol/L	140,5
24	**K**	Potasemie	3,5-5,10 mmol/L	4,3
25	**LDL. Col**	Low-density lipoprotein cholesterol	20-130 mg/dl	75
26	**At RISK**	Atherogenic risk	0,1-5,0	2,55
27	**Ion CA.**	Ionized calcium	4,0-5,2 mg/dl	4,6
28	**Albumin**	Albumin	50,0-65,0%	57,5
29	**Alpha 1**	α 1 globulin	1,0- 6,0 %	3,5
30	**Alpha 2**	α 2 globulin	6,0-13,0%	9,5
31	**Beta**	β globulin	8,0-16,0%	12
32	**Gamma**	γ globulin	12,5-21 %	16,75
33	**A/G**	albumin/globulin ratio	1,2-1,5	1,35
34	**PTH**	Parathyroid hormone	11- 67 pg/ml	39
35	**ACTH**	Adrenocorticotropic hormone	7,2-63,3 pg/ml	35.25
36	**Free Rad.**	Free Radicals	250-300 U.Carr	275
37	**Serotonin**	Serum serotonin	40-200 µg/l	170
38	**Compl C3**	Serum complement C3	83-183 mg/dl	133
39	**Compl C4**	Serum complement C4	15-57 mg/dl	36
40	**S.Cortisol**	Serum cortisol	3,7-19,4 µg/dl	11,55
41	**C reac. Prot.**	C reactive protein	0,10-6,00 mg/L	3,05
42	**Free T4**	Free thyroxine	0,89-1,74 ng/dl	1,315
43	**TSH**	Thyroid stimulating hormone	0,4-4 UI/ml	2,2

Table 9 Reference value ranges of conducted analyzes and their average values

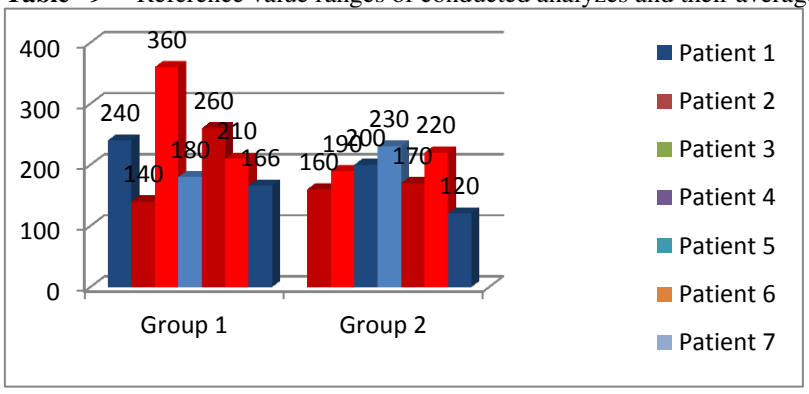


Figure 62 Initial surface (before treatment) of the psoriatic lesions (in cm²) Before starting treatment, the surface area of skin affected by psoriasis patients vary between 140 cm² and 360 cm² with an average of 203.28 cm². For group 1, the average was 222.28 cm², and for group 2, the average was 184.28 cm².

For the statistical processing of the data obtained after performing laboratory investigations, we have achieved the tables containing the test values before treatment and after 6 weeks of treatment.

Patient nr.	Average value	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Sex		♂	♀	♀	♂	♀	♀	♂	♀	♀	♂	♂	♀	♀	♂
Age years		43	28	53	32	51	48	42	36	63	53	58	27	28	57
Area cm²		240	140	360	180	260	210	166	160	190	200	230	170	220	120
GLU	90	81	82	77	90	63	83	86	75	235	78	90	72	61	88
ALT	22,5	33	24	11	28	38	46	38	9	35	84	30	13	12	19
AST	23,5	24	20	17	27	40	32	24	12	25	26	23	13	19	22
P	383	304	399	341	493	353	360	391	317	375	399	446	299	342	398
CK	106	50	223	69	85	112	94	49	81	121	109	240	42	60	54
UREA	32,5	44	29	31	52	48	42	21	19	41	23	49	17	28	36
CR-S	0,95	0,83	0,73	0,91	1,13	1,10	0,86	0,68	0,68	0,78	0,91	1,46	0,66	0.8	1,21
URIC	4,9	6,9	4,6	3,8	7,5	4,0	5,2	4,7	3,1	5,0	6,9	4,4	2,1	5,0	5,5
CHOL	175	249	220	234	233	191	237	258	178	211	318	128	187	129	232
TG-B	107,5	543	275	126	111	94,0	104	164	80	228	446	71	64	64	115
HDLD	50	34,2	37,6	57,9	80,6	55,6	56,4	55,8	50,5	41,4	37,1	36,2	59,9	40,6	47,9
AMY7	75	37	58	88	51	48	62,8	43	65	43	85	133	41	76	49
ALP	62	49	51	36	70	52	48	41	34	58	32	40	39	60	36
GGT	35,5	40	19	10	87	36	38	25	10	30	34	18	12	9	16
TBIL	0,7	0,8	0,7	0,6	0,7	0,4	0,7	0,7	0,7	0,5	0,6	0,8	0,6	0.8	0,8
DBIL	0,1	0,1	0,1	0,1	0,1	0,1	0,1	0.0	0,1	0,1	0,1	0,1	0.1	0.1	0,1
ALB	42,5	46	48	40	42	46	48	42	43	43	42	47	41	45	41
TP	7,55	7,5	7,9	7,7	7,6	7,9	8,2	7,0	7,4	7,8	7,6	8,3	7,5	7.6	7,2
CA	9,45	9,6	9,8	9,8	9,7	9,3	9,2	10,0	9,7	10,1	9,7	9,9	9,5	9,8	9,9
MG	2,25	2,1	2,3	1,9	2,4	2,2	2,1	2,0	2,2	1,9	2,2	2,3	1,9	2,0	2,2
FE	115	94	81	78	99	86	110	113	166	45	74	89	73	90	100
PHS	3,55	2,5	2,3	2,9	3,4	3.2	2,8	2,9	2,3	2,0	2,4	2,3	2,4	3,5	3,4
NA	140,5	138,2	138,9	137,0	143,2	142,0	138,4	136,6	140,4	138,1	139,0	141,6	139,6	139,6	144
K	4,3	4,16	4,67	4,42	4,69	4,40	4,10	3,86	4,14	3,93	5,04	5,18	4,47	4,51	3,50
LDL.Col	75	106,2	127,4	150,9	130,2	122,4	118,6	169,4	111,5	124,0	191,7	77,6	114,3	75,6	161,1
At.RISK	2,55	7,3	5,9	4,0	2,9	4,5	3,6	4,6	3.5	5,1	8,6	3,5	3,1	3,2	4,8
Ion CA.	4,6	4,1	4,0	4,1	4,1	4,3	4,2	4,4	4,2	4,2	4,1	4,0	4,0	4,1	4,3
Albumin	57,5	52,42	55,37	50,73	53,73	51,66	54,05	54,48	52,70	51,23	53,20	54,84	53,31	55,92	55,88
Alpha 1	3,5	3,35	3,52	4,41	4,46	3,5	3,6	3,85	6,35	4,87	5,61	4,37	4,07	3,36	4,00
Alpha 2	9,5	11,83	9,85	11,49	19,49	9,48	10	10,51	10,99	11,48	11,67	7,30	7,64	9,01	8,93
Beta	12	15,92	13,99	13,10	13,51	12,78	13,27	15,01	14,95	15,10	15,43	12,30	15,18	13,35	14,98
Gamma	16,75	16,51	17,26	20,27	17,82	22,62	13,06	16,16	15,00	17,32	14,09	21,19	19,79	18,37	16,20
A/G	1,35	1,10	1,24	1,03	1,16	1,1	0,89	1,20	1,11	1,05	1,14	1,21	1,14	1,27	1,27
PTH	39	14,6	29,4	46,0	34,8	24,8	11,8	18,4	16,2	22,4	48,8	52,4	14,6	28,8	32,8
ACTH	35.25	12,6	16,8	32,4	9,3	14,0	8,83	16,6	28,2	39,8	10,16	42,4	13,8	29,4	44.30
Free Rad.	275	280	310	288	260	229	550	220	268	220	300	280	288	260	280
Serotonin	170	120,4	190,3	86,8	170,9	167,2	144,5	183,6	98,6	110,8	189,7	88,4	110,3	163,3	146.2
Compl. C3	133	88	96	110	98	102	130	109	162	86	138	116	148	164	86
Compl. C4	36	56,3	48,2	18,9	36,8	30,5	37,6	42,3	52,4	19,7	28,3	42,8	39,6	19,8	44,3
S. Cortisol	11,55	3,8	6,4	12,6	4,4	7,4	11,4	8,2	12,8	14,2	10,6	8,4	4,8	12,8	11,4
C reac. Prot.	3,05	1,22	2,40	4,8	0,82	1,44	1,60	0,96	3,60	2,46	1,22	1,66	5,64	4,32	2,14
Rheumat. Fct.	5	< 8	< 8	< 8	< 8	<8	<8	< 8	< 8	< 8	< 8	< 8	< 8	< 8	< 8
Free T4	1,315	0,94	1,26	1,16	1,42	1,44	1,43	1,44	1,64	1,14	1,52	1,28	0,99	1,38	1,12
TSH	2,2	2,40	0,82	1,48	2,16	0,988	1,46	1,36	3,22	1,08	2,16	2,82	1,22	1,86	1,78

Table 10 Biochemical investigations of 14 cases with psoriasis vulgaris before treatment

(Values above the normal range are red, while those below the range are blue)

Reference ranges for normal laboratory test values were entered in a table, to which I added their average values, then used as a calculus basis by comparing the values obtained from study included patients.

Patient nr.	Average value	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Sex		♂	♀	♀	♂	♀	♀	♂	♀	♀	♂	♂	♀	♀	♂
Age/years		43	28	53	32	51	48	42	36	63	53	58	27	28	57
Area/cm²		20	12	28	0	10	14	0	22	28	30	42	20	36	16
Treatment		Sc+Kh	Sc+Kh	Sc+Kh	Sc+Kh	Sc+Kh	Sc+Kh	Sc+Kh	Sc	Sc	Sc	Sc	Sc	Sc	Sc
GLU	90	110	75	95	110	84	95	103	96	132	122	132	89	94	110
ALT	22,5	22	18	8	32	36	44	35	9	25	73	12	19	7	13
AST	23,5	24	19	15	36	38	30	24	14	25	39	14	27	17	18
D-P	383	295	393	295	480	388	420	288	307	412	433	396	295	323	307
CK	106	60	277	93	80	140	110	38	47	82	231	215	413	47	69
UREA	32,5	29	32	24	50	54	38	28	21	36	29	39	23	32	63
CR-S	0,95	0,85	0,70	0,77	1,10	1,00	0,95	0,60	0,67	0,79	1,05	1,22	0,76	0,89	1,18
URIC	4,9	6,2	5.0	3,6	7,00	5,2	5,4	4,3	3,1	6,1	7,7	3,9	2,6	6,6	6,4
CHOL	175	298	233	225	240	175	232	269	216	274	345	151	187	145	225
TG-B	107,5	853	171	83	110	127	80	158	72	270	476	92	33	64	229
HDLD	50	28,8	43,8	51,7	78,6	62,4	60,4	59,1	50,4	46,6	44,3	38,5	58,1	49.1	47,1
AMY7	75	34	52	80	48	50	75	38	69	54	83	144	48	67	47
ALP	62	43	56	33	68	58	42	36	35	49	36	39	33	52	36
GGT	35,5	55	17	11	84	48	42	24	12	34	54	20	8	8	17
TBIL	0,7	0,6	0,7	0,5	0,6	0,5	0,8	0,8	0,6	0,6	0,9	0,8	0,7	0,7	1,0
DBIL	0,1	0,0	0,2	0,1	0,2	0,1	0,3	0,0	0,1	0,1	0,1	0,1	0,1	0.1	0.1
ALB	42,5	44	48	40	45	43	45	42	41	41	45	45	43	45	41
TP	7,55	7,3	8,1	7,3	7,8	7,6	8,0	7,9	7,3	7,5	8,3	8,2	7,8	8,3	7,0
CA	9,45	9,8	9,6	9,4	9,4	9,2	9,0	9,6	9,7	9,7	9,7	8,9	9,3	9,8	9,5
MG	2,25	2,3	2,4	1,9	2,6	2,4	2,2	2,1	2,0	2,0	2,3	2,2	2,0	2,3	2,3
FE	115	44	83	75	110	92	106	130	86	49	107	83	76	118	143
PHS	3,55	3,3	3,7	3,5	3,9	4,0	3,7	3,9	2,9	2,4	3,1	3,3	2,9	3,9	3,2
NA	140,5	140,6	140	139,2	144,0	146,4	144,5	137,5	138,9	142,6	142,7	143,6	140,5	145,3	144,1
K	4,3	5,0	4,9	4,8	5,0	4,8	4,0	4,5	4,7	4,1	4,5	4,4	4,1	4,9	4,7
LDL.Col	75	98,6	155,0	156,7	140,4	136	126,6	178,3	151,2	173,4	205,5	94,1	122,3	83,1	132,1
At.RISK	2,55	10,3	5,3	4,4	3,6	5,0	4,2	4,6	4,3	5,9	7,8	3,9	3,2	3,0	4,8
Ion CA.	4,6	4,2	3,9	4,1	3,8	4,0	4,4	4,0	4,2	4,1	3,9	3,6	3,9	3,9	4,2
Albumin	57,5	53,77	52,24	54,31	52,23	47	54,22	54,14	50,37	52,13	51,15	53,10	53,20	51,01	51,23
Alpha 1	3,5	3,85	3,93	3,64	4,83	2,2	3,8	4,16	6,42	4,61	5,46	4,85	4,48	4,07	4,13
Alpha 2	9,5	12,16	9,33	10,80	20,14	12,5	10,40	10,89	11,16	11,83	12,36	7,85	8,83	10,44	9,33
Beta	12	16,40	13,9	12,7	14,23	13,1	13,10	14,74	16,18	14,33	17,23	12,77	13,86	13,52	16,19
Gamma	16,75	18,63	17,61	19,9	14,28	25,2	18,47	17,23	16,12	17,86	16,13	21,42	21,16	20,96	16,12
A/G	1,35	1,05	1,17	1,15	0,97	0,89	1,18	1,15	1,00	1,07	1,00	1,13	1,10	1,04	1,12
PTH	39	16,8	28,2	48,6	32,4	25,9	16,8	28,2	20,4	26,2	55,6	48,2	16,8	32,6	30,6
ACTH	35.25	14,80	20,60	30,2	16,4	17,41	17,4	22,6	32,3	40,2	16,2	38,6	18,4	32,8	40,20
Free Rad.	275	320	360	300	280	310	501	274	296	260	380	360	260	320	340
Serotonin	170	98,3	167,2	110,4	149,3	144,5	113,57	163,8	120,6	98,7	144,3	116,8	100,7	141,9	110,8
Compl. C3	133	96	108	124	104	106	134	128	146	98	140	122	124	153	93
Compl. C4	36	54,7	40,3	20,2	40,2	30	37,5	47,7	55,6	24,3	32,4	46,2	40,8	26,9	50,6
S. Cortisol	11,55	4,8	8,6	16,2	10,8	18,8	14,2	18,6	18,2	22,4	16,2	14,8	6,2	12,4	20,2
C reac. Prot.	3,05	1,46	2,20	6,00	0,84	0,80	2,01	1,14	3,40	2,72	1,04	1,48	5,40	4,66	2,83
Rheuma. Fct.	5	< 8	< 8	< 8	< 8	9	<8	< 8	< 8	< 8	< 8	< 8	< 8	< 8	< 8
Free T4	1,315	1,16	1,20	1,10	1,36	1,51	1,47	1,52	1,54	1,10	1,66	1,38	0,88	1,26	1,40
TSH	2,2	3,66	1,24	1,58	2,66	1,46	3,06	2,44	3,14	1,28	2,40	3,20	1,44	2,40	2,20

Table 11 Biochemical investigations of 14 cases with psoriasis vulgaris after treatment

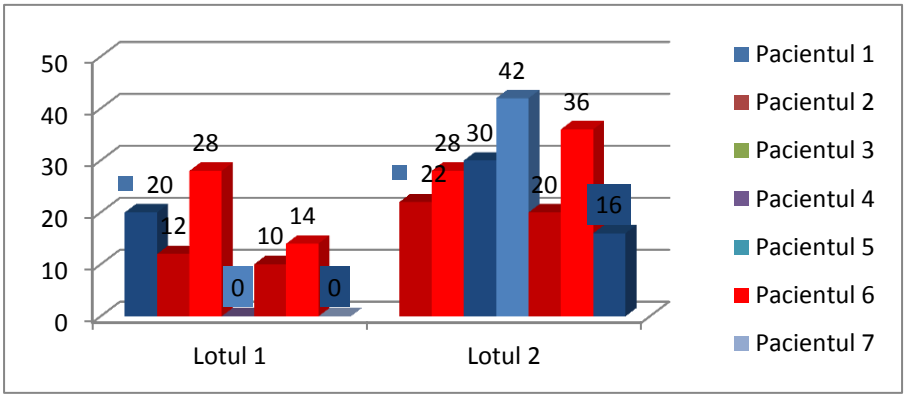


Figure 63 The area of the psoriatic lesions after treatment in cm²

Unhealed lesions area after treatment varies between 0 cm² and 42 cm².

