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CLINICAL

SUMMARY

Atopic Dermatitis is a chronic inflammatory skin reaction characterised by pruritus and typical skin eruptions. The clinical syndrome is present in patients with familial and immunological traits of atopy characterised by the hyperproduction of IgE in direct response to environmental inhalants and food allergens.

In our experience, numerous patients diagnosed with Atopic Dermatitis present with normal IgE levels but positive skin prick test reactions to food allergens. Our hypothesis is that asymptomatic sensitization occurs via the intervention of the intestinal immune system (Gut-associated Lymphoid Tissue - GALT) and that the skin is simply the target organ. The patients who took part in our study obtained rapid improvements in symptomology when prescribed a food intolerance exclusion diet based on the EAV test. They frequently test positive for some food additives and toxins, suggesting a possible aptenic role.

We would advise the following therapy for Atopic Dermatitis: correction of intestinal dysbiosis, and homotoxicological drainage in synergy with a food intolerance exclusion and biological diet.

KEY WORDS

ATOPIC DERMATITIS, INTESTINAL IMMUNE SYSTEM (GUT-ASSOCIATED LYMPHOID TISSUE (GALT)), FOOD INTOLERANCE TEST, BIOLOGICAL DIET

THE ROLE OF FOOD INTOLERANCE IN THE PATHOGENESIS OF ATOPIC DERMATITIS

MINUTES OF THE XVI NATIONAL CONFERENCE ON HOMEOPATHY, HOMOTOXICOLOGY AND BIOLOGICAL MEDICINE

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Atopic Dermatitis (AD) (Neuro dermatitis, Atopic eczema) is a common chronic pathology of the skin, which manifests itself in patients with the usual immune signs of atopy (FIGURE 1). The main symptom of AD is the inflammatory reaction of the skin characterised by Pruritus and typical skin eruptions such as desquamative erythemic and oedematous papules or patches. The distribution of the lesions is age dependent: in infants, they usually affect the frontal region, cheeks and the extensor surfaces of the limbs. The lesions subsequently spread across the flexor regions, particularly the popliteal muscle areas, the neck and the face.

AD is generally regarded as a cutaneous form of atopy, as a result of the usual association with Allergic Rhinitis and

Asthma (often both present) and the high serum concentrations of IgE (FIGURE 2).

The severity of the dermatitis, however, does not always correlate with exposure to allergens to which the patient has a positive reaction in skin tests. Desensitization is often ineffective.

The recurring question in clinical practice is how AD, which is regarded as the prototype of spontaneous Hypersensitivity, can easily develop into chronicity. It is likely that many accompanying factors may interact with the basic immune processes (1) (FIGURE 3).

The disease almost always begins in infancy and, in some cases, clears up before the age of 2. Persistence into se-

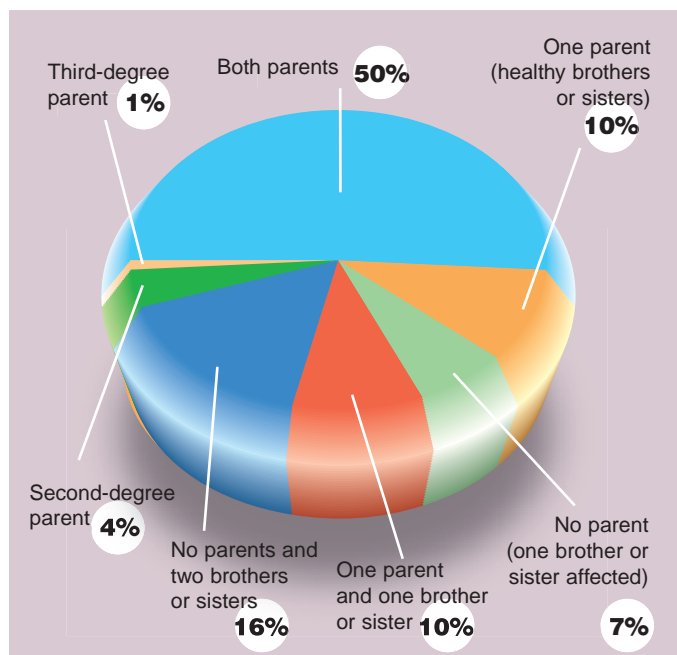


Figure 1

Hereditary Predisposition of Atopic Dermatitis.
 From W. Watson et Al. - The Genetics of Psoriasis. Arch Dermatol 1972; 102: 197-207.

