

# The Treatment of Infection Predisposition with Biotherapeutic Medication

by K.H. Ricken, M.D.

Reprinted from Medical Journal for Naturopathy, Organ for the Association of Physicians for Naturopathy No. 9, September 1990, Volume 31, Pages 693-701

## Summary

10 male and 10 female patients with predisposition to infections (recurrent sinobronchitis) were treated with antihomotoxic preparations. After 6 months, the lowered T lymphocytes and T helper cells increased significantly. This increase was consistent with the absence of recidivity. The levels of the immunoglobulins (IgG, IgM, and IgA) were essentially unchanged. The lowered lysozyme levels increased which reflected an increase in phagocytosis. The use of homotoxicological agents represents a valuable addition to the possibilities for treating predisposition to infections.

## Introduction

Every general practitioner, pediatrician, and internist knows the kind of problem the treatment of predisposition to infections represents. 10% of adults with an infection predisposition (sinobronchitis) have had their disorder since their first year of life. Signs of an increased predisposition are as follows:

- Frequency of illness recidivation.
- Severity of illness.
- Duration of illness.
- Inadequate response to an otherwise efficacious treatment.
- Change in localization.

Whereas the methods of traditional medicine basically represent either counteraction therapy (antibiotics, antipyretics, virostatics) or substitution

therapy (immunoglobulin preparations), natural and holistic treatment methods should be regarded as stimulation therapy in the sense of their being regulative measures. With biotherapeutic/ antihomotoxic medication, we are carrying out an immune modulation which gives the body's own defense system the chance to correct disturbed immune balances.

### A. Allopathic Methods

- Suppressive therapy (e.g. antipyretics)
- Antimicrobial (e.g. antibiotics)
- Substitution therapy (e.g. immunoglobulin preparations)
- Chemical immune therapy (virostatics)
- Derivatives of micro-organisms (e.g. bacteria lysate)
- Biological preparations (e.g. transfer factors, interferon)

### B. Natural Healing Methods

- Nonspecific stimulus therapy (e.g. physical therapy)
- Specific therapy (e.g. homeopathy)
- Phytotherapy (e.g. plant extracts)
- Thymus extract

Figure 1: Treatment possibilities for predispositions to infection

We distinguish between a nonspecific and a specific immune system. In addition to the T lymphocytes (= carriers of cellular immunity) and the B lymphocytes (= carriers of humoral immunity), there is a third group, the null lymphocytes, to which the NK cells (= natural killer cells) belong. Furthermore, the first and most primitive defensive reaction is phagocytosis whose reaction capability can be assessed by determining the lysozyme level in the serum.

## Our studies

### Methodology

10 female patients aged between 65 and 74 years and 10 male patients aged between 66 and 75 years with predisposition to infections were examined and then antihomotoxically treated for 6 months. The patients were those who have suffered from infections of the respiratory tract at least 5 to 6 times per year over the course of the last 5 years. A precondition for the treatment was a lowered T lymphocyte level in the serum. A control group composed of 10 female and 10 male patients with no anamnestic or clinical indications of a predisposition to infections was available. The treatment of the infection predisposition of the patients was carried out according the following scheme:

### Analysis of the total lymphocytes (T, B, null)

The lymphocytes in the peripheral blood were differentiated and the percentages of T cells, B cells, and null cells were estimated.

### Principle

The Bio-Rad Quantigen T & B cell assay served to identify and quantitatively assess the lymphocyte subpopulations. In this procedure, antibodies which are bound to microbeads serve as cell-marking reagents. Yellow-brown immunobeads bind on B cells and form rosettes. In the same test tubes, colorless immunobeads bind on T cells and also form rosettes. The remaining unmarked cells represent the

null cell population.

### Reagents

Cell washing concentrate: A bottle with 300 ml of solution contains a 5-times concentrated Dulbeccos phosphate-buffered sodium chloride solution without  $\text{Ca}^{++}$  and  $\text{Mg}^{++}$  ions, 0.5 mM sodium edetate, and 0.05% sodium azide.

Immunobead reagent: A vial contains 5 ml of a suspension of monoclonal anti-T cell antibodies, covalent bound on polymeric, colorless beads, and rabbit anti-human immunoglobulin antibodies, covalent bound on yellow-brown, polymeric beads, in phosphate-buffered sodium chloride solution, 0.1% BSA (bovine serum albumin), and 0.01% sodium azide.

