Specific IgE Antibodies To *Sarcoptes Scabiei* In Scabetic Patients Also Recognize House Dust Mites Antigens

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**Summery**

**Background:** Scabies is relatively a highly contagious inflammatory skin disease caused by infestation with a parasite *Sarcoptes scabiei*. The pathogenesis of scabies is mainly due to & starts after sensitisation of the host to the mite & its products which provoke an allergic response, responsible for a wide spectrum of clinical & histological manifestations.

**Objective:** To determine whether specific IgE against scabies can show a cross reactivity with house dust mites allergens.

**Study Design:** The level of specific IgE against *Sarcoptes scabiei var hominis*, Dermatophagoides farinae & Dermatophagoides pteronyssinus was measured in 30 scabietic patients by ELIZA method & the results was compared using t test with that from the control group.

**Result:** There was a high statistical correlation (P<0.001) between the data of specific IgE against *Sarcoptes scabiei var hominis*, Dermatophagoides farinae & Dermatophagoides pteronyssinus in the patient group in comparison with that from the control group.

**Conclusion:** Some antigens from *Sarcoptes scabiei* body & its fecal pellets cross react with house dust mites body & feaces, since they are related phylogenetically. So specific IgE against scabies can cross react with house dust mites, *Dermatophagoides farinae* & *Dermatophagoides pteronyssinus*, which are important allergens responsible for extrinsic asthma. This cross reactivity may explain the association of severe form of scabies infestation with atopic diseases & the persistence of symptoms in some cases even after proper treatment of the disease.

**Introduction:**

Scabies is a contagious inflammatory skin disease caused by infestation of *Sarcoptes scabiei var hominis* a parasite that colonizes the human epidermis (1). The disease is undoubtedly an ancient problem, far in the period of Babylon & later on Roman era. Ruth in I lot Springs was well known to be curative. In the early Islamic era many Muslims physicians described & treated scabies by painting the whole body with yellow sulphur alone or mixed with oil (2).

Several centuries later in 1687 Bonomo & Cestoni in Italy described the parasite & marked scabies as the first infective agent to be related to a specific disease in man (3), but unfortunately it took two centuries before this was generally accepted & definitely proven a mite *Sarcoptes scabiei* causes human mange by Raspail & his Corsican student Renacci in 1834 (4).

Transmission of scabies usually occurs by direct close physical contact including sexual contact, with an infested person’s lesion. This contact must be enough to allow the acarus to pass from one host to another & to establish itself by burrowing in the skin (5).

However the surrounding area can be a source of living mites as well, these include bedding, clothes, underwear, blankets, dusts & rugs (3).

Human scabies is caused by the mile *Sarcoptes scabiei var hominis* an arthropod of the Class Arachnida, which differ from the insects in the absence of the wings. Order Acarina that includes ticks & mites where the head, thorax & abdomen are fused in an unsegmented body (4).

Suborder Astigmata which includes the parasitic mite (*Sarcoptes scabiei*) & the free living mites (*Dermatophagoides farinae* & *Dermatophagoides pteronyssinus*) which are associated with house dust allergies (3).

Family Sarcoptidae, Genoits sarcptes, which have more than 30 species & 15 varieties infesting human & other mammals separately & named from the host in which they live as *Sarcoptes scabiei var hominis*, bovis, equi, suis & cams for human, cattle, horse, swine, & dog respectively (7).

When the mites enter the skin, it is quite accepted that, they evoke an immune response to scabies infestation which may be humoral &/ or cellular mediated immunity (8,9) however non of these reactions have been shown to eliminate all mites from the skin surface, though locally they may prevent the epidemic multiplication of scabies organisms on the skin (10). The pathogenesis of scabies is mainly due to & starts after sensitization of the host to the mite & its products which provokes an allergic response, responsible for a wide spectrum of clinical & histological manifestations that ranges from subjective.
symptoms with few parasites & hardly any visible lesions to a severe hyperkeratotic form of the disease, where several thousands of mites are digging into the skin (11). Moreover at least 6 antigens from Sarcoptes scabiei body & 3 antigens from its fecal pellets cross react with house dust mites body & feces respectively (12,13) since they are related phylogenetically (12,14,15). These mites, Dermatophagoides farinae & Dermatophagoides pteronyssinus, are important allergens responsible for extrinsic asthma, conjunctivitis & allergic rhinitis (16). This cross reactivity may explain the association of severe form of scabies infestation with atopic diseases & the persistence of symptoms in some cases even after proper treatment of the disease (17).

The itching & scratching in scabies is mainly due to immediate type hypersensitivity reaction (14), through the release of histamine & other cellular products from the skin, onward, the clinical manifestations of scabies are seen & felt after an induction phase from 2-8 weeks (11). This is suggested by:

-- An elevation of total serum IgE antibody among patients with scabies & fall after successful treatment (14,18). Moreover there is a statistical correlation between IgE level & the degree of lesion extension (16). Those with severe form of Norwegian scabies have an extreme elevation of serum IgE level (19), simulating those with atopic diseases (20).

-- Antigen specific IgE in the scrum was elevated in scabetic patients as determinate by radio-allergio-sorbent test (RAST), using crude scabies antigen, that cross react with antigen from the common house dust mite (12,20,21).

-- Intracutaneous skin test with extracts of adult mite causes a wheal reactions in the individuals who have had scabies but not normal volunteers (20), & this can be passively transferred by a factor in the serum (Praussnylz-Kustner) (19).

-- Scabietic patients serum could cause histamine release using basophil degranulation test (22).

