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# PSYCHOSOCIAL STUDY OF BRONCHIAL ASTHMA IN MALE CHILDREN

THESIS

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The Master Degree

IN

CHILDHOOD STUDIES  
PSYCHOMEDICAL DEPARTMENT

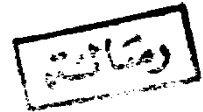


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TO MY MOTHER

WHO GIVES ME A LOT



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INTRODUCTION  
&  
AIM OF THE WORK

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Asthma is a complex disorder in which biochemical, immunologic, infectious, endocrinal and psychological factors play roles of varying degrees of importance in different individuals.

It is one of the leading causes of chronic illness in childhood, affecting 2.5 - 5% of the general population, the vast majority of the cases "approximately 60%", consists of children under the age of 17 years, who are sufficiently debilitated that they are responsible for nearly 25% of total school days missed by all children (Purcell et al., 1972; and Weitzman et al., 1982). This disruption suggests that the disease is going to involve a major social-psychological factor, no matter what its aetiology (Aaronson, 1972).

In our country, the incidence of asthma among diseased children presenting to the out-patient department, Children's Hospital, Cairo University, was found to be 2.2% (El Hefny, 1964).

Clinicians have known for a very long time, that asthma is often accompanied by emotional disturbances and that emotional stress can be responsible for precipitating and aggravating attacks of asthma (Graham and Rutter, 1970; McLean and Ching, 1973), but it is very rare to find asthma caused exclusively by psychological factors (Brown, 1973).

This study is a trial to highlight the possible contributing psychosocial factors to bronchial asthma in children, and this can be cleared in the light of these questions:

1. Are there differences between asthmatics and controls in family influences?
2. Are there differences between asthmatics and controls as regards their developmental history?
3. Are there differences between asthmatics and controls in psychiatric and behavioural disorders?
4. Are there differences between asthmatics and controls in personality trait?.

REVIEW  
OF  
LITERATURE

DEFINITIONS:

Asthma is a Greek word that means "breath hard" (Kuzemko, 1976). This meaning remained intact through centuries although the pathogenesis of the disease is still poorly understood.

The United States National Tuberculosis Association (1967) defined asthma as "a disease characterised by an increased responsiveness of the trachea and bronchi to various stimuli, and made manifest by difficulty in breathing due to generalised narrowing of the airways. This narrowing is dynamic and changes in degree, either spontaneously or as a result of therapy (Williams and Phelan, 1975). Periods of complete or almost complete freedom from symptoms may occur either spontaneously or following treatment. Attacks of asthma may be brief and mild or severe and prolonged reaching the stage of status asthmaticus (Freedman et al., 1975).

The Committee on Diagnostic Standards for Non-tuberculous Respiratory Diseases proposed the following definition based on clinical terms "an illness that is manifested clinically by intermittent

episodes of wheezing and dyspnoea, generally associated with a hyper-responsive state of the bronchi which may be antigen mediated. It is differentiated from other obstructive airway diseases by its usual irreversibility (Aaronson, 1972).

PREVALENCE:

Asthma occurs more commonly in children than in adults and in boys than in girls by a ratio of about 2 : 1 (Apley and McKeith, 1962; Graham et al., 1967; and Purcell et al., 1972). Estimate of prevalence ranges from less than 1% up to 12%; 0.8% in Copenhagen school children aged 7 to 14 years (Frandsen, 1958); 0.85% in rural and urban community in West England (Peltonen et al., 1955); 1.2% in the 7 year old children in a survey in England (Lee et al., 1983); 1.4% in Stockholm school children aged 7 - 14 years (Kraepelien, 1954), 1.8% in Birmingham school children aged to 15 years (Morrison-Smith et al., 1971), 1.69% in children aged 5 - 6 years in Switzerland (Varonier, 1970); 2.3% in Isle of Wight children aged 9 - 11 years (Graham et al., 1967); 4.8% in Aberdeen school children aged 10 - 15 years (Dawson et al., 1969); 4.9 in Peterborough school children aged 5 - 11 years (Kuzenko, 1976); and 12.1% in American children aged 10 - 14 years (Broder et al., 1962).

These differences may be real or may reflect

methodological differences. There has been no uniformity in sample selection, definition of asthma, method of examination and follow up in various studies.

In Egypt, the incidence of asthma among diseased children presenting to the outpatient department Children's Hospital, Cairo University, was found to be 2.2% (El Hefny, 1964).

## PSYCHOPATHOLOGY

(Causal, Contributing and Maintaining Factors)

### I. BIOLOGICAL MODEL:

#### 1. Immunologic Concept of Asthma:

In 1908, Von Pirquet developed the concept of hypersensitivity and in 1923, Coca and Cook, the concept of atopy. These concepts were rapidly applied to explain asthmatic attacks in atopic subjects and those who were shown to be sensitive to pollens of grasses, house dust, foods, animal furs and dander and fungi. Pepys (1973) used the term "atopy" to mean the capacity to produce reaginic antibodies (IgE) as evidenced by skin test or serum IgE level. Atopic diseases tend to be associated with raised serum IgE levels. About 50% of patients with allergic asthma have markedly raised IgE levels (Johansson et al., 1975). These antibodies react with the antigens on the surface of the mast cells in the bronchial wall and release histamine, slow reacting substance of anaphylaxis (SRS-A), and other pharmacologically active substances, which are responsible for bronchial muscle

contraction and inflammatory reaction in the mucosa and submucosa (Fig. a). Tomioka and Ishizaka (1971) showed that the mast cell is the only cell in primate lung to which IgE myeloma protein will bind. Salvato (1961) demonstrated that fewer intact mast cells could be detected in the lung during an acute asthmatic attack than in normal individuals.

Histamine is the major pharmacological mediator stored in mast cells. In vitro, it can be shown to constrict bronchial smooth muscles and it can be detected in asthmatic lungs after in vitro challenges with specific antigen (Schild et al., 1951; Brocklehurst, 1960). Despite of these findings, it is doubtful whether histamine plays a significant role in causing bronchospasm experienced by asthmatics (Austen and Blannerhasset, 1968). Lichenstein and Bourne (1971) found that histamine can act also in an indirect way by activating the adenyl cyclase enzyme which catalyses the formation of 3', 5' AMP from adenosine triphosphatase (ATP) (Sutherland and Robinson, 1966), which then effectively inhibit further production of amine, thus histamine can control the magnitude of the response of sensitized tissue to antigen.