

World Allergy Organization Guidelines for Prevention of Allergy and Allergic Asthma

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The prevalence of asthma and allergy is increasing. It is estimated that over 20% of the world's population suffer from immunoglobulin E (IgE)-mediated allergic diseases such as asthma, rhinoconjunctivitis, eczema, and anaphylaxis. Asthma, which in more than 50% of adults and in at least 80% of affected children is allergic, occurs in around 5–15% of the pediatric population. Asthma is estimated by the World Health Organization (WHO) to affect 150 million people worldwide, placing an enormous strain on health resources in many countries, and is a major cause of hospitalizations for chronic diseases in children in the western world.

Prevention of Allergy and Allergic Asthma was a collaborative project between the World Allergy Organization (WAO) and the World Health Organization (WHO) [1]. This document is a condensed version of the full Prevention of Allergy and Allergic Asthma Report [2].

The strategic guidelines in this document have been developed to provide a sound basis for practical action to be taken by governments, health care professionals, lay organizations, and patients.

Keywords: allergy, asthma, prevention, education, strategy

Allergy Clin Immunol Int – J World Allergy Org 2004; 16:176–185

Introduction

In the westernized world, allergy and asthma have increased two- to threefold over the last forty years and have reached epidemic proportions. The increase in these diseases has occurred

over a period of persistent environmental and lifestyle changes. Epidemiological studies have shown great variation in the prevalence of asthma and allergies between different nations. Valuable information may be derived from areas where a rapid increase in disease has occurred, to form the basis for prevention strategies in areas where the prevalence of these diseases is still low.

The strategic guidelines in this document have been developed to provide a sound basis for practical action to be taken by governments, health care professionals, lay organizations, and patients at a national level. Much of the research on which the guidelines are based has been conducted in countries that have experienced this rapid increase in allergies and asthma, and so will require interpretation and adaptation to meet different local circumstances. Within any country, climatic and environmental variations, and differences between affluent and non affluent groups may require a broad range of strategies and advice to be available. It is hoped that the general principles of prevention proposed in this document will provide a useful basis for local guidelines to be developed. Further actions are recommended in each section to assist the strategic development of national and international research programs.

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TABLE 1
WHO CATEGORIES OF EVIDENCE

Ia	Evidence from meta-analysis of randomized controlled trials
Ib	Evidence from at least one randomized controlled trial
IIa	Evidence from at least one controlled study without randomization
IIb	Evidence from at least one other type of quasi-experimental study
III	Evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies and case-control studies
IV	Expert opinion of the PAAA working group

This document focuses mainly on primary prevention of the allergen-specific IgE immunological sensitization that is the basis for atopic allergy and for much of asthma. Primary prevention measures should be implemented if they meet the following criteria:

- they should be of potential benefit to the majority of the population;
- they should be of no known harm to anyone;
- they should not involve unreasonable costs.

Secondary prevention is also covered in some detail since adequate diagnosis and treatment of allergic diseases such as eczema and allergic rhinitis at a young age can prevent the later development of asthma.

Tertiary prevention is mentioned briefly; it is covered in detail elsewhere, for example in the Global Resources in Allergy (GLORIA) program of the World Allergy Organization (WAO), the Global Initiative for Asthma (GINA) document [3], and the Allergic Rhinitis – its Impact on Asthma (ARIA) document [4].

Nonallergic asthma is mentioned in this report since some secondary prevention strategies may be of value in this disorder. Although the causes are less well defined, similar inflammation is present in the airways of patients with nonallergic asthma as in allergic asthmatics.

