BMJ Best Practice

Allergic rhinitis

Straight to the point of care

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Table of Contents

Summary

Allergic rhinitis is an immunoglobulin E (IgE)-mediated inflammatory nasal condition resulting from allergen introduction in a sensitized individual.

Presumptive diagnosis of allergic rhinitis is usually clinical and is made if someone experiences one or more of rhinorrhea, sneezing, itching of nose/palate/eyes, or nasal congestion in response to allergen exposure. Cough is also a common symptom.

The diagnosis is confirmed with demonstration of specific IgE reactive to environmental allergens. Food allergy testing is not recommended in the routine evaluation of rhinitis.

Treatment includes allergen avoidance (reducing exposure to relevant allergens such as dander, dust mite, and pollen), pharmacotherapy, and immunotherapy.

Intranasal corticosteroids remain the single most effective class of medications for treating allergic rhinitis.

Immunotherapy is often recommended for patients with persistent symptoms.

Definition

Allergic rhinitis (AR) is an immunoglobulin E (IgE)-mediated inflammatory nasal condition resulting from allergen exposure in a sensitized individual. AR is a common inflammatory condition of the upper respiratory tract, characterized by nasal pruritus, sneezing, rhinorrhea, and nasal congestion. Frequently, there is associated palate, throat, ear, and eye itching as well as eye redness, puffiness, and watery discharge.

AR may sometimes be due to occupational or hobby exposures to proteins that do not commonly provoke IgE-mediated responses. Examples include woodworkers becoming sensitized to wood dusts, or food service workers becoming sensitized to grain dusts.

Epidemiology

AR is a common disease that affects up to 30% of adults and up to 40% of children in industrialized countries worldwide.[6] The prevalence of AR varies significantly between countries.[7]

AR is believed to affect up to 60 million people in the US annually.[3] Prevalence of physician-diagnosed AR in the US has been reported to be 14% in adults and 7% in children.[8]

In Europe, the prevalence of AR has been reported as 13%.[9]

AR affects people of all ages, but approximately 80% of individuals diagnosed with AR develop symptoms before the age of 20 years.[10] [11] During childhood, older children have a higher prevalence of AR than younger children, with a peak occurring at ages 13 to 14 years.[12] During childhood, boys are more likely than girls to be affected by AR; beginning in puberty, girls have a higher rate of new AR symptoms, and by age >20 years the prevalence of AR is equal among men and women.[13]

AR has a major impact on quality of life. It is commonly associated with and can exacerbate asthma, allergic conjunctivitis, rhinosinusitis, and sleep disturbances.[3]

AR is found in approximately twice as many children with sleep-disordered breathing as those without.[14] The presence of AR in adults with obstructive sleep apnea does not appear to affect sleep parameters. Further exploration of the interaction between these conditions is indicated.[14]

Etiology

AR is a multifactorial disease with genetic and environmental components. AR is highly heritable.[15]

The risk of developing atopic disease in the absence of parental family history has been reported to be 13%.[16] The risk increased to 29% if one parent or sibling is atopic, 47% if both parents are atopic, and 72% if both parents have the same atopic manifestation.[16]

One genome-wide association study, which included 59,762 cases of European ancestry, identified 41 risk loci for AR. Genes involved in various immune pathways were implicated in AR pathogenesis; fine mapping of the HLA region suggested specific amino acid variants that are important for antigen binding.[17]

Environmental factors and gene-environment interactions may play an important role in AR development.[18] [19] [20] Epidemiologic studies implicate exposure to aeroallergens, indoor dampness-related exposures, and living in an urban environment.[21] [22] [23] [24] Changes to the intestinal microbiome may contribute to increased prevalence of allergic disease in industrialized nations (the "microflora hypothesis").[25] [26] [27] [28] [29] According to this hypothesis, the intestinal microbiome of Westerners may be altered by certain lifestyle factors (e.g., antibiotic use and dietary factors) that may predispose to the development of allergy.[25]

Pathophysiology

In susceptible individuals, exposure to a variety of environmental aeroallergens leads to allergic sensitization, which is characterized by the production of specific IgE directed against these proteins. This process begins with the binding of the allergen by antigen-presenting cells, such as dendritic cells, in the nasal mucosa that process the captured allergen and present it to T cells. Ultimately, this may lead to the production of

allergen-specific IgE, which binds to high-affinity IgE receptors present on the surface of mast cells in the nasal mucosa.

Once mast cells are sensitized to specific allergens, re-exposure to the allergen in quantities sufficient to cause cross-linking between allergen and adjacent IgE molecules will lead to degranulation of the mast cell and initiation of various pro-inflammatory events, such as synthesis of interleukins and inflammatory cell infiltration. The effects of mast cell activation may be separated into two separate processes termed the early phase and the late-phase response.

The early phase begins within minutes of allergen exposure and is primarily due to mast cell release of preformed mediators, including histamine, tryptase, chymase, kinins, and heparin. Other substances that are rapidly synthesized include cysteinyl leukotrienes (CysLTs) and interleukins, among others. Clinically, this process results in mucus gland stimulation leading to rhinorrhea, sensory nerve stimulation (which leads to sneezing and itching), and vasodilation (which results in mucosal and sinusoidal swelling and nasal obstruction).

Over 4 to 8 hours, the events of the early phase result in the recruitment and migration of other inflammatory cells to the nasal mucosa, including eosinophils, lymphocytes, and macrophages. These cells become activated and release their mediators into the milieu, perpetuating and amplifying the inflammatory process. Symptoms of the late-phase response are characterized less by sneezing and itching than the early phase and more by congestion and mucus production.

Classification

Allergic Rhinitis and its Impact on Asthma (ARIA) classification[1] [2]

The ARIA classification of AR is based on severity, duration of symptoms, and impact of social life, school, and work.

Intermittent: symptoms are present

- <4 days a week or for <4 consecutive weeks/year.

Persistent: the symptoms are present

- ≥4 days a week and for ≥4 consecutive weeks/year.

Mild: none of the following items are present

- Sleep disturbance
- Impairment of daily activities, leisure, and/or sport
- Impairment of school or work
- Troublesome symptoms.

Moderate/severe: one or more of the following items are present

- Sleep disturbance
- Impairment of daily activities, leisure, and/or sport
- Impairment of school or work
- Troublesome symptoms.

Classification by symptom severity[1] [3] [4]

Mild rhinitis severity: present when symptoms are not interfering with quality of life such as impairment of daily activities, work or school performance, and sleep.

Moderate/severe rhinitis: present when symptoms are troublesome or there is a negative impact on any of these parameters.

Classification by temporal pattern of allergen exposure[3] [5]

AR can be classified as being seasonal or perennial, depending on whether an individual was sensitized to cyclic pollen or year-round allergens such as dust mites, pets, cockroaches, and molds.

This classification scheme has been shown to be artificial and often inconsistent, because, depending on the locale, allergic sensitization to multiple seasonal allergens can result in year-round disease, and, conversely, allergic sensitization to "perennial" allergens such as animal dander can result in symptoms during only a limited period of time. Thus, medical management of these patients is better informed by considering patients as having intermittent or persistent symptoms.

Case history

Case history #1

A 22-year-old student presents with a 5-year history of worsening nasal congestion, sneezing, and nasal itching. Symptoms are year-round but worse during the spring season. On further questioning it is revealed that he has significant eye itching, redness, and tearing as well as palatal and throat itching during the spring season. He remembers that his mother told him at some point that he used to have eczema in infancy.

Approach

AR is mediated by an immunoglobulin (Ig)E-associated response to indoor and outdoor environmental allergens. However, none of these individual findings are very sensitive or specific, so their presence does not distinguish between allergic and nonallergic nasal disease. Similarly, the absence of pertinent findings does not rule out allergic disease, so determination of specific IgE reactivity using skin-prick testing or in vitro specific IgE determination represents the only confirmed means of diagnosing AR.

Signs and symptoms

Nasal signs and symptoms:

- Pruritus
- Sneezing
- Rhinorrhea
- Nasal congestion, often the most bothersome.

Associated signs and symptoms:

- Palate, throat, ear, and eye itching
- Eye redness, puffiness, and watery discharge.

Constitutional signs and symptoms:

- Fatigue
- Irritability.

The diagnosis of AR may be made presumptively based on the types of signs and symptoms and the history of allergen triggers. Patients should also be asked about the presence of chest symptoms, food allergies, and atopic dermatitis (eczema).

Unilateral rhinorrhea should prompt evaluation for cerebrospinal fluid leak. Unilateral disease, with the exception of unilateral nasal congestion due to a deviated septum, should warrant referral to an ear, nose, and throat physician.