Average for all these areas / patient for both groups in total is 19.86 cm².

Average for all these areas / patient for the first group is 12 cm².

Average for all these areas / patient for the second group is 27,71cm².

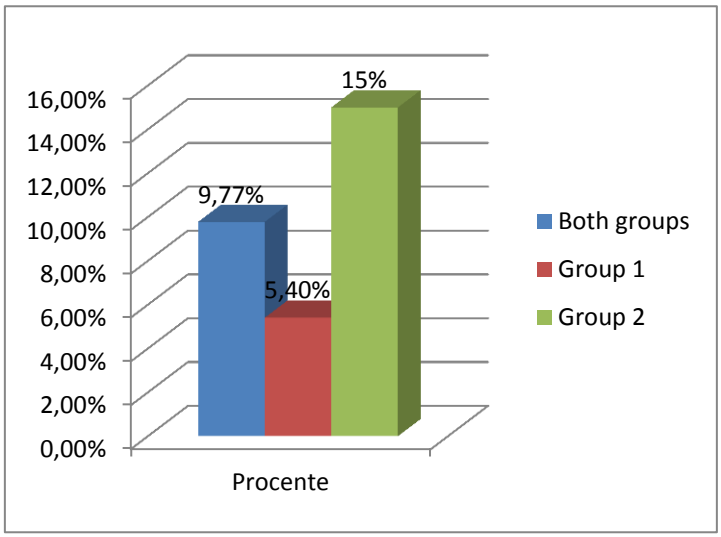


Figure 64 The residual area (after treatment) of the psoriatic lesions (in percent) Average percentage of the total of these surfaces / patient for both groups is 9.77
Average percentage of the total of these surfaces / patient for their first is 5.40%.
Average percentage of the total of these surfaces / patient for the second group is 15%.

	Stesclin derma + Klinhaem			Stesclin derma		
Analyze	The average value pre-treatment	The average value post-treatment	Difference percentage %	The average value pre-treatment	The average value post-treatment	Difference percentage %
GLU	80,28	96	↑ 19,58	99,85	110,7	↑10,86
ALT	31,14	27,85	↓10,56	28,85	22,57	↓21,76
AST	26,28	26,57	↑1,08	20	22	↑10
LD-P	377,28	365,57	↓3,10	368	353,28	↓4
CK	97,43	114	↑17	101	157,71	↑56,1
UREA	38,14	36,43	↓4,48	30,42	34,71	↑14,10
CR-S	0,89	0,85	↓4,49	0,92	0,94	↑2,17
URIC	5,24	5,04	↓3,81	4,57	5,2	↑13,78
CHOL	231,71	238,85	↑3,08	197,57	220,42	↑11,56
TG-B	202,42	226	↑11,6	152,57	176,57	↑15,73
HDLD	45,95	54,97	↑19,63	44,8	47,72	↑6,52
AMY7	55,4	53,85	↓2,80	70,28	73,14	↑4,06
ALP	49,57	48	↓3,17	42,71	40	↓6.34
GGT	36,43	40,14	↑10,18	18,42	21,85	↑18,62
TBIL	0,66	0,64	↓3.06	0,68	0,87	↑27,94
DBIL	0,09	0,13	↑44,44	0,1	0,1	0
ALB	44,57	43,85	↓1,61	43,14	43	↓0,32
TP	7,68	7,71	↑0,39	7,63	7,77	↑1,83
CA	9,63	9,42	↓2,18	9,80	9,51	↓2,95
MG	2,14	2,27	↑6,07	2,10	2,16	↑2,85
FE	82,34	91,43	↑11,03	91	94,57	↑3,92
PHS	2,86	3,71	↑29,72	2,61	3,1	↑18,77
NA	139,18	141,74	↑1,84	140,33	142,52	↑1,56
K	4,33	4,71	↑8,77	4,40	4,49	↑2,04
LDL.Col	132,16	141,65	↑7,18	122,26	137,38	↑12,36
At.RISK	4,69	5,34	↑13,85	4,54	4,7	↑3,52
Ion CA.	4,17	4,06	↓2,63	4,13	3,97	↓3,87
Albumin	53,20	52,56	↓1,20	53,87	51,74	↓3,95
Alpha 1	3,81	3,77	↓1,04	4,66	4,86	↑4,29
Alpha 2	11,80	12,32	↑4,40	9,57	10,26	↑7,21
Beta	13,94	14,02	↑0,57	14,47	14,86	↑2,69
Gamma	17,67	18,76	↑6,16	17,42	18,53	↑6,37
A/G	1,10	1,08	↓1,81	1,17	1,07	↓8,54
PTH	25,68	28,13	↑9,54	30,85	32,91	↑6,67
ACTH	15,79	19,92	↑26,15	29,72	31,24	↑5,11
Free Rad.	305,28	335	↑9,73	270,85	316.6	↑16,89
Serotonin	151,5	135,3	↓10,6	129,61	119,11	↓8,10
Compl. C3	104,71	114,29	↑9,14	128,57	125,14	↓2,66
Compl. C4	38,65	38,66	↑0,02	36,27	39,54	↑9,01
S. Cortisol	7,74	13,14	↑69,76	10,70	15,77	↑47,38
C reac. Prot.	1,89	2,06	↑8,99	3,00	3,07	↑2,33
Free T4	1,30	1,33	↑2,30	1,30	1,32	↑1,53
TSH	1,52	2,3	↑51,31	2,02	2,29	↑13,36

Table 12 The average values of analyzes before and after treatment, the differences in percentage

	Treatment with Stesclin derma + Klinhaem			
	Analyze	The average value pre-treatment	The average value post-treatment	Difference percentage %
1	s.Cortisol	7,74	13,14	↑ 69,76
2	TSH	1,52	2,3	↑ 51,31
3	DBIL	0,09	0,13	↑ 44,44
4	PHS	2,86	3,71	↑ 29,72
5	ACTH	15,79	19,92	↑ 26,15
6	HDLD	45,95	54,97	↑ 19,63
7	GLU	80,28	96	↑ 19,58
8	CK	97,43	114	↑ 17,00
9	At.RISK	4,69	5,34	↑ 13,85
10	TG-B	202,42	226	↑ 11,60
11	FE	82,34	91,43	↑ 11,03
12	GGT	36,43	40,14	↑ 10,18
13	Free Rad.	305,28	335	↑ 9,73
14	PTH	25,68	28,13	↑ 9,54
15	Compl. C3	104,71	114,29	↑ 9,14
16	C reac. Prot.	1,89	2,06	↑ 8,99
17	K	4,33	4,71	↑ 8,77
18	LDL.Col	132,16	141,65	↑ 7,18
19	Gamma	17,67	18,76	↑ 6,16
20	MG	2,14	2,27	↑ 6,07
21	Alpha 2	11,80	12,32	↑ 4,40
22	CHOL	231,71	238,85	↑ 3,08
23	Free T4	1,30	1,33	↑ 2,30
24	NA	139,18	141,74	↑ 1,84
25	AST	26,28	26,57	↑ 1,08
26	Beta	13,94	14,02	↑ 0,57
27	TP	7,68	7,71	↑ 0,39
28	Compl. C4	38,65	38,66	↑ 0,02
29	Alpha 1	3,81	3,77	↓ 1,04
30	Albumin	53,20	52,56	↓ 1,20
31	ALB	44,57	43,85	↓ 1,61
32	A/G	1,10	1,08	↓ 1,81
33	CA	9,63	9,42	↓ 2,18
34	Ion CA.	4,17	4,06	↓ 2,63
35	AMY7	55,4	53,85	↓ 2,80
36	TBIL	0,66	0,64	↓ 3,06
37	LD-P	377,28	365,57	↓ 3,10
38	ALP	49,57	48	↓ 3,17
39	URIC	5,24	5,04	↓ 3,81
40	UREA	38,14	36,43	↓ 4,48
41	CR-S	0,89	0,85	↓ 4,49
42	ALT	31,14	27,85	↓ 10,56
43	Serotonin	151,5	135,3	↓ 10,6

Table 13 Average values and percentage differences of group 1 analyzes

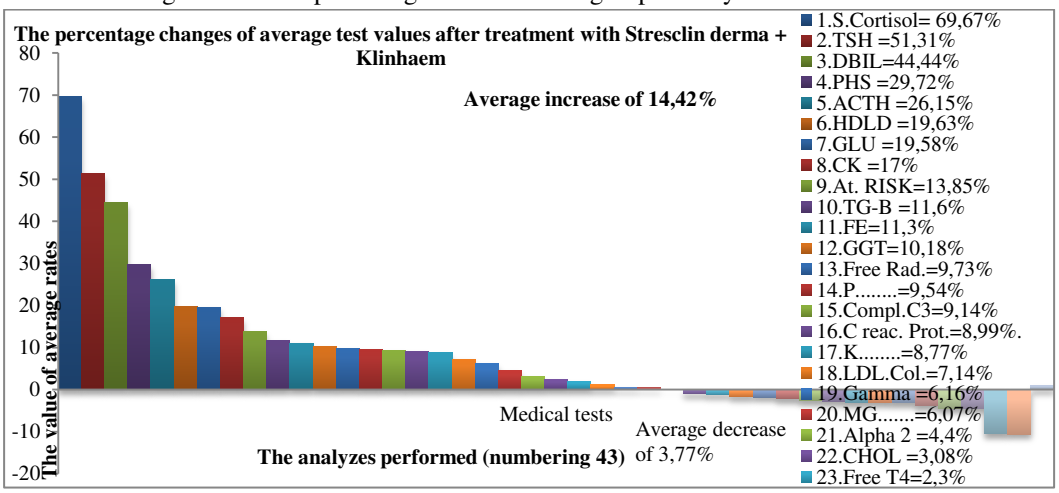


Figure 108

Nr.	Treatment with Stresclin derma			
	Analyses	The average value pre-treatment	The average value post-treatment	Difference percentage %
1	CK	101	157,71	↑ 56,1
2	S. Cortisol	10,70	15,77	↑ 47,38
3	TBIL	0,68	0,87	↑ 27,94
4	PHS	2,61	3,1	↑ 18,77
5	GGT	18,42	21,85	↑ 18,62
6	Free Rad.	270,85	316.6	↑ 16,89
7	TG-B	152,57	176,57	↑ 15,73
8	UREA	30,42	34,71	↑ 14,10
9	URIC	4,57	5,2	↑ 13,78
10	TSH	2,02	2,29	↑ 13,36
11	LDL.Col	122,26	137,38	↑ 12,36
12	CHOL	197,57	220,42	↑ 11,56
13	GLU	99,85	110,7	↑ 10,86
14	AST	20	22	↑ 10
15	Compl. C4	36,27	39,54	↑ 9,01
16	Alpha 2	9,57	10,26	↑ 7,21
17	PTH	30,85	32,91	↑ 6,67
18	Gamma	17,42	18,53	↑ 6,37
19	HDLD	44,8	47,72	↑ 6,52
20	ACTH	29,72	31,24	↑ 5,11
21	Alpha 1	4,66	4,86	↑ 4,29
22	AMY7	70,28	73,14	↑ 4,06
23	FE	91	94,57	↑ 3,92
24	At.RISK	4,54	4,7	↑ 3,52
25	MG	2,10	2,16	↑ 2,85
26	Beta	14,47	14,86	↑ 2,69
27	C reac. Prot.	3,00	3,07	↑ 2,33
28	K	4,40	4,49	↑ 2,04
29	CR-S	0,92	0,94	↑ 2,17
30	NA	140,33	142,52	↑ 1,56
31	TP	7,63	7,77	↑ 1,83
32	Free T4	1,30	1,32	↑ 1,53
33	DBIL	0,1	0,1	0
34	ALB	43,14	43	↓ 0,32
35	Compl. C3	128,57	125,14	↓ 2,66
36	CA	9,80	9,51	↓ 2,95
37	Ion. CA	4,13	3,97	↓ 3,87
38	Albumin	53,87	51,74	↓ 3,95
39	LD-P	368	353,28	↓ 4
40	ALP	42,71	40	↓ 6.34
41	Serotonin	129,61	119,11	↓ 8,10
42	A/G	1,17	1,07	↓ 8,54
43	ALT	28,85	22,57	↓ 21,76

Table 14 Average values and percentage differences of group 2 analyzes

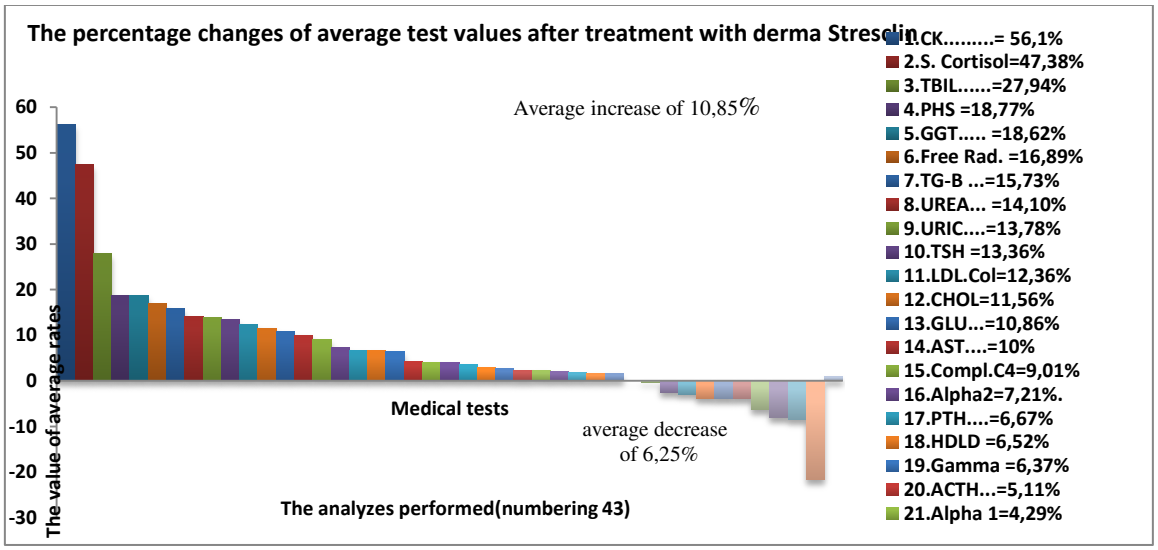


Figure 109

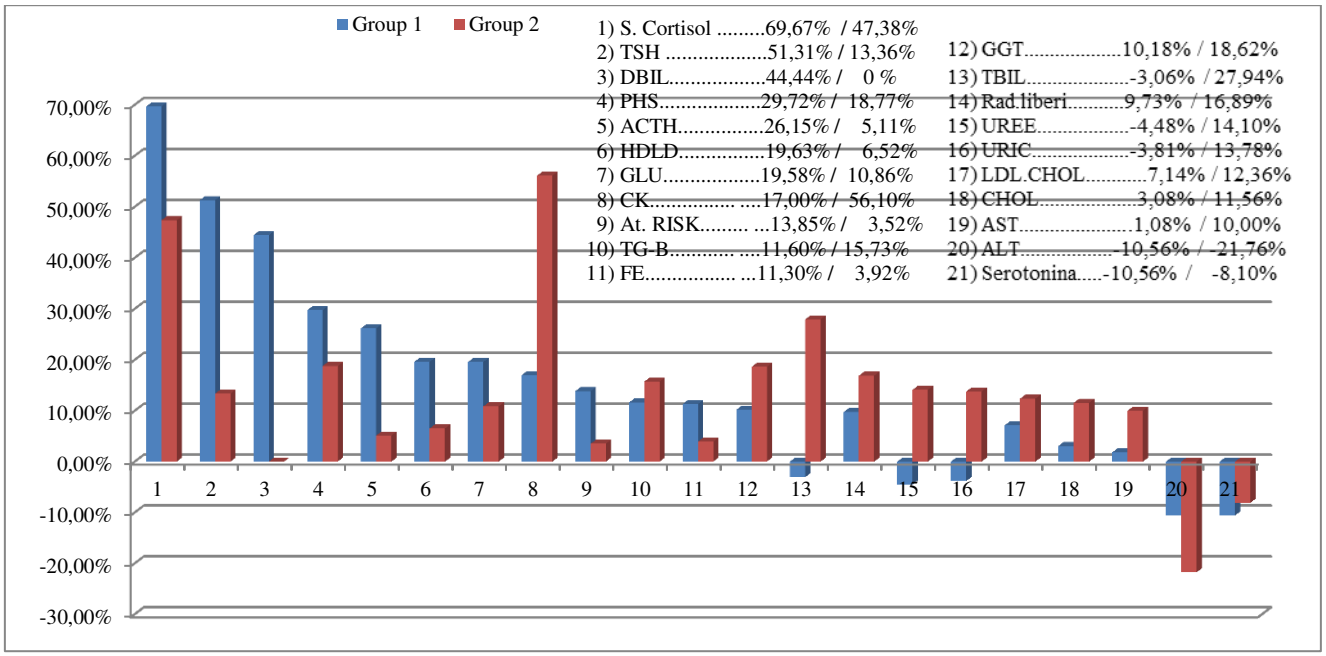


Figure 110 Variations of percentage average values of tests after treatment exceeding average values of 10% compared to average values before the treatment

From the graph shown in Figure 110 it makes clear that significant changes were observed in the average values test results of the study.