Evidence Base

In creating the full Prevention of Allergy and Allergic Asthma document, authors were asked to code referenced documents in accordance with the World Health Organization (WHO) Categories of Evidence. On the basis of the categorization of evidence, a strength of recommendation may be awarded to the ensuing guidelines. The Categories of Evidence are more easily applied to therapeutic interventions than to epidemiological and basic research studies, where randomization or blinding may not be possible. It may not be possible, for example, to code a well-designed physiological study by these criteria. Thus,

TABLE 2
WHO STRENGTH OF RECOMMENDATIONS

A	Directly based on category I evidence
B	Directly based on category II evidence or extrapolated recommendation from category I evidence
C	Directly based on category III evidence or extrapolated recommendation from category I or II evidence
D	Directly based on category IV evidence or extrapolated recommendation from category I, II or III evidence

a recommendation awarded a 'b' cannot always be interpreted as a lesser recommendation than one coded 'a' – it is an indication only of the type of studies which have created the evidence base; a recommendation awarded 'd' means only that at the present time there is an absence of evidence. Strength of recommendation categories have been restricted to the guidelines on Preventive Measures and Education, where a clear interpretation of the evidence base is possible.

Glossary and Definitions

The terminology proposed by the European Academy of Allergology and Clinical Immunology (EAACI) publication, "A Revised Nomenclature for Allergy" [5] has been updated by WAO in its publication "A Revised Nomenclature for Allergy for Global Use" [6]. The WAO nomenclature is used throughout this document.

Glossary

Allergy: Allergy is a hypersensitivity reaction initiated by immunological mechanisms. Allergy can be antibody- or cell-mediated. In the majority of cases, the antibody typically responsible for an allergic reaction belongs to the IgE isotype and these individuals may be referred to as suffering from an IgE-mediated allergy. Not all IgE-mediated allergic reactions occur in "atopic" subjects. In non-IgE-mediated allergy, the antibody can belong to the IgG isotype, e.g., anaphylaxis due to immune complexes containing dextran, and the classical, nowadays rare, serum sickness previously referred to as a Type II reaction. Both IgE and IgG antibodies are found in allergic bronchial pulmonary aspergilliosis (ABPA). Allergic contact dermatitis is representative of allergic diseases mediated by lymphocytes.

Allergens: Allergens are antigens which cause allergy. Most allergens reacting with IgE and IgG antibody are proteins, often with carbohydrate side chains, but in certain circumstances pure carbohydrates have been postulated to be allergens. In rare instances, low molecular weight chemicals, e.g., isocyanates and anhydrides acting as haptens, are still referred to as allergens for IgE antibodies. In the case of allergic contact dermatitis, the classical allergens are low molecular weight chemicals, e.g., chromium, nickel, and formaldehyde, reacting with T cells.

Atopy: Atopy is a personal and/or familial tendency, usually in childhood or adolescence, to become sensitized and produce IgE antibodies in response to ordinary exposure to allergens, usually proteins. As a consequence, such individuals can develop typical symptoms of asthma, rhinoconjunctivitis, or eczema. The terms *atopy* and *atopic* should be reserved to describe the genetic predisposition to become IgE-sensitized to allergens commonly occurring in the environment and to which everyone is exposed but to which the majority do not produce a prolonged IgE antibody response. Thus, atopy is a clinical definition of an IgE antibody high-responder. The term atopy cannot be used until an IgE sensitization has been documented by IgE antibodies in serum or by a positive skin prick test. Allergic symptoms in a typical atopic individual can be referred to as atopic, e.g., atopic asthma. However IgE-mediated asthma in general should not be called atopic asthma. Neither a positive skin prick test nor presence of IgE antibody to a less common allergen, e.g., *Hymenoptera* sting or a drug, which are high-dose exposures, is a diagnostic criterion for atopy.

Hypersensitivity: Hypersensitivity causes objectively reproducible symptoms or signs, initiated by exposure to a defined stimulus that is tolerated by normal subjects.

Prevention: *Primary Prevention* is the prevention of immunological sensitization (ie, the development of IgE antibodies). *Secondary Prevention* prevents the development of an allergic disease following sensitization (and the progression from eczema or rhinoconjunctivitis into severe diseases such as asthma). *Tertiary Prevention* is the treatment of allergic diseases and asthma.

Disease Nomenclature

Asthma (as defined by GINA): Asthma is a chronic inflammatory disorder of the airways in which many cells play a role, in particular mast cells, eosinophils, and T lymphocytes. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and/or in the early morning. These symptoms are usually associated with widespread but variable airflow limitation that is at least partly reversible either spontaneously or with treatment. This inflammation also causes an associated increase in airway responsiveness to a variety of stimuli. *Occupational asthma* is characterized by variable airflow limitation and/or airways hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace.

