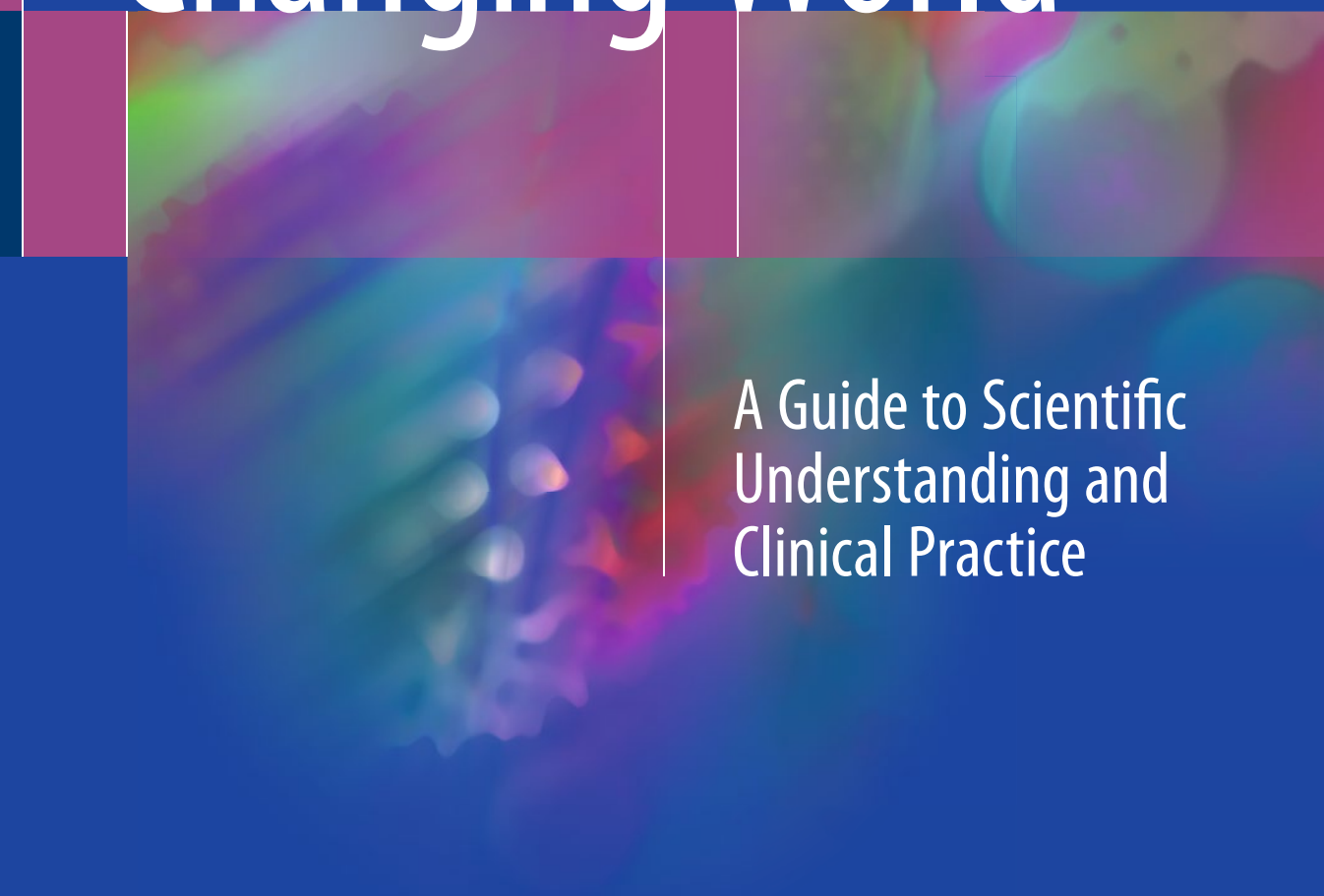
Jae-Won Oh

Pollen Allergy in a Changing World

A Guide to Scientific Understanding and Clinical Practice



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This book is dedicated to my dear Mrs. Eul-Won Moon, Ms. Seung-Yeon Oh, and Ms. Seung-Eun Oh for their heartful support and inspiration.

