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Dust Allergy: Cause & Concern

Indian Perspective

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Respectfully dedicated to the sweet memory
of my beloved father Sri Gopinath Saha
who inspired me in every step of my career

Preface

Dust allergy is a fairly common problem among individuals sensitive to inhalation of dust leading to various allergic manifestations such as allergic rhinitis, hay fever, atopic eczema, urticaria and in severe cases bronchial asthma. It is a fact that a threefold increase in the prevalence of different allergic disorders has been noticed in recent times throughout the globe and India is no exception. Such an exponential increase is mostly attributed to the rapid industrialization, unplanned urbanization, metamorphic change in lifestyle pattern and food habits, increase in air pollution, etc. Irrespective of the standard of living and the health consciousness, people of all cross sections of the society in different countries come in contact with dust of varying quality and quantity. As a consequence, sensitive people are susceptible to the detrimental effects of dust, most commonly in the form of dust allergy. Perusal of literature show that the information available in this respect is mostly from Western countries. However, data from Third-World countries like India are still fragmentary, and in comparison with documents for other allergic diseases, systematic compilation on dust allergy is still inadequate. In such a backdrop, the present attempt is a timely intervention. World is facing an unprecedented increase in the prevalence of allergic diseases with greater complexity and severity day by day. It is a public health problem, and extensive efforts should be made towards its understanding, particularly on its causes and consequences, diagnosis, prevention and optimal treatment. Therefore, an integrated approach is needed combining different branches of sciences such as entomology, immunology, molecular biology, epidemiology, pharmacology, genomics and computational biology for better understanding of disease pathogenesis, proper diagnosis and treatment of allergic diseases.

The whole content of the present compilation is divided into twelve distinct chapters, and each of them is rich in information based on supportive data and elaborately presented with necessary illustrations and proper referencing. An exhaustive review of the existing literature on different aspects of dust allergy in the field of researches in India and abroad with special reference to house dust mites has been provided. The present monograph also documents an appraisal

of the responsible allergens in our surroundings, house dust mites in particular, their diversity, distribution pattern, seasonality, life history traits and their clinical relevance with the disease pathogenesis and overall their allergenic potentiality. Insights on the hereditary links with dust allergy are extremely useful for in-depth understanding of the causes and concerns of dust allergy problem. The extent of topics covered in the book appears to fill up the vacant niche between the disease and its proper management. Overall, the extent of discussions on highly focused topics and the sequential appearance of the topics are perfectly blended to attract students of different disciplines apart from the general public to be knowledgeable about dust allergy. The length of the book and the matter discussed are fully matched to qualify as a document for easy reading and understanding the subject matter. A prior knowledge about the causes and symptoms of a disease facilitates smooth treatment process as well as formulation of prevention strategies. Thus, the present compilation appears to be a pioneer effort and fair approach to fulfil the requirements of the target audience through portraying different dimensions of dust allergy for the general readers as well as academicians interested in the field of allergy and immunology. Besides, there are ample scopes and potential to generate further information on dust allergy-related problems, particularly in developing countries with exceptionally large population at the same time with high parasitic load, where it has recently been came to the surface due to some anthropogenic activities. Thus, it provides an excellent opportunity for future researchers in this field of work. I am confident that this concise yet informative compilation would certainly make this book acceptable for general as well as medical students, researchers, health professionals and experts in the field of allergic research.

Kolkata, India Goutam Kumar Saha

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About the Author

Goutam Kumar Saha passed B.Sc. (Honours in Zoology) from Burdwan University securing first class first position in the year 1981. In M.Sc. examination too, he stood first among the successful candidates in Zoology and was awarded prestigious university gold medal. He was awarded the Ph.D. degree on dust mite allergy in 1991 from Calcutta School of Tropical Medicine. He has been actively engaged in research on allergy and immunology during the last 30 years and published 163 research articles in different journals of international repute and has authored twelve books/technical monographs to his credit. He has actively participated in the scientific proceedings of the World Allergy Congress for four times (2003, 2005, 2007 and 2015) and visited several countries such as Canada, Germany, France, Australia, the Netherlands, Belgium, Italy, China, Thailand, South Korea and Singapore in connection with his research works. He has successfully conducted a good number of research projects sponsored by national and international funding agencies. Professor Saha started his professional carrier as a lecturer in Zoology at the Post-Graduate Department of Zoology, Darjeeling Government College, and is currently working as a professor of Zoology, University of Calcutta. In recognition of his outstanding contribution in the field of allergy and applied immunology, he was awarded honorary fellowship of the Indian College of Allergy and Applied Immunology and The Zoological Society, Kolkata. Besides his excellence in the academic field, he is a good sportsman and represented the university as blue and participated in several national games.

Chapter 1
Allergy—Definition

1.1 Self and Non-self

It is a unique property and extraordinary ability of the immune system to distinguish between the self (product of the individuals own) and non-self (different origin or foreign molecules) macromolecules with the help of lymphocytes and antibody molecules. When a foreign molecule enters into the body system, it is designated as antigen and stimulates the immune system towards that molecule or a part of it called epitope and ultimately elicit the production of specific antibody.

Upon entering the body, the antigen stimulates the naive B lymphocytes and transformed them into plasma cells (Effector B cells), which are capable of synthesizing specific antibody. On the other hand, antigenic stimulation of naive T lymphocytes transformed them into activated T cells, which secrete a series of biologically active proteins—called cytokines. In case of allergic mechanism, T helper cells and their signal protein cytokines, play an important role in both IgE synthesis and activating inflammatory cells in the IgE mediated allergic reaction. Contrary to this, T cytotoxic cells kill intruding foreign particles by direct contact or through release of cytokines.

1.2 An Optimum Balance Between Th1 and Th2 Cells

It is a fact that T cells regulate the immune response and two distinct classes of CD4 T helper cells namely, Th1 and Th2 cells have been identified on the basis of their cytokine release pattern. Th1 cells are responsible for the release of IL 2, interferon gamma (IFNg) and supposed to play a vital role in inflammatory delayed-type hypersensitivity. On the other hand, Th2 cells release IL 4, IL 5, IL 6, IL 9, IL 13 etc. and mediates humoral responses. Both Th1 and Th2 originate from the naive T cells and differentiates either to Th1 or Th2 as and when comes in initial contact with the antigen. In case of normal individuals, there is a delicate balance between Th1 and Th2 immune responses and if there is a tilting in Th2/Th1

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balance towards Th2 type, allergy results. Whereas, if the shifting is towards Th1 type—it may lead to autoimmune disorders. It is now well documented that allergic individuals have more Th2 cells and high levels of IL 4, IL 5, IL 6, IL 9, IL 13 in their blood. IL 4 and IL 13 induce specific B cells to undergo isotype switch and produce IgE—which is the key molecule in allergic response.

1.3 The Meaning of Allergy

Literally, the word allergy means intolerance or hypersensitivity to certain external substances popularly known as allergens. The term "allergy" was coined by Von Pirquet in the year 1906 from the greek word 'allos' meaning different or changed and 'ergos' meaning work or action, hence "other action" or "altered reaction". He defined it as a changed responsiveness in individuals, who have previously been exposed to an antigen (allergen). Subsequent exposure to the said antigen resulted in an altered responsiveness and the term allergy was proposed to explain this unexpected changed reactivity. The majority, however, prefer to describe allergy as an immunologically mediated state of hypersensitivity that is damaging to the body. From the immunological point of view, Middleton et al. in the year 1978 defined it as 'an antigen-antibody related abnormal reaction in some parts of the body of sensitive individuals to some external substances—allergens'. Allergy can develop at any age, possibly even inside the mother's womb, generally occur during childhood, however, symptoms may appear for the first time in adulthood too. The parts of the body that are more prone to allergic reactions include the eyes, nose, lungs, skin and stomach. In case of allergy, the common questions are—why some are sensitive to certain allergens while most are not? Why do allergic individuals produce more IgE antibody than that of the non-allergic ones? The major cause is obviously the hereditary factors. However, genetic factors alone are unlikely to explain the unusual high prevalence of allergic diseases as genetic shift in the population cannot occur in such a short duration. Changes in lifestyle, living conditions, food habits and environmental factors are implicated in the increased prevalence of allergic diseases. Other important contributing factors are smoking, increasing pollution load, infection and hormonal interference. Allergens may be inhaled, ingested, simply applied to the skin or injected as medication or through insect stinging.

To aid in classifying the phenomenon of hypersensitivity, Coca (1922) coined a term "Atopy" to describe a form of clinical allergy which is of inherited nature. Patients of atopic constitution have hereditary tendency to produce reaginic antibodies (IgE) in greater amount after natural exposure to substances (atopens), which are usually harmless to normal persons. Atopic patients have an increased risk of developing asthma, urticaria, atopic eczema and rhino-conjunctivitis.

1.4 A Note on Historical Perspective

Von Pirquet in the year 1906 noted that an initial doze of injection, often developed attacks of urticaria and asthma and he designated the phenomenon as allergy or a changed state. Later, in the year 1910, Meltzer first pointed out that asthma is an allergic disease. Since then, the knowledge of allergy advanced at a rapid pace. During sixties, several workers, throughout the globe made substantial contributions on the causes and mechanism of different allergic diseases and established the subject "Allergology" as separate branch of medicine. A major breakthrough took place in the field of allergy with the identification of a new class of immunoglobulin protein—called IgE, separately by Professor Johansson' group from Sweden and Dr. Isizaka' group from Japan in the year 1966. Subsequently, development of method for estimation and characterization of this new antibody opened a new vista in the field of diagnosis and treatment of allergic disorders.

Chapter 2
Allergy—Fact File

2.1 Global Burden of Allergy—An Estimate

In most of the developed countries, allergy has become one of the most common and widely effecting disorders and nowadays is considered to be a common clinical problem. An increasing trend in the occurrence of allergies is observed as societies become more prosperous and urbanized. At present, about 30 % of the adult human population and 40 % of the children suffers from either of the different types of this malady, like allergic rhinitis, urticaria, eczema, food and contact allergies and asthma during their normal life span. In the United States of America, allergy is considered to be the fifth leading chronic disease and when taken together, an estimated fifty million Americans suffer from all types of allergies. It is estimated that allergic rhinitis, contact dermatitis and asthma are among the fifteen most common diseases diagnosed by the physicians and health experts. Moreover, thirty six million people per year in USA suffer from seasonal allergy that leads to nearly loss of four million days at work and school with a national burden of $18 billion. Recent survey suggests that allergy is diagnosed almost one in every four individuals and among all those manifestations, allergic rhinitis is the most prevalent one (30 %), particularly among young adults.

The risk of allergic sensitization and its development differs with age, with the young children being at the maximum risk. Sensitization to allergen plays a crucial role in the development of atopic disorders. Available information have shown that IgE levels are highest during childhood and fall rapidly between the ages of 10 and 30 years. As diagnosis of IgE-mediated disorders is challenging, physicians rely on a thorough interview on clinical symptoms, but allergy symptoms can arise from many different organ systems and vary in severity, and symptoms can coincide with several other diseases. Children and young adults are most susceptible to the hay fever and the occurrence of asthma is maximum among the children below ten years. The peak age for the onset of hay fever is in adolescence, and about 63 % of students are reported to have suffered from hay fever symptoms. In general, the boys are more prone to develop allergy than the girls, whereas, the young adult females are more likely to be affected by asthma. Sex differences tend

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to decrease during the process of adulthood. The chance of having an allergy varies radically based on race and ethnicity. It was observed that ethnicity plays an important role in some allergies such as asthma; however it was not possible to separate racial factors from environmental influences and changes that occur due to migration. Interestingly, it has been suggested that different genetic loci are responsible for asthma, specifically, among people of Caucasian, Hispanic, Asian, and African origins. The death rate due to asthma among Afro-Americans is nearly 4–6 times higher than among Native Americans, and Hispanic populations especially Puerto Ricans, have much higher asthma prevalence and greater morbidity and mortality.

In general, asthma is more prevalent in ill developed urban settlements than their rural counterparts, specially among industrialized communities, which may be attributed to the poor air quality, increased air pollution, excessive automobile exhausts, change in life style and food habits and intolerable psychological stress, which is more pronounced among urban inhabitants. Urban air pollution has been one of the significant environmental and extrinsic etiologic agents responsible for allergic diseases. Ozone, nitrogen dioxide, as well as particulate matter generated by automobile traffic and industry contribute towards air pollution and subsequently asthma and other allergic disorders. Empirical data on public health and hygiene have shown that the industrialized and more urbanized countries have a higher incidence of allergic diseases, while the occurrence of such disease are quite stable in low or middle income developing countries (ISAAC 1998). In USA, Canada, UK, Ireland, New Zealand and Australia, the incidence of diseases related to nasobronchial allergies between children of age groups 13–14 years is highest in the world and accounts to nearly 32 % of allergic diseases(Gangal and Chowgule 2009).

Strong epidemiological evidence supports a relationship between air pollution and the exacerbation of asthma and other allergic diseases around the world, including under developed and developing counties. In the recent past, increase in air pollution or decrease in ambient air quality, metamorphic changes in lifestyle, use of fast foods and changes in dietary habit as a result of strong western influence, lack of proper treatment and negligence at the initial stages of complaints have led to an alarming rise in the incidence of allergic diseases round the world. Allergic asthma is a heterogeneous disease with interplay between genetic and environment factors, asthma is chronic inflammatory disorder causes recurrent episodes of wheezing, breathlessness, chest tightness and coughing particularly at night and early in morning. There has been a substantial increase in the incidence and prevalence of nasobronchial allergic disorders among all age, sex and racial groups, affecting 12–20 % of the population worldwide (Smith 1983). The situation is worst in most of the less prosperous and developing countries with weak economic condition. There are many factors such as living habits, occupation, humidity, climate, home standards and ventilation which have been attributed to such an increase.

2.2 Socio Economic Impact of Allergy

Allergic diseases are very common multi-factorial disease complex and have a noteworthy impact on the society as well as economy. Allergies impart a negative impact on health and well being as well as education and career achievement and thus had emerged as a major health and economic burden. They adversely affect the quality of life of the patient including their parents as well as other family members thereby leading to weaker school performance and lower productivity at work. In Western Europe the direct and indirect annual estimated costs is nearly 45 billion Euros. In USA, an estimated 40 million i.e. 25 % of the normal population have allergic diseases, and the annual medical cost associated only with allergic rhinitis is estimated to be nearly $53.4 billion, excluding 20 million lost school days and 3.5 million lost workdays each year, that amounts to $154 million direct wages loss only due to seasonal nasal allergies. As a matter of fact, there are up to 20 million days of decreased productivity from either due to symptoms or due to side effects of medication. For adults, allergy is the fifth leading chronic disease and a major cause of absenteeism, linked to depression and anxiety, affecting cognition and learning (Fineman 2002).

2.3 Allergy Is More Common in Urban Landscape

Evidences round the globe show that the allergic diseases are more common in more developed and civilized countries as compared to agricultural based or traditional countries. Within the developed countries, the incidence is higher among urban population, particularly among those living in the posh areas of the cities. It is estimated that in urban areas, nearly one in three children are allergic and 30–50 % of them develop asthma. It was also found that a number of different factors, like the poor air quality in urban settings due to increased environmental pollution, greater chances of indoor allergen exposure, dietary changes, housing architecture, water supply and highly stressed lifestyles had significantly contributed towards the higher incidence of allergic diseases in the urban areas (Ellwood et al. 2001; Pearce et al. 2007; Wood and Gibson 2009). Apart from these, several house hold management practices such as cleaning, dusting, keeping the pet indoor, smoking has also added towards the development of the disease. Several case studies related to public health and hygiene have demonstrated that, increased levels of vehicle emissions and westernized lifestyle are significantly associated with the increased frequency of respiratory allergic manifestations among people living urban areas compared with their rural counterparts. Evidences suggest that air pollution associated with increased level of ozone, nitrogen dioxide and sulphur dioxide in the atmosphere as a result of excessive diesel exhaust, is the main culprit behind the increased occurrence of the allergic diseases (D'Amato et al. 2005). Van Zijverden et al. (2000) demonstrated that a positive correlation exists between

these pollutants and increased allergen specific IgE production by stimulating T helper 2 cell (Th2)-mediated immune responses to common antigens and alter normal Th1/Th2 ratio towards a predominant Th2. Although it is known that Th2-mediated responses are involved in respiratory allergy to common allergens, carbonaceous particles like diesel exhaust particles and carbon black particles could play a stimulatory role in this process. It was noted that diesel exhaust particles can even promote primary allergen sensitization towards a neoallergen (Diaz-Sanchez et al. 1999) and also appear to twist cytokine release in favour of Th2 pattern. In fact, diesel exhaust particles act as immune-modulating agent and perform the function of an adjuvant during the sensitization phase of allergen response as well as aggravate the symptoms in sensitized individuals (Peden 2005).

2.4 Allergy: A Lifestyle Disorder

There are a number of evidences which suggest that many aspects of lifestyle and the environment may lead to the development of allergic diseases. Population-based surveys in the developed world indicate that the more affluent sections of the community have the highest prevalence of allergic sensitization and associated diseases. According to the hygiene hypothesis, as proposed by Strachan (1989), changes in lifestyle among industrialized countries have led to a decrease of the infectious burden and are associated with the rise of allergic and Autoimmune diseases. Allergic diseases are more common in western countries than in developing countries and the prevalence of allergic diseases has been increased at a rapid pace over the last few decades in the developing world too. In fact, higher prevalence of these diseases within western communities suggests that allergy is an ‘illness of wealth’. However, it has started to rise in other countries which are becoming more developed. Even in the developing countries like India, the prosperous urban population, following western lifestyle appears to be more susceptible to allergic response, leading to increased incidence of allergic disorders. It is assumed that this rapid increase in its prevalence in genetically stable populations must be attributed to some other factors, possibly environmental or lifestyle factors. Evidences support that the underlying causes are different aspects of modern living, including more sterile surroundings, changing diet, air pollution, increased consumption of processed foods, possibly even obesity and sedentary lifestyles. The habit of smoking, snuffing, drinking, and tobacco chewing along with the use of unnecessary drugs can also contribute to this unwelcome situation in a more intensified manner. The formaldehyde present in rugs, upholstered furniture, wall paper, draperies, paints initiates coughing, sneezing, wheezing, sore on nasal passage, flu like symptoms, aggravates asthma, irritates throat and causes watery eyes, are being used indiscriminately in our day to day life. Increased use of foam mattress instead of conventional cotton mattress favours the growth and multiplication of house dust mites, which ultimately increases the chance and duration of exposure to that offensive indoor allergen.

2.5 Allergy Is a Big Burden in Western Communities—Controversies and Realities

In western lifestyle practices, clean environment, changes in dietary habits, indiscriminate use of antimicrobials, widespread vaccinations and indoor air quality problems lead to a pro-allergic condition. In other words, individuals living in too sterile environment are not exposed to enough pathogens to keep the immune system busy and active. As suggested by hygiene hypothesis, allergic diseases are initiated as a result of inappropriate immunological responses to apparently harmless antigens driven by a Th2 mediated immune response. As proposed by hygiene hypothesis, at first, insufficient stimulation of the Th1 arm of the immune system lead to an overactive Th2 arm, which in turn led to allergic response. Beside, the endotoxin (lipopolysaccharide) and allergen often occur together in indoor air, which may add to the inflammation process. In general, immunological and autoimmune diseases are less common in the developing world than the industrialized countries. However, an increasing trend in the incidence of immunological disorders have been noticed from a number of third world countries, as the country grows more affluent and presumably cleaner. Empirical evidences suggest that the indiscriminate use of antibacterial cleaning products augment the situation and as a result reduced exposure to microorganisms due to this sort of lifestyle changes, in developed countries is associated with increased prevalence of allergic disorders. At the same time infections in early childhood may protect against allergic sensitization (Strachan et al. 1996), particularly, exposure to food and orofaecal pathogens like Hepatitis A, *Toxoplasma gondii* and *Helicobacter pylori* reduce the risk of atopy (Chen and Blaser 2008). However, some gastrointestinal bacteria such as *Campylobacter jejuni*, *Yersinia* and *Clostridium difficile* were associated with higher prevalence of atopy. Although, bacterial, fungal and helminth infections under certain condition have a protective role against allergic diseases, the role of viruses is still controversial. It has been hypothesized that exposure to viral and bacterial infections and some vaccines in early life can enhance type 1 (Th1) immunity and the production of cytokines (IFN-g) that inhibit directly the allergy promoting type 2 (Th2) immune responses (Barreto et al. 2006).

Thus it may be presumed that low socio-economic condition, high temperature, poor standard of living including housing conditions, lack of proper attention towards health and hygiene in most of the developing countries, predispose the inhabitants to infections and possibly higher prevalence of helminth infestations protect them from allergic sensitization.

2.6 Allergy: A Stress Related Phenomenon

Empirical evidences clearly indicate that the allergic responses are very much stress dependant and psychological stressors have direct and substantial adverse effect on existing allergic patients (Kilpelainen et al. 2002; Montoro et al. 2009).

To accommodate themselves with the uneven rat-race, human beings are generating more and more stress day by day. A close relation between high incidence of allergic disorders and Depressive symptoms as well as anxiety related disorders have also been documented by Lietzén et al. (2011). In fact, stress and depression enhance humoral immunity instead of cell-mediated immunity, favouring more IgE production and shift from Th1 to Th2, thus initiate allergic response. Sugerman et al. (1982) reported that psychiatric patients, particularly depressed one, showed higher IgE levels to specific allergen, and there are reports of substantial improvement of allergic skin disorders due to administration of anxiety medications. Recent view suggests that mood and psychological stressors can modulate allergic response, like atopic dermatitis (Novak et al. 2003).

The possible effect of sedentary lifestyle to lung function and daily activity have received little attention until recently (Platts-Mills et al. 1997). Spending excessive time in front of video screens, televisions and computers by most of the children, decreased in physical activity, which could influence the lung function. Thus decline in physical activity must be considered as a possible factor contributing to the severity of asthma (Platts-Mills et al. 2000).

Chapter 3
Possible Allergens in Our Surroundings

Antigen that initiates an IgE mediated allergic reactions is called allergen. An allergen molecule consists of a number of antigenic determinants called epitopes which are pieces of small polypeptide in the large protein molecule. Allergens are high molecular weight proteins or polysaccharides which when introduced into the body system may trigger the production of allergen specific antibodies. Different allergens may have some similarities in their amino acid sequences, which may sometimes lead to cross-reactivity.

Among the possible allergens in our surroundings, the most importants are pollens, fungi, mould, spores, animal dander, fur, feathers, microorganisms etc. Certain food stuff like, brinjal, egg, milk, peanut, some cosmetics, perfumes, some drugs and chemicals like penicillin, sulphur containing drugs may also initiate allergic reaction. Besides, allergy to bee sting, contact dermatitis in contact with some metals like nickel, chromium, copper, etc. exposures to certain industrial fumes, asbestos, etc. are not uncommon.

3.1 Role of Pollens and Moulds

Pollen grains are relatively large particles of 20–30 μm in size, usually trapped in upper airway passage resulting allergic rhinitis or asthma. They are released from plants during pollination season and are transferred from one plant to others either through insects or mechanically by wind. Pollens of different deciduous plants like birch, olive, Japanese cedar, etc. are of great concern. Additionally, different wild and cultivated grasses like timothy, rye grass, orchid grasses may produce allergenic pollens. Weeds, specially ragweed pollens are important source of allergy mostly during late summer and early autumn.

Moulds, also known as microfungi are microscopic organisms usually produce enormous number of small spores of 2–5 μm size. They usually affect lower airway passage resulting asthma. Most important allergen producing moulds include *Cladosporium*, *Alternaria*, *Aspergillus*, *Penicillium*, *Mucor*, etc. In warm, humid

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climate, moulds grow rapidly all year round and mostly during late summer. Inhalation of very small amount of allergenic mould spores may initiate IgE mediated reaction and results asthma. On the other hand, exposure to massive amount of saprophytic moulds can evoke both IgE and IgG mediated response leading to broncho-pulmonary aspergillosis. In some cases, they may lead to allergic alveolitis of extrinsic origin.

3.2 Allergens of Animal Origin—Insects, Mites and Pets

Allergic response to cockroaches is very common among urban dwellers, specially among people of low socio-economic status. A large number of urban asthmatic patients show sensitivity towards cockroaches (Tandon et al. 1990). Allergic response to non-biting midges (chironomidae) was also suspected long before. The first indication that adult chironomid could be immunologically significant, came from recognition of a relationship between seasonal midge swarms and allergy among Nilotic people of Sudan (Cranston et al. 1981). Subsequently, the chironomid induced allergy was also reported from Egypt (Cranston et al. 1983), USA (Kagan et al. 1984), Sweden (Eriksson et al. 1985), Japan (Sasa 1985) and India (Nandi et al. 2014). Japanese group reported a case of bronchial asthma induced by direct inhalation of small midge, *Tanytarsus oyamai*. Later, Ito et al. (1986) reported that about 40 % of asthmatics in Japan are sensitive to chironomids and demonstrated a high titre of specific IgE antibody towards chironomid allergen. In recent years, the chironomid induce hypersensitivity has become a serious health problem throughout the world (Cranston et al. 1983; Tee et al. 1985; Cranston 1988). It has been estimated that the incidence of chironomid hypersensitivity is next to that of house dust mite. It is now established that haemoglobin of the chironomid larvae actually constitutes the potent allergen for human (Cranston et al. 1983). As the metamorphosis progress, haemoglobin undergoes enzymatic degradation reaching minimal in the adult. According to Baur et al. (1986), both larvae and adults contain similar allergenic determinants and therefore, it would be pertinent to consider both larvae and adult midge as source of allergic etiology.

It is now well documented that mites belonging to the genus *Dermatophagoides* under the family Pyroglyphidae which are present in the house dust are considered to be the most potent allergen leading to various allergic manifestations like allergic rhinitis, hay fever, urticaria and in extreme cases bronchial asthma. A large number of storage mites such as *Glycyphagus* sp., *Tyrophagus* sp., *Acarus siro*, *Suidasia* sp. etc. infesting stored hay and grains are frequent causes of allergy among persons handling stored product and farmers.

Allergy to pet animals like cat, dog, guinea-pig, hamsters are not uncommon. The common manifestations include rhino-conjunctivitis, allergic rhinitis and asthma. Cat allergens are mainly produced by the salivary gland, while major dog allergens are associated with saliva, epidermal cells and urine. Contrary to the common belief, hairs, as such are not allergenic. In case of mice, rats, guinea

pigs and hamsters the urine is the main source of allergen, often producing allergic conjunctivitis, rhinitis and asthma.

Allergy to bee and wasp sting is a common phenomenon and it is estimated that over two million Americans are allergic to insect sting. However, intensity of reaction varies from person to person. Usual symptoms include intense itching, hives, flushing of the skin, tingling, nausea and vomiting and even anaphylaxis leading to difficulty in breathing, swallowing, hoarseness, swelling of the tongue, dizziness and ultimately fainting. Prompt treatment is necessary to avoid casualty.

3.3 Food Allergy-Types and Consequences

Different common food stuffs like eggs, nuts, milk, peanuts, some fishes, shellfishes, tomatoes are considered to be the major cause of childhood allergy resulting nausea, vomiting, diarrhea, severe abdominal pain, itching, hives, eczema, asthma and even anaphylaxis. In case of egg, major allergenic determinants are in the egg white and such proteins are mostly heat resistant. As a matter of fact, both raw and cooked eggs are equally potent as allergen producer. A large number of fish species are inducer for IgE mediated response resulting angio-edema, urticaria, gastrointestinal symptoms, asthma including anaphylactic reaction. Some shell fishes, specially crustaceans are noted for their potentiality to cause violent allergic response and even anaphylactic shock. Several cereal products like wheat, rye, barley are occasionally involved in allergic manifestations, mostly gastrointestinal symptoms. In several western countries, allergy to peanuts and cashew nuts are very common and often leads to life threatening anaphylactic reaction. Certain dyes used in beverages, many preservatives mostly sulphites, food additives like tartrazine, some flavour enhancer or taste makers like monosodium glutamate specially used in Chinese preparations have been accused of causing urticaria, asthma and anaphylaxis.

3.4 Importance of Chemicals, Drugs and Medicines, Cosmetics, Latex etc.

Many drugs such as antibiotics like penicillin, cephalosporin and sulfa, some anti convulsants, insulin of both pork and beef sources frequently leads to allergic response. Immediately after administration of the allergic drugs, difficulty in breathing, increased heart rate and decreased blood pressure including anaphylactic shock can be noticed. Mild symptoms include atopic dermatitis, urticaria, digestive symptoms, asthma etc. Symptoms may appear within seconds or several days after drug intake and may disappear three to five days after drug withdrawal. Drug allergy is not simply a drug “side effect” rather it is an abnormal over reaction of the immune system.

Products like moisturizers, shampoos, deodorants, colognes, etc. may cause contact dermatitis among sensitive individuals. In fact, certain ingredients specially some fragrances and preservatives can act as antigen and trigger allergic reaction. Beauty parlor workers are at risk of developing skin allergy due to constant use of hair dyes, nail polish remover, etc. Pungent room freshener also acts as potent irritants. Symptoms from cosmetic allergy include swelling, redness of skin, itching, burning sensations, etc. The face, lips, eyes, ears and neck are commonly affected.