Physical exam

Physical exam of the nose is supportive. Findings may include swelling of the turbinates and nasal mucosa, the presence of clear nasal mucus and/or a pale nasal mucosa, and nasal crease (a transverse crease resulting from repetitive nose rubbing and manipulation).

Physical exam of the face may identify allergic shiners (bluish discoloration in the infraorbital region), conjunctival injection, ocular mucoid discharge, and, less commonly, Dennie-Morgan lines (creases present under the lower eyelids).

Do not routinely perform sinonasal imaging in patients with symptoms suggesting a diagnosis of allergic rhinitis alone. The use of imaging for allergic rhinitis is unproven.[50] [51] [52][53]

Trial of therapy

A trial of symptom-driven pharmacotherapy with intranasal corticosteroids, or oral or intranasal antihistamine, may be a pragmatic and reasonable first step. A trial of intranasal corticosteroids should be considered a preferred first choice in moderate or severe AR, especially if symptoms are persistent or

nasal obstruction is an issue. A sufficient response to the chosen medication should be assessed after 1 to 2 weeks of therapy (at least 2 weeks for an intranasal corticosteroid).

Because symptoms of AR are often very subjective, the patient's perceived improvement in symptoms and quality of life is paramount in determining whether there has been an adequate response or whether further investigation is required. Validated quality of life questionnaires specific for AR can be used to assess therapy response (e.g., Rhinoconjunctivitis Quality of Life Questionnaire [RQLQ] and Rhinitis Control Assessment Test [RCAT]).[54] [55] [56]

Allergy testing

Determination of specific IgE reactivity using skin-prick testing or in vitro specific IgE determination is advisable if there is an inadequate response to a trial of therapy.[57]

- Skin prick testing or in vitro IgE determination usually suffice, although on some occasions both tests are used for optimal management.
- The choice of test often depends on availability. In vitro IgE determination tends to be more readily available but is more expensive, and provides less sensitivity and specificity than skin testing. However, in vitro IgE determination may be preferable in patients with severe eczema affecting the testing area, in patients with significant dermatographism, or for those unwilling or unable to stop their antihistamines or medications with antihistamine properties (e.g., tricyclic antidepressants).
- Most medical laboratories offer a variety of allergen panels that should incorporate the most important perennial allergens such as animal danders and dust mites as well as geographically important local pollens such as grasses, weeds, and trees. However, tests should be ordered judiciously in the context of the patient's symptoms and exposures (e.g., do not order tests for tropical grasses in northern climates where these grasses do not grow); the "shotgun approach" - ordering numerous tests to see if any come back positive - is inappropriate.
- Panels including foods have no role in the diagnosis of AR in the absence of systemic symptoms (e.g., urticaria or anaphylaxis) that occur in conjunction with the consumption of certain foods.
- Results may not only confirm the presence of allergic disease but also help in directing environmental control interventions and determining whether a patient may be suitable for immunotherapy.
- Approximately 26% of patients previously considered nonallergic based on negative tests for specific IgE may have positive nasal allergen provocation tests.[58] Although cumbersome, nasal allergen provocation tests might be an option for confirming the diagnosis in patients who have symptoms of AR, and negative specific IgE in vivo or in vitro tests, and who fail empiric therapy for AR based on the characteristics of their symptoms.

Do not perform any unproven diagnostic tests, such as immunoglobulin G (IgG) or a battery of nonspecific IgE tests, in the evaluation of allergy. IgG and indiscriminate IgE testing is unproven and can lead to inappropriate diagnosis and treatment.[44]

DIAGNOSIS

History and exam

Key diagnostic factors

sneezing (common)

- More likely in AR than nonallergic rhinitis.

nasal pruritus (common)

- More likely in AR than nonallergic rhinitis.

Other diagnostic factors

palate, throat, ear, and eye itching (common)

- Usually occurs in the early phase of AR.

eye redness, puffiness, and watery discharge (common)

- Usually occurs in the early phase of AR.

fatigue and irritability (common)

- May be due to poor quality sleep resulting from nasal congestion.

nasal congestion (common)

- Usually occurs in the late phase of AR.

rhinorrhea (common)

- Unilateral rhinorrhea should prompt evaluation for cerebrospinal fluid leak. Unilateral disease, with the exception of unilateral nasal congestion due to a deviated septum, should warrant referral to an ear, nose, and throat physician.

allergic shiners (common)

- Bluish discoloration in the infraorbital region.

conjunctival injection (common)

- May be identified on physical exam of the face.

ocular mucoid discharge (common)

- May be identified on physical exam of the face.

nasal crease (common)

- A transverse crease on the exterior of the nose resulting from repetitive nose rubbing and manipulation.

pale nasal mucosa (common)

- May be identified on physical exam of the nose.

swelling of the nasal mucosa and turbinates (common)

- May be identified on physical exam of the nose.

abundant clear nasal secretions (common)

- May be identified on physical exam of the nose.

Dennie-Morgan lines (creases present under the lower eyelids) (uncommon)

- Identified on physical exam of the face, although uncommon. May be due to nasal congestion and poor blood circulation in the ocular region.[59]

Risk factors

Strong

other atopic conditions or family history of atopy

- The best established risk factor is a family history of atopic disease, especially AR.[30] Various modalities, such as genetic linkage analysis, have been employed to collectively identify a multitude of genetic loci associated with a higher incidence of AR.[31]
- The risk of developing atopic disease in the absence of parental family history has been reported to be 13%.[16] The risk increased to 29% if one parent or sibling is atopic, 47% if both parents are atopic, and 72% if both parents have the same atopic manifestation.[16]

age <20 years

- Approximately 80% of individuals diagnosed as having AR develop symptoms before the age of 20 years.[10] [11] Onset after age 20 years raises suspicion of nonallergic rhinitis rather than AR.

exposure to aeroallergens (pollen, molds, house dust mites, pollution)

- Epidemiologic studies implicate exposure to aeroallergens, indoor dampness-related exposures, and living in an urban environment.[21] [22] [23]
- A statistically significant correlation has been reported between the incidence and/or prevalence of AR and living in an urban or industrialized environment with associated increased levels of traffic and ambient polycyclic aromatic hydrocarbons.[23] [32] [33] [34]
- The International Study of Asthma and Allergies in Childhood established that local environmental or ecologic factors may contribute to the varying prevalence of asthma, rhinoconjunctivitis, and atopic dermatitis.[12]
- One meta-analysis demonstrated a modest increased risk of AR associated with exposure to cigarette smoke.[35]

exposure to animal dander

- Cat and dog hair are common allergens, and may cause perennial AR symptoms.[36] [37]
- Exposure to a dog during the first year of life may reduce the risk of AR and asthma.[38] [39]
- Children of families with exposure to a farming lifestyle (farming animals, unpasteurized milk) in rural areas of Germany, Austria, and Switzerland were less likely to develop allergic sensitization, hay fever, and asthma than children of nonfarmers living in the same localities.[40] Continual long-term exposure to stables until age 5 years was associated with the lowest frequencies of allergic sensitization and disease.[40]

Weak

ethnicity

- In the US, atopic sensitization (including asthma) appears to be more prevalent among certain ethnic groups, such as African-Americans and Puerto Ricans.[41] [42]
- In one UK study, significantly more West Indian women consulted primary care physicians for AR compared with the white British population.[43]

positive allergen skin-prick tests

- Even though sensitization to a specific allergen does not always equate with clinical disease, allergen reactivity remains one of the strongest risk factors for the presence of upper (as well as lower) allergic respiratory disease. False positive results exist, so it is essential that positive tests of specific allergen reactivity be correlated with the clinical history. Unproven diagnostic tests, such as immunoglobulin G (IgG) or a battery of nonspecific immunoglobulin E (IgE) tests, are not recommended in the evaluation of allergy.[44]

Tests

1st test to order

Test	Result
therapeutic trial of antihistamine or intranasal corticosteroid • Oral or intranasal antihistamine can be used. In moderate or severe AR, a trial of an intranasal corticosteroid should be considered a preferred first choice, especially if nasal obstruction is an issue. Reassess after a 1- or 2-week trial. • Amelioration of symptoms and improvement in quality-of-life parameters such as sleep quantity and quality, activities of daily living, and ability to pursue hobbies, sports, and social activities without limitation should guide the clinician in determining whether a specific treatment regimen represents a failure. Validated quality of life questionnaires specific for AR can be used to assess therapy response (e.g., Rhinoconjunctivitis Quality of Life Questionnaire [RQLQ] and Rhinitis Control Assessment Test [RCAT]).[54] [55] [56]	**clinical improvement**