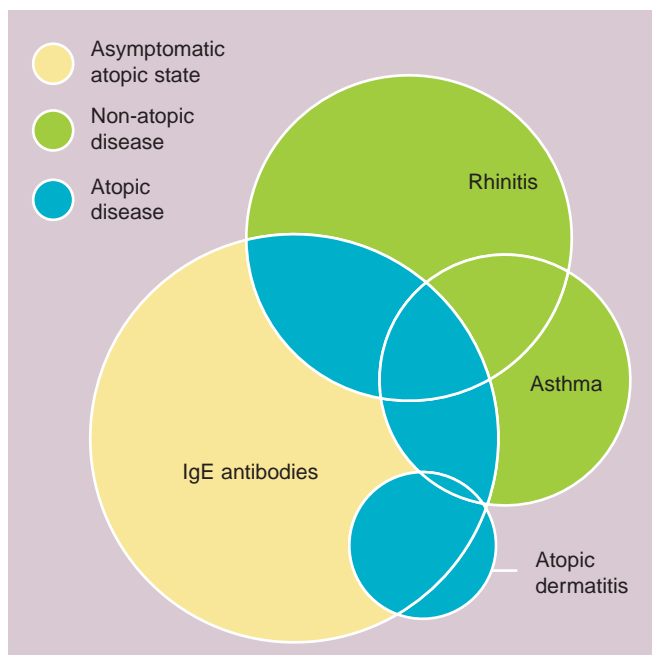


Figure 2

Interrelation between Atopy, Atopic diseases and IgE antibodies.

cond infancy and adulthood is characterised by frequent cycles of remission and relapse. Pruritus is the cardinal symptom - often worse at night and triggered by aspecific stimuli such as temperature changes, sweating, physical strain and emotional stress.

The ingestion of allergenic foods can cause the disease to worsen (2). At macroscopic level, acute lesions are characterised by oedema and dermal infiltrates with the presence of mononucleus cells, degranulated mast cells and Langerhans cells.

In Homotoxicology, Atopic dermatitis or Eczema is classed under the Deposit Phase, with an overload of the Matrix (3).

CHANGES IN HUMORAL IMMUNITY

AD manifests itself by means of the heightened capacity of the B Lymphocytes to produce IgE antibodies against

allergens which trigger off the immune response after contact, inhalation or ingestion (4), (5).

However, the cutaneous lesions are not, in general, reactivated during the pollen season. Food allergy is strongly implicated in Atopic Dermatitis in children. Milk, eggs, wheat, maize, crustacea, hazelnuts, almonds and peanuts are the most common allergens, but all these foods can potentially play an allergenic role (6), (7).

One of the most common problems in routine procedure is the limited use of RAST (Radioallergosorbent Test) in the diagnosis of Atopic Dermatitis. Our studies have often proved RAST-negative in patients affected by true Atopic Dermatitis. Several authors established two types of AD, on the basis of a RAST-positive or -negative result (8).

The way in which a food is cooked also influences its level of allergenicity. In general, allergens of animal origin continue their activity for longer, whereas

vegetable allergens are more easily broken down by cooking or by other processes (9), (10).

There have been recent examples of the role of food additives and preservatives as allergens. Some vegetable gums, carmine red, Ethylvanillin, Vanilla and Tartrazine (11), (12) can directly trigger an IgE mediator response.

IgG STS (Short Term Sensitizing) antibodies can also cause allergic reactions; they activate the degranulation of Mastocytes and Basophils, leading to the deposit of immunocomplexes with complement mediator phlogosis (13).