Preface

This book is primarily focused on allergic pollen which are the most common and important causative and triggering agents in respiratory allergy, and thus is written for the suffering people who are tormented by these interesting but often inconspicuous plants in a changing world with environmental change. A growing number of people are recently contracting allergic diseases caused by pollen because of climate change in the world. Moreover the seasonal and regional variations of pollen have been changed in Asian pacific countries as well as in Europe and America. As the sensitization rate to pollen has increased in children recently, allergic plants which are rapidly proliferating, has emerged as a dangerous element to allergic children. It is now essential to survey pollens around the patient for the management of pollen allergy.

Plants causing allergies are difficult to identify because they seldom have prominent flowers. It is strongly proposed that the reason behind this is not only the rapid proliferation of weeds and trees, but also air pollution such as greenhouse gases caused by the increase of traffic and the construction of apartments and factories, and increased temperature resulted from climate change which expedites the proliferation of plants such as ragweed and -provides a harmful environment to allergies. Nobody can be protected from allergy plants by staying in the cities as well as in rural areas. Allergenic plants grow abundantly in areas where man is disturbing the natural environment for his houses, roadways, even highways, and agricultural activities. They grow best in soil disturbed by human's activities and deforestation with a changing world. It is almost impossible to avoid plants which cause allergies, because pollen can travel many kilometers on the breezes or winds. However, the ability to avoid large doses is critical for sensitive people. The intensity of the allergic reaction depends on the amount of exposure.

Weather conditions, including rainfall, atmospheric temperature, humidity, wind speed and wind direction, may alter the concentrations of plant pollens and other allergens, which can subsequently influence the occurrence of allergic diseases such as asthma, allergic rhinitis, allergic conjunctivitis, and even atopic dermatitis. Many studies have demonstrated that CO_2 concentration and increased atmospheric temperature increase pollen concentration in the world. Most work on the impact of climate change on aeroallergens can be divided into a number of distinct areas, including impact on pollen amount, pollen allergenicity, pollen season, plants and pollen distribution. Although few observations and estimates were reported regarding season start and length of allergic pollen season in other countries including the USA, earlier

start dates and rising pollen concentrations have been reported widely in many European countries. The growing degree hour model was used to establish a relationship between start and end dates of pollen production and differential temperature sums using observed hourly temperatures from surrounding meteorology stations. Studies of climate change effects on distributions of allergenic pollens have focused typically on analysis of observed pollen counts and their regression relationships with local meteorological and climatic factors. The onset, duration and intensity (i.e. abundance of pollen grains in the air) of the pollen season vary from year to year. Weather variables, mainly air temperature, sunlight and rainfall, together with CO_2 are among the main factors affecting phenology (that means the times of the appearance of first leaves, first flowers, autumn leaf coloration and so on) and pollen production by plant. In addition weather patterns influence the movement and dispersion of all aeroallergens in the atmosphere through the action of winds, rainfall and depending on the atmospheric stability.

This book is not only designed to assist allergists, physicians, and medical students to guide to clinical practice of pollen allergy with information of climate change in the world, but also published to help allergic sufferers recognize the cause of their misery, the allergenic plants. Scientific names must be used to be certain of the identity of plants and rare usual botanical words have been avoided if possible. It would be more comfortable to use common names, but there are so many different common names for most plants that the use of these names might cause confusion.

Seoul, South Korea Jae-Won Oh

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Contents

Allergy and Pollen

1

1.1 What Is Allergy?

The understanding of allergy is growing exponentially with the ongoing expansion in our knowledge of the immune system. The immune system is the capacity to distinguish harmful non-self molecules from self-molecules, a characteristic that exists in a delicate balance between tolerance to self and response or rejection of non-self. This system is focused on host defense and is composed of specific cellular and protein components that develop and function in a highly complex manner, in order to neutralize or destroy dangerous non-self while preserving self.

Allergic responses are clear examples of the detrimental side of the immune system, because they represent immunological reactions to innocuous environmental elements, resulting in annoying and sometimes debilitating effects in the affected individuals. The allergic response involves hypersensitivity reactions directed at allergens, in contrast to similar hypersensitivity mechanisms that provide host protection to parasitic infections. Autoimmunity defines a state in which tolerance to self is lost and the immune response is activated against host tissues. Otherwise, allergic and hypersensitivity reactions are the result of immune responses to innocuous non-self molecules.