Erythrosine B solution: A vial contains 3 ml of a solution of 0.03% erythrosine B in a phosphate-buffered sodium chloride solution and 0.01% sodium azide.

Lymphocyte isolation medium: 4 ml per sample is required.

### Procedure

5 ml of heparinized blood is processed within 6 hours after the sample was taken. After lymphocyte isolation, wash once with cell washing solution. Incubate for 30 min. at  $37^{\circ}\text{C}$  to remove all of the cytophilous immunoglobulins. Wash twice with cell washing solution. The number of cells is adjusted to  $0.75$  to  $1.25 \times 10^7$  cells/ml.

100  $\mu\text{l}$  of the cell suspension is mixed with 200  $\mu\text{l}$  immunobead reagent. Centrifuge for 3 minutes at  $150 \times g$ . Incubate for 30 min. at  $37^{\circ}\text{C}$ . The precipitate is resuspended. 100  $\mu\text{l}$  of erythrosine B solution is added. Microscopic analysis with  $\times 40$  magnification.

### Calculations

The number of each type of lymphocyte is divided by the total number of lymphocytes counted and then multiplied by 100 to obtain the percentage of each cell type.

Number of T (B, or null) lymphocytes / Total number of lymphocytes  $\times 100$  = % T (B, or null)

Normal value (after adding Bio-Rad):

T cells: 77.36% ( $\pm 2.89$ )

B cells: 11.68% ( $\pm 1.94$ )

Null cells: 10.94% ( $\pm 2.46$ )

### Analysis of the lymphocyte subpopulations (T4/T8)

The T lymphocyte subpopulations are analyzed in the same way (Bio-Rad Quantigen T4/T8 cell assay):

T4 cells = T helper lymphocytes are bound by means of colorless microbeads.

T8 cells = T suppressor lymphocytes are marked with yellow-brown microbeads

Accepted normal values:

T4 cells: 65%, T8 cells 35%

T4/T8 quotient = 2:1

### Analysis of lysozyme in the serum

The lysozyme was determined using the turbidimetric test at 546 nm (Testomar-Lysozym, Beringwerke).

Normal values: 3.0-9.0 mg/l

### Analysis of immunoglobulins

The immunoglobulins M, G, and A were assessed using simple radial immunodiffusion (Nor-Partigen, Behringwerke).

Normal values: IgG: 800-1800 mg%

IgA: 90-450 mg%

IgM: 70-280 mg% (m)

60-250 mg% (f)

### Results

#### Phagocytosis

The influence on phagocytosis was indirectly monitored by means of the lysozyme analysis (Fig. 3).

As a result of the antihomotoxic therapy, the lysozyme mean of 1.62 mg/l before treatment increased to 3.45 mg/l after treatment in the 20 patients investigated (mean for female patients: 1.42 mg/l before and 3.28 mg/l after treatment; mean for male patients: 1.82 mg/l before and 3.61 mg/l after treatment).

#### Humoral immunity

The analysis of the immunoglobulins for the 20 patients resulted in a mean for IgG of 1274.2 mg% before and 1312.0 mg% after the antihomotoxic treatment.

The IgA mean increased from 197.3 mg% before to 255.2 mg% after the treatment. The IgM mean increased from 148.9 mg% before to 188.4 mg% after treatment (Fig. 4).

#### Cellular immunity

Because the normal values which are given in the literature for T, B, and null lymphocytes vary greatly, 20 control persons from our own patient population were analyzed. The values for males and females were first examined separately because earlier studies have shown clear sex differences. An overview of the means calculated for all 20 patients was then presented in Fig. 5.

Whereas the T lymphocyte means of 54.6% before and 62.52% after treatment showed an increase, the means for B lymphocytes of 30.13% before and 22.75% after treatment showed a decrease. The null lymphocyte means were 15.28% before and 14.73% after treatment. The analysis of the T lymphocyte subpopulations yielded an increase from 42.68% before to 52.83% after treatment for the T helper cells (T4 cells), whereas the T suppressor cells (T8 cells) decreased from 56.83% before to 47.17% after the treatment.

