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Immunological Studies of Ginseng

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Abstract

One of the major effects of *Panax ginseng*, the best known traditional medicine in the Far East, is the enhancement of host resistance against infections, which could depend on an influence from the immune system. The studies presented have been carried out with extracts from Korean ginseng roots which were examined for immunological activity *in vitro* and *in vivo* . The results obtained in a double-blind clinical study with humans confirmed results obtained *in vitro* with human granulocytes and *in vivo* with mice. The ginseng extracts showed a significant stimulatory action on the immune response.

Introduction

One of the programs which have been undertaken by our company for knowing better the properties of Ginseng, is the clinical research. During the last 23 years, with the cooperation of physicians and researchers working in hospitals and clinical universities, our laboratories have published 42 clinical trials on the standardized Ginseng Extract G115. These clinical trials have been carried out on 3535 patients. Thanks to these studies our products GINSANA and GERIAVIT PHARMATON, containing the G115 as active ingredient, could be registered as a medicine in 53 countries all over the world (Tables 1 and 2).

These clinical trials have given statistically significant results which can be summarized in two main actions: on the central nervous system and on the cells involved in the physical work. These two actions are globally interpreted as demonstrating improvements in physical and mental performance.

The immunological studies carried out with Ginseng on animals after 1980 (1-13) and the encouraging results which have been obtained, have brought ourselves to begin clinical studies for checking the immunomodulatory activity of the standardized Ginseng extract G115 in the man. In the controlled double-blind study, which we are presenting, the activities of the standardized Ginseng Extract G115 and the activities of another extract have been compared one to another and both to placebo.

Materials and Methods

Staff: The study has been performed under the direction of Prof. F. Fraschini, director of the Chemotherapy

Table 1 Clinical trials with GINSANA

Author (s)	Year	No. of participants	No. of women	No. of men	Age (year) b=below a=above	Average of age	Dosage	Time of treatment
M.von Ardenne/W.Klemm	1987	63	·	·			2x1	4 weeks
L.D'Angelo, et al.	1986	32	--	32	20-24		2x1	12 weeks
I.Forgo/G.Schimert	1985	28	--	28	20-30	24.4-24.7	2x1	9 weeks
A.C.Gianoli/D.Riebenfeld	1984	83	59	24		37.7	2x1	4 months
I.Forgo	1983	30	--	30	19-31	24.2	2x1	9 weeks
H.A.Quiroga	1982	45	22	23	40-76	58	2x1	90 days
I.Forgo/A.M.Kirchdorfer	1982	30	--	30	18-31	23.5	2	9 weeks
I.Forgo/A.M.Kirchdorfer	1981	20	--	20	18-31	22.8	2x1	9 weeks
I.Forgo, et al.	1981	120	60	60	30-60		2x1	12 weeks
E.Dorling, et al.	1980	60	·	·	22-80		2x1	90 days
I.Forgo	1980	12	--	12	18-35		2	14 days
H.A.Quiroga/A.E.Imbriano	1979	200 (-66)	45 (-15)	155 (-51)	41-70	57	2x1/1x1	1 month/2 months
U.J.Schmidt, et al.	1978	540	·	·			1x1	

Number of participants : 1263
Unusual participants : 91
Effective estimated participants : 1197