Allergy is a kind of disorders in which this balance is disrupted, which is an abnormal reaction to a very small amount of a specific substance, called an allergen which is usually proteins or glycoproteins of a molecular weight of 10,000–60,000 Da. This substance is harmless to general people who do not have this particular allergy. Allergens stimulate the production of allergic antibodies or of sensitized cells. This response is mediated by immunoglobulin (Ig) E antibody specific to the allergen. Mast cells and basophils are activated after IgE binding, starting a series of cellular and molecular events that results in the clinical manifestations of allergic disease. Allergens usually contained in pollens, molds, animal skin cells, danders, or hairs, house dust, insects, medication, or even foods such as milk, eggs, soy, wheat or nuts, fish, shellfish. These allergens cause trouble when they are inhaled, or swallowed, or injected into the body [1].

Allergic sensitization can be arisen *de novo* at any stage of life. However it is much more common for these diseases to appear initially in a mild form during childhood. Indeed, particularly in the case of allergy, the transient appearance of IgE against ubiquitous environmental allergens in early childhood is so frequent within the overall population that it can be classed as normal and it is only in a small subset of children that these responses fail to resolve spontaneously and instead persist and consolidate, leading to clinically significant allergic symptoms. It is also becoming increasingly evident that this period in early life represents a crucial window period of opportunity for modulation of these responses before they become persistent. There is accordingly interest

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in definition of the underlying regulatory mechanisms at play within the immature immune system at this time, as these constitute potential therapeutic targets. Moreover, although atopic sensitization is an important risk factor for diseases such as asthma, it is also evident which infers that other important cofactor are involved that operate against the atopic background to produce allergy.

A reaction upon exposure to these pollen allergens was called hay fever in the past; however, the expression is a misnomer. Hay fever is neither caused by hay nor is there any fever although the weakened body may develop various infections. This condition must be seasonal allergic rhinitis in which the lining of the nose becomes swollen and exudes a watery discharge, the nose and palate itch, and there are frequent sneezes. The eyes may become itchy, reddened, and runny, and some peoples may proceed to chronic rhinitis with conjunctivitis or chronic ear infections. Another reaction to allergens is asthma. In asthma there may be hard to breath with wheezing as the air passes through narrowed bronchus or bronchioles. In addition, there are other allergic reactions such as urticaria, angioedema, and anaphylactic shock. These seldom occur from ordinary exposure to allergic plants. Allergens are so small that they are not usually seen, and thus it often seems mysterious that the allergic person can detect such a small amount of the allergen [2].

1.2 The Overview of Allergic Immune Mechanism

Allergic diseases such as allergic rhinitis, asthma, and food allergy are characterized by the ability to make an IgE antibody response to an environmental allergen. There is both a strong genetic and environmental contribution to the development of allergic disease. IgE-mediated allergic responses most frequently occur on mucosal tissue in nose, conjunctiva, airway, and gastrointestinal tract or skin surfaces as these anatomical sites contain high levels of mast cells to which IgE is linked.

Initial exposure of a genetically predisposed individual to low levels of allergens such as tree, grass or weed pollens results in uptake of the pollen allergen by antigen-presenting cells (APCs), intracellular digestion of the allergen into peptide fragments, and display of the allergen peptide fragments in a human leukocyte antigen (HLA) groove on the APC surface. When circulating T cells (expressing an antigen cell surface receptor specific for the allergen peptide) interact with the APC, the interaction activates the T cell to express cytokines characterized by a helper T cell type 2 (Th2) cytokine profile. Th2 cytokines such as interleukin (IL)-4 play an important role in inducing B cells to switch class and express IgE, induce eosinophil proliferation in the bone marrow induced by IL-5, and upregulate adhesion molecules on blood vessels to promote tissue infiltration of circulating inflammatory cells associated with allergic inflammation such as eosinophils and basophils. The allergen-specific IgE (induced by initial exposure to allergen) binds to high-affinity IgE receptors on mast cells and basophils. These IgE sensitized mast cells upon re-exposure to specific allergen are activated to release histamine, cysteinyl leukotriene, and many other proinflammatory mediators that contribute to the allergic inflammatory response. Although this induction of a Th2 response is characteristic of allergic inflammation, it is increasingly evident that additional immune and inflammatory responses contribute to allergic inflammation.