Test to avoid

Recommendations	Rationale
immunoglobulin G (IgG) testing • Do not perform IgG testing in the evaluation of allergy.[44]	• **IgG testing is unproven and can lead to inappropriate diagnosis and treatment. Appropriate diagnosis of allergy requires specific IgE testing in a patient with a relevant medical history.[44]**
sinonasal imaging • Do not routinely perform sinonasal imaging in patients with symptoms suggesting a diagnosis of allergic rhinitis alone.[50] [51] [52] [53]	• **The use of imaging for allergic rhinitis is unproven.[50]** • **Any potential benefits are outweighed by potential significant adverse events, unnecessary costs, and exposure to ionizing radiation.[51]**

Other tests to consider

Test	Result
allergen skin-prick testing • Provides superior sensitivity and specificity compared with in vitro specific IgE determination testing, as long as the test is performed by a properly trained individual. • Also offers rapid results and lower costs, especially if the number of individual skin tests performed is limited. A battery of nonspecific IgE testing in the evaluation of allergy is not recommended.[44] • However, it is not suitable for patients with severe eczema affecting the testing area, those with significant dermatographism, or those unwilling or unable to stop their antihistamines or medications with antihistamine properties (e.g., tricyclic antidepressants). • Most medical laboratories offer a variety of allergen panels that should incorporate the most important perennial allergens such as animal danders and dust mites as well as geographically important local pollens such as grasses, weeds, and trees. Tests should be ordered judiciously in the context of the patient's symptoms and exposures (e.g., do not order tests for tropical grasses in northern climates where these grasses do not grow). • Interpretation of results can be challenging. Individuals with very high IgE levels may have multiple positives that may or may not be clinically relevant. • Clinical correlation with symptoms by an individual highly trained in the interpretation of allergy testing is recommended. • Size of individual wheal and flare responses do not necessarily correlate with clinical reactivity to that particular allergen.	**wheal and flare reaction after specific allergen is introduced into the skin is 3 mm larger than negative (saline) control**
in vitro specific IgE determination • Sandwich-type assay that utilizes the patient's serum to bind to specific allergens present on a solid phase/chip. • Tends to be more expensive than skin-prick testing, and results are not immediately available. However, can be employed in patients with severe eczema, significant dermatographism, or those unwilling or unable to stop their antihistamines (because antihistamine use may cause false negative results in skin-prick tests). • Interpretation can be challenging and should be performed by an individual trained in the interpretation of specific IgE testing in the clinical context. A battery of nonspecific IgE testing in the evaluation of allergy is not recommended.[44] • IgE levels do not necessarily correlate with clinical reactivity to that particular allergen. • RAST results, and to a lesser degree skin tests, can be difficult to interpret in individuals with very high IgE levels as they often have nonspecific reactivity that may or may not be clinically relevant.	**specific allergen response**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Nonallergic rhinitis	• Sporadic or persistent perennial symptoms not resulting from IgE-mediated immunopathologic events. • Pain/pressure and a postnasal drip sensation are common. • Presence of nasal itching and sneezing less likely. • Nonallergic triggers such as strong odors, perfumes, cigarette smoke, and weather-related changes may be present. • Not common in children. Onset of symptoms after age 20 years more likely.[60] [61] [62]	• Other than the absence of positive allergy tests, no single, specific differentiating feature exists.
Acute sinusitis	• Acute (<2 weeks), subacute (2 to 6 weeks). • Acute disease often due to an infectious cause. • May present with cough, discolored nasal mucus, and facial pressure/pain.[63]	• Diagnosis is usually clinical.
Chronic sinusitis	• Symptoms >12 weeks. Usually diagnosed with the aid of radiologic studies. One of the more common clinical characteristics of chronic sinusitis is the presence of hyposmia or anosmia. • More commonly characterized by chronic inflammation than a bacterial infection, especially in adults.[63] • Frequently characterized as chronic sinusitis with nasal polyposis, and chronic sinusitis without nasal polyposis.	• Sinus CT scans are abnormal, by definition, in people with chronic sinusitis.
Infectious rhinosinusitis	• Viral infection may result in acute (<2 weeks) episode of rhinitis presenting with nasal congestion, rhinorrhea, sneezing, and varying degrees of nasal pruritus. May present with a sore	• Diagnosis is usually clinical.

Condition	Differentiating signs / symptoms	Differentiating tests
	throat, myalgias, headaches, discolored mucus, and fever. More common during the fall to spring months.	

Criteria

Allergic Rhinitis and its Impact on Asthma (ARIA) criteria[1] [2]

Intermittent: symptoms are present

- <4 days a week or for <4 consecutive weeks/year.

Persistent: symptoms are present

- >4 days a week and for ≥4 consecutive weeks/year.

AR is considered mild if none of the following items are present, but classified as moderate/severe when one or more items are present:

- Sleep disturbance
- Impairment of daily activities, leisure, and/or sport
- Impairment of school or work
- Troublesome symptoms.

Classification by symptom severity[1] [3] [4]

Mild rhinitis severity: present when symptoms are not interfering with quality of life, such as daily activities, work or school performance, and sleep.

Moderate/severe rhinitis: present when symptoms are troublesome or there is a negative impact on any of these parameters.

Approach

The goal of symptom relief or cessation is achieved by institution of allergen avoidance measures, pharmacotherapy, immunotherapy, or a combination thereof. The clinician should ask about nasal, palate, and eye symptoms, so that pharmacotherapy can be selected to target all of the affected areas. Common non-nasal symptoms contribute to impaired quality of life but may be unintentionally overlooked.

- Allergen avoidance is one of the guiding principles of treatment. While environmental control measures can sometimes lead to complete symptom control (e.g., by removing a pet), they can at other times be difficult to implement.
- After an initial pharmacologic treatment regimen has been initiated, follow-up should occur within 7-14 days and therapy be stepped up or stepped down, as deemed necessary.[67]
- Second-generation, nonsedating antihistamines are recommended for initial treatment of mild symptoms.[3] They are considered to be safe and relatively free of adverse effects. Sedation associated with the use of first-generation antihistamines diminishes their utility.
- Intranasal corticosteroids remain the single most effective class of medications for treating AR.[3] [4] However, for many patients, especially those with mild symptoms, antihistamines may be trialed prior to starting intranasal corticosteroids.[52]
- Second-generation antihistamines (e.g., loratadine and cetirizine) are preferred for breast-feeding mothers.[68] Caution is advised if use of first-generation antihistamines is required in breast-feeding mothers.[68] Case reports and studies report somnolence and irritability in breastfed infants when first-generation antihistamines are taken by the mother.[69]
- Leukotriene receptor antagonists (e.g., montelukast) are an option for patients who have failed or are intolerant of other agents, but they are used infrequently due to lack of efficacy and possible risk of neuropsychiatric events.[3] [70]

Allergen avoidance and control

Pollen (grasses, trees, weeds)[71]

- Keep windows of homes and cars closed and employ an air conditioner in the recycling/indoor mode.
- Minimize time spent outdoors during times of high pollen count, when practical.
- Avoid activities known to cause exposure to pollen, such as mowing grass.
- Shower after outdoor activities where exposure to pollen is high.
- Use recirculated air in the car when pollen levels are high.
- Wear sunglasses (to protect eyes from airborne pollen).
- Dry bedding and clothing inside or in a tumble dryer.

House dust mite minimization[71] [72]

- Physical measures include heating ventilation, freezing, washing, and barrier methods.
- Wash bedding weekly in hot water (>140°F [>60°C]) to kill dust mites and denature the allergens they produce. Hot tumble drying of washed items for an additional 10 minutes after they are dry will kill dust mites.
- Cover mattress, pillow, and quilt with dust mite-resistant covers. Wash covers every 2 months.
- Remove all soft toys and woolen bedding from the bedroom. Freezing soft toys overnight kills mites but does not remove the allergen; they can then be tumbled in the dryer to help with this.
- Where possible replace carpets with hard floors.
- Damp dust or use cloths to clean hard surfaces (including hard floors) weekly.

- Vacuum carpets weekly.
- Reduce humidity – have a dry and well ventilated house, and adequate floor and wall insulation.
- Venetian blinds or flat blinds are easier to clean than heavy curtains.
- Consider leather or vinyl sofas instead of fabric.