### Discussion

The effect of the administration of biotherapeutic/antihomotoxic medication to 20 patients with a predisposition to infections has been assessed. The precondition for inclusion in the clinical study was a lowered initial values of total T lymphocytes. The first analysis of all of the laboratory parameters occurred before the beginning of the 6-month treatment and the second analysis was carried out after the treatment ended.

Phagocytosis represents the most important nonspecific defensive reaction. In this respect, the lysozyme increase should be regarded as an important macrophage resistance factor. Although the values of the immunoglobulins increased, variations were within normal physiological limits. The increase of total T lymphocytes indicates stimulation of cellular defense

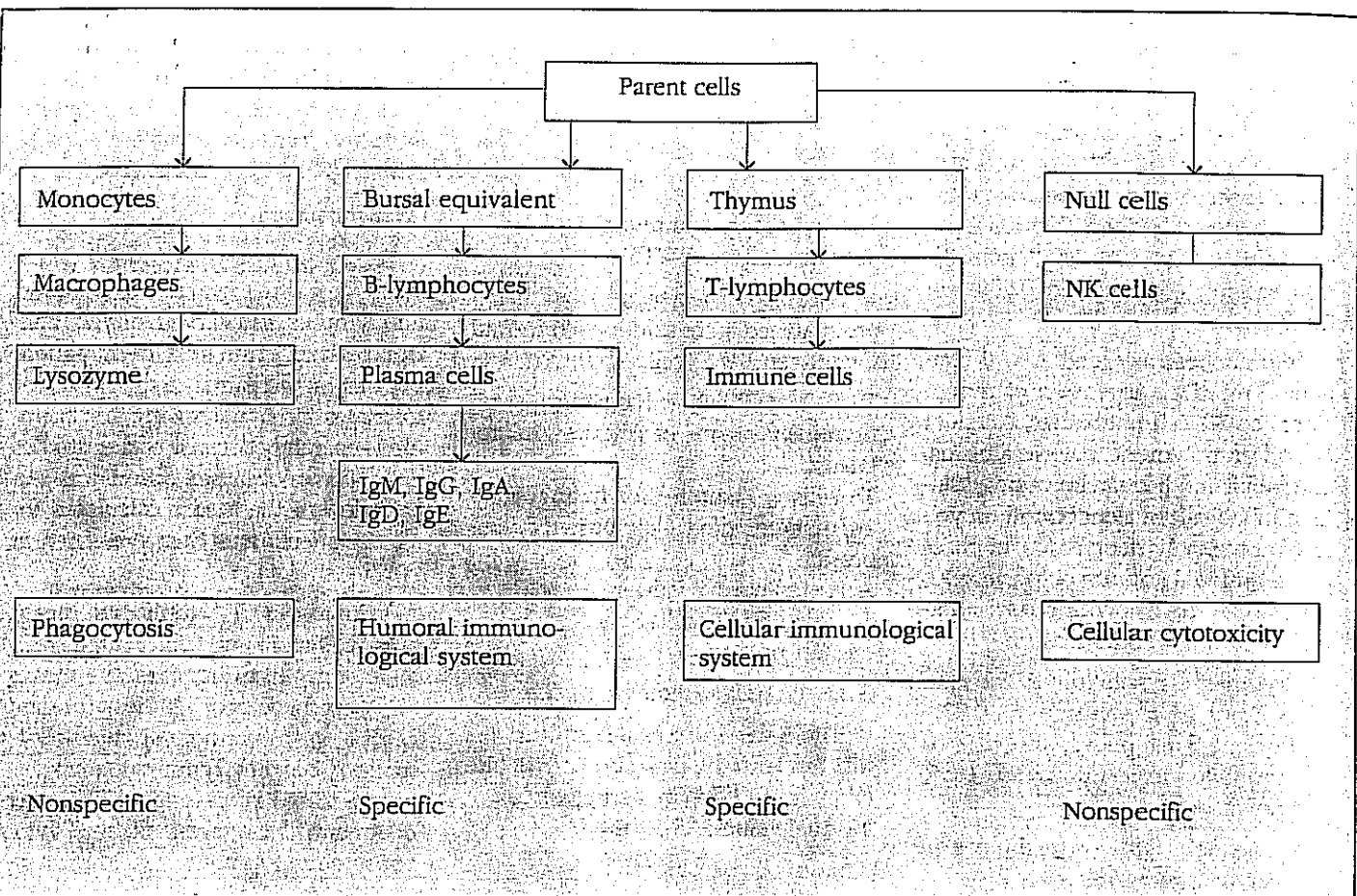


Fig. 2: Model of the immunological system, modified from Ricken.