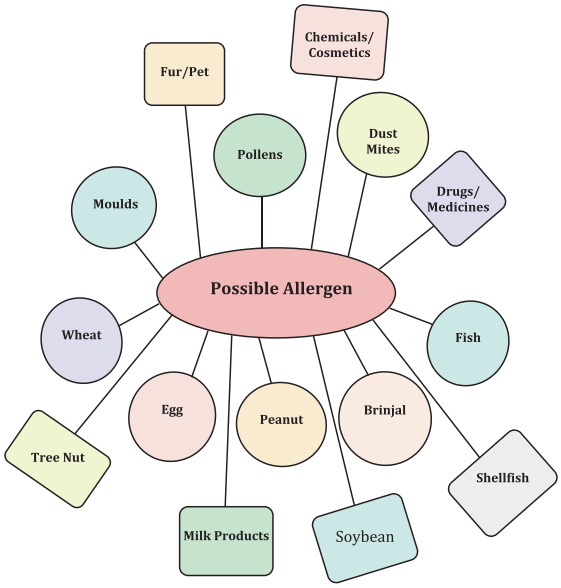
It is well documented that robber gloves may cause contact eczema belonging to type IV immunologic response. However, an IgE mediated immediate type allergic reaction to latex protein has been reported in the recent past (Bousquet et al. 2006). The phenomenon is very much evident as an occupational allergy mainly among health care personnel. Latex is a milky sap extracted from rubber tree and a variety of articles are produced from it like, gloves, medical heath care materials, catheters, tubes, balloons, condoms etc. Around 10–20 % of the health workers are allergic to latex.

3.5 Occupational Allergy at Working Places

It occurs as a result of constant exposure to different irritants originated and used as industrial dusts and chemical fumes in different working places. Over the last few decades, the prevalence of occupational allergy has been increased considerably due to introduction of highly reactive chemicals such as isocyanites and acid anhydrides in the manufacture of synthetic materials, specially plastic industry. Polyurethane foam makers, spray painters are the worst sufferers.

Laboratory workers handling animals often develop allergic conjunctivitis, rhinitis and asthma. Workers engaged in food processing industries, metal refineries and photographic works indiscriminately use certain chemicals like platinum salts and enzymes used in detergent industry initiate allergic manifestations including asthma. Besides, exposure to extraordinary load of organic dust by grain workers and pig-breeders, occupational exposure to moulds in bakery industry during manufacture of alcoholic drinks, bread, beverages, cheese, beer, wine, even during the preparation of enzymes, antibiotics and steroids in drug manufacture industries may result a variety of allergic disorders. Rhinitis and asthma are very common among millers and bakers—specially designated as Baker's asthma due to the sensitivity to wheat flour. Workers in the cotton industry, library workers often suffer from asthmatic complaints due to inhalation of cotton and paper dust respectively in their working places. Incidence of asthma is prevalent among workers exposed to nickel, asbestos, chromium, vanadium, etc. Moreover, a good number of inhalants like sulphur dioxide, nitrogen dioxide, ozone, ammonia and halogen gases may aggravate asthmatic symptoms.

Common allergens



Chapter 4
Mechanism of Allergic Reaction

4.1 Different Components Involved in Allergic Reaction—Immunoglobulin E (IgE) Antibody, Mast Cells, Eosinophils and Basophils

Immunoglobulin E is released from the lymphocytes and plasma cells in the airway passage, gastrointestinal tracts, and regional lymph nodes. It is present in a very low concentration and comprises less than 0.001 % of the circulating Immunoglobulin. IgE is a distinct class of serum protein consisting of four polypeptide chains, two identical heavy chains and two identical light chains, held together by disulfide bonds and non-covalent forces. The light chains, kappa or lambda are shared in common with other immunoglobulin classes but the heavy chains are characteristics of IgE—called epsilon chain "E" or it binds to target cells (i.e. tissue mast cells and circulating basophils) via the Fc region of the molecule, while the Fab fragments contain the antigen binding sites. IgE carries biological properties of skin sensitizing reaginic antibody and once IgE affixes itself to the surface of cells, subsequent binding of a specific allergen triggers a biochemical reaction leading to the release of biologically active substances from the granules of the mast cell.

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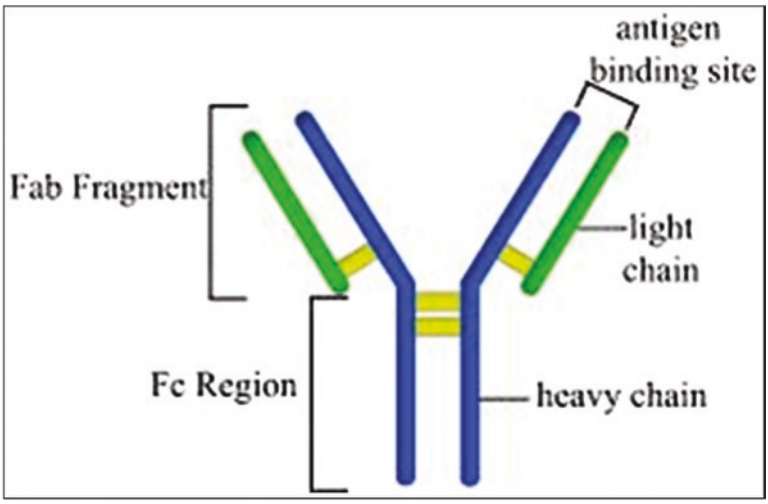


Structure of IgE antibody

Elevated serum IgE level is an important indicator of allergic diseases resulting from immediate type hypersensitivity. Serum IgE levels get elevated in patients with extrinsic asthma and other atopic diseases suggesting that the quantification of serum IgE may be significant in the diagnosis of allergic disorders. Thus, determination of total IgE in serum has been considered to be an important decisive factor for predicting future atopic manifestations and may be used as a screening tool specially in case of children of atopic parents (Kjellman 1977).

Mast cell and basophils are considered to be the initiating cells for IgE mediated allergic reactions with high affinity receptors for IgE. They are responsible for synthesis and release of histamine and other chemical mediators responsible for various allergic manifestations. Mast cells are kind of tissue cells and are predominantly present in the skin, airways and gastrointestinal tract and contains granules of histamine, leukotrienes and prostaglandins. While basophils acts as a 'circulating mast cell' and mediates systemic allergic reactions.

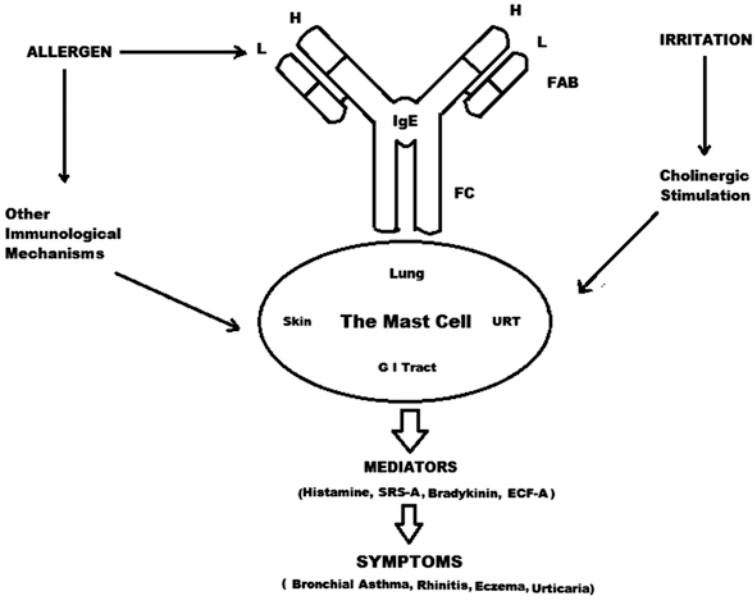
Eosinophil leucocyte is an important component in the process of allergic inflammation. It contains granules of eosinophil specific proteins of cytotoxic nature responsible for tissue damage. Thus damages the epithelial cells and as a result sensory nerves are more easily be exposed to irritants.

4.2 Basic Mechanism and Types

When an individual is immunologically primed, further contact with antigen leads to secondary boosting of the immune response. However, the reaction may be excessive leading to gross tissue damage (hypersensitivity), if the antigen is

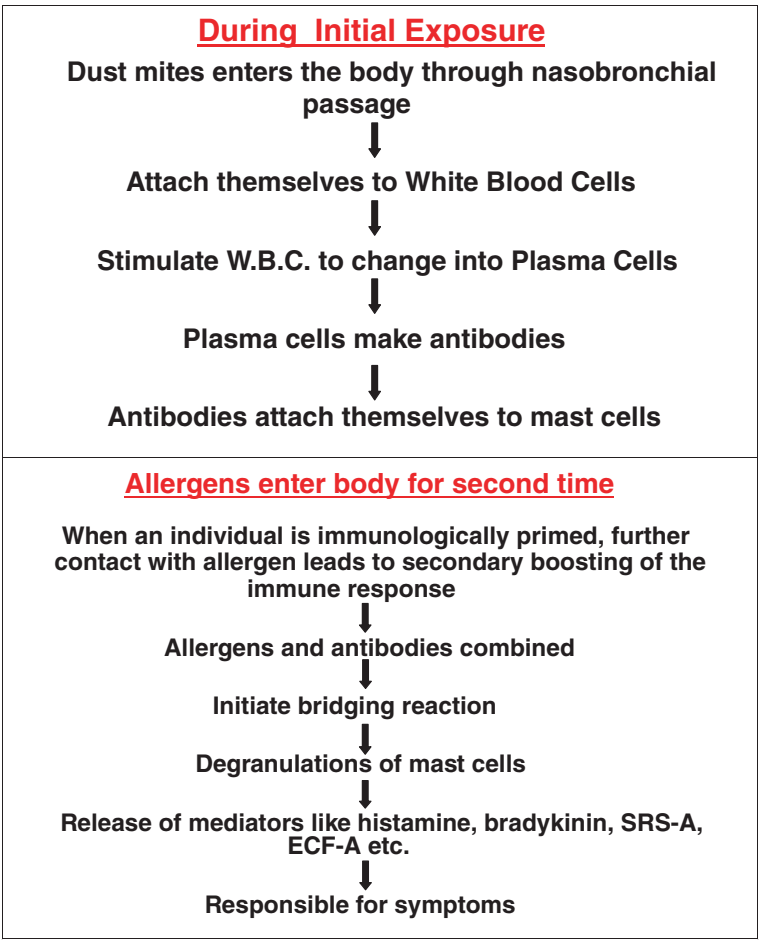
present in relatively large amounts or if the humoral and cellular immune state is at a higher level.

During initial exposure, allergens must be sufficient to activate the mast cells bound IgE molecules. Once the IgE molecules are sensitized, even a minute amount can let loose the cascade of chemical mediators, by bridging the gap between two IgE molecules and thus initiate the degranulation of the mast cells. The reactions between reagin and allergen are immunologically specific. Stimulation of mast cells by antigens release powerful chemical mediators that trigger a sequence of physiologic events, eliciting the symptoms of immediate hypersensitivity. These powerful mediators act on different organs resulting in various manifestations of allergic diseases. Among them, the lung is recognized as the most important and vulnerable target for allergic manifestations and when this reaction occurs in the lung, the resultant effect is asthma. The bronchi of subjects with asthma are hypersensitive to many external stimuli.



Schematic presentation of allergic mechanism

According to Cockcroft et al. (1977) the degree of hyper-responsiveness may increase in subjects with asthma after deliberate exposure to inhaled allergens. The resultant mediators then cause dilation of blood vessels and capillary permeability increases resulting in swelling. In addition, they also contract the bronchial smooth muscles, swells the membrane, produces thick mucus and ultimately results in breathing trouble particularly during expiration. Externally, it produces wheal and flare reactions on the skin i.e. elevated patches with itching and surrounding redness. A similar observation is made in urticaria. Initially, histamine gives the typical symptoms of hay fever in the nose and eyes mainly sneezing, blocked nose, swollen and itching eyes.



Schematic presentation of mechanism of dust mite allergy

On the basis of immunological mechanisms, Gell et al. (1977) classified the allergic reactions into four distinct classes.

I. **Type I reaction (Immediate hypersensitivity)**: These reactions are mediated primarily by IgE antibody and is referred as reagin or skin sensitizing antibody. It can circulate freely or bind to basophils or mast cells at specific Fc receptor site. When mast cell bound IgE combines with antigen, it initiates the characteristic events of immediate type hypersensitivity within seconds, leading to the release of mediators like histamine, leukotrines, eosinophil and neutrophil chemotactic factors from the granules of mast cells. Clinical conditions associated with type I hypersensitivity includes extrinsic bronchial asthma, allergic rhinitis,

some cases of urticaria, food allergies, certain drug allergies, reactions to stinging insects and systemic anaphylaxis.

II. **Type II reaction (Cytotoxic)**: It involves the binding IgE antibody to cell bound antigen. Antigen-antibody binding results in activation of the complement cascade and the destruction of the cell (cytolysis) to which the antigen is bound, e.g. Immuno haemolytic anaemia and Rh haemolytic disease in new born child.

III. **Immune complex mediated reactions**: These reactions are caused by free circulating antibodies, precipitins, belonging to the immunoglobulin G. The complement system is activated when these antibodies react with locally introduced antigens to produce an antigen-antibody complex. This activation of complement together with complement induced granulocytes, accumulation and release of histamine as anaphylatoxin occurs, thus results in tissue damage. e.g. Allergic alveolitis, certain types of nephritis, serum sickness.

IV. **Delayed hypersensitivity**: This type of reaction is caused by immune competent cells (lymphocytes) and as a rule symptoms occur 12–48 h after exposure to the allergen. These lymphocytes are immunologically specific with receptors for the antigens. The classical examples of type IV reactions are the positive tuberculin reactions, contact dermatitis and rejection of tissues transplanted from other individuals.

Besides usual four types of reactions, recently, an additional type of hypersensitivity reaction, called "Stimulatory hypersensitivity" has been proposed. Many cells receive instruction by agents such as hormones through surface receptors, which specifically bind to external agents presumably through complementarily of structure. This combination may lead to allosteric changes in configuration of the receptor or of adjacent molecules which become activated and transmit a signal to the cell interior.

4.3 Immunoglobulin E (IgE) and Its Biological Role in Allergy

The observation that instant wheal and flare skin reactions could be elicited by applying small quantities of pollen or pollen extracts to the skin of sensitive patients was first made over century ago. During 1920s, Prausnitz and Kustner established that the factor responsible for the allergic skin reaction could be passively transferred by serum of a sensitive person to a non sensitive recipient. This factor was called 'reagin' or 'skin sensitizing antibody'. A major breakthrough in search of the nature, immunochemical and biological properties of IgE and its association with allergic diseases occurred during mid 1960s, when Dr. Kimishige and Teruko Ishizaka and Drs. Johansson and his associates independently discovered that the skin sensitizing antibody belonged to the unrecognised IgE immunoglobulin class.

Ishizaka and Ishizaka (1967) isolated IgE from the serum of allergic patients and demonstrated that it was the carrier of reaginic activity. Simultaneously, Johansson (1967) recognised a patient with an IgE myeloma and isolated IgE (originally called IgND) from his serum. Collaborative studies by the two groups later revealed that IgND and gamma E globulin were in fact the same molecule and it was named immunoglobulin E (IgE) at a WHO meeting held at Geneva in 1968.

The discovery of a new immunoglobulin class IgE and subsequent development of new technique to measure total IgE protein and detection of allergen specific IgE antibodies opened a new vista in the field of clinical allergology. The serum concentration of IgE was found significantly elevated in most patients with allergic diseases such as hay fever, atopic eczema and extrinsic asthma. Further, the patients suffering from parasitic disorders such as intestinal capillariasis, bilharziasis, hookworm, ascariasis and echinococcosis were reported to have highly increased IgE levels that were much higher than that caused by an atopic allergy (Berg and Johansson 1969; Juhlin et al. 1969).

The elevated serum IgE levels in atopic individuals and questionably atopic patients have been demonstrated by Johansson et al. (1970), Dockhorn (1982) and Nelson (2003). Johansson et al. (1970) while comparing the data obtained from children with that of seasonal and perennial sufferers observed that the later group had the highest IgE levels. Spitz et al. (1972) studied serum IgE levels among asthmatics in relation to the spectrum of the disease and noted that as the atopic score increased the serum IgE level also spurted up.

Kjellman et al. (1976) observed that the determination of total IgE provide a valuable means for predicting future atopic manifestations which might be used as a screening procedure especially in children of atopic parents. They demonstrated that a child having a high IgE level above the geometric mean are very likely to develop a clinically significant atopic allergic disorder. Sears et al. (1989) suggested that the healthy subjects with a family history of allergy could feature higher IgE levels than those without. According to Dockhorn (1982), 75 % patients with allergic asthma possessed elevated total serum IgE levels. In the recent past, serum IgE and IgG4 antibodies in adults and children with mite sensitive bronchial asthma were measured by different workers.

On the other hand, some workers like Gleich et al. (1971) and Orgel et al. (1975) claimed that the elevated serum IgE levels and atopic diseases are not invariably associated with each other. Henderson et al. (1971) first of all, raised doubt about the usefulness of measurement of total serum IgE in the diagnosis of atopic diseases, specially in adults. Additionally, Bennich et al. (1968) noticed that in some allergic asthmatic subjects, circulating serum IgE levels may remain within normal limits. Orgel et al. (1977) observed low levels of IgE frequently in the subjects with atopic diseases, while the subjects without such disorders often showed high level. Although many studies confirmed the existence of high IgE values in atopic disorders, a considerable overlap between the levels of total IgE in healthy subjects and allergic patients has also been reported by Zetterstorm and Johansson (1981).

King et al. (1985) considered IgE levels as poor predictors of whether or not a person was allergic to house dust mites, or for that matter whether a person was allergic at all. According to Nagaya (1979) there was a tendency to have more positive RAST scores with higher levels of serum IgE but again there was a broad overlap. He identified 42 patients with typical signs and symptoms of bronchial asthma, positive skin tests and RAST results but with total IgE less than 100 IU/ml. In their studies, Bousquet et al. (1982) also reported that total serum IgE levels were within normal range in 38 % of the allergic patients. According to Nelson (2003) there is no fixed level of IgE which could be used to rule out the presence of allergic disease. He further stated that determination of serum IgE did not in any practical way help the management of those patients.

According to Wide et al. (1967), "the more allergens the patient is allergic to, the higher the serum IgE value seems to be". Subjects with multiple severe allergies exhibited higher levels of serum IgE than subjects with single allergen sensitivity. Loeffler et al. (1973) demonstrated a significant correlation between categories of skin test reactivity and the group mean serum IgE level. However, when the individual values were examined, an extremely broad range of serum IgE occurred with each degree of skin reactivity. Hogarth-Scott et al. (1973) made the curious observation that house dust RAST scores were statistically correlated with total IgE levels. Zetterstorm and Johansson (1981) observed that the number of allergens positive to RAST were strongly correlated ($P < 0.001$) to the level of total IgE. Among patients with three or more positive RAST tests, 87 % had an IgE level above 150 IU/L. The results of skin tests was also correlated strongly ($P < 0.001$) with the total IgE level. Bousquet et al. (1982) suggested that patients who presented multiple sensitization have higher total mean serum IgE concentrations than those suffering from animal dander or pollen allergy only. Batabyal et al. (1986) also reported significantly increased levels of IgE in patients with multiple sensitivity.

It is also observed that IgE levels are influenced by sex as well as age. There are ample examples that the total serum IgE levels are affected by sex. A more detailed study demonstrate age and sex related differences in total IgE and mite specific RAST scores in the rural but not in urban population (Turner et al. 1986). Attempts to establish a correlation between mean serum IgE level of patients and the duration of the disease were made by several workers and it is proved that the serum IgE levels remained virtually unchanged irrespective of the duration of disease. Besides, the presence or absence of personal and/or family history of atopic disease contributed significantly to the serum IgE levels of the patients.

On the other hand, Berg and Johansson (1969) observed no differences in IgE concentration, which could be considered as sex dependent. There exist conflicting reports that the total IgE levels were (Wood and Oliver 1972; Barbee et al. 1981) and were not (Nye et al. 1975; Zetterstorm and Johansson 1981) dependent on sex as well as age. In the opinion of Bousquet et al. (1982), mean IgE concentration were not significantly different in men and women. In an earlier investigation, Salkie and Weimer (1984) showed that the total serum IgE levels were influenced by sex, though it was contradicted by Batabyal et al. (1986), who found

no significant difference in serum IgE levels in different age groups and sexes. A more detailed study undertaken by Turner et al. (1986) demonstrated age and sex related differences in total IgE and mite specific RAST scores in the rural but not in urban population. Khatua et al. (1987) suggested that with increase in the age of patients, the total IgE and RAST positive cases increased, so also the sensitivity with increased number of allergens.

4.4 Immunoglobulins Other Than IgE in Allergy

IgE is not the only immunoglobulin responsible for immediate allergic reactions. Available report demonstrate that Immunoglobulin G (IgG) antibodies were responsible for immediate allergic reactions and called them "Short term anaphylactic IgG antibodies". Assem and McAllen (1970) noticed positive reaction of patients' leucocytes with anti IgG, anti IgM and anti IgA sera, suggesting that the antibodies belonging to these immunoglobulin classes also play role in causing immediate allergic reactions. According to some authorities, immediate allergic reactions were due to IgE antibodies in most asthmatic patients, yet there was a group with reactions due to short term anaphylactic IgG antibodies and suggested that, "Short term anaphylactic IgG antibodies" belonged to the IgG_4 sub class.

Chapter 5
House Dust Mite Allergy—An Environmental Enigma

House dust mite allergy is a significant risk factor for asthma in many countries and high exposure to the allergens contributes to airway inflammation and asthma exacerbations. Dust allergy is more or less a common problem among individuals sensitive to dust inhalation and sensitizations to house dust mites trigger intense allergen-induced inflammation of the skin and airways mucosa of atopic subjects resulting in various allergic manifestations like atopic dermatitis, atopic eczema as well as allergic rhinitis and in extreme cases the bronchial asthma. The association between house dust and asthma has long been suspected as was documented by Sir John Floyer (1698) in his book 'Treatise on Asthma' and distinguished asthma from other forms of breathlessness, and gave a detailed account of the disease. The bronchi of asthmatics are sensitive to various aero allergens specially inhalants namely pollens, moulds, feathers, furs, animal dander, dust, house dust mites etc., repeated exposures to which may trigger off bronchial spasm in susceptible individuals.

House dust is a complex assortment of substances of both plant and animal origin, and consists of animal and human dander, debris from wool, feathers, hairs, insects, cotton, silk, jute and synthetic fibres, carpets, beddings, furniture and upholstery. Apart from these fungal spores, bacteria, other microorganisms as well as shedding from other house hold articles also contribute to its composition. Although house dust has long been suspected to cause sneezing and wheezing in sensitive subjects, the exact nature of the main allergen in house dust was unknown for quite a long time. Kern (1921) and Cooke (1922) pointed out the involvement of house dust in respiratory allergy and claimed the presence of distinct allergen within it. It is now well documented that a tiny organism called mites, present in the house dust are the main source of allergen. Dust mites can directly or indirectly affect the human health and there are substantial evidences that the people handling materials heavily infested with mites are known to suffer from various allergic manifestations such as allergic rhinitis, bronchial asthma etc. Voorhorst et al. (1964) first pointed out that house dust mites belonging to the genus *Dermatophagoides* of the family Pyroglyphidae is the most potent allergen in house dust responsible for various allergic manifestations. Subsequently, allergy

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skin tests and nasal and bronchial challenge tests have clearly established that pyroglyphid mites are chiefly responsible for house dust allergy. These findings were further confirmed by the estimation of total serum IgE level and detection of mite specific IgE antibodies present in patients' sera (Aas and Johansson 1971; Saha 1993).

The earliest report on the presence of mites in homes especially, in the dust of bedrooms and mattresses came from the work of Dekker (1928). Later, following the isolation of it from floor and mattress dust by Baker et al. (1956), a number of studies in the field of Acarology on various aspects of this group of mites have been performed. Thus, during the past few decades, the mite fauna of house dust in different parts of the world has received considerable attention.

5.1 The Mighty Mites and Their Medical Importance

Mites are usually minute (0.6–2.0 mm in length) creatures, difficult to detect with naked eye. There are about 48,000 species of mites that have been identified so far around the globe (Schauff 2000). They belong to the subclass Acari under class Arachnida and phylum Arthropoda and are characterized by the absence of antennae and mandibles and the presence of simple eyes, four pairs of walking legs and two pairs of specialized mouthparts—chelicerae and padipalpi. Mites are very diverse and wide spread organisms found almost in all the habitats known on the earth, thus they are "ubiquitous, inconspicuous, harmful and helpful" (Schauff 2000). These tiny and delicate organisms exhibit extreme diversity in morphology, habitat and behaviour. Although, mostly they are plant feeders, fungivorous, algivorous, saprophagus, blood and lymph sucker, coprophagous or carnivorous, there are many whose feeding habits are still unknown. They mostly occur in soil, litter, humus, compost heaps, moss, lichens growing over tree stumps, twigs, leaves, flowers and buds, granaries of stored foods, house dust, nests of birds, caves, nests of mammals and various other places, viz. pasture soils, coniferous taiga forest, arctic tundra and even in sub-antarctic zones. A few species are slightly hydrophilic and still fewer are known to inhabit the sea.

Though several classifications (Baker and Wharton 1952; Evans et al. 1961; Hughes 1961, 1976; Krantz 1978) on the systematic position of mites under phylum Arthropoda have been put forward, the most widely accepted one is of Evans et al. (1961) and Hughes (1976). The schematic representation of the classification proposed by Hughes (1976) is given below:

Phylum	Class	Sub-class	Order
Arthropoda	Arachnida	Acari	Notostrigmata, Tetrastigmata, Metastigmata, Mesostigmata, Prostigmata, Astigmata, Cryptostigmata

Among mites there is a taxonomically and ecologically defined assemblage called "House dust mite" consisting chiefly of genus *Dermatophagoides* (family Pyroglyphidae). In the house dust mites, the cuticle is soft thus enabling the body to change size and the dorsal part of the cuticle is striated which provides the base for its species specific identification.

Mites have created both interests and concerns among medical entomologists and acarologists worldwide and received considerable attention because of their importance as pest of agricultural crops as well as in veterinary and human health. The majority of mites are free living, but thousands of species parasitize plants and animals, infesting internal organs such as inner and middle ear, respiratory passage, lung, skin and intestine of vertebrates, while others serve as both reservoir and vector of serious pathogens (viz. rickettsial pox and scrub typhus etc.).

5.2 Mites as Parasite

A large number of mite species are known to parasitize diverse group of animals causing damage to their internal organs. Most of the mites are fairly host specific, developing successfully on only one species or group of closely related animals. For example, *Myobia musculi* and *Radfordia ensifera* are blood-suckers of house mouse and white rats, respectively. The tropical rat-mite (*Ornithonyssus bacoti*), the northern fowl-mite (*O. sylviarum*) and the tropical fowl-mite (*O. bursa*) are important blood-sucking forms living on domestic animals, while, *Dermanyssus gallinae*, the chicken-mite, parasitizes chicken and *Acarophenax lacunatus* parasitizes coleopteran insects *Rhyzopertha dominica*. Krametter-Froetscher et al. (2006) studied the occurrence of cattle-ear-mite, *Raillietia auris*, which is not a parasite, rather feed on ear wax and sloughed epidermal cells. *Sternostoma tracheacolum* is known as the canary lung mite and is found in the tracheae, air-sacs, bronchi and parenchyma of the lungs. *Trombicula* larvae feed on lymph and contents of disintegrated cells of their vertebrate hosts. *Cheyletiella parasitivorax* (the rabbit-fur-mite) reportedly infests sheep in Australia; *Psorergates ovis* causes itching and irritation in sheep, while *Dermoglyphus* spp., *Falculifer* spp., *Megninia* spp. etc. live in the quills of feathers and cause nuisance and itching in birds. There are several mites that are parasitic to the invertebrates too, viz. *Varroa destructor* poses a serious threat to honey bees worldwide, *Acarapis woodii* lives in the tracheae of bees and can cause their death; "straw-itch" mites can cause serious problems in insect cultures. Members of the family Macrochelidae (*Macrocheles* sp.) are associated with house Flies (*Musca domestica*) and live on their eggs.