Allergic Asthma: This is the basic term for asthma mediated by immunological mechanisms. When there is evidence of IgE-mediated mechanisms, the term *IgE-mediated asthma* is recommended. IgE antibodies can initiate both an immediate and a late asthmatic reaction. However, as in other allergic disorders, T cell-associated reactions seem to be of importance in the late and delayed reactions. Depending on duration of symptoms, asthma can be referred to as either intermittent or persistent (as recommended in the ARIA document [3]).

Non-Allergic Asthma: This is the preferred term for nonimmunological types of asthma. It is recommended that the old terminologies, “extrinsic,” “intrinsic,” “exogenous,” and “endogenous” should no longer be used to differentiate between the allergic and nonallergic subgroups of asthma.

Rhinoconjunctivitis: Symptoms of an immunologically mediated hypersensitivity reaction in the nose and conjunctiva should be referred to as allergic rhinoconjunctivitis. Most cases are IgE mediated. Based on duration of symptoms, it can be useful to differentiate between intermittent and persistent allergic rhinoconjunctivitis, as described in the ARIA document [3].

Dermatitis: The umbrella term for a local inflammation of the skin should be dermatitis. What is generally known as “atopic eczema/dermatitis” is not one, single disease but rather an aggregation of several diseases with certain characteristics in common. A more appropriate term is eczema. The subgroup related to allergic asthma and rhinoconjunctivitis, i.e., eczema in a person of the atopic constitution, should be called atopic eczema. Close contact with low molecular weight chemicals may provoke a predominantly T-helper cell type 1 (Th1) lymphocyte-mediated allergic contact dermatitis. The nonallergic variety can also be described by terms like irritant/toxic contact dermatitis.

Genetics

At present, no genetic markers for IgE sensitization are known. Genes involved in the pathogenesis of allergy, and of asthma in general, have not been identified with certainty, although potential candidate genes have been defined. No firm practical recommendations to physicians or patients can yet be made from the results of genetic studies, but may be possible in the future.

Current Knowledge

- The atopic constitution is a major risk factor for the development of IgE sensitization
- Individuals with a family history of atopy have an increased risk of developing allergic diseases such as allergic asthma, allergic rhinoconjunctivitis, or atopic eczema
- The risk of developing allergic disease in a particular organ is related to family history of that disease
- The contribution of genetic factors to the development of IgE-sensitization, and to family history of an IgE-mediated disease, is 70–80%

Further Actions Required

Identification of genetic variants that predispose to allergic disease may result in several outcomes:

- Early identification of susceptible individuals would allow them to be targeted at an early age to prevent IgE sensitization, and for avoidance of allergen exposure. Genetic screening in early life may become useful in preventing allergic disease
- Identification of environmental factors that interact with susceptibility genes to trigger allergic disease, and the periods of life in which this occurs, may give opportunities for environmental modification
- Understanding susceptibility factors will help to find new drugs to treat and prevent allergic diseases. Genetic variants influence response to therapy and their identification will improve the efficacy of therapeutic measures

TABLE 3
IS THE ATOPIC CONSTITUTION HERITABLE?

Individuals with a family history of atopy have an increased risk of developing IgE sensitization
The risk of developing IgE antibody-mediated diseases (asthma, rhinitis, eczema) is related to family history of such disease
Studies have estimated the contribution of genetic factors to the development of IgE sensitization, and to family history of an IgE-mediated disease, at 70–80%

Environmental Influences

Environmental factors initiate and aggravate allergic diseases and asthma. It was initially thought that early infections increased the risk of allergic symptoms. However, by the late 1980s the hygiene hypothesis proposed that infections may prevent, rather than cause, allergic diseases, though their role is still not fully understood. In the early 1980s, outdoor air pollution was related to troublesome asthma and also considered as a possible cause of the increasing asthma incidence. Although outdoor air pollution may aggravate established asthma, it has not been shown to increase the incidence.

