## SHORY TERM RESEARCH PROJECT REPORT:

DEFECTION OF ALLEMGERS IN ALLERGIC REPORTS

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UNIVERSITI SAINS MALAYSIA SHORT TERM RESEARCH PROJECT REPORT: DETECTION OF ALLERGENS IN ALLERGIC RHINITIS BY **ELANGO** PUROHIT ZAHARA MANAP HILMI RAZA HOSPITAL UNIVERSITI SCHOOL OF MEDICAL SCIENCES UNIVERSITI SAINS MALAYSIA KUBANG KERIAN KELANTAN 1988 (Supported by a Universiti Sains Malaysia Short Term Research Grant)

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#### 1. INTRODUCTION:

The study of the detection of allergens in allergic rhinitis requires clear understanding of concepts of allergy and the various diagnostic procedures used in their study.

Allergy may be considered to be a pathological hypersensitivity on the part of the allergic individual to substances (Allergens) or situations to which a non allergic individual does not react or reacts minimally. Allergens are those substances which elicit an allergic response manifested by a hypersensitivity reaction. Allergic reaction is mediated by the presence of an antibody which has been formed by the allergic individuals in response to exposure to an antigen. Antibodies may be found circulating freely in the serum (humoral antibodies) or attached to body cells (somatic antibodies) of the affected individual. Allergic reactions associated with humoral antibody are spoken of as immediate allergic reaction and a re characterized by the familiar skin wheal and erythema reaction. The examples of immediate allergic reactions are Hay fever, asthma and urticaria and in these conditions positive skin tests are elicited by intradermal injections of the appropriate antigen. The particular antibody which is responsible for wheal formation in the immediate reaction is termed reaginic antibody and possesses the characteristics of attaching itself to the skin cells. Delayed allergic reactions include tuberculin reaction, contact dermatitis and they involve somatic antibodies. These conditions do not give wheal and erythema formations.

Allerny itself is not considered to be hereditary; rather, the existence of a family predisposition to allergic disease may be present. Reaginic antibodies may be genetically related. Familial allergic disease is commonly spoken of as atopic allerny.

Allergic rhinitis is an IgE mediated hypersensitivity disease of the mucous membranes of the nasal airways characterized by sneezing, nasal blockage and discharge. Conjunctivitis and bronchial constriction often accompany these symptoms. Allergic rhinitis is commonly found in association with exposure to aeroallergens. Minority of susceptible individuals develops a similar syndrome following ingestion of certain foods, but an immunological basis for these reactions is yet to be established. Allergic rhinitis may be seasonal or perennial. Seasonal allergic rhinitis (Hay fever) is allergy to the pollens of grasses, flowers, trees and shrubs. Perennial allergic rhinitis is caused by house dust, house dust mite and moulds. The common food allergens are sea foods (Crab, shrimp), cow's milk proteins, eggs etc.

IgE comprises only 0.004% of the total serum immunoglobulins. It is composed of two heavy chains (E) and two light chains (K or $\lambda$ ).(Fig. 1)

Immunoglobulin E has the unique property of binding reversibly to high affinity receptors on mast cells and basophils. When two such antibody molecules are bridged by an appropriate allergen biological events are initiated which lead to the degranulation of mast cells, with release of several powerful chemical mediators like histamine, eosinophilic chemotactic factor, slow reacting substance of anaphylaxis, heparin and platelet activating factor.(Fig. 2) Following the release of these mediators, there is an increase in vascular permeability, contraction of smooth muscle in the respiratory and gastrointestinal tract and an influx of eosinophils. This results in the clinical manisfestations of allergic disorders of the immediate type. Most patients with atopic allergy had consistently elevated serum concentrations of IqE. More than 99% of the patients' IgE is free in the circulation and not fixed to mast cells or basophils, thus, a direct measurement of serum IgE should reflect clinical allergy in respiratory and gastrointestinal tract.