Bird and rodent mites normally live on the host or in their nests, but will migrate to human occupied areas under sudden emergencies (if their host dies or leaves the nest). Several species of mites cause dermatitis, urinary, pulmonary and intestinal acariasis, itching and allergic diseases in humans. Several species of mites have been implicated as causal factors of contact dermatitis among

persons handling infested grains. The more familiar examples are: copra-itch (*Tyrophagus putrescentiae*), wheat-pollard-itch (*Suidasia nesbitti*), Baker's-itch (*Acarus siro*.), dried-fruit-dermatitis (*Carpoglyphus lactis*) and grocer's itch (*Glycyphagus domesticus*). *Dermatophagoides* spp. cause severe scalp and facial dermititis in man; *Cheyletiella yasguri* also causes pruritic dermatities in man (Rack 1971). According to Numata et al. (1979) *Dermatophagoides farinae* plays a positive role in causing chronic urticaria in man. The grain-itch-mite (*Pyemotes ventricosus*), tropical rat-mite (*Ornithonyssus bacoti*) and tropical fowl-mite (*O. bursa*) reportedly bite man. Yoshikaya et al. (1983) have shown that *Cheyletus malaccensis* bites human skin and causes an itching papule. *Tyrophagus longior* has been found in both the digestive and urinary tracts of man. Sasa (1950) reported *Dermatophagoides saitoi* from the sputum of a female patient with typical Loeffler's syndrome. The same species has been isolated from the urine of a male patient with urinary acariasis (Sasa 1951). *Dermatophagoides takeuchi*, D. *scheremetewskyi*, *Pyemotes* sp. *Glycyphagus* sp., *Rhizoglyphus* sp., *Tarsonemus* sp. and *Cheyletus eruditus* have also been reported from the urine of patients (Bernhard et al. 1986).

5.3 Mites as Vectors of Pathogens

The vast majority of the (approximately 48,000 mite species) identified so far worldwide are harmless to humans or beneficial to ecosystems (Hoy 2009). Only about 250 mite species are recognized as to cause health-related problems for humans and domestic animals. Some species of mites are known to transmit pathogens of several diseases in man. Rickettsial pox is caused by *Rickettsia akari* transmitted by the mite, *Liponyssoides sanguineus*. This mite normally parasitizes the house-mouse, *Mus musculus*, and transmits the disease to man through biting. *Androlaelaps casalis* has been found infected with the rickettsiae of Q-fever (*Coxiella burnetii*). Species of chiggers, namely, *Trombicula akamushi*, *T. deliensis* have been reported as vectors of the scrub typhus (causative organism is *Rickettsia tsutsugamushi*). In the opinion of Mulvey (1972), there exists a strong correlation between the cot-death syndrome and the presence of *D. pteronyssinus* in the bedding of infants. Fujimoto et al. (1982) opined that both *D. pteronyssinus* and *D. farinae* play important role in the pathogenesis of Kawasaki disease in some patients. Yamashita et al. (1994) observed the transmission dynamics of *Rickettsia tsutsugamushi* strains among humans, wild rodents and trombiculid mites in Japan where tsutsugamushi diseases was becoming endemic. *Dermanyssus gallinae* is suspected as being a biological vector of *Salmonella enterica* and is responsible for persistence of salmonellosis infection between successive flocks. Some ectoparasitic mites, who feed on insect haemolymph, can serve as interspecific vectors of *Spiroplasma poulsonii*, a male-killing endosymbiont of *Drosophila* (Jaenike et al. 2007). However, recent evidence suggests that *Leptotrombidium* mites harbor and acts as a vector of other pathogens

such as Hantavirus (Houck et al. 2001). Another report reveals that *Bartonella* species act as the causative agent of bartonellosis in humans (Kabeya et al. 2010); it also documents that *Schoengastia* and *Blankarrtia* belonging to the family Trombiculidae can harbor *Bartonella* spp. Furthermore, other mites belonging to the Trombiculid family, *Neotrombicula* and *Eushoengastia*, can harbor *O. tsutsugamushi*. Therefore, these observations suggest that mites may play a greater role in disease transmission.

5.4 Mites as Source of Potent Allergen

Although Sir John Floyer observed mites in dust in 1698, it was only in the 1960s when their associations with the allergies were reported. Over the years, dust mites have become a subject of extensive research for their association with allergies and respiratory ailments.

Dust mites are microscopic organisms found in homes and are 0.2–0.3 mm long and translucent. Because of this, they are essentially invisible to the unaided human eye. Allergens have been described from the families Acaridae, Glycophagidae and Pyroglyphidae. The acarid mites *Acarus siro* and *Tyrophagus putrescentiae* are minor sources of house dust allergens. For Glycyphagidae, *Blomia tropicalis* has emerged as a prominent house dust mite in tropical and subtropical regions. The pyroglyphid mite *Dermatophagoides* spp. is distributed from temperate to tropical regions, while *D. farinae* is noted in the drier regions and *Euroglyphus maynei* is found in temperate regions but in lesser abundance. The house-dust mites *Dermatophagoides farinae*, *D. pheronyssinus* and *Euroglyphus maynei* are cosmopolitan inhabitants of human houses worldwide. These mites are the sources of multiple potent allergens that trigger allergic reactions in house-dust-mite-sensitive individuals. House dust mites are nearly universal in occurrence—although they do not bite, their waste products, when inhaled by sensitive people, cause allergy symptoms. It is actually the excretory products of these mites that causes the allergic reactions, thus even the dead dust mites are responsible for similar manifestations. Food is rarely a problem for the house dust mites. Their primary food is skin scales (dander) contained in house dust. All humans shed about 5–10 grams of dead skin each week. Since the greatest fallout occurs in areas of human activity, the mites tend to be most numerous in beds, overstuffed sofas and chairs, and adjacent carpeted areas. About 80 % of the material seen floating in a sunbeam is actually the flakes of skin. As relative humidity tends to be higher in those areas due to perspiration and exhalation, the occurrence of the dust mites are highest where human sleep and lie about. Mattresses, sofas, carpet, and other soft furnishings trap and accumulate dust, dander, and moisture, making them ideal microhabitats for mite development. House dust mites go through five major life stages: egg, larva, protonymph, tritonymph and adult. Between the life-stages the mites molt, shedding their outer skin. When temperature and humidity are optimum, development from egg to adult takes about one month while their

longevity varies for about 2–4 months. Nevertheless, during its short life span, a single dust mite can produce waste product that is about 200 times its weight and can lay up to 300 eggs. So it is quite natural to find thousands of mites in a gram of house dust. The mites thrive best in warm, humid conditions where their populations can explode. Unfortunately, for humans, the beds in which we sleep are warmer and most humid places year round. Dust mites live in mattresses, pillows, carpets, fabric furniture and stuffed toys and like to burrow into the fabric and escape the light. As and when sensitive individuals are exposed to the allergenic by-products of dust mites, allergic reaction initiates and thus responsible for year-round allergic manifestations.

House dust is often heavily contaminated with the allergenic components comprising of fecal pellets and cast skins (Pepys et al. 1968). Case studies reveal that dust mites are accountable for 50–80 % of asthmatics, as well as in countless cases of eczema, hay fever, and other allergic ailments. Symptoms usually include sneezing, itching, watery eyes, wheezing etc. and appearance of red rash around the neck. Other allergic reactions may include headache, fatigue, and depression (Lyon 1991). Inhalations of dust mite allergens by hypersensitive individuals may also result in acute attacks of bronchial asthma, accompanied by wheezing, shortness of breath, and even death. Diagnostic tests and clinical studies have shown house dust mite to be the most common allergen and "root cause" for the development of asthma in young children. Recent studies suggest that at least 45 % of young people with asthma are allergic to house dust mites. Unlike "seasonal" allergies caused by molds and pollen, people who are allergic to dust mites often will have symptoms year round. The role of house dust mites inducing allergy has been increasingly recognized by allergologist and aerobiologists. However, clinical investigations of house-dust mite allergy in tropics are few (Voorhorst 1969; Anand 1981; Hurtado and Mava 1987). Group 1 allergens of the mites *D. farinae* (Der f1) and *D. pteronyssinus* (Der p1) are the most significant allergens; 80–95 % of patients allergic to dust mites have an elevated IgE response to them (Peng et al. 1998).

Review of literature revealed that extensive research work on house dust mites specially *Dermatophagoides* spp. in relation to nasobronchial allergic disorders dated back to the early 20th century. According to Kern (1921) and Strom van Leeuwen (1924), house dust contained an allergen, responsible for sneezing and wheezing in sensitive subjects. Since then, investigators in different parts of the world were engaged searching relentlessly for the source of the allergen in house dust. The view that mites could be held responsible for asthma was first put forward by the Italian physician Giacome Ancona in 1923, on the basis of his observation on grain workers suffering from asthma and dermatities on the neck, breast and arms. In the past, several cases of allergic diseases have been described in people handling materials heavily infested with mites. Voorhorst et al. (1964), was the first to claim that house dust mites of the genus *Dermatophagoides* was the commonest single allergen responsible for causing allergic rhinitis, atopic dermatitis and bronchial asthma. Such a claim of Voorhorst et al. (1964) was subsequently confirmed by several workers from different corners of the world, such as

Maunsell et al. (1968) from England; Bernecker (1968) from Germany; Spieksma et al. (1967) from Netherlands; Miyamoto et al. (1974) from Japan; Brownswijk and Sinha (1971) from Canada and Mulla et al. (1975) from U.S.A. Presence of house dust mites and their association with the aetiology of asthma were also reported from the Tropics (Buchanan and Jones 1972) and Nigeria (Somorin et al. 1978). According to Virchow et al. (1976) about 90 % cases of house dust allergy were due to mites and *D. farinae* plays a positive role in causing chronic urticaria in man and the same has been recently confirmed by Mahesh et al. (2005). In the recent past, Saha (1993, 1997) and Poddar et al. (2006b) have pointed out that house dust mites particularly the genus *Dermatophagoides* is responsible for naso-bronchial allergic disorders in susceptible persons in Kolkata, India.

Tovey et al. (1981) showed that more than 95 % of the allergens accumulating in mite cultures was associated with their faecal particles and emphasized their role as the major source of house dust allergens. Excrement particles obtained from mite's intestines by microsurgery were found highly allergenic in patients, allergic to house dust as was reported also by Halmai and Alexander (1971). Muto et al. (1985) emphasized the role of house dust mite in the dwelling houses as a trigger of asthmatic attack, by measuring Anti- DP specific IgE antibodies. They further opined that allergy to house dust mites is a significant feature in the disease pathogenesis and is associated with modifications to traditional lifestyles such as introduction of cover less blankets, foam mattresses instead of traditional cotton mattresses, changes in sleeping habits that favours more fertile environment for growth and multiplication of mites.

Beds are a prime habitat for dust mites (where 1/3 of life occurs), and an used mattress may have up to 10 million mites inside (Lyon 1991). Also, bedroom carpeting and household upholstery support high mite populations to a great extent (Lyon 1991). Mites prefer warm, moist surroundings such as the inside of a mattress when someone is on it.

Chapter 6
House Dust Mites—A Precarious Indoor Allergen

6.1 Systematic Account and Identifying Features of House Dust Mites

Family Pyroglyphidae

Family Pyroglyphidae was created by Cunliffe in the year 1958. Subsequently, Hughes in the year 1961 synonymized the Pyroglyphidae with the family Mealiidae and the family Epidermoptidae. Fain (1965) first gave a detailed description of the family as "small mites with cuticle finely or coarsely wrinkled; tarsi ending in a globular pulvillus and a small claw, anus ventral; vestigial genital sense organs present in both sexes; vulva of the female reverse Y or V shaped; oil glands present and open between L2 and L3; vertical setae absent". According to Fain (1965), this family consisted of 5 nidicolous and detritivorous species including the common house dust mite, *Euroglyphus maynei*. However, in 1967, Fain extended the family to include the genera *Dermatophagoides and Sturnophagoides* in a separate sub-family Dermatophagoidinae. The family Pyroglyphidae now contains 36 species under 15 genera and 2 subfamilies (Mulla and Medina 1980), of which, approximately one third have been recorded from domestic dust (Wharton 1976).

Genus-*Dermatophagoides*

The genus *Dermatophagoides* belonging to the family Pyroglyphidae was erected by Bogdanov in 1864 for *D. scheremetewski,* and are found on human skin. Fain (1967) divided the family Pyroglyphidae into two subfamilies (a) Pyroglyphinae and (b) Dermatophagoidinae, the later include the genera *Dermatophagoides* and *Sturnophagoides.* The genus *Dermatophagoides* was reported for the first time in floor and mattress dust in the year 1956 by Baker et al. and was the most prevalent and abundant pyroglyphid mite recovered from house dust.

They are free living and occupy every niche of the house, more abundant in beds than elsewhere, because beds are rich in human skin scales on which they feed (Spieksma 1967; Bronswijk 1973) and provide the ideal microclimate such as temperature, humidity etc. Among several species of *Dermatophagoides*

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mites, *D. pteronyssinus* and *D. farinae are* most common and ubiquitous in distribution (Wharton 1976). House dust mites generally prefer warm moist places (e.g. mattresses, stuffed furniture, pillows, blankets, carpets, quilts, soft toys, curtains etc.) with plenty of shed skin scales of human and breed there. *D. pteronyssinus* has been reported from England, Wales, Netherlands, Canada, Denmark, Belgium, Itally, France, Russia, Barbados, India, Pakistan, Germany, Finland, Iran, Australia, Brazil, Surinam, Malaysia, etc. Because of its wide spread distribution, especially in the European countries, it has been designated as the "European house dust mite".

D. farinae is another cosmopolitan species of pyroglyphids and is reported to be dominant in house dust from USA (Bronswijk and Sinha 1971), Czechoslovakia (Dusbabek 1975), Egypt (Frankland and EI-Hefny 1971). This species has been designated as the "American house dust mite". It is the only species found in all habitats and is probably the least ecologically specialised of all pyroglyphids.

Identifying features

1. Pyroglyphids with the anterior edge of the propodosoma not overhanging the gnathosoma, the external scapular setae longer than the internals.
2. Two pairs of long setae projecting from the posterior margin of the body.
3. Cuticle finely striated, in the female posterior genital shield of average size, not sclerotised and lacking a bifid anterior margin.
4. Absence of hysterosomal shield.

6.2 Mites in House Dust at Global Scale

The evidence of the presence of mites in house dust dates back to seventeenth century. Since then, a number of researchers have described many species of mites from the house dust samples. During the last few decades the mite fauna of house dust in different parts of the world has received considerable attention. In an earlier report, Oboussier (1939) reported *Carpoglyphus lactis*, *Glycyphagus domesticus*, *Glycyphagus privatus* and *Tyrophagus longior* from Hamburg (Germany) house dust samples. Solomon (1961) in England confirmed the presence of moss-mites (Oribatei), spider-mites (Tetranychidae), blood-sucking mites e.g. the chieken mite, *Dermanyssus gallinae* and other Gamasides as possible inhabitants or invaders of houses, shops and other occupied buildings. *Glycyphagus domesticus* occuring on damp walls was referred by him as the typical house mite. Solomon (1962) further reported the presence of certain species of mites particularly associated with food stuffs in houses e.g. the flour-mite, *Acarus siro*; the dried fruit mite, *Carpoglyphus lactis*, *Thyreophagus entomophagus*, *Glycyphagus destructor*, *Gohieria fusca*, several species of *Tyrophagus* and the common predatory mites of the genus *Cheyletus*, especially *Cheyletus eruditus*. In addition, some unidentified mites were also reported to be present in abundance in house dust

samples. Oshima (1964) classified those mites as the genus *Dermatophagoides* and subsequently, Fain in the year 1966, identified them to the species level as *Dermatophagoides pteronyssinus.* It has been reported that *D. pteronyssinus* was almost invariably present in the dust samples and constituted about 70 % of the total mite fauna (Voorhorst and Spieksma 1973).

Another species belonging to the same genus, namely *D. farinae* was first found in infested poultry and pig rearing meal near Bristol, England (Williams 1961). The adult forms were described by Hughes (1961) and later, redescribed in greater detail by Fain (1967). In some parts of the world this species of house-dust mite occurred in greater number than *D. pteronyssinus* (Halmai 1984).

Although mites have been reported from variety of habitats, the mite fauna of house dust has not been fully explored. The available literature documented approximately 130 species of mites from the house dust samples all over the world (Zheltikova et al. 1985). Different authorities from several corners of the globe indentified and documented different numbers and variety of mites from house dust sample and excerpts of the same is depicted below.

Country	No. of species	Reference
Netherlands	10 species	Spieksma and Spieksma-Boezeman (1967)
United Kingdom	8 species	Maunsell et al. (1968)
United Kingdom	5 families	Blythe et al. (1975)
United Kingdom	31 species	Colloff (1987)
Japan	65 species	Sharp and Haramoto (1970)
Japan	36 species	Oshima (1970)
Thailand	14 fam.	Wongsathuaythong and Lakshna (1972)
USA	9 species	Shamiyeh et al. (1973)
Panama	29 species	Miranda et al. (2002)
Poland	29 species	Solarz et al. (2004)
Switzerland	38 species	Mumcouglu (1976)
Brazil	9 species	Cao Lopez and Estruch fajardo (1981)
Brazil	10 species	Galvao and Guitton (1986)
Havana, Cuba	5 species	De Moreira and Filagonio (1980)
Germany	35 species	Keil (1983)
Italy	23 species	Castagnoli et al. (1983)
USSR	10 species	Ivanova and Petrova (1984)
USSR	22 species	Dubinina (1984)
Malaysia	6 species	Rueda (1985)
Czechoslovakia	16 species	Vobrazkova et al. (1986)
Spain	6 species	Del Rey Calero and Lomas (1972)

6.3 Predominance of *Dermatophagoides* Species in House Dust

The occurrence of a variety of mites in house dust have been reported by earlier workers, among which mites of the genus *Dermatophagoides* predominated (Wharton 1976). Maunsell et al. (1968) reported twenty species from fourteen genera belonging to six families, of which the most common species was *D. pteronyssinus*. Oshima (1970) conducted a faunistic survey of the mites in house dust of Japan and Taiwan and reported the preponderance of the genus *Dermatophagoides* to the extent of 90 % in house dust among thirty six different species of mites isolated. Boquete (2006) collected 332 dust samples with a portable vacuum cleaner from the surface of the mattress used by allergic patients as well as from controls. According to them, *Dermatophagoides pteronyssinus* was the most common species among thirty two mite species identified, followed by the predatory mite *Cheyletus* sp., *Lepidoglyphus destructor*, *Euroglyphus maynei*, *Chortoglyphus arcuatus* and *D. farinae*. A recent investigation of house dust mite incidence in relation to the social factors in Turky was made by Aycan et al. (2007) and they obsereved the predominance of *Dermatophagoides* mites in all instances. Predominance of *Dermatophagoides pteronyssinus* and *D. farinae* in the house dust samples almost all over the world have been reported by many authors, namely, Spieksma and Spieksma-Boezeman (1967) and Bronswijk (1973) of Netherland; Oshima (1970) of Japan; Maunsell et al. (1968), Blythe et al. (1975) and Colloff (1987) of United Kingdom; Massey and Massey (1984) of Hawaii islands; Rey Calero and Lomas (1972) and Gomez et al. (1981) of Spain; Guy et al. (1972) and Araujo-Fontaine et al. (1973) of France; Mumcouglu (1976) of Switzerland; Charlet et al. (1977) of Colombia; Abbott et al. (1981) of New Zealand; Green (1983) of Australia; Korsgaard (1983) of Denmark; Ivanova and Petrova (1984) of USSR; Feldman-Muhsan et al. (1985) of Israel; Vobrazkova et al. (1986) of Czechoslovakia, Hurtado and Mava (1987) of Venezuela, and Chang and Hsieh (1989) of Taipei. All of them arrived at a commonalty that *D. pteronyssinus* is the most predominant mite species in the house dust sample and it constituted 42.3–92.6 % of the total population depending on the geographical location. In contrast, Wharton (1970) of U.S.A.; Frankland and El-Hefny (1971) of Egypt; Wongsathuaythong and Lakshana (1972) of Thailand; Dusbabek (1975) and Makovcova et al. (1982) of Czechoslovakia; De Moreira De Moreira (1982) of Brazil; Hallas and Korsgaard (1983) of Denmark; Halmai (1984) of Hungary; Solarz (2004) of Poland reported D. *farinae* as the predominant mite species which accounted for 33.64–51.77 % of all the mites isolated from the house dust samples. Mariana et al. (2000) reported *Blomia tropicalis* was the most common and abundant house dust mite in tropical and subtropical areas. According to them the much shorter development period of all the stages in the life cycle of *Blomia tropicalis* compared to *D. pteronyssinus* may appear as an advantage for the species to become more abundant. In a recent study, Catanghal and Paller (2012) showed that dust mites are commonly found in carpets and mattresses in houses

of Philippines. They identified twenty three species of mites belonging to 7 families, of which, *Blomia tropicalis* of family Glycyphagidae and *Dermatophagoides farinae* of family Pyroglyphidae were the most prevalent and abundant species. In a more recent study, Gill and Kaur (2014), while working on the occurrence, prevalence and composition of mite fauna in human dwellings of Patiala City, Punjab, India, claimed that although allergenic mites belonging to Astigmata was found in maximum number in all the infested samples, *Blomia tropicalis* was the predominant one followed by *D. farinae.* For this reason Spieksma (1967) proposed "the house dust mite" as the common name for *D. pteronyssinus*, but Wharton (1970) preferred to record *D. farinae* as "the American house dust mite" and *D. pteronyssinus* as "the European house dust mite".

Several views have been put forward regarding the variation in the density of mites in the dust samples collected from the houses of bronchial asthma/allergic patients and healthy individuals and in fact, there was hardly any agreement. Studies on the variation of house dust mite population density in the houses of asthmatic and control subjects have been made and similar levels of infestation were reported by several workers (Spieksma 1967; Maunsell et al. 1968; Bronswijk and Sinha 1971; Sesay and Dobson 1972). According to Takaoka et al. (1977), Ishii et al. (1979), Mulla and Medina (1980) and Ming (1983) the mite densities as well as the mite fauna in houses of asthmatics were not conspicuous and significantly different from that found in houses of normal individuals (non-asthmatics). A significant difference between total mite population and pyroglyphid mite densities in houses occupied by asthmatic and non asthmatic individuals have also been reported by some workers (Charlet et al.1977; Murton and Madden 1977). In the opinion of Charlet et al. (1977) the density of mites in dust samples taken from floors and mattresses of allergic individuals was higher than the corresponding habitats of non-allergic individuals and a significant difference was revealed in floor dust. According to Korsgaard (1983), the density of the house dust mite (*D. pteronyssinus*) was higher in the dust sample taken from houses of asthmatic patients. Dubinina (1984) reported that the mite fauna in the houses of asthma patients was abundant in respect of both the number of species and the density of the mites as a whole. On the other hand, in the opinion of Colloff (1987) the dust samples taken from houses of atopic asthmatics contained significantly fewer mites than those from non-asthmatics and he attributed this disparity partly due to regular vacuum cleaning of mattresses and carpets and partly due to the use of some antidotes for avoiding house dust mite by some of the patients.

6.4 Does Mite Density Influence the Frequency of Disease?

It could be a normal expectation that the persons exhibiting allergic symptoms with a history of sensitivity to dust would be expected to have long term exposure to population of house dust mites prevailing in the dust samples of their homes.

It has been claimed by several workers (Spieksma 1967; Mitchell et al. 1969) that there exist a correlation between the number of mites and the allergen content in samples of house dust. According to Voorhorst et al. (1967), Maunsell et al. (1968) and Voorhorst (1969) the degree of house dust antigenicity was proportional to the concentration of pyroglyphids in the dust. Larson et al. (1969) observed a significant ($p < 0.05$) correlation between pyroglyphids in dust and positive skin reactions towards that dust sample. Brown and Fillar (1968) also reported the correlation between intense reaction and concentration of mites in dust samples obtained from homes of asthmatic patients. According to Bear and Wharton (1970), the load of mites itself could determine the specific allergenicity and positive cutaneous reaction towards these species of mites. However, in the opinion of Mulla and Medina, (1980) no direct relationship exists between the number of acari in dwelling houses and the frequency of respiratory allergy at all times; only when the acarine population was high correspondingly there was a high level of IgE in the serum of allergic patients. It is now well documented that the allergenicity of the dust sample depends on the number/load of mites specially the pyroglyphid contained in the dust (Saha 1994). Warner et al. (1998) used the limit of 100 mites/gm of dust as the risk level for sensitization.

6.5 Mites Other Than *Dermatophagoides* in Allergy

Allergy due to mites is not confined only to the *Dermatophagoides* mites. The symptoms of asthma are also reported to be associated with other species of mites viz. *Pyemotes ventricosus*, *Acarus siro* and *Glycyphagus domesticus*. Saxena et al. (1981) reported from India that *Caloglyphus berlesei* might be one of the contributing factors towards house dust allergy. In the recent past, several cases of mite allergies caused by *Lepidoglyphus destructor* and *A. siro* were reported among farmers handling infested hay (Hillerdal 1982). Further more, the grain storage mite, *Tyroglyphus* sp. was identified as a cause of occupational allergic respiratory disease among farm workers (Wharton 1976). Lutsky et al. (1984) emphasized the probable role of northern fowl mite *Ornithonyssus sylviarum* in occupationally related respiratory disease more precisely among poultry workers in Israel. Macan et al. (2012) identified some less common mites such as *Spelaeorhynchus* sp, *Melichares agilis, Cheyletus spp.* and *Hirstia passericola*. Although they were present in relatively lower densities compared to *Blomia tropicalis, D. pteronyssinus* and *D. farinae*, these mites have been reported to produce allergens evoking allergic reactions. The species *Blomia tropicalis*, *Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, *Acarus siro* and *Tyrophagus* spp. possess strong allergenic properties and represent the main cause of mite allergy (Macan et al. 2012). From India *B. tropicalis* has been incremunated as major source of allergen in house dust by Podder et al. (2010b).

6.6 Isolation and Extraction of Mites from House Dust

A variety of methods were used by many workers to isolate the mite fauna from the house dust samples across the globe. Muller (1958) used a combination of floatation and centrifugation techniques for their collection. A new method for isolation of mite fauna from the house dust samples was described by Spieksma and Spieksma-Boezeman (1967). They used sieves of different meshes to sieve the dust samples, boiled the resultants with 90 % lactic acid, centrifuged the solution and collected the supernatant on a filter paper. Saturated solution of aqueous sodium chloride was used in the residue, again centrifuged and filtered and the mites were finally collected from the filter paper(s) under a Stereomicroscope. The advantage of this method, as the authors claimed, was negligible (3 %) loss of mites due to sieving.

Oshima (1967) sieved dust samples and mixed it with a mixture of ethyl ether and carbon tetrachloride (CCl_4) having different specific gravities in the order of 1.3, 1.4, 1.5 and finally with pure CCl_4 of specific gravity 1.6. The mixtures were centrifuged and the supernatants were collected in the filter paper(s) and the mites were eventually recovered from the filter paper(s) under a Stereomicroscope.

Maunsell et al. (1968) added dichloromethane (specific gravity 1.324–1.327) to the dust sample, centrifuged and filtered. The mites so collected were transferred to lactic acid and kept for 24 h for further studies.

A very simple method for isolating mites from the house-dust samples was described by Larson and his co workers in the year 1969. The collected dust sample was sieved through different sieves, kept in a watch glass containing either ether or alcohol and mites were isolated mechanically with the help of a fine brush under a Stereomicroscope.

Voorhorst (1969) used two sieves (2.4 and 0.075 mm), one placed on top of other fitted to rotating machine. The dust samples, containing mites, were collected from the second sieve and centrifuged after adding 90 % lactic acid. The supernatant was filtered in a Buchner funnel and the mites were isolated from the samples under a Stereomicroscope.

Shamiyeh et al. (1973) used five drops of liquid soap and 30 ml salt solution on 0.1 g of dust. This was stirred and the suspension was placed in an ultrasonic cleaner for 20 min, centrifuged and the upper liquid layer was poured into a sieve to trap the mites for staining and counting.

Takaoka et al. (1977) combined floatation and centrifugation techniques with Derling's solution to isolate mite fauna from the house dust samples. Fain and Hart (1986) demonstrated a new floatation method for extracting mites from house dust cultures etc. by successive extraction with 80 % ethanol and a saturated solution of sodium chloride.

6.7 Biology of House Dust Mites

Oshima and Sugita (1966) first of all successfully completed the life cycle of *D. farinae* in laboratory condition with dried *Daphnia* as food. Mean duration (in days) of different development stages were 6.0 for eggs, 5.8 for larvae, 5.6 for protonymphs and 6.8 for tritonymphs and the whole cycle took 24.2 days to complete.

