MOLDS ARE A MAJOR SOURCE OF HOUSE DUST ALLERGENS

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BY

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THIS WORK IS DEDICATED TO THE
MEMORY OF MY FATHER

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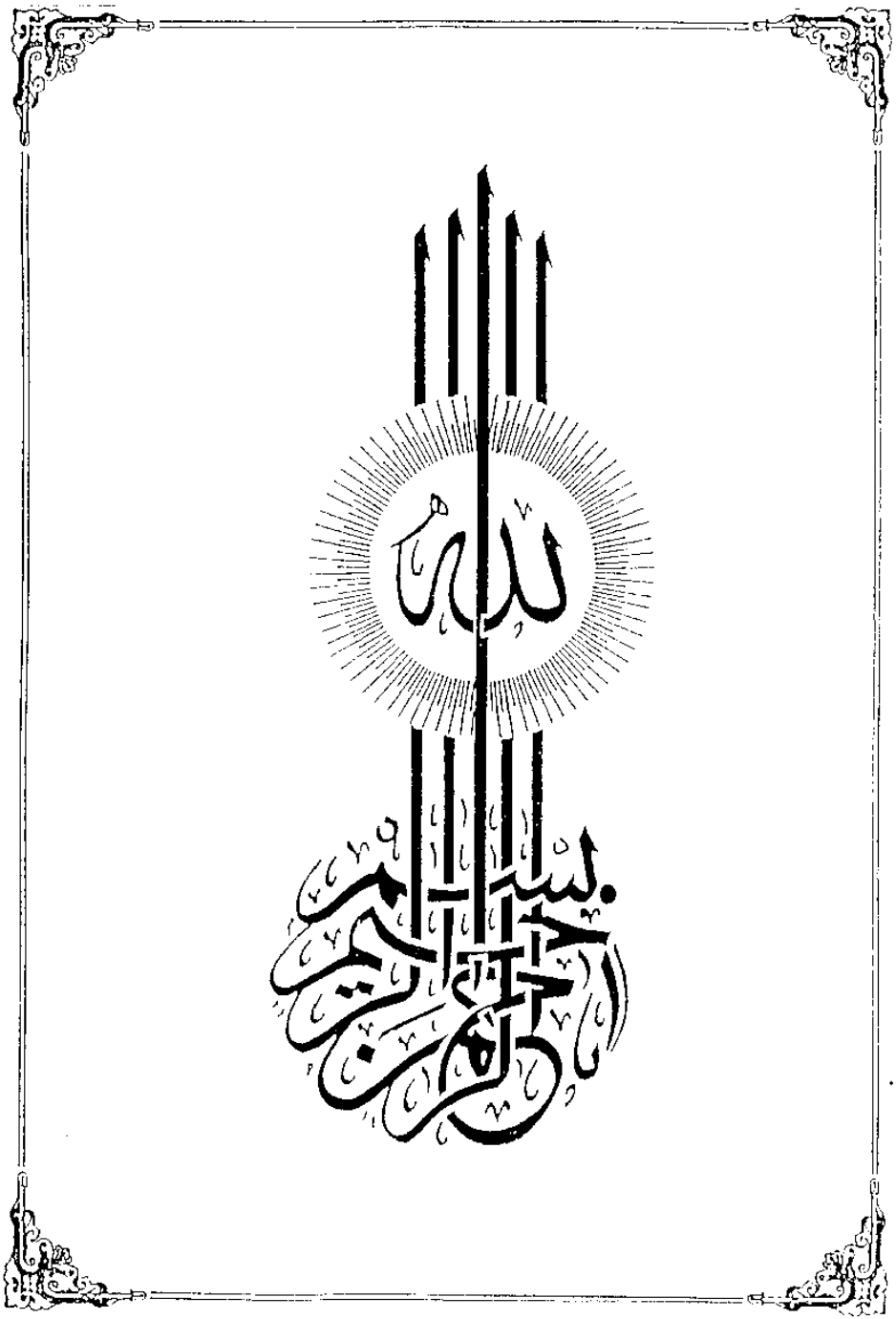

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INTRODUCTION AND AIM OF THE WORK

Allergic disease due to a composite of house dust (H.D.) allergens is a common problem. Common components such as mites, pollen, molds, feathers, animal and human dander, textiles and insect emanations were all possible allergens. Airborne mold spores must take their place along with pollen and dust as an important cause of inhalant allergens. It has been established since long time [Blackley, 1873; Storm Van Leeuwen, 1924; Fraenkel, 1938; Ripe, 1966 and Salvaggio and Seabury, 1971] that the respiratory allergic symptoms can be caused by inhalation of the airborne mold spores.