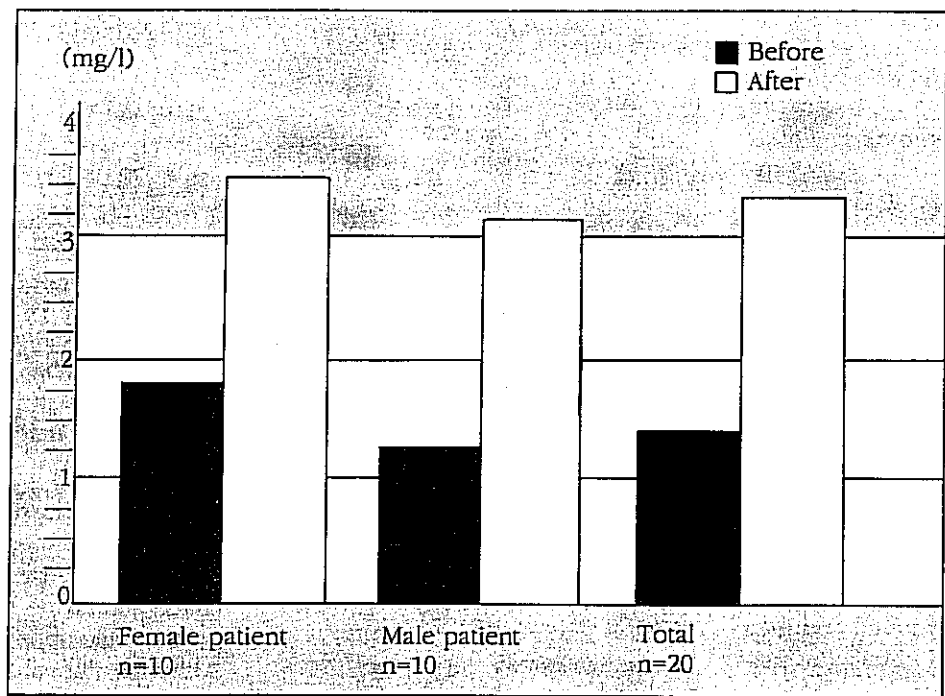


Fig. 3: Lysozyme means (mg/l) for patients with infection predisposition before and after treatment.

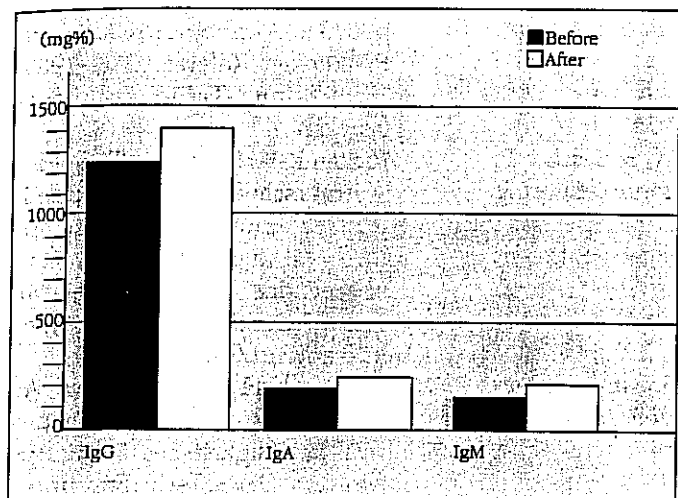


Fig. 4: Immunoglobulin (mg% means) for 20 patients with infection predisposition before and after treatment.