In the opinion of Spieksma and Spieksma-Boezeman (1967), at 25 °C temperature and 80 % RH, the female *D. pteronyssinus* produced 25–30 eggs in the first oviposition period commencing 3–4 days after the first copulation, and 15–30 eggs in the second oviposition period following a second mating. A third oviposition period might also occur producing fewer eggs. In such a physical environment, the adult male and female lived for 60–80 days and 100–150 days respectively. Larson et al. (1969) cultured *D. farinae* mites successfully on dog-food at room temperature and 75 % RH. They observed that development from egg to egg took 24–35 days (on average 30 days), with the egg stage lasting for 6–8 days. The females did not lay eggs unless fertilized; they mated more than once and laid 1–2 eggs per day for at least 30 days in succession. According to Mulvey (1972), the life cycle of *D. pteronyssinus* was completed in 14–20 days (minimum 13 days and maximum 30 days) at 80 % RH. The female produced 25–30 eggs (maximum 79) in 2–3 oviposition periods. The incubation period of egg lasted for 6 days. The larval stage for completion took 5–6 days; protonymph took 4–7 days, while tritonymph took 4-8 days. Furumizo (1975) worked out the life history of *D. farinae* and noted that the life cycle consisted of 7 developmental stages namely egg, active larva, quiescent larva, active protonymph, quiescent protonymph, active tritonymph and quescent tritonumph. The life cycle was faster at higher temperature and egg production was relatively less at lower temperature. Conversely, longevity increased at lower temperature. Unmated mites lived longer. Under optimum conditions of 26.6 °C and 75 % RH and reared on *Daphnia,* life cycle was completed in 23 days. The life span of adult male varied from 4 to 5 weeks and the female mites lived longer than their male counterpart. On an average, a female lived for 36 days, deposited 50 eggs, and copulated 4 times. Males lived for 23 days, fertilizing as many as 5 females during this period. The mortality rate was highest in larval stages (44 %) and gradually decreases as the development progressed. Parthenogenesis was not observed in *D. farinae*. Viviparity was highly improbable. De Moreira and Filagonio (1980) during laboratory rearing of *D. farinae* and *D. pteronyssinus*, reported the presence of a quiescent deutonymphal stage and the occurrence of such a stage in pyroglyphids was not previously recorded. Majority of the workers reported that the life cycle of *D. pteronnysinus* shows seven distinct developmental stages namely egg, larva, protonymph, resting protonymph, tritonymph, resting tritonymph and adult (Ho and Nadchatram 1984; Modak et al. 2000). However, Bronswijk and Sinha (1971) earlier reported the presence of only five life stages and excluded the possibility of any resting or pharate proto and

tritonymphal stages. It usually takes 14–16 days to complete the life cycle at a temperature ranging from 25 to 33 °C and RH varying from 57–89 % without any deutonymphal stage (Modak et al. 2000). Earlier workers (Bronswijk and Sinha 1971, Ho and Nadchatram 1984) reported a longer period (average 33 days) in both *D. farinae* and *D. pteronyssinus* while according to Matsumoto et al. (1986) the life cycle of *D. pteronyssinus* takes 37 days to complete.

Gamal-Eddin et al. (1983) reported the effects of different temperature regimes (15–40 °C) and relative humidity (50–90 %) on the duration of the pre-imaginal period (from egg to tritonymph), oviposition period, fecundity and duration of the egg stage and larval stage of the house dust mites—*D. farinae* and *D. pteronyssinus*. The results showed that the most suitable conditions for development of all four stages were 25 °C and 75 % RH for *D. farinae* and 25 °C and 80 % RH for *D. pteronyssinus*.

Ho and Nadchatram (1984) studied the life-cycle and longevity of *D. pteronyssinus in* different room temperatures and also in an air-conditioned room. Room temperatures ranged from 23 to 38.5 °C and in air-conditioned room, the temperature varied from 19.5 to 26 °C, while RH was maintained at 75 %. They found pyroglyphids having 7 distinct life stages: egg, larva, pharate protonymph, protonymph, pharate tritonymph, tritonymph and pharate adult. The eggs took 24 to 43 days (average of 33 days) to develop into adults. 31 % eggs hatched out successfully to adults, 19 % eggs did not hatch. Mortality was found in all except the pharate stages. The higher morality (42.4 %) was observed in the larval stage. The mortality rate was 23.7 and 13.8 % for protonymphs and tritonymphs, respectively, and the mortality rate decreased as development progressed. The average development time in non air-conditioned room and in the incubator was similar, whereas, it was longer in the air-conditioned room. Females and males lived for 23 days on an average. There was no significant difference between the sexes in terms of longevity.

Matsumoto et al. (1986) studied the effect of relative humidities on the life cycle of the pyroglyphids (*D. farinae* and *D. pteronyssinus*) at 25 °C temperature. At 76 % RH, the average developmental period of individuals of *D. farinae* was 39.6 days; 8.1 days for eggs; 8.2 days for larvae; 17.0 days for protonymphs and 6.6 days for tritonymphs. Two types of protonymphal stage, a "regular protonymph" developing in 5.10 days, and a "prolonged protonymph" taking more than 15 days to develop, were observed at 61, 76 and 80 % RH. A few "prolonged tritonymphs" requiring more than 20 days to develop were observed at 61 % RH. Females laid an average of 1–2 eggs per day and a maximum of 6 per day. Fecundity was highest at 86 % RH, with a total of 80.6 eggs per female. The lifespan of females was longest, averaging 188.8 days in unmated individuals at 76 % RH. The developmental period of individuals of *D. pteronyssinus* was shortest at 76 % RH, averaging 37.1 days. Both pyroglyphids died before the protonymphal stage when reared at 36 % RH.

6.8 Culture of House Dust Mites

Several authors attempted to culture the house dust mite mainly *D. pteronyssinus* and *D. farinae* using a variety of nutrient materials, and in fluctuating temperature and relative humidity. According to Spieksma (1967), human skin scales and powdered yeast were highly suitable nutrient media for the successful culture of both *D. pteronyssinus* and *D. farinae*. Pepys et al. (1968) cultured *D. pteronyssinus* on human scales and yeast and *D. culinae* on dog-meal. Miyamoto et al. (1968) used dried *Daphnia* as nutrient media for *D. farinae* and obtained positive results. Larson et al. (1969) cultured *D. farinae* mites successfully on dog-food at room temperature and 75 % relative humidity. Sasa et al. (1970) claimed that powdered laboratory food for rats and mice was the best culture medium for *D. farinae, Tyrophagus putrescentiae* and *Aleuroglyphus ovatus* and equal quantities of dried yeast and sugar served for the best purpose for *Carpoglyphus lactis*. The optimum relative humidity during culture should be 12 % for *D. farinae*, 15 % for *T. putrescentiae* and *A. ovatus* and 25–35 % for *C. lactis*.

According to Hall et al. (1971), the best culture media at 25 °C temperature and 80 % RH were yeast and gelatine or yeast alone. Penaud et al. (1972) obtained good results with a culture mixture composed of powdered human skin scales and yeast at 75 % RH and 25 °C temperature for *D. pteronyssinus* and at 75 % RH and 20 °C temperature for *D. farinae*. Waki and Matsumoto (1973) obtained the highest yield at 25 °C temperature and 60 % RH with a culture medium consisting of equal parts of dried yeast and fish meal for *D. farinae*. According to Miyamoto et al. (1975) the mixture composed of two parts of each of powdered laboratory animal food and dried yeast and one part of dried fish powder with 16 % humidity served the best purpose. Murton and Madden (1977) successfully reared *D. pteronyssinus* population in the laboratory using electric razor shavings from human beared growth as food substrate. *D. farinae* was cultured successfully by Arlian et al. (1979) with a culture medium of human hair and Fleischman's active dry yeast at 75 % RH. Equal proportions of dried yeast and powdered mouse food were used by Ishii et al. (1982) for the successful culture of *D. farinae* and *D. pteronyssinus.* Ivanova and Petrova (1984) compared 7 media for rearing *D. pteronyssinus* and observed that hairs from electric shavers combined with yeast powder in a ratio of 9:1 yielded the best result.

6.9 Habitat Preferences of House Dust Mites

Several workers (Spieksma 1967; Korsgaard 1983 and Feldman-Muhsan et al. 1985) observed a correlation between the degree of dampness and the number of mites (specially *D. pteronyssinus*) in the houses, presumably because damp houses provided better conditions for the growth of mites than dry ones. Although, report of higher numbers of mites initially came from floor dust, several studies revealed

that *Dermatophagoides* mites were much more abundant in mattress dust than any where else in the houses examined. According to Maunsell et al. (1968) mattress dust contained 100 times more mites than living room dust. Workers like, Cunnington (1967), Guy et al. (1972), Sesay and Dobson (1972), Blythe et al. (1975), Rao et al. (1975), Gamel-Eddin et al. (1982), Ivanova and Petrova (1984), Mosbech et al. (1991), Solarz (1997), Chew et al. (1999), Schei et al. (2002) and Pujari-Vandana et al. (2007) also observed higher mite abundance in mattress dust. In a more recent study, Podder et al. (2010a) while comparing the mite densities in mud and concrete house dust samples reported that the mite densities are higher in the mud houses than in the concrete houses. This study also agrees with the earlier findings of Mumcuoglu et al. (1999) and Macan et al. (2003), who also observed that a correlation exists between the mite numbers and climatic condition in the settlement. According to them the number of mites is higher in the damp environment than dry one. The mud house contains higher mite density than concrete house which may be due to the availability of proper microclimate like relative humidity and temperature for the multiplication of mite species in the former than the later (Bronswijk 1973). Beside, the use of the vacuum cleaner for dusting the floor is almost absent in the mud houses. On the other hand, it is frequently used by the inhabitants of the concrete houses. This ensures a stable food supply in the former condition. Otani et al. (1984) reported that the density of mite population was significantly higher in old and damp houses and they were much more abundant in mattress dust than anywhere else in the house. Ivanova and Petrova (1984) also made similar observation and found a correlation between density of mites and the age of the house and they also reported that the mite density is significantly higher in mud houses than concrete houses. Henzel et al. (2010) suggested that pyroglyphids per gram of dust were higher in mud house in comparison to concrete house. Nayar et al. (1974) and Maurya and Jamil (1980) from India also observed that old and damp houses contained higher densities of mites than the new houses. Several workers have reported that humidity is usually the limiting factor for mite growth (Dobson 1979; Korsgaard 1983). According to Chmielewski (1991) and Hart (1998), the growth of mite was correlated with temperature and relative humidity. Tiana et al. (2006) concluded that differences in housing characteristics could affect mite densities.

In another study, Ho and Nadchatram (1984) reported significant differences in mite densities in foam mattress as compared to cotton mattress. Schei et al. (2002) showed that the chance of finding mite fauna was four times higher in foam mattress as compared to spring mattress. According to Blythe (1976) and de Oliveira and Daemon (2003), light, temperature, RH, mechanical disturbances, predation and parasitism, inter and intra-specific competition and different building materials used may influence the population size of domiciliary dust mites. Sufficient and appropriate food is the main requirements for the successful growth and multiplication of these mites. More specifically, among different types of mattresses used, the foam mattress harbour significantly higher mite densities than conventionally used cotton mattress. This is probably due to the seldom cleaning of the former and availability of ideal micro-climatic conditions (Ho and Nadchatram

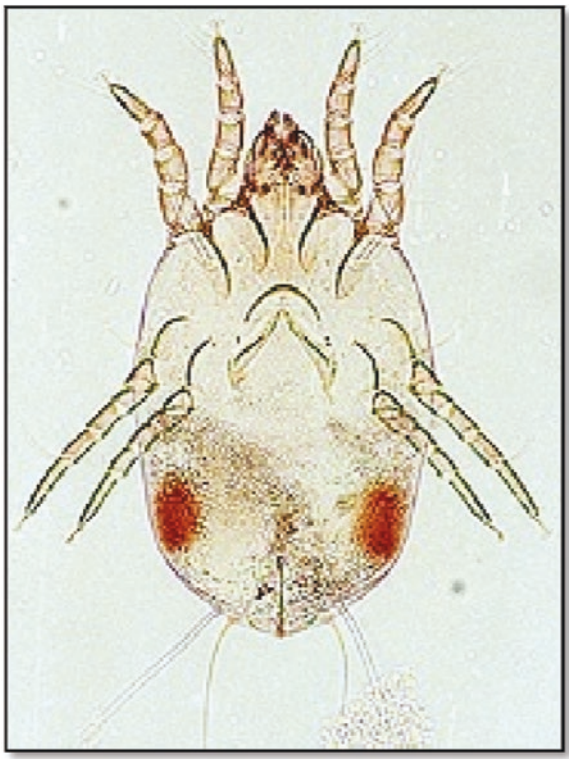
1984). Tiana et al. (2006) investigated the nature of the dust mite fauna on beds used by individuals with different socioeconomic backgrounds in Salvador, Brazil. According to their report, *B. tropicalis* was found with a similar frequency in beds of the two socioeconomic groups (poor vs. wealthy). But interestingly, *D. pteronyssinus* was found more frequently in the beds of the wealthy than of the poor group.

6.10 Seasonal Fluctuations of House Dust Mites

Regarding the seasonal abundance and fluctuation of total number of mites as well as *Dermatophagaides* mites, several authors were of different opinion. The maximum number of mites were isolated in the months of July to October and minimum in February to April by Spieksma (1968) from Netherlands; Arujo-Fontaine et al. (1973) from France; Shamiyeh et al. (1973) from U.S.A.; Takaoka and Okada (1984) from Japan; Murray and Zuk (1979) from Canada; Makovcova et al. (1982) from Czechoslovakia. According to Sharp and Haramoto (1970) of U.K., the mite population was much higher during December to February than during July to October. Domrow (1970) working from Brisbane, Australia found maximum population (live and dead mites) in February and minimum in June and July. Gamal-Eddin et al. (1982) from Egypt reported that *Dermatophagoides* mites had a peak abundance in May followed by a slightly lower occurrence during September. Arlian et al. (1983) studied the density of *D. farinae* and *D. pteronyssinus* in homes of dust allergic patients in Ohio, USA and showed a seasonal fluctuation with peaks during the humid summer months and during the period of maximum domestic heating in the winter. They suggested that house dust mite population survived the dry winter months indoor as pharate tritonymphs, which being resistant to desiccation provided the major source of breeding mites for population growth in the spring to be followed. Takaoka and Okada (1984) from Japan, further pointed out that *D. pteronyssinus* and *D. farinae* were most abundant in the months of August to September and in July to August, respectively. Feldman-Mushasm et al. (1985) from Israel observed relatively small number of mites in April as compared with the rest of the year. Yagofarov and Galikeev (1987) claimed that autumn is the favourable season for the reproduction of mites, while Chang and Hsieh (1989) reported that the mean number of mites was higher between August to November and lowest in July. Solarz (1997) investigated seasonal dynamics and age structures of pyroglyphid population in Poland and observed that with the increase of indoor humidity, *Dermatophagoides farinae* was the numerically dominant species (62.7 % of the total mites), followed by *D. pteronyssinus*.

It has been recognized that temperature and humidity are the two important key factors controlling mite abundance. Bronswijk (1973) and Arlian et al. (1983) reported that the annual periodicity is positively correlated with humidity cycle inside the house. According to Spieksma (1970) the mites are very sensitive

to variations in humidity and under RH conditions lower than 70 %, the population stops growing. It may be concluded that a temperature of 25 to 30 °C and RH between 65–75 % are optimum for the successful growth and multiplication of mites and any variation in these factors are detrimental and ultimately fatal to these small arthropods.



Dermatophagoides farinae the American house dust mite

D. pteronyssinus, the European house dust mite

Chapter 7
Dust Mite Allergy—Evaluation Procedures

The association between house dust and nasobronchial allergic manifestations have been recognised for over two decades. Available reports demonstrate that the specific allergen in the house dust are mites of the genus *Dermatophagoides*. Mites are known to be ubiquitous in dust samples all over the world and *D. pteronyssinus* and *D. farinae* seem to be the most abundant species of mites in house dust everywhere. The results of bed dust obtained from patients and control subjects' houses revealed that more than 90 % of the samples were positive for the presence of mites. It is now well documented that the average number of all types of mite fauna/g of dust, isolated from patients' beds was significantly higher than control subjects beds as evident from the work of Charlet et al. (1977), Maurya and Jamil (1980), Korsgaard (1983). However, several workers (Spieksma et al. 1967; Maunsell et al. 1968; Bronswijk and Sinha 1971; Sesay and Dobhson 1972; Dixit and Mehta 1973; Takaoka et al. 1977 and Ho and Nadchatram 1984) did not observe any difference in densities of mites between houses of asthma and non asthmatic occupants.

The genus *Dermatophagoides* was the most common mites encountered in patients' and control subject's bed dust and interestingly, patients' bed dust contained significantly greater concentration of these mites than control subject' bed dust. Both *D. pteronyssinus* and *D. farinae* mites were present in patients' and control subjects' beds in the ratio of approximately 2:1, indicating the predominance of the former species over the latter as evident from the work of several workers. It is suggested that the dominance of one species over the other was probably influenced by climatic conditions.

Analysis and comparison of results in respect to all types of mites as well as *Dermatophagoides* mites in bed dust and bed-room floor dust samples of patients revealed higher densities of mites in beds in comparison to floors and the differences were statistically significant in both cases ($p < 0.01$). According to Maunsell et al. (1968), mattress dust contained 100 times more mites than living room dust as mattresses are ideal place for the development of pyroglyphid population, since it constitutes a less disturbed habitat in contrast to floors which are cleaned occasionally. Thus, the physical appearance of higher densities of *Dermatophagoides*

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population present in bed dust samples increased the chances and duration of exposure to those offending allergens and might be responsible for allergic aetiology.

7.1 Primary Screening Through Skin Prick Test

Skin sensitivity test had been considered as an indicator for diagnostic evaluation of various types of allergic diseases and established a good correlation between the clinical history of house dust allergy, asthma and a positive skin reaction to extracts of mites and house dust. A good correlation between the clinical history of house dust allergy and positive skin response with mite extract has been established by various investigators (Maunsell et al. 1968; Pickering and Gabriel 1973). A method for the preparation of antigenic extract by the separation of mites from the culture media was developed by Maunsell et al. (1968). A year later, he performed skin tests with extract prepared from *D. pteronyssinus* mites, present abundantly in mattress dust of patients' beds and reported that 95 % of the house dust sensitive patients showed positive skin reaction to the extracts of particular species of mites.

Smith et al. (1969) highlighted the clinical significance of skin reactions to mite extracts in children with asthma and noted that most of the patients, sensitive to house dust were also positive to *Dermatophagoides* spp. Pickering and Gabriel (1973) observed a high incidence of skin sensitivity to house dust mites among patients of atopic asthma in Hongkong and the occurrence of relatively large numbers of these mites in their samples of house dust. A close and quantitative relation between the frequency of allergic respiratory problem and severity of allergy prick test reaction was suggested by Burrows et al. (1976). Tan and Teoh (1979) from Singapore reported that all patients in their study group who gave positive skin reaction, responded to house dust mite, thus establishing the importance of house dust mite as an allergen in asthmatics. Lawrence (1982) from Jamaica, reported that more than 86 % patients responded positively to prick skin test against house dust mites (either *D. pteronyssinus* and or *D. farinae*). A highly significant association between a positive prick test to mite and history of respiratory problems of patients getting worse with further exposure to dusting, sweeping, vacuum cleaning etc. was reported by Murray et al. (1983).

7.2 Cross Reactivity Between Different Mite Allergens

A good deal of cross-reactivity between different species of mites have been noted by Spieksma (1969). He also reported a high correlation between skin test reactivity to extracts of *D. pteronyssinus, D. farinae,* Euroglyphus maynei *and house*

dust. Dasgupta and Cunlifee (1970) demonstrated the presence of at least one and probably two antigens common to two species of mites (D. pteronyssinus and *D. farinae*) explaining why individuals reacted in a similar manner to the two extracts. According to Barbee et al. (1981) a positive prick test to one allergen markedly increased the likelihood of a positive reaction to a second/third allergen. However, Murray et al. (1985) suggested incomplete cross-reactivity between *D. farinae* and *D. Pteronyssinus.*

On analysing the skin prick test results against house dust and house dust mite allergens, it is well established that most of the patients responded to house dust and house dust mite allergens, thus establishing the role of mite in the aetiology of various allergic manifestations. In this context, one is tempted to infer that the main allergen in the house dust was probably derived from the mite *Dermatophagoides*, highlighting the importance of house dust mites in house dust allergy. Analysis of skin prick test results against house dust and mite allergens showed a good correlation ($p \ll 0.01$), indicating the presence of a common allergen between them. A comparison between the results of RAST with house dust and mite allergen showed a good correlation ($p < 0.10$) in their frequency of positive response to two allergenic extracts, which further confirms that the mites (*Dermatophagoides* spp.) and house dust had a common allergen.

The non-responsive patients, who did not respond to any of the battery of allergens tested, indicate that their allergic condition was either of intrinsic type or emotional in origin. The possibility of these patients being sensitive to allergens other than those commonly included in the allergen list also could not be ruled out. Withdrawal of antihistaminic drugs, not at proper time may also interfere with the test results.

On analysing the skin prick test results against house dust mites in relation to the time of onset of asthmatic attack, it is evident that the frequency of positive reaction was higher in patients whose asthmatic attack worsened after midnight as compared to other groups of patients. This might be attributed to the patients exposure to mites which were abundant in bed dust samples. Evaluation of the effect of age, sex and duration of disease on skin prick test result against house dust and house dust mites indicate that the frequency of positive skin response was not either age or sex dependent. The duration of disease, however, influenced the frequency of positive reaction and the rate was noted to be higher in patients suffering for longer duration. However, several workers emphasized that the rate of skin reaction was highly age dependent and peak reactivity occurred during young adulthood, with a decline in the older age group. Murray et al. (1983) interpreted it as the decline in reactivity which occurs in older subjects consequent upon either actual decrease in immunological reactivity or a decrease in the capacity of the skin to respond to immunologic challenge.

7.3 Eosinophil Profile in Pathophysiology of Allergic Disorders

The presence of eosiniphils in the sputum was classically attributed to allergy, being mediated by eosinotactic factors and the importance of presence of eosinophils in the sputum in the differential diagnosis of asthma and obstructive chronic bronchitis has already been stressed. However, others opined that eosinophils might not be constantly present in the asthmatic sputum.

The correlation between peripheral eosinophil count and bronchial asthma had also been stressed by earlier workers. Eosinophils normally constitute a small proportion of the circulating leukocytes with normal values ranging from about 350/mm^3 and a count exceeding 350/mm^3 indicated a clinically significant elevation of total eosinophil count and associated with airway obstruction. However, there might be patients with bronchial asthma without eosinophilia as a result of unknown mechanisms (false negative results). Honsinger et al. (1972) suggested that, although eosinophil was known to be associated with allergic disease, its precise function is still not well understood.

It had already been suggested that allergic reactions in the target tissues caused the liberation of various pharmacologically active chemical mediators including the eosinophil chemotactic factor of anaphylaxis (ECF-A), which leads to the congregation of eosinophil leucocytes in the local sites and these chemo-attractants potentially would attract eosinophils to the allergic reaction sites and explained their presence in asthmatic lung tissue. It is stated that eosinophil leukocytes, derived from the blood stream were responsible for the eosinipenia, which ultimately stimulate bone marrow with consequent eosinopoiesis. Such an increased production of eosiniphils, being in excess of demand, might flood the blood, resulting eosinophilia in the blood. However, mechanisms causing blood eosinophilia still remained obscure.

7.4 Estimation of Total Serum IgE Level and Detection of Allergen Specific IgE Antibodies

IgE was considered as the prime carrier of reaginic hypersensitivity in humans and serum IgE levels were elevated in patients with allergic asthma. IgE levels were elevated (above 300 IU/ml) in allergic patients and the mean value was significantly higher in comparison to control subjects ($p \ll 0.001$), although there is a wide range of variations. The basis of variations in IgE levels was polygenic along with the possible effect of environment and socio-economic factors. It is further suggested that the immunogenic response of individuals exposed to similar environment might not be similar and the effect of socio-economic factors on total serum IgE levels should not be totally bypassed. However, in some allergic asthmatic subjects, circulating serum IgE levels remained within normal limits.

Although many studies confirmed the existence of high IgE values in atopic disorders, a considerable overlap between the levels of total IgE in healthy subjects and allergic patients have also been reported. There may be some patients with typical signs and symptoms of allergic rhinitis, positive skin tests and RAST positive response, but with total IgE less than 100 IU/ml which is in accordance with the fact that normal IgE level may occur in allergic patients. The extremely low level of serum IgE in some patients might be explained on the basis of the findings that one fraction of IgG antibodies (IgG4) might mediate immediate hypersensitivity reactions in such atopic patients.

Though skin testing is an excellent screening test, demonstration of specific IgE antibodies to a defined allergen through Radioallergosorbent test is a definite proof of IgE mediated diseases. Hence, the determination of specific IgE provides important information regarding the therapy to be adopted. Since specific IgE determination is completely unaffected by the intake of antihistamines or steroids, chances of obtaining false negative results are almost negligible. Analysis of the RAST results against house dust and house dust mites in relation to daily time of onset of asthmatic attack, it was observed that the frequency of RAST positivity was higher against mites in patients whose asthmatic attack was triggered after midnight as compared to those whose asthmatic bouts developed early morning and at no particular time of the day which corroborated well with the findings observed through skin test results.

It had already been suggested that the more allergens the patient was allergic to, the higher the serum IgE value seemed to be. Subjects with multiple severe allergies contain higher levels of serum IgE than subjects with single allergen sensitivity. Similarly, there is a tendency to have more positive RAST scores with higher levels of serum IgE and the level correlated well ($p < 0.01$) with the number of allergens positive in RAST.

Of the two diagnostic methods employed for predicting immediate type of hypersensitivity, the RAST is more sensitive and efficient. The patient with extremely low sensitivity towards a particular allergen, might not be easily detected by skin prick test method, but could be recognised by RAST. However, in general a good degree of correlation existed between skin prick test and RAST results and the correlation between RAST and skin prick test is entirely allergen dependent.

Studies to correlate the mean serum IgE values with absolute blood eosinophil count indicated that the group mean serum IgE level is higher in case of patients with elevated (>500/mm^3) blood eosinophil count and a higher IgE levels is significantly associated with a peripheral count of 500/mm^3 or more. Thus it may be presumed that the presence of elevated total serum IgE level in combination with elevated absolute blood eosinophil count further confirmed the atopic status of patients.

In the late sixties, Maunsell et al. (1968), Miyamoto et al. (1968) and Voorhorst (1969) observed that the degree of house dust antigenicity was proportional to the concentration of pyroglyphids in the dust. Later, Bear and Wharton (1970) and Gabriel et al. (1982) also noted that the load of mites could determine specific

antibodies and positive cutaneous test response against *D. pteronyssinus* and *D. farinae* mites. Mulla and Medina (1980) in their study also reported that in cases where high population of acari prevailed, a high level of IgE was found in the allergic patients' serum.

On correlating the density of *D. farinae* mites/g of dust to total mean serum IgE levels, RAST and skin prick test results, the mean serum IgE levels are observed to be significantly higher in patients whose bed dust contained more than 100 *D. farinae/*g of dust ($p < 0.05$) in contrast to those whose bed dust contained less than 100 *D. farinae/*g of dust. The frequency of skin prick test and RAST positivity to mite allergen was also noted to be higher ($p < 0.05$) in the former group (Saha 1994). In conclusion, it may be stated that with an increase in mite density, a gradual increase in the frequency of positive RAST and skin prick test response along with a marked elevation in the total serum IgE concentration occurred. Thus one might be inclined to draw an inference that the total mean serum IgE and the frequency of RAST and skin prick test is directly proportional to the specific mite density.

Chapter 8
House Dust Mite Allergy—Indian Perspective

An increasing trend in the incidence of allergic diseases has been noticed from different corners of the globe, specially in the developing countries due to diverse factors (Ciftci et al. 2006). Available reports demonstrate that a particular group of mite present in the house dust is considered to be the major source of allergens that cause sensitization and development of allergic diseases like allergic rhinitis, hay fever, skin rash and in extreme cases bronchial asthma all around the world. A rise in the prevalence and incidence of nasobronchial allergic disorders has been recorded in the recent past in India too. However, data on the prevalence of allergic diseases in Indian context is still fragmentary, except few scattered information from different pockets. According to the earliest available information, the prevalence to be 2.78 % among middle aged urban population of Delhi (Viswanathan 1966). However, a more recent study among school children in Delhi reported asthma incidence to be 10–13 % (Chhabra et al. 1998). In a rough estimate, in India, 250 million people are suffering from one or more allergic manifestations. Frequent change in lifestyle, environmental factors and dietary habits are blamed for such an increased occurrence and frequent recurrences in India. The growing concern is the increase rate of incidence and prevalence of some allergic diseases, specially allergic asthma in a rapid pace and are compounded by acquisition of western lifestyle, including standard of indoor environment, diet, air pollution and intolerable psychological stress.