CHANGES IN CELLULAR IMMUNITY (FIGURE 4)

According to recent studies, the hyperproduction of allergen-specific IgE could be due to defective regulation of the T-Lymphocyte line, particularly when there is a predominant population of T-helper lymphocytes with a TH2-type

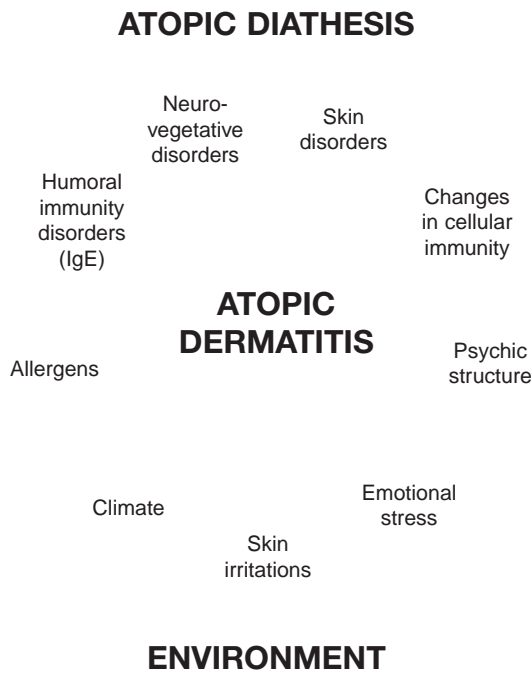


Figure 3

Multifactorial Pathogenesis of Atopic Dermatitis.

cytokine profile. An anomalous population of T-helper CD4+ lymphocytes may explain the inadequate function of the TCD8 lymphocytes in suppressing IgE (14).

Many diagnosis fields such as AD, display delayed or cell mediator Hypersensitivity,

rather than an acute IgE reaction.

The reaction may occur in the Langerhans cells, which, in patients affected by Atopic Dermatitis, have higher levels of IgE receptors – floating in the lymph nodes, they behave like cells producing antigen and cause Th2 allergen-specific reactions.

With Atopic Dermatitis, the typical IgE atopic response is converted into a cellular reaction (15).

The increase in the production of IL-4 and the shortage of Interferon-g also play a major role. Higher percentages of T Clones have been observed in the blood samples of subjects with AD (16). The Phenotype Th2 has a high capacity for producing IL-4 and IL-5 and very little or no capacity for producing IFN g (17). It has not yet been established why, in atopic subjects, the allergens “expand” the CD4 + TH2 clones.

Our hypothesis, which is one possible interpretation of this phenomenon, is that the intestine represents the apparatus for T clone selection via numerous mechanisms. For example, various clinical panels, in conjunction with our studies over the last few years, illustrate the association between AD and Intestinal Dysbiosis. We often obtained a positive EAV test result for Helminths.

Helminths selectively activate TH2 s, releasing many proteolytic enzymes.

As a result of the abnormal putrefactive fermentation in Intestinal Dysbiosis, polyphenolic compounds, capable of activating the TH2 line, are released. However, the mechanism which we consider to be more quantitatively significant in determining AD, is the “fracture” of oral tolerance.

FOOD INTOLERANCES

Oral tolerance is a specialised function of the immune system of the intestinal mucosa (GALT), which is usually unresponsive to protein antigens introduced per os. The phenomenon is mediated by the T cells. The factors which influence its development are the dosage and frequency of exposure to the antigen (usually protein), the genetic equipment of the host (Biotype and diathetic field), previous immunisations and, finally, the overall level of immunological activation of the mucous membranes (Immu-