Table 2. Clinical trials with GERIAVIT PHARMATON

Author (s)	Year	No. of participants	No. of women	No. of men	Age (year) b=below a=above	Average of age	Dosage	Time of treament
D.J.Garay Lillo, et al.	1987							6 months
M.Zuin, et al.	1987	24	10	14		66.8 (±8.5)	2x1	12 weeks
P.A.Tesch, et al.	1987	38	--	38	50-54		1x1	8 weeks
M.Le Faou	1985	12 (-1)	2	9	31-55	39.72	2x1	6 weeks
G.Mor	1985	126 (-31)	72	54	24-86	58	2x1	min. of 3 weeks
S.Murano/R.R.Lo Russo	1984	65	·	·	18-70		2x1/1x1	30 days/30days
D.J.Garay Lillo, et al.	1984	60 (-2)	·	·	35-65		2x1	8 weeks
M.S.Seif El-Nasr, et al	1982	201	·	·			2x1	3 months at 21 days
R.Hugonot, et al.	1981	98 (-11)	56 (-7)	42 (-4)	a 50		2x1	2 months
J.E.Curutchet Ragusin, et al.	1980	100 (-2)	·	·	60-82		2x1/1x1	3 weeks/1 week
U.J.Schmidt, et al.	1978	540	·	·			1x1	90 days
A.C.Gianoli	1977	170 (-30)	·	·			2x1	21 days
W.C.M.Simon	1977	22 (-6)	·	·	58-65		1x1	90 days
W.J.Revers, et al.	1976	30	24	6	53-90	75.8	1x1	100 days
A.C.Gianoli	1975	59 (-8)	25	26	50-80		2x1/1x1	14 days/66 days
F.Sandberg	1974	30	·	·	22-26		2x1	33 days
W.C.M.Simon	1974	20	--	20	50-60		2x1	6 weeks
G.Warnecke	1974	72	72	--	38-47	43.5	3x1/2x1/1x1	8 days/8 days/134 days
A.Cascone	1973	50	32	18	59-87	76	2x1	30 days
L.Schrüffer	1973	66	36	30	31-60		2x1/1x1	2 months/1 month
R.Viguie	1972	145	62	83	39-84		2x1	15 days-2 months
E.Poggi, et al.	1972	60	25	35	41-85		2x1	30 days
A.Alessandrini	1971	60	29	31	49-93	77	2x1	60 days
G.Warnecke	1970	66	66	--	30-73		1x1	8-12 weeks
G.Schimert	1970	60	·	·	45-75		1x1	5-6 months
R.Colombi	1970	40	8	32	43-85	68	1x1	21 days
P.Luth	1968	70	·	·	b 50			
F.Stengel/H.Listabarth	1968	95	78	17	b 60/a 90		1x1	3-10 months
P:Luth	1965	ca. 50	·	·	a 40		1-2	4 weeks

Number of participants : 2429
Unusual participants : 91
Effective estimated participants : 2338

Department at the University of Milan (Italy). The following researchers, to whom we also express our thanks, have been involved in the execution: Dr. F. Scaglione, Dr. F. Ferrara, Dr. S. Dugnani, Dr. L. Ripamonti and Dr. M. Falchi.

Volunteers' selection: 60 healthy volunteers, who had been informed about the purpose of the study and gave their consent, were admitted to the study.

Criteria of inclusion: Subjects of either sex with an age lower than 50 years.

Criteria of exclusion:

— Subjects not complying with the criteria of inclusion
— Patients having been treated with corticosteroids, immunomodulating agents or radiotherapy in the 12 months preceeding the study
— Subjects who underwent vaccinations other than the anti-influenza one in the 20 days prior to the study
— Patients with neoplasies
— Uremic patients (creatinemia higher than 2.5 mg/100ml)
— Subjects with suspected or ascertained hypersensitivity towards the drug under investigation and/or its excipients

Clinical laboratory methods: Chemotaxis of circulating polymorphonuclear leukocytes (PMN), carried out on agarose according to the method described by Nelson and coll. (14). Phagocytosis and intracellular killing

according to the method described by Lehrer and Cline (15).

Total lymphocytes (T3) and lymphocyte subsets: T helper (T4) and T suppressor (T8), obtained by means of the use of monoclonal antibodies (Ortho) according to the method described by Faure and coll. (16). Blastogenesis of circulating lymphocytes, employing Concanavalin A (Con-A) and Pokeweed (PWM) as mitogens, according to the method described by Martelli and coll. (17), using H3-thymidine labelling and expressing the results as cpm of the stimulated sample, by subtracting the cpm due to the sample without mitogens. NK activity determined according to the method described by Bray and coll. (18).

Experimental procedures: The commercially available GINSANA capsules, Batch No. 601/397 (containing 100 mg standardized Ginseng Extract G115 as active ingredient), the placebo capsules (containing lactose and caramel) and the capsules Batch No. 15239 (containing 100 mg Ginseng Extract PKC 169/79) were supplied by Pharmaton SA, Lugano-Bioggio, Switzerland. The three kinds of capsules were totally undistinguishable in form, shape and colour.

The packages containing the capsules were identified with the letters A, B and C. The key for the indentification was put into an envelope, which was closed and opened only at the end of the research. The volunteers were divided into three groups of 20 subjects each and randomly allotted to treatment A, B or C. The double-blind treatment consisted of the administration of one capsule every 12 hours for 8 weeks. All the parameters mentioned above were determined on leukocytes from venous blood, before and 4 and 8 weeks after the beginning of the treatment.