From the average percentage variations, we selected the values that exceeded by 10% the initial baseline average for each particular analysis, positively and negatively.

Variations of average percentage results which exceed +/- 10%.

Action of plant extracts

The obvious increase of these averages after completion of therapy, is explained by the content of plant extracts included in the two therapeutic products:

For Klinhaem

- anti-psoriatic action of tuberous roots extract of Smilax china - **250** mg/5 ml;
- immunomodulation action of Rubia cordifolia roots extract - **612,5** mg/5 ml;
- anti-inflammatory action of Acacia catechu roots extract - **612,5** mg/5 ml;
- anti-inflammatory action of Azadirachta indica extract - **250** mg/5ml;
- immunostimulant action of Tinospora cordifolia extract – **87,5** mg /5ml;
- anti-inflammatory and antioxidant action of theTurmeric rhizome extract (Curcuma Longa) – **75** mg/5ml.

For Stresclin derma

- anti-inflammatory and antioxidant action of theTurmeric rhizome extract (Curcuma Longa) – **250** mg/capsule;
- action to combat free radicals Camellia sinensis extract (green tea) – **70** mg/capsule;
- anti-inflammatory, antioxidant action and free radical annihilation of Bacopa monnieri extract – **35** mg/capsule;
- anti-inflammatory action of Acacia catechu roots extract - **35** mg/capsule;
- anti-inflammatory action of Azadirachta indica extract - **25** mg/capsule;
- immunostimulatory and anti-inflammatory action of Ocimum sanctum extract, fruits – **25** mg/capsule;
- blood cleanser, detoxifying, calming, sedative action of Taraxacum officinale (dandelion), roots extract - 25 mg/capsule;

In group 1, patients treated with Klinhaem and Stresclin derma, I recorded the following significant variations compared with patients group 2 (values increased by over 10% from the initial average values) :

Serum cortisol

It increased from the average baseline by 69.76% (from 7.74 mg / dL to 13.4 mg / dl) for the first group and by 47.38% (from 10.70 mg / dl to 15 77 mg / dl) for the second group.

Blood samples were collected in the morning, at the same time. Patients were not treated previously blood samples collection with drugs that could iatrogenic increase serum cortisol value.

None of the patients was pregnant.

During treatment, none of the patients did not change lifestyle, nor subjected to special stressful situations.

Cortisol is part of glucocorticosteroids.

In blood, 90% of cortisol is linked to "corticosteroid binding globulin" and albumin, and 10% of cortisol is free to interact with receptors.

The main action is to increase blood glucose (by stimulating gluconeogenesis) and anti-inflammatory and immunosuppressive action.

Controlling the synthesis and secretion of cortisol is provided by the hypothalamic-pituitary-adrenal axis negative feedback.

Secretion stimulating is achieved due to the hormone corticotropin releasing (corticotropin releasing hormone) secreted by the hypothalamus through the release of pituitary ACTH.

At the same time, the stress increases cortisol secretion.

Serum cortisol levels varies during the day. Highs are seen in the morning hours and the minimum recorded in the afternoon and evening.

In pregnancy, elevated cortisolmay appear.

Medicamente mai frecvent cortisolo-stimulante sunt : aspirina, contraceptive orale, diclofenac, insulina, ranitidina, estrogeni, cortizon, atropina, anticonvulsivante, metoclopramid, spironolactona, hidrocortizon, benzodiazepine, etc. (http://www.synevo.ro).

Serum TSH (Thyroid stimulating hormone)

It increased compared to average reference for the first group by 51.31% and by 13.36% for the second group.

Thyroid stimulating hormone (TSH, thyrotropin) is created by the anterior pituitary cells and has a circadian secretory sequence controlled by TSH-releasing hormone (TRH) produced by the hypothalamus.

TSH has a stimulatory action on the formation and secretion of thyroid hormones and proliferative action.

The patients were not under influence of drugs that can cause increased levels of TSH: amiodarone, atenolol, calcitonin, carbamazepine, chlorpromazine, clomiphene, conjugated estrogens, phenytoin, potassium iodide, L-thyroxine, lithium, lovastatin, methimazole, metoclopramide, morphine, prazosin prednisone, propranolol, rifampin, sumatriptan, tamoxifen (http://www.synevo.ro).

TBIL levels (direct bilirubin)

It increased compared to average reference for the first group by 44.44% and by 0% for the second group.

Serum bilirubin levels increase when production exceeds its metabolism and excretion.

Elevated levels of direct bilirubin in serum is associated with reduced excretion of conjugated bile pigment by the liver and by the gallbladder, and occurs in cholestatic or hepatocellular jaundice.

In the laboratory two parts of the pigment are measured - the conjugated water-soluble fraction, which gives direct reaction with the diazo reagent and which is conjugated bilirubin (in the form of mono- and diglucuronid) and fat-soluble fraction which is the unconjugated bilirubin.

Conjugated bilirubin, soluble and highly reactive, gives a color reaction with diazo reagent, being known as the direct bilirubin. Elevated serum value of direct bilirubin is associated with reduced excretion of conjugate pigment from the liver and occurs in cholestatic and hepatocellular jaundice. Pathological increase of direct bilirubin leads to appearence of the pigment in urine (http://www.synevo.ro).

Phosphorus - serum

It increased compared to average reference for the first group by 29.72% and by 18.77% for the second group.

The main regulators of phosphoremy are the kidneys. Parathyroid hormone (PTH) stimulates renal excretion of phosphate. Other hormones that play a role in phosphate homeostasis (insulin, growth hormone, 1,25 (OH) 2D3, IGF1, thyroid hormones), reduce the excretion of phosphate. On the other hand, glucocorticoids, calcitonin, atrial natriuretic factor, EGF, TGF-α, increase the excretory effect.

Level phosphoremy has to be evaluated together with serum calcium; there is an inverse relationship between the two elements: increasing one of the two electrolytes in the blood increase the urinary excretion of the other electrolyte. Many of the causes that increase the level of calcium also decrease phosphoremy.

None of the patients had received treatment with a medication that can increase phosphoremy, ex: tetracycline (nephrotoxicity), anabolic steroids, androgens, tacrolimus, beta adrenergic blockers (acebutolol, pindolol), vitamin D2, erythropoietin, ergocalciferol, furosemide, growth hormone, hidrocloratiazida, Staphylococcus aureus (nephrotoxicity), phosphates, sodium etidronate (http://www.synevo.ro).

Serum ACTH (adrenocorticotropic hormone)

It increased compared to average reference by 26,15% for the first group and just by 5.11% for the second group.

ACTH controls the development and hormone secretion of the adrenal glands. By stimulating adrenal fasciculata area it activates the synthesis and secretion of glucocorticoids (cortisol and corticosterone). ACTH secretion-stimulating effects of cAMP are mediated by the adrenal cortex.

ACTH secondary properties are due to the metabolic effects of glucocorticoid hormones, activating carbohydrate, lipid and protidic metabolism.

Adjusting ACTH secretion occurs via neurohumoral path with indirect participation of the hypothalamus. The main humoral regulating factor is the blood levels of glucocorticoid hormones, especially cortisol. Under its influence, the secretion of ACTH decreases, by a negative feedback mechanism and, respectively, increases if circulating glucocorticoids decrease.

Serum HDLD (High density lipoprotein cholesterol)

It increased compared to average reference for the first group by 19.63% and by only 6.52% for the second group.

HDL cholesterol (HDL-D) is a group of synthesized and hepatocyte secreted lipoprotein.

HDL plays an important role in cholesterol metabolism, participating in its transportation from extrahepatic tissues to the liver for catabolism and excretion. Together with the LDL it contributes to the maintenance of cell cholesterol level. HDL cholesterol and apolipoprotein concentrations, are positive risk factors in atherosclerosis. Pacienții cu nivele ridicate ale HDL-D sunt protejați, având un risc scăzut de a face ateroscleroză. Patients with high levels of HDL-D are protected, having a low risk of atherosclerosis. Increases in HDLD occur in different situations: moderate alcohol consumption, treatment with insulin, intens and long exercise, the consumption of drugs: cimetidine, cyclofenil, oral contraceptives, doxazosin, estrogen, fibric acid derivatives (clofibrate, gemfibrozil) lovastatin, pravastatin, simvastatin, niacin, phenobarbital, phenytoin, prazosin, terazosin, terbutaline, captopril, carbamazepine, coenzyme Q10, furosemide, insulin, ketoconazole, medroxyprogesterone, niacin, nifedipine, verapamil (http://www.synevo.ro)

Blood sugar (serum glucose)

It increased compared to average reference for the first group by 19.58% and by 10.86% for the second group.

The hormone that regulate blood glucose levels and insulin is glucagon. Glucagon speeds up the conversion of glycogen into glucose, causing blood sugar rise. Insulin increases the permeability of cell membranes to glucose,transports the glucose to cells (metabolism), stimulates the formation of glycogen and reduces the blood glucose concentration. Other hormones that play an important role in glucose metabolism are: ACTH, glucocorticoids, epinephrine, thyroxine.

Increases in blood sugar can be caused by external intake: caffeine, ACTH, corticosteroids, asparaginase agonists, beta adrenergic (ex. Albuterol, isoproterenol, terbutaline), calcitonin, diazoxide, diuretics (acetazolamide, chlorthalidone, ethacrynic acid, furosemide, thiazides, triamterene), dopamine, epinephrine, estrogens, fructose, glucagon, indomethacin, lithium carbonate, morphine, nicotinic acid (high dose), octreotide (somatostatin), oral contraceptives, phenothiazines, phenytoin, rifampin, streptozotocin, theophylline, thiabendazole, D-thyroxine (http://www.synevo.ro).

CK (serum creatine kinase)

It increased compared to average reference by 17% for the first group and by 56.1% for the second group.

Creatine kinase is an enzyme which is found in high concentrations in skeletal muscles and myocardium and in much lower concentrations in the brain.

Elevations of creatine kinase is determined by the external supply of: aminocaproic acid, amphotericin B, captopril, carbenoxolone, carteolol hydrochloride, clindamycin, chlorpromazine, clofibrate, clonidine, colchicine, cyclopropane, danazol, diclofenac, digoxin, gemfibrozil, haloperidol, inhibitors of 3-hydroxy-3- -metilglutaril CoA reductase inhibitors (eg, lovastatin), insulin, isotretinoin, labetalol, lamivudine, levamisole, lidocaine, lithium, D-penicillamine, pindolol, prochlorperazine, propranolol, streptokinase, Trimethoprim, zalcitabine (http://www.synevo.ro).

Atherogenic risk

It increased compared to average reference for the first by 13.85% and by only 3.52% for the second group.

Blood TG (triglycerides)

It increased compared to the average reference by 11.6% for the first group and by 15.73% for the second group.

Transient elevations may occur after ingestion of alcohol and rich lunch. Elevated levels may occur in pregnancy, obesity, physical inactivity, smoking.

Increases occur in genetic hyperlipidaemia (types I, II, III, IV, V, deficiency of apo C-II) and secondary, gout, pancreatitis, liver disease, alcoholism, nephrotic syndrome, renal disease, acute: MI - reach the maximum in three weeks and may persist for a year, hypothyroidism, diabetes, glycogenosis (von Gierke disease), Down syndrome, anorexia nervosa.

Increased levels occur after the intake of: beta blockers, catecholamines, cholestyramine, corticosteroids, cyclosporine, danazol, diazepam, diuretics, estrogen, ethanol, etretinate, interferon, isotretinoin, retinol, miconazole (http://www.synevo.ro).

Serum iron - sideremia

It increased compared to average reference by 11.3% for the first group and just by 3.92% for the second group.

Ferroportin is a basolateral transporter through which iron leaves enterocitul and is the site of action for hepcidin, recently discovered peptide hormone composed of 25 amino acids, synthesized in the liver, which interacts with ferropoetin and induces internalization and degradation of it.

Increased sideremy can occur in chronic liver disease, premenstrual periods, or after treatments: aspirin, cefotaxime, chemotherapy, chloramphenicol, cisplatin, oral contraceptives, iron dextran; Staphylococcus aureus, methimazole, methotrexate, multivitamins containing iron, pyrazinamide (http://www.synevo.ro).

GGT (serum gamma glutamyl transferase)

It increased compared to average reference for the first group by 10.18% and by 18.62% for the second group.

GGT synthesis in the liver can be induced by cholestasis, chronic alcohol consumption and therapeutic doses of certain drugs, such as phenytoin. GGT level is increased in tumor cells of hepatoma in hepatocytes and liver tumors compressed by the areas of regenerative cirrhotic liver. The increase in serum levels of GGT is also caused by cell membrane damage by toxic drugs (including alcohol), ischemia, infection, or enzyme removal from the cell membrane , as a result of the detergent action of bile acids. GGT is an enzyme specific to the liver and bile ducts.

GGT is most sensitive indicator to detect alcoholism, the enzyme whose increase exceeds other routinely dosed liver enzyme.

In hepatobiliary diseases, GGT correlates with alkaline phosphatase levels. The increases however are not specific and may be associated with pancreatic, cardiac, renal, or diabetes disorders.

Elevations in GGT can occur under certain circumstances. Liver pathology:

- Acute viral hepatitis, chronic active hepatitis, viral or autoimmune in acute alcoholic hepatitis, liver cirrhosis, biliary cirrhosis in fatty liver, cholestasis syndrome, primitive liver tumors, liver metastases, liver congestion.

Isolated elevations of GGT: anticonvulsant medication, fatty liver, subclinical biliary obstruction, replacement space by formations in liver, heart failure, ethanol etiology.

Other causes of increased GGT: acute pancreatitis, acute myocardial infarction, acute renal failure, nephrotic syndrome, graft versus host impairment (moderate increases), diabetes (slightly higher), tumors and brain hemorrhage (slight increase), neoplasms - especially melanoma, breast cancer and lung cancer.

Slight elevation in GGT can occur in obesity, kidney disease, heart disease, postoperative states.

Some drugs can cause increases of GGT value : acetaminophen, barbiturates, captopril (rare), cephalosporins, estrogens, oral contraceptives, phenytoin, primidone, propoxyphene, streptokinase acid, ASA, amitriptyline, anabolic steroids, androgens, azotioprina, benzodiazepines, carbamazepine, carbazona , chlorothiazide, chlorpropamide, clavulanic acid, dapsone, acetaminophen, allopurinol acid, ASA, amiodarone, amitriptyline, androgens, asparaginase, aspirin, azathioprine, carbamazepine, chenadiol, chlorambucil, chloramphenicol, chlorpropamide, cimetidine, cyclosporine, danazol, dantrolene, dapsone, diclofenac , disulfiram, erythromycin, estrogens, etc. (http://www.synevo.ro).