The disease allergic rhinitis is extremely common in some countries and is said to affect 10 to 20% of North Americans and 10 to 15% of North Europeans. The incidence and rate of allergic rhinitis and the allergen causing it varies from country to country. Even in Malaysia it is one of the common ENT disease. A review of the literature shows a paucity of investigation on the disease allergic rhinitis in Malaysia. Only one study had been done correlating the clinical findings with the radio allergosorbent test (PAST) results. We are particularly interested to find out the common allergen causing allergic rhinitis in Malaysia and to find out the usefulness of the different diagnostic procedures in the diagnosis of allergic rhinitis.

### 2. OBJECTIVES:

(i) General:

Prospective study on allergic rhinitis in Kelantan

### (ii) Specific

- a. To find out what are the common allergens in Kelantan which are causing allergic rhinitis
- b. Correlation of the different dianostic procedures in the diagnosis of allergic rhinitis.
- c. Having found out the allergens to advise the patient to avoid the particular allergens

The flow chart of data collection is as follows:

History taking (Proforma)

ENT Examination

Allergen skin prick test

Investigations

Data collected was subsequently processed by computer.

### 5. STUDY METHOD:

- 5.1 History taking (Proforma)
- 5.2 ENT Examination Thorough examination of ears, nose and throat were done for all cases.

### 5.3 Allergen skin prick test

- 5.3.1 This is an objective method based on the specific immediate sensitivity conferred by IgE antibodies.
- 5.3.2 Patients were advised to stop antihistamines and other drugs for 72 hours prior to testing.

### 5.3.3 Preparation of the site of the test:

Flexor aspect of both forearms were used for the testing. Patient was asked to clean the test site with soap and water. A ball pen was used to mark the skin adjacent to planned test sites to identify the allergen and control solution used.

5.3.4 Twelve common allergens and one control solution were used for testing. Bencard prick test solutions (England) were used for the test.

### 5.3.5 Prick test method:

Using the applicator attached to the vial cap, one drop of control solution was placed on the skin of the flexor aspect of the forearm. Then in the same way one drop of each prick test solution was placed on the forearm at about 3 cm intervals. Hypodermic needle was used for the pricking. needle was held almost parallel to the skin with its tip in the drop of control solution and pushed until the tip just enters the superficial skin layer. The tip was then slightly raised and then withdrawn. The procedure was repeated with each test solution using fresh hypodermic needle for each time. Excess test solution was wiped away using a fresh piece of cotton wool for each test. The reactions were read after 15 minutes. The strength of each reaction was assessed after that by the degree of erythema and the area of the wheal formed. Strength of each reaction was recorded as recommended by the manufacturer as follows:-

- No wheal. Erythema absent or less than 1mm diameter.
- + Wheal absent or very slight. Erythema present, but not more than 3 mm in diameter.
- ++ Wheal not more than 3 mm in diameter, with associated erythema.
- +++ Wheal between 3 mm and 5 mm in diameter, with erythema.
- ++++ Any larger reaction or one with pseudopodia.

Reactions no greater than that of the control solution was ignored. Allowance was made for the size of the control solution, i.e. if a reaction of + to control solution was shown and a reaction of +++ to the allergen then a true measure of sensitivity for the allergen was taken as ++.

- 5.4 <u>Investigations</u> The following investigation were done for all the cases.
  - 5.4.1 Total white cell count
  - 5.4.2 Absolute eosinophil count
  - 5.4.3 X\_ray paranasal sinuses.

### RESULTS:

The results are presented under the following headings:

- 1. Background Information.
- 2. Clinical History.
- 3. ENT Examination.
- 4. Investigations.

### 1. BACKGROUND INFORMATION:

#### Table 1:

### 1.1 AGE GROUP:

No. Of Patients					
Male	Female				
2	0				
7	5				
27	18				
15	9				
5	2				
3	2				
	2 7 27 15				

Peak incidence is in the third decade (45 patients \_ 47.36%)

### 1.2 SEX:

There were 59 male (62.10%) and 36 (37.9%) female patients.