Statistical evaluation of data: The gathered data underwent statistical analysis, comparing the two treatments by means of the analysis of variance.

Results

In table 3 the results concerning chemotaxis, phagocytes and intracellular killing of circulating PML are reported.

Chemotaxis shows a behaviour comparable in the group treated with the PKC 167/79-extract (group A) and in the group treated with the G115-extract (group C), with a statistically significant increase occurring after 4 weeks of treatment. In the G115-group it increases with a further significant enhancement after the eighth week. In the group treated with placebo (group B) no variation can be observed.

The phagocytosis index (PHI) shows a significant increase at the end of the eighth week of treatment with PKC 167/79, while with the G115-extract it shows a significant rise already after the fourth week of treatment and maintains high levels also at the end of the eighth week.

The phagocytosis fraction (PHF) shows an increase with PKC 167/79, which proves to be statistically significant at the end of the eighth week of treatment, while with the G115-extract it is already significant after the fourth week and keeps at high levels up to the eighth week. In the placebo group, no modification of the two parameters considered can be observed.

The intracellular killing shows a comparable behaviour in the PKC 167/79 and G115 groups with a strong significant increase occurring at the end of the eighth week.

The total T lymphocytes show a significant increase in the fourth week of treatment, which keeps at high levels at the end of the eighth week of treatment in the PKC 167/79 group, as well as in the G115 group (Table 4).

***The T-helper* lymphocytes** show a significant increase at the end of the eighth week in the PKC 167/79 group, while in the G115 group a significant rise can already be seen at the fourth week, which keeps at high levels

Table 3. Activity of circulating polymorphonuclear leukocytes (PMN) in 3 groups of 20 subjects each, before and 4 and 8 weeks after administration of the standadized Ginseng extract G115, the Ginseng extract PKC 167/79 or placebo. Chemotaxis is expressed as the induced migration in millimeters after subtracting the spontaneous migration. The phagocytosis index PHI is expressed as number of phagocyted microorganisms/total PMN. The phagocytosis fraction PHF is expressed as phagocyting PMN/total PMN. The intracellular killing is expressed as per cent of killed microorganisms.

		PKC 167/79 (Mean ± SD)	PLACEBO (Mean ± SD)	G115 (Mean ± SD)
Chemotaxis	Basal	1.92 ± 0.65	2.03 ± 0.66	1.74 ± 0.49
	4 weeks	*2.31 ± 0.52	2.01 ± 0.63	*2.20 ± 0.54
	8 weeks	*2.52 ± 0.53	2.00 ± 0.55	**2.53 ± 0.54
PHI	Basal	0.49 ± 0.26	0.42 ± 0.18	0.38 ± 0.13
	4 weeks	0.57 ± 0.13	0.46 ± 0.19	**0.56 ± 0.13
	8 weeks	*0.69 ± 0.24	0.44 ± 0.18	**0.63 ± 0.13
PHF	Basal	0.27 ± 0.11	0.24 ± 0.09	0.21 ± 0.06
	4 weeks	0.30 ± 0.06	0.26 ± 0.09	**0.34 ± 0.08
	8 weeks	*0.37 ± 0.10	0.28 ± 0.16	**0.41 ± 0.09
Killing	Basal	21.04 ± 10.74	35.73 ± 18.80	26.78 ± 11.57
	4 weeks	*28.54 ± 9.71	44.22 ± 15.05	*39.16 ± 14.05
	8 weeks	**34.54 ± 12.47	*47.49 ± 14.44	**43.84 ± 13.32

*$p<0.05$ **$p<0.001$ vs basal value

Table 4. Lymphocyte subsets and T-helper / T-suppressor ratio in 3 groups of 20 subjects each, before and 4 and 8 weeks after administration of the standardized Ginseng extract G115, the Ginseng extract PKC 167/79 or placebo. Unit of measurement per cent.