In group 2, patients treated with Stresclin derma, I record the following significant variations compared with group 1 (values increased by over 10% from the initial average values):

TBIL (serum total bilirubin)

It increased from the average reference by 27.94% for the second group and decreased below the average reference by -3.06% for the first group.

Bilirubin serum levels increase when production exceeds its metabolism and excretion.

Total serum bilirubin elevated presented in the following cases: uncomplicated hemolysis, hepatocellular jaundice, extrahepatic biliary obstruction, viral hepatitis, alcoholic hepatitis.

Medicinal products that cause increases: acebutolol, acetaminophen, acetazolamide, acyclovir, albendazole, allopurinol, alprazolam, amiloride, etc..

Medicinal products that cause decreases: amikacin, anticonvulsants, prednisone, theophylline, thioridazine, ursodiol (http://www.synevo.ro).

Free radicals

It increased compared to the average of reference for the second broup by 16.89% and by 9.73% for the first group.

Free radicals in the body primarily come from oxidation processes performed by the body on the background of stress and negative emotional states, but also from the toxins of all kinds from the outside and reached the body in different ways (alcohol, tobacco, drugs drugs, pollution, malnutrition).

Free radicals are highly reactive and may alter the structure of the macromolecules: proteins, lipids, nucleic acids (DNA, RNA).

Free radicals are known to alter cell membranes by lipid peroxidation. Significant action of free radicals is focused on unsaturated fatty acids in cell membranes.

The consequence of membrane lipid peroxidation: increasing the stiffness, decreased activity of membrane enzymes, impaired permeability and altered receptor activity present in the membrane.

Free radicals can directly attack also membrane proteins, causing changes in the structure and activity of cell membranes.

Urea (blood urea nitrogen)

It increased from the average reference by 14.10% for the second group and decreased below the average reference by -4.49% for the first group.

Urea in the blood and urine varies in direct proportion with nutrition and inversely proportional to cell anabolism in protein gain state, pregnancy, convalescence. Also, the concentration of serum urea depends on renal perfusion: in the presence of urine output, blood urea rerun from distal renal tubules is minimal, a large amount of urea is excreted in urine and serum urea levels remain low; if antidiuresis exists, as thirsty exsicoza, oliguria heart failure, urea difuse from distal tubules to blood flow, and blood urea levels rise.

Medicines that cause increases: acetaminophen acetazolamide, aminocaproic acid, nalidixic acid, acyclovir, dexamethasone, dextran, diazepam, diazoxide, diclofenac, methylprednisolone, metoprolol, etc..

Medicines that cause decreases: ascorbic acid, phenothiazines (http://www.synevo.ro).

URIC (serum uric acid)

It increased from the average reference by 13.78% for the second group and decresed below the average reference by -3.81% for the first group.

Increases in serum uric acid is recorded in different situations: renal failure, gout, asymptomatic hyperuricemia, neoplasia, antineoplastic chemotherapy, hemolytic anemia, pernicious anemia, toxemia of pregnancy, psoriasis (1 / 3out patients); •

salicylates (<4 g / day), poisoning (barbiturates, methanol, ammonia, carbon monoxide), substances that cause low renal clearance or tubular secretion (thiazides, furosemide), nephrotoxicity (mitomycin C), metabolic acidosis, diabetic ketoacidosis; hypertriglyceridemia, diet: protein diet with high molecular weight decrease, nucleoprotein excess, alcohol consumption. Various causes: lead poisoning, von Gierke's disease, Nyhan syndrome Lesch- (hereditary gout), Down syndrome, polycystic kidney disease, hypoparathyroidism, primary hyperparathyroidism, hypothyroidism, sarcoidosis.

Medicines that cause increases: nicotinic acid (high dose), beta-blockers (atenolol, propranolol, nadolol, timolol), corticosteroids (acute leukemia), cyclosporine, diazoxide, diuretics (acetazolamide, chlorthalidone, ethacrynic acid, furosemide, thiazides triamterene), epinephrine, ethanol, ethambutol, phenothiazines, norepinephrine, pyrazinamide, salicylates (low dose), some anticancer (asparaginase, cisplatin, chlorambucil, fludarabine, hydroxyurea, idarubicin, mechlorethamine, vincristine), theophylline.

Medicines that cause decreases: ascorbic acid, alfametildopa, allopurinol, aspirin, desferoxamin, corticosteroids, diethylstilbestrol, enalapril, ibuprofen, indomethacin, mannitol, probenecid, spironolactone, verapamil (http://www.synevo.ro).

LDL.C0l (Low-density lipoprotein cholesterol levels)

It increased compared to average reference for the second group by 12.36% and by7.14% for the first group.

Increases: postprandial, steroid administration: anabolic steroids, beta blockers, antihypertensives, progestogens, carbamazepine familial hypercholesterolemia (type II), hyperlipoproteinemia II b and III, rich diet in cholesterol and saturated fat, hypothyroidism, nephrotic syndrome, diabetes , multiple myeloma and other dysgammaglobulinemia, cholestasis, chronic kidney disease, porphyria, anorexia nervosa (http://www.synevo.ro)

CHOL (serum cholesterol levels)

It increased compared to average reference for the second group by 11.56% and by 3.08% for the first group.

Increases: hyperlipoproteinaemia type IIb, III, V, familial hypercholesterolemia type IIa, cholestasis, biliary cirrhosis, nephrosis, pancreatic disease, pancreatic and prostate cancer, hypothyroidism, diabetes, alcoholism, von Gierke disease (glycogenosis), rich diet in fat and cholesterol, obesity.

Individual variation (4-10%); Seasonal variations: higher values up to 8% in winter than in summer; cholesterol diet and pregnancy causes increases in cholesterol. Medicines that cause increases: ascorbic acid, chenodeoxycholic, amiodarone, androgens, aspirin, catecholamines, antibiotics, beta-blockers, carbamazepine, clonidine, clopidogrel, cyclosporine, glycogen corticosteroids, ibuprofen, vitamin D, cyclosporine, disulfiram, diuretics (small effect) , ergocalciferol (high dose), etretinate, isotretinoin, levodopa, miconazole (http://www.synevo.ro).

AST (serum aspartate aminotransferase)

It increased compared to average reference by 10% for the second group and by 1.08% for the first group.

Apart from liver disease with necrosis of hepatocytes, moderate increases are recorded in some forms of muscular dystrophy in dermatomyositis, trauma, surgery, intramuscular, eclampsia, acute pancreatitis, intestinal lesions,local post irradiation pulmonary infarction, cerebral infarction, myocardial kidney , burns, hypothermia, hyperthermia, intoxication (mushrooms), hemolytic anemia; hypothyroidism.

Drugs that may cause elevated AST**:**

Drugs causing cholestasis: ASA acid, amitriptyline, anabolic steroids, androgens, azathioprine, etc.

Drugs causing hepatocellular damage: acetaminophen, allopurinol, aminosalicylic acid, amiodarone, amitriptyline, anabolic steroids, androgens, asparaginase, aspirin, etc.

Many other drugs can cause increases which are generally transient, but in some cases indicate hepatotoxicity. Among these are included: acebutolol, aminoglutethimide, aminoglycosides, azithromycin, etc. (http://www.synevo.ro).

In group 2, patients treated with Stresclin dermis, I recorded the following significant variations compared with group 1 (lower values by more than 10% from the initial average values):

ALT (serum alanine aminotransferase)

It decreased compared to the average reference by -21.76% for the second group and by -10.56% for the first group.

ALT is the most commonly explored cytolisis indicator and most appropriate to detect even minimal liver damage. ALT is more specific for liver disease than AST . Absolute values of ALT however do not correlate directly with the severity and prognosis of liver damage, and therefore serial measurements are very useful.

Decreases in ALT are recorded in several pathologies as: urinary infections, malignancies, pyridoxal phosphate deficiency (malnutrition, alcohol).

Overall, ALT and AST levels are parallel. Exception is alcoholic hepatitis, where the ratio AST / ALT (De Ritis) may be greater than 2 due to reduced tissue of liver by ALT (deficiency of pyridoxal phosphate) and occasionally ratio AST / ALT may increase in patients with fatty liver infiltration in pregnancy.

Drugs that may decrease ALT: aspirin, cyclosporine, phenothiazines, interferon, ketoprofen, simvastatin, ursodiol (http://www.synevo.ro).

In group 1, patients treated with Klinhaem and Stresclin derma, I recorded the following significant variations compared with group 2 (lower values by more than 10% from the initial average values) :

Serotonin (serum serotonin)

It decreased compared to the average reference by -10.56% for the first group and by -8.10% for the second group.

Foods that contain serotonin do not significantly influence the test.

Drugs that can affect the concentration of serotonin are monoamine oxidase inhibitors, lithium preparations, methyldopa, morphine and reserpine.

Low levels of serotonin may be associated with depressive syndromes.

Correlations between detected biochemical markers of patients diagnosed with Psoriasis (Data to be published in scientific journals)

Results To determine whether studied markers were significant statistically differences in patients who received some type of treatment, I analyzed these data using IBM SPSS Statistics 19.S software program. Significant results were obtained for the following markers in the treatment with Derma Stresclin:

5.6.1. A/G –albumin/globulin ratio

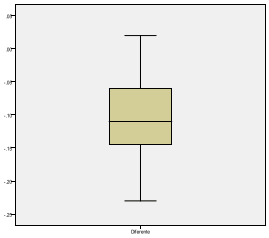
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
A/G differences	.143	7	.200*	.991	7	.996

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
A/G differences	7	100.0%	0	.0%	7	100.0%



b. **Step 2** - After applying parametric test Paired Samples T Test it *resulted in existence of statistically significant decreasing of the albumin / globulin ratio after applying the treatment* (t=3,406, 6 degrees of freedom, p=0,014<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	A/G pre-treatment	1.1700	7	.08307	.03140
	A/G post-treatment	1.0657	7	.05412	.02045

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	A/G pre-treatment & A/G post-treatment	7	.363	.423

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	A/G pre-treatment – A/G post-treatment	.10429	.08101	.03062	.02937	.17920	3.406	6	.014

5.6.2. Albumin

a. Step 1

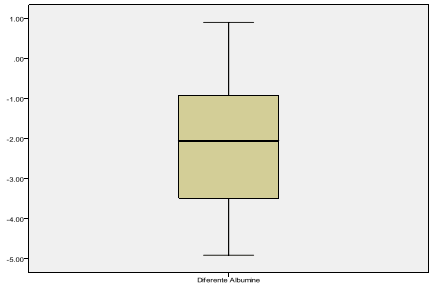
Tests of Normality

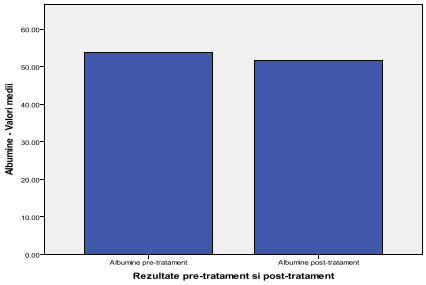
	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Albumin differences	.177	7	.200*	.937	7	.610

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Albumin differences	7	100.0%	0	.0%	7	100.0%

Step 2 - After applying parametric test Paired Samples T Test resulted the *existence of statistically significant*





decrease of albumin after treatment (t=2,627, 6 degrees of freedom, p=0,039<0,05).

Paired Samples Statistics

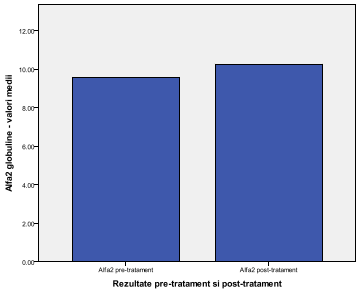
		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Albumin pre-treatment	53.8686	7	1.74568	.65981
	Albumin post-treatment	51.7414	7	1.09173	.41264

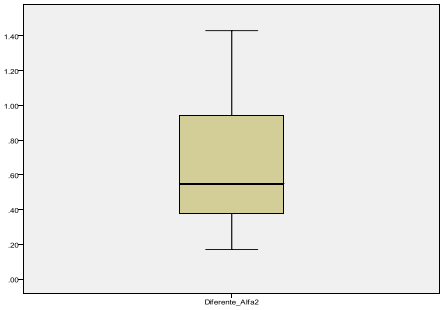
Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Albumin pre-treatment & Albumin post-treatment	7	-.092	.845

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Albumin pre-treatment - Albumin post-treatment	2.12714	2.14237	.80974	.14578	4.10851	2.627	6	.039





5.6.3. Alpha 2 globulins

a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Alpha2 differences	.208	7	.200*	.907	7	.378

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Alpha2 differences	7	100.0%	0	.0%	7	100.0%

b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of Alpha 2 globulin after treatment* (t=-3,901, 6 degrees of freedom, p=0,008<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Alpha2 pre-treatment	9.5743	7	1.81086	.68444
	Alpha2 post-treatment	10.2571	7	1.65505	.62555

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Alpha2 pre-treatment & Alpha2 post-treatment	7	.968	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Alpha2 pre treatment - Alpha2 post-treatment	-.68286	.46313	.17505	-1.11118	-.25453	-3.901	6	.008

5.6.4. Ionized calcium

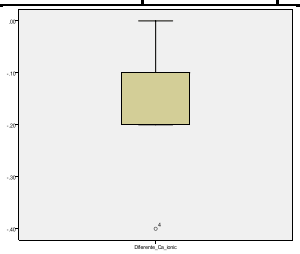
a. Step 1

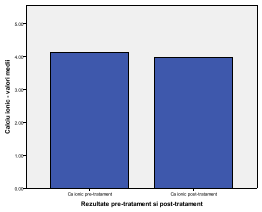
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Ionized calcium differences	.245	7	.200*	.888	7	.263

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Ionized calcium differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test *resulted in existence of a statistically significant decrease of ionized calcium after treatment* (t=3,267, 6 degrees of freedom, p=0,017<0,05). And after applying non-parametric test Related Samples Wilcoxon Signed Rank Test *also resulted in existence of statistically significant decrease* (p=0,026<0,05).

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between Ca ionic pre-tratament and Ca ionic post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.026	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Ionized calcium pre-treatment	4.1286	7	.11127	.04206
	Ionized calcium post-treatment	3.9714	7	.21381	.08081

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Ionized calcium pre-treatment & Ca ionic post-treatment	7	.881	.009

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Ionized calcium pre-treatment - Ionized calcium post-treatment	.15714	.12724	.04809	.03946	.27482	3.267	6	.017

5.6.5. Total cholesterol

a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Total cholesterol differences	.144	7	.200*	.968	7	.885

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Total cholesterol differences	7	100.0%	0	.0%	7	100.0%

b. Step 2 - After applying parametric test Paired Samples T Test , resulted the *existence of a statistically significant increase of total Cholesterolului after treatment* (t=-2,571, 6 degrees of freedom, p=0,042<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Total cholesterol pre-treatment	197.57	7	65.709	24.836
	Total cholesterol post-treatment	220.43	7	70.776	26.751

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Total cholesterol pre-treatment & Total cholesterol post-treatment	7	.943	.001

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Total cholesterol pre-treatment - Total cholesterol post-treatment	-22.857	23.519	8.889	-44.609	-1.106	-2.571	6	.042

5.6.6. Serum complement C4

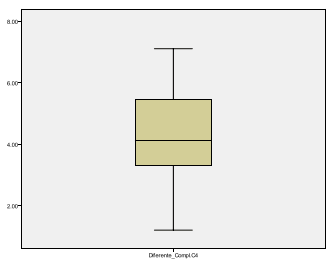
a. Step 1.