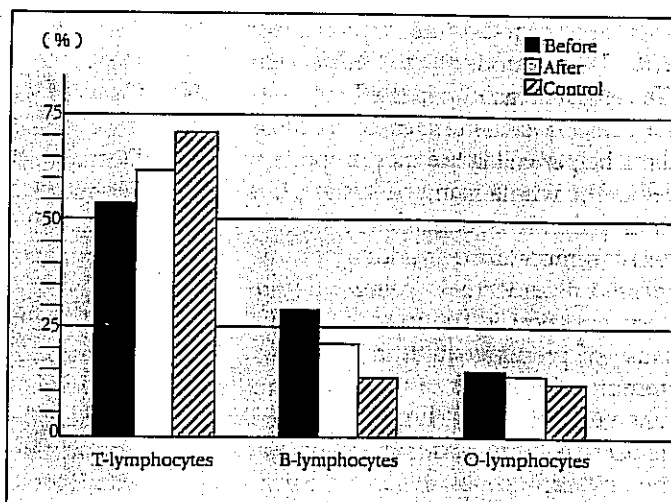


Fig. 5: Lymphocytes (mean %) with 20 patients with infection predisposition before and after treatment as well as for 20 control persons.

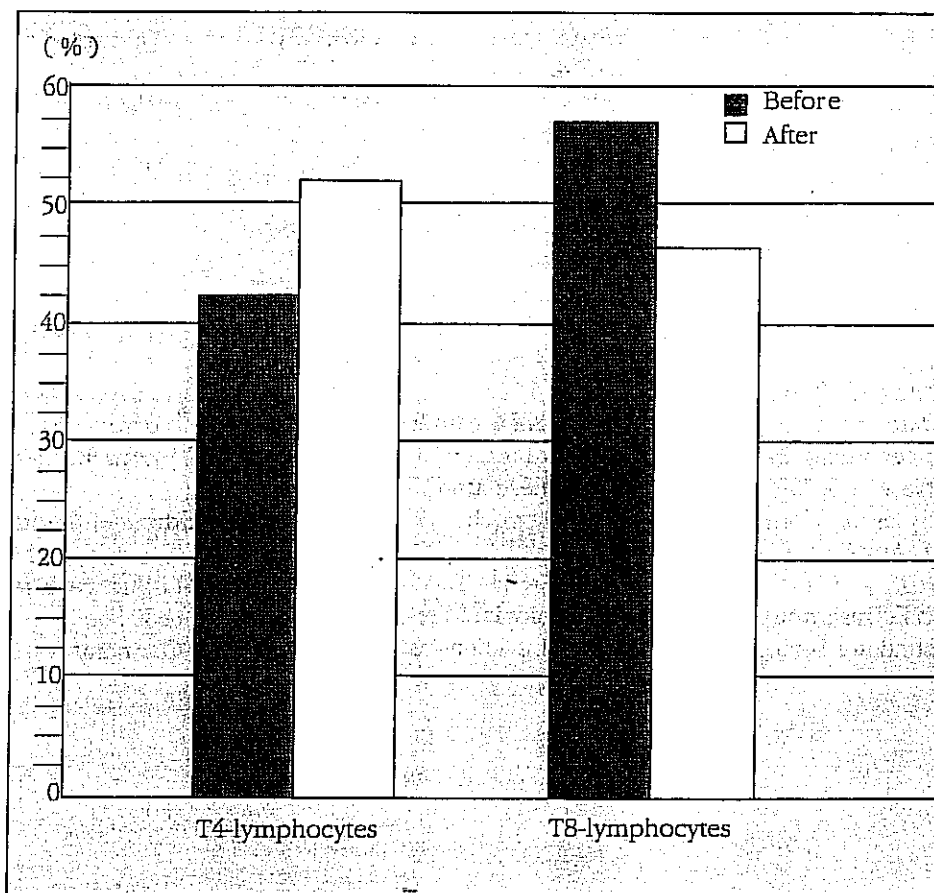


Fig. 6: T4 and T8 lymphocytes (mean %) for 20 patients with infection predisposition before and after treatment.

against infections due to the antihomotoxic therapy. With many patients, normal values were even reached. An examination of the T lymphocyte subpopulations yielded an improvement in the T4/T8 quotient: the T helper cells (T4 cells) increased and the T suppressor cells (T8 cells) decreased. Because the T helper cell is the most important cell in the whole immune system, this finding should be regarded as being of decisive importance. In addition to suppressed phagocytosis, cellular immunosuppression is the most important cause of predisposition to infections. Immunostimulation of the cellular defensive system (T lymphocytes, T helper cells) by means of antihomotoxic therapy represents the most crucial finding of this clinical study. The investigation confirms the efficacy of antihomotoxic therapy using the methods of traditional medicine. It represents a valuable addition to the treatment possibilities for predisposition to infections.