		PKC 167/79 (Mean ± SD)	PLACEBO (Mean ± SD)	G115 (Mean ± SD)
Total	Basal	82.22 ± 4.78	83.69 ± 7.16	80.97 ± 4.82
T-Lymphocytes	4 weeks	*87.29 ± 5.17	80.82 ± 11.89	*87.12 ± 8.45
	8 weeks	**88.69 ± 4.57	82.87 ± 10.27	**88.61 ± 8.83
T-helper	Basal	54.67 ± 5.91	52.73 ± 7.87	57.60 ± 4.36
	4 weeks	56.98 ± 4.88	51.63 ± 7.58	*61.66 ± 4.28
	8 weeks	*58.16 ± 4.68	52.76 ± 7.58	**62.50 ± 4.20
T-suppressor	Basal	30.45 ± 6.99	26.09 ± 4.88	30.74 ± 3.35
	4 weeks	28.46 ± 5.22	25.98 ± 4.69	29.81 ± 4.43
	8 weeks	29.97 ± 3.87	26.78 ± 5.12	30.74 ± 3.58
H/S-Ratio	Basal	1.88 ± 0.24	2.02 ± 0.41	1.88 ± 0.23
	4 weeks	2.06 ± 0.40	2.01 ± 0.29	*2.10 ± 0.34
	8 weeks	1.96 ± 0.29	2.02 ± 0.46	*2.05 ± 0.28

*$p<0.05$ **$p<0.001$ vs basal value

Table 5. Lymphocyte stimulation in 3 groups of 20 subjects each, before and 4 and 8 weeks after administration of the standardized Ginseng extract G115, the Ginseng extract PKC 167/79 or placebo. Unit of measurement : counts per minute (cpm) of incorporated 3H thymidine.

		PKC 167/79 (Mean ± SD)	PLACEBO (Mean ± SD)	G115 (Mean ± SD)
Concanavalin A	Basal	31600 ± 12967	34286 ± 14251	32345 ± 16573
	4 weeks	38483 ± 14802	33877 ± 15367	*46827 ± 19101
	8 weeks	*40895 ± 13885	33342 ± 13746	*50504 ± 18648
Pokeweed mitogen	Basal	17553 ± 6628	16070 ± 7240	18900 ± 8942
	4 weeks	19322 ± 9395	16386 ± 6523	*27485 ± 13179
	8 weeks	*22917 ± 6802	16187 ± 6795	*30254 ± 14315

*$p<0.05$ **$p<0.001$ vs basal value

Table 6. Natural killer cells activity in 3 groups of 20 subjects each, before and 4 and 8 weeks after administration of the standardized Ginseng extract G115, the Ginseng extract PCK 167/79 or placebo. Unit of measurment per cent of 51Cr released in tumoral K562 target cells.

		PKC 167/79 (Mean ± SD)	PLACEBO (Mean ± SD)	G115 (Mean ± SD)
NK-activity	Basal	35.73 ± 18.80	35.31 ± 16.01	30.71 ± 14.82
	4 weeks	44.22 ± 15.05	36.81 ± 14.59	*43.95 ± 16.77
	8 weeks	*47.49 ± 14.44	35.68 ± 15.46	**48.22 ± 16.32

*$p<0.05$ **$p<0.001$ vs basal value

at the end of the eighth week. In the placebo group, no modification of the parameter considered can be observed.

The T-suppressor lymphocytes do not show any variation.

The T-helper / T-suppressor ratio does not show significant variations in the PKC 167/79 group and in the placebo group, while in the G115 group a statistically significant increase can be seen in the fourth week of treatment, which keeps at high levels also at the end of the eighth week. In table 5 the results concerning the **lymphocyte stimulation induced by mitogens** are reported. The response to Concanavalin A and to Pokeweed mitogen shows a statistically significant increase after the eighth week of the treatment in the PKC 167/79 group and already from the fourth week in the G115 group.

The natural killer cells activity does not show any significant variations in the placebo group, while in the G115 group a statistically significant increase of the activity can be observed in the fourth week of treatment, which keeps to high levels up to the end of the eighth week (Table 6).

Discussion

Four major immune systems assist our organism in the defense against a constant assault by viral, bacterial, fungal, protozoal agents which have the potential of producing infection and disease. These systems consist of antibody-mediated (B cell) immunity, cell mediated (T cell) immunity, phagocytosis and complement. Each system may act independently or in concert with the others. The clinical features associated with immunodeficiency are related to the degree of deficiency and the particular system which is deficient in function (Table 7). Numerous advances have recently been made in the diagnosis of specific immunodeficiency disorders (Table 8 gives their classification).