8.1 Diversity and Distribution of Mites in Indian House Dust

The diversity and distribution of house dust mite fauna was investigated in depth by several workers from time to time throughout India. The first report of house dust mites from India was of Krishna Rao and Channa Basavanna (1973), who recorded three species of pyroglyphid mites from house dust samples of Bangalore and believed that these mites played an important role in causing respiratory

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allergy and rhinitis. Subsequently, studies on the house dust mite fauna have been conducted in different parts of the country and the presence of a varying number of species of dust mites have been reported by different workers (Dixit and Mehta 1973; Gupta and Dutta Ray 1975; Dar and Gupta 1979; Krishna Rao et al. 1981; Maurya and Jamil 1980; Tripathi and Parikh 1983; Channa Basavanna et al. 1984; Tandon et al. 1988, 1990; Anand 1981; Kumar et al. 1988; Kumud et al. 1988). Dar and Gupta (1979) collected dust samples from houses of bronchial asthma patients from 19 states of India and identified 23 species of mites belonging to 18 families. From Karnataka, Krishna Rao et al. (1981) described 23 species of mites belonging to six families; 20 species of mites under six families and two orders have been reported by Channa Basavanna et al. (1984) from Bangalore; 27 mite species distributed over 21 genera and 10 families have been reported from Punjab and Himachal Pradesh by Kumar et al. (1988). Kumud et al. (1988) reported the presence of 17 species under 14 genera and 10 families from 200 dust samples collected from Haryana. From West Bengal, Tandon et al. (1988) identified 22 species of mites under 19 genera and 13 families from the dust samples of houses of asthmatic patients of Kolkata, West Bengal. Modak et al. (1995) in a comparative study reported the presence of 54 species from three different districts of West Bengal, namely 24 Parganas, Burdwan and Calcutta. Among them, maximum variety was isolated from Kolkata metropolis. Simultaneously, Lal et al. (1973) reported the presence of *D. farinae* mite in the house dust sample of patients suffering from bronchial asthma in Delhi and showed a high incidence of positive skin reaction to extracts of mites and house dust among the patients. Dixit and Mehta (1973) and Nayar et al. (1974) while conducting their studies in the industrial belts of Madhya Pradesh found that *D. pteronyssinus* was predominantly present in all the samples of house dust and established a good correlation with skin test reactions to the extract of house dust and *D. farinae* mites. Jamil and Maurya (1981) from Lucknow and Tripathi and Parikh (1983) from Bombay reported the abundance of *D. farinae* and incriminated it as the main cause of respiratory allergy. Valandiker and Channa Basavanna (1992) made faunistic studies on house dust mites in Karnataka and reported 11 species under eight genera and six families, while Lakshmi and Haq (1999) from Kerala reported 17 species under 13 genera and eight families. In Madras city, Kannan et al. (1996) conducted a survey on house dust mites in dust samples collected from mattresses of 172 houses and revealed that *Dermatophagoides pteronyssinus* was the predominant mite species identified (97.09 %), followed by other species of mites. Modak et al. (2003) published a paper on faunal diversity and habitat preference of house dust mites of West Bengal. A recent report was published by Chaudhury et al. (2005) in which they dealt with synanthropic mite fauna of Kolkata metropolis and its adjoining areas and reported 25 species of house dust mites belonging to 13 genera, six families and three orders. In the subsequent year, Poddar et al. (2006a) described a new species of *Grallacheles* De Leon (Acari: Cheyletidae) from bedroom floor dust in Kolkata, India. Pujari-Vandana et al. (2007) studied the role of environmental factors on house dust mites under different living conditions and also highlighted the seasonal occurrence of different mite species. Baring, West Bengal

(Saha 1993, 1997; Modak et al. 2003; Poddar et al. 2006b), Delhi (Dar et al. 1973; Dar and Gupta 1979) and Maharastra (Tripathi and Parikh 1983; Mahesh et al. 2005; Shah and Bapat 2006), very limited number of works on indoor dust mites survey has been carried out in other parts of India.

During last 25 years, the house dust mite fauna of West Bengal were studied by several workers (Tandon et al. 1988, 1990; Modak et al. 1987, 1991, 2000, 2003; Saha et al. 1989, 1995a, b, 1997; Saha 1993, 1994, 1997, 2002, 2005; Podder et al. 2005, 2006a, b, 2009a, b, 2010a, b; Chaudhury et al. 2005, 2011, 2012, 2013). A total of fourteen districts of West Bengal including Kolkata metropolis have been searched for mite fauna. A total of 68 species of mites belonging to 25 families under 44 genera and three orders namely, Astigmata, Prostigmata and Mesostigmata have been reported including a good number of unidentified cryptostigmatids. Among them six species appeared to be new to science while another 20 species were reported for the first time from Indian house dust samples. It is interesting to note that maximum number of mite species (47) were isolated form dust samples of Kolkata followed by Burdwan (40) and 24 Parganas (33) and least (6) from Birbhum district of West Bengal (Saha 2005). In the recent past, attempts have been made to investigate the variety and variability of dust mites in the floor and bed dust of atopic allergic patients and to correlate it with the severity of allergic diseases in selected patients from Assam by Sharma et al. (2009, 2011). They conducted a quantitative and qualitative survey on indoor dust mites at the residence of atopic allergic patients from four different districts of Assam. Six different genus of house dust mites were identified during the said survey. The dominant genus was *Dermatophagoides* followed by *Blomia, Acarus* and *Cheyletus*. Among *Dermatophagoides, D. farinae* followed by *D. pteronyssinus* was found to be the dominant species and *B. tropicalis* and *Dermatophagoides* spp. were found higher in the bed as compared to floor dust.

8.2 Isolation and Culture of House Dust Mites

In India, several workers performed experiments for successful culture of the mite species in the laboratory. Nayar et al. (1974) obtained satisfactory results on rat-food and Gaine's dog-food, but poor yield of *D. farinae* was noticed when mites were reared on dried yeast powder. Maurya et al. (1983) used two culture media for rearing *D. farinae:* (a) a mixture of 35 % rat-food powder with 35 % chicken-food and 30 % dried yeast and (b) the rat-food alone. These media yielded the highest number of living mites at the end of 13th week at 26 °C temperature and 13 % RH. At 25 °C temperature and 80 % RH, Dar and Gupta (1979) by using Gaine's dog-food mixed with 20 % yeast obtained good result.

According to Jamil and Maurya (1981), chicken-food was the best culture medium for *D. farinae,* the second and third in order of performance level are being the rat-food and Gaine's dog-food. Channa Basavanna et al. (1984) used a mixture of diet consisting of sprouted Bengal gram powder 50 %, dog-biscuit

powder 20 %, human skin scales 4 %, dry prawn powder 10 % and yeast 16 % by weight with a relative humidity of 75 %. According to them, this diet was quite good for *D. pteronyssinus* and claimed that there was no need to add human skin scales for culturing *D. farinae*.

In India, Channa Basavanna et al. (1984) introduced an improved method for isolation of mites from the house dust samples. This method was adopted from Oshima (1967) with little modification in which ether was replaced by kerosene oil and the authors sieved dust samples after washing them in kerosene oil. Further modification of this method was made by Modak et al. (1987). They sieved dry dust by using an electrically operated sieve-shaker having sieves of five different mesh sizes (2.36 mm, 1.00 mm, 500 μm, 75 μm and 45 μm) instead of one (500 μm). Dust collected on 75 μm sieve was processed using a mixture of kerosene oil and carbon tetra chloride (sp. gr. 1.5), instead of using pure carbon tetra chloride.

8.3 Differences in Mite Abundance Between Allergic and Non-allergic Homes

In India, Dixit and Mehta (1973) and Nayar et al. (1974) observed that numerically the mites in the houses of asthmatic and the control subjects were almost identical and the difference being statistically insignificant. In contrast, according to Maurya and Jamil (1980) the density of mites in the mattress and floor dust samples of allergic persons was significantly higher than samples recovered from the houses of non-allergic persons. Tripathi and Parikh (1983) found no difference in the mite contents of the houses of allergic and non-allergic persons, except *Dermatophagoides* which were present in greater number in the houses of allergic persons. Tandon et al. (1988) reported higher number of mites and *Dermatophagoides* spp. in the dust samples of houses belonging to asthmatic patients and the difference was statistically significant.

8.4 Habitat Preference of House Dust Mites

Dominance of *Dermatophagoides, Blomia,* and *Cheyletus* in dust samples are well reported from different parts of the world (Binotti et al. 2001) including India (Shah and Bapat 2006). Mites prefer warm, moist surroundings such as the inside of a mattress, occupied by asthmatic individual for most of their daily activities and shed about 1/5 oz of dander (dead skin) each week. Dander being the favorite mite's food (both human and animal skin flakes), thus ensure food source. Besides, bedroom carpeting and household upholstery support high mite populations. The number of allergenic mites in the bed and floor dust of the patients is well correlated with intensity of the skin test reactions (Jogdand 1987).

Podder et al. (2010a) was of the opinion that, although mites can exist in different niches in a particular house, their density depends on some basic ecological requirements, both physical and biological. The physical factors like light, temperature, relative humidity, mechanical disturbances and biological factors like predation or parasitism, inter- and intraspecific competition along with sufficient and right food are the prime requisite for the successful growth and multiplication of mite population. A more recent study (Gill and Kaur 2013) suggested that dust mites are commonly found in carpets and mattresses in houses and more mites were found in carpeted living rooms, when compared to non-carpeted living rooms. Chaudhury et al. (2013) was of the opinion that the bed dust contained significantly higher mite population ($p < 0.01$) than the corresponding bedroom floor dust and the density of total mites and glycyphagids were significantly higher in rural houses in comparison to those of urban houses. In contrast, rural houses contained least mite number of pyroglyphids/g of dust. They also reported that the density of total mites as well as pyroglyphids/g of dust was higher in mud house in comparisons to concrete house, as well as, the density of total mites, pyroglyphids and glycyphagids are higher in cotton mattress in comparisons to that of foam mattress. However, Modak and Saha (2002) showed that density of total mites as well as pyroglyphid mites/g of dust were significantly higher in houses of middle income group people in comparison to those of high and low income group. In a more recent study, Podder et al. (2010a) while comparing the mite densities in mud and concrete house dust samples reported that the mite densities are higher in the mud houses than in the concrete houses. This study also agrees with the earlier findings of Mumcuoglu et al. (1999) and Macan et al. (2003), who also observed that a correlation exists between the mite numbers and climatic condition in the settlement. According to them the number of mites is higher in the damp environment than dry one. The mud house contains higher mite density than concrete house which may be due to the availability of proper microclimate like relative humidity and temperature for the multiplication of mite species in the former than the later (Bronswijk 1973). Beside, the use of the vacuum cleaner for dusting the floor is almost absent in the mud houses. On the other hand, it is frequently used by the inhabitants of the concrete houses. This ensures a stable food supply in the former condition. Otani et al. (1984) reported that the density of mite population was significantly higher in old and damp houses and they were much more abundant in mattress dust than anywhere else in the house. Nayar et al. (1974) and Maurya and Jamil (1980) from India also observed that old and damp houses contained higher densities of mites than the new houses. Modak and Saha (2002) from Kolkata, India also recorded that foam mattresses contained higher mite densities than those of cotton mattresses. However, Kannan et al. (1996) from Madras city, India, demonstrated that the mite density was higher in cotton mattresses than that of the foam mattresses. In a survey of homes with dust mites in and around Kolkata city, 60 % of the dust mite population is reported from the bed, mattresses, and pillows, 30 % in upholstery, and 10 % in carpet (Modak et al. 1991). Concentration and varieties of mites in the dust samples collected from selected patients house were found to vary due to the difference in the structure

and materials of the buildings, socioeconomic status of the individuals, type of mattresses used, standard of hygiene maintained, and difference in the microclimatic conditions that contributes to the higher accumulation of mites in the house dust (Modak et al. 1987). The distribution of mites on the basis of housing pattern indicates that RCC type of buildings supports maximum dust mite population followed by Assam type (semi-RCC) buildings, and the lowest count was observed in wooden houses as pointed from Assam by Sharma et al. (2011). Environmental factors like temperature, rainfall, and relative humidity are found to determine the diversity and abundance of indoor mite population. Similarly, jute bags used as mat in bamboo and wooden houses in most of the Indian villages might facilitate higher dust mites growth.

8.5 Seasonal Population Structure of House Dust Mite

In India, the seasonal abundance of house dust mites is yet to be studied in a comprehensive manner. Shivpuri (1962, 1981) was the first Indian scientist to conduct extensive and intensive studies on mites and recorded that mites could grow well in house dust at 25 °C temperature and 80 % relative humidity (Shivpuri 1962, 1981; Shivpuri and Dua 1974). However, notable contributions in this area came from Dar et al. (1974) and Dar and Gupta (1979). According to them, the mite density reached maximum in the months of August and September while it was minimum in March–May. From West Bengal, Banerjee (1988) reported that the mite population was maximum in July and August (monsoon) and minimum in May (summer). However, Datta and Joy (1987) from West Bengal in a more detail study claimed that maximum number of *D. farinae* was observed during winter in buildings and during rainy season in cottages. According to Kumar et al. (1988), the mites were most abundant in warm season (July–October) and their density declined in hot season (March–June) and cold season (November–February). Ranganath and Channa Basavanna (1988) observed maximum mite population in December and minimum in March–July. On analyzing the seasonal variation of mite density from Madras, Kannan (1996) reported that the mite density was high during the rainy season and low during the summer. In a more recent study from West Bengal, Podder et al. (2009b) reported that the density of all types of mites in bed and bedroom floor dust were maximum in autumn (September–November) and lowest in monsoon (June–August). Similar trend was also observed in case of pyroglyphids but only in bed dust sample. However, in bedroom floor dust samples, the density of pyroglyphids was observed to be maximum in summer (March–May) and minimum in monsoon (June–August).

From Assam, Sharma et al. (2011) while conducting seasonal studies on the distribution of dust mites showed maximum concentration between June and August, a time when the atmospheric temperature, relative humidity and rainfall were maximum, and it accounts for 37.7 % of total mite population. *Acarus* spp. was found maximum during the month of August followed by

May, whereas, *Blomia* spp. was maximum during the month of July followed by June and March respectively. Similarly, *Dermatophagoides* spp. was found maximum during the month of July followed by March. Least population was recorded from November to February, that is, the winter season. Indoor dust mite counts showed significant correlation with the monthly mean rainfall and monthly mean maximum temperature, while insignificant positive correlation with relative humidity was observed. Higher mite population is encountered during the summer and early autumn months from Assam by Sharma et al. (2011). Andrews et al. (1980) observed that mite positive patients had aggravation of respiratory symptoms in the colder months when mites were dead and had disintegrated to dust and had no seasonal changes in symptoms. That is why, Colloff and Spieksma (1992) pointed out that mite population are generally recorded maximum during the rainy seasons, but their effects are found maximum during the dry and cooler months of the year. Physical interventions (namely, steam cleaning plus vacuuming and vacuuming alone) offer practical, effective means of reducing house dust mite allergen levels in low-income, urban home environments (Hofter 2006).

8.6 Dust Mite—A Potent Allergen of Indian House Dust

Extensive clinical research to identify the offending allergen(s) playing a positive role in the aetiology of asthma and allergic rhinitis have been carried out since long back in India (Shivpuri and Agarwal 1969; Shivpuri et al. 1977). On the basis of the outcome of their research works, they have claimed that the mites are perhaps the sole potent allergen responsible for precipitation of attack of bronchial asthma. Since then, the studies on house dust mite fauna and their probable role in aetiopathogenesis of bronchial asthma have been reported from different parts of India (Dixit and Mehta 1973; Lal et al. 1973; Nayar et al. 1974; Dar and Gupta 1979; Jamil and Maurya 1981; Channa Basavanna et al. 1984; Roy et al. 1987; Tandon et al. 1988, 1990; Modak et al. 1987, 1991, 2000, 2003; Saha et al. 1989, 1995a, b, 1997; Saha 1993, 1994, 1997, 2002; Podder et al. 2005, 2006a, b, 2009a, b and 2010a, b; Chaudhury 2005, 2011, 2012, 2013). There was also on record analysis of prick skin test in patients of nasobronchial allergy with common offending allergens (Lal et al. 1973; Shivpuri et al. 1977). Shivpuri et al. (1977) reported the clinical significance of skin reactions to house dust and house dust mite extracts on patients with bronchial asthma. In an investigation, the number of allergenic mites in the bed and floor dust of the patients was well correlated with intensity of the skin test reactions (Jogdand 1987). The importance of skin tests as the standard method for demonstrating the presence of skin sensitizing antibodies was established by Roy et al. (1987). Data available on allergy tests on Indian samples collected from 650 collection centres across India shows that more than 80 % people tested were allergic to dust mites. Sundaram et al. (1980) showed that prick test with *D. pteronyssinus* extract was positive in 77.7 % of the patients. He also observed that majority of patients had a history of asthma attack

occurring after midnight and 77.3 % of such patients produced positive skin reactions with the house dust mite extracts. Kathuria et al. (1992) while identifying the clinical importance of pollens, fungi and house dust and house dust mites in nasobronchial allergy on North Indian population opined that house dust mites gave highest number of positive reactions compared to any other allergens. According to Mahesh et al. (2005), among patients with chronic urticaria, 64 % (78/122) had skin sensitivity to house dust mites as evidenced from prick skin test results suggesting possible association of house dust mite sensitivity with chronic urticaria. Those with the most common form of eczema—atopic eczema closely linked to asthma—are also affected by the tiny mites. In the recent past a study on patients with atopic dermatitis and chronic urticaria in Kolkata showed that among various aeroallergens tested, house dust mite (HDM), pollens, *Aspergillus fumigatus* and insects were closely linked to asthma and were found to be most commonly positive (Dhar and Banerjee 2010). Simplício et al. (2007) also reported *Dermatophagoides farinae* (Der f1) as major allergens in bedding dust samples and stated that Der f1 represents an important risk factor for exacerbation of allergic symptoms in previously mite-sensitized guests.

Allergy triggers in India are quite different from the west. While dust mites cause 80 % of all allergies in India, dust is the second common allergen in the US and Europe, where pollen allergy accounts for 20 % of allergies. The allergenic proteins responsible for causing symptoms are contained not only within the mites themselves but also in their shed skins and especially in their excreta. In a rough estimate, a single dust mite can produce up to 20 fecal pellets per day, and it may produce approximately 2000 fecal pellets during its active lifetime of up to 3 or 4 months (Hofter 2006). Thus capable of producing excreta about 200 times of their body weight and a typical used mattress may have up to 10 million mites inside (Lyon 1991). Routine human activity such as house cleaning and dusting, walking or playing on carpet, or making the bed, causes the tiny dead/live mites or their fecal particles to become airborne and inhaled. According to The British Allergy Foundation, house dust mites are one of the three most common triggers for allergies and up to 85 % of people with asthma are affected by the droppings of house dust mites. Allergen load of mites collected from the dust samples were found directly proportional to the severity of allergic attack in some cases (Andrews et al. 1980). In the recent past, scientists from Banaras Hindu University, India opined that house dust mites are fast emerging as most notorious indoor allergen (Agarwal 2009) and it takes 100 mites per gram of dust to produce sensitivity and 500 mites per gram of dust to produce asthma attack. He further added that around 15 % rise in asthma cases amongst children is witnessed every year in India and indoor allergens play active role in it. A number of household articles including carpets, curtains, sofa cover, mattresses and soft toys (fur-type) seem to attract these mites and initiate allergic response. Besides, fast food especially junk food were also contributing to the steady rise in the problems in children. In an another report, Dr. Paramesh (2009), President of the Indian Academy of Allergy said that “At least half of the asthma attacks are caused by house-dust mites, a problem to which very little attention has been given and today, indoor

pollution is where the danger is and dust-mite-related pollution is one of the major causes of allergy." According to him, about 100 mites per gram of dust are enough to set off your allergies and 500 dust mites per gram are enough to cause wheezing. However, studies have found that an average Indian household furnished with carpets, curtain drapes, indoor plants, even stuffed toys, harbour about 4000 dust mites/g of dust. When children are exposed to dust mite allergies at an early age, life-long asthma can be triggered. A study carried out to find the allergy pattern in Mumbai shows house dust mite was the cause of allergies in 43 % of patients in Mumbai owing to the hot and humid climate and 50 % of children diagnosed with allergies and allergies precede or coincide with asthma in approximately 80 % children and 60 % adults. Most of the dust mites die when exposed to low humidity levels or extreme temperature, but they leave their waste behind, which continues to cause allergic reactions. In a worm humid house, dust mites can survive easily year round.

Estimation of total serum IgE level and detection of specific IgE antibodies against offending allergen, by Radio Immuno Assay (RIA), as an alternative and confirmative method for diagnosis of extrinsic allergy had been reported by Lal et al. (1973). They estimated total and specific serum IgE concentration in patients of bronchial asthma in relation to house dust and house dust mites. Estimation of serum immunoglobulins including IgE in the sera and bronchial aspirates of children and adults with wheezy bronchitis, bronchial asthma and pulmonary eosinophilia was made by different workers (Kumar et al. 1971; Batabyal et al. 1986; Khatua et al. 1986, 1987; Saha et al. 1989; Saha 1993, 1995a, b).

Though specific serum IgE antibodies against the offending allergens (pollen, *Aspergillus* and house dust) in patient of bronchial asthma had been estimated (Batabyal et al. 1986; Khatua et al. 1986; Roy et al. 1987), information with regard to the level of specific serum IgE antibodies against house dust, *D. pteronysinus* and *D. farinae* mite antigens was lacking for quite a long time. Batabyal et al. (1986) however, presented some data on the diagnosis of allergic disorders involving skin testing, analysis of the serum for total IgE and allergen specific IgE antibodies against pollen grains and total house dust. Khatua et al. (1986) made an immunological study of bronchial asthma in children between 1 and 5 years of age, wherein a relation between asthma with total and specific IgE and the common allergens (mostly pollens) responsible to precipitate the attack was made. Shah and Bapat (2006) worked on in utero exposure to house dust mites (Der P1 and Blo t5) and both the allergens were measured in matched maternal and umbilical cord blood samples by EIA method. The allergens were detectable in 50 % matched maternal and umbilical cord blood, which suggest that Der P1 and Blo t5 allergens can cross the placenta to reach the umbilical cord blood and may initiate sensitization in utero.

Saha (1993, 1995a, b), Saha and associates (1994, 1997) and Podder et al. (2006a, b) have carried out extensive research on different aspects of dust mite allergy in Kolkata, India including entomological, clinical and immunological parameters in a comprehensive manner since more than twenty five years and established the role of house dust mites in house dust allergy.

8.7 House Dust Mite Allergy in Kolkata—A Case Study

The incidence of nasobronchial allergic disorders due to dust inhalation is on increasing trend in Kolkata and its adjoining areas, but no authentic up to date information on its incidence is available. Serious attempt to identify the causative agents in the dust responsible for nasobronchial allergic disorders and bronchial asthma in individuals susceptible to dust inhalation in Kolkata has not been made so far. As mentioned earlier, mites belonging to the genus *Dermatophagoides* (fam. *Pyroglyphidae*) have been reported to be the most potent source of allergen in house dust globally. Though some valuable information in the relevant field is available from other parts of the country, little attention has been paid to this subject in West Bengal. It may be worthnoting that no systematic and comprehensive study implicating *Darmatophagoides* mites as the aetiopathological agents of naosbronchial allergic disorders has so far been made in West Bengal. Lack of information on the immunological aspects of house dust mite sensitive patients is also badly felt.

In the aforesaid backdrop, a comprehensive study was undertaken to elucidate the role of house dust mites (*Dermatophagoides* spp.) in the precipitation of bronchial asthma and to assess the immunological profile of such patients residing in and around Kolkata.

The notable findings are

- A total of 54 species belonging to 34 genera and 13 families under 3 orders have been identified.
- All the reported allergenic mites namely *Dermatophagoides pteronyssinus, D. farinae, Austroglucyphagus geniculatus,* Blomia tropicalis*,* Acarus siro*,* Glycyphagus domesticus*,* Euroglyphus mayenei*., Tyrophagus putrescentiae were isolated.*
- *Dermatophagoides pteronyssinus* was found to be the most predominant mite species in all the dust samples screened, comprising 47 % of total acarine fauna.
- Species diversity Index indicates that the maximum number of species are found in pre-monsoon period and minimum in winter both in bed dust and corresponding bedroom floor dust.
- Seasonal fluctuation of mites in bed dust and corresponding bed room floor dust reveals that the number of total mites as well as four important allergenic mites, namely *D. pteronyssinus, D. farinae,* Blomia tropicalis *and Austroglycyphagus geniculatus* are higher in pre-monsoon period and lower in winter period.
- A positive correlation between the number of mites with prevailing room temperature and relative humidity was also observed.
- Patients bed contains higher mite population than the corresponding bed-room floor dust except *D. farinae*.
- The number of mites in mud house is significantly higher than concrete house ($p < 0.05$).
- Foam mattress contains significantly higher mite population than the traditional cotton mattress ($p < 0.005$).

- The life cycle of *D. pteronyssinus* (egg to egg) is completed in 9–14 days under laboratory conditions at 25 °C temperature and 80 % relative humidity.
- The average duration of different life stages are as follows: larva-1.57 days, protonymph-2.64 days, tritonymph-2.46 days and egg to adult-11.26 days.
- Mortality rate is highest in larval stage and as the development progressed, the mortality rate decreases.
- The rate of fecundity of *D. pteronyssinus* females ranges between 1.3 and 6.4/day.
- Adult mites live about 5–6 weeks in laboratory conditions, females lived longer than males.
- Skin prick test was carried out against 5 pollens, 5 molds and 8 other allergens including house dust and three species of mites such as *Dermatophagoides pteronyssinus*, *D. farinae* and *Blomia tropicalis*.
- Results of skin prick test reveals that more than 72 % patients are sensitive to pollens and 22 % to molds.
- The sensitivity towards house dust and house dust mites are 96 % and more than 75 % patients are sensitive to *Dermatophagoides pteronyssinus* mite, 72 % to *Blomia tropicalis* 63 % to *D. farinae* and mite allergens.
- The sensitivity to *Blomia tropicalis* mite is new report in Indian population.
- Results of skin prick test between two sexes reveals that the frequency of positive skin reaction does not differ significantly between male and female.
- Difference in density of *Dermatophagoides* mites and *Blomia tropicalis* mite population between patient and control subject bedroom dust is statistically significant ($p < 0.0005$).
- The difference between patients and control mean serum IgE level is statistically significant ($p < 0.05$). 93.5 % patients are with elevated (300 IU/ml) serum IgE level.
- The mean serum IgE level neither depends on age nor sex.
- Identification of allergen specific IgE antibodies by Immuno CAP System shows that 79.77 % patients respond to HD allergen, 71.91 % to DP and 88.76 % to DF allergens while 90 % patients respond to BT allergen which further confirms the result of skin prick test. The sensitization to this *B. tropicalis* mite has been established in different parts of the world (Arlian et al. 1993; Simpson et al. 2003; Flores et al. 2003).

Chapter 9
Allergy and Heredity—Genetic Links

It has been proved that the entire immune system and the production of antibodies are governed by some genetic factors and heredity plays an important role in the development of allergic manifestations. The tendency to produce specific IgE antibodies and susceptibility of the organ to the allergic disease is governed by family history and genetical make up of the individual. Most family studies report a positive family history in approximately 50 to 75 % of the cases (David et al. 1980). If any of the parents has allergies of any type, there is chance of developing allergy among children at least one in three. If both parents are allergic, the chances are more (7 in 10). Allergic diseases are strongly familial, identical twins are likely to have the same allergic diseases about 70 % of the time; the same allergy occurs about 40 % of the time in non-identical twins. Allergic parents are more likely to have allergic children and their allergies are likely to be more severe than those from non-allergic parents.

Kjellman (1977) proved that a child with atopic parents is six times more susceptible to allergies than the children with no parental history of atopic disorders. Atopy is very high when both the parents have suffered from same disease, affecting same organ such as skin, respiratory tract, etc. The following table shows the chance of occurrence of atopy in children from parents with different degrees of atopy as observed by Kjellman (1977).

Sl	Atopy in parents	Frequency of atopy in children (%)
1	No atopy	12.5
2	One parent atopic	19.8
3	Both parents atopic	42.9
4	Both atopic with same manifestation	72.2

Although there is no doubt that the capacity to become allergic is an inherited tendency, most likely polygenic in nature and a number of factors are responsible to initiate and favours the allergic response (Ono 2000). These include presence of specific genes acquired from the parents, exposure to the offending allergen(s) and degree and length of exposure. The nature of inheritance is polygenic and unlike

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many other diseases, fifty different genes are involved. Thus, instead of single effecter gene, phenotypic expression of allergic diseases occurs through complex interaction with other genes and environmental risk factors (Ono 2000). However, studies of a possible relationship of geographic and racial factors in the occurrence of common allergic diseases suggest that environmental factors are more significant than racial and genetic factors. Recent studies identified the gene CHI 3L1 having a portion called 131C or 131G associated with asthma. The kids who inherit two copies of the faulty 131C variation have twice the chance of developing asthma than those without it. On the contrary, children who inherit one or two copies of the faulty 131G variation have half the risk of the disease (Ober 1998).

Chapter 10
Allergic Manifestations and Their Diagnosis

10.1 Manifestations

The common allergic manifestations include allergic rhinitis, hay fever, atopic dermatitis, urticaria, atopic eczema, allergic rhinoconjunctivitis and in extreme cases bronchial asthma. All these complaints may occur either singly or in combination with each other.

Asthma

Asthma is an expression of immediate hypersensitive reaction in the lung, characterized by difficulties in breathing and wheezing. Asthma, being a chronic debilitating disease, effects the socio-economic pattern of the diseased individual. According to available literature, it is defined as "wide spread bronchoconstriction, resolving either spontaneously or as a response to bronchodilator therapy, manifesting as paroxysms of wheeziness, dyspnoea and cough in an atopic individual". In physiologic terms it is also defined as a "reversible obstructive airway disease (ROAD)". Respiratory allergic disorders occur predominantly in early life and are more common among older children and adults. During childhood, asthma is about 30 % more common in males than in females and the disease tends to be more severe in male children.