IgE may act to protect the host against parasites through mediators released by triggered mast cells, that affect the parasite directly or via increasing vascular permeability together with the releasing of eosinophil chemotactic factor. All these could lead to accumulating the necessary antibodies & cells to attack the parasite, in addition IgH immune complexes can induce macrophage mediated cytotoxicity to parasites its products (23,24,25).

Subjects:
This study was conducted in the , Baghdad teaching hospital /Department of Dermatology & in Al-Rasheed Military Hospital, in a period between August 2002, & February 2003, gathering a total sum of 60 individuals of both sexes, different age groups comprising all the patient group & the control group. From each individual a full history & clinical assessment was done. All the people in the study & their family were free from history of allergic diseases & Parasitic diseases.

The patient study grope (30 individual) were diagnosed clinically as having scabies by a history of contact with a satieties patient & finding the burrows in their skin. All the patients had the disease for the first time, some of the patients developed the lesions recently, while other patients had the disease for more than one month without treatment. AH skin lesions were clinically free from secondary bacterial Infection.

The control groups (30 individual) apparently normal persons were age & sex matched ill the patient group.

From each individual in the study 5 ml of blood was aspirated, few drops of blood were put on clean slides for blood film, the rest of the aspirated blood were allowed to clot, then centrifuged & the separated serum was divided into A aliquots & stored in a deep freeze at (-20 C°) till used. Thawing of each frozen serum was allowed only once at the time of its used in the test.

Method:
Ultra Allergy Enzyme Immunoassay specific IgE-Using Miniprep 75/2 ( Biomaghereb) This procedure uses an enzyme immunoassay (EIA) method for the semi-quantitative determination of allergen-specific IgE concentration in human serum.

The serum is incubated in a first step with an allergen covalently bound to the solid support of a paper disc, if the sample contains antibody directed against this allergen it will be bound to the solid phase.

Washing the support with the washing solution removes all the antibodies having not reacted with the disc. Incubation of the washed paper disc with enzyme labeled antiserum human IgE allows the antiserum to fix the allergen-specific IgE antibodies to be bound to the solid support.

After a second washing (ie disc, a chromogenic solution is added & incubated. This results in the development of a yellow color which is measured spectrophotometrically at 405 nm, after stopping the reaction & measuring in a colorimeter, the level of allergen-specific IgE is calculated.

Compare the absorbances of each control & patient samples with the absorbances for the references sera IXC,B.A.I 1. Assign the class value to be sample as follows:

<table>
<thead>
<tr>
<th>Class</th>
<th>Count rate</th>
<th>Specific IgE conc.</th>
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A semi-quantitative assessment can be done alternatively by assigning I.A,B,C&D the values of 52.5,1 7.5,3,5,0,7 & 0.35EU/ml respectively.

On semilog paper (included in (he kid) plot (JD average for each tandard(Y axis) versus concentration (X axis) of references sera. Read 'allies of controls or patients directly from the standard curve. Value elow 0.35 EU/ml are nor, detectable. Values increases with increasing concentration of allergenspecific IgE.

Results:
Total of 30 scabietic patients were studied their ages ranged between 12-47 years with a mean age ±S.D. 21.45.19.18 years. The predominance of 20(66.6%) male patients over 10(33.3%) female patients made the male to female ratio2:1. The distribution of cases by their age groups is shown in Fig. 1, which shows the maximum number of patients were in their second decade of life (10-19 years).

Serum specific IgE against Sarcoptes scabiei var homins was elevated in all patients (100%) above the upper normal value of the control group (3.5 EU/ml) & there was a high statistical significance P<0.001 between the mean value of serum specific IgE in patient group when compared with that of the control group. Moreover this specific IgE significantly cross react with specific IgF against house dust mites (Tab.1).

Discussion:
Scabies is undoubtedly an ancient disease, & in the present work some of the clinic-immunological characteristics of the disease were evaluated; Our study showed that scabies still affecting most age groups but it tends to have a higher incidence in younger people than elderly & the prevalence was decreased with increasing age groups, this drop in incidence may be due to acquired resistance , similar results were reported by some other studies Buxton (1941) (1), Beaver et al (1984)(5) , while different from another study by Christopherson (1078 )who reported that scabies was common in all age groups“6. This study also noticed that scabies was more frequent at the second decade of age because most of the patients were at 10-1(> years of age, while previous study by Al-Rawi (1900) reported that scabies was more predominant in children below 10 years(39.5%), & only (27.5%) were at second decade of life (1> , the reason for this difference may be due to that , the previous study did not included the military hospitals in their work, while in this work (50%) of the patients included in the test were military persons, & for the same reason in this study there was a male predominance (2:1), which is agree with !pstein( 1955 f . Srlvastara et al ('950)'6) , while Al-Rawi (1990) reported that the disease was equal between males & females’.

Most of the female patients were students , while most of the males were military persons which is in agree with what was reported by Rpstein(1955){27}.

Specific serum IgF was elevated in scabies patients which is coincided with what was reported previously itching & skin lesions in scabies max be mainly due to immune hypersensitivity reactions through the release of histamine & other cellular mediators from triggered mast cells which may act to help the host against parasites either directly or via increasing vascular permeability to enhance the accumulation of the necessary antibodies & cells that attack the
This study showed that there was cross reaction between specific IgE against scabies & anyogens present in house dust mites, since they are related phylogenetically. These mites, Dermatophagoides farinae & Dermatophagoides pteronyssinus, are important allergens responsible for extrinsic asthma, conjunctivitis & allergic rhinitis. This cross reactivity may explain the association of severe form of scabies infestation with atopic diseases & the persistence of symptoms in some cases even after proper treatment of the disease.

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15. Stenius B & Wide L. Reaginic antibody (IgF) skin & provocation test to Dermatophagoides fariniae & house dust mite in respiratory allergy Lancet (1969); ii: 455.