Research has moved from the study of factors that aggravate allergies and asthma, and examination of risk factors within populations, to studying differences between populations that might explain the development of these conditions. The majority of studies come from developed countries, where rates of asthma and allergic disease are generally higher than in developing countries. Studies such as the International Study of Asthma and Allergies in Childhood (ISAAC) [7] (see <http://isaac.auckland.ac.nz/> for details) will continue to bring important information from places where these conditions are rising in prevalence as lifestyle and environments change.

Current Knowledge

- Cigarette smoking in pregnancy deteriorates fetal lung development and increases the risk of wheezing in infancy
- Environmental tobacco smoke causes wheezing in infants and aggravates asthma. All children benefit from avoidance of environmental tobacco smoke
- Avoidance of allergen exposure has only partially been successful in prevention of IgE-sensitization. Avoidance is difficult to implement, benefits are small, and long-term effects doubtful
- Infants at high allergy risk (allergic disease in atopic parents or siblings) may benefit from avoidance of pets and dust mites during the first year of life
- Some early respiratory infections, e.g., pertussis and respiratory syncytial virus (RSV) bronchiolitis, as well as some forms of gastro-enteritis may enhance the development of allergic diseases and asthma
- Relative lack of microbial exposure (both gastro-intestinal and respiratory), particularly early in life, may enhance the development of allergic diseases

Further Actions Required

Study priorities in allergy related environmental issues are:

- Exploration of the environmental factors associated with residence in high versus low prevalence of allergy locations
- Effect of modulating the diet of pregnant and breastfeeding mothers (including increased intake of vegetables, fruit, cereals, nuts, starches, and fish)
- Effect of allergen avoidance in primary and secondary prevention, and in the treatment of allergic diseases (tertiary prevention)
- Protective effect of allergen exposure in the first year of life
- Effect of indoor air as well as ambient air pollutants (e.g., NO_x, ozone, fine particulates) in the development of allergic diseases and their aggravation

Early Immunological Influences

Current Knowledge

- Allergen-specific T-helper cell programming begins early in life and consolidates into the adult pattern during the preschool years. This programming is directly driven by dietary and inhalant allergens that are ubiquitous in the environment
- Early exposure to high levels of dietary or inhalant allergens usually results in high-zone tolerance, which protects against IgE sensitization. Postnatal development of allergen-specific responses is modulated by Th1 function, which develops through contact with microbial signs absent from the fetal environment.

- Early exposure to low levels of inhalant allergens in the absence of sufficient microbial exposure may trigger low-zone tolerance resulting in either weak Th1 immunity or Th2-polarized responses. This may increase the risk of IgE sensitization.

Further Actions Required

Study priorities are:

- Safe intervention strategies for infants at high allergy risk. As allergic manifestations are also increasingly common in children without family history of allergy, there is a need for easy-to-implement guidelines for all infants. Allergic diseases are difficult to modify once established
- Role of nutritional factors in infants in development of the immune and respiratory system
- Possibilities to induce postnatal high-zone tolerance to inhalant allergens by administering high doses of allergens
- Inhibition of Th2 function during the early stages of allergy development by new antagonists (against IgE, interleukin 4 (IL-4), IL-12, etc.) administered alone or in combination with allergen-specific immunotherapy
- Mechanisms underlying interactions between respiratory allergy and respiratory infections during infancy, in relation to long-term asthma outcomes
- Efficacy of antiviral agents in treatment of RSV-bronchiolitis and prevention of asthma in young children at high risk

Predictive and Early Diagnosis

Symptoms of the upper or lower airways related to IgE sensitization are not detectable at birth and rarely develop during the first two years of life. In many cases, airways symptoms are preceded by eczema, which has the highest incidence during the first three months of life and reaches the highest prevalence during the third year of life. Atopic eczema usually presents in children with a positive family history of atopy and allergic asthma or allergic rhinoconjunctivitis and is considered a prodromal state of later allergic respiratory allergy. Episodic wheezing is frequently observed in children from infancy onwards; in the majority of cases it is transient and does not result in persistent disease. A range of cofactors, both host and environmental, are involved in IgE-mediated diseases such as asthma; only a subset of school age children with signs of IgE sensitization develop allergic disease.

