CLINICAL SIGNIFICANCE OF GOAT WOOL ANTIGEN & SHEEP WOOL ANTIGEN IN EGYPTIAN ASTHMATICS

THESIS

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(Medicine)

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بسم الله الرحمن الرحيم
قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم.
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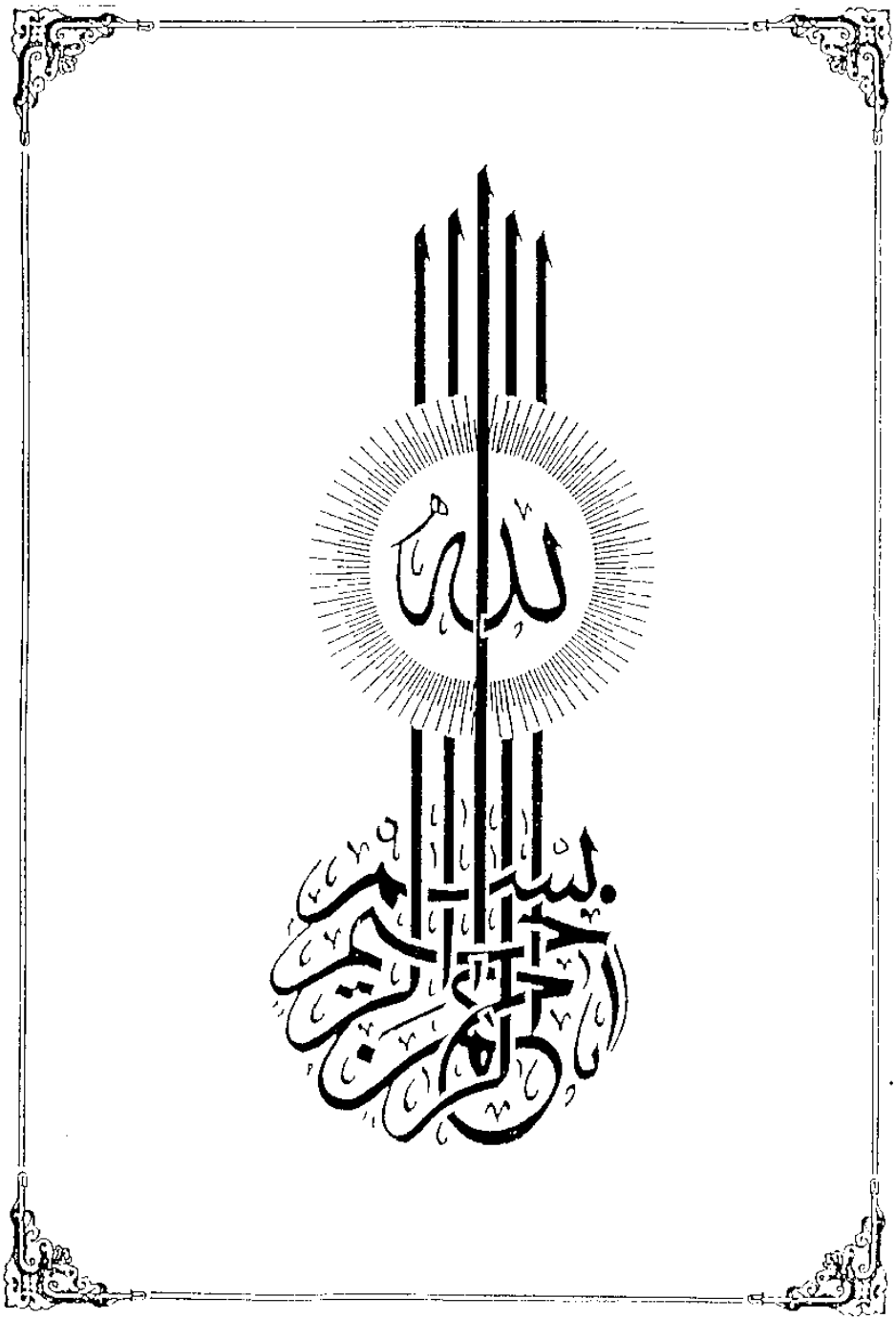

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To My Parents,
My Husband
&
My Daughter

ACKNOWLEDGMENT

FIRST AND FOR MOST THANKS ARE DUE TO ALLAH

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ABBREVIATIONS

AA	*Arachidonic Acid*
AB	*Antibody*
BAL	*Bronchoalveolar lavage*
cAMP	*Cyclic Adenosine Monophosphate*
C.D.	*Cotton Dust*
D.H.	*Dog Hair*
FEV_1	*Forced Expiratory Volume in the First Second*
H.D.	*House Dust*
MBP	*Major Basic Proteins*
M.M.	*Mixed Moulds*
M.P.	*Mixed Pollens*
NANC	*Non Adenergic non Cholenergic Nerves*
NKA	*Neurokinin A*
PAF	*Platelet Activating Factor*
PEFR	*Peak Expiratory Flow Rate*
PHM	*Peptide histidine Methionine*
RAST	*Radio-Allergen Sorbant Test*
SP	*Substance P*
SRS-A	*Slow Releasing Substances of Anaphylaxis*
VIP	*Vasoactive Intestinal Peptide*

CONTENTS

INTRODUCTION
AND
AIM OF THE WORK

INTRODUCTION:

It has been noticed during allergy practice among Egyptian asthmatics that goat wool allergy is distinct from sheep wool allergy since a positive skin test to any of them was observed to occur separately from one another.

