

# **CHILDHOOD ATOPY**

**Essay submitted for fulfillment of  
master degree in Pediatrics**

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## **ABSTRACT**

A child with atopy produces IgE antibodies after exposure to common environmental allergens. Both genetic and environmental factors determine the development of atopic disease. The presence of specific IgE antibodies to environmental allergens is determined with skin prick or radioallergosorbent testing in children with atopy. Preventing atopic disease in high-risk infants and hindering progression of disease in children with established disease are the areas of active research. Management of atopic disease is frequently symptomatic, but it is important to avoid identified allergen triggers. Immunotherapy may be considered in selected school-age children with severe rhinoconjunctivitis.

**Key words :** Childhood, Atopy.

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## ABBREVIATIONS

<b><math>\alpha_1</math>A</b>	: $\alpha_1$ antitrypsin
<b>AD</b>	: Atopic Dermatitis.
<b>CAPS</b>	: Children's Asthma Prevention Study (Australia).
<b>CFC</b>	: Chlorofluorocarbon
<b>CMPA</b>	: Cow's Milk Protein Allergy
<b>Der P/f</b>	: Dermatophagoids Pteronyssinus / farinae
<b>DPI</b>	: Dry-powder inhaler.
<b>DSCG</b>	: Disodium cromoglycate
<b>EHWFs</b>	: Extensively hydrolyzed whey formulas
<b>ETS</b>	: Environmental tobacco smoke
<b>FDA</b>	: Food and Drug Administration
<b>FEV<sub>1</sub></b>	: Forced expiratory volume in one second
<b>HDM</b>	: House dust mite
<b>HFA</b>	: Hydro fluoro alkane
<b>IgE</b>	: Immunoglobulin E
<b>ISAAC</b>	: The International Study of Asthma and Allergy in childhood.
<b>LGG</b>	: Lactobacillus GG
<b>MAAS</b>	: Manchester Allergy and Asthma Study.
<b>MDI</b>	: Metered dose inhaler.
<b>PEF</b>	: Peak expiratory flow
<b>PIAMA</b>	: The Prevention of the Incidence of Asthma and Mite Allergy (Netherlands).
<b>PUFA</b>	: Polyunsaturated fatty acid

<b>RAST</b>	: Radioallergosorbent testing
<b>RCT</b>	: Randomized controlled trial
<b>SCORAD</b>	: Scoring Atopic Dermatitis
<b>SLIT</b>	: Sublingual Immunotherapy.
<b>SPACE</b>	: The study of prevnetion of Allergy in children in Europe.
<b>SPT</b>	: Skin Prick tests.
<b>TNF-<math>\alpha</math></b>	: Tumour necrosis factor- $\alpha$ .

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## INTRODUCTION

Pediatric allergic diseases comprise a large component of general pediatric practice. The most common allergic diseases seen are those comprising the allergic march: atopic eczema, allergic rhinitis, and asthma. Management of these disorders has improved over the past several years. More effective medications and treatments are now available together with the development of guidelines to assist management. It has also become clear in recent years that environmental exposures during the first years of life are critical for the pathogenesis of atopic diseases in genetically predisposed children.

Food allergies also comprise a large component of pediatric practice and are increasing in prevalence. The diagnosis of food allergies places limitations on children and families that can lead to nutritional insufficiency and diminished quality of life. As a result, great care must be taken to accurately diagnose food allergies and avoid mislabeling children as allergic to particular foods (*Stone, 1997*). There are no symptoms pathognomonic for food allergy and no single laboratory tests are diagnostic. Therefore, the diagnosis has to be based on controlled elimination and challenge procedures (*Muraro et al., 1997*).

Food allergy is often related to atopic dermatitis and vice versa in early childhood. In many studies atopic dermatitis has been evaluated and reported without a proper evaluation of a possible coexisting food allergy. (*Johansson et al., 1997*).