Pet dander

- Individuals allergic to cats and dogs have few effective ways to reduce their exposure to pet allergens short of ridding themselves of the animals. It is important to counsel patients that pet allergen levels only slowly decline over several months when a pet is removed from the home; therefore, rapid improvement is not expected.[71] Ultimately though, the affected individual willing to part with their cat or dog will frequently experience significant symptom relief.
- While some people may react differently to individual dogs, "hypoallergenic" dogs are a myth that has been debunked.[73] [74]
- Washing of cats has not been shown to be an effective approach to reducing allergen exposure. Although weekly washings can reduce allergens, clinical studies have shown neither a persistent reduction of airborne allergens nor a clear reduction in rhinitis symptoms.[75] [76]
- High-efficiency particulate air (HEPA) filters did not lead to significant symptom improvement in controlled trials of cat-sensitized individuals.[77] [78]

Cockroach infestation

- Cockroach infestation is associated with AR and asthma, especially in the inner city.[79]
- Control measures are based on eliminating suitable environments and restricting access by sealing, caulking, and controlling the food supply as well as using chemical control and traps.
- While cockroach extermination by professionals may reduce allergen levels by 80% to 90%, the clinical significance of this finding requires further research.[80]
- Reinfestation from adjacent apartments is a frequent problem, and thus extermination efforts will likely need to be repeated and extended beyond the affected space.

Molds[71]

- Mold-allergic individuals should carefully inspect their home for mold damage, with special attention to more humid areas of the dwelling.
- Appropriate steps should be taken to mitigate or prevent sources of humidity and/or water ingress associated with indoor mold growth. Ensure adequate natural ventilation, including the use of extractor fans; seal leaks in bathrooms and roofs.
- Clear overflowing gutters and blocked under-floor vents.
- Remove indoor pot plants (which promote mold growth).
- Dry or remove wet carpets.
- Avoid working with garden compost or mulch, or mowing lawns.
- Remove localized mold growth with a dilute bleach solution. More extensive mold damage may require aggressive measures such as replacing the affected surface/material.

Mild or intermittent symptoms

First-line management is with either an intranasal corticosteroid or a nonsedating oral antihistamine.[67] Monotherapy with an intranasal corticosteroid is generally recommended because oral antihistamines are less effective.[67] [81] However, many patients may prefer oral drugs.

Oral antihistamines are effective for rhinorrhea, sneezing, and itching, but have only a modest effect on nasal congestion.[52] Cetirizine, a second-generation antihistamine, has been found to be particularly

effective in AR, but may cause some mild sedation.[82] Second-generation oral antihistamines are preferred to first-generation agents because they cause less sedation, dizziness, and incoordination.[3] Paradoxical hyperactivity with use of sedating antihistamines has been reported, particularly in children.[83]

Intranasal antihistamines are another first-line option when symptoms are intermittent and do not require daily medication.[3] Intranasal antihistamines are particularly effective for rhinorrhea and congestion, but they do not improve symptoms at non-nasal sites.[53] They have a fast onset of action after initial dosing (usually 15-30 minutes, and no later than 3 hours) and are effective over a 12-hour period.[3] [67] Intranasal antihistamines may cause sedation.

Patient reassessment

The patient should be reassessed after a trial of monotherapy with an intranasal corticosteroid or oral antihistamine (ideally within 5 to 7 days).[3] If the patient remains symptomatic, an alternative first-line monotherapy should be used.[3] Failing this, first-line treatment options (from different drug classes) may be combined. For example, an intranasal corticosteroid or intranasal antihistamine could be added to an oral antihistamine. If symptoms are persistent, an intranasal corticosteroid and intranasal antihistamine may be continued in combination.[67]

When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67] The dose of intranasal sprays can be reduced as long as symptoms continue to be controlled. If multiple pharmacologic agents are used, discontinuation of the medication added to the intranasal corticosteroid may be considered.

Patients may have tried using over-the-counter decongestants, which may provide temporary relief from symptoms; however, there is no evidence to support their ongoing use in allergic rhinitis, and guidelines do not recommend them.

Persistent and moderate or severe symptoms

Intranasal corticosteroids should be the first consideration if symptoms are persistent and moderate or severe.[3] They may provide additional benefit in reducing AR-associated ocular symptoms.[84] [85] Oral and intranasal antihistamines are also first-line options in these patients.

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When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67] The dose of intranasal sprays can be reduced as long as symptoms continue to be controlled. If multiple pharmacologic agents are used, discontinuation of the medication added to the intranasal corticosteroid may be considered.

Immunotherapy

Immunotherapy may be offered by an allergy specialist (through a shared decision-making model) to a patient who remains symptomatic despite allergen avoidance measures and pharmacotherapy.[3]

Immunotherapy is also commonly used by patients either unwilling to take or unable to tolerate medications.[3] Immunotherapy should be targeted to include allergens that are clinically relevant to both the patient and the geographic locale.

Allergen immunotherapy has been shown to improve symptoms, medication use, and combined symptom/medication use scores in patients with rhinoconjunctivitis.[86] [87] Subcutaneous immunotherapy (SCIT) may alter the natural history of allergic disease (induce long-term remission after discontinuation of therapy and prevent new sensitizations), as well as reduce the progression from AR to asthma when given in children ages 6 to 14 years for a minimum of 3 years.[88]

Sublingual immunotherapy (SLIT), an alternative to SCIT depending on the allergen involved, is effective in treating AR in adults and children and may also have disease-modifying potential.[87] [89] [90] [91] [92] [93] It is considered to be safer than SCIT because adverse effects are usually limited to mucosal symptoms, and it is easier to administer (patient self-administers).[94] However, SLIT may be less effective than SCIT.[93] Direct comparisons using standardized and validated outcome measures between SLIT and SCIT are not available.[95] SLIT is more appropriately used in monosensitized patients, especially those sensitized to dust mites, grass, or ragweed.[96] [97] [98] [99] [100]

If immunotherapy is not available or there is a significant wait, a short course (7 days) of an oral corticosteroid may also be considered if symptoms are severe.[67]

Usual therapy not effective

Evaluation by an allergy specialist is advisable when there is:

- An incomplete response to trial of therapy of environmental and pharmacologic interventions, and a persistent and significant impact on quality of life (interference with hobbies, family life, activities of daily living, sleep, emotional well-being)
- An inability to adequately control associated conditions such as asthma or sinus disease.

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
mild or intermittent symptoms		
	1st	**intranasal corticosteroid**
	plus	**allergen avoidance**
	1st	**oral antihistamine**
	plus	**allergen avoidance**
	1st	**intranasal antihistamine**
	plus	**allergen avoidance**
	2nd	**alternative first-line monotherapy or combination therapy**
	plus	**allergen avoidance**
persistent and moderate to severe symptoms		
	1st	**intranasal corticosteroid**
	plus	**allergen avoidance**
	1st	**oral antihistamine**
	plus	**allergen avoidance**
	1st	**intranasal antihistamine**
	plus	**allergen avoidance**
	2nd	**alternative first-line monotherapy or combination therapy**
	plus	**allergen avoidance**
	3rd	**sublingual immunotherapy (SLIT) or subcutaneous immunotherapy (SCIT)**
	plus	**allergen avoidance**
	4th	**oral corticosteroid**
	plus	**allergen avoidance**

Ongoing		(summary)
usual therapy ineffective or poorly tolerated		
	1st	**sublingual immunotherapy (SLIT) or subcutaneous immunotherapy (SCIT)**
	plus	**allergen avoidance**

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

mild or intermittent symptoms

1st **intranasal corticosteroid**

Primary options

» beclomethasone dipropionate nasal: (42 micrograms/spray aqueous) children ≥6 years of age and adults: 42-84 micrograms (1-2 sprays) in each nostril twice daily

OR

» budesonide nasal: (32 micrograms/spray) children ≥6 years of age: 32-64 micrograms (1-2 sprays) in each nostril once daily; children ≥12 years of age and adults: 32-128 micrograms (1-4 sprays) in each nostril once daily

OR

» fluticasone propionate nasal: (50 micrograms/spray) children ≥4 years of age and adults: 50-100 micrograms (1-2 sprays) in each nostril once daily

OR

» mometasone nasal: (50 micrograms/spray) children ≥2 years of age: 50 micrograms (1 spray) in each nostril once daily; children ≥12 years of age and adults: 100 micrograms (2 sprays) in each nostril once daily

» In patients with mild or intermittent symptoms, intranasal corticosteroids are a first-line treatment option.[67]

» When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67] The dose of intranasal sprays can be reduced as long as symptoms continue to be controlled.

» Examples of suitable intranasal corticosteroids are provided here; however, this list is not exhaustive and many other options are available.

plus **allergen avoidance**

Acute

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

1st **oral antihistamine**

Primary options

» cetirizine: children ≥6 months of age: 2.5 to 5 mg orally once daily when required; children ≥6 years of age and adults: 5-10 mg orally once daily when required

OR

» desloratadine: children ≥6 months of age: 1 to 2.5 mg orally once daily when required; children ≥12 years of age and adults: 5 mg orally once daily when required

OR

» fexofenadine: children ≥2 years of age: 30 mg orally twice daily when required; children ≥12 years of age and adults: 60 mg orally twice daily or 180 mg once daily when required

OR

» levocetirizine: children ≥6 months of age: 1.25 to 2.5 mg orally once daily when required; children ≥12 years of age and adults: 2.5 to 5 mg orally once daily when required

OR

» loratadine: children ≥2 years of age: 5 mg orally once daily when required; children ≥6 years of age and adults: 10 mg orally once daily when required

» In patients with mild or intermittent symptoms, a nonsedating antihistamine is a first-line treatment option.[67]

» Monotherapy with an intranasal corticosteroid is generally recommended because oral antihistamines are less effective.[67] [81] However, many patients may prefer oral drugs.