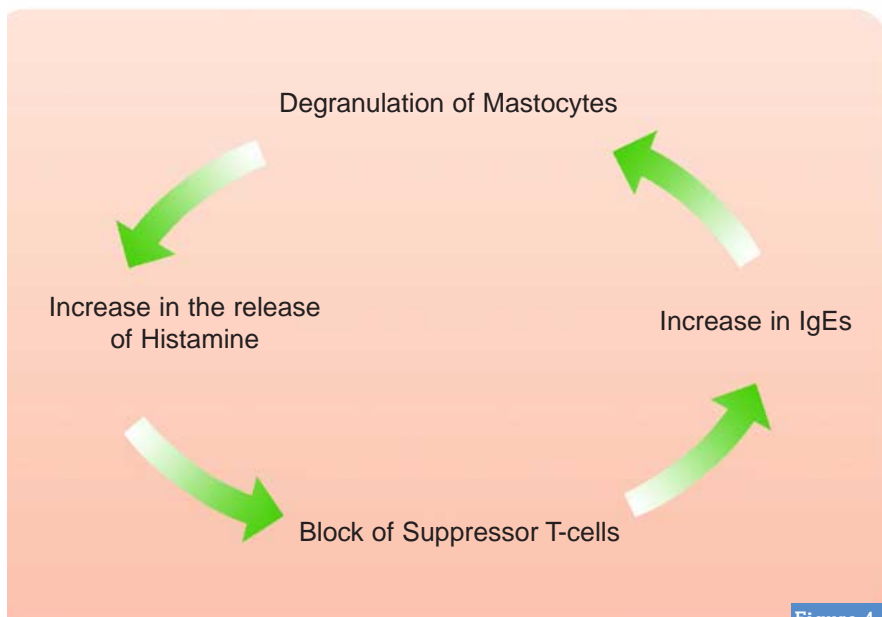


Figure 4

FACTORS WHICH INFLUENCE ORAL TOLERANCE

- QUANTITY OF FOOD ANTIGEN
- FREQUENCY OF EXPOSURE TO FOOD ANTIGEN
- GENETIC EQUIPMENT (COMPOSITION, DIATHESIC FIELD)
- PREVIOUS IMMUNIZATIONS (VACCINES)
- OVERALL IMMUNOLOGICAL ACTIVATION OF THE MUCOUS MEMBRANE (CHRONIC IMMUNOPHLOGOSIS OF MALT SYSTEM)

Figure 5

nophlogosis of the MALT (Mucosa-associated Lymphoid Tissue system) (FIGURE 5).

Various mechanisms operate in the development of oral tolerance (FIGURE 6) (18).

β The suppressor T cells act as an aspecific antigen producing TGF_β and probably other aspecific suppressive factors. As a result of this antigen aspecificity, the oral tolerance produced by the ingestion of one antigen may cause the suppression of the reaction against a second antigen administered by another means (oral, parenteral, aerial, cutaneous), when administered simultaneously (19).

In theory, this suppression could explain how the possible "fracture" of oral tolerance towards a food antigen, may cause immune-pathological reactions against "cross reacting" antigens which have entered by other means, such as via the skin or mucous membrane.

A second oral tolerance mechanism involves the induction of anergy and the removal of the T cell in the laboratory animal (mouse): it has been proved that the frequent ingestion of large quantities of the same food antigen can lead to a breakdown in the capacity for a "suppressor" reaction. (20).

One may, therefore, hypothesise that diets which trigger weak TH1 reactions (such as the ingestion of low doses of

Antigen) may promote the development of T cells producing TGF_β and, therefore, oral tolerance. Diets that provoke intense reactions (such as those containing high doses of antigens administered very frequently) do not stimulate the formation of cells producing TGF_β and, therefore, lead to food intolerances.

The studies carried out in our General Outpatients' Clinic of Biological Medicine, corroborate the oral tolerance mechanism. In fact, the identification of these food intolerances, as they are known, i.e. foods containing Antigens which have been able to overcome oral tolerance, and the ensuing exclusion diet, work simultaneously on the dose and frequency of contact with food allergens.

Diets formulated on the basis of food intolerances work by "resetting" the oral tolerance mechanism – i.e. they restore the function of the suppressor system.

The EAV test allows intolerated foods to be identified quickly (21). The elimination of these foods from the patient suffering from AD leads to an immediate improvement in skin symptoms. The subsequent reintroduction of the food with a rotation diet every 4 days, allows good clinical control of AD (22).

BREAST FEEDING

An important aspect of mucosal immunity is the capacity of the BlgA cells to penetrate the galactophorous ducts and secrete IgA, and then be transported into the mammary secretions (colostrum, milk), where the concentration is extremely high (average: 50mg/ml) (23).

In addition to the anti-infective effects, the secretory IgA develops a protective role, regulating the absorption of food proteins in the first days of life, a time when the body is prone to developing long-lasting IgE mediator allergic reactions. Early exposure to certain food antigens leads to the development of allergies and not being breastfed, causing a deficiency in protective IgAs, has been linked with Atopy. (FIGURE 7).

* "Blue Fish" is the term in Italian used to describe fish such as Sardines, Pilchards, Anchovies, mackerel etc.