Common symptoms: Airway obstruction, breathing becomes difficult, specially expiration, chest congestion accompanied by wheezy dyspnoea and troublesome cough.

Types of asthma

For better understanding of the disease pathogenesis, differential diagnosis of the disease and to provide best possible treatment regime, a detailed and confirmed information regarding the type of asthma is of prime importance. In fact, the heterogeneity of asthma makes its classification a difficult proposition. However, Lawlor and Tashkin (1981) classify asthma into intrinsic, extrinsic and mixed type based on etiologic factors.

- **Intrinsic**: Non-allergic type, in such cases symptoms are unrelated to allergen exposure and are provoked or aggravated by infection, exhaustion, stress,

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psychological stimuli and specific climatic or environmental changes; more common in adults. The cause may be an irritation to the nerves or muscles in the airway. This type represents about 10 % of all cases and usually develops after the age of thirty. Women are generally more affected, difficult to treat and symptoms are often chronic and occurs year-round.

- **Extrinsic**: Allergic type where asthma initiates due to immunological response to external inhaled agents like pollens, moulds, animal danders, dust mites etc. This type is more common (90 % of all cases) and typically develops in childhood.
- **Mixed**: Is a mixture of both extrinsic and intrinsic type.

Categories of Asthma
Asthma has further been categorized into following types:

Nocturnal—When asthmatic attack worsens at midnight and should be taken seriously, otherwise it often leads to respiratory arrest and ultimately death.
Exercise induced—Noticed during or shortly after exercise, characterized by wheezy dyspnoea and short of breath.
Occupational—Develops after starting a new job and interestingly symptoms usually subsides during holidays and when out of work.
Seasonal—Prevalent during flowering or pollen season and change in weather.
Cough variant—Accompanied with persistent and nagging cough.
Silent—Without giving any prior intimation or warning, with less wheezing, usually with severe asthmatic bouts.

Allergic rhinitis or Hay fever
Common symptoms: Lining of nose swollen; runny discharge along with spells of sneezing; itchiness of throat and palate. In addition to exerts its own damaging activities, allergic rhinitis very often leads to the most difficult to treat stage of allergic manifestation termed as allergic asthma. This phenomenon is popularly known as "**Allergic March**" (Bousquet et al. 2003). It is estimated that a children with allergic rhinitis and a positive history of atopy, the risk of development of allergic asthma is increased from 20 to 60 % (Corren 2000) and when accompanied by atopic dermatitis and food allergy the chances may increased up to 80 % (Eichenfield et al. 2003).

Eczema
Common symptoms: Severe skin irritation, develop rashes which may be wet, dry or chapped, followed by unbearable itching; usually on elbow, backs of knees, neck, ankles, wrists, back of hands etc.

Urticaria
Common symptoms: Appears suddenly; skin becomes reddish and warm with intense itching which lasts for a while.

Conjunctivitis
Common symptoms: Redness, itchiness and swelling of the eye; eyes are rubbed frequently.

Anaphylactic Shock
It is considered to be the worst form of allergic reaction and mostly fatal. The condition typically occurs due to ingestion of some food stuffs or due to insect stinging. Notable symptoms include reddish discoloration of the skin, nasal congestion, swelling of the throat, stomach pain, nausea, vomiting, shortness of breath, low blood pressure or shock-characterised by insufficient blood circulation.

10.2 Diagnostic Procedures

Allergic diseases are diagnosed primarily on the basis of history and characteristic clinical observations and confirmed by definite laboratory tests. Effective management of allergic diseases require reliable identification of offending allergen and its subsequent avoidance by the patient. For this reason, a detailed case history of the patient including the history of atopy is of prime importance. Sometimes, but certainly not always, it is unambiguous. In certain cases, the case history can be difficult to interpret or directly misleading. Clinical diagnosis involves skin testing, analysis of serum for total IgE and allergen specific IgE antibodies and different types of challenge tests. The diagnosis must be accurate. Doubtful test results should not be accepted. At the same time the diagnostic techniques should not put the patient at risk.

10.2.1 Documentation of Case History and Family History

A detailed case history of the patients including personal and family history of atopy, present clinical condition, age at onset of the symptoms, lifestyle at home and work place, house condition etc., should be maintained in a well prepared proforma. The pertinent history also includes the presence of pets, seasonality of symptoms, history of specific exposure, recurrence of symptoms etc.

10.2.2 Physical Examinations

Thorough examinations of different target organs like eyes, nose, chest, ears, throat and skin has to be made. At the same time X-ray of chest and sinuses and pulmonary function test must be done to confirm airway obstructions.

10.2.3 Allergen Provocation Test or Bronchial Challenge Test

Although it was of great use in earlier days, the method is now rarely used for clinical diagnosis as the method is tedious, time consuming and high degree of risk involvement.

10.2.4 Skin Prick Test as Basic Screening Procedure

The direct introduction of an antigen into the skin of a patient could provide a simple and efficient technique for determining IgE antibodies to specific antigens. This diagnostic technique was introduced in 1873 as a practical clinical tool by C.H. Blackley, who showed that patients suffering from hay fever reacted by forming urticarial wheals after scratch tests with pollen. Since then, the association between allergic symptoms and immediate skin test reactivity had been confirmed in several studies (Hogarth-Scott et al. 1973; Roy et al. 1987; Saha et al. 1995 a, b) and thus allergy skin test became a routine clinical procedure. Tuft and Heck (1974) regarded skin test as the major and most commonly used diagnostic tool in clinical practice of allergy. Skin test detects reagins fixed to the mast cells in the skin and offers an important tool for allergy diagnosis. It can be performed either as a prick or scratch test or as an intracutaneous test. Among them, prick testing is most accepted procedure at present to be recommended both for children and adults. It is highly specific from an immunological point of view and involves minimal risk. It is easy to perform and standardize and gives little pain to the patient. The results are taken twenty minutes after testing and graded as 1^+, 2^+, 3^+, 4^+ or negative on the basis of intensity of reaction and erythema formation as compared to positive (histamine) and negative (normal saline) control as suggested by Grater et al. (1982). Although, intracutaneous testing is approximately 10–100 times more sensitive than prick testing, more often, it gives non-specific reactions and involves risk of anaphylactic shocks and is also painful for the patients.

Grater et al. (1982) preferred skin test because of its inexpensiveness, simplicity to perform and capacity to produce instant results. However, the results were reportedly influenced by drugs and by the patient's responsiveness to mediators. One of the greatest difficulties encountered in making skin tests precise, specific and reproducible was the lack of sufficiently potent, stable and well standardised allergen extracts of many important allergens (Aas and Belin 1972).

As stated earlier children could develop an increasing capacity to elicit skin sensitizing antibodies with increasing age and exposure. In his study on the relation of skin reactivity to age and sex of the patients, Pearson (1937) noted that a spontaneous desensitization occurred with advancing age, but there was no difference in the prevalence of skin reactivity between the two sexes. In contrast, Lindbladd and Farr (1961) conceived no relation between age and occurrence of skin reactivity of an individual, and in their opinion, the decline in reactivity which occurred in older subjects, may be the result of a true decline in immunological reactivity or a decrease in the capacity of the skin to respond to immunological challenge. However, the postulate of skin reactivity increasing with age was also suggested by Hannaway and Hyde (1970).

The first comprehensive study on the prevalence of immediate skin test reactivity in the general population in relation to age and sex was made by Barbee et al. (1976). Subsequently, they described the relationship between respiratory allergy, skin test reactivity and serum IgE level. The distribution of IgE in relation

to age, sex and allergen skin reactivity was also evaluated by Barbee et al. (1981). No significant difference in skin test reaction rates were noted between male and female subjects. The reaction rates were highly age dependent, for each of the individual allergen and also for total reaction. The reaction rate increased through the teens, reaching a peak of 52 % in the first half of the third decade of the age. Subsequently, the rate dropped slowly until the attainment of 50 years and thereafter it dropped rapidly till 60, reaching the minimum (16 %) in subjects older than 75 years of age. According to them, maximum reactivity occurred during the young adulthood.

Lawrence (1982) from Jamaica reported that neither sex nor age of the patients' had any consistent relationship to the groups of allergens producing positive skin reactions. According to Chan Yeung et al. (1985) the prevalence of atopy was highly age dependent and confirmed the findings of Barbee et al. (1976) that sex had no influence on the prevalence of atopy.

10.2.5 Role of Peripheral Blood and Sputum Eosinophil Count on Diagnosis

Eosinophil is prime mediator of the pathophysiology of bronchial asthma (Holgate 2008). Eosinophilia in association with reversible obstructive airway diseases was first documented by Ellis (1908). Huber and Koessler (1922) recorded the massive blood and tissue eosinophilia in the lungs of patients dying of asthma and opined that the coincidence of sputum and blood eosinophilia in the same individual seemed to be a pathognomonic symptom of asthmatic state. Lowell (1967) was of the opinion that reliance should be placed chiefly on the total eosinophil count rather than examination of sputum or nasal exudate because of either non availability or poor quality of the sputum and nasal samples. Subsequently, Middleton et al. (1978) claimed that the usefulness of blood or tissue eosinophilia in establishing an allergic aetiology for any manifestation of disease in an individual patient was marginal. Eosinophilia might occur in absence of any apparent illness and was sometimes congenital. On the contrary, there are patients with bronchial asthma without eosinophilia as a result of unknown mechanisms (false negative results), though the percentage of such cases is very low. Connell (1968) found that eosinophils were present in higher numbers at the site of an allergic reaction and in increased numbers in peripheral blood of some atopic individuals. Since then, the association between peripheral eosinophil count and bronchial asthma had been stressed by different workers (Honsinger et al. 1972; Horn et al. 1957). Honsinger et al. (1972) observed that the improvement in pulmonary function coincides with decreases in total eosinophil counts and higher IgE levels were also significantly associated with a peripheral eosinophil count of 500/mm^3 or more. Franklin (1974) stated that the measurement of total eosinophils was indeed useful in the management of difficult-to-treat asthma. According to Horn et al. (1957),

the relief of bronchospasm as a result of therapy was associated with concomitant fall in the blood eosinophil count.

Several workers suggested that different mediators (histamine, ECF-A) released as a result of antigen-antibody reaction are known to attract eosinophils to allergic reaction sites, however, the mechanism involved in blood eosinophilia is yet to be properly understood.

It was reported long back that the hallmark of the pathology of asthma was a massive infiltration of the lung parenchyma by eosinophils and destruction of mucociliary apparatus. Brown (1958) suggested the presence of large number of eosinophil leukocytes and sometimes Charcot-Leyden crystals as the characteristic of the sputum of asthmatic patients. Later, several authors suggested that the presence of eosinotactic factors was classically attributed to allergy (Kay et al. 1978). In fact, anything more than the occasional eosinophil in the sputum would promote a search for an allergic causes and possible airway obstruction as suggested by Keil (1983). On the basis of bronchoalveolar lavage fluid analyses among asthmatic patients, Metzger et al. (1985) reported a predominant increase in eosinophilic and neutrophilic granulocytes. Raynolds and Newball (1974) pointed out that in the bronchial passage of normal individuals eosinophil cells were not present and different chemoattractants such as SRS-A, ECF-A, NCF-A and PAF-A, potentially would attract blood leukocytes and explain their presence in asthmatic lung tissue. In contrast, Dulfano (1973) was of the opinion that eosinophils might not be constantly present in asthmatic sputum and several specimens should be examined as they might decrease or disappear at the beginning of an allergic episode.

10.2.6 Quantification of Total Serum IgE Level

The quantity of IgE normally present in serum is insufficient to produce visible precipitin reactions in gel. The problem of detection and quantification of IgE levels in serum was resolved by the adoption of Radio Immuno Assay Technique (RIA) with the help of radio-labelled reagents. The initial method "RIST" was introduced by Johansson et al. (1968, 1972). Subsequently the double antibody RIA method was introduced by Gleich et al. (1971). Polmar et al. (1973) considered this method to be the most sensitive and satisfactory of all RIA procedures which could measure IgE concentration as low as 0.1 μg/ml. Recent studies revealed that measurement of total IgE is easier and less costly and also to be an accurate screening test for sensitization of common inhalants (Eriksson et al. 1985). To measure low level of IgE in serum sensitive RIA methods have been developed such as, Double Antibody technique, Radio Immuno Sorbent Test (RIST) and Paper Radio Immuno Sorbent Test (PRIST) as proposed by Kjellman et al. (1976). In Calcutta population, Saha et al. (1989) and Saha (1993) reported that 92 % asthma patients showed elevated serum IgE level (above 300 IU/ml) and the mean value was significantly higher in comparison to control subjects sera ($p \ll 0.001$).

10.2.7 Detection of Allergen Specific IgE Antibodies as Confirmative Test

RAST was introduced by Wide et al. (1967) as an in vitro method for detection of allergen specific IgE antibodies in allergic diseases. Besides, enzyme linked immunosorbent assays (ELISA), fluorescent allergosorbent test (FAST) and radioimmunosorbent test (RIST) are also used. Atopic individuals with allergen specific IgE antibodies were thought to develop a variety of symptoms when exposed to that allergen. According to Stenius and Wide (1969), till date, the RAST appeared to be the most widely used method to measure serum IgE antibodies to a variety of allergens including house dust and house dust mites. Identification of the specific allergen by RAST, helps the patient to avoid exposure to it or to obtain appropriate therapy. Close correlation had been established between results obtained from the RAST and clinical provocation tests (Berg et al. 1971). However, Aas and Johansson (1971) reported that specific IgE antibodies were not found in 25 % of those who showed positive bronchial response to specific allergen. The main obstacle in obtaining precise and reliable RAST results in all instances is perhaps the use of heterogenous allergenic extracts as suggested by Aas and Belin (1972) and were of the opinion that controversy regarding the appropriate use of RAST for in vitro diagnosis of IgE mediated disorders could not be resolved till date.

According to Pelikan and Pelikan (1982) introduction of the quantitative determination of specific IgE antibodies in serum by RAST and its wide practical use in the diagnosis of allergic diseases due to type I hypersensitivity could be regarded as one of the most important development in clinical allergy during the last decade. Recent studies revealed that the classical RAST method is now being replaced by the more sensitive and highly specific recent method introduced by Pharmacia Immuno CAP system (Kherkof et al. 2003). Further, introduction of the quantitative determination of specific IgE antibodies in serum by Pharmacia CAP System is world wide in use (William et al. 2000; Uchio et al. 2001).

10.2.8 Diagnostic Efficacy of Skin Test and RAST and Their Limitations

Following the introduction of RAST method, many studies were undertaken to compare the results of RAST with skin test (Norman et al. 1973). Appreciably good agreements (79–100 %) between these two diagnostic methods against several common allergens, including house dust mites was documented (Roy et al. 1987). Srivastava et al. (1983) screened the sera of asthmatic patients for allergen specific IgE against *D. farinae* mite by RAST and reported that patients positive to *D. farinae* by skin test had RAST scores of 3^+ and 4^+, while skin test negative patients and control subjects both had a RAST score of 0. However, correlation between in vivo and in vitro tests with different allergens was found to

be unsatisfactory by some researchers namely, Aas and Johansson (1971) and Hogarth-Scott et al. (1973). Aas and Johansson (1971) showed that the correlation between RAST and skin test was allergen dependent. However, Rappaport et al. (1979) suggested lack of correlation between the skin test and RAST was mainly due to a surprisingly large number of skin test negative/RAST positive disagreements.

The relative diagnostic efficacy of skin test and RAST remained controversial. Some investigators (Santrach et al. 1980) claimed equal diagnostic value of those two tests where as others (De Filippie et al. 1981; Fink 1985) believe that the skin test was superior to RAST. According to Aas and Belin (1972), the case history and allergy skin test might not be reliable in all instances, in such cases, the use of RAST could render additional information about the immunological specificity of the skin test and help in appropriate diagnosis. Though skin and RAST studies correlated variably with each other, the decision as to which test should be used would depend on the availability, cost, discomfort or risk to the patients (Herbert et al. 1982). Pascual et al. (1977) opined that RAST should be performed on patients with histories and physical findings consistent with atopic diseases even when the skin tests show negative results. Fink (1985) suggested that although it might not be as sensitive as the skin test, the RAST could be used when skin testing is not feasible. Roy et al. (1987) however, observed that each method had its own merits and demerits. Though detection of specific IgE antibodies in serum by RAST overcame some of the disadvantages of skin test, it required special equipment, radioactive chemicals and is highly expensive.

Chapter 11
Dust Mite Allergy Management—Common Practices

Dust mite allergens are associated with fine particles that tend to have a wide range of aerodynamic behavior, having the property to settle down within 15 min of disturbance. Almost no mite allergen is found in the air of undisturbed rooms. Generally, the mite allergens are found in the dust that settled down in the carpets, beddings, and upholstered furniture; clothing also appears to be an important source of mite allergen exposure, particularly if the clothing is not washed very frequently. Allergy is one of the major factors associated with the cause and persistence of allergic rhinitis, eczema and asthma. Identifying the allergens causing the symptoms is an essential part of treating allergic diseases.

Allergy is considered to be an escalating problem in developing countries and can be tackled through multidisciplinary approaches with broad and integrated management strategies. Though there are no cures for allergic manifestations, the symptoms, if identified at an early stage, can be reduced and managed through appropriate preventive measures and treatment. However, before starting any treatment schedule, following important aspects should be taken into consideration.

11.1 Allergen Avoidance

In order to prevent allergy, primarily two important attributes should be taken into consideration. Firstly, proper identification of offending allergen(s) to which the person is sensitive to and secondly, simple avoidance of such allergen or their subsequent reduction or elimination from the environment of the concerned subject (Platts-Mills 2003). For example, if a person shows allergic symptoms like swelling of lips and throats associated with other gastrointestinal problems after having shell fish or other similar food stuffs in their diet, it is wise and advisable to avoid such food in future rather than to apply any medication. However, in many instances there are many allergens such as dusts, mould spores, pollens etc. which cannot be avoided completely; in those cases minimizing the exposure to such allergens having great therapeutic benefit is advisable.

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11.2 Change in Lifestyle

Non-medical treatment is often as effective managing allergies as applying medications. Managing allergies may require a few changes in lifestyle in order to avoid the allergens. Changing lifestyles such as having a well balanced diet, avoidance of both active and passive smoking, regular physical work-out can improve their allergic manifestations.

11.3 Medication

Antihistamines and decongestants are the common medicines used to mitigate allergic manifestations. Antihistamines have been used for years to treat allergy symptoms. They can be taken as pills, liquid, nasal spray, or eye drops. Antihistamines are used against rashes, hives, sneezing, itching and runny nose. Eye drops containing antihistamine are used to reduce itching, tearing and swelling of eyes. The mode of action of antihistamines include blockage of histamine that is released during an allergic reaction. Decongestants are used for quick, temporary relief of nasal and sinus congestion and are often prescribed along with antihistamines for allergies. They can come in nasal spray, eye drop, liquid, or pill form. Decongestant tablets, sprays, and nasal drops are used to reduce stuffiness of the nose. Oral decongestants relieve nasal and sinus congestion caused by allergic rhinitis. Corticosteroids relieve symptoms by suppressing allergy-related inflammation. Corticosteroid nasal sprays are used to reduce inflammation inside the nose. Inhaled corticosteroids are often used every day as part of treatment for asthma caused or complicated by reactions to airborne allergens. Cromolyn Sodium prevents the inflammation that causes nasal congestion. Oral corticosteroids may be used with great care to avoid possible side effects. Epinephrine reduces the constriction of the blood vessels, decreases swelling and helps to increase blood pressure; it also increases the heart's contraction and heart rate, which can help to prevent or reverse cardiovascular collapse. Epinephrine relaxes the muscles around the airways in the lungs, helping the airways to open up. Epinephrine is the only drug to overcome life threatening anaphylactic shock. For therapeutic treatment of asthma, the use of bronchodilators such as sodium cromoglycate in the form of spray, tablet or injection are recommended. The sprays are commonly used to give immediate relief. Bronchodilators relax the muscle bands that tighten around the airways and rapidly open the airways, letting more air in and out of the lungs, thereby improving breathing. In case of severe asthma steroids may be used and should be regularly taken even when the allergy symptoms are not felt. Antihistamines are the mainstay of drug treatment for urticaria.

11.4 Immunotherapy or Desensitization

Allergy vaccine has now gained wide recommendation specially for hay fever, insect stings etc. It is functional through systemic administration of increasingly higher doses of prepared and purified offending allergen (Durham and Till 1998; Muller 2003). The process is to build up the dose of allergen given and then to continue with a dose which will cause the immune system to tolerate the substance. It has been shown to alter the Th1-Th2 cytokine imbalance that occurred due to allergic response. Besides, it exerts direct effect on eosinophils and mast cell and makes them inactive towards allergic response (Greenberger 2002). However, its effectiveness decreases against food, drug and feather allergy. Recent experimental evidences suggest desensitization with mite extracts can be successfully used to treat individuals sensitive to asthma caused by house dust mite allergens (Pichler et al. 2001; Frati et al. 2012).

11.5 Prevention

Though any specific and effective ways to check mite borne allergic diseases are lacking, the symptoms may be relieved or minimized and in best cases eliminated with the adoption of combination of suitable preventive as well as curative measures. If the patient be relieved of the allergic symptoms, the chances of increasing the tolerance with time improve and thus the risk of developing new allergies minimizes. However, the tendency towards spontaneous development of tolerance varies distinctly with age, type of symptoms and nature of allergens.

Some plain-simple practices and habits with a little modification and certain changes in lifestyle at residence, office, school, during outdoor activities and even during travel may prevent and reduce the allergic discomforts to a great extent. Frequent dusting, preferably wiping of surfaces with wet cloth and frequent washing of beds to check dust mite population gives better result. To avoid the possible risk of dusting and cleaning, wear mask during house work or use vacuum cleaner. Avoidance of direct contacts with the pets especially with feathers or fur such as birds, dogs, cats etc. may also give better results. Newer approaches include environmental management to minimize allergen exposure, stress management and therapeutic interventions. As the mould spores grow well in the moist areas, it is highly recommended to reduce the moisture level through dehumidifiers specially in the bathroom and kitchen. Children susceptible to allergic manifestations may get exposed to allergens in school and play ground, thus it is prudent to restrict their outdoor activities as far as possible and stay inside during peak pollen season and avoid visit areas with high pollen density. During travel, early morning or late in the evening, when air quality is comparatively better should be preferred, the car windows should be kept close, a facemask should be worn, and travelling on windy days should be avoided.

11.6 Management Practices

House dust mites are tiny microscopic creatures, about a quarter of a millimetre long. They prefer to live on mattresses, bedding, upholstered furniture, carpets and curtains. In spite of the best efforts, even the cleanest house harbours these organisms. National Institute of Environmental Health Sciences have suggested certain proposals following which the density and occurrence of the mites can be effectively reduced.

- Maintenance of lower humidity—Dehumidifier or air conditioner should be used to maintain relative humidity within or below 50 %. Researches have shown that as the air-conditioning system reduces the humidity that the dust mites need to thrive, air-conditioned homes have ten times fewer dust mite allergens as compared to non air-conditioned homes. Using an electric blanket for 8 h each day reduces dust mites by 50 % in a month. Using a vent fan for removing moisture in bathrooms and the kitchen, repairing any source of water leaks therein are also effective in reducing the mites.
- Avoiding furry or feathered pets—Pets with fur or feathers contribute to the accumulation of dander in the dust and thus increase resource for mites to survive. So the pets should be kept out of reach of the allergic person's bedroom at all times.
- Reducing air infiltration—Aeration of the house with open windows allows entry of pollens and increase in humidity. Thus person with allergic problems should be cautious of the pollen allergen.
- House dust mites prefer warm moist environments with ideal temperature between 18 and 24 °C. Thus the parents are advised to keep the temperature in their children's bedrooms below 18 °C to deter the mites. Allergic children should not sleep in the bottom bunk bed where allergen can fall onto them.
- Proper sanitation, hygiene and a general cleanliness of the bed and bedroom should be maintained.
- Avoidance of suspected offending allergens as far as practicable.
- House dust mites seem to thrive best on human skin scales; supply of food materials to the mites should be restricted in every possible way.
- All bedding and blankets should be washed once a week in hot water (at least 130–140 °F) to kill dust mites. Non-washable bedding can be frozen overnight to kill dust mites.
- Mattress and pillows should be encased in dust-proof or allergen impermeable covers. These should be breathable and should completely enclose the item.
- Wool or feathered bedding must be replaced with synthetic materials and traditional stuffed animals with washable ones. Alternatively, if the toy cannot be washed at 130–140 °F, they should be placed in a plastic bag in the freezer for at least 12 h once a month and then washed at the recommended temperature.
- Foam mattresses favour growth and multiplication of house dust mites so it is highly recommended to use conventional cotton mattresses.

- Homes with bare floors have up to 90 % less dust in them than homes with carpets. All carpeting from concrete floors should be removed as such floors trap moisture allowing dust mites and mould spores to thrive. The floor must be sealed with a vapour barrier, and then can be covered with a washable surface such as vinyl or linoleum.
- Conventional dusting may stir up dust, resulting more circulating allergens around. Dry cloth must not be used as this swirl up the mite allergens. So, it is recommended to use a damp mop or rag to remove dust. By spraying furniture polish or dusting liquid directly on surface, airborne particles can be reduced by 93 %.
- Vacuuming—The most important tool for managing house dust and dust mites is the vacuum cleaner. Systematic and regular vacuuming of carpets, furniture, textiles and other home furnishings will help to keep dust mite populations low. Vacuum cleaner with either a double-layered microfilter bag or a HEPA filter should be used to trap allergens. Where carpets cannot be removed, vacuum cleaning must be done regularly with a high-filtration vacuum cleaner with filters capable of retaining a high proportion of the smallest particles. High-temperature steam-cleaner kill mites quite effectively.
- Person susceptible to dust mites must wear a mask while vacuuming to avoid inhaling allergens, and stay out of the vacuumed area for 20 min to allow any dust and allergens to settle after vacuuming.
- As ventilation reduces humidity and thus do not provide congenial environment for the mites to grow, the house must be kept well ventilated. Use of trickle-vents in double-glazing, or open windows is a good option.
- Use of acaricides—The use of acaricide products contribute to the elimination of house dust mites in the household environment, especially those found in places that are hard to reach by vacuum cleaner or to otherwise clean. Till date no acaricides are found to successfully control the dust mites. However, though benzyl benzoate or tannic acid may worsen allergies in some people, they are reported to reduce the levels of dust mites. Therefore cleaning and non-toxic approaches using non-chemical dust mite control measures should be given priority rather than to use the chemicals that may prove detrimental for some with serious allergies.

The harm caused by house dust mites is enormous which affect the socio-economic condition of the individual. Allergy is a disabling disease, sometimes dangerous or even fatal. This is now double challenge—designing an intervention strategy using the best evidence—base from the most promising methods, and to test the impact in realistic scenario. It is therefore recommended that proper identification of the causative allergen and subsequently the reduction of load of these offending allergens from the patients' environment may be helpful for the prophylactic management of various allergic disorders.