### 1.3 Ethnic group

Malays constituted the majority (54 patients - 56.84%), followed by Chinese (30 patients - 31.59%) and Indians (10 patients or 10.53%)

#### 2. CLINICAL HISTORY

### 2.1 Response to history of sneezing

71 patients (74.74%) were sneezers compared to 24 (25.26%) non sneezers.

### 2.2 Response to history of Nasal blockage

81 patients (85.26%) had nasal blockage

### 2.3 Response to hisotry of Rhinorrhoea

89 patients (93.68%) had rhinorrhoea. Of which only one patient had purulent discharge, rest of the patients had mucoid discharge.

### 2.4 Response to history of Itching

83 patients (87.36%) had itchiness in either nose, ears, throat or eyes.

### 2.5 Response to history of Cough

39 patients had a history of cough.

### 2.6 Response to family history of atopy

44 patients (46.32%) had a family history of atopy among parents or grand parents.

### 2.7 Response to patient having other atopic diseases

There were 25 positive responses (26.31%).

### 2.8 Occupation:

Majority of the patients (42.11%) were  $\cap$ ffice Workers, field workers forming only 10.53%).

### 2.9 Response to specific contacts

27 patients had specific contact to cats and 22 patients to feathers.

### 2.10 Response to specific factors increasing symptoms:

71 patients had house dust as the specific factor increasing their symptoms followed by cold air in 61 patients

#### 3. ENT EXAMINATION

#### 3.1 Result of Nose Examination

- 3.1.1 <u>Nasal Mucosa</u> was pale in 61 patients (64.21%), purplish in 22 patients (23.16%) and normal in 12 patients (12.63%).
- 3.1.2 <u>Turbinates</u> were normal in 10 patients (10.53%) and hypertrophied in 85 patients (89.47%). There was minimal hypertrophy in 21 patients moderate hypertrophy in 58 patients and marked hypertrophy in 6 patients.
- 3.1.3 Nasal polyp was present in 4 patients (4.21%).
- 3.1.4 Nasal septum was in midline in 53 patients (55.79%) and deviated in 42 patients (44.21%).

### 3.2 Results of throat examination

Throat was normal in 76 patients (80%) and abnormal in 19 patients (20%).

### 3.3 Results of post nasal examination

Post nasal drip was present in 17 patients (17.89%) and absent in 78 patients (82.11%).

### 3.4 Results of ear examination

Ears were normal in 83 patients (86.32%) and abnormal in 13 patients (13.68%).

3.5 <u>Sinus Tenderness</u> was present in 4 patients and absent In 91 patients.

### 4.1 Results of Total White Cell Count

Total count was within normal range in 93 patients (97.89%) and was raised in 2 patients (2.11%).

### 4.2 Results of Absolute eosinophils count

Absolute eosinophils count was whithin normal range in 55 patients and was raised in 40 patients (42.10%).

### 4.3 X ray paranasal sinuses

Table 2.

Finding	No. of Patients	Percentage
Normal Haziness Mucosal thickening Fluid level	39 39 16 1	41.05% 41.05% 16.84% 1.06%

### 4.4 SKIN PRICK TEST

### Table 3

T2	- Straw dust	T8 - Cat Fur
T3	- Candida albican	T9 - Whole egg
T4	- Penicillium notatum	T10 - Lobster
T5	- House dust	T11 - Crab
T6	- House dust mite	T12 - Shrimp
T7	- Poultry feather	T13 - Sardine

- 0 No wheal. Erythema absent or less than 1 mm diameter.
- 1+ Wheal absent or very slight. Erythema present not more than 3 mm diameter.
- 2+ Wheal not more than 3 mm in diameter with associated erythema
- 3+ Wheal between 3 5 mm in diameter with erythema.
- 4+ Any larger reaction possibly with pseudopodia.

