

# SKIN TESTING OF NORMAL CHILDREN AND CHILDREN WITH BRONCHIAL ASTHMA AND ASTHMATIC BRONCHITIS

## THESIS

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## **AIM OF THE WORK**

- 1 -  
AIM OF THE WORK

Bronchial asthma and asthmatic bronchitis are common conditions encountered in infants and children. Many such cases may be an allergic manifestation to various foods or other allergenes. The aim of our study is to determine the response of;

- normal children,
- children with asthmatic bronchitis, and
- those with bronchial asthma, to skin testing with :
  - . Egg albumin.
  - . Milk proteins;
    - × casein,
    - × alpha-lactalbumin ( $\alpha$ -L.A.),
    - × beta-lactoglobulin ( $\beta$ -L.G.), and
    - × bovine serum albumin (B.S.A.).

# **INTRODUCTION**

## INTRODUCTION

### Definition of bronchial asthma :-

Bronchial asthma is a variable, intermittent, reversible, obstructive pulmonary syndrome, that includes many bronchospastic airway disorders. It is characterized by recurrent, periodic episodes of paroxysmal wheezing frequently associated with dyspnea and cough (Creip, 1976).

### Etiology of bronchial asthma :-

Prior to puberty, asthma is twice as common in boys as in girls, and this is called "early onset asthma", but it affects males and females equally during adolescence and thereafter and it is called "late onset asthma" (Ellis, 1973).

Bronchial asthma is a complex disorder in which biochemical, neurological, immunological, infectious, endocrine and psychological factors play a role of varying degrees of importance in different individuals (Vaughan, et al, 1979).



(1) Neurological and biochemical factors :-

Patients with bronchial asthma show abnormal biochemical response to injection of epinephrine. Normally, it stimulates beta 2 receptors in muscles and liver leading to hyperglycaemia and lactacidemia, but in asthmatics it does not do these effects (Moller, 1975). Rackemann and Edwards, (1962), postulated that there is a generalized abnormality in structure and function of the beta adrenergic receptors of the adenylyl cyclase complex, in patients with bronchial asthma. Additional support for this concept derives from studies in vitro which show that leukocytes from asthmatics have deficient synthesis of cyclic adenosine monophosphate (cyclic AMP) after stimulation by catecholamines, when compared with leukocytes from normal individuals (Kelly, et al, 1974).

(2) Immunologic Factors :-

The immune system consists of cells and cell products (Leddy, et al, 1977). The principle cells of immune system are lymphocytes, plasma cells and macrophages, which are collectively organized into lymphoid tissue (Harrison, et al., 1980). The two

populations of immuno competent lymphocytes are referred to as B-cells and T-cells (Krupp, et al, 1979). B-lymphocytes have receptors on their surfaces for particular antigens (Parker, 1975). When the antigen binds to the cell, the cell is stimulated to be divided, and its daughter cells are transformed into plasma-cells, which secrete large quantities of immunoglobulins (Ganong, 1979). The T-cells are responsible for cell-mediated immunity (Jones, 1976). Once they are activated, lymphokines will be produced, that participate in attacking foreign proteins (Spitzer, 1977). Various subsets of T-cells are recognized according to function known as helper cells, suppressor cells, and cytotoxic effector cells (Harrison, et al , 1980). Macrophages are involved in the initial ingestion and processing of some antigens, before interaction with lymphocytes (Bellanti, 1978). Specific adaptive immunity is in large part due to antibodies or immunoglobulins, a family of glycoproteins which are able to combine with foreign antigens (Goldman and Goldblum, 1977). Five immunoglobulin classes (Ig G, IgA, IgM, IgE, and IgD) are recognized on the basis of structural

difference of their heavy chains (H-chains), including the aminoacid sequence and the length of the polypeptide chain (Roitt, 1977).

The immune system protects the individual from the attacks of microorganisms by preventing their entry or facilitating their removal. The same immunologic mechanisms, however, may cause damage to normal tissues, especially if the immune response is excessive or prolonged (Holborow and Reeves, 1977). Five mechanisms of immune injury have been known. Type-I (anaphylactic reaction) in which pharmacologically active substances are released from mast cells or basophils as a result of the binding of the antigens to the IgE antibody attached to the surface of these cells (Middelton, et al , 1978). Asthma, allergic rhinitis, and urticaria are examples of this type of reaction (Fudenberg, 1978). Type II, III, IV and the recently discovered type V reactions are all unrelated to our study.

Asthma may be extrinsic, intrinsic or mixed. The extrinsic (atopic or allergic) asthma occurs in atopic persons, with positive family history of

allergy, increased titre of skin-sensitizing antibodies or reagins (IgE), positive skin tests and attacks appear after exposure to some environmental factors that may be either inhalants, as pollens, dust, animal dander and feather, or ingestants, as eggs, milk, fish, wheat and yeasts, or contactants, as wool, nylon and some make-up products and creams (Phelan, 1972). The inhalant enters with the inspired air, to react with an IgE antibody that was previously formed on previous exposure to the same inhalant (Williams and Phelan, 1975). Type-I reaction takes place and histamine, slow reacting substance of anaphylaxis (SRS-A), and probably other mediators are released from the mast cells (Gell, et al, 1975). The ingestants reach the blood after being absorbed from the gut and follow the same way as inhalants. Ingested non-protein substances, particularly drugs, of which asprine is a notable example, occasionally cause asthma by forming haptens (Benacerraf, et al , 1972).

(3) Infectious Factors :-

Respiratory infection commonly provokes asthmatic attacks, especially in intrinsic asthma. In contrast to extrinsic asthma, intrinsic asthma occurs in non-atopic individuals, without family history of allergy to exogenous substances. Patients with intrinsic asthma show negative skin test and low IgE levels. It occurs most frequently in the first two years of life (Kempe, et al , 1980). In addition, respiratory tract infection is often present and males are more frequently affected than females. It is sometimes referred to as asthma due to infection, asthmatic bronchitis, infective asthma, non atopic or non allergic asthma (Mac Loed, 1974). Bacterial infection play no role, but certain viral infections especially respiratory syncytial virus (R.S.V.) and para influenza virus have been shown to cause exacerbation of wheezing in children with asthma (Speer, 1966, and Kempe ,et al, 1980).

The term mixed asthma is used when a clear precise separation of asthma into one of the extrinsic or intrinsic categories is not possible (Horowitz and Hong, 1977).

(4) Endocrine Factors :-

Little is known about the role of endocrine factors in the etiology of asthma. It was found that asthma improves in some children at puberty. It is said that there is an increased incidence in Addison's disease, and that asthmatics with thyrotoxicosis do not respond to treatment until the endocrine problem is brought under control (Lowell, 1978).

(5) Psychologic Factors :-

Asthma has long been thought to be a disorder influenced to a great extent by emotional factors, but Ellis (1977), showed that emotional incidents play an important role as precipitants in many children, rather than being an etiologic role. Williams and Phelan (1975), found that behavioural disturbances were uncommon in most asthmatic children, although they found them less socially mature and more demanding for mother's attention. Also these patients, especially those with severe asthma, show more anxiety, more worry than other children and reacting to stress by an aggressive behaviour.

(6) Seasonal Factors :-

Bronchial asthma seems to be more common in the winter and spring times (Gerrard, 1980). Weather factors, including temperature, humidity, barometric pressure, wind velocity and air pollution may influence the development of asthma. How these various factors work, is remaining unclear (Forfar and Arneil, 1978).

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