The prevalence of sources of goat wool and sheep wool antigens are common in our environment, oriental carpet does contain goat and sheep wool and seems to be decisive in generating goat wool allergy as well as sheep wool allergy. The intrinsic allergenicity of goat wool antigen seems to be very high, so that a very close association with goat is not necessary to develop hypersensitivity among susceptible subjects.

AIM OF THE WORK:

The aim of the present work is to study the prevalence and clinical significance of goat wool antigen and sheep wool antigen among Egyptian asthmatics in an attempt to investigate the importance of the former antigen as an inhaler allergen among Egyptian asthmatic population and to define the role of goat wool allergen in provoking various hypersensitivity reactions, either alone or in association with other allergens.

REVIEW OF LITERATURE

BRONCHIAL ASTHMA

Definition:

Warwick (1978) states that asthma is a state of airway hyper reactivity with reversible attacks of airway obstruction with episodic wheezing , dyspnea and cough. It is provoked by many factors as allergens (inhalers, ingestants and injectant), exercise, infection, drug (as aspirin) and emotional factors. All these factors will act through mediator release ***(Steim and Fulginiti, 1980)***. This narrowing is dynamic, and changes in degree either spontaneously or because of therapy ***(Nicholas, 1980)***.

Bronchial asthma is a complex disease, involving abnormalities of the immune system, airway autonomic receptor and other pathways ***(Austen et al., 1977)***. This reaction develops not only due to release of mediators but also exaggerated response to these mediators ***(Warren and Tse, 1974)***. ***Scadding (1983)*** defined bronchial asthma as a disease characterized by wide variation over short period of time in resistance to flow in intrapulmonary airways. ***Druce (1985)*** stated that bronchial asthma is a chronic disease that is episodic in nature with acute exacerbation and symptoms free period of variable duration.

Asthmatic patients are mostly diagnosed by a triad of episodic cough, wheezing and dyspnea, which are variable in severity and duration *(Daniele, 1988)*. *Holtzman (1982)* stated that allergy is the predominant factor which precipitates bronchospasm in 35% of asthmatics and is contributory in another 30-60%.

Other predominant factor precipitating bronchospasm is infection; in young children, respiratory syncytial virus infection is a common cause of wheezy episodes while in adults, rinoviruses are the cause *(Horn et al., 1979)*. Also bronchopulmonary aspergillosis, polyarteritis nodosa and parasite induced asthma during pulmonary migration *(Lawler and Fischer, 1981)*.

TABLE (1): TYPES OF ASTHMA

Quoted from Wyngaarden and smith (1988)

TYPE	INITIATION FACTORS
EXTRINSIC	IgE mediated external allergen
INTRINSIC (CRYPTOGENIC)	? non antigenic stimuli
MIXED (ADULT ONSET)	?
EXERCISE INDUCED	Alteration in airway temperature and humidity; mediator release
ASPIRIN SENSITIVE (ASSOCIATED WITH NASAL POLYPS)	Aspirin and other non steroidal anti inflammatory drugs
ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS (ABPA)	hypersensitivity to aspergillus species (not infection)
OCCUPATIONAL	Metal salts (Platinum, Chrome, Nickel) antibiotic powder (penicillin, sulfathiazole and tetracyclin) Toluene di-isocyanate (TDI) Flour, wood dust, cotton dust (Byssinosis) Animal proteins

I. Extrinsic:

Its onset is usually in childhood (5 year) or early adult life (before 30 year), There is a known external allergen as dust, pollen and danders; positive immediate skin test to specific

antigen. IgE is raised in 50%-60% of cases and gives positive response to provocation tests involving the inhalation of specific antigen.

It is intermittent in attacks and in 50% there is family history of multiple allergies (atopy) as asthma, hay fever and ecczyma. It is subdivided into atopic and non atopic by ***Pepys and Frankland (1970).***

TABLE (2): TYPES OF EXTRINSIC ASTHMA

	EXTERNAL ATOPIC	EXTERNAL NON ATOPIC
AGE OF ONSET	mainly early in life	mainly adult (before 30 years)
DETERMINED BY	constitutional factors	environmental factors (occupational)
SKIN TEST REACTION	prick test and usually multiple to common allergens	Intradermal and single to particular antigen
INHALATION TEST		
- *Asthma*	immediate	late (after 4-6 hours)
- *Fever*	-	+
- *Leucocytosis*	-	+
- *Eosinophilia*	+	±
- *Antibody specific IgE*	+	?
- *Precipitins*	-	+

II. Intrinsic:

Previously, this type of asthma was known as intrinsic asthma but since it represents a heterogenous group of asthmatic patients in whom different mechanisms, many of which are unknown may play a role in triggering attacks, it is more appropriate to be classified as cryptogenic asthma (*Bonini, 1980*). It gives negative skin test, IgE level is normal or even low. Onset is usually in older adults after 30 years and course is usually continuous, Family history is less common than extrinsic 20%. *Parker (1980)* found that in both groups of asthma, there is blood and sputum eosinophilia. Airway hypersensitivity in intrinsic groups may be due to release of triggering substances from mast cells or due to abnormal neurogenic reflexes or both as a result of nonspecific stimuli as infection, pollution, exercise, cold or psychogenic *(Nadel et al., 1984)*. It was found that in asthmatic patients, there is lowered threshold for stimulation of irritant receptors of vagus afferent nerves and viral infection has the capacity to lower the threshold for stimulation of irritant receptors *(Kaliner, 1985)*.