Autoimmune Diseases – New Therapy Approaches

Dr. med. Ursula Jacob

Introduction

Autoimmune diseases are caused by an overreaction of the immune system against the body's own tissues. The immune system erroneously recognizes endogenous tissue as "non-self" and attacks it, causing severe inflammatory reactions, which may lead to damages in various organs.

The immune system is responsible for the recognition and the defense of microorganisms, viruses, foreign substances (like chemicals and metals), here specifically "trained" T-cells are playing an important role.

In autoimmune diseases the T-cells react abnormally by recognizing body-own structures as "non-self", i. e. they attack endogenous tissues, whereas the repair mechanisms of the body try to regenerate damaged organ parts wherever possible.

Without treatment this process will continue throughout the lifecycle or until the complete destruction of the organ.

There are various hypotheses about the cause of this disease:

- genetic disposition in combination with external impacts (like stress, infections, etc.)
- too little exposition to environmental bacteria could encourage the development of an autoimmune disease
- chronic viral infections and unfavorable environmental influences

Through the autoimmune reaction auto-antigens, cell membrane particles (micelles), DNA fragments or endogenous proteins are circulating in the body's blood stream.

L-lymphocytes and other immune cells erroneously recognize them as "non-self" and express inflammation promoting substances (so called cytokines) and disturb the cell communication (like IL-6, IL 8, TNF α etc.).

More and more immune cells are attracted and the false information is carried on. B-cells differentiate to plasma cells and begin to produce auto-antibodies which are released into the blood stream.

So they reach each part of the body, they bind to their specific antigens, these antigens dock with the target structures of the autoimmune reaction and mark those cells as having to be eliminated by phagocytes and CD 8 T-cells, which then leads to the destruction of the respective organ (like in multiple sclerosis).

Also specific pathogens, like bacteria (streptococci) are able to do this (like in rheumatoid fever). This can be seen in serological auto antibody titers (as e. g. ANA, ANCA, anti phospholipid antibody) and also in the amount of circulating immune complexes.

Standard Therapies

There is no possibility for a causal therapy, as the exact triggers cannot be defined.

The disease can only be treated symptomatically (immune suppression, anti-inflammatory) in the form of cortisone or, most recently, with antibodies.

Alternative Therapies

As already mentioned, there is discussion of various triggers which might be able to cause an autoimmune disease, like

- Environmental intoxification through heavy metals, which still are main components of dental fillings, like mecury, arsenic, aluminium, etc.
- Chronic inflammatory problems like bacterial/ viral burdens and allergies.
- Stress related problems which may lead to a metabolic dysfunctions and an immune suppression due to depressed adrenal gland function.

Therapy Options with Natural Substances

For some time now, there have been therapeutic approaches to treat autoimmune diseases with natural substances. Well known is the therapy in Hashimoto's (autoimmune disease of the thyroid gland) with selenium *[Journal of clinical endocrinology and metabolism 87(4):1687–1691, 2002].*

Also incense preparations (boswellia carterii) are given, for example in rheumatoid arthritis. These prevent chronic inflammatory reactions in the body, but show no effect in acute inflammations.

They inhibit in a reversible manner 5-lipoxygenase and thus stop part of the inflammation cascade. They also suppress NF-kb activation, which is responsible for chronic inflammatory reactions. Through this they have a positive effect on the involved pain symptoms. Vitamin E, primrose oils, omega 3/6 as well as other antioxidant combinations can influence detoxification and regeneration ability of the damaged organs.

New Therapy Approaches

As autoimmune diseases affect complete organ systems, disturb the metabolism, hormones and the function of the adrenal glands, a therapy success can unfortunately not be achieved by one substance alone, but only by an appropriate combination of different substances.

In order to determine these substances, we must get an overview of the immune status, the metabolic condition and the hormonal function.

For this we determine individual profiles in specialized laboratories, like

- Inflammatory cytokines panel (interleukins, like 6, 8, 12, 10, etc.) as well as TH17-inflammtion, detailed immune status.
- Also circulating immune complexes, ANA, ANCA and parameters related to the affected organs.
- Amino acids, fatty acids, essential vitamins, antioxidants and minerals.
- Possible triggers like heavy metals, intolerances, viruses, etc..

Depending on the results, an individual micronutrient combination is prepared for the patient to achieve a metabolic balance. This is combined with different detox therapies and targeted antiviral therapies.

To support these micronutrients we use regenerative therapies employing different peptides (according to organ specificity).