So the aim of this work is to study the relationship between the H.D. asthmatic patients and the mold allergens.

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Review of Literature

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REVIEW OF LITERATURE

Bronchial asthma is a chronic pulmonary disease characterized by increased bronchial reactivity to various stimuli and manifested by recurrent episodes of more or less generalized airway obstruction. The condition is usually reversible either spontaneously or following administration of appropriate therapy.

The aetiology of bronchial asthma is multifactorial. Altered immunologic state heads the list of the encriminated factors. Various excitants may evoke hypersensitivity reactions in susciptible subjects and lead to transient airway obstruction that characterizes bronchial asthma [Seoudi, et al. 1974]. This reaction may result from inhalation of allergens, the ingestion of foods such as: fish, eggs, milk and some fruits or drugs as: beta-blockers, cholinomimetics, histamine and histamine liberators as morphine and d-tubocurarine, also prostaglandin synthetase inhibitor drugs as aspirin, indomethacin, ibuprofen,.....etc.

It is now more than a century since it was demonstrated that bronchial asthma can be precipitated by the inhalation of allergens [Blackley, 1873] and more than half a century has elapsed after the demonstration that H.D. is important in this respect [Kern, 1921 and Cooke, 1922]. Cooke in 1922 reported that in 327 cases of bronchial asthma a reaction to H.D. of diagnostic importance was found in 109 (33%). Cooke's observations on the frequency of H.D. sensitiviness in bronchial asthma have been abundantly confirmed. McLaughlin

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in 1927 reported that, out of 1186 patients, 63% were H.D. positive and 126 of them reacted to H.D. alone. Rowe in 1927 reported 160 cases, of which 42% were H.D. positive. He thought that the reaction were probably explainable by the presence in the dust of such substances as animal dander, hair, orris or glue and that there was no substance present specific for the H.D.

The importance of H.D. allergens for bronchial asthma must be considered in the light of the frequency and importance of allergic factors as a whole in bronchial asthma. This is very much dependent on patient population, for allergy plays a more important role in children and young people than in adult patients. In a study of 809 children with bronchial asthma, allergy was found to be present as a precipitating factor in about 85% [Aas, 1969] and the allergological investigation of 1035 children with asthma showed that 63% of them reacted to the inhalation of H.D. allergens [Aas, 1970]. Thus such allergens seemto be the most important in extrinsic asthma.

H.D. has long been known to cause sneezing and wheezing in sensitive subjects [Maunsell, et al. 1968; Freedman,1970 and Aaronson 1972]. However, aggravation of these allergic respiratory symptoms is extremely common by exposure to various types of dust. Inorganic dust such as most road dust precipitates symptoms on an irritant basis. Organic dust may act in the same way or may evoke immunologically mediated responses. Dust of household origin is considered

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as a cause of allergic rhinitis and asthma [Mathews, 1978].

Patients with allergy to H.D. typically have rhinitis, conjunctivitis and asthma which is triggered by cleaning the house, dusting, beating rugs or making beds. These patients have continuous symptoms of varying severity and improve in the summer months during periods of less intense exposure. The diagnosis is confirmed by demonstrating the presence of Immunoglobulin E(IgE) antibody to a mixture of H.D. allergens. It is advisable to consider the use of two sources of H.D. extract in order to screen for a broad spectrum of antigen reactivity [Stechschulte, 1982].

Özkaragöz in 1967 performed skin test with pollen, mold spores and H.D. on 106 children having bronchial asthma in Turkey. He found that 66.1% of the patients were sensitive to H.D.

Voorhorst [1972] in Holland, recorded that 50% of the pollinosis patients gave positive prick skin test reactions to H.D. extracts.

More than 80% of a group of 133 asthmatic children in Britain also reacted to H.D. [Sarsfield, 1974].