MANAGEMENT

Acute

» Oral antihistamines are effective for rhinorrhea, sneezing, and itching, but have only a modest effect on nasal congestion.[52] Cetirizine, a second-generation antihistamine, has been found to be particularly effective in AR, but may cause some mild sedation.[82] Second-generation oral antihistamines are preferred to first-generation agents because they cause less sedation, dizziness, and incoordination.[3]

» Paradoxical hyperactivity with use of sedating antihistamines has been reported, particularly in children.[83]

» When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67]

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

1st **intranasal antihistamine**

Primary options

» azelastine nasal: (137 micrograms/spray) children ≥5 years of age: 137 micrograms (1 spray) in each nostril twice daily; children ≥12 years of age and adults: 137-274 micrograms (1-2 sprays) in each nostril twice daily; (205.5 micrograms/spray) children ≥6 years of age: 205.5 micrograms (1 spray) in each nostril twice daily; children ≥12 years of age and adults: 205.5 to 411 micrograms (1-2 sprays) in each nostril once to twice daily

OR

» olopatadine nasal: (665 micrograms/spray) children ≥6 years of age: 665 micrograms (1 spray) in each nostril twice daily; children ≥12 years of age and adults: 1330 micrograms (2 sprays) in each nostril twice daily

» Intranasal antihistamines (e.g., azelastine, olopatadine) are another first-line option when symptoms are intermittent and do not require daily medication.[3]

» Intranasal antihistamines are particularly effective for rhinorrhea and nasal congestion,

Acute

but they do not improve symptoms at non-nasal sites.[53] They have a fast onset of action after initial dosing (usually 15-30 minutes, and no later than 3 hours) and are effective over a 12-hour period.[3] [67]

» Intranasal antihistamines may cause sedation.

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

2nd **alternative first-line monotherapy or combination therapy**

» The patient should be reassessed after a trial of monotherapy with an intranasal corticosteroid or oral antihistamine (ideally within 5-7 days).[3] If the patient remains symptomatic, an alternative first-line monotherapy should be used.[3] Failing this, first-line treatment options (from different drug classes) may be combined. For example, an intranasal corticosteroid or intranasal antihistamine could be added to an oral antihistamine. If symptoms are persistent, an intranasal corticosteroid and intranasal antihistamine may be continued in combination.[67]

» When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67] The dose of intranasal sprays can be reduced as long as symptoms continue to be controlled. If multiple pharmacologic agents are used, discontinuation of the medication added to the intranasal corticosteroid may be considered.

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

persistent and moderate to severe symptoms

1st **intranasal corticosteroid**

Acute

Primary options

» beclomethasone dipropionate nasal: (42 micrograms/spray aqueous) children ≥6 years of age and adults: 42-84 micrograms (1-2 sprays) in each nostril twice daily

OR

» budesonide nasal: (32 micrograms/spray) children ≥6 years of age: 32-64 micrograms (1-2 sprays) in each nostril once daily; children ≥12 years of age and adults: 32-128 micrograms (1-4 sprays) in each nostril once daily

OR

» fluticasone propionate nasal: (50 micrograms/spray) children ≥4 years of age and adults: 50-100 micrograms (1-2 sprays) in each nostril once daily

OR

» mometasone nasal: (50 micrograms/spray) children ≥2 years of age: 50 micrograms (1 spray) in each nostril once daily; children ≥12 years of age and adults: 100 micrograms (2 sprays) in each nostril once daily

» Intranasal corticosteroids should be the first consideration if symptoms are persistent and moderate or severe.[3] They may provide additional benefit in reducing AR-associated ocular symptoms.[84] [85]

» When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67] The dose of intranasal sprays can be reduced as long as symptoms continue to be controlled.

» Examples of suitable intranasal corticosteroids are provided here; however, this list is not exhaustive and many other options are available.

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

Acute

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

1st **oral antihistamine**

Primary options

» cetirizine: children ≥6 months of age: 2.5 to 5 mg orally once daily when required; children ≥6 years of age and adults: 5-10 mg orally once daily when required

OR

» desloratadine: children ≥6 months of age: 1 to 2.5 mg orally once daily when required; children ≥12 years of age and adults: 5 mg orally once daily when required

OR

» fexofenadine: children ≥2 years of age: 30 mg orally twice daily when required; children ≥12 years of age and adults: 60 mg orally twice daily or 180 mg once daily when required

OR

» levocetirizine: children ≥6 months of age: 1.25 to 2.5 mg orally once daily when required; children ≥12 years of age and adults: 2.5 to 5 mg orally once daily when required

OR

» loratadine: children ≥2 years of age: 5 mg orally once daily when required; children ≥6 years of age and adults: 10 mg orally once daily when required

» Oral antihistamines are a first-line option if symptoms are persistent and moderate or severe.

» Monotherapy with an intranasal corticosteroid is generally recommended because oral antihistamines are less effective.[67] [81] However, many patients may prefer oral drugs.

» Oral antihistamines are effective for rhinorrhea, sneezing, and itching, but have only a modest effect on nasal congestion.[52] Cetirizine, a second-generation antihistamine, has been found to be particularly effective in AR, but

Acute

may cause some mild sedation.[82] Second-generation oral antihistamines are preferred to first-generation agents because they cause less sedation, dizziness, and incoordination.[3]

» Paradoxical hyperactivity with use of sedating antihistamines has been reported, particularly in children.[83]

» When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67]

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

1st **intranasal antihistamine**

Primary options

» azelastine nasal: (137 micrograms/spray) children ≥5 years of age: 137 micrograms (1 spray) in each nostril twice daily; children ≥12 years of age and adults: 137-274 micrograms (1-2 sprays) in each nostril twice daily; (205.5 micrograms/spray) children ≥6 years of age: 205.5 micrograms (1 spray) in each nostril twice daily; children ≥12 years of age and adults: 205.5 to 411 micrograms (1-2 sprays) in each nostril once to twice daily

OR

» olopatadine nasal: (665 micrograms/spray) children ≥6 years of age: 665 micrograms (1 spray) in each nostril twice daily; children ≥12 years of age and adults: 1330 micrograms (2 sprays) in each nostril twice daily

» Intranasal antihistamines (e.g., azelastine, olopatadine) are particularly effective for rhinorrhea and nasal congestion, but they do not improve symptoms at non-nasal sites.[53] They have a fast onset of action after initial dosing (usually 15-30 minutes, and no later than 3 hours) and are effective over a 12-hour period.[3] [67]

» Intranasal antihistamines may cause sedation.

plus **allergen avoidance**

Acute

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

2nd **alternative first-line monotherapy or combination therapy**

» The patient should be reassessed after a trial of monotherapy with an intranasal corticosteroid or oral antihistamine (ideally within 5-7 days).[3] If the patient remains symptomatic, an alternative first-line monotherapy should be used.[3] Failing this, first-line treatment options (from different drug classes) may be combined. For example, an intranasal corticosteroid or intranasal antihistamine could be added to an oral antihistamine. If symptoms are persistent, an intranasal corticosteroid and intranasal antihistamine may be continued in combination.[67]

» When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67] The dose of intranasal sprays can be reduced as long as symptoms continue to be controlled. If multiple pharmacologic agents are used, discontinuation of the medication added to the intranasal corticosteroid may be considered.

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

3rd **sublingual immunotherapy (SLIT) or subcutaneous immunotherapy (SCIT)**

Primary options

» house dust mite allergen extract: consult specialist for guidance on sublingual dose

OR

» mixed grass pollens allergen extract: consult specialist for guidance on sublingual dose

Acute

OR

» timothy grass pollen allergen extract: consult specialist for guidance on sublingual dose

OR

» short ragweed pollen allergen extract: consult specialist for guidance on sublingual dose

» Immunotherapy is the only treatment modality to potentially have a disease-modifying effect.[101] It should be targeted to include allergens that are clinically relevant to both the patient and the geographic locale.