0 and 1+ taken as negative.

2+, 3+ and 4+ taken as positive in this study.

	NO. OF PATIENTS											
Class	Ţ2	Т3	T4	T5	T6	T7						
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о .	24	84	81	16	20	14						
1+	5	4	2	3	6	4						
2+	34	5	7	18	12	27						
3+	28	2	5	40	20	39						
4+	4	0	0	18	37	11						

Class	NO. OF PATIENTS										
	T8 T9 T10 T		T11	T12	T13						
0	15	83	38	32	37	69					
1+	3	1	5	4	5-	0.					
2+	.13	8	23	24	19	10					
3+	35	2	23	28	29	16					
4+	29	1	6	7	5	0					

No. of patients negative to all allergens - 6 (6.31%)

No. of patients positive to one allergens - 4 (4.21%)

No. of patients positive to 2 allergens - 5 (5.26%)

No. of patients positive to more than two allergens  $-80 \ (84.21\%)$ 

# - 15]-| 9x 1:0010x TESTS

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#### Skin Prick Test T2 Straw dust T9 Wholeegg T3 Candida Albican T10 Lobster IT4 Penicillum Notatum Til Crab House dust T12 Shrimo T13 Sandine House dust mite Poultry feather cat fur: SNO **T5** T8 T10T11T12T13 T3 T4 T6 Τ7 Ţ9 25 3 3 3 4 0 0 0 Ū 0 27 23 <u>0</u> 3 2 ) 1 i 1 ) () 0 () Ċ 3 4 34. 35 <u>...</u>. .<u>..</u>. 4 Ü ... 3 3 (, <u> 5</u> つ

### SKIN PAIGN TESTS

Skin P	rick	Tes	t						:			
T2	Str	aw di	ust				T9	Who	ie e	gg		
T3	Can	dida	Albi	can	:		T10	Lob	ster			
T4	Per	icilli	ım N	otati	um		711	Crai	0	•		
T5	Hou	ise di	ust				T12	Shri	mp	•	:	
T5	Ноц	se di	ust n	nite			713	Sarc	dine	:		
17	Pou	itry	feat	her	:		:					
T8	cat	fur	•	:	:					· · · · · · · · · · · · · · · · · · ·	:	
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### SKIN PRICK TESTS

Skin Pi	rick	Tes	t									
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T3.	Can	dida	Albi	can			T10	Lob	ster			
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75	Hou	ise d	ust				T12	Shr	np			
T6	Hou	ise di	ust n	nite			T13	San	dine		:	
17	Pou	ltry	feat	her							:	
T8	cat	fur	:		:							
										:	-	
SNO	T2	Т3	T4	T5	T6	T7	. <b></b>	T9	T10	T11	T12	T13
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Fig.1. Schematic diagram of the lg E molecule.