Table 7. Clinical features associated with immunodeficiency*

A. Features frequently present and highly suspicious :
1. Chronic infection
2. Recurrent infection (more than expected)
3. Unusual infecting agents
4. Incomplete clearing between episodes of infection or incomplete response to treatment

B. Features frequently present and moderately suspicious :
1. Skin rash (eczema, *Candida*, etc)
2. Diarrhea (chronic)
3. Growth failure
4. Hepatosplenomegaly
5. Recurrent abscesses
6. Recurrent osteomyelitis
7. Evidence of autoimmunity

C. Features associated with specific immunodeficiency disorders :
1. Ataxia
2. Telangiectasia
3. Short-limbed dwarfism
4. Cartilage-hair hypoplasia
5. Idiopathic endocrinopathy
6. Partial albinism
7. Thrombocytopenia
8. Eczema
9. Tetany

*from Amman A and Wara D., Evaluation of infants and children with recurrent infections. Curr. Probl. Pediatr. 5 (1975)

The precise screening tests available for each component of the immune system and the commercialization of sophysticated instruments such as the fluorescence-activated cell sorters (FACS) in combination with production of monoclonal antibodies, have made important contribution to our understanding immunodeficiency

Table 8. Classification of immunodeficiency disorders*

I. Antibody (B cell) immunodeficiency diseases
X-linked hypogammaglobulinemia (congenital hypogammaglobulinemia)
Transient hypogammaglobulinemia of infancy
Common, variable unclassifiable immunodeficiency (acquired hypogammaglobulinemia)
Immunodeficiency with hyper-IgM
Selective IgA deficiency
Selective IgM deficiency
Selective deficiency of IgG subclasses
Secondary B cell immunodeficiency associated with drugs, protein-losing states
B cell immunodeficiency associated with 5'-nucleotidase deficiency
X-linked lymphoproliferative disease

II. Cellular (T cell) immunodeficiency diseases
Congenital thymic aplasia (DiGeorge syndrome)
Chronic mucocutaneous candidiasis (with or without endocrinopathy)
T cell deficiency associated with purine nucleoside phosphorylase deficiency
T cell deficiency associated with absent membrane glycoprotein
T cell deficiency associated with absent class IMHC antigens

III. Combined antibody-mediated (B cell) and cell-mediated (T cell) immunodeficiency diseases
Severe combined immunodeficiency disease (autosomal recessive, X-linked, sporadic)
Cellular immunodeficiency with abnormal immunoglobulin synthesis (Nezelof's syndrome)
Immunodeficiency with ataxia-telangiectasia
Immunodeficiency with eczema and thrombocytopenia (Wiskott-Aldrich syndrome)
Immunodeficiency with thymoma
Immunodeficiency with short-limbed dwarfism
Immunodeficiency with adenosine deaminase deficiency
Episodic lymphopenia with lymphotoxin
GVH disease
Acquired immunodeficiency syndrome (AIDS)

IV. Phagocytic dysfunction
Chronic granulomatous disease
Glucose-6-phosphate dehydrogenase deficiency
Myeloperoxidase deficiency
Chediak-Higashi syndrome
Job's syndrome
Tuftsin deficiency
"Lazy leukocyte syndrom"
Elevated IgE, defective chemotaxis, eczema, and recurrent infections

V. Complement abnormalities and immunodeficiency diseases
C1q, C1r, and C1s deficiency
C2 deficiency
C3 deficiency (type I, type II)
C4 deficiency
C5 dysfunction, C5 deficiency
C6 deficiency
C7 deficiency
C8 deficiency
C9 deficiency

*from Stites D. P., Stobo J. D., Wells J. V., Basic and Clinical Immunology. Ed. Appleton & Lange, Norwalk, Connecticut / Los Altos, California 1987

disorders, autoimmune diseases, tumor immunity, infectious disease immunity and the activity of medicines on the immune system (19).

On circulating phagocytes, the standardized Ginseng Extract G115 shows a more prompt action if compared to the extract PKC 167/79. The phagocytosis increases in fact in a significant way already in the fourth week in the group treated with G115, while in the group treated with PKC 167/79, a significant rise occurs only at the end of the eighth week. These results, obtained on the man, confirmed the ones obtained by Matsuda and coll. on the mice (11). The intracellular killing is of difficult interpretation, as it proves, in the eighth week, to be statistically enhanced also in the group treated with placebo.

The T-helper lymphocytes are significantly enhanced in the fourth week in the group treated with the G115-extract, but only in the eighth week in the group treated with the PKC 167/79-extract. The more effective action of the G115 on the lymphocyte T helper subset, further demonstrated by an enhancement of the helper/suppressor ratio, shows that the G115-extract can induce a higher immune response.