As a specific therapy option to influence pathological cytokines (chronic inflammation), autoimmune complexes and the immune system, an extract from fermented soybeans was examined and given, which has a targeted influence on interleukin-6, -8, NK-cells and TNF-alpha, as you can see in the following table.

Disease	Affected Organ Tissue
Alopecia areate	Hair
Autoimmune enteropathy	Small and larage intestine enterocytes
Autoimune hepatitis	Liver
APECED	Pancreas, adrenal cortex, thymus
Bullous pemphigoid	Hemidesmosomes of basal keratinocyte cells of skin, also often mouth mucosa
Chronic gastritits	Stomach
Churg-Strauss syndrome	Vessels
CIDP	Myelin layer of nerves of the peripheral
Colitis ulcerosa	Large intestines or rectum
Darmatomyositis	Cutaneous muscles
Diabetes mellitus type II	Beta cells of the pancreas
Dermatitis herpetiformis Duhring	Skin, small intestines
Epidermolysis bullosa acquisita	Skin, small intestines
Glomerulonephritis	Kidneys
Goodpastuere syndrome	Basal membranes of kidneys and lung
Guillain-Barré syndrome	Myelin layer of nerves of the peripheral nerve system
Hashimoto's thyroiditis	Thyroid gland
Idiopthic thrombozytopenic purpura	Thrombozytes
Lichen sclerosus	Skin, small intestines
Lichen Mucosae	Mucosa
Linear IgA dermatosis	Skin, small intestines
Lupus erythematodes	Inner organs and skin
Microscopic polyangiitis	Skin, kidneys, lung
Adamantiades-Behçet syndrome	Skin, iris
Grave's disease	TSH receptors of the thyroid

Case study

Patient AL.K.H. male *16.4.52

Patient has chronic viral load, prediabetes

Therapy until now: fermented soybean products, Q10, minerals

Results: to date – decrease in ANA, no new infections

Lab parameters	9/12	1/13	4/13
IL-8 pg/ml < 35	20,7	53,1	21
IL-6 pg/ml < 2	3,5	1,6	1
TNF-α pg/ml < 1	0,1	1,6	0,1
NK absolute per ml >100-	61	66	122
CRP sen mg/dl < 0,3	0,7	k.M	0,04

Variation of Anti-oxidation Capacity:
See table 5. GSH were increased remarkably in all the three groups after therapy.
GSH-PX were increased remarkably in all the three groups after therapy.
LPO in all the three groups decreased remarkably therapy.
Mn-SOD increased remarkably in all the three groups after therapy.
Cu-Zn-SOD were increased remarkably in groups A, groups B after therapy
TAA were increased remarkably only in groups C after therapy, increased not remarkably in group A, which may result from the high level before therapy

Table 5. Analysis on change of Anti-oxidation Capacity

Item	Group	Case	Pretreatment	Post-treatment	Changes before and after
GSH (mg/gHb)	A	67	2.430±0.459	3.242±0.760	(+)0.812±0.772△△
	B	67	2.453±0.331	3.129±0.478	(+)0.676±0.483△△
	C	128	2.382±0.575	3.192±0.668	(+)0.810±0.696△△
GSH-PX (μ/gHb)	A	67	678.2±203.5	780.9±115.4	(+)102.7±195.3△△
	B	67	590.7±159.8	814.3±125.4	(+)223.6±192.2△△
	C	113	678.1±170.7	843.3±150.1	(+)166.2±222.5△
LPO (μmol/ml)	A	67	5.477±1.435	4.126±0.805	(-)1.351±1.475△△
	B	67	5.460±1.780	4.110±0.860	(-)1.350±1.940△△
	C	127	5.650±2.173	4.259±0.901	(-)1.381±2.250△△
Mn-SOD (Nu/ml)	A	67	48.07±10.72	61.21±5.997	(+)13.14±2.197△△
	B	67	45.42±10.51	59.25±7.300	(+)13.83±12.80△△
	C	127	49.29±9.920	53.89±11.19	(+)4.50±18.37△△
Cu-Zn-SOD (u/gHb)	A	66	1162±163.0	1344±81.81	(+)182±187.0△△
	B	67	1158±173.9	1293±120.8	(+)135±236.3△△
	C	127	1289±787.8	1355±882.4	(+)66±1173
Total antioxidative activity	A	68	30.47±4.328	31.31±3.408	(+)0.84±4.470
	B	66	28.80±4.720	30.71±3.160	(+)1.91±4.520
	C	127	28.18±4.910	30.68±3.449	(+)2.50±5.402△

Change of T Lymphocyte:
See table 8. CD_3 increased remarkably in all the three groups after therapy,
CD_4 increased remarkably in all the three groups after therapy
CD_8 increased remarkably only in Group A after therapy, not change remarkably in Group C, which may be the high level before therapy.
CD_4/CD_8 increased remarkably only in Group C.