» Immunotherapy may be offered by an allergy specialist (through a shared decision-making model) to a patient who remains symptomatic despite allergen avoidance measures and pharmacotherapy.[3] Immunotherapy is also commonly used by patients either unwilling to take or unable to tolerate medications.[3]

» SLIT is effective in treating AR in both adults and children.[87] [89] [90] [91] [92] [93] It is considered to be safer than SCIT because adverse effects are usually limited to mucosal symptoms, and it is easier to administer (patient self-administers). However, SLIT may be less effective than SCIT.[93]

» SLIT is more appropriately used in monosensitized patients, especially those sensitized to dust mites, grass, or ragweed.[96] [97] [98] [99] [100] For polysensitized patients, SLIT with multiple allergens is sometimes employed, although no commercially available formulation containing more than one allergen currently exists.

» SLIT formulations can be employed in two different manners. One involves taking SLIT for approximately 12 weeks before and throughout the pollen season, stopping thereafter. Alternatively, SLIT can be taken daily for 3 years to provide a sustained effect for a fourth year, even after discontinuation.[101] [102]

» SCIT is used less frequently than SLIT. Improvement requires several months of treatment. It is generally accepted that a 1-year trial will determine who will and who will not respond to SCIT.

Acute

» Local and systemic reactions to SCIT may occur.[103] [104] Systemic reactions can vary from mild to life-threatening; fatal reactions after receiving an allergy vaccine are estimated to occur at a rate of 1 in 2 to 2.5 million injections.[105] [106] SCIT may reduce the progression from AR to asthma when given in children ages 6 to 14 years for a minimum of 3 years.[88] Various extract manufacturers and dosing regimens exist for SCIT.

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

4th **oral corticosteroid**

Primary options

» prednisone: 5-60 mg/day orally

» If immunotherapy is not available or there is a significant wait, a short course (7 days) of an oral corticosteroid may also be considered if symptoms are severe.[67]

plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the relevant allergens of concern for a particular patient.

Ongoing

usual therapy ineffective or poorly tolerated

1st **sublingual immunotherapy (SLIT) or subcutaneous immunotherapy (SCIT)**

Primary options

» house dust mite allergen extract: consult specialist for guidance on sublingual dose

OR

» mixed grass pollens allergen extract: consult specialist for guidance on sublingual dose

OR

» timothy grass pollen allergen extract: consult specialist for guidance on sublingual dose

OR

» short ragweed pollen allergen extract: consult specialist for guidance on sublingual dose

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Ongoing

formulation containing more than one allergen currently exists.

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plus **allergen avoidance**

Treatment recommended for ALL patients in selected patient group

» Allergen avoidance should be attempted by all patients with AR.

» Allergy testing can be helpful in identifying the allergens of concern for a particular patient.

Primary prevention

Cat and dog hair are common allergens, and may cause perennial AR symptoms.[36] [37] Exposure to a dog during the first year of life may reduce the risk of AR and asthma.[38] [39]

Pregnancy and early life

Current evidence does not support an antigen avoidance diet for pregnant women, including those at high risk for giving birth to a child with atopic disease.[45] [46]

While currently available evidence does not indicate that probiotic supplementation reduces the risk of atopic disorders, the World Allergy Organization recommends the use of probiotics in: pregnant women at high risk for having an allergic child; women who are breast-feeding infants at high risk of developing allergy; and infants at high risk of developing allergy.[28] [47]

There is insufficient evidence to determine the relationship between the age at which complementary foods are first introduced and risk of developing AR during childhood.[48]

In a multicenter allergy study, no modifiable aspects of early-life environment or lifestyle were identified as targets for primary prevention of AR.[49]

Secondary prevention

Avoidance of allergens known or suspected to trigger AR symptoms may minimize (or fully alleviate) symptoms and reduce medication use. However, this may not be the case for all AR patients.

Patient discussions

An initial official consultation for AR should include:

- Information on allergen avoidance (only if allergy testing has been performed - there is no value in discussing avoidance of "typical" allergens without confirming that they are relevant to the patient)
- An action plan detailing various aspects of the chosen pharmacologic agent(s)
- Expected clinical outcomes.

Details on medications should include:

- Appropriate dosing frequency and technique (including teaching on proper administration of nasal sprays)
- Whether treatments should be used on a schedule or as needed
- Expected time to clinical improvement
- Possible adverse effects (e.g., sedation with first-generation antihistamines).

Plans for appropriate follow-up should be made. The patient should be reassessed after a trial of monotherapy with an intranasal corticosteroid or oral antihistamine (ideally within 5-7 days).[3]

FOLLOW UP

Monitoring

Monitoring

Plans for appropriate follow-up should be made. The patient should be reassessed after a trial of monotherapy with an intranasal corticosteroid or oral antihistamine (ideally within 5-7 days).[3]

When symptoms improve, decreasing or discontinuing treatment may be considered.[3] [67]

Complications

Complications	Timeframe	Likelihood
acute conjunctivitis (allergic)	**variable**	**medium**
Appropriate therapy with oral or ophthalmic antihistamines or ophthalmic cromolyn may be required to achieve a successful and comprehensive therapeutic outcome.		
asthma	**variable**	**medium**
Poorly controlled AR may result in the development of lower respiratory tract symptoms or loss of asthma control in certain individuals. If this occurs, the physician will need to address both airways equally to gain adequate disease control.		
food-pollen syndrome	**variable**	**medium**
Symptoms such as itching and swelling triggered by certain fruits or vegetables in patients with allergic rhinitis are usually confined to the oropharynx and will generally resolve without treatment. However, a nonsedating antihistamine may be appropriate for troublesome symptoms. Anaphylaxis is rare but an epinephrine (adrenaline) autoinjector is recommended for patients who are at increased risk of severe reactions. Patient education about avoidance of the responsible foods is recommended.[111]		
antihistamine-associated sedation	**variable**	**medium**
Use of first-generation antihistamines may result in both drowsiness and a global reduction or impairment in intellectual and motor performance, such as learning a new task or driving an automobile. This can also occur with some second-generation antihistamines (e.g., cetirizine and levocetirizine), but is less common than with first-generation agents. Paradoxical hyperactivity with use of sedating antihistamines has been reported, particularly in children.[83]		
adverse effects of nasal decongestants	**variable**	**medium**
Include nasal burning, stinging, dryness, and less commonly, mucosal ulceration.		
rhinitis medicamentosa (rebound nasal congestion)	**variable**	**medium**
Can occur when intranasal decongestants are used for longer than 3 to 5 days.		
adverse effects of oral decongestants	**variable**	**medium**
Adverse effects of oral decongestants include central nervous stimulation such as insomnia, which may occur in up to one third of people; nervousness; anxiety; tremors; tachycardia; palpitations; and increases in BP.		
adverse effects of leukotriene receptor antagonists	**variable**	**medium**
Leukotriene receptor antagonists (e.g., montelukast) are associated with an increased risk of neuropsychiatric events (e.g., agitation, depression, sleeping problems, and suicidal thoughts and		

Complications	Timeframe	Likelihood
actions). The risk of neuropsychiatric events may outweigh the benefits, particularly for patients with symptoms that are mild and treatable with alternative agents. Leukotriene receptor antagonists should be reserved for those who have failed or are intolerant of other agents.[3] [70]		
chronic sinusitis	**variable**	**medium**
There is considerable epidemiologic evidence to support an association between sinus disease and AR. Studies have noted that 40% to 67% of patients with unilateral chronic sinusitis and up to 80% with chronic bilateral sinusitis have AR.		
acute sinusitis	**variable**	**medium**
There is considerable epidemiologic evidence to support an association between sinus disease and AR. Studies have noted that 25% to 30% of patients with acute sinusitis have AR.		

Prognosis

Natural history of disease

AR prevalence increases in young childhood and peaks in childhood and adolescence until decreasing with advancing age.

Studies suggest that AR symptoms may improve or resolve with time in approximately 10% to 50% of affected individuals.[109] [110] However, it is a condition that may persist throughout life.[1]

Variability of severity

As with other chronic diseases, AR can vary in severity over time. While a long-term trend for the improvement or resolution of symptoms exists, the perceived severity of disease can increase or decrease, wax and wane, or even change unpredictably over a short time span. Possible factors explaining severity variability may be divided into being external or internal. External factors include time of the day, location, and season, because they all affect pollen counts. Internal factors may include circadian rhythms, mood, and affect, as well as immunologic changes that occur over time.