Hendrich, et al. [1975] studied 656 asthmatic patients. 544 (84%) gave positive immediate skin prick tests to at least one of 22 common allergens used routinely. The positive reactions were most

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commonly seen to H.D. (82%), followed by pollen (66%), animal danders (38%), food (16%), Aspergillus fumigatus (16%) and other molds (21%). There was a highly significant association of positive history of allergy with positive prick skin tests for all allergens studied.

106 asthmatic patients were studied in Nigeria. 87% of them were positive to prick skin tests with one or more allergens. The commonest reactions were to H.D. (58%), house dust mite (H.D.M.) (45%). 51% of a group of controls were also positive on skin testing but the pattern of responses was different from the asthmatic patients [Warrell, et al. 1975].

The high prevalence of skin test reactivity to the H.D. extracts among the allergic subjects has fostered the presumption that H.D. allergy is common, particularly in subjects with perennial respiratory allergy. Such observations make it necessary to identify the nature of H.D. allergenicity. Some authors considered that, H.D. containes a unique (but undefined) allergen [Kern, 1921; Cooke, 1922 and Storm van Leeuwen, 1929], others believed that house dust's allergic activity merely reflect the sum of its component allergens [Rowe, 1927; Berrens, 1970; Aas, 1976; Mathews, 1978 and Stechschulte, 1982]. Since H.D. is a mixture of numerous different components, there is good reasons for confusion regarding the origin of its precise allergenic properties. The dust may be composed of large particles and microscopic fragments of textile fibres, hair and skin detritus from human individuals and

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animals, degradation product of feather, kapok, minerals, paints and insecticides, natural and synthetic items of the daily household, plants, food and insects. Added to this, other organic or nonorganic contaminants such as pollen, mold spores and minerals from the outdoor atmosphere dependent on geographic situation, season, weather, and wind factors. All or most of these material are found in different stages of denaturation and may provide nutritive sources for numerous microorganisms such as viruses, bacteria, algae, molds and fungi, as well as many species of insects and mites. Also there may be microscopic organisms not yet defined or characterized to date. Thus it is no wonder that susceptible individuals become allergic to one or several of these components which are inhaled regularly through several hours every day [Aas, 1976].

The allergen content of commercial H.D. extracts was examined by some authors [Voorhorst, 1977; Carlsen, et al. 1979; Pauli, et al. 1979 and Mansfield and Nelson, 1982]. Mite, cat and dog danders were the major allergens in the extracts and the endo dust was different from the H.D. extracts. There were significant apparent differences in potency between H.D. extracts as obtained from the manufacturer [Storm van Leeuwen, 1929 and Sarsfield, 1974]. Human dander and cotton linters, represent only minor contributors to H.D. allergenicity [Voorhorst, et al. 1967].

Many researchers reported that there was almost perfect

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identity between H.D. allergen and mite allergen. In 1928 Dekker reported that mattresses contained a virtual mite detritus and the mites were the dust component responsible for asthma and rhinitis in individuals who are atopic to H.D. This is supported also by Voorhorst, et al.[1964]; Maunsell et al. [1970]; D'Souza, et al. [1973] and Miyamoto et al. [1974]. In 1967 Voorhorst, et al. established a primary role for Dermatophagoides pteronyssinus (a mite commonly found in the H.D. of Dutch homes) as the common allergen of H.D. Also Maunsell, et al. in 1968 supported their hypothesis. Moreover, they found that an increase in the number of Dermatophagoides pteronyssinus (D. pteronyssinus) present in H.D. led to an increase of allergenic potency. Mite extracts have also been studied by several other techniques. Bronchial inhalation challenge tests with D. pteronyssinus or D. farinae have been positive in the large majority of H.D. sensitive asthmatics [McAllen, et al. 1970]. Several investigators have produced nasal symptoms in H.D. sensitive subjects by nasal challenge tests with mite extracts [McAllen, et al. 1970 and Sarsfield, 1974]. Conjunctival reactions can also be elicited. Several laboratories have obtained positive RAST (radioallergosorbent test) with D. pteronyssinus and/or D. farinae antigens and sera of H.D. sensitive patients [Holford-Strevens, et al. 1970 and Sarsfield, 1974].

Voorhorst, et al. 1967 suggested that secretory and excretory products of the mites may play a part. This may well be so, but it should also be remembered that H.D. contains other allergenic

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