Also concerning the response to mitogens, a different behaviour can be noticed. The G115-extract induces an enhancement of the response to the mitogens employed already after four weeks of treatment, while the PKC 167/79-extract enhances the response of lymphocytes only after eight weeks of treatment. These data are not in agreement with those obtained by Jie Y.H. and coll. (8) *in vitro* with spleen cells of mice, but are in agreement with those obtained by Tong L. and Chao (2), who observed a promoted mitosis in cultured human lymphocytes activated by Concanavalin A.

Concerning the enhancement of the natural killer cell activity against the erythroleukemia cell line K562 as target cells, the treatment obtained by treating humans with the standardized Ginseng Extract G115 confirms the results that Singh V.K. and coll. have reported at the 4th International Ginseng Symposium, using the mice as model (7).

The results obtained in this clinical trial confirm those reached on animals: It is now statistically proved that *Panax Ginseng* extracts, administered orally, stimulate the immune response in the man. Considering that the standardized Ginseng Extract G115 does not provoke any toxicity on the man (20), while other known immunostimulants such as Levamisol, Aristolochiaacid and Nomifenesin cause undersired side-effects, the obtained results are encouraging (21).

Evidently, as in the case of each scientific research, further questions orignated from these results. Therefore we are now programming to carry out studies on ill patients, whose immunitary system is debilitated. We also want to check for how long the immunostimulant effect remains in the organism, after having finished the therapy with the standardized Ginseng Extract G115.

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Y. S. Yun : How did you array the helper and suppressor T-cells?

F. Soldati : We have done it according to the method of Faure et al. published in "Clinical Immunology" and Immunopathology. 22. 139 (1982). This method is based on the indirect immunofluorescence using monoclonal antiboby and fluorescence conjugated with goat antimouse Ig G immunoglobulins. The T-cell subsets can be identified the marker (glycoproteins) specific for the total T-lymphocytes with OKT3 monoclonal antibody. for the T-helper/inducer lymphocytes with OKT4 monoclonal antibodies and for the T-suppressor/cytotoxic cells with OKT8 monoclonal antibodies.

Y.S. Yun : Until 1985 it was known that all helper cells bear OKT4. However. in 1986 it was discovered that helper T-cells induced by MHC clan I have OKT8 and helper T-cells induced by MHC clan II have OKT4. Therefore. it is not possible to determine the surface markers but they can be determined by functional kits.

F. Soldati : The method and antibodies that we have utilized are those which are currently used in hospitals to determine the T-helper subsets. the T-suppressor subsets and total T-lymphocytes. As I told at the begining of my presentation. this study has been performed on healthy volunteers for clinical reason. Now we know that the G115 extract enhances the immunological response without side effects and in the next study we will test functional activity in patients whose immune system is debilitated. We know that *in vitro* tests with pure ginsenosides give different immunological responses. for instances chemotaxis. interleukins. that rest in animals or humans. In my opinion if one wants to check the immunological activity of ginseng or ginseng extract you have to do *in vivo* with man. after oral administration of normal doses of ginseng. One absolutely can not extrapolate *in vitro* results or animal results on humans. Also from ginseng till 1988 one have isolated 103 different compounds and personally I don't believe that the ginsenosides alone are responsible for the beforementioned immunomodulation.

F. Sandberg : The adaptogenic effect of ginseng promulgated by A.A Brekhmann of Russia was first mentioned 1974 at the first Ginseng Symposium in 1974. That adaptogenic effect could partly be explained by the immunomodulating effect of ginseng. At that time the experimental methods was not so developed but now Soldati and Fahim group have given experimental evidence for this theory.

인삼의 면역학적 연구

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인삼은 극동지역에서 잘알려진 민간약이다. 주 효능중 한가지인 감염에 대한 숙주 저항성의 증진은 면역작용에 미치는 영향에 따라 좌우될 수 있다. 우리가 제시한 이 연구는 한국인삼의 뿌리에서 추출한 추출물을 가지고 행하였다. 이 추출물의 면역학적 활성은 시험관내에서. 그리고 생쥐와 사람을 이용한 생체내 실험으로 조사되었다. 사람을 이용한 이중맹검시험에서 얻어진 결과는 사람의 과립구를 이용한 시험관내 실험과 생쥐를 이용한 생체 실험에서 얻어진 결과에 의하여 뒷받침 되었다. 즉 인삼 추출물은 유의성 있는 면역촉진 작용을 나타냈다.