Immunotherapy outcomes

In patients receiving grass allergen immunotherapy for a period of 3 to 4 years, approximately 50% of patients continued to derive clinical benefits 3 years after immunotherapy had been discontinued.[101] [102]

Diagnostic guidelines

International

Clinical practice guideline: immunotherapy for inhalant allergy (https://www.entnet.org/quality-practice/quality-products/clinical-practice-guidelines) [64]

Published by: American Academy of Otolaryngology; Head and Neck Surgery Foundation **Last published:** 2024

Rhinitis 2020: a practice parameter update (https://www.aaaai.org/Allergist-Resources/Statements-Practice-Parameters/Practice-Parameters-Guidelines) [3]

Published by: American Academy of Allergy, Asthma, and Immunology **Last published:** 2020

Practical guide to skin prick tests in allergy to aeroallergens (https://pubmed.ncbi.nlm.nih.gov/22050279) [57]

Published by: Global Allergy and Asthma European Network **Last published:** 2012

Japanese guidelines for allergic rhinitis 2020 (https://www.sciencedirect.com/science/article/pii/S1323893020300502) [65]

Published by: Japanese Society of Allergology **Last published:** 2020

Australasian guidelines for allergic rhinitis 2022 (https://www.allergy.org.au/hp/papers) [66]

Published by: Australasian Society of Clinical Immunology and Allergy **Last published:** 2022

BSACI guidelines: immunotherapy for the management of allergic and non-allergic rhinitis (revised edition) (https://www.bsaci.org/guidelines/bsaci-guidelines) [52]

Published by: British Society for Allergy and Clinical Immunology **Last published:** 2017

Treatment guidelines

International

Clinical practice guideline: immunotherapy for inhalant allergy (https://www.entnet.org/quality-practice/quality-products/clinical-practice-guidelines) [64]

Published by: American Academy of Otolaryngology; Head and Neck Surgery Foundation **Last published:** 2024

Rhinitis 2020: a practice parameter update (https://www.aaaai.org/Allergist-Resources/Statements-Practice-Parameters/Practice-Parameters-Guidelines) [3]

Published by: American Academy of Allergy, Asthma, and Immunology **Last published:** 2020

Sublingual immunotherapy: a focused allergen immunotherapy practice parameter update (https://www.aaaai.org/Allergist-Resources/Statements-Practice-Parameters/Practice-Parameters-Guidelines) [107]

Published by: American Academy of Allergy, Asthma, and Immunology; American College of Allergy, Asthma, and Immunology **Last published:** 2017

Next-generation Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines for allergic rhinitis based on Grading of Recommendations Assessment, Development and Evaluation (GRADE) and real-world evidence (https://www.jacionline.org/article/S0091-6749(19)31187-X/fulltext) [67]

Published by: Centre for Empowering Patients and Communities; European Academy of Allergy and Clinical Immunology; European Institute for Innovation and Technology EIT Health; European Federation of Allergy and Airways Diseases Patients' Associations; European Respiratory Society; European Forum for Research and Education in Allergy and Airways Diseases; Global Allergy and Asthma European Network; Global Alliance against Chronic Respiratory Diseases; Global Initiative for Asthma; Impact of Air Pollution in Asthma and Rhinitis; Société Française d'Allergologie; Societé de Pneumologie de Langue Française; World Allergy Organization **Last published:** 2020

Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines: 2016 revision (https://www.jacionline.org/article/S0091-6749(17)30919-3/fulltext) [2]

Published by: Global Allergy and Asthma European Network; Grading of Recommendations Assessment, Development and Evaluation Working Group; American Academy of Allergy, Asthma & Immunology **Last published:** 2016

Japanese guidelines for allergic rhinitis 2020 (https://www.sciencedirect.com/science/article/pii/S1323893020300502) [65]

Published by: Japanese Society of Allergology **Last published:** 2020

EAACI guidelines on allergen immunotherapy: allergic rhinoconjunctivitis (https://onlinelibrary.wiley.com/doi/full/10.1111/all.13317) [108]

Published by: European Academy of Allergy and Clinical Immunology **Last published:** 2018

International

Australasian guidelines for allergic rhinitis 2022 (https://www.allergy.org.au/hp/papers) [66]

Published by: Australasian Society of Clinical Immunology and Allergy **Last published:** 2022

BSACI guidelines: immunotherapy for the management of allergic and non-allergic rhinitis (revised edition) (https://www.bsaci.org/guidelines/bsaci-guidelines) [52]

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REFERENCES

Key articles

- Dykewicz MS, Wallace DV, Amrol DJ, et al. Rhinitis 2020: a practice parameter update. J Allergy Clin Immunol. 2020 Oct;146(4):721-67. Full text (https://www.jacionline.org/article/S0091-6749(20)31023-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32707227?tool=bestpractice.bmj.com)
- Scadding GK, Kariyawasam HH, Scadding G, et al. BSACI guideline for the diagnosis and management of allergic and non-allergic rhinitis (revised edition 2017). Clin Exp Allergy. 2017 July;47(7):856-89. Full text (https://onlinelibrary.wiley.com/doi/10.1111/cea.12953) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30239057?tool=bestpractice.bmj.com)
- Wise SK, Lin SY, Toskala E, et al. International consensus statement on allergy and rhinology: allergic rhinitis. Int Forum Allergy Rhinol. 2018 Feb;8(2):108-352. Full text (https://onlinelibrary.wiley.com/doi/full/10.1002/alr.22073) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29438602?tool=bestpractice.bmj.com)
- Bousquet J, Schünemann HJ, Togias A, et al. Next-generation Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines for allergic rhinitis based on Grading of Recommendations Assessment, Development and Evaluation (GRADE) and real-world evidence. J Allergy Clin Immunol. 2020 Jan;145(1):70-80.e3. Full text (https://www.jacionline.org/article/S0091-6749(19)31187-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31627910?tool=bestpractice.bmj.com)

References

1. Bousquet J, Khaltaev N, Cruz AA, et al. Allergic rhinitis and its impact on asthma (ARIA) 2008. Allergy. 2008 Apr;63 Suppl 86:8-160. Full text (https://onlinelibrary.wiley.com/doi/full/10.1111/j.1398-9995.2007.01620.x) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18331513?tool=bestpractice.bmj.com)
2. Brożek JL, Bousquet J, Agache I, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines - 2016 revision. J Allergy Clin Immunol. 2017 Oct;140(4):950-8. Full text (https://www.jacionline.org/article/S0091-6749(17)30919-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28602936?tool=bestpractice.bmj.com)
3. Dykewicz MS, Wallace DV, Amrol DJ, et al. Rhinitis 2020: a practice parameter update. J Allergy Clin Immunol. 2020 Oct;146(4):721-67. Full text (https://www.jacionline.org/article/S0091-6749(20)31023-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32707227?tool=bestpractice.bmj.com)
4. Wallace DV, Dykewicz MS, Bernstein DI, et al. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol. 2008 Aug;122(2 Suppl):S1-84. Full text (https://www.jacionline.org/article/S0091-6749(08)01123-8/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18662584?tool=bestpractice.bmj.com)
5. World Health Organization. International classification of diseases, 10th revision. 2019 [internet publication]. Full text (https://icd.who.int/browse10/2019/en#/J30)

6. European Academy of Allergy and Clinical Immunology. Global atlas of allergic rhinitis and chronic rhinosinusitis. Zurich, Switzerland: EAACI; 2015. Full text (https://medialibrary.eaaci.org/mediatheque/media.aspx?mediaId=60232&channel=8518)

7. Asher MI, Montefort S, Bjorksten B, et al. ISAAC Phase Three Study Group. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. Lancet. 2006 Aug 26;368(9537):733-43. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/16935684?tool=bestpractice.bmj.com)

8. Meltzer EO, Blaiss MS, Naclerio RM, et al. Burden of allergic rhinitis: allergies in America, Latin America, and Asia-Pacific adult surveys. Allergy Asthma Proc. 2012 Sep-Oct;33(suppl 1):S113-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22981425?tool=bestpractice.bmj.com)

9. Bauchau V, Durham SR. Prevalence and rate of diagnosis of allergic rhinitis in Europe. Eur Respir J. 2004 Nov;24(5):758-64. Full text (https://erj.ersjournals.com/content/24/5/758.long) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15516669?tool=bestpractice.bmj.com)

10. Skoner DP. Allergic rhinitis: definition, epidemiology, pathophysiology, detection, and diagnosis. J Allergy Clin Immunol. 2001 Jul;108(1 Suppl):S2-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/11449200?tool=bestpractice.bmj.com)

11. Pawankar R, Canonica GW, Colgate ST, et al. World Allergy Organization (WAO) white book on allergy: update 2013. Milwaukee, WI: World Allergy Organization; 2013. Full text (https://www.worldallergy.org/UserFiles/file/WhiteBook2-2013-v8.pdf)

12. Mallol J, Crane J, von Mutius E, et al; ISAAC Phase Three Study Group. The International Study of Asthma and Allergies in Childhood (ISAAC) phase three: a global synthesis. Allergol Immunopathol (Madr). 2013 Mar-Apr;41(2):73-85. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22771150?tool=bestpractice.bmj.com)

13. Pinart M, Keller T, Reich A, et al. Sex-related allergic rhinitis prevalence switch from childhood to adulthood: a systematic review and meta-analysis. Int Arch Allergy Immunol. 2017;172(4):224-35. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28456795?tool=bestpractice.bmj.com)

14. Cao Y, Wu S, Zhang L, et al. Association of allergic rhinitis with obstructive sleep apnea: a meta-analysis. Medicine (Baltimore). 2018 Dec;97(51):e13783. Full text (https://journals.lww.com/md-journal/Fulltext/2018/12210/Association_of_allergic_rhinitis_with_obstructive.113.aspx) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30572534?tool=bestpractice.bmj.com)

15. Dávila I, Mullol J, Ferrer M, et al. Genetic aspects of allergic rhinitis. J Investig Allergol Clin Immunol. 2009;19 Suppl 1:25-31. Full text (http://www.jiaci.org/issues/vol19s1/5.pdf) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19476051?tool=bestpractice.bmj.com)

16. Evans R. Epidemiology and natural history of asthma, allergic rhinitis, and atopic dermatitis (eczema). In: Middleton E, Reed C, Ellis E, eds. Allergy: principles and practice. 4th ed. St Louis, MO: Mosby; 1993:1109-36.