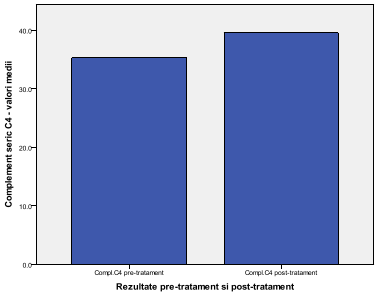
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Compl.C4 differences	.152	7	.200*	.970	7	.902

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Compl.C4 differences	7	100.0%	0	.0%	7	100.0%





b. Pasul 2 - After applying parametric test Paired Samples T Test, resulted the existence of a statistically significant increase of *serum complement C4 after treatment*(t=-5,691, 6 degrees of freedom, p=0,001<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Compl.C4 pre-treatment	35.271	7	12.7851	4.8323
	Compl.C4 post-treatment	39.543	7	12.0360	4.5492

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Compl.C4 pre-treatment & Compl.C4 post-treatment	7	.989	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Compl.C4 pre-treatment - Compl.C4 post-treatment	-4.2714	1.9830	.7495	-6.1054	-2.4374	-5.699	6	.001

5.6.7. Cortisol

a. Step 1

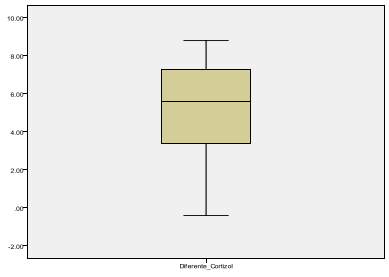
Tests of Normality

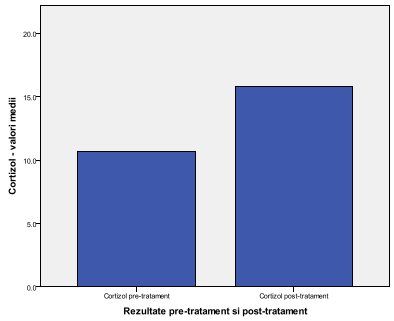
	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Cortisol differences	.254	7	.190	.909	7	.389

a. Lilliefors Significance Correction

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Cortisol differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test, *resulted the existence of a statistically significant increase of Cortisol after treatment*(t=-3,938, 6 degrees of freedom, p=0,008<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Cortisol pre-treatment	10.714	7	3.2059	1.2117
	Cortisol post-treatment	15.771	7	5.3783	2.0328

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Cortisol pre-treatment & Cortisol post-treatment	7	.802	.030

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Cortisol pre-treatment - Cortisol post-treatment	-5.0571	3.3975	1.2841	-8.1993	-1.9150	-3.938	6	.008

5.6.8. Gamma globulins

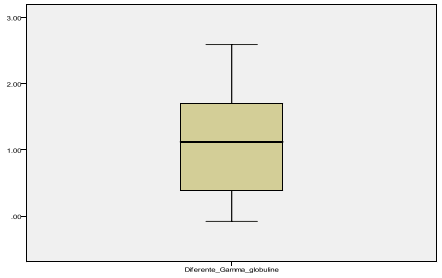
a. Step 1

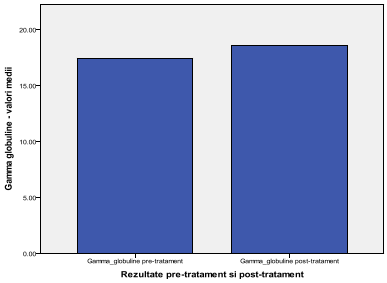
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Gamma globulins differences	.152	7	.200*	.962	7	.838

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Gamma globulins differences	7	100.0%	0	.0%	7	100.0%





. Step 2 - After applying parametric test Paired Samples T Test , resulted the existence of a statistically significant increase of *Gamma globulins after treatment* (t=-3,045, 6 degrees of freedom, p=0,023<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Gamma globulins pre-treatment	17.4229	7	2.55525	.96580
	Gamma globulins post-treatment	18.5386	7	2.54946	.96361

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Gamma globulins pre-treatment & Gamma globulins post-treatment	7	.928	.003

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Gamma globulins pre-treatment - Gamma globulins post-treatment	-1.11571	.96938	.36639	-2.01224	-.21919	-3.045	6	.023

Phosphoremia.

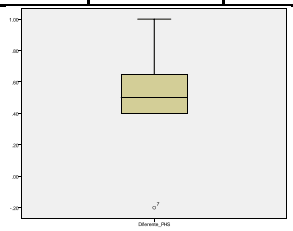
Step 1

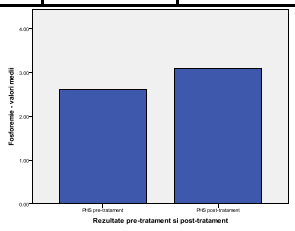
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
PHS differences	.265	7	.148	.929	7	.545

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
PHS differences	7	100.0%	0	.0%	7	100.0%





Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between PHS pre-tratament and PHS post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.028	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

b. Pasul 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of phosphoremia after treatment* (t=-3,501, 6 degrees of freedom, p=0,013<0,05). And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of phosphoremia* (p=0,028<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PHS pre-treatment	2.6143	7	.58716	.22193
	PHS post-treatment	3.1000	7	.45826	.17321

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PHS pre-tratament & PHS post-tratament	7	.780	.038

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PHS pre-treatment - PHS post-treatment	-.48571	.36710	.13875	-.82522	-.14620	-3.501	6	.013

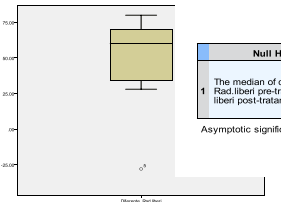
5.6.10. Free Radicals

a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Free radicals differences	.219	7	.200*	.864	7	.163

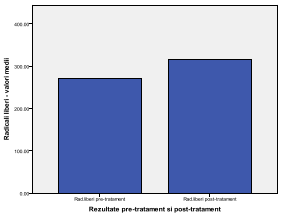
	Case **Processing Summary** Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Free radicals differences	7	100.0%	0	.0%	7	100.0%



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decis
1	The median of differences between Rad.liberi pre-tratament and Rad. liberi post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.034	Reject the null hypothe

Asymptotic significances are displayed. The significance level is .05.



b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of free radicals after treatment*(t=-3,206, 6 degrees of freedom, p=0,018<0,05). And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of Free radicals*(p=0,034<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Free radicals pre-treatment	270.8571	7	25.89355	9.78684
	Free radicals post-treatment	316.5714	7	47.07036	17.79092

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Free radicals pre-treatment & Free radicals post-treatment	7	.600	.154

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Free radicals pre treat- Free radicals post-treat.	-45.71429	37.72583	14.25902	-80.60486	-10.82371	-3.206	6	.018

5.6.11. TSH

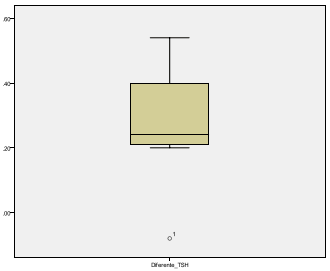
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
TSH differences	.212	7	.200*	.950	7	.726

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
TSH differences	7	100.0%	0	.0%	7	100.0%



b. Step 2 - After applying parametric test Paired Samples T Test , resulted the existence of a statistically significant increase of *TSH after treatment*(t=-3,642, 6 degrees of freedom, p=0,011<0,05 And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test resulted also that there is a statistically significant increase *of TSH*(p=0,028<0,05).

Paired Samples Statistics

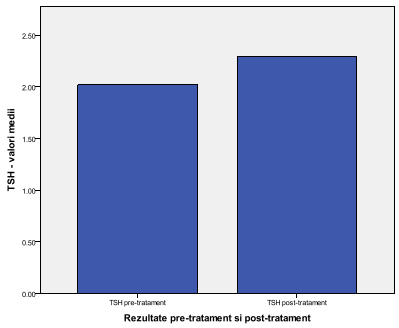
		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	TSH pre-treatment	2.0200	7	.78596	.29707
	TSH post-treatment	2.2943	7	.74447	.28138

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	TSH pre-treatment & TSH post-treatment	7	.968	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. De-vi ation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	TSH pre-tratament - TSH post-trat.	-.27429	.19924	.07530	-.45855	-.09002	-3.642	6	.011



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between TSH pre-tratament and TSH post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.028	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

Significant results for the following markers in the treatment with Stresclin Derma + Klinhaem:

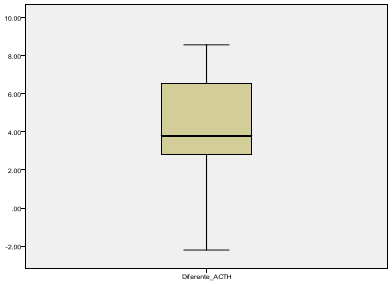
5.6.12. ACTH a. Step 1

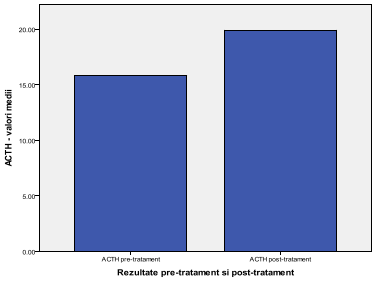
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
ACTH differences	.152	7	.200*	.959	7	.807

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
ACTH differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of ACTH after treatment*(t=-3,058, 6 degrees of freedom, p=0,022<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	ACTH pre-treatment	15.7900	7	7.97402	3.01390
	ACTH post-treatment	19.9157	7	5.23734	1.97953

Paired Samples Correlations

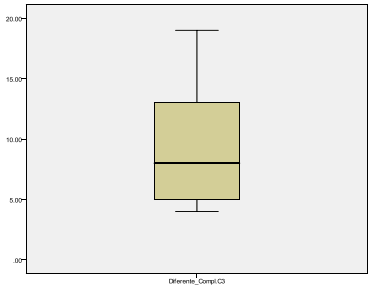
		N	Correlation	Sig.
Pair 1	ACTH pre-treatment & ACTH post-treatment	7	.937	.002

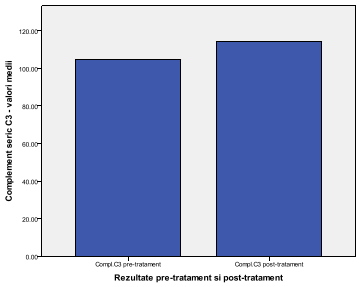
Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	ACTH pre-treatment - ACTH post-treatment	-4.12571	3.56960	1.34918	-7.42705	-.82438	-3.058	6	.022

5.6.13. Serum complement C3

a. Step 1





Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Compl.C3 differences	.181	7	.200*	.912	7	.411

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Compl.C3 differences	7	100.0%	0	.0%	7	100.0%

b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of serum complement C3 after treatment* (t=-4,480, 6 degrees of freedom , p=0,004<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Compl.C3 pre-treatment	104.7143	7	13.49956	5.10235
	Compl.C3 post-treatment	114.2857	7	14.25616	5.38832

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Compl.C3 pre-tratament & Compl.C3 post-tratament	7	.918	.003

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
	-9.57143	Pair 1	Compl.C3 pre-treatment - Compl.C3 post-treatment	-14.79925	-4.34361	-4.480	6	.004

5.6.14. Cortisol

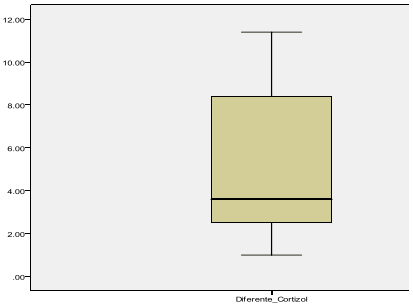
a. Step 1

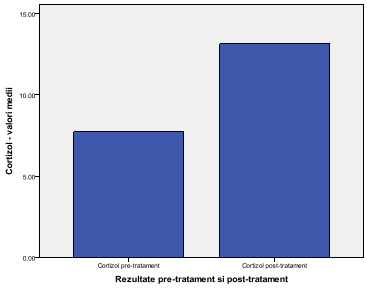
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Cortisol differences	.241	7	.200*	.883	7	.241

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Cortisol differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test, *resulted the existence of a statistically significant increase of Cortisol after treatment* (t=-3,472, 6 degrees of freedom, p=0,013<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Cortisol pre-tratament	7.7429	7	3.31203	1.25183
	Cortisol post-tratament	13.1429	7	5.29114	1.99986

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Cortisol pre-tratament & Cortisol post-tratament	7	.629	.131

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Cortisol pre-tratament - Cortisol post-tratament	-5.40000	4.11501	1.55533	-9.20575	-1.59425	-3.472	6	.013

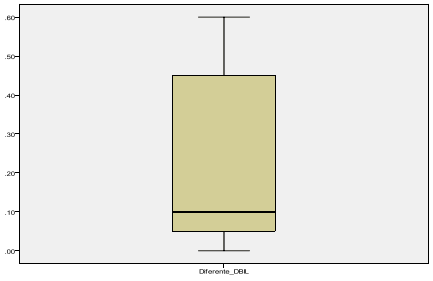
5.6.15. Direct bilirubin

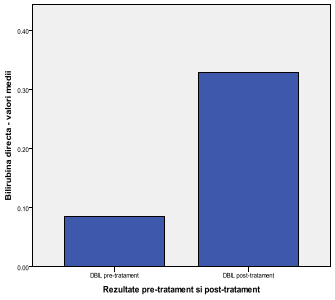
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
DBIL differences	.287	7	.084	.855	7	.135

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
DBIL differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test, *resulted the existence of a statistically significant increase of direct bilirubin after treatment* (t=-2,563, 6 degrees of freedom, p=0,043<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	DBIL pre-treatment	.0857	7	.03780	.01429
	DBIL post-treatment	.3286	7	.26277	.09932

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	DBIL pre-treatment & DBIL post-treatment	7	.384	.396

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	DBIL pre-treatment - DBIL post-treatment	-.24286	.25071	.09476	-.47473	-.01099	-2.563	6	.043

5.6.16. Blood sugar (glucose)

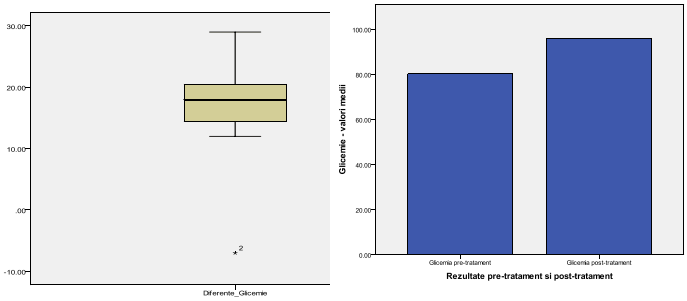
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
GLU differences	.260	7	.168	.862	7	.157

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
GLU differences	7	100.0%	0	.0%	7	100.0%



b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of blood glucose after treatment* (p=0,028<0,05).

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between Glicemia pre-tratament and Glicemia post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.028	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

5.6.17. Kalemia

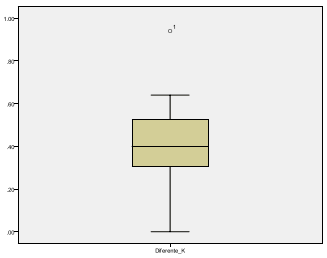
b. Step 1

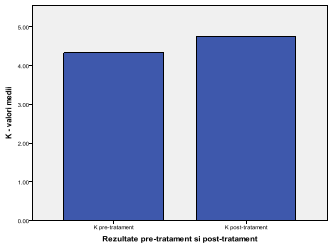
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
K differences	.239	7	.200*	.957	7	.794

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
K differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test, *resulted the existence of a statistically significant increase of kalemia after treatment* (t=-3,805, 6 degrees of freedom, p=0,009<0,01). And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of kalemia after treatment* (p=0,028<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	K pre-treatment	4.3286	7	.30575	.11556
	K post-treatment	4.7571	7	.35523	.13427

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	K pre-treatment & K post-treatment	7	.602	.152

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	K pre-treatment - K post-treatment	-.42857	.29802	.11264	-.70419	-.15295	-3.805	6	.009

5.6.18. LDL Cholesterol

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between K pre-tratament and K post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.028	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

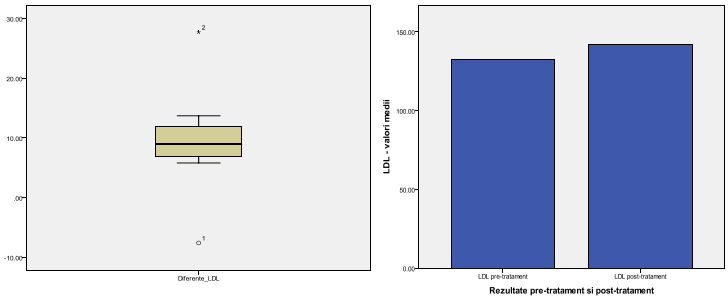
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
LDL differences	.218	7	.200*	.927	7	.526

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
LDL differences	7	100.0%	0	.0%	7	100.0%



b. Step 2 - And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of LDL colesterolulu, after treatment* (p=0,043<0,05).