Recent studies, according to our anamnestic and clinical data, showed that breastfeeding for 6 months significantly prevents the onset of atopy (24).

FOOD TOXINS

As previously mentioned, some additives can directly cause IgE mediator reactions. However, other substances added to the foods cause allergic skin reactions via various mechanisms. Some additives are composed of polypeptides or large proteins and, therefore, are intrinsically immunogenic. Additives of this type include vegetable gums, carob flour (E410), guar seed flour (E412), etc. Other additives with low molecular weight - such as Sodium Metabisulphite (E223), Sodium Benzoate (E210-219), Potassium Sorbate (E202) etc. - appear to interact with both the tissutal proteins and the food proteins, causing a humoral immune reaction.

The Antibodies most frequently encountered are IgG STS and IgA. Via the deposit of immune complexes, they

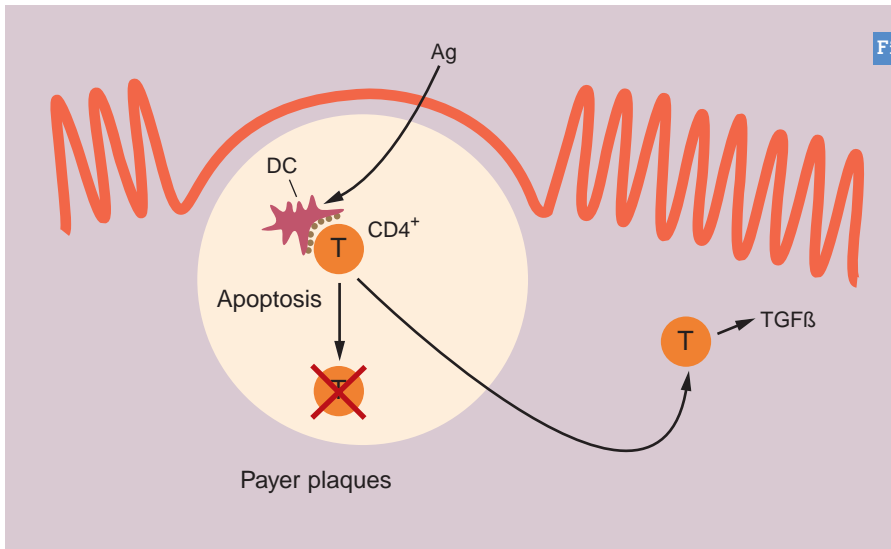


Figure 6

Induction of oral tolerance.
The protein antigens which penetrate the Payer plaques are captured by the dendritic cells (DC), which then cause apoptosis (Removal mechanism) amongst the antigen-specific T cells or differentiation in the suppressor T cells (suppressor mechanism) which produce Transforming Growth Factor-β. (TGF-β).

produce complement mediator phlogosis with the involvement of the integumental apparatus. Our study indicates that patients suffering from AD with a RAST-negative result recognise this mechanism on the basis of the chronic recurrence of skin symptoms (25).

For drug allergies, the same phenomenon is the cause. The now constant presence of antibiotics and hormones in foods of animal origin for general consumption, is another aggravating factor in patients with AD.

A recent Swedish study (26) (27), shows that in a group of children with parents of anthroposophic cultural extraction, those fed from a very young age with breast milk and vegetables with added, strictly natural, milk enzymes and cereals, did not suffer with atopy or AD.

parent is atopic, then such intervention is justified. We recommend the use of miasmatic remedies and nosodes (Sulphur, Thuya, Tuberculinum-Injeel, Psorinum-Injeel, Luesinum-Injeel). During pregnancy, it is advisable to combine mesenchymal drainage remedies as well (Galium - Heel®, Lymphomyosot®, Pulsatilla comp.).