The innate immunity presents the first line of host defense. The specificity of innate immunity is based on pattern recognition, involving molecules that are shared by multiple microbes, however not present in the host. The innate immune system comprises cellular elements that are both resident in tissues (i.e., epithelium, macrophages, mast cells) for a rapid response and circulating leukocytes that are recruited from the blood stream such as neutrophils, eosinophils, basophils, mononuclear cells, natural killer (NK) cells, and NKT cells. In addition to the cellular response the innate immune system has humoral elements, which provides a mechanism for an immediate response to infection that is not anti-

gen specific and does not have immunological memory. The group of proteins that circulate in the blood called the complement system and diverse blood cells contact with a pathogen or an infected cell, either directly induce cytotoxicity or initiate a phagocytosis process and intracellular killing.

Adaptive immunity mechanisms are designed to specifically recognize and distinguish a large number of molecules, together with an ability to amplify the response with repeated exposures to the same pathogen or molecule. Thus, recognition of a particular pathogen by the adaptive immunity may result in enhanced killing by phagocytes. Conversely, activation of complement proteins facilitates chemotaxis and migration of lymphocytes, which develop the adaptive response. A remarkable aspect of the adaptive immune system is its property of memory. The adaptive immune response generated by its component T and B cells is slower to respond to infections but has the advantage of exhibiting antigen specificity and immunological memory. Immunologic memory is made possible by the clonal expansion of lymphocytes in response to allergen stimulation. Since the human immune system first begins to differentiate in fetal life, uniquely reacting lymphocytes are created by the recombination of genes encoding antigen receptors expressed on the lymphocyte cell membrane. Through the expression of these receptors, each lymphocyte has the ability to bind to and become activated by a specific antigen, either natural or artificial. Interaction with antigen not only activates the lymphocytes but also results in the generation of long-lived antigen-specific memory cell clones. Thus, when the same antigen enters the body, there is immediate recognition by these memory cells. Both cellular and humoral responses to the antigen are produced more rapidly than in the first encounter, and more memory cells are generated. This process of expansion of clonal populations of uniquely reacting lymphocytes first explained the B cell origin of antibody diversity and applies to cellular immune responses as well. Thus, the cellular and molecular mechanisms through which an aberrant immune response to low levels of otherwise innocuous and ubiquitous environmental exposures such as pollens or foods may trigger a range of allergic responses from chronic symptoms affecting quality of life to acute severe allergic reactions that are life threatening [3, 4].

1.3 Who Has Allergic?

It is unusual to be allergic at birth. Most allergic individuals inherit only the capacity to become allergic. They usually become sensitized to the allergen during an infection or when exposed to excessive air pollution. The mucous membranes along the respiratory tract and intestinal tract are quite efficient in keeping foreign materials from entering the body's tissues during normal health. However, inflammation caused by infections or air pollution disrupts the mucous blanket and allows penetration of the foreign proteins in an unaltered state. The body's immune system then makes antibodies which are specific for these allergens. On later exposure to these allergens, even when the mucous membrane is intact, the immune system stimulates the release of agents by specialized cells such as B, T cells, mast cells, and so on to prevent invasion by these foreign substances. These agents produce edema, congestion with increased blood flow to the local tissues, and watery secretions. These responses result in the allergic symptoms, which really are somewhat like common cold symptoms. On repeated exposure to the allergens, some patients become more and more highly sensitized. Other patients tend to do better in time.

Some of the possible causes accounting for a number of observations of the increased prevalence of allergic disorders in urban communities are related to the environment, such as ambient pollution, increased concentration of indoor allergens, diet, and the decrease of childhood infections. The hygiene hypothesis is based on the possible immunomodulation induced by bacterial and viral infections early in infancy, modifying the chances of developing an allergic response. However, environmental factors do not fully explain the increase of allergic disease. Genetic predisposition to allergic disorders has also been extensively explored recently, as

it has been known that children of allergic parents are more critical to develop allergic disease. Genetic studies, including linkage analysis of large families, have identified several possible loci containing candidate genes that may confer increased susceptibility to allergic disease. It is not unexpected that allergic disorders, with their variety of presentations, may result from a complex interplay between a genetic predisposition of particular populations and specific environmental factors [5–11].