Table 8 Analysis on change of immunity indicators

	Group	Case	Pretreatment	Post-treatment	Changes before and after
CD_3 %	A	67	56.82±10.49	65.61±9.236	(+)8.79±10.87△△
	B	66	59.00±10.40	67.60±8.400	(+)8.60±12.20△△
	C	120	55.22±14.55	63.28±12.11	(+)8.06±16.83△△
CD_4 %	A	67	39.06±7.228	45.46±7.846	(+)6.40±9.306△△
	B	66	39.17±9.000	45.60±6.800	(+)6.43±10.79△△
	C	121	38.50±8.545	43.93±9.548	(+)5.43±10.70△△
CD_8 %	A	67	27.36±8.056	31.43±6.589	(+)4.07±8.093△△
	B	66	28.41±7.030	30.98±6.463	(+)2.57±10.382
	C	120	29.18±7.974	29.19±8.337	(+)0.01±11.67
CD_4/CD_8	A	67	1.550±0.569	1.494±0.351	(-)0.056±0.606
	B	66	1.441±0.413	1.531±0.361	(+)0.091±0.570
	C	119	1.401±0.410	1.526±0.430	(-)0.125±0.603△

Ⅱ Change of Anti-oxidation Capacity:
See table 5. GSH, GSH-PX, Cu-Zn-SOD, Mn-SOD and TAA were all increased after therapy, and the values below normal almost all recovered. LPO in all the three groups decreased remarkably, and the values above normal almost all recovered.

Table 5. Analysis on change of Anti-oxidation Capacity

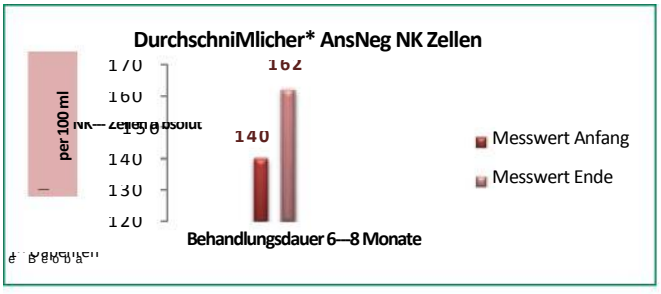
Item	Group	Case	Pretreatment	Post-treatment	Changes before and after
GSH (mg/gHb)	A	63	2.434±0.322	2.803±0.419	(+)0.369±0.456△△
	B	61	2.327±0.396	2.967±0.774	(+)0.640±0.801△△
	C	74	2.540±0.670	3.430±1.790	(+)0.890±1.720△△
GSH-PX (μ/gHb)	A	63	696.9±183.1	799.8±143.1	(+)102.9±312.3△△
	B	61	695.0±165.1	816.6±134.4	(+)121.6±182.3△△
	C	74	759.0±238.9	831.4±148.6	(+) 72.4±257.9△
LPO (μmol/ml)	A	63	5.861±1.760	4.084±0.895	(-)1.777±1.965△△
	B	61	5.082±1.642	4.296±0.979	(-)0.786±1.866△△
	C	74	5.690±1.690	4.300±0.990	(-) 1.390±1.89△△
Mn-SOD (Nu/ml)	A	63	43.07±9.961	60.91±8.713	(+)17.84±13.06△△
	B	61	45.00±10.63	60.44±9.658	(+)15.44±13.61△△
	C	74	43.25±9.740	60.74±9.190	(+)17.49±13.10△△
Cu-Zn-SOD (u/gHb)	A	63	1131±168.0	1293±127.7	(+)162±227.8△△
	B	61	1149±164.0	1269±155.3	(+)120±257.8△△
	C	74	1120±154.3	1268±127.0	(+)148±202.6△△
Total antioxidative activity	A	63	29.06±5.522	30.56±3.481	(+)1.50±5.728△△
	B	61	27.53±4.509	29.91±3.268	(+)2.38±4.424△△
	C	74	27.77±4.700	30.71±3.640	(+)2.94±5.170△△