A prerequisite for secondary prevention is a correct diagnosis of individuals who have developed IgE sensitization. IgE antibody should be measured in the serum by an appropriate immunoassay or indicated in the skin by the skin prick test, using

standardized allergen extracts. In specialist clinics, target organ reactivity can be determined by challenge tests, e.g., nasal, conjunctival, and bronchial allergen challenge.

While we lack confirmed genetic and immunologic markers, primary prevention of IgE sensitization is difficult to achieve.

Current Knowledge

- Family history, taken by an experienced clinician, is the most reliable predictor of development of allergy and asthma in infants. If both parents have allergic asthma, allergic rhinoconjunctivitis, or atopic eczema, the child has a four-fold risk of developing allergies compared with a child whose parents do not have allergies. If only one parent has allergic disease, the risk is twofold
- Early signs of an allergic disease, especially atopic eczema, and presence of IgE antibodies to inhalant allergens are important risk factors for later respiratory allergy. IgE antibodies in infant serum to basic food proteins (e.g., hen's egg) may predict the so-called allergic march
- Current immunological markers obtained during the neonatal period are not specific or sensitive enough to predict allergic disease. High IgE concentration in cord blood or infant serum is specific for subsequent allergic disease but has a low sensitivity, and measuring total IgE is not recommended for screening

Further Actions Required

Mechanisms of initial IgE sensitization should be explored to develop markers necessary for early prediction.

Preventive Measures

Risk factors associated with asthma have been partially identified. Evidence is most complete for indoor allergens, particularly for house dust mites. Preventive measures to reduce mite exposure and other focused intervention strategies are appropriate for those at high risk. Tobacco smoke is a known risk factor for childhood respiratory disease and for occupational sensitization.

Guidelines

WHO strength of recommendations is given in brackets.

Primary Prevention

- Avoid smoking and exposure to environmental tobacco smoke, particularly during pregnancy and early childhood (B). Tobacco smoke should be also removed from work places (B)

- Avoid damp housing conditions (C), and reduce indoor air pollutants (C)
- Breastfeed exclusively until 4–6 months (B)*. No special diet for the lactating mother (A)
- Reduce exposure to inhalant allergens in young children at high risk (dust mites, cockroaches, furred pets) (B)
- Eliminate sensitizing and highly irritating agents in occupational environments (C). If this is not possible, implement measures to prevent employee exposure

Secondary Prevention

- Treat atopic eczema in infants and children to try to prevent respiratory allergy (D)
- Treat upper airways disease (rhinoconjunctivitis) to reduce risk of development of asthma (D)
- In young children already sensitized to house dust mites, pets, or cockroaches, exposure should be reduced to prevent onset of allergic disease (B)
- Remove employees from occupational exposure if they have developed symptoms caused by occupational allergic sensitization (C)

Tertiary Prevention

- Infants with cow's milk allergy should avoid cow's milk proteins; if a supplement is needed, use hypoallergenic formula, if available/affordable, to improve symptom control (B)
- Patients with asthma, rhinoconjunctivitis, or eczema who are allergic to indoor allergens such as dust mites, cockroaches, and animal danders should eliminate or reduce the exposure to improve symptom control and prevent exacerbations (A–B)
- Aim pharmacotherapy primarily towards the underlying inflammatory process (A)
- Avoid strictly acetyl salicylic acid or other nonsteroidal anti-inflammatory drugs (NSAIDs) in patients who have been diagnosed as being sensitive to them (C)

Allergen avoidance advice sheets are provided in Appendix 1.

Further Actions Required

Study priorities are:

- Detection of potent allergens in marketed readymade foods (also hidden allergens); declaration of the food allergen content should be improved

* Based on allergy prevention studies, exclusively breastfeeding and avoidance of solid foods for at least 4 months seems to be effective. WHO dietary guidelines recommend exclusively breastfeeding for 6 months in general. Infants who are not breastfed because their mothers are HIV positive, and who have consequently developed allergy to cow's milk, should receive hypoallergenic formula, if available, to improve symptom control (B).