1.4 What Is Aeroallergen?

Aeroallergens are airborne particles that can cause respiratory or conjunctival allergy. Aeroallergens are named using nomenclature established by an International Union of Immunologic Societies subcommittee: the first three letters of the genus, followed by the first letter of the species and an Arabic numeral. Allergens from the same group are often given the same numeral [12]. For example, Lol p 1 and Phi p 1 are both group I grass allergens (Table 1.1).

For a particle to be clinically significant as an aeroallergen, it must be buoyant, present in significant numbers, and allergenic such as ragweed pollen. Pine pollen, by contrast, is abundant in certain regions like Korea, Japan, and China, and is buoyant, but because it does not readily elicit IgE antibodies, it is not a significant aeroallergen. In general, the insect-pollinated plants do not produce appreciable amounts of airborne pollen, as opposed to wind-pollinated plants, which, by necessity, produce particles that travel for miles. Fungal spores are ubiquitous, highly allergenic, and may be more numerous than pollen grains in the air, even during the height of the pollen season. The house dust mite is very common and important indoor allergen. The above allergens are emphasized because they are the ones most commonly encountered, and they are considered responsible for most of the morbidity among atopic patients. Certain aeroallergens, such as animal dander, feathers, and epidermal antigens, may be localized to individual places. Others may be associated with occupational exposures, as is the case in veterinarians who work with certain animals, in farmers who encounter a variety of pollens and fungi in hay and stored grains, and in bakers who inhale flour [13].

Observations that allergic symptoms due to ragweed may persist for days after intact airborne pollen is no longer detectable, spurred studies demonstrating aeroallergens in submicronic particles, presumably from fragmented pollen grains as submicronic allergenic particles. Airborne birch allergenic activity also has been demonstrated on particles smaller than 2.4 μm. Starch granules are prominent in the cytoplasm of certain pollens such as the grasses (Poaceae) and docks (Rumex, species of Polygonaceae). Such grass starch granules have heavy concentrations of major allergen groups. The force of storm-driven raindrops may disrupt pollen grains, releasing large amounts of respirable allergen-laden particles [14–18].

Fungi constitute one of the kingdoms of living organism. Fungi are eukaryotic organisms with chromosomes within membrane-bound nuclei, dividing through mitosis. These organisms have chitin-containing cell walls, a polysaccharide found also in insect exoskeletons. Fungi may be unicellular; syncytial, with many nuclei not divided into separate cells; or multicellular, with nuclei separated by septa. Life cycles frequently are very complex, with multiple life stages having either sexual or asexual reproduction. Holomorph refers to the whole fungus with its varying life stages, which is comprised of the anamorph (asexual reproductive) stages plus the teleomorph (sexual reproductive) stages. Frequently, only the anamorph or the teleomorph is identified, and the alternate life stages are not known. Other major phyla of fungi include Ascomycota, characterized by spores developing in sacs, or asci, in groups of eight; Zygomycota, containing typical bread molds, and producing sporangia containing numerous spores; and Basidiomycota, containing mushrooms, puffballs, smuts, and rusts, with basidiospores produced in quartets on basidia. Oomycota, containing powdery mildew (Oidium), has been reclassified out of the fun-