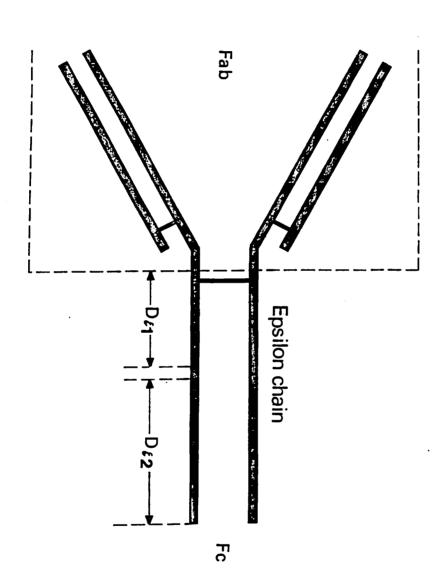
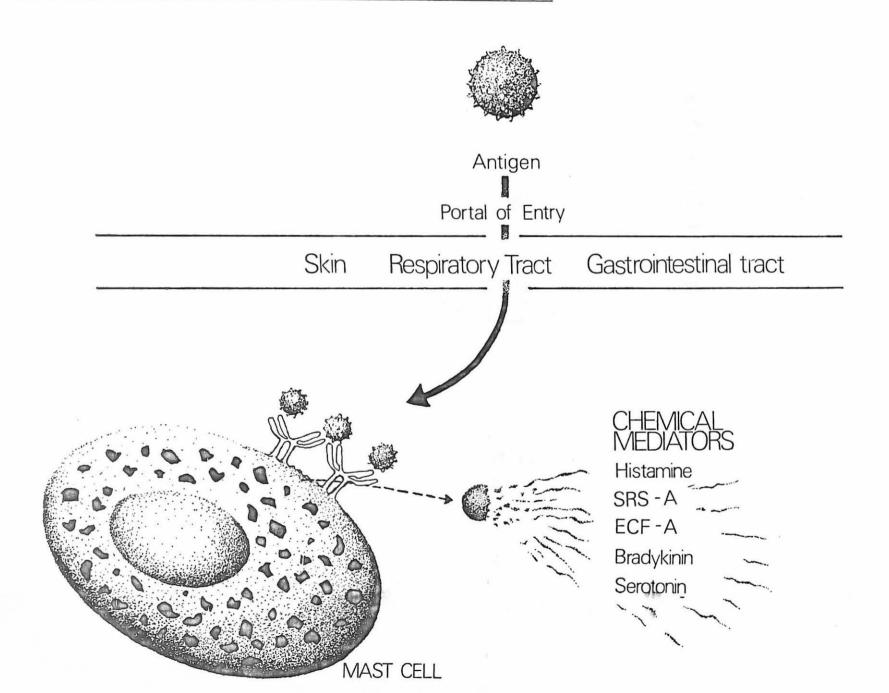
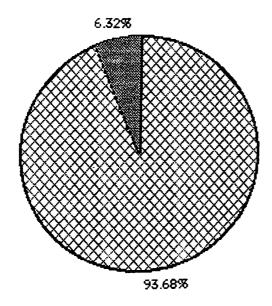


Fig.2. Antigen, antibody complex & degranulation of the mast cells



# RHINORRHOEA

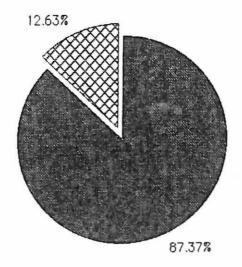




Non Rhinorrhoea

FIG. 3

### **ITCHING**



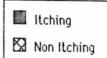
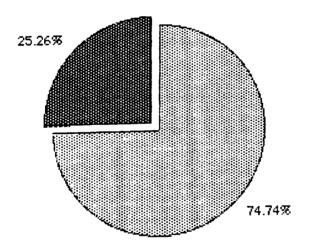


FIG. 4

# **SNEEZERS**



Sneezers
Non Sneezers

FIG. 5

# SNEEZERS & BLOCKERS

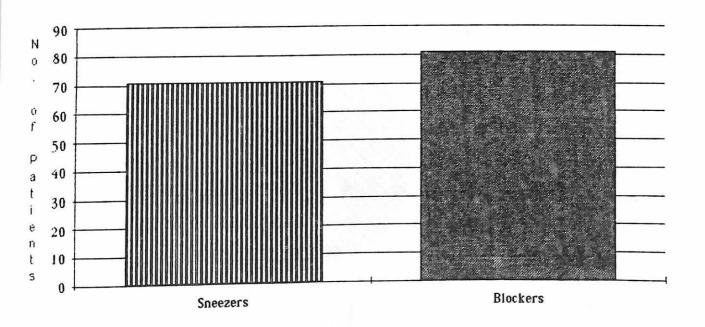


FIG. 6 Sneeters and Nasal Blockers