- Effect of human milk, and the role of milk constituents (e.g., n3/n6 fatty acids, alpha-linolenic acid, cytokines, etc.) in the development of sensitization and clinical manifestations of allergy
- Effect of dietary factors in the development of allergy and asthma, e.g., salt intake, antioxidants, and n3/n6 fatty acids
- Effect of intestinal microbial flora, antibiotics, pro- and prebiotics, and microbial vaccinations on the development of sensitization and clinical manifestations of allergy
- Effect of low allergen domestic environments in sensitized children and in patients with allergic diseases. Environmental measures should be studied in terms of clinical benefit, feasibility, compliance, cost, and the interventions should be of adequate length, e.g., 12 months
- Effect of dust mite avoidance on allergic symptoms, exacerbation rates, need for medication, and health costs
- Role of indoor air and ambient air pollutants in the development of sensitization and clinical manifestations of allergy

Further Actions for Occupational Allergies

- Risks of occupational allergy should be monitored and epidemiological information collected by a globally agreed questionnaire
- High risk for allergy environments should be identified (e.g., enzymes, flours, latex, animals). In atopic employees who work in these environments, detection of sensitization by skin prick tests or IgE antibody measurements could prevent the development of clinical allergy
- General principles of prevention of occupational asthma, rhinoconjunctivitis, and dermatitis (hand eczema, contact dermatitis) should be published by national regulatory and advisory bodies

Education

Patient education programs in allergic disease and asthma have proven value in optimizing disease control and are cost-effective. The resulting increase in drug costs observed in some asthma studies, especially in low-income populations, and in the number of physician visits, are usually outweighed by the reduction in emergency visits and hospitalizations.

Guidelines

(WHO strength of recommendations is given in brackets.)

- Patient education regarding precipitants of allergic symptoms, asthma, and anaphylaxis is essential. Guided self-management to prevent, assess, and treat symptoms is the key to optimizing disease control (A)
- School policies on asthma and anaphylaxis management are essential (D)

Further Actions Required

Initiation of an International Coalition for Allergy and Asthma Prevention. The aims are:

- To collect effective programs into a database for all countries
- To establish an annual convention on allergy and asthma prevention, education and applied research for medical professionals from participating countries
- To establish an international allergy and asthma prevention & education promotion fund, supported by public and private funding
- To determine criteria to distribute funds as:
 - seed money to initiate program activities, and
 - matching funds to supplement the available funding
- To determine the criteria for membership of the International Coalition as an Allergy and Asthma Preventing & Education Promoting Country

Recommended topics for inclusion in patient education programs are shown in Appendix 2.

Costs of Allergies and Asthma

Asthma and other allergic diseases represent health conditions from which millions suffer physical impairments, reduction in quality of life, and economic impact. Prevention will lead to more effective use of health care resources.

The cost of asthma in the USA in the late 1990s was US \$12.7 billion; the indirect costs for allergic rhinitis accounted for an additional US \$1.2–7 billion annually and accounted for 36 million physician visits. In Australia, 1 million days are missed from school each year due to asthma. There is evidence that the costs are increasing, and are switching from hospital costs to medication costs. The economic impact of allergic diseases in developing countries with large populations – such as India, Indonesia, and China – has not been quantified.

Guideline

- When considering any intervention for prevention or treatment, take into account not only the evidence showing the beneficial effect of a certain product or measure but also the costs. Use the most cost-effective product or measure.

Further Actions Required

Study priorities are:

- Health economic impact of asthma, rhinitis, eczema, and the conditions together, especially in terms of indirect costs
- Societal costs of allergy and asthma in developing countries, and the development of these costs

TABLE 4
SUMMARY OF GUIDELINES
(WHO STRENGTH OF RECOMMENDATIONS IS GIVEN
IN BRACKETS)

Primary prevention

- Avoid smoking and exposure to environmental tobacco smoke, particularly during pregnancy and early childhood (B). Tobacco smoke should be also removed from work places (B)
- Avoid damp housing conditions (C), and reduce indoor air pollutants (C)
- Breastfeed exclusively until 4–6 months (B)*. No special diet for the lactating mother (A)
- Reduce exposure to inhalant allergens in young children at high risk (dust mites, cockroaches, furred pets) (B)
- Eliminate sensitizing and highly irritating agents in occupational environments (C). If this is not possible, implement measures to prevent employee exposure

Secondary prevention

- Treat atopic eczema in infants and children to try to prevent respiratory allergy (D).
- Treat upper airways disease (rhinoconjunctivitis) to reduce risk of development of asthma (D).
- In young children already sensitized to house dust mites, pets, or cockroaches, exposure should be reduced to prevent onset of allergic disease (B).
- Remove employees from occupational exposure if they have developed symptoms caused by occupational allergic sensitization (C).