The expression of allergic diseases may vary with age. Symptoms may disappear or be replaced by other symptoms. In infancy the main atopic symptoms are atopic eczema, gastrointestinal symptoms, and recurrent wheezing, whereas bronchial asthma and allergic rhino

conjunctivitis are the main problems later in childhood. Adverse reactions to food, mainly Cow's milk protein and hen's egg, are most common in the first year of life, and correspondingly sensitization to these allergens occurs early in life. In general, sensitization and development of clinical food allergy develop in the order of exposure (*Host and Halkens', 1998*).

The hygiene hypothesis proposes that exposure to certain infections and vaccines might influence the direction of immune responses and protect against the development of atopy (*McCune et al., 1998* – *Medeiros et al., 1998* - *Gruber et al., 1998* ). However, not all infections are protective. Recurrent lower respiratory tract infections in early childhood are a recognized risk factor for asthma in later childhood (*Arshed et al., 1998* ). The most common respiratory pathogen in infancy is respiratory syncytial virus and this has been linked to later development of wheeze, asthma, and airway obstruction (*Korppi et al., 1998* ; *Stein and Martinez, 1998* ).

## **AIM OF THE WORK**

This work will attempt to approach the immunologic basis, diagnosis and treatment of atopic diseases, with particular focus on food allergy. Also primary and secondary prevention of them will be targeted.

## ATOPIC DISEASES IN CHILDHOOD

### **Definitions :**

- Allergy:** is the clinical manifestations of immunologically mediated reactions to foreign substances, usually proteins.
- Atopy:** is the genetic propensity to produce IgE antibodies after exposure to allergen. This can be confirmed by means of skin prick test or measurement of specific IgE antibodies in the serum (allergic sensitization) (*Arshad et al., 2000*).
- Atopic Diseases:** are the clinical syndromes of the atopic diseases such as eczema, asthma and rhinoconjunctivitis defined by a group of symptoms and signs.
  - \* **Atopy and atopic disease:** The distinction between atopy and atopic disease is important. A child with atopy produces specific IgE antibodies after exposure to common environmental allergens and is said to be sensitized to that allergen. The presence of specific IgE antibodies is measured by means of a skin prick test or radioallergosorbent testing (RAST). While eczema, asthma and rhinoconjunctivitis are clinical syndromes each defined by a collection of symptoms and signs and are commonly referred to as the atopic diseases. While most children with these conditions are atopic, some are not, and, conversely, some children with atopy may not manifest atopic disease (*Gold and Kamp, 2000*). Atopy is only one of many factors involved in the pathogenesis of these disorders.

**-At-risk population:**

Those who have a genetic predisposition and are at higher risk of development of allergic diseases. At present, this is usually assessed through family history of allergy, presence of other allergic diseases, or allergic sensitization.

## EPIDEMIOLOGY OF ATOPY

The International Study of Asthma and Allergies in Childhood (ISAAC) Phase One demonstrated that there are large variations in the prevalence of allergic diseases varying between asthma, allergic rhinoconjunctivitis and atopic eczema throughout the world (differences of between 20-fold and 60-fold between centres) (*Williams et al., 1994*).

Perhaps more importantly, it showed that the international patterns of disease prevalence cannot be explained by the current understanding of the etiology of these conditions. A consistent finding in Phase One was the marked differences in asthma prevalence in populations with similar genetic or ethnic backgrounds (*Beasley et al., 1996*), suggesting that environmental factors in the broadest sense are the major determinants of the prevalence of asthma in a community.

Ecological analyses using Phase One data found weak positive associations with economic development and dietary trans fatty acids for all three diseases, while negative associations were found for tuberculosis and greater plant intake in the diet. In contrast, no clear associations with pollen, immunizations, tobacco, climate or antibiotics were shown (*Weiland, et al., 1994* – *Foliaki et al., 1996*).

These findings suggest that the protective effects of dietary factors (consumption of cereals, starch and vegetables) and exposure to infection (tuberculosis) are worthy of further exploration.

ISAAC Phase Three showed that there was marked variation in the global prevalence of atopic disease. This variation occurs not only between countries but also regionally within countries, with the highest prevalence being in westernized, industrialized countries. In these countries the prevalence of atopic disease has been rising since the latter