References

Aas K, Belin L (1972) Standardization of diagnostic work in allergy. Acta Allergol 27:439–468

Aas K, Johansson SGO (1971) The radioallergosorbent test in the in-vitro diagnosis of multiple reaginic allergy. J Allergy Clin Immunol 48:134–142

Abott J, Cameron J, Taylor B (1981) House dust mite counts in different types of mattresses, sheep skins and carpets and a comparison of brushing and vacuuming collection methods. Clin Allergy 11(6):589–595

Agarwal S (2009) TNN, 4 May 2009

Anand P (1981) Dust mites as an offending allergen in nasobronchial allergy. Aspect Allergy Appl Immunol 14:34–36

Andrews B, Murray AC, Farguson BM (1980) The seasonal variations of allergic respiratory symptoms induced by house dust mites. Ann Allergy 45:347–350

Araujo-Fontaine A, Wagner M, Kremer M (1973) Contribution to the study of house dust mites in Alsace. Relations to conditions in the habitat. C R Seances Soc Biol Fil 167(2):371–378

Arlian LG, Bernstein IL, Johnson CL et al (1979) A technique for separation of house-dust mites from culture media. J Med Entomol 16(2):128–132

Arlian LG, Woodford PJ, Bernstein IL (1983) Seasonal population structure of house dust mites, *Dermatophagoides* spp. (Acari: Pyroglyphidae). J Med Entomol 20:99–102

Arlian LG, Vyszenski-Moler DL, Fernandez-Caldas E (1993) Allergenicity of the mite *Blomia tropicalis*. J Allergy Clin Immunol 91:1042–1050

Assem ESK, McAllen MK (1970) Serum reagin and leukocyte response in patients with house dust mite allergy. Br Med J 2:504–507

Aycan OK, Atambay M, Dalal UN (2007) Investigation of house dust mite incidence according to social factors. Turkiye Parazitol Derg 31(3):219–224

Baker EW, Wharton GW (1952) An introduction to Acarology. The Macmillan Company, New York

Baker EW, Evans TM, Gould DJ et al (1956) A manual of parasitic mites of medical and economic importance. National Pest Control Association, New York, p 170

Banerjee S (1988) Seasonal fluctuations of allergic pyroglyphid mites (Acari) of Birbhum district, West Bengal. (Abs.). In: Proceedings of VIIIth National Congress of Parasitology, Calcutta

Barbee RA, Lebowitz MD, Thompson HC, Burrows B (1976) Immediate skin test reactivity in a general population sample. Ann Intern Med 84:129–133

Barbee RA, Brown WG, Kaftenborn W et al (1981) Allergy skin test reactivity in a community population sample: correlation with age, histamine skin reactions and serum Immunoglobulin E. J Allergy Clin Immunol 68:106–111

G.K. Saha, *Dust Allergy: Cause & Concern*, DOI 10.1007/978-981-10-1825-1

Barreto ML, Cunha SS, Alcântara-Neves N et al (2006) Risk factors and immunological pathways for asthma and other allergic diseases in children: background and methodology of a longitudinal study in a large urban center in North eastern Brazil (Salvador-SCAALA study). BMC Pulm Med 6:15

Batabyal SK, Ghosh JM, Kundu S et al (1986) Studies on skin sensitizing tests and serum IgE levels in normal and other allergic disorders. Antiseptic 37–42

Baur X, Aschauer H, Mazur G et al (1986) Structure, antigenic determinants of some clinically important insect allergens: chironomid haemoglobins. Science 233:351–354

Bear H, Wharton GW (1970) Antigens of the house-dust mite. Excerpta Medica, International Series, 211

Bennich H, Ishizaka T, Johansson SGO et al (1968) Immunoglobulin E: a new class of human immunoglobulin. Bull World Health Org 38:151–152

Berg T, Johansson SGO (1969) IgE concentrations in children with atopic diseases. Int Arch Allergy 36:219

Berg T, Bennich H, Johansson SGO (1971) In vitro diagnosis of atopic allergy, I. A comparison between provocation tests and the radio allergosorbent test. Int Arch Allergy 40:770–778

Bernecker C (1968) Mites and house dust allergy. Lancet 2:1145

Bernhard K, Karg W, Steinbrink H (1986) House dust mites in bed dust and on the body. Angew Parasitol 27(1):49–52

Binotti RS, Muniz JR, Paschoal IA et al (2001) House dust mites in Brazil—an annotated bibliography. Mem Inst Oswaldo Cruz 96(8):1177–1184

Blythe ME (1976) Some aspects of the ecological study of the house dust mites. Br J Dis Chest 70:3–31

Blythe ME, Ubaidi W, Smith J (1975) Study of dust mites in three Birmingham hospitals. Br Med J 1:62–64

Boquete M (2006) House dust mite species and allergen levels in Galicia, Spain: a cross-sectional, multicenter, comparative Study. J Investig Allergol Clin Immunol 16(3):169–176

Bousquet J, Coulomb Y, Arrendal H et al (1982) Total serum IgE concentrations in adolescents and adults using the Phadebas IgE PRIST technique. Allergy 37:397–406

Bousquet J, Vignola AM, Demoly P (2003) Links between rhinitis and asthma. Allergy 58:691–706

Bousquet J, Flahault A, Vandenplas O et al (2006) Natural rubber latex allergy among health care workers: a systematic review of the evidence. J Allergy Clin Immunol 118(2):447–454

Bronswijk JE MH Van (1973) *Dematophagoides pteronyssinus* (Trouessart, 1897) in mattress and floor dust in a temperate climate (Acari: Pyroglyphidae). J Med Entomol 10:63–70

Bronswijk JE MH Van, Sinha RN (1971) Pyroglyphid mites (Acari) and house dust allergy. J Allergy 47:31–52

Brown HM (1958) Treatment of chronic asthma with prednisolone: significance of eosinophils in sputum. Lancet 2:1245–1247

Brown HM, Fillar JL (1968) Role of mites in allergy to house dust. Br Med J 3:646–647

Buchanan DJ, Jones IG (1972) Allergy to house dust mites in the tropics. Br Med J 3:764

Burrows B, Lebowitz MD, Barbee RA (1976) Respiratory disorders and allergy skin test reactions. Ann Intern Med 84:134–139

Cao Lopez J, Estruch fajardo I (1981) Preliminary study of the acarine fauna of house-dust of the metropolitan area of the city of Havana. Revista Cubana de Higiene Y Epidemiologis 19(3):237–243

Castagnoli M, Liguori M, Nannelli R (1983) House dust mites in Italy. In: Atti XIII Congresso Nazionale Italiano di Entomologia, Turin, Italy; Instituo di Entomologia Agraria e Apicoltura, Universita di Torino, pp 577–582

Catanghal RAM, Paller VGV (2012) Mite fauna and mite antigen detection in house dust found in residential areas in Los Banos, Laguna, Philippines. Southeast Asian J Trop Med Public Health 43(5):1114–1121

Chan Yeung M, Vedal S, Lam S et al (1985) Immediate skin reactivity and its relationship to age, sex, smoking and occupational exposure. Arch Environ Health 40(1):53–57

Chang YC, Hsieh KH (1989) The study of house dust mites in Taiwan. Allergy 62(2):101–106
Channa Basavanna GP, Krishna Rao NS, Ranganath HR (1984) House dust mites from human and animal habitations in India. Acarology 6(2):1149–1153
Chapman MD, Heymann PW, Wilkins SR (1987) Monoclonal immunoassays for major dust mite (*Dermatophagoides*) allergens, Der p I and Der f I, and quantitative analysis of the allergen content of mite and house dust extracts. J Allergy Clin Immunol 80(2):184–194
Charlet LD, Mulla MS, Sanchez-Medina M (1977) Domestic Acarina of Colombia: occurrence and distribution of Acari in house dust. Acarologia 19:302–317
Chaudhury S, Roy I, Podder S et al (2005) Diversity of synanthropic mites in Kolkata Metropolis, India. Rec Zool Surv India 104(3–4):151–159
Chaudhury S, Gupta SK, Saha GK (2011) Seasonal variations in synanthropic mite community of Kolkata, India. Proc Zool Soc 64(1):35–39
Chaudhury S, Gupta SK, Saha GK (2012) Synanthropic acarine population associated with bird nest. J Threatened Taxa 4(5):2553–2616
Chaudhury S, Gupta SK, Saha GK (2013) Synanthropic mites of Kolkata—an ecological appraisal. Proc Zool Soc 66:56–63
Chen Yu, Blaser MJ (2008) *Helicobacter pylori* colonization is inversely associated with childhood asthma. J Infect Dis 198(4):553–560
Chew FT, Zhang L, Ho TM et al (1999) House dust mite fauna in tropical Singapore. Clin Exp Allergy 29:201–206
Chhabra SK, Gupta CK, Chhabra P et al (1998) Prevalence of bronchial asthma in school children in Delhi. J Asthma 3:291–296
Chmielewski W (1991) Biological observations of allergenic mites *Suidasia nesbetti*. Wiad Parazytol 37(1):133–136
Ciftci IH, Cetinkaya Z, Atamboy M et al (2006) House dust mite fauna in western Anatolia, Turkey. Korean J Parasitol 44(3):259–264
Coca AF (1922) Studies in specific hypersensitiveness V. The preparation of fluid extracts and solutions for use in the diagnosis and treatment of the allergies with notes on the collection of pollens. J Immunol 7:163–178
Cockcroft DW, Ruffin RE, Dolovich J et al (1977) Allergen induced increase in non-allergic bronchial reactivity. Clin Allergy 7:503–513
Colloff MJ (1987) Effects of temperature and relative humidity on development times and mortality of eggs from laboratory and wild populations of European house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Exp Appl Acarol 3:279–289
Colloff MJ, Spieksma FThM (1992) Pictorial keys for the identification of domestic mites. Clinical and Experimental Allergy 22:823–830
Connell JT (1968) Morphologic changes in eosinophils in allergic diseases. J Allergy 41:1
Cooke RA (1922) Studies in specific hypersensitiveness IV. New etiologic factors in bronchial asthma. J Immunol 7:147
Corren J (2000) The association between allergic rhinitis and asthma in children and adolescents: epidemiologic considerations. Pediatr Ann 29:400–402
Cranston PS (1988) Allergen of non-biting midges (Diptera: Chironomidae): systematic survey of chironomid haemoglobins. Med Vet Entomol 2:117–127
Cranston PS, Gad El Rab MO, Kay AB (1981) Chironomid midges as a cause of allergy in the Sudan. Trans R Soc Trop Med Hyg 75:1–4
Cranston PS, Gad El Rab MO, Tee RD et al (1983) Immediate-type skin reactivity to extracts of the 'green nimitti' midge (*Cladotanytar*sus *lewki*) and other chironomids in asthmatic subjects in the Sudan and Egypt. Ann Trop Med Parasitol 71:521–533
Cunliffe F (1958) *Pyriglyphus morlani*, a new genus and species forming a new family, Pyroglyphidae, in the Acaridae, Acarina, Sarcoptiformes. Proc Ento Soc Wash 60:85–86
Cunnington AM (1967) The mite fauna of house dust. Acta Allergologica 22:415
D'Amato GG, Liccardi M, D'Amato M et al (2005) Environmental risk factors and allergic bronchial asthma. Clin Exp Allergy 35:1113–1124

Dar N, Gupta VK (1979) Studies on the house dust mites of India and their role in causation of bronchial asthma and allergic rhinitis. Part—I. The mites. Oriental Insects 13(3–4):261–298
Dar N, Menon MPS, Shivpuri DN et al (1973) The mite fauna of Indian house dusts. Aspects Allergy Appl Immunol 6:51–60
Dar N, Shivpuri DN, Menon MPS (1974) Seasonal periodicity of house dust mite population. Aspects Allergy Appl Immunol 7:63–70
Dasgupta A, Cunliffe AC (1970) Common antigenic determinants in extracts of house dust and *Dermatophagoides* sp. Clin Exp Immunol 6:891–898
Datta S, Joy VC (1987) Incidence of potential allergy causing house dust mite, *Dermatophagoides farinae* Hughes at Santiniketan. Abstract. 1st National Seminar on Acarology, Kalyani, West Bengal
David GM, Susan HH, Hussain R et al (1980) Genetics of human immune response to allergens. J Allergy Clin Immunol 65:322–332
De Filippi I, Ymen L, Schroder H (1981) Clinical accuracy of updated version of Phadebas RAST test. Ann Allergy 46(5):249–255
De Moreira NS, Filagonio CJB (1980) On the occurrence of the deutonymphal stage in pyroglyphid mites Cunlife, 1958 of the genus *Dermatophagoides* Bogdanov, 1864 (Sarcoptiformes). Ludiana 1:135–141
de Oliveria AJ, Daemon E (2003) Qualitative and quantitative assessment of mites (Acari) in domiciliary dust in rural dwellings in the "Zona da Mata" region, Minas Gerais, Brazil. Rev Bras Zool 20(4):675–679
Dekker H (1928) Asthma and Milben. Nunchener Med Wochensch 12:515–516
Del Rey Calero J, Lomas GD (1972) Respiratory allergy provoked by the sensitising mites of house dust. Rev Clin Esp 126(3):215–222
Dhar S, Banerjee R (2010) Atopic dermatitis in infants and children in India. Indian J Dermatol Venereol Leprol 76:504–513
Diaz-Sanchez D, Garcia MP, Wang M et al (1999) Nasal challenge with diesel exhaust particles can induce sensitization to a neoallergen in the human mucosa. J Allergy Clin Immunol 104:1183–1188
Dixit IP, Mehta RS (1973) Prevalence of *Dermatophagoides* species in India and its role in the causation of bronchial asthma. J Assoc Phys India 21:31–37
Dobson PM (1979) Some effects of microclimate on the longevity and development of *Dermatophagoides pteronyssinus*. Acarologia 21:482–486
Dockhorn R (1982) Using the RAST with an overview of clinical significance. Ann Allergy 49:1–8
Domrow R (1970) Seasonal variation in numbers of the house dust mite in Brisbane. Med J Aust 2:1248–1250
Dubinina EV (1984) Mite fauna of the dust in the dwellings of bronchial asthma patients in the Lithuanian SSR. Med Parazitol (Mosk) 5:63–67
Dulfano MJ (1973) Sputum: fundamentals and clinical pathology. Carles C Thomas, Springfield
Durham SR, Till SJ (1998) Immunologic changes associated with allergen immunotherapy. J Allergy Clin Immunol 102:157–164
Dusbabek F (1975) Population structure and dynamics of the house-dust mite *D. farinae* in Czechoslovakia. Folia Parasitol Prague 22:219–231
Eichenfield LF, Hanifin JM, Beck LA et al (2003) Atopic dermatitis and asthma: parallels in the evolution of treatment. Pediatrics 111:608–616
Ellis AG (1908) The pathological anatomy of bronchial asthma. Am J Med Sci 136:404–429
Ellwood P, Asher MI, Björkstén B et al (2001) Diet and asthma, allergic rhinoconjunctivitis and atopic eczema symptom prevalence: an ecological analysis of the international study of endosymbiotic *Spiroplasma* by mites. Biol Lett 3:23–25
Eriksson NE, Peterson I, Vedal S et al (1985) Allergy among farmers. Abstract 137 of annual meeting of the European Academy of Allergology and Clinical Immunology, Stockholm, p 199
Evans GO, Sheals JG, Macfarlane D (1961) The terrestrial Acari of the British Isles. An introduction to their morphology, biology and classification, vol 1. Introduction and biology. British Museum (Natural History), London, p 219

Fain A (1965) Les acariens nidicoles et detriticoles de la famille Pyroglyphidae Cunliffe (Sarcoptiformes). Revue Zool Bot Afr 72:257–288
Fain A (1966) Nouvelle description de *Dermatophagoides pteronyssinus*. Importance de cet acarien en pathologie humaine. Acarologia 8:302
Fain A (1967) Deux nouvelles especes de Dermatophagoidinae rattachment de cette sous – famille aux Pyroglyphidae (Sarcoptiformes). Acarologia 9:870–881
Fain A, Hart BJ (1986) A new simple technique for extraction of mites, using the difference in density between ethanol and saturated NaCl (preliminary note). Acarologia 27(3):255–256
Feldman-Mushan B, Mumcuoglu Y, Osterovich T (1985) A survey of house dust mites (Acari: Pyroglyphidae and Cheyletidae) in Israel. J Med Entomol 22(6):663–669
Fineman SM (2002) The burden of allergic rhinitis: beyond dollars and cents. Ann Allergy Asthma Immunol 88:2–7
Fink JN (1985) Immunotherapy of asthma. J Allergy Clin Immunol 76(2):402–404
Flores I, Mora C, Rivera E et al (2003) Cloning and molecular characterization of a cDNA from *Blomia tropicalis* homologous to dust mite group 3 allergens (trypsin-like proteases). Int Arch Allergy Immunol 130:12–16
Frankland AW, El-Hefney A (1971) House dust and mites as causes of inhalant allergic problem in the United Arab Republic. Clin Allergy 1(3):257–260
Franklin W (1974) Treatment of severe asthma. New Engl J Med 290:1469–1473
Frati F, Incorvaia C, David M et al (2012) Requirements for acquiring a high-quality house dust mite extract for allergen immunotherapy. Drug Des Dev Ther 6:117–123
Fujimoto T, Kato H, Ichiose E et al (1982) Immune complex and mite antigen in Kawasaki disease. Lancet ii:980–981
Furumizo RT (1975) Laboratory observations on the life history and biology of the American house dust mite *Dermatophagoides farinae* (Acarina: Pyroglyphidae). Calif Vector Views 22:49–60
Gabriel M, Cunnington AM, Allan WGLC et al (1982) Mite allergy in Hong Kong. Clin Exp Allergy 12(2):157–171
Galvao AB, Guitton N (1986) Mites in house dust from the capitals of Brazilian states and the Island of Fernando de Noronha. Mem Inst Oswaldo Cruz 81(4):417–430
Gamal-Eddin FM, Tayel SE, Abou-Senna FM et al (1982) Present status and ecology of house dust mites in Egypt: as approaches to environmental control of mites and preparation of specific diagnostic antigen before resort to any desensitizing vaccine. J Egypt Soc Parasitol 12(1):253–282
Gamal-Eddin FM, Abou-Senna FM, Tayel SE et al (1983) Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled temperatures and relative humidities to pave the way in front of the workers in the field of house dust mite bronchial asthma. 1. Pre-imaginal period. J Egypt Soc Parasitol 13(2):319–334
Gangal SV, Chowgule R (2009) Infections in early life and susceptibility to allergic diseases: relevance of hygiene hypothesis. Curr Sci 96:784–793
Gell PGH, Coombs RRA, Lachman PJ (1977) Clinical aspects of immunology, 3rd edn. Blackwell Scientific Publication, Oxford
Gill NK, Kaur H (2014) A study on the occurrence, prevalence and composition of mite fauna in human dwellings of Patiala City, Punjab (India). Indian J Sci Res 8(1):91–97
Gleich GI, Averbeck AK, Swedlund HA (1971) Measurement of IgE in normal and allergic serum by radioimmunoassay. J Lab Clin Med 77:690–698
Gomez MS, Portus M, Gallego J (1981) Factors that influence the composition of the mite fauna of house dust. IV. Altitude. Allergol Immunopathol 9(2):123–130
Grater WC, Dockhorn R, Boggs P et al (1982) Report of the American College of Allergists Committee on Standarisation of Allergic Extracts. Ann Allergy 49:49–54
Green WF (1983) The house dust mite, *Dermatophagoides farinae*, in Australia. Med J Aust 2(6):259–260
Greenberger PA (2002) Immunotherapy update: mechanisms of action. Allergy Asthma Proc 23:373–376

Gupta SK, Datta Roy RK (1975) Occurrence of house dust mites in West Bengal (India). Newsl Zool Surv India 1(3):48–50
Guy Y, Rioux JA, Guin JJ et al (1972) The mite fauna of house dust. Results of the survey in Languedoc-Roussillon. Bull de la Soc de Pathol Exot 65(3):472–481
Hall CC Jr, McMahon B, Sams JT (1971) Collection and rearing of *Dermatophagoides farinae* Hughes, a mite from house dust. Ann Allergy 29(2):81–85
Hallas TE, Korsgaard J (1983) Annual fluctuations of mite and fungi in Danish house dust: an example. Allergol Immunopathol 11(3):195–200
Halmai Z (1984) Changes in the composition of house dust mite fauna in Hungary. Parasitol Hung 17:59–70
Halmai Z, Allexander FAR (1971) Studies on the house dust allergen. Allerg Immunol 17:69–71
Hannaway PJ, Hyde JS (1970) Scratch and intradermal skin testing. A comparative study in 250 atopic children. Ann Allergy 28:413–419
Hart BJ (1998) Life-cycle and reproduction of house dust mites: environmental factors influencing mites populations. Allergy 53:13–17
Henderson LL, Swedlund HA, Van Dellen RG (1971) Evaluation of IgE tests in an allergy practice. J Allergy Clin Immunol 48:361–365
Henzel L, Kalisinska E, Kosik-Bogacka DI (2010) Mites in dust samples collected from sleeping places in apartments. Polish J Environ Stud 19(4):723–730
Herbert FA, Weimer N, Salkie ML (1982) RAST and skin test screening in the investigation of asthma. Ann Allergy 49:311–317
Hillerdal G (1982) Mites living in hay: an important allergic source. Allergy 37:475–489
Ho TM, Nadchatram M (1984) Life-cycle and longevity of *Dermatophagoides pteronyssinus*, Trouessart (Acarina: Astigmata: Pyroglyphidae) in a tropical laboratory. Trop Biomed 1:159–162
Hofter T (2006) The necessary need for mattress cleaning: dust mites, asthma, allergies and allergens. http://www.sterilmattress.com
Hogarth-Scott RS, MeNieol RN, Williams HE et al (1973) Diagnosis of allergy in vitro: a comparison between skin testing and serum levels of specific IgE antibody in children. Med J Aust 1:1293–1297
Holgate ST (2008) Pathogenesis of asthma. Clin Exp Allergy 38(6):872–897
Honsinger RW, Silverstein D, Van Arsdel PP (1972) The eosinophil and allergy: why? J Allergy Clin Immunol 49(3):142–155
Horn BR, Robin ED, Theodore J et al (1957) Total eosinophil counts in the management of bronchial asthma. New Engl J Med 292:1152–1155
Houck MA, Qin H, Roberts HR (2001) Hantavirus transmission: potential role of ectoparasites. Vector-borne Zoonotic Dis 1:75–79
Hoy MA (2009) The predatory mite Metaseiulus occidentalis: mitey small and mitey large genomes. Bioessays 31(5):581–590
Huber HL, Kossler KK (1922) The pathology of bronchial asthma. Arch Int Med 30:689–760
Hughes AM (1961) The mites of stored food. Mins Agric Fish Food Tech Bull 9:287
Hughes AM (1976) The mites of stored food and houses. HMSO, London
Hurtado I, Mava P (1987) House dust mites in Caracas, Venezuela. Ann Allergy 59(2):128–130
ISAAC Steering Committee (1998) Worldwide variation in prevalence of asthma, allergic rhinoconjunctivitis and atopic eczema. Lancet 351:1225–1232
Ishii A, Takaoka M, Ichinoe M et al (1979) Mite fauna and fungal flora in house dust from houses from asthmatic children. Allergy 34:379–387
Ishii A, Shimomura H, Hashiguchi J et al (1982) Biochemical and allergenic properties of the house dust mite extract *Dermatophagoides pteronyssinus*. Allergy 37:285–290
Ishizaka K, Ishizaka T (1967) Identification of Y-E antibodies as a carrier of reaginic activity. J Immunol 99:1187–1198
Ishizaka K, Ishizaka T, Hornbrook M (1966) Physicochemical properties of human reaginic antibody. IV: Presence of a unique immunoglobulin as a carrier of reaginic activity. J Immunol 97:75–85

Ito K, Miyamoto T, Shibuya T et al (1986) Skin test and radioallergosorbent test with extracts of larval and adult midges of *Tokunagayusurik a akamusi* Tokunaga (Diptera: Chironomidae) in asthematic patients of the metropolitan area of Tokyo. Ann Allergy 57:199–204

Ivanova LN, Petrova YuI (1984) The ecology and fauna of house-dust mites. Meditsinskaya Parazitologiya i Parazitarnye Bolezni 5:78–82

Jaenike J, Polak M, Fiskin A et al (2007) Interspecific transmission of haemoglobins. Science 233:351–354

Jamil Z, Maurya R (1981) Exploration of the requisites for the mass culture of *Dermatophagoides farinae*. In: Channa Basavanna GP (ed) Contributions in Acarology in India, vol 27, pp 111–115

Jogdand SB (1987) Air spora at Aurangabad. Dissertation, Marathwara University, Aurangabad, India

Johansson SGO, Bennich H, Berg T (1972) The clinical significance of IgE. Progr Clin Immunol 1:157–181

Johansson SGO (1967) Raised levels of new immunoglobulin class (IgND) in asthma. Lancet 2:951

Johansson SGO, Bennich H, Wide L (1968) A new class of Immunoglobulin in human sera. Immunology 14:265–272

Johansson SGO, Bennich H, Berg T et al (1970) Some factors influencing the serum IgE levels in atopic diseases. Clin Exp Immunol 6:43–47

Juhlin L, Johansson SGO, Bennich H et al (1969) Immunoglobulin E in dermatoses. Arch Derm 100:12

Kabeya H, Colborn JM, Bai Y et al (2010) Detection of *Bartonella tamiae* DNA in ectoparasites from rodents in Thailand and their sequence similarity with bacterial cultures from Thai patients. Vector-borne Zoonotic Dis 10:429-434

Kagan SL, Yunginger JW, Johnson R (1984) Lake fly allergy: incidence of chironomid sensitivity in an atopic population. J Allergy Clin Immunol 73:187

Kannan I, Rajendran P, Selvarani P et al (1996) A survey of house dust mites in Madras (India) in relation to mite density, seasonal variation and niche. Biomedicine 16:58–62

Kathuria PC, Sharma N, Sharma N (1992) Clinically important pollen, fungi and house dust mite (*D. farinae*) and dusts in nasobronchial allergy. Indian J Allergy Appl Immunol 6(1&2):27–30

Kay AB, Gad El Rab MO, Stewart J et al (1978) Widespread IgE-mediated hypersensitivity in Northern Sudan to the chironomid *Cladotanytarsus lewisi* ('green nimitti'). Clin Exp Immunol 34:106–110

Keil H (1983) Ecological-faunastic studies on house dust mites in Hamburg with particular reference to *D. pteronyssinus* Trouessart, 1897 and *D. farinae* Hughes, 1961 (Acari: Pyroglyphidae). Entomologische Mitteilungen aus dem Zoologischen Museum Hamburg 7(118):343–386

Kern RA (1921) Dust sensitization in bronchial asthma. Med Clin North Am 5:751–758

Khatua SP, Khatua S, Batabayal SK et al (1986) Immunological study of bronchial asthma. Indian Pediatr 23:677–680

Khatua SP, Khatua S, Batabyal SK et al (1987) Immunological study of bronchial asthma in children. J Pediatr Clin India 29–33

Kherkhof M, Dubois AEJ, Postma DS et al (2003) Role and interpretation of total serum IgE measurements in the diagnosis of allergic airway diseases in adults. Allergy 58:905–911

Kilpelainen M, Koskenvuo M, Heleniusm H et al (2002) Stressful life events promote the manifestation of asthma and atopic diseases. Clin Exp Allergy 32:256–263

King MJ, Betts L, Stock M et al (1985) House dust mite allergy in military personel. Milit Med 150(1):19–22

Kjellman NIM (1977) Atopic disease in seven-year-old children. Acta Paediatr Scand 66:465

Kjellman NIM, Johansson SGO, Roth A (1976) Serum IgE levels in healthy children quantified by a sandwich technique (PRIST). Clin Allergy 6:51–59

Korsgaard J (1983) House dust mites and absolute indoor humidity. Allergy 38:85–92
Krametter-Froetscher R, Leschnik M, Hoegler S et al (2006) Occurrence of ear-mite *Raillietia auris* in cattle in Austria. Vet J 17(1):186–188
Krantz GW (1978) A manual of Acarology, 2nd edn. Oregon State University, Corvallis
Krishna Rao NS, Channa Basavanna GP (1973) Pyroglyphid mites in man and his surroundings. Curr Sci 42:33
Krishna Rao NS, Ranganath HR, Channa Basavanna GP (1981) House dust mites from India. Indian J Acarol 5:85–94
Kumar L, Newcomb RW, Hornbrook M (1971) A year round study of serum IgE levels in asthmatic children. J Allergy 48:305–312
Kumar NR, Kumar R, Singh T (1988) Faunistic and ecological studies on the Acaro fauna of habitation of the socio-economically poor dwellers of the Punjab and Himachal Pradesh, India. Prog Acarol 1:169–172
Kumud R, Mathur B, Mathur S (1988) A preliminary report on the occurrence of house dust mites from Hissar, the subtropical region of Haryana, India. Prog Acarol 1:157–162
Lakshmi R, Haq MA (1999) Survey on dust mites of Calicut University Campus. J Acarol 15:55–63
Lal MA, Dasgupta A, Pande JM et al (1973) Clinical significance of skin reaction to mite extracts in individuals with bronchial asthma. Indian J Med Res 61:672–675
Larson DG, Mitchell WF, Wharton GW (1969) Preliminary studies on *Dermatophagoides farinae* Hughes, 1961 (Acari) and house dust allergy. J Med Entomol 6:295–299
Lawlor GJ Jr, Tashkin DP (1981) Asthma. In: Lawlor GJ Jr, Fischer TJ (eds) Manual of allergy and immunology: diagnosis and therapy. Little Brown & Company, Boston, pp 113–156
Lawrence AWW (1982) Clinical aspects of respiratory tract allergic diseases in Jamaica, West Indies. Ann Allergy 49:225–228
Lietzén R, Virtanen P, Kivimäki M et al (2011) Stressful life events and the onset of asthma. Eur Respir J 37(6):1360–1365
Lindbladd JH, Farr RP (1961) The incidence of positive intradermal reactions in humans without history of rhinitis or asthma. J Allergy 32:392–401
Loeffler JA, Cawley LP, Moeder M (1973) Serum IgE levels: correlation with skin test sensitivity. Ann Allergy 31:130–140
Lowell FC (1967) Clinical aspects of eosinophilia in atopic disease. J Am Med Assoc 202:875–878
Lutsky I, Teichtahl H, Bar-Sela S (1984) Occupational asthma due to poultry mites. J Allergy Clin Immunol 73:56–60
Lyon WF (1991) House dust mites. Ohio State University Extension Fact Sheet, HYG-2157-97
Macan J, Kanceljak B, Plavec D et al (2003) Difference in mite fauna between the continental and mediterranean climates of Croatia: microscopy and Dust screen™ test findings. Allergy 58:780–783
Macan J, Kanceljak-Macan B, Milković-Kraus S (2012) Pyroglyphid mites as a source of work-related allergens. Arh Hig Rada Toksikol 63(1):57–66
Mahesh PA, Kushalappa PA, Holla AD et al (2005) House dust mite sensitivity is a factor in chronic urticaria. Indian J Dermatol Venereol Leprol 71:99–101
Makovcova S, Samsinak K, Vobrazkova E (1982) Biological aspects of mites from house dust and their importance in house dust allergy. Part-I. Fysiatricky a Reumatologicky Vestnik 60(2):69–74
Mariana A, Ho TM, Sofian-Azirun M et al (2000) House dust mite fauna in the Klang valley, Malaysia. Southeast Asian J Trop Med Pub Health 31(4):712–721
Massey JE, Massey DG (1984) Effect of vacuum cleaning on house dust mites. Hawaii Med J 43(11):404–406
Matsumoto K, Okamoto M, Wada Y (1986) Effects of relative humidity on life cycle of the house dust mites, *D. farinae* and *D. pteronyssinus*. Jap J Sanit Zool 37(1):79–90
Maunsell K, Wraith DG, Cunnington AM (1968) Mites and house dust allergy in bronchial asthma. Lancet 1:1267–1270