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between LDL pre-tratament and LDL post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.043	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

5.6.19. Magnesemia

a. Step 1

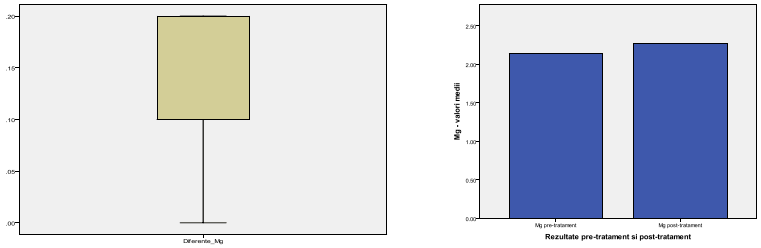
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Mg differences	.256	7	.182	.833	7	.086

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Mg differences	7	100.0%	0	.0%	7	100.0%

b. Step 2 - *After applying parametric test Paired Samples T Test , resulted the existence of a statistically significant increase of magnesemia after treatment* (t=-4,50, 6 degrees of freedom, p=0,004<0,01).



Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Mg pre-treatment	2.1429	7	.17182	.06494
	Mg post-treatment	2.2714	7	.22887	.08650

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Mg pre-treatment & Mg post-treatment	7	.969	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Mg pre-treatment - Mg post-treatment	-.12857	.07559	.02857	-.19848	-.05866	-4.500	6	.004

5.6.20. Natremiea

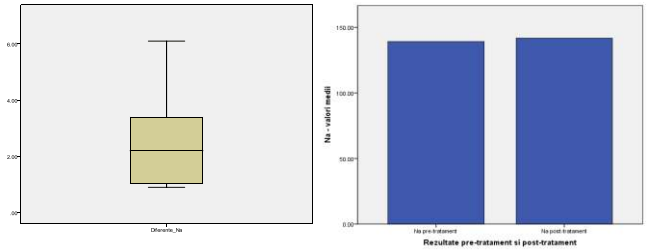
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Na differences	.252	7	.200*	.853	7	.131

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Na differences	7	100.0%	0	.0%	7	100.0%



b. Step 2 - After applying parametric test Paired Samples T Test , resulted the existence of a statistically significant increase of *natriemia after treatment* (t=-3,462, 6 degrees of freedom, p=0,013<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Na pre-treatment	139.1857	7	2.48893	.94073
	Na post-treatment	141.7714	7	3.24844	1.22780

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Na pre-treatment & Na post-treatment	7	.794	.033

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Na pre-treat Na post-treat	-2.58571	1.97605	.74688	-4.41325	-.75818	-3.462	6	.013

5.6.21. Phosphoremia

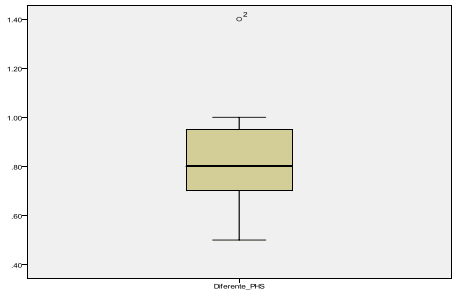
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
PHS differences	.170	7	.200*	.937	7	.612

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
PHS differences	7	100.0%	0	.0%	7	100.0%



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between PHS pre-tratament and PHS post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.018	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

Step 2 After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of phosphoremia after treatment*(t=-7,725, 6 degrees of freedom, p<0,001). And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test resulted also that there is a statistically significant increase of *phosphoremia after treatment*(p=0,018<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PHS pre- treatment	2.8571	7	.37796	.14286
	PHS post- treatment	3.7143	7	.24785	.09368

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PHS pre- treatment & PHS post- treatment	7	.630	.129

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PHS pre-treat.PHS post-treat.	-.85714	.29358	.11096	-1.12866	-.58562	-7.725	6	.000

5.6.22. TSH

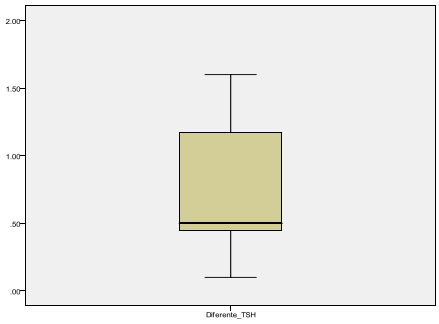
a. Step 1

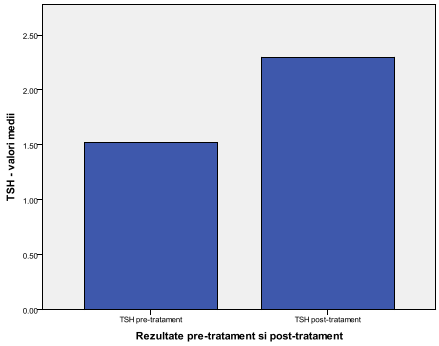
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
TSH differences	.266	7	.144	.924	7	.505

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
TSH differences	7	100.0%	0	.0%	7	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of TSH, after treatment*(t=-3,790, 6 degrees of freedom, p=0,009<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	TSH pre-treatment	1.5243	7	.57526	.21743
	TSH post-treatment	2.3000	7	.90591	.34240

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	TSH pre-treatment & TSH post-treatment	7	.824	.023

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	TSH pre-treatment- TSH post- treatment	-.77571	.54148	.20466	-1.27650	-.27493	-3.790	6	.009

Differences in statistical results, compared between the treatment with Stresclin Derma and the treatment with Stresclin derma associated with Klinhaem

5.6.23. ACTH

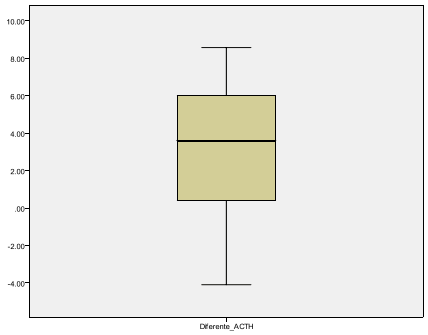
a. Step 1

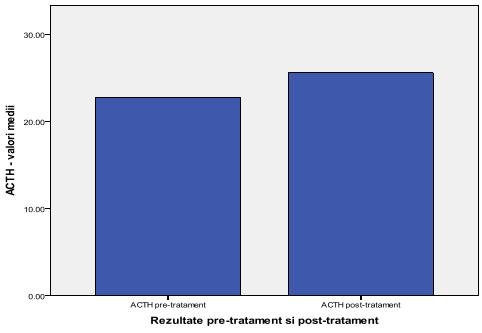
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
ACTH differences	.201	14	.130	.927	14	.273

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
ACTH differences	14	100.0%	0	.0%	14	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of ACTH after treatment*(t=-2,683, 13 degrees of freedom, p=0,019<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	ACTH pre-treatment	22.7564	14	12.93286	3.45645
	ACTH post-treatment	25.5793	14	9.69933	2.59225

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	ACTH pre-treatment & ACTH post-treatment	14	.980	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	ACTH pre-treatment - ACTH post-treatment	-2.82286	3.93666	1.05212	-5.09582	-.54989	-2.683	13	.019

5.6.24. ALT

a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
ALT differences	.138	14	.200*	.972	14	.900

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
ALT differences	14	100.0%	0	.0%	14	100.0%

b. Step 2 - After applying parametric test Paired Samples T Test , resulted the existence of a statistically significant decrease of *ALT after treatment*(t=2,830, 13 degrees of freedom, p=0,014<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	ALT pre-treatment	30.0000	14	19.43431	5.19404
	ALT post-treatment	25.2143	14	17.99649	4.80976

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	ALT pre-treatment & ALT post-treatment	14	.946	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	ALT pre-treatment - ALT post-treatment	4.78571	6.32673	1.69089	1.13277	8.43866	2.830	13	.014

5.6.25. Alpha 2

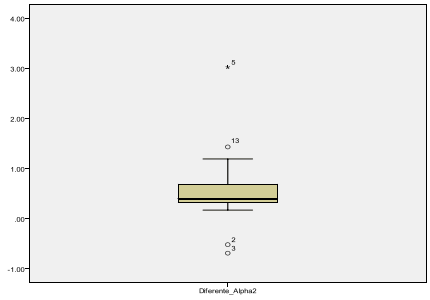
a. Step 1 - After applying the test of normality of distribution (Kolmogorov Smirnov and Shapiro-Wilk) resulted values of p lower (!) than 0.05, which *implies a different distribution other than normal data for differences between the values of pre-treatment and post-treatment.*

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Alpha2 differences	.244	14	.024	.853	14	.024

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Alpha2 differences	14	100.0%	0	.0%	14	100.0%



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between ALPHA2 pre-tratament and ALPHA2 post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.028	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of Alpha 2 globulins after treatment* (p=0,028<0,05).

5.6.26. Total Cholesterol

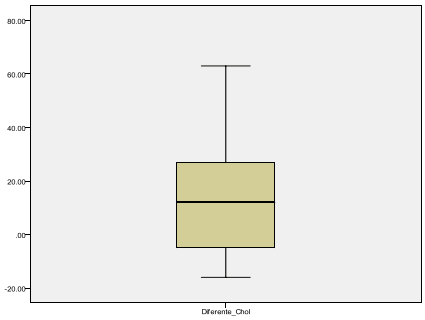
a. Step 1

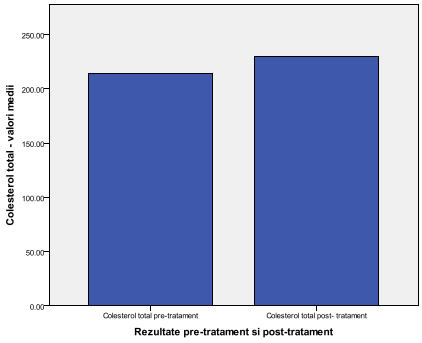
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Chol differences	.126	14	.200*	.950	14	.556

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Chol differences	14	100.0%	0	.0%	14	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of total cholesterol after treatment*(t=-2,431, 13 degrees of freedom, p=0,03<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	total cholesterol pre-treatment	214.6429	14	50.23423	13.42566
	total cholesterol post-treatment	229.6429	14	55.47225	14.82558

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	total cholesterol pre-treatment & total cholesterol post-treatment	14	.909	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	total cholesterol pre-treatment - total cholesterol post-treatment	-15.00000	23.08513	6.16976	-28.32896	-1.67104	-2.431	13	.030

5.6.27. Calcium

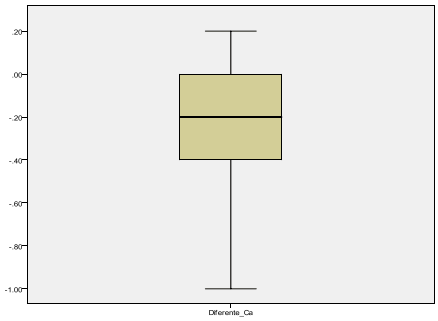
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Ca differences	.221	14	.063	.890	14	.080

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Ca differences	14	100.0%	0	.0%	14	100.0%



b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant decrease of calcium after treatment*(t=3,161, 13 degrees of freedom, p=0,008<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Ca pre-treatment	9.7143	14	.25071	.06701
	Ca post-treatment	9.4714	14	.28670	.07662

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Ca pre-treatment & Ca post-treatment	14	.434	.121

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Ca pre-treatment - Ca post-treatment	.24286	.28747	.07683	.07688	.40884	3.161	13	.008

5.6.28. Ionized Ca

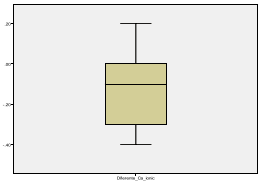
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Ionized Ca differences	.151	14	.200*	.958	14	.697

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Ionized Ca differences	14	100.0%	0	.0%	14	100.0%



b. Pasul 2 - After applying parametric test Paired Samples T Test , r*esulted the existence of a statistically significant decrease of*

Ionized Ca after treatment(t=2,852, 13 degrees of freedom, p=0,014<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Ionized Ca pre-treatment	4.1500	14	.12247	.03273
	Ionized Ca post-treatment	4.0143	14	.20327	.05433

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Ionized Ca pre-treatment & Ionized Ca post-treatment	14	.494	.072

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Ca ionic pre-treatment - Ca ionic post-treatment	.13571	.17805	.04759	.03291	.23852	2.852	13	.014

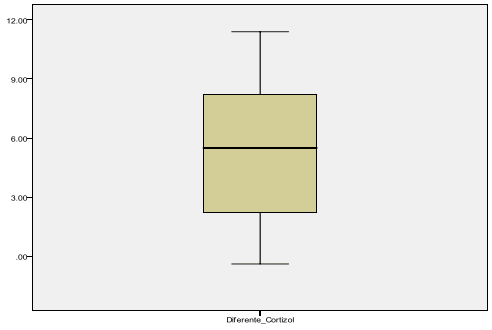
5.6.29. Cortisol a. Step 1

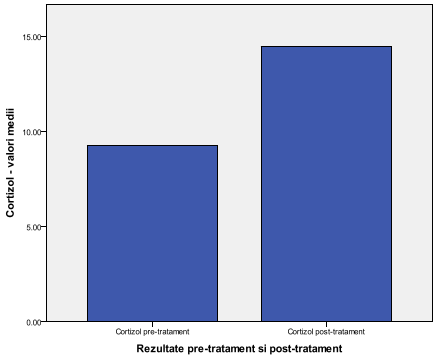
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Cortisol differences	.105	14	.200*	.967	14	.838

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Cortisol differences	14	100.0%	0	.0%	14	100.0%





b. Step 2 - After applying parametric test Paired Samples T Test , resulted the existence of a statistically significant increase of *cortisol after treatment* (t=-5,390, 13 degrees of freedom, p<0,001).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Cortisol pre-treatment	9.2286	14	3.49052	.93288
	Cortisol post-treatment	14.4571	14	5.30395	1.41754

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Cortisol pre-treatment & Cortisol post-treatment	14	.733	.003

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Cortisol pre-treatment - Cortisol post-treatment	-5.22857	3.62967	.97007	-7.32428	-3.13286	-5.390	13	.000

5.6.30. Direct bilirubin

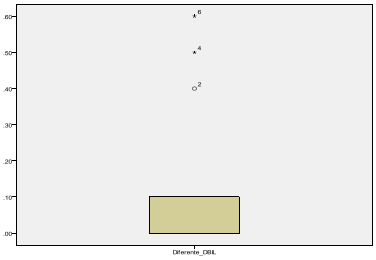
a. Step 1 - After applying the test of normality of distribution (Kolmogorov Smirnov and Shapiro-Wilk) *resulted values of p lower (!) than 0.05, which implies a different distribution other than normal data for differences between the values of pre-treatment and post-treatment.*

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
DBIL differences	.360	14	.000	.637	14	.000

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
DBIL differences	14	100.0%	0	.0%	14	100.0%



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between DBIL pre-tratament and DBIL post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.042	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of direct bilirubin after treatment* (p=0,042<0,05).

5.6.31. LDL

a. Step 1 - After applying the test of normality of distribution (Kolmogorov Smirnov and Shapiro-Wilk) *resulted values of p lower (!) than 0.05, which implies a different distribution other than normal data for differences between the values of pre-treatment and post-treatment*. But further analysis of data on the different outlook revealed the existence of several "outliers" with significant influence, resulting the need of nonparametric Wilcoxon test application to step 2.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
LDL differences	.222	14	.061	.918	14	.207

a. Lilliefors Significance Correction

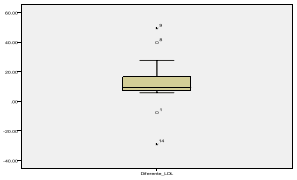
Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
LDL differences	14	100.0%	0	.0%	14	100.0%

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between LDL pre-tratament and LDL post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.019	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.



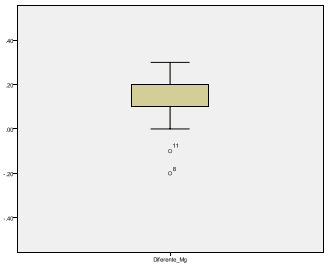
b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of LDL cholesterol after treatment* (p=0,019<0,05).

5.6.32. Mg

a. Step 1 - After applying the test of normality of distribution (Kolmogorov Smirnov and Shapiro-Wilk) resulted values of p lower (!) than 0.05, But further analysis of data on the different outlook revealed the existence of several "outliers" with significant influence, resulting the need of nonparametric Wilcoxon test application to step 2.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Mg differences	.308	14	.001	.880	14	.059



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between MG pre-tratament and MG post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.028	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Mg differences	14	100.0%	0	.0%	14	100.0%

b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of magnesemia after treatment* (p=0,028<0,05).