Tertiary prevention

- Infants with cow's milk allergy should avoid cow's milk proteins; if a supplement is needed, use hypoallergenic formula, if available/affordable, to improve symptom control (B)
- Patients with asthma, rhinoconjunctivitis or eczema who are allergic to indoor allergens such as dust mites, cockroaches, and animal danders should eliminate or reduce the exposure to improve symptom control and prevent exacerbations (A–B)
- Aim pharmacotherapy primarily towards the underlying inflammatory process (A)
- Avoid strictly acetyl salicylic acid or other non-steroidal anti-inflammatory drugs (NSAIDs) in patients who have been diagnosed as being sensitive to them (C)

Education

- Patient education regarding precipitants of allergic symptoms, asthma, and anaphylaxis is essential. Guided self-management to prevent, assess and treat symptoms is the key to optimizing disease control (A)
- School policies on asthma and anaphylaxis management are essential (D)

*See footnote page 181.

- Health economic evaluations in all controlled studies exploring early treatment of allergic disease, especially asthma
- Health economic impact of measures taken to control environmental exposure compared with medication management in prevention and treatment of allergies and asthma
- Development of more advanced models to study economic impact of allergic diseases on health

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Appendix 1: Patient Information Sheets on Practical Allergen Avoidance Advice

House dust mite allergen reduction

Aims to reduce the amount of mite allergens in the home

Major strategies (WHO Strength of Recommendation A)

- Wash bedding regularly (every 1–2 weeks) at 55–60°C, if possible, to kill mites (washing with cold water removes 90% of mite allergens; washing at 55–60°C kills mites)
- Wash pillows and duvets in hot water 55–60°C and encase pillows and mattresses with documented protective coverings
- Sufficient ventilation of dwellings to decrease humidity; aim to reduce indoor relative humidity to below 50% and avoid damp housing conditions

Additional strategies

- Use a good quality vacuum cleaner (if possible, one fitted with HEPA filter)
- Use a damp duster when dusting and cleaning surfaces
- Replace wall to wall carpets with linoleum or wooden floors which can be wiped clean
- Remove/reduce curtains and soft furnishings in the bedroom
- Replace fabric-covered seating with leather or vinyl
- Remove soft toys from the bedroom; wash them at 55–60°C or freeze them (in a kitchen deep-freezer) to kill house dust mites
- Do not allow pets in the bedroom
- House dust mites are transparent and have no natural protection against sunlight. Exposure of mattresses, rugs, and carpets to direct strong sunlight (for more than 3 h) kills mites and can be used in appropriate regions.
- A hammock, easily washable and susceptible to air and sun drying, is used in many areas of the world

Cockroach allergen avoidance

Removes the cockroaches, eliminates the places and conditions in which they can live, and removes allergens

- Eradicate cockroaches with appropriate insecticides
- Seal cracks in floors and ceilings
- Remove sources of food
- Control dampness
- Scrub floors with water and detergent to remove allergens
- Bedding, curtains, and clothing can be contaminated and must be washed

Severe Reactions, Allergic Anaphylaxis

Aims to prevent contact with the allergens that induce anaphylaxis in susceptible individuals, and to provide strategies for dealing with episodes of allergic anaphylaxis