2 Breastfeeding for at least the first six months of life. During breastfeeding, atopic mothers' diets should consist of organic foods, frequently varied according to rotation diet principles, on

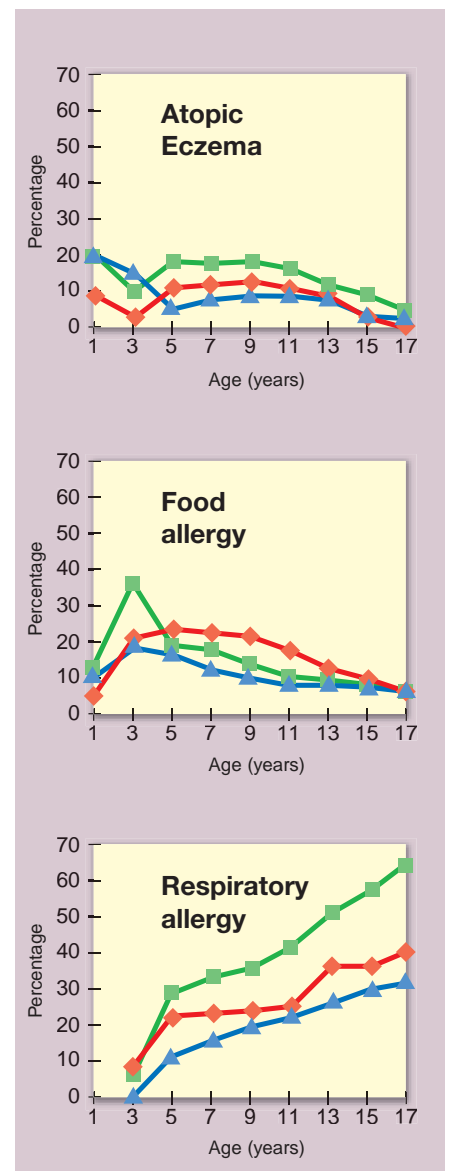


Figure 7

Prevalence of allergic symptoms (Atopic eczema, food allergy, respiratory allergy) according to the duration of breastfeeding: from nought to one month (in green), one-six months (in blue), and six months + (in red).

PREVENTION OF ATOPIC DERMATITIS

We recommend the following interventions:

1 Eugenic homotoxicological treatment in people at risk of atopy before conception. If both parents are atopic, eugenic intervention is essential. Our study indicated that even if only one