5.6.33. Na

a. Step 1 - After applying the test of normality of distribution (Kolmogorov Smirnov and Shapiro-Wilk) resulted values of p lower (!) than 0.05, which implies a different distribution other than normal data for differences between the values of pre-treatment and post-treatment. But further analysis of data on the different outlook did not reveal the existence of several "outliers" with significant influence, resulting the need of parametric Student test application to step 2.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Na differences	.142	14	.200*	.958	14	.693

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Na differences	14	100.0%	0	.0%	14	100.0%

b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of natremia after treatment* (t=-4,049, 13 degrees of freedom, p=0,001<0,01).

Paired Samples Statistics

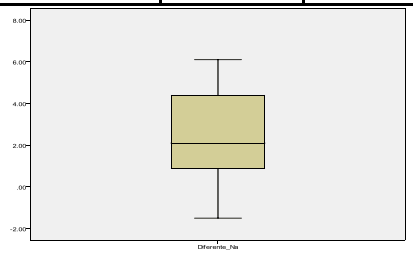
		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Na pre-treatment	139.7571	14	2.22977	.59593
	Na post-treatment	142.1500	14	2.68808	.71842

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Na pre-treatment & Na post-treatment	14	.610	.021

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Na pre-treatment - Na post-treatment	-2.39286	2.21132	.59100	-3.66964	-1.11608	-4.049	13	.001



5.6.34. PHS

a. Step 1

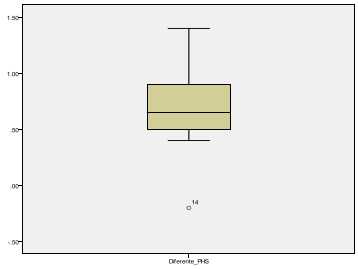
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
PHS differences	.162	14	.200*	.953	14	.610

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
PHS differences	14	100.0%	0	.0%	14	100.0%

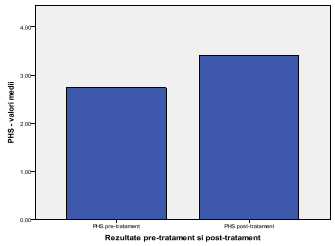
b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of phosphoremia after treatment* (t=-6,735 13 degrees of freedom, p<0,001). And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test r*esulted also that there is a statistically significant increase of phosphoremia after treatment* (p=0,001<0,01).



Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between PHS pre-tratament and PHS post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.001	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.



Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PHS pre-treatment	2.7357	14	.49085	.13119
	PHS post-treatment	3.4071	14	.47631	.12730

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PHS pre-treatment & PHS post-treatment	14	.703	.005

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PHS pre-treatment - PHS post-treatment	-.67143	.37299	.09969	-.88679	-.45607	-6.735	13	.000

5.6.35. PTH

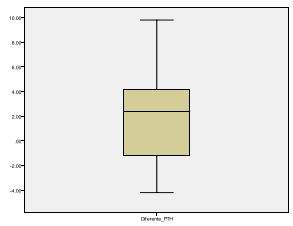
a. Step 1

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
PTH differences	.138	14	.200*	.973	14	.918

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
PTH differences	14	100.0%	0	.0%	14	100.0%



b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of*

PTH after treatment (t=-2,201, 13 degrees of freedom, p=0,046<0,05).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PTH pre-treatment	28.2714	14	13.35149	3.56833
	PTH post-treatment	30.5214	14	12.38561	3.31019

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PTH pre-treatment & PTH post-treatment	14	.959	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PTH pre-treatment - PTH post-treatment	-2.25000	3.82557	1.02243	-4.45882	-.04118	-2.201	13	.046

5.6.36. Free radicals

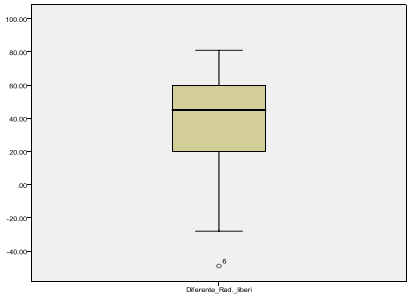
a. Step 1.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Free radicals differences	.166	14	.200*	.898	14	.105

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Free radicals differences	14	100.0%	0	.0%	14	100.0%



b. Step 2 - After applying parametric test Paired Samples T Test , *resulted the existence of a statistically significant increase of free radicals after treatment*(t=-3,618 13 degrees of freedom, p=0,003<0,01). And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of free radicals after treatment*(p=0,008<0,01).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Free radicals pre-treatment	288.0714	14	80.46066	21.50402
	Free radicals post-treatment	325.7857	14	62.93119	16.81907

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Free radicals pre-treatment & Free radicals post-treatment	14	.880	.000

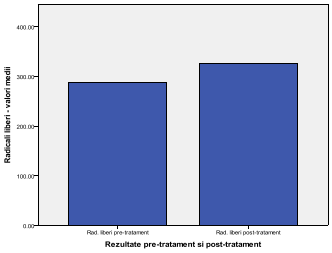
Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Free radicals pre-treatment - Free radicals post-treatment	-37.71429	39.00380	10.42421	-60.23441	-15.19416	-3.618	13	.003

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between Rad. liberi pre-tratament and Rad. liberi post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.008	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.



5.6.37. TSH

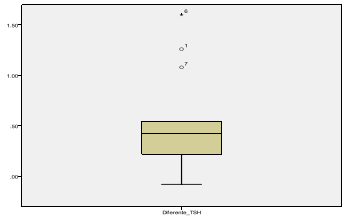
a. Step 1 - After applying the test of normality of distribution (Kolmogorov Smirnov and Shapiro-Wilk) *resulted values of p lower (!) than 0.05, which implies a different distribution other than normal data for differences between the values of pre-treatment and post-treatment*. But further analysis of data on the different outlook revealed the existence of several "outliers" with significant influence, resulting the need of nonparametric Wilcoxon test application to step 2.

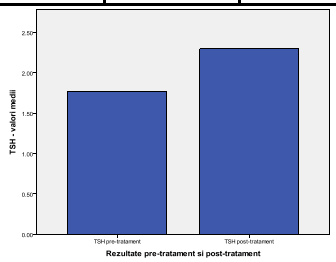
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
TSH differences	.273	14	.006	.868	14	.039

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
TSH differences	14	100.0%	0	.0%	14	100.0%





Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between TSH pre-tratament and TSH post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.001	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of TSH after treatment* (p=0,001<0,01).

5.6.38. Gamma globulins

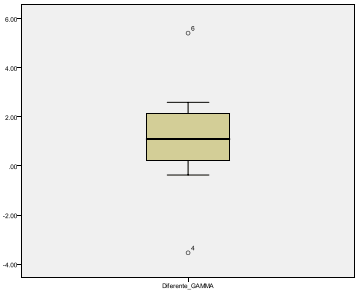
a. Step 1

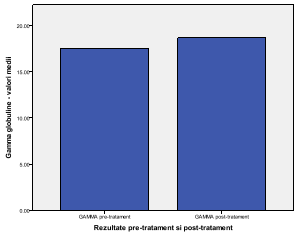
Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
GAMMA differences	.159	14	.200*	.934	14	.342

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
GAMMA differences	14	100.0%	0	.0%	14	100.0%





Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The median of differences between GAMMA pre-tratament and GAMMA post-tratament equals 0.	Related-Samples Wilcoxon Signed Rank Test	.030	Reject the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

b. Step 2 – And after applying the nonparametric test Related Samples Wilcoxon Signed Rank Test *resulted also that there is a statistically significant increase of gamma globulins after treatment* (p=0,03<0,05).

In conclusion, following statistical analysis of the values results of laboratory investigations conducted on the two groups of patients, after 6 weeks of treatment, following significant increases or decreases are noticed :

A) **Group 1 (treated with Stresclin Derma in association with Klinhaem) - increased:**
- ACTH, C3, Cortisol., DBIL, GLU, K, LDL Col., Mg, Na, PHS and TSH.

B) **In group 2 (treated with Stresclin Derma) - increased:**
- Alpha 2, CHOL, Compl.C4, Cortisol s., Gamma, PHYS, Rad. free TSH.

- decreased - A / G, ALB, CA.

C**) Differences in results of statistical comparison between group 1 (treated withStresclin Derma in association with Klinhaem) and group 2 (treated with Stresclin Derma):**

- are higher - ACTH, alpha 2, CHOL, serum Cortisol., DBIL, Mg, Na, PHS, PTH, free rad., TSH, Gamma.

- are lower: - ALT, CA, CA .Ion, LDL.

Correlations between biochemical markers of patients diagnosed with psoriasis

Correlations between biochemical and hormonal markers in patients from both groups (Group 1 treated with Stresclin Derma and Klinhaem and group 2 received only Stresclin Derma)

The data was processed using two tests, one parametric and other nonparametric - Pearson and Spearman.

S.1. Correlations synthesis between hormonal and biochemical markers in patients from both groups (Group 1 treated with Stresclin Derma and Klinhaem and group 2 treated with Stresclin Derma)

Relevant results were obtained in two cases:

a. In 7 of these pairs, the correlation obtained is positive (two members of the markers pair decrease or increase in the same time):

1. **ALT-CHOL** - Pearson correlation coefficient 0,645 to p=0.013, Spearman correlation coefficient 0,585 to p=0,028; Hepatotoxicity.
2. **MG - PHS -** Pearson correlation coefficient 0,569 to p=0,034, Spearman correlation coefficient 0,566 to p=0,035; beneficial action .
3. **NA - MG -** Pearson correlation coefficient 0,566 to p=0,035, Spearman correlation coefficient 0,570 to p=0,033; beneficial action.
4. **Serotonina - MG** - Pearson correlation coefficient 0,536 to p=0,048, Spearman 0,550 to p=0,041; beneficial action.
5. **Serotonina - PHS** - Pearson correlation coefficient 0,677 to p=0,008, Spearman 0,633 to p=0,015; beneficial action.
6. **CHOL – LDL** - Pearson correlation coefficient 0,688 to p=0,006, Spearman 0,625 l p=0,017; Hepatotoxicity.
7. **ACTH - Cortizol** - Pearson correlation coefficient 0,579 to p=0,03, Spearman 0,562 to p=0,037. beneficial action.

b. Pentru 2 dintre aceste perechi, s-au obținut corelații negative (când unul dintre markeri creşte, celălalt scade şi invers):

1. **GAMMA - CHOL** - Pearson correlation coefficient -0,617 to p=0,019, Spearman -0,558 to p=0,038; beneficial action.
2. **ACTH-ALT -** Pearson correlation coefficient -0,591 to p=0,026, Spearman -0,623 to p=0,017. beneficial action.

S.2. Sinteza corelațiilor dintre markerii biochimici și hormonali la pacienții lotului 1 (tratați cu Stresclin Derma și Klinhaem)

a. For 22 of these pairs, obtained correlation are positive (two members of the markers pair decrease or increase at the same time):

1. **ALT – AST** - p=0.03275 1<0.05 Spearman correlation coefficient: 0.795
2. **Cr-S – AST** - p=0.03993 1<0.05 Spearman correlation coefficient:..... 0.777
3. **GGT- AST** - p=0.03993 1<0.05 Spearman correlation coefficient:..... 0.777
4. **NA – AST -** p= 0.02636 1<0.05 Spearman correlation coefficient:..... 0.768
5. **TSH – GLU** - p=0.02345 1<0.05 Spearman correlation coefficient: 0.813
6. **UREA - CR**-S - p=0.03624 1<0.05 Spearman correlation coefficient:..... 0.786
7. **ALP – UREA** - p=0.01370 1<0.05 Spearman correlation coefficient: 0.857
8. **NA - CR-S** - p=0.01370 1<0.05 Spearman correlation coefficient: 0.857
9. **Alpha 2** – **GGT** - p=0.03624 1<0.05 Spearman correlation coefficient: 0.786
10. **ALP – MG** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.991
11. **Beta – CA** - p=0.04790 1<0.05 Spearman correlation coefficient: 0.759
12. **AT- TG-B Risk** - p=0.01370 1<0.05 Spearman correlation coefficient: 0.857
13. **PHS – HDLD** - p=0.02946 1<0.05 Spearman correlation coefficient: 0.804
14. **UREA - AST** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.884

15. **Compl.C4** – Beta - p=< 0.001 <0.05 Spearman correlation coefficient: 0.955
16. **DBIL- LD-P** - p=< 0.007 <0.05 Spearman correlation coefficient: 0.802
17. **GGT – URIC** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.929
18. **NA – UREa** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.893
19. **Beta – CHOL** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.893
20. **Compl.C4 – CHOL** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.929
21. **Free T4 – ALT** - p=0.023 1 <0.05 Spearman correlation coefficient: 0.917
22. **Free T4 – PHS** - p=0.023 1<0.05 Spearman correlation coefficient: 0.806

b. For nine of these pairs, negative correlations were obtained (when one of the markers increases, the other decreases and vice versa):

1. **TP – CA. Ion** - p=< 0.001 <0.05 Spearman correlation coefficient: .....-0.92
2. **Beta – AMY7** - p=< 0.001 <0.05 Spearman correlation coefficient: .....-0.92
3. **ACTH – GGT** - p=< 0.001 <0.05 Spearman correlation coefficient: .....-0.893
4. **GLU – CK** - p=0.01820 1<0.05 Spearman correlation coefficient: ..... -0.839
5. **Albumins – UREA** - p=0.03624 1<0.05 Spearman correlation coefficient: .....-0.786
6. **ACTH – Uric -** p=< 0.001 <0.05 Spearman correlation coefficient: .....-0.964
7. **Compl.C3 – K** - p=6.807e-3 1<0.05 Spearman correlation coefficient: .....-0.893
8. **Gamma - Alpha 1** - p=< 0.001 <0.05 Spearman correlation coefficient:..... -0.964
9. **Compl.C4 - AMY 7** - p=0.02345 1<0.05 Spearman correlation coefficient:..... -0.821

S.3. Correlations synthesis between hormonal and biochemical markers in patients from group 2 (Treated with Stresclin Derma)

a. For 26 of these pairs, the obtained correlation is positive (two members of the pair of markers decrease or increase at the same time):

1. **GLU - LD-P** - p=0.0438 1<0.05 Spearman correlation coefficient: 0.768
2. **AST – ALT** - p=0.01586 1<0.05 Spearman correlation coefficient: 0.848
3. **CHOL – ALT** - p=0.02345 1<0.05 Spearman correlation coefficient:..... 0.821
4. **CHOL - TG-B** - p=0.02345 1<0.05 Spearman correlation coefficient:..... 0.821
5. **GGT - CHOL** - p=0.04790 1<0.05 Spearman correlation coefficient:..... 0.759
6. **GGT - TG-B** - p=< 0.001 <0.05 Spearman correlation coefficient: 0.955
7. **TBIL - CR-S** - =0.02946 1<0.05 Spearman correlation coefficient: 0.804
8. **LDL – CHOL** - =< 0.001 <0.05 Spearman correlation coefficient: 0.964
9. **At Risk. – CHOL**- p=< 0.001 <0.05 Spearman correlation coefficient: 0.964
10. **At. Risk. - TG-B** - p=2.519e-3 1<0.05 Spearman correlation coefficient:.....0.929
11. **Beta – CHOL** - p=0.01370 1<0.05 Spearman correlation coefficient: 0.857
12. **Free Rad. - CR-S** - p=0.03993 1<0.05 Spearman correlation coefficient:.....0.777
13. **LD-P – GGT** - p=0.01820 1<0.05 Spearman correlation coefficient:.....0.839
14. **MG – TBIL** - p=0.02946 1<0.05 Spearman correlation coefficient: 0.804
15. **At Risk– GGT** - p=8.274e-3 1<0.05 Spearman correlation coefficient: 0.884
16. **C re. Prot.- HDLD** - p=2.519e-3 1<0.05 Spearman correlation coefficient: ..0.929
17. **ALB – TP** - p=1.813e-3 1<0.05 Spearman correlation coefficient:.....0.938
18. **MG – Fe** - p=0.01370 1<0.05 Spearman correlation coefficient:.....0.857
19. **Mg – Na** - p=0.01370 1<0.05 Spearman correlation coefficient: 0.857
20. **K – Fe** - p=9.909e-3 1<0.05 Spearman correlation coefficient: 0.875
21. **Free Rad. – MG** - p=0.03275 1<0.05 Spearman correlation coefficient:.....0.795
22. **At Risc– LDL** - p=2.519e-3 1<0.05 Spearman correlation coefficient: 0.929
23. **Alpha 2 – LDL** - p=0.03624 1<0.05 Spearman correlation coefficient: 0.786
24. **Beta – LDL** - p=0.02345 1<0.05 Spearman correlation coefficient:..... 0.821
25. **Free Rad.** – PTH - p=5.504e-3 1<0.05 Spearman correlation coefficient:... 0.902
26. **Free T4** - Prot.Totale - p=2.519e-3 1<0.05 Spearman correlation coefficient:..0.929

b. In 7 of these pairs, negative correlations were obtained (when one of the markers increases, the other decreases and vice versa):

1. **HDLD – GLU** - p= 8.274e-3<0.05 Spearman correlation coefficient:.....-0.884
2. **C Re. Prot.– GLU** - p=0.01172<0.05 Spearman correlation coefficient: ..... -0.866
3. **C Re. Prot.- LD-P** -p=0.02073 1<0.05 Spearman correlation coefficient: .....-0.830
4. **PTH – HDLD** - p=0.02345 1<0.05 Spearman correlation coefficient: .....-0.821
5. **C Re. Prot.– GGT** - p=1.813e-3 1<0.05 Spearman correlation coefficient: .....-0.938
6. **GLU - LD-P -** p=0.02636 1<0.05 Spearman correlation coefficient: .....-0.813
7. **Beta – Gamma** - p=0.02073 1<0.05 Spearman correlation coefficient: .....-0.830

CONCLUSIONS

Analyzing the data obtained in this thesis, the following assessment, comments and conclusions can be formulated:

Experiment 1. Shows principles and a technical variant of harvesting and processing of skin biopsy specimens, technique which can be improved by: improving the method of skin slicing, improving materials used as gauges, use of small but efficient thermostats, bath time shortening.