The present investigation is a pilot study carried out in a clinical setting. Hopefully it will lead to further studies under more highly controlled conditions which will provide further evidence of the immunostimulatory effect of antihomotoxic therapy.

## References

1. Albrecht H., Franz G. Naturheilverfahren. Zum Stand der Forschung. Springer-Verlag Berlin, Heidelberg, New York 1990.
2. Bianchi I. Principles of Homotoxicology, Vol. I. Aurelia-Verlag Baden-Baden, 1989.
3. Bieringer K, Zoch E. Das Immunsystem unter besonderer Berücksichtigung des thymusabhängigen T-Zell-Systems. Hufeland-Journal 1988; 4:3-9.

4. Claussen C-F. Homotoxikologie. Aurelia-Verlag Baden-Baden, 1988.
5. Comsa J. Thymushormone. Med. Welt 1980; 31: 533-536.
6. Dostal V, Bayer W, Schleicher P, Schmidt K.H. Immunmonitoring und additive Immuntherapie. Hippokrates-Verlag Stuttgart, 1990.
7. Goldstein G. Die Thymushormone. Triangel 1972; 11: 7-14.
8. Gürtler L. Reaktionsmuster des Körpers bei angeborener und erworbener Immunschwäche. Mta 1989; 4:1143-1148.
9. John J. Erkältungskrankheiten, grippale Infekte, Grippe (Influenza). Welche Therapie? Biol. Med. 1978; 5: 228-232.
10. Klimbel K-H. Verordnungshäufigkeit von Immunstimulanzien. Münch. med. Wschr. 1989; 131: 512-514.
11. Kollmer E.P. Regulierung der Abwehr mit Nosoden. Naturmedizin heute 1987; 1: 475-477.
12. Manger B. Immundefekterkrankungen. Therapiewoche 1989; 39: 2437-2446.
13. Meyer-Langdorff H. Der immuntherapeutische Stoß. Biol. Med. 1985; 2: 408-410; 3: 470-477. 1986; 2: 69-77; 4:169-177.
14. Ricken K-H. Stufenschema der Immunologie in Phylogenese, Ontogenese und Pathogenese. Therapiewoche 1989; 29: 2071-2075.
15. Ricken K-H. Taschenatlas der Immunologie, Allergie und allgemeinen Infektionslehre. Verlag für Medizin Dr. E. Fischer, Heidelberg 1981.
16. Ricken K-H. Die Behandlung der Infektanfälligkeit mit Biotherapeutika/Antihomotoxika nach Reckeweg. Referat 03-16-1989, Heel-Round-Table in Saarbrücken.
17. Ricken K-H. Die Behandlung der Abwehrschwäche im Alter durch gezielte Immunstimulation mit Biotherapeutika/Antihomotoxika. Referat 03-10-1990, Wissenschaftl. Heel-Symposium in Frankfurt/Main.
18. Ricken K-H., Kindermann W. Behandlungsmöglichkeiten der Infektanfälligkeit des Leistungssportlers. Dtsch. Zschr. Sportmed. 1986; 37: 146-150.
19. Schleicher P, Schmidt K. Grundzüge der Immundiagnostik und therapie. Hippokrates-Verlag, Stuttgart 1989.
20. Schmidt F. Antihomotoxische Therapie-Standortbestimmung. Biol. Med. 1990; 1: 75-77.
21. Wagner H. Neue Untersuchungen über die immunstimulierende Wirkung einiger pflanzlicher Homöopathika. Biol. Med. 1985; 2: 399-407.
22. Wagner H, Jurcic K, Doenicke A, Rosenhuber E, Behrens N. Die Beeinflussung der Phagozytosefähigkeit von Granulozyten durch homöopathische Arzneipräparate. Arzneim.-Forsch. Drug Res. 1986; 36: 1421-1425.

Address of the author:  
K.H. Ricken, MD  
Lothringer Strasse 1  
6630 Saarlouis  
Germany