Table 1.1 Classification of allergenic plants

Class	General name	Species (genus name plus specific epithet)	Allergenicity
Fagaceae	Oriental chestnut oak Daimyo oak Oak Oriental white oak Mongolian oak Beech	*Quercus acutissima* Carruth. *Q. dentata* Thunb. *Q. variavilis* Bl. *Q. aliena* Bl. *Q. mongolica* Fisch. ex Ledeb. *Fagus engleriana* Seem. ex Diels	+++
Betulaceae	Birch Alder Hazelnut	*Betula platyphylla* var. japonica H. *Alnus japonica* (Thunb.) Steud. *Corylus heterothylla* Fisch. ex Trautv.	+++
Salicaceae	Korean willow Italian poplar	*Salix koreensis* Ander. *Populus euramericana* Guinier	+
Ulmaceae	Japanese elm Hackberry	*Ulmus davidiana* var. japonica Nakai *Celtis sinensis* Persoon	++
Platanaceae	Planetree	*Platanus occidentalis* L.	+
Aceraceae	Maple	*Acer palmatum* Thunb.	+
Juglandaceae	Walnut	*Juglan sinensis* Dode	+
Oleaceae	Korean ash	*Fraxinus rhynchophylla* Hance	+
Taxodiaceae	Japanese cedar	*Cryptomeria japonica* (L. f) D. Don	+++
Pinaceae	Pine	*Pinus densiflora* Siebold & Zucc.	+
Asteraceae	Common ragweed Giant ragweed Sagegrush Wormwood Mugwort	*Ambrosia artemisiifolia* L. *A. trifida* L. var. trifida *A. trifida* for. integrifolia (Muhi.) Fern. *A. montana* (Nakai) Pamp. *A. tridentata* Nutt. *A. absinthium* L. *A. vulgaris* L.	+++
Amaranthaceae	Green amaranth Wild amaranth Slender amaranth	*Amaranthus mangostanus* L. *A. retroflexus* L. *A. lividus* L. *A. viridis* L.	++
Chenopodiaceae	Goosefoot Mexican tea	*Chenopodium album* var. centrorubrum Makino *C. ambrosioides* L. *C. glaucum* L. *C. bryoniaefolium* Bunge	++
Cannabaceae	Japanese hop	*Humulus japonicus* Siebold et Zucc.	+++
Plantaginaceae	Asian plantain English plantain	*Plantago asiatica* L. *P. lanceolata* L.	+++
Polygonaceae	Common sorrel Sheep sorrel Curly dock	*Rumex acetosa* L. *R. acetosella* L. *R. crispus* L.	+
Urticaceae	Nettle	*Urtica thunbergiana* Siebold et Zucc.	±
Gramiceae	Lawngrass Bermuda grass Timothy grass Orchard grass	*Zoysia japonica* Steud. *Cynodon dactylon* (L.) Pers. *Phleum pratense* L. *Dactylis glomerata* L.	++

gal kingdom and into the kingdom Chromista, owing to a lack of chitin in cell walls. Slime molds, Myxomycetes, are likewise not classified as true fungi. Both of these latter two groups have members incriminated as allergens [19].

Although animal sources are primarily indoors as pets, some may be significant outdoor allergens as well. Heavy hatches of caddis flies or mayflies or miller moth infestations have been reported to induce allergic symptoms. Occupational expo-

sures to sewer flies, municipal sanitation workers. Horse dander allergen may be sampled outdoors downstream of stables [20, 21].

1.5 Pollen As Aeroallergen

Pollen size and weight as aeroallergens as they fall in the 20–60 μm range of most particulate matter and their allergenic constituents are protein with a proteins with a molecular weight between 10,000 and 40,000 Da. Protective mechanisms in the nasal mucosa and upper tracheobronchial passages remove most of the larger particles, so only those 5 μm or smaller reach the alveoli of the lungs. These are considerations in the pathogenesis of allergic rhinitis, bronchial asthma, and hypersensitivity pneumonitis as well as the irritant effects of chemical and particulate atmospheric pollutants [22].

The development of asthma after pollen exposure is enigmatic because pollen grains are deposited in the upper airways as a result of their large particle size. Experimental evidence suggests that rhinitis, but not asthma, is caused by inhalation of whole pollen in amounts encountered naturally. Pollen asthma may be caused by the inhalation of pollen debris that is small enough to access the bronchial tree. Evidence supports this hypothesis. Extracts of materials collected on an 8-μm filter that excludes ragweed pollen grains induced positive skin test results in ragweed-sensitive subjects. Using an immunochemical method of identifying atmospheric allergens, Amb a 1 was found to exist in ambient air in the absence of ragweed pollen grains. Positive bronchial challenge test was induced with pollen grains that had been fragmented in a ball mill, but was not induced by inhalation of whole ragweed pollen grains. Exposure of grass pollen grains to water creates rupture into smaller, respirable size starch granules with intact group V allergens, possibly explaining the phenomenon of thunderstorm asthma during grass pollen seasons. However, despite the generally accepted limitations previously mentioned, examination of tracheobronchial aspirates and surgical lung specimens has revealed large numbers of whole pollen grains in the lower respiratory tract [22, 23].