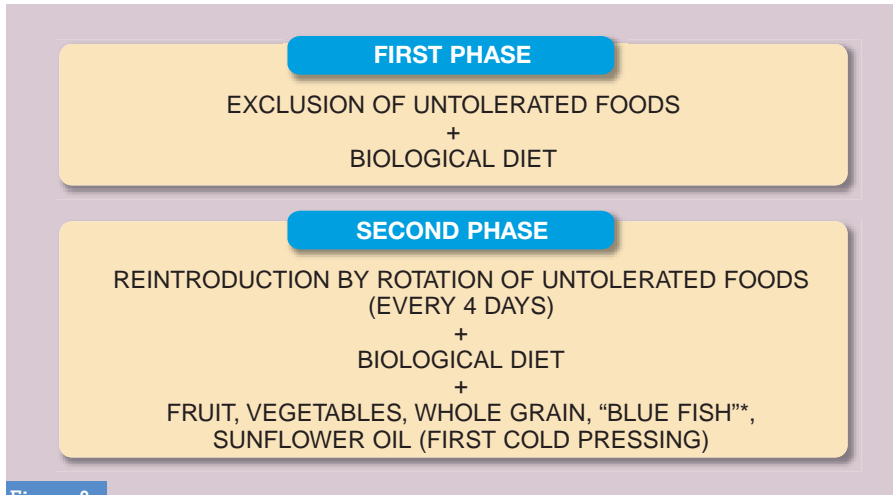


Figure 8

**Atopic Dermatitis
 DIETETIC TREATMENT.**

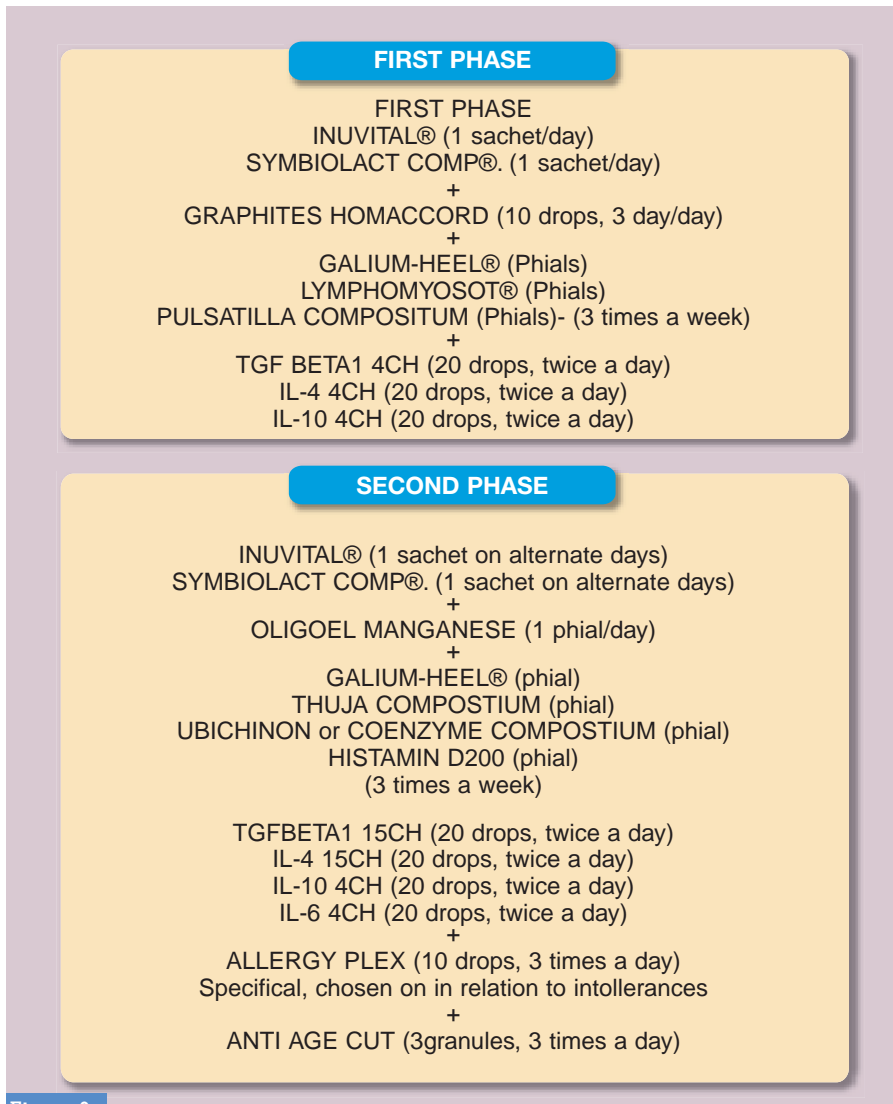


Figure 9

**Atopic Dermatitis
 BIOLOGICAL TREATMENT.**

the basis of their individual food intolerances.

TREATMENT OF ATOPIC DERMATITIS

DIET
 (FIGURE 8)

We regard the desensitisation of the intestinal immune system as being the first level of therapeutic intervention.

A diet that eliminates those food intolerances tested with EAV should be prescribed and this should be continued for at least two months. At the next test, subject to a negative test result, we reintroduce the food with a rotation diet of every four days. It should be recommended to patients suffering from AD that they use foods of strictly organic origin, particularly for fruit, vegetables and whole grains. We also recommend the frequent use of "blue fish"* and sunflower oil (first cold pressing), for their high content of -3 and -6 polyunsaturated fatty acids, particularly effective in skin inflammatory pathologies (28).

BIOLOGICAL THERAPY
 (FIGURE 9)

We correct frequent dysbiosis with Inuvital® and Symbiolact comp.®. For children, we use the Symbioflor® "programme".

For "resetting" the immune system, in accordance with the physiopathological considerations previously illustrated, we recommend the use of Cytokines. In particular the association of:

- TGF B1, 4CH
- IL4, 4CH
- IL6, 4CH
- IL10, 4CH

Through its diathetic immunomodulation action, Oligoel Manganese is particularly effective in atopic patients.

We also intervene with homotoxicological treatment for regulating the skin

reaction phase and mesenchymal drainage.

It is important to sustain the cutaneous trophism with Anti Age Cut.

At the second consultation, we also prescribe the Allergy Plex series according to the food(s) that still give a positive result in the EAV test.

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