Maurya KR, Jamil Z (1980) Factors affecting the distribution of house dust mites under domestic conditions in Lucknow. Indian J Med Res 72:284–290
Maurya KR, Jamil Z, Dev B (1983) Prevalence of astigmatid mites (Acarina) in the domestic environment of north-eastern and northern India. Biol Mem 8(1/2):207–215
Meltzer SJ (1910) Bronchial asthma as a phenomenon of anaphylaxis. JAMA 55:1021–1024
Metzger WJ, Nugent K, Richerson HB et al (1985) Methods for bronchoalveolar lavage in asthematic patients following bronchoprovocation and local antigen challenge. Chest 87:16–19
Middleton E Jr, Reed C, Ellis E (eds) (1978) Allergy: principles and practice. Mosby and Company, St. Louis
Ming HT (1983) Division of Acarology. Annual report, 1983, Institute for Medical Research, Kuala Lumpur, Malaysia, pp 25–26
Miranda RJ, Diomedes QA, Alejandro A (2002) House dust mites from urban and rural houses on the lowland pacific slopes of Panama. Syst Appl Acarol 7:23–30
Mitchell WW, Warton GW, Larson DG et al (1969) House dust, mites and insects. Ann Allergy 23:93
Miyamoto T, Oshima S, Ishizaki T et al (1968) Allergenic identity between the common floor mite (*Dermatophagoides farinae* Hughes, 1961) and house dust as a causative antigen in bronchial asthma. J Allergy 42:14–28
Miyamoto T, Johansson SGO, Ito K et al (1974) Atopic allergy in Japanese subjects: studies primarily with radioallergosorbent test. J Allergy Clin Immunol 53:9–19
Miyamoto T, Ishii A, Sasa M (1975) A successful method for mass culture of the house dust mite, *Dermatophagoides pteronyssinus* (Trouessart, 1897). Jap J Exp Med 45(2):133–138
Modak A, Saha GK (2002) Effect of certain socio-ecological factors on the population density of house dust mites in mattress dust of asthmatic patients of Calcutta, India. Aerobiologia 18:239–244
Modak A, Saha GK, Tandon N et al (1987) House dust mite fauna in asthmatics with special reference to socio-economic status of the patients. Indian Med Gaz CXXII(10):346–349
Modak A, Saha GK, Tandon N et al (1991) Dust mite fauna in houses of bronchial asthma patients-comparative study of three zones of West Bengal (India). Entomon 16:115–120
Modak A, Saha GK, Gupta SK et al (1995) Occurrence of some uncommon dust mites from the houses of respiratory allergic patients in West Bengal, India. J Acarol 13:29–34
Modak A, Saha GK, Tandon N (2000) Laboratory observation of the life cycle of *Dermatophagoides pteronyssinus* (Acarina: Pyroglyphidae): a common allergic mite of house dust. Proc Zool Soc 53(2):105–108
Modak A, Saha GK, Gupta SK et al (2003) Faunal diversity and habitat preference of house dust mites of West Bengal in relation to nasobronchial allergic disorders. Rec Zool Surv India 102(1&2):1–11
Montoro J, Mullol J, Jáuregui I et al (2009) Stress and allergy. J Investig Allergol Clin Immunol 19(1):40–47
Mosbech H, Jensen A, Heining JH et al (1991) House dust mite allergens on different types of mattresses. Clin Exp Allergy 21:351–355
Mulla MS, Medina MS (1980) Domestic acari of Colombia-bionomics, ecology and distribution of allergenic mites, their role in allergic diseases. Department of Entomology, University of California, Riverside, USA, p 270
Mulla MS, Harkrider JR, Galant SP et al (1975) Some house dust control measures and abundance of *Dermatophagoides* mites in Southern California (Acari: Pyroglyphidae). J Med Ent 12:5–9
Muller G (1958) A centrifugal flotation extraction technique and its comparison with two funnel extractors. In: Murphy PW (ed) Progress in Soil Zoology (1962), London, p 207
Muller UR (2003) Recent developments and future strategies for immunotherapy of insect venom allergy. Curr Opin Allergy Clin Immunol 3:299–303
Mulvey PM (1972) Cot death survey. Anaphylaxis and the house dust mite. Med J Aust 2:1240–1244

Mumcuoglu KY (1976) House dust mites in Switzerland. Distribution and taxonomy. J Med Entomol 13:361–373
Mumcuoglu KY, Gat Z, Horowitz T et al (1999) Abundance of house dust mites in relation to climate in contrasting agricultural settlements in Israel. Med Vet Entomol 13:252–258
Murray AB, Zak P (1979) The seasonal variation in a population of house dust mites in a North American City. J Allergy Clin Immunol 64(4):266–269
Murray AB, Simons FE, Ferguson AC et al (1983) Skin testing for allergy in children. Can Med Assoc J 129:824–830
Murray AB, Ferguson AC, Morrison BJ (1985) Sensitization to house dust mites in different climatic areas. J Allergy Clin Immunol 76(1):108–112
Murton JJ, Madden JL (1977) Observations on the biology, behaviour and ecology of the house dust mite, *Dermatophagoides pteronyssinus* (Trouessart) (Acarina: Pyroglyphidae) in Tasmania. J Aust Ent Soc 16:281–287
Muto K, Takai H, Hiratani M et al (1985) The increase in serum IgE and anti *Dermatophagoides pteronyssinus* (anti-DP) specific IgE levels during stay at home in institutionalized asthmatic children: The role of house dust mite in the dwelling as a trigger of asthma attack in mite sensitive patients. Arerugi 34(3):173–183
Nagaya H (1979) Relationship between antigen specific IgE antibody (RAST) and total serum IgE levels. Ann Allergy 43:267–270
Nandi S, Aditya G, Saha GK (2014) Chironomid midges as allergens: evidence from two species of Kolkata, India. Indian J Med Res 139:142–147
Nayar E, Lal M, Das Gupta A (1974) The prevalence of mite *Dermatophagoides pteronyssinus* and its association with house dust allergy. Indian J Med Res 62(1):11–14
Nelson HS (2003) Advances in upper airway diseases and allergen immunotherapy. J Allergy Clin Immunol 111:S793–S798
Norman PS, Lichenstein LM, Ishizaka K (1973) Diagnostic tests in ragweed hay fever. J Allergy Clin Immunol 52:210–224
Novak N, Bieber T, Leung DY (2003) Immune mechanisms leading to atopic dermatitis. J Allergy Clin Immunol 112:S128–S139
Numata T, Yamamoto S, Yamura T (1979) The role of mite allergen in chronic urticaria. Ann Allergy 43:356
Nye L, Merrett TG, Landon J et al (1975) A detailed investigation of circulating IgE levels in a normal population. Clin Allergy 1:13–24
Ober C (1998) Do genetics play a role in the pathogenesis of asthma? J Allergy Clin Immunol 101:S417–S420
Oboussier H (1939) Beitrage Zur biologie and anatomie der wohnugsmilben. Zeit Aug Entomol 26:253–296
Ono SJ (2000) Molecular genetics of allergic diseases. Ann Rev Immunol 18:347–366
Orgel HA, Hamburger RN, Bazaral M et al (1975) Development of IgE and allergy in infancy. J Allergy Clin Immunol 56:296–307
Orgel HA, Kemp JP, Meltzer EO et al (1977) Atopy and IgE in a pediatric allergy practice. Ann Allergy 39:161–168
Oshima S (1964) Observations on floor mites collected in Yokohoma. I: On the mites found in several schools in summer. Jap J Sanit Zool 15:233–244
Oshima S (1967) Studies on the genus *Dermatophagoides* (Psoroptidae: Acarina) as floor mites with special reference to the medical importance. Jap J Sanit Zool 18:213–215
Oshima S (1970) Studies on the mite fauna of the house dust of Japan and Taiwan with special reference to house dust mites in the houses of asthmatic children in Japan. Allergy (Arerugi) 33(8):454–462
Oshima S, Sugita K (1966) Notes on the life history of *Dermatophagoides farinae* Hughes, 1961. Bull Yokohama Munic Inst Publ Health 4:66–69
Otani T, Kinugawa N, Likura Y et al (1984) Distribution of house dust mites in the houses of asthmatic children in Japan. Allergy (Arerugi) 33(8):454–462

Paramesh H (2009) TNN, 4 May 2009
Pascual HC, Reddy PM, Nagaya H et al (1977) Agreement between radioallergosorbent test and skin test. Ann Allergy 39:325–327
Pearce N, Khaled NA, Beasley R et al (2007) Worldwide trends in the prevalence of asthma symptoms: phase III of the International Study of Asthma and Allergies in Childhood (ISAAC). Thorax 62:758–766
Pearson RS (1937) Observations on skin sensitivity in asthmatic and control subjects. Q J Med 6:165–179
Peden DB (2005) The epidemiology and genetics of asthma risk associated with air pollution. J Allergy Clin Immunol 115(2):213–219
Pelikan Z, Pelikan Filipek M (1982) A diagnostic study of immediate hypersensitivity in asthmatic patients: a comparison of bronchial challenge and serum RAST. Ann Allergy 49:112–117
Penaud A, Nourrit J, Autran P et al (1972) Recent data on pyroglyphid mites in house dust. Ann de parasitologie Humaine et compare 47(4):631–662
Peng Z, Xu W, Simons FE (1998) Highly sensitive and specific ELISA with monoclonal antibody capture to measure *Dermatophagoides farinae* 1-specific IgE. Ann Allergy Asthma Immunol 80(3):274–278
Pepys J, Chan M, Hargreeve FE (1968) Mites and house dust allergy. Lancet 1:1270–1272
Pichler CE, Helbling A, Pichler WJ (2001) Three years of specific immunotherapy with house-dust-mite extracts in patients with rhinitis and asthma: significant improvement of allergen-specific parameters and of nonspecific bronchial hyper-reactivity. Allergy 56(4):301–306
Pickering CA, Gabriei M (1973) The pattern of atopic asthma in the Chinese of Hong Kong. Bull HK Med Assoc 25:95–99
Platts-Mills TAE (2003) Allergen avoidance in the treatment of asthma and rhinitis. N Engl J Med 349:207–208
Platts-Mills TAE, Vervloet D, Thomas WR et al (1997) Indoor allergens and asthma. Third international workshop, Spain. J Allergy Clin Immunol 100:S1–S24
Platts-Mills TA, Rakes G, Heymann PW (2000) The relevance of allergen express to the development of asthma in childhood. J Allergy Clin Immunol 105:503–508
Poddar S, Saha GK, Gupta SK (2005) Some new species and new records of dust mites from Kolkata, India. Rec Zool Surv India 104(3&4):1–6
Podder S, Gupta SK, Saha GK (2006a) Description of one new species of *Grallacheles* De Leon (Acari: Cheyletidae) and new records of *Pachygnathus* Duges (Acari: Pachygnathidae) from floor dust in India. Entomon 31(4):333–338
Podder S, Choudhury I, Das A et al (2006b) Occurrence of immediate hypersensitivity to common inhalants—an investigation of nasobronchial allergy patients in Kolkata, India. Allergy Clin Immunol Int 18(3):114–119
Podder S, Biswas H, Gupta SK et al (2009a) Life cycle of house dust mite *Dermatophagoides pteronyssinus* (Acari:Pyrogylphidae) under laboratory condition in Kolkata metropolis. Acarina 17(2):219–222
Podder S, Gupta SK, Saha GK (2009b) Seasonal prevalence of allergenic mites in house dust of Kolkata Metropolis, India. Aerobiologia 25(1):39–47
Podder S, Gupta SK, Saha GK (2010a) House dust mites in relation to different habitat conditions of Kolkata metropolis, India. Acarina 18(1):91–95
Podder S, Gupta SK, Saha GK (2010b) Incrimination of *Blomia tropicalis* as a potent allergen in house dust and its role in allergic asthma in Kolkata metropolis, India. Allergy Clin Immunol Int 3(5):182–187
Polmar SH, Waldmann TA, Terry WB (1973) A comparison of three radioimmunoassay techniques for the measurement of serum IgE. J Immunol 110:1253–1261
Prausnitz C, Kustner H (1921) Studies on supersensitivity. Zbl Bakt (I Abt Orig) 86:160–169
Pujari-Vandana A, Rao S, Chandrasekhar CP (2007) Occurrence of house dust mites in relation to season, type of house and occupation. J Acarol 16:112–116

Rack G (1971) *Cheyletiella yasguri* Smiley. 1965. (Acarina, Cheyletiellidae), ein facultative menschenpathogener parasit des Hundes. Z Parasitenk 36:321–334
Ranganath HR, Channa Basavanna GP (1988) Seasonal incidence of house dust mites in Bangalore, India. Prog Acarol 1:153–156
Rao VR, Dean BV, Seaton A et al (1975) A comparison of mite populations in mattress dust from hospitals and from private houses in Cardiff, Wales. Clin Allergy 5:209–215
Rappaport I, De Ponce D, Sogn D et al (1979) On the correlation between RAST and the allergy intradermal test. Ann Allergy 43:1–7
Raynolds HY, Newball HH (1974) Analysis of proteins and respiratory cells obtained from human lungs by bronchial lavage. J Lab Clin Med 84:559–573
Rey Calero J del, Garcia de Lomas J (1972) Respiratory allergy provoked by the sensitising mites of house dust. Revista Clinica Espanola 126(3):215–222
Roy S, Sharma A, Chetty A et al (1987) Skin test and RAST in children allergic to common allergens. Indian J Pediatr 5:437–440
Rueda LM (1985) House dust mites (Acari) in Malayasia. Philippines Entomologists 6(4):428–434
Saha GK (1993) House dust mite allergy in Calcutta, India: evaluation by RAST. Ann Allergy 70:305–309
Saha GK (1994) Relationship between *Dermatophagoides* mite density and specific immune response in asthmatic patients. Ann Allergy 73:429–433
Saha GK (1997) House dust mite sensitivity among rural and urban asthmatics of West Bengal, India: a comparison. Aerobiologia 13:269–273
Saha GK (2002) House dust allergy—an environmental enigma. In: Recent advances in life sciences. University of Calcutta, pp 11–13
Saha GK (2005) House dust mite allergy—an environmental enigma. In: Mukhopadhyay A, De AK (eds) Perspective in environmental health—vector and vector borne diseases. Originals, New Delhi, pp 133–147
Saha GK, Modak A, Batabayal SK et al (1989) Clinical significance of IgE in bronchial asthma. J Indian Med Assoc 87:155–157
Saha GK, Modak A, Tandon N et al (1995a) Prevalence of house dust mites (*Dermatophagoides* spp.) in homes of asthmatic patients of Calcutta. Ann Entomol 12:21–25
Saha GK, Modak A, Tandon N et al (1995b) The evaluation of skin prick test in house dust mite allergy in Calcutta, India. Trop Geogr Med 47(6):278–281
Saha GK, Modak A, Tandon N (1997) Comparative study of dust mite fauna in Calcutta and its suburbs with special reference to *Dermatophagoides* species. Ann Entomol 15:18
Salkie ML, Weimer N (1984) The influence of season and of sex on the serum of total IgE and on the distribution of allergen specific IgE. Clin Biochem 17(6):362–366
Santrach PJ, Peterson RN, Yunginger W (1980) Comparison of diagnostic test for hymenoptera sting allergy. Ann Allergy 45:130–136
Sasa M (1950) Mites of the genus *Dermatophagoides* Bogdanov 1864, found from three cases of human acariasis. Jap J Exp Med 20:519–525
Sasa M (1951) Further notes on mites of the genus *Dermatophagoides* Bondanov, found from human acariasis. Jap J Exp Med 21:199–203
Sasa M (1985) Chironomid midges as the cause of bronchial asthma. Kankyo Eisei 32:8–14
Sasa M, Miyamoto T, Shinohara S et al (1970) Studies on mass culture and isolation of *Dermatophagoides farinae* and some other mites associated with house dust and stored food. Jap J Exp Med 40(5):367–382
Saxena RK, Jamil Z, Saxena KC (1981) Role of *Caloglyphus barlesi* in house dust allergy. In: Channa Basavanna GP (ed) Contributions to Acarology in India, vol 27, pp 115–121
Schauff ME (2000) Mighty mites ubiquitous, inconspicuous, harmful, helpful. Agric Res 48(10):2
Schei MA, Hissen JO, Lund E (2002) House dust mites and mattresses. Allergy 57:538–542
Sears MR, Hervison GP, Holdaway MD et al (1989) The relative risks of sensitivity to grass pollen, house dust mite, and cat dander in the development of childhood asthma. Clin Exp Allergy 19:419–424

Sesay HR, Dobson RM (1972) Studies on the mite fauna of Scotland with special reference to that of bedding. Acarologia 14(3):384–392
Shah P, Bapat MM (2006) Improper window air-conditioning of home and occurrence of house dust mite allergen infestation in Mumbai City women. Indian J Med Sci 60(11):472–474
Shamiyeh NB, Hornsby RP, Bennett SE et al (1973) Distribution of house dust mites in environs of the Tennessee River Valley (USA). J Econ Entomol 66(4):998–999
Sharma D, Dutta BK, Singh AB (2009) Pollen, fungus and house dust mites survey at the residence of 90 allergic patients in greater Silchar area of Assam, India. Res J Allergy 1:1–11
Sharma D, Dutta BK, Singh AB (2011) Dust mites population in indoor houses of suspected allergic patients of South Assam. ISRN Allergy, India. doi:10.5402/2011/576849
Sharp JL, Haramoto FH (1970) *Dermatophagoides pteronyssinus* (Trouessart) and other Acarina in house dust in Hawaii. Proc Hawaiian Entomol Soc 20(3):583–589
Shivpuri DN (1962) Comparative evaluation of the sensitivity of common methods of diagnostic antigen tests in patients of respiratory allergy. Indian J Chest Dis 4:102–108
Shivpuri DN (1981) House dust mite allergy in India. Aspects Allergy Appl Immunol 14:19–35
Shivpuri DN, Agarwal MK (1969) Studies on the allergenic fungal spores of Delhi (India) metropolitan area. J Allergy 44:204
Shivpuri DN, Dua KL (1974) Seasonal periodicity of house dust mite population. Allergy Appl Immunol 7:63–74
Shivpuri DN, Dar N, Menon MPS (1977) House mite asthma in India. In: Advances in chronic obstructive lung diseases, pp 143–150
Simplício EC, Silva DAO, Braga IA et al (2007) Mite and pet allergen exposure in hotels in Uberlândia, Midwestern Brazil. Indoor Air 17(4):278–283
Simpson A, Green R, Custovic A et al (2003) Skin test reactivity to natural and recombinant *Blomia* and *Dermatophagoides* spp. allergens among mite allergic patients in the UK. Allergy 58:53–56
Smith JM (1983) Epidemiology and natural history of asthma, allergic rhinitis and atopic dermatitis. In: Middleton E, Rud CE, Ellis EF (eds) Allergy: principles and practice, 2nd edn. St. Louis, Mosby, pp 633–658
Smith JM, Disney ME, Williams JD et al (1969) Clinical significance of skin reactions to mite extracts in children in asthma. Br Med J 2:723
Solarz K (1997) Seasonal dynamics of house dust mite populations in bed/ mattress dust from two dwellings in Sosnowiec (upper Silesia, Poland): an attempt to assess exposure. Ann Agric Environ Med 4:253–261
Solarz K, Szilman P, Szilman E (2004) Occupational exposure to allergenic mites in a Polish Zoo. Ann Agric Environ Med 11:27–33
Solomon ME (1961) Mites in houses, shops and other occupied buildings. The Sanatarian
Solomon ME (1962) Ecology of the flour mite, *Acarus siro* L. (=*Tyroglyphus farinae* DeG.). Ann Appl Biol 50:178
Somorin AO, Wasu-Hunponu OO, Mumcuoglu KY, Heiner DC (1978) Mite allergy in Nigerians; studies on house dust mites in houses of allergic patients in Lagos. Trish J Med Sci 147:26–30
Spieksma FThM (1967) Occurrence and properties of the house dust mite *Dermatophagoides pteronyssinus*. Int Rhinol 5:162–167
Spieksma FThM (1968) The house dust mite *Dermatophagoides pteronyssinus*, the cause of house dust allergy. Ent Ber Amst 28(2):27–38
Spieksma FThM (1969) Cultivation of house dust mites (letter). J Allergy 43:151–152
Spieksma FThM (1970) Biological aspects of the house dust mite (*Dermatophagoides pteronyssinus*) in relation to house dust atopy. Clin Exp Immunol 6(1):61–70
Spieksma FThM, Spieksma-Boezeman MIA (1967) The mite fauna of house dust with particular reference to the house dust mite *Dermatophagoides pteronyssinus* (Trt., 1897) (Psoroptidae, Sarcoptiformes). Acarologia 1:226–241
Spitz E, Gelfand EW, Sheffer AL et al (1972) Serum IgE in clinical immunology and allergy. J Allergy Clin Immunol 49:337–347

Srivastava N, Gupta SP, Srivastava LM (1983) Effect of house dust mite on serum complement activation and total haemolytic activity in vitro in normal subjects and in patients with bronchial asthma. Clin Allergy 13(1):43–50

Steinus B, Wide L (1969) Reaginic antibody (IgE), skin and provocation tests to *D. culinae* and house dust in respiratory allergy. Lancet 2:455–458

Storm van Leeuwen W, Bien Z, Varekamp H (1924) Experimentelle allergische Krankheiten (Asthama Bronchiale, Rhinitis Vasomotoria). Z Immunf Exp Ther 40:552–553

Strachan DP (1989) Hay fever, hygiene, and household size. BMJ 299:1259–1260

Strachan DP, Taylor EM, Carpenter RG (1996) Family structure, neonatal infection, and hay fever in adolescence. Arch Dis Child 74:422–426

Sugerman AA, Southern DL, Curran JF (1982) A study of antibody levels in alcoholic, depressive and schizophrenic patients. Ann Allergy 48:166–171

Sundaram P, Ravindran P, Jorhi M (1980) House dust mite allergy and bronchial asthma. Aspects Allergy Appl Immunol 13–14:30–33

Takaoka M, Okada S (1984) Ecological studies on the house dust mites in Saitama prefecture Japan; seasonal occurrence of the pyroglyphid mites in house dusts in 1981 to 1982. Jap J Sanit Zool 35(2):129–137

Takaoka M, Ishii A, Kabasawa Y et al (1977) Mite fauna in house dust collected from the residence of asthmatic children. Jap J Sanit Zool 28(2):237–244

Tan WC, Teoh PC (1979) An analysis of skin prick test reaction in asthmatics in Singapore. Ann Allergy 43:44–46

Tandon N, Batabyal SK, Saha GK et al (1988) Dust mites in relation to naso-bronchial allergy in Calcutta—a preliminary report. Indian J Allergy Appl Immunol 2(1):11–14

Tandon N, Mitra SB, Saha GK et al (1990) Role of cockroaches in allergy to house dust in Calcutta, India. Ann Allergy 64:155–157

Tee RD, Cranston PS, Dewair M et al (1985) Evidence for haemoglobins as common allergenic determinants in IgE-mediated hypersensitivity to chironomids (non-biting midges). Clin Allergy 15:335–343

Tiana B, Fernando MC, Carolina FR et al (2006) Dust mite species and allergen concentrations in beds of individuals belonging to different urban socioeconomic groups in Brazil. J Asthma 43(2):101–105

Tovey ER, Chapman MD, Platts-Mills TAE (1981) Mite faeces are a major source of house dust allergens. Nature 289:592–593

Tripathi DM, Parikh KM (1983) Mite fauna and other allergens present in the house dust in Bombay. Lung India 1(4):147–151

Troussart EL (1901) *Surdeux especes*, format in genre noureae de sarcoptides detriticotes parasites des fourrures. Bull Soc Zool France 26:83–84

Tuft L, Heck VM (1974) Mites as antigenic excitants of house dust allergy-report of some allergy studies. Ann Allergy 33:325

Turner KJ, Dowse GK, Stewart GA et al (1986) Studies on bronchial hyperreactivity, allergic responsiveness and asthma in rural and urban children of the highlands of Papua New Guinea. J Allergy Clin Immunol 77(4):558–566

Uchio E, Matsura N, Matsumoto S et al (2001) Histamine release test and measurement of antigen—specific IgE antibody in the diagnosis of allergic conjunctival diseases. J Clin Lab Anal 15:71–75

Valandikar SC, Channa Basavanna GP (1992) Some faunistic studies on house dust mites in Karnataka. In: Haq MA, Ramani N (eds) Man, mites & environments. Anjengo Pub, Calicut, pp 111–118

Van Zijverden MA, Van der Pijl M, Bol M et al (2000) Diesel exhaust, carbon black, and silica particles display distinct Th1/Th2 modulating activity. Toxicol Appl Pharmacol 168:131–139

Virchow C, Roth A, Moller E (1976) IgE antibodies to house dust, mite, animal allergens and moulds in house dust hypersensitivity. Clin Allergy 6:147–154

Viswanathan R (1966) Epidemiology of asthma in an urban population, a random morbidity survey. J Med Assoc India 46:480–483

Vobrazkova E, Kasiakova A, Samsinak K (1986) Analysis of dust samples from the clinical environment of children with eczemas. Angew Parasitol 27(1):53–55

Von Pirquet C (1906) Allergy. Munchen Med Wschr 53:1457–1458

Voorhorst R (1969) Further research on house dust atopy in relation to the house dust mite. Respiration 26:151–162

Voorhorst R, Spieksma FThM (1973) House dust atopy and the house dust mite *Dermatophagoides*. Allergol Immunopathol 1(1):13–24

Voorhorst R, Spieksma-Boezeman MIA, Spieksma FThM (1964) Is a mite (*Dermatophagoides* sp.) the producer of house dust allergen? Allergy Asthma 10:329–334

Voorhorst R, Spieksma FThM, Varekamp H et al (1967) The house dust mite (*Dermatophagoides pteronyssinus*) and the allergens it produces. Identity with the house dust allergy. J Allergy 39:325–335

Waki S, Matsumoto K (1973) Studies on the environmental requirements for the feeding of the dust mite, *Dermatophagoides farinae* Hughes 1961. Part I. Observations on the breeding under various temperature and humidity conditions. Jap J Sanit Zool 23:159–163

Warner A, Bostrom S, Moller G et al (1998) Mite fauna in the home and sensitivity to house dust and storage mites. Allergy 54:681–690

Wharton GW (1970) Mites and commercial extracts of house dust. Science 167(3923):1382–1383

Wharton GW (1976) House dust mites. J Med Entomol 12:577–621

Wide L, Bennich H, Johansson SGO (1967) Diagnosis of allergy by an *in vitro* test for allergen antibodies. Lancet 2:1105–1107

Williams GC (1961) Infestation in feed mills. Agr Merchant 41:72–80

Williams P, Barnes JH, Szeinbach SL et al (2000) Analytic precision and accuracy of commercial immuno-assays for specific IgE: establishing a standard. J Allergy Clin Immunol 105:1221–1230

Wongsathuaythong S, Lakshana P (1972) House dust mite survey in Bangkok and other provinces in Thailand. J Med Assoc Thailand 55(5):272–286

Wood LG, Gibson PG (2009) Dietary factors lead to innate immune activation in asthma. Pharmacol Ther 123(1):37–53

Wood CBS, Oliver J (1972) Serum IgE in asthmatic children. Relation to age, sex, eczema and skin sensitivity. Int Arch Dis Child 47:890–896

Yagofarov FE, Galikeev KHL (1987) Some data on the ecology of house dust mites in the conditions of the town of Semipalatinsk. Parazitologiya 21(2):148–151

Yamashita T, Kasuya S, Noda N et al (1994) Transmission of *Rickettsia tsutsugamushi* strains among humans, wild rodents, and trombiculid mites in an area of Japan in which tsutsugamushi disease is newly endemic. J Clin Microbiol 32(11):2780–2785

Yoshikaya M, Hanaoka K, Yamada Y et al (1983) Experimental proof of itching papules caused by *Cheyletus malaccensis* (Oudeman). Ann Rep Tokyo Metrop Res Lab Publ Health 34:264–276

Zetterstorm O, Johansson SGO (1981) The concentrations measured by PRIST in serum of healthy adults and in patients with respiratory allergy. Allergy 36:537–547

Zheltikova TM, Petrova-Nikitina AD, Kanchurin AKh et al (1985) House dust mites and allergies in man. Nauchnye Doklady Vysshei Shkoly, Biologicheskie Nauki. 2:12–30

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