Experiment 2. Corroborating lysosomal changes with augmentation in the disease, but also with posttherapeutic free radicals, noted in a previous paper, it can promote the hypothesis that the pathogenesis of psoriasis vulgaris includes lysosomes involvement, closely related to the release of free radicals.

Experiment 3. It can be said that the treatment recommended for patients with psoriasis, even if it has definite indication in arthropathyc form and dysmetabolic affections, produces in the first phase mobilization of possible deposits of free radicals that can cause negative changes of symptoms and of some laboratory biochemical parameters or of inflammation.

Experiment 4. Treatment of psoriasis with common herbal preparations "Stresclin derma" and "Klinhaem" can cause the disappearance of specific lesions of psoriasis vulgaris.

Combined therapeutic effect of the two products can be explained by the complementary action of two means: immunological ("Klinhaem") and neuro-endocrine ("Stresclin derma").

The main biochemical changes observed in both patients are increased cortisol levels, TSH and blood glucose. Noteworthy is the change in the value of free radicals, which increased in patients with low initial values, but decreased significantly in patients with very high initial values.

Experiment 5. Analyzing graphs in conjunction with the studied literature, we can draw the following conclusions:

- Treatment caused marked reduction of initial skin lesion, reaching an average area of residual psoriatic lesions for all patients of 9.77% lower in the first group (5.4%) compared to the second group (15%). Reduction of skin lesions is more evident in men, encompassing an area of the remaining lesion of 108 cm² (20 cm² for the first group and 88 cm² for the second group) compared to 170 cm² (64 cm² for the first group and 106 cm² for the second group) in women,
- Treatment with both products causes significant variations of biochemical and hormonal studied values.
- It is found predominantly increasing average values, evidenced in group 1 (treated with Stresclin derma and Klinhaem), which accounted for an average increase by 14.42% and an average decrease by only 3.77%, compared with group 2 (treated only with Stresclin derma), which earned an average increase by 10.85% and an average decrease by 6.25%. Adding the two values obtained for each group, it can be said that the combined treatment resulted in an increase of the average value by 10.65% analyzes, and simple therapy, their average value increased by only 4.6%.

In terms of statistical calculations assessments, it can be concluded:

a) It emerged clearly the existence of significant differences ante / post-treatment: glucose, ALT, Total Cholesterol, Ca, Ca ion, Mg, phosphoremia, Na, LDL, Alpha2 globulin, free radicals, TSH and serum cortisol.

b) In several cases, results have come out well in some tests (indicating significant differences ante / post-treatment), but require reservations due to the presence of different results from other tests, which pursued the same goal. Therefore, may be considered to be positive the following parameters: PTH, ACTH and Serotonin.

c) I found significant correlations between certain parameters developments after treatment, respectively: 7 pairs obtained positive correlation (both members of the pair of markers decrease or increase at the same time):

ALT-CHOL (liver injury), MG - PHS (beneficial), NA - MG (beneficial),
Serotonin - Mg (beneficial), Serotonin - PHS (beneficial);
CHOL - LDL (liver injury), ACTH - Cortisol (beneficial) .

d) I got two pairs of negative correlations (when one of the markers increases, the other decreases and vice versa): GAMMA - CHOL (beneficial) and ACTH-ALT (beneficial).

e) For the two treatment regimens, results for statistically significant differences are the following:

- For Stresclin derma + Klinhaem scheme:- ACTH - Compl.C - Cortisol - DBIL,
(11 analysis) - Glu, - K, - LDL, - Mg, - Na, - PHS,- TSH.
- For stresclin derma scheme:- AG, - Albumins, - Alpha 2, - Ca ion,- Chol,- Compl C4,
(11 analysis) - Cortisol, - Gamma,- PHS,- Free Rad., - TSH.

e) For some markers: cortisol, TSH and PHS there are significant differences before and after treatment, in both treatment regimens.

The highest average values, which exceeded by minimum 10% the initial average values, before treatment, they are in the following order:

Cortisol

It has the highest average value after treatment, increasing by 69.67% for the first group and by 47.38% for the second group.

By working with other data presented in the paper, I may assume that the beneficial mechanism of action of the two drugs, but especially Klinhaem's, is to stimulate endogenous serum cortisol production. Increased levels of this hormone may explain the favorable effect, not only anti-inflammatory, but also changes of some other blood values. Administered preparations action is due to extracts from the plants mentioned in Chapter 5.5.2., which have the accumulated effect of antipsoriatic, immunomodulatory, anti-inflammatory, antioxidant and fight free radicals.

CK (creatine kinase)

It increased compared to the average reference by 17% for the first group and by 56.10% for the second group. Due to its release from muscle, I suppose that this increase is the result of its participation in the energy process of muscle contraction. The enzyme is involved in the reversible reaction of phosphorylation and dephosphorylation of the creatine; creatine phosphate (macroergic compound) being an energy reserve in muscle.

The fact that the average values of the enzyme are much higher in patients from group 2, which had been treated only with Stresclin derma, extract from the tuberous roots of Smilax china (250 mg / 5 ml) contained in Klinhaem can exert an inhibitory effect of the muscle creatine kinase release.

Serum TSH (Thyroid stimulating hormone)

It increased compared to the average reference for the first group by 51.31% and by 13.36% for the second group.

It is known that corticosteroid hormones may increase serum TSH.

DBIL levels (direct bilirubin)

It increased compared to the average reference for the first group by 44.44% and by 0% for the second group.

The increase of this biochemical parameter in patients treated with both products could be explained by the occurrence of a degree of liver cholestasis, determined by the action from plant extracts contained in the Klinhaem product.

Serum PHS (serum phosphorus)

It increased compared to the average reference for the first group by 29.72% and by 18.77% for the second group.

The stimulative action of serum phosphorus formation by glucocorticoid hormones is proved in this case. Also, it`s proved the effect of decreasing serum calcium (by -2.18% for the first group and by -2.95% for the second group),together with phosphoremy increasing.

TBIL (serum total bilirubin)

It increased compared to the average reference by 27.94% for the second group and it decreased below the average reference by -3.06% for the first group.

Increasing the average value for this parameter in group 2, patients who received treatment only with Stresclin derma, can be explained by protective immunomodulation effect of extract from the roots of cordiofolia Rubio (612.5 mg / 5ml) contained in the Klinhaem product.

Serum ACTH (adrenocorticotropic hormone)

It increased compared to the average reference by 26,15% for the first group and by just 5.11% for the second group.

Given the increase in plasma cortisol, normally, the average amount of ACTH after treatment should be lower than that recorded value before treatment, explained by a negative feedback mechanism. I assume that this decrease will occur after a longer period of time.

Serum HDLD (High density lipoprotein cholesterol)

It increased compared to the average reference for the first group by 19.63% and by only 6.52% for the second group.

Since HDL cholesterol (HDL) is a group of lipoproteins synthesized and secreted by hepatocytes, I assume that increasing its level in plasma, especially in patients from the first group, who followed the combined treatment with both products, could be explained by the anti-inflammatory action on hepatocytes. Increasing effect of HDL may be secondary to hepatic lipid metabolism increase.

GLU (serum glucose)

It increased compared to the average reference for the first group by 19.58% and by 10.86% for the second group.

It is known that glucocorticoids increase determine definitely the increased blood sugar for the same period. For this reason, the preparations should not be recommended for diabetic patients, and for others recommend a hypocaloric and hypoglucidic diet.

GGT (serum gamma glutamyl transferase) (Gamma-glutamyl transpeptidase)

It increased compared to the average reference for the first group by 10.18% and by 18.62% for the second group.

As anabolic steroids can increase GGT, it is plausible the same increase effect in serum cortisol during therapeutic stimulation.

Increasing this biochemical parameter in treated patients from both groups could be explained by the occurrence of a degree of liver cholestasis by the action of stimulating the metabolism of liver cells.

Free radicals

They increased compared to the average of reference for the second group by 16.89% and by 9.73% for the first group.

Free radicals in the body come primarily from oxidation processes performed in the body and oxidative stress to which it is exposed. Free radicals are molecules or molecular fragments containing one or more odd electron. By accepting the hypothesis that preparations administered to patients causes "oxidative stress" is expected to increase this indicator of metabolic status.

The free radicals are involved in the onset of some diseases. Among these, may be mentioned: acute inflammation, some cardiovascular diseases (especially in the atherogenic process evolution), liver failure, respiratory failure, etc..

Increased free radical evident from the second group, which has undergone treatment only with Stresclin derma, can be justified by the protective immunomodulatory action of extract from the cordiofolia Rubio roots (612.5 mg / 5ml) contained in the Klinhaem product.

TG-B (serum triglycerides)

They increased compared to the average reference by 11.6% for the first group and by 15.73% for the second group.

It was proved that anabolic steroids may increase TG-B; it is plausible that increased serum cortisol have the same effect during therapeutic stimulation.

Increasing this biochemical parameter in treated patients from both groups could be explained by the occurrence of a degree of liver cholestasis.

Urea (blood urea nitrogen)

It increased compared to the average reference by 14.10% for the second group and decreased below the reference average by -4.49% for the first group.

As anabolic steroids may increase serum urea, it is plausible that increased serum cortisol has the same effect during therapeutic stimulation. The explanation may be the protein catabolism increase, pronounced in the second group.

It looks like the antipsoriatic effect of tuberous root extract Smilax china (250 mg / 5 ml), coupled with immunomodulation action of the roots cordiofolia Rubia extract (612.5 mg / 5 ml) has a decreasing effect of producing blood urea nitrogen.

Aterogen risk

It increased compared to reference average for the first group by 13.85% and by only 3.52% for the second group. This is the consequence of dyslipidemia found by lipid metabolism parameters (increased HDL, LDL.CHOL, TG-B)

Uric acid (serum uric acid)

It increased compared to the average reference by 13.78% for the second group and decreased below the average reference by -3.81% for the first group.

Uric acid is the end product of purine metabolism of nitrogenous bases (adenine and guanine).

Uric acid is a mild oxidant and its ability to capture free radicals is incriminated as a protective factor against continue oxidative aggression facing the most body tissues.

Whereas the administration of corticosteroids may reduce serum uric acid, it goes without saying that higher amounts of corticosteroids highlighted in the first group may explain the decrease in the average values of this blood parameter.

Increased serum uric acid in the second batch, can be explained by the metabolic mechanisms of determined action of Camellia sinensis extract(green tea), which is characterized by combating free radicals and increased catabolism purine nitrogenous bases (adenine and guanine).

LDL.Col (Low-density lipoprotein cholesterol levels)

It increased compared to average reference for the second group by 12.36% and by 7.14% for the first group.

It is shown that anabolic steroids can increase HDL cholesterol, it is also plausible that the increase in serum cortisol has the same effect during therapeutic stimulation.

Increasing this biochemical parameter in treated patients from both groups could be explained by the occurrence of a degree of liver cholestasis.

CHOL (cholesterol levels)

It increased compared to the average reference for the second group by 11.56% and by 3.08% for the first group.

Glucocorticoid administration may increase CHOL; it is plausible that increased serum cortisol has the same effect during therapeutic stimulation.

The increase of this biochemical parameter proportional with the increase of serum cortisol in patients from both groups could be explained by the action of stimulating liver cell metabolism, meaning increasing lipid anabolism.

FE (Serum iron - sideremia)

It Increased compared to reference average by 11.3% for the first group and by just 3.92% for the second group. Increased serum iron can be linked with increased analysis that highlights hepatic dysfunction (TBIL, GGT, AST, dyslipidemia), also the increase in serum cortisol.

AST (serum aspartate aminotransferase)

It increased compared to average reference by 10% for the second group and by 1.08% for the first group.

Intake of anabolic steroids can cause both cholestasis and hepatocellular damage. Both effects may increase serum AST.

The marked difference between the values of the two groups of patients can be explained by increasing average value for this parameter more in group 2, patients who received treatment only Stresclin derma, due to the protective immunomodulatory action extract from the Rubio cordiofolia roots (612.5 mg / 5ml) contained in the Klinhaem product.

The lowest average values, which decreased by at least 10% of the initial average values, pre-treatment, are in the following order :

ALT (serum alanine aminotransferase)

It decreased compared to the average reference by -21.76% for the second group and by -10.56% for the first group.

ALT is more specific than AST for liver disease. Since the absolute values of ALT do not correlate directly with the severity and prognosis of liver damage, meaning that this parameter blood drop can be interpreted as evidence of the absence of organic changes, histo-pathological hepatocytes.

Serotonin (serum serotonin)

It is a biogenic amine derivative of tryptophan (5-hydroxytryptamine). Manifests important pharmacological actions: vasoconstrictor properties (hypertension); stimulates peristalsis; stimulates smooth muscle contraction, **favors the transmission of nervous influx; is assigned a central role in psycho-behavioral states.**

It decreased compared to the average reference by -10.56% for the first group and by -8.10% for the second group. So obvious decrease explanation for this blood parameter may consist in the action of the roots extract of Taraxacum officinale (dandelion) (25 mg / cps.): blood cleanser, detoxifying, calming, sedative.

In conclusion, we can say the following:

- In the treatment of psoriasis vulgaris studied natural therapeutic products can be recommended : Tinefcon, Omega 3 Derma Klinhaem and Stresclin.
- The mechanism of action is mainly to stimulate the release of endogenous cortisol.
- Avoid recommendation of these products to patients who have dysmetabolic associated diseases: diabetes, liver diseases, endocrine disorders with metabolic implications.
- Due to the increasing trend of the Ritis coefficient (AST / ALT), calculated before and after treatment from average values of the two groups of patients (group 1 increased from 0.84 to 1.05, and in group 2 from 0.69 to 0.97), although not exceeded the threshold of 1.33, it can be said that the administered treatment produce some degree of improvement in liver function.
- Hepatoprotective drugs will be recommend during therapeutic periods (silymarin, Hepato protect, etc.)
- Patients will be biochemical and hormonal monitored throughout the therapy, and in the case of alterations, therapeutic doses will be reduced or will be suspended until the entry into normality.

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PARTICIPATION AS MEMBER AND LEADER OF FOLLOWING RESEARCH COLLECTIVES

1. "ENVIRONMENTAL INFLUENCE CONCERNING DIVERS HEALTH", theme code TS – 208 from Sectoral Plan for Research and Development of MapN year **2007**- ended at 23.11.2007 și PSCD al MapN for year **2008**- ended at 21.11.2008.
2. "DEVELOPMENT OF THEORETICAL AND APPLICATIVE STUDIES CONCERNING CALORIC DIVER DIET", theme code TS – 224 from Internal Plan of Research and Development of Divers Center, **2009 – 2010** in period.

PARTICIPATION AS A MEMBER AND RULER OF THE FOLLOWING DRAFTING MANUALS COLLECTIVES, MILITARY TRAINING AND REGULATIONS

1. HYPERBARIC MEDICINE COURSE., developed as a "responsible course" for obtaining the **"Certificate of complementary studies in Hyperbaric Medicine",** under the leadership of the National Center for Health Professional Development, Ministry of Health, Constanta, **(2009)**
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