Another consideration is the rapidity with which various allergens are leached out of the whole pollen grains. The mucous blanket of the respiratory tract has been estimated to transport pollens into the gastrointestinal tract in less than 10 min. The allergens of grass pollens and ragweed Amb a 5 are extracted rapidly from the pollen grains in aqueous solutions and can be absorbed through the respiratory mucosa before the pollen grains are swallowed. But ragweed Amb a 1 is extracted slowly, and only a small percentage of the total extractable Amb a 1 is released from the pollen grain in this time frame. This observation has not been reconciled with the presumed importance of Amb a 1 in clinical allergy, but absorption may be more rapid in the more alkaline mucus found in allergic rhinitis [24].

1.6 Evaluation of Pollen Allergy

Pollens stimulate immunoglobulin E (IgE) antibody production in susceptible persons when they impact mucous membrane with specific allergen, which can result in a reaction between the allergen and the fixed IgE antibodies, causing a release of mast cell mediators of acute inflammation leading to clinical allergic disease. Allergic rhinitis and asthma are referred to as IgE-mediated diseases because total IgE levels are elevated and specific IgE sensitization is present. Allergic rhinitis, suggested by a patient's history of symptoms that seemingly follow a seasonal pattern, should not be assumed unless confirmed by appropriated laboratory tests.

Laboratory tests for IgE antibodies are of two types: direct skin test and the quantitative serum test for allergen-specific IgE such as Unicap system, RAST (radioallergosorbent test), MAST (multiple allergosorbent test). Increased levels of a pollen allergen-specific serum IgE are an indication of sensitization of the pollen. However, as with skin tests, the clinical relevance of a positive test still needs to be determined. Each attempts to diagnose a patients' sensitization to allergens.

Skin testing is done usually by either the prick or intradermal technique and has the advantage of being relatively inexpensive, easy to perform, and is usually sensitive and reliable. Prick testing is the safest of two, but may produce less reliable results. Intradermal testing can, on occasion, produce dangerous reactions; false positive reactions may occur if the antigens are too concentrated. Serum testing is costly but is less traumatic and may be indicated when patients have eczema or other skin problems that would adversely affect skin testing in the affected area. Perhaps the greatest advantage of skin testing is that the results are almost immediately available, while serum test results may take a considerable length of time to obtain (See Chapter 7.5).

For allergy testing, a good patient history can help isolate suspected allergens, but it must be as accurate as memory and medical records can make it. Because inhalant allergens and their numbers can change dramatically from day to day, a patient's faulty memory can send his physician off on a wild goose chase and prevent identification of the offensive allergen and delay necessary therapy. It is helpful if patients maintain a daily record of overall symptoms index along with the symptoms they experienced on specific days [25].

1.7 Factors Influencing Clinical Significance of Pollen

Pollens are typical frequently encountered aeroallergens. In general, entomophilous (insect-pollinated) plants produce scant, heavy, or sticky pollens that do not become airborne. Anemophilous (wind-pollinated) plants represent only about one-tenth of the more than 250,000 pollen-producing species. Depending on their season, anemophilous spores can reach concentrations over 100 grains/m^3, can remain airborne for days, and can be carried hundreds of miles from their point of origin. In contrast to entomophilous plants, anemophilous plants possess large stamens borne on long, well-exposed filaments, often organized as catkins. Their flowers usually lack color, scent, and nectar and release large quantities of pollen in warm, dry weather. Particle size is a critical physical attribute of aeroallergens and an important consideration in the pathogenesis of allergic rhinitis, asthma, hypersensitivity pneumonitis. Protective mechanisms in the nasal mucosa and upper tracheobronchial passages remove larger particles, so only those of 5 μm or less reach the alveoli of the lungs. Pollen grains range in size from about 15–75 μm. Thus the conjunctivae and upper respiratory tract are exposed to the highest dose of aeroallergens [3].

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