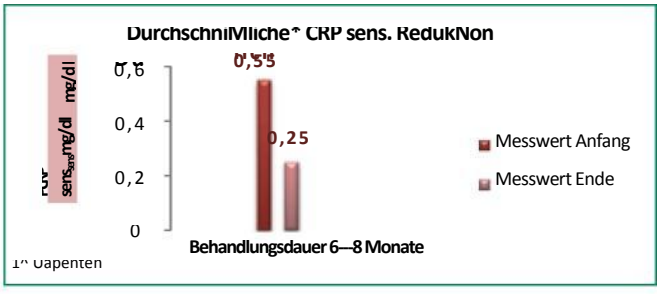
Change of T Lymphocyte:
See table 8. CD_3 in Group A and Group C increased remarkably, while that in Group B did not increased remarkably, which may result from the high level before therapy.
CD_4 increased remarkably only in Group A. There appeared a rising trend of CD_8 in Group A and Group B, while the trend is opposite in Group B. CD_4/CD_8 did not change remarkably in all three groups.

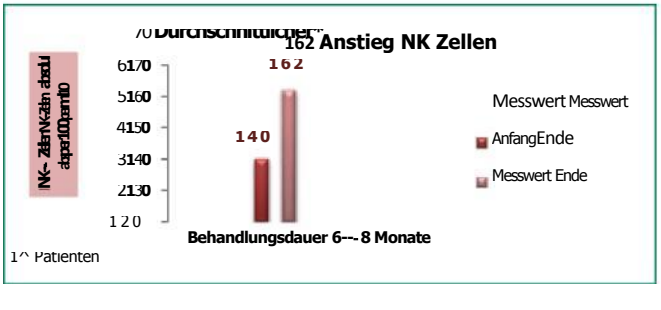
Table 8 Analysis on change of immunity indicators

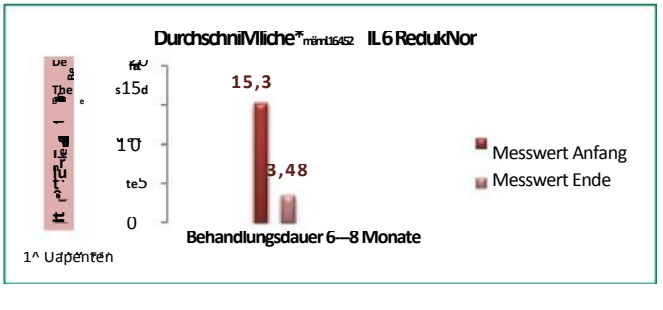
	Group	Case	Pretreatment	Post-treatment	Changes before and after
CD_3 %	A	63	57.44±9.891	62.83±9.698	(+)5.39±13.24△△
	B	60	60.97±9.433	64.12±12.00	(+)3.15±13.56
	C	53	59.70±6.100	63.90±5.700	(+)4.2±13.5
CD_4 %	A	62	38.52±8.931	43.39±7.273	(+)4.87±11.87△△
	B	60	42.63±7.667	43.18±9.355	(+)0.55±11.38
	C	53	40.70±6.800	43.00±8.600	(+)2.3±11.10
CD_8 %	A	62	26.10±7.889	27.65±7.084	(+)1.55±11.91
	B	60	30.53±7.199	28.98±6.445	(-)1.35±8.559
	C	53	28.10±7.600	29.80±6.800	(+)1.7±8.400
CD_4/CD_8	A	62	1.473±0.5378	1.582±0.4267	(+)0.109±0.6678
	B	60	1.451±0.3211	1.559±0.5530	(+)0.108±0.5860
	C	53	1.560±0.5400	1.490±0.3800	(-)0.07±0.6400

Abb. 1 *Nutrition 2013 Feb;28(2):154-9. Doi: 10.1016/j.nut2011.05.008. Epub 2011 Aug 20. Effects of soybean on immune function, brain function, and neurochemistry in healthy volunteers.[Yimit D, Hoxur P, Amat N, Uchikawa K, Yamaquchi N. Research Institute, Xinjiang Medical University, Urumqi, People's Republic of China]*









Conclusion

The fermented soybean extract proved to have a positive influence on various autoimmune diseases by down regulating the chronic inflammation and by stimulating the immune system without any side effects, also over an extended period of time. In addition we could note decreasing autoimmune parameters (like ANA, CIC, ANCA) and thus see a marked improvement of the patient's life quality.

Dr. med. Ursula Jacob
Geibelstrasse 15
81679 Munich | Germany
phone+49 (0)89.46229502
fax +49 (0)89.46223399
mobile +49 (0)160.7277580
info@doc-jacob.com
www.doc-jacob.com