- Carry an epinephrine autoinjector and know how and when to use it; always have a spare autoinjector available
- Carry an emergency pager or mobile telephone to call assistance
- Carry/wear Medic-Alert information
- When travelling abroad, carry an anaphylaxis alert card in the language of the country being visited, detailing food, drug, and insect allergies
- Avoid stinging insects, and learn how not to attract them:
 - do not wear perfumes or bright colours
 - do not pick ripe fruit, avoid refuse bins and compost heaps which attract insects
 - keep car windows closed when driving
- Avoid allergenic ingredients in ready-made food by learning how to interpret ingredient lists
- If eating out, check with the chef that allergenic foods/oils are not used in dishes; explain the significance of avoiding allergenic ingredients

In the school environment

- Ensure that parents, teachers, fellow students, and school administrators are aware of the necessity to provide a safe environment for children at risk of anaphylaxis to foods or insect stings.
- Create a no-food area of the school playground
- Identify a supervisor to carry a telephone for emergencies

In the occupational environment

- Avoid contact with airborne or contact allergens; for example, airborne latex can be avoided by coworkers using powder-free latex gloves

Pollen avoidance

Provides mechanical barriers to pollen contact

- Keep windows closed at peak pollen times, e.g., in the evening when airborne pollen descends to lower altitudes
- Wear glasses or sunglasses to prevent pollen entering the eyes
- Consider wearing a mask over nose and mouth to prevent inhalation of pollen at peak times
- Pollen-allergic individuals should not cut grass
- Keep windows closed when the grass has been mown
- Use airconditioning, if possible
- Install car pollen filters, if possible

Pet allergen avoidance

Reduces the amount of pet allergen indoors

- If possible, find another home for the pet, and do not bring new animals into the home
- Exclude pets from bedrooms and, if possible, keep pets outdoors
- Vacuum carpets, mattresses, and upholstery regularly, if a power source and equipment are available
- Change clothes before going to school/work if you have attended your horse/cat/dog

Mould allergen avoidance

Prevents mould from growing, and mould spores from becoming airborne during mould removal

Indoors

- Use dehumidifiers in the home if relative humidity is constantly high (above 50%)
- Ensure heating, ventilation, or air conditioning systems are properly maintained
- Use 5% ammonia solution to remove mould from bathrooms and other contaminated surfaces
- Replace carpets with hard flooring; replace wallpaper with paint
- Repair indoor water damage immediately

Outdoors

- Avoid cutting grass in late summer when mould spores are present in decaying vegetation

Appendix 2: Content of Educational Programs in Allergy and Allergic Asthma

Allergic asthma and allergic rhinitis

Appropriate actions for guided self-management include:

1. Developing good communication between patient and physician to improve patient compliance
2. Promoting understanding about the basic facts, causes and triggers of allergic asthma/ allergic rhinitis
3. Identifying and controlling factors that aggravate asthma/rhinitis symptoms and provoke exacerbations
4. Following a written action plan to avoid or handle exacerbations
5. Emphasizing the importance of proper drug use and correct use of spacers and inhalers for long-term control
6. Monitoring symptoms and peak flow values in persistent asthma and adjusting medication accordingly
7. Educating to decrease reliance on unproven treatments

Severe Reactions, Allergic Anaphylaxis

Each patient should have an individual management protocol to include:

1. Carrying an epinephrine autoinjector and knowing how and when to use it; always having a spare autoinjector available
2. Carrying an emergency pager
3. Carrying/wearing Medic-Alert information
4. Avoiding stinging insects, learning how not to attract them
5. Avoiding allergenic ingredients in ready-made food

In the school environment

Education of parents, teachers, fellow students, and school administrators is necessary to provide a safe environment for children at risk of anaphylaxis to foods or insect stings.

6. Creating a no-food area of the school playground
7. Identifying a supervisor to carry a telephone for emergencies

In the occupational environment

8. Health and Safety measures should be introduced to prevent exposure of affected workers to airborne or contact allergens

Eczema

Educational programs in eczema should include:

1. Informing about nature, heredity, causes, and triggers of eczema
2. Identification and avoidance of individual provocation factors, skin care, and treatment options, including complementary therapies
3. Discussion of diagnosis and treatment of food allergies and adequate nutrition in childhood
4. Behaviour-oriented psychological intervention to interrupt the itching-scratching cycle.
5. Training to improve stress management and reduce the negative social effects of illness-specific problems