REFERENCES

17. Waage J, Standl M, Curtin JA, et al. Genome-wide association and HLA fine-mapping studies identify risk loci and genetic pathways underlying allergic rhinitis. Nat Genet. 2018 Aug;50(8):1072-80. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7068780) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30013184?tool=bestpractice.bmj.com)

18. Chen HI, Lin YT, Jung CR, et al. Interaction between catalase gene promoter polymorphisms and indoor environmental exposure in childhood allergic rhinitis. Epidemiology. 2017 Oct;28 Suppl 1:S126-32. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29028686?tool=bestpractice.bmj.com)

19. Li CH, Sayeau K, Ellis AK. Air pollution and allergic rhinitis: role in symptom exacerbation and strategies for management. J Asthma Allergy. 2020 Aug 26;13:285-92. Full text (https://www.dovepress.com/air-pollution-and-allergic-rhinitis-role-in-symptom-exacerbation-and-s-peer-reviewed-fulltext-article-JAA) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32922045?tool=bestpractice.bmj.com)

20. Zhang Y, Tan M, Qian X, et al. Interaction between early-life pet exposure and methylation pattern of ADAM33 on allergic rhinitis among children aged 3-6 years in China. Allergy Asthma Clin Immunol. 2021 May 1;17(1):44. Full text (https://aacijournal.biomedcentral.com/articles/10.1186/s13223-021-00526-5) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33933154?tool=bestpractice.bmj.com)

21. Dunlop J, Matsui E, Sharma HP. Allergic rhinitis: environmental determinants. Immunol Allergy Clin North Am. 2016 May;36(2):367-77. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27083109?tool=bestpractice.bmj.com)

22. Jaakkola MS, Quansah R, Hugg TT, et al. Association of indoor dampness and molds with rhinitis risk: a systematic review and meta-analysis. J Allergy Clin Immunol. 2013 Nov;132(5):1099-1110.e18. Full text (https://www.jacionline.org/article/S0091-6749(13)01153-6/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24028857?tool=bestpractice.bmj.com)

23. Christensen SH, Timm S, Janson C, et al. A clear urban-rural gradient of allergic rhinitis in a population-based study in Northern Europe. Eur Clin Respir J. 2016 Nov 26;3:33463. Full text (https://www.tandfonline.com/doi/full/10.3402/ecrj.v3.33463) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27890047?tool=bestpractice.bmj.com)

24. Burbank AJ, Hernandez ML, Jefferson A, et al. Environmental justice and allergic disease: a Work Group report of the AAAAI Environmental Exposure and Respiratory Health Committee and the Diversity, Equity and Inclusion Committee. J Allergy Clin Immunol. 2023 Mar;151(3):656-70. Full text (https://www.jacionline.org/article/S0091-6749(22)02555-6/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/36584926?tool=bestpractice.bmj.com)

25. Shreiner A, Huffnagle GB, Noverr MC. The "Microflora Hypothesis" of allergic disease. Adv Exp Med Biol. 2008;635:113-34. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18841708?tool=bestpractice.bmj.com)

26. Martinez FD, Holt PG. Role of microbial burden in aetiology of allergy and asthma. Lancet. 1999 Sep;354 (suppl 2):SII12-5. Full text (https://www.thelancet.com/journals/lancet/article/

PIIS0140-6736(99)90437-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/10507253?tool=bestpractice.bmj.com)

27. Björkstén B. Effects of intestinal microflora and the environment on the development of asthma and allergy. Springer Semin Immunopathol. 2004 Feb;25(3-4):257-70. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15007630?tool=bestpractice.bmj.com)

28. Fiocchi A, Pawankar R, Cuello-Garcia C, et al. World Allergy Organization-McMaster University guidelines for allergic disease prevention (GLAD-P): probiotics. World Allergy Organ J. 2015 Jan 27;8(1):4. Full text (https://waojournal.biomedcentral.com/articles/10.1186/s40413-015-0055-2) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25628773?tool=bestpractice.bmj.com)

29. Cuello-Garcia CA, Fiocchi A, Pawankar R, et al. World Allergy Organization-McMaster University guidelines for allergic disease prevention (GLAD-P): prebiotics. World Allergy Organ J. 2016 Mar 1;9:10. Full text (https://waojournal.biomedcentral.com/articles/10.1186/s40413-016-0102-7) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26962387?tool=bestpractice.bmj.com)

30. Wang DY. Risk factors of allergic rhinitis: genetic or environmental? Ther Clin Risk Manag. 2005 Jun;1(2):115-23. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1661616) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18360551?tool=bestpractice.bmj.com)

31. Barnes KC, Marsh DG. The genetics and complexity of allergy and asthma. Immunol Today. 1998 Jul;19(7):325-32. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/9666606?tool=bestpractice.bmj.com)

32. Testa D, DI Bari M, Nunziata M, et al. Allergic rhinitis and asthma assessment of risk factors in pediatric patients: a systematic review. Int J Pediatr Otorhinolaryngol. 2020 Feb;129:109759. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31734564?tool=bestpractice.bmj.com)

33. Wang XD, Zheng M, Lou HF, et al. An increased prevalence of self-reported allergic rhinitis in major Chinese cities from 2005 to 2011. Allergy. 2016 Aug;71(8):1170-80. Full text (https://onlinelibrary.wiley.com/doi/10.1111/all.12874) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26948849?tool=bestpractice.bmj.com)

34. Hassoun Y, James C, Bernstein DI. The effects of air pollution on the development of atopic disease. Clin Rev Allergy Immunol. 2019 Dec;57(3):403-14. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8215519) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30806950?tool=bestpractice.bmj.com)

35. Saulyte J, Regueira C, Montes-Martínez A, et al. Active or passive exposure to tobacco smoking and allergic rhinitis, allergic dermatitis, and food allergy in adults and children: a systematic review and meta-analysis. PLoS Med. 2014 Mar;11(3):e1001611. Full text (https://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.1001611) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24618794?tool=bestpractice.bmj.com)

36. Wallace DV. Pet dander and perennial allergic rhinitis: therapeutic options. Allergy Asthma Proc. 2009 Nov-Dec;30(6):573-83. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20031003?tool=bestpractice.bmj.com)

37. National Institute for Health and Care Excellence. Clinical knowledge summaries: allergic rhinitis. Aug 2021 [internet publication]. Full text (https://cks.nice.org.uk/topics/allergic-rhinitis)

38. Ojwang V, Nwaru BI, Takkinen HM, et al. Early exposure to cats, dogs and farm animals and the risk of childhood asthma and allergy. Pediatr Allergy Immunol. 2020 Apr;31(3):265-72. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31829464?tool=bestpractice.bmj.com)

39. Fall T, Lundholm C, Örtqvist AK, et al. Early exposure to dogs and farm animals and the risk of childhood asthma. JAMA Pediatr. 2015 Nov;169(11):e153219. Full text (https://jamanetwork.com/journals/jamapediatrics/fullarticle/2467334) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26523822?tool=bestpractice.bmj.com)

40. Riedler J, Braun-Fahrlander C, Eder W, et al. Exposure to farming in early life and development of asthma and allergy: a cross-sectional survey. Lancet. 2001 Oct 6;358(9288):1129-33. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/11597666?tool=bestpractice.bmj.com)

41. Stevenson MD, Sellins S, Grube E, et al. Aeroallergen sensitization in healthy children: racial and socioeconomic correlates. J Pediatr. 2007 Aug;151(2):187-91. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17643776?tool=bestpractice.bmj.com)

42. Szentpetery SE, Forno E, Canino G, et al. Asthma in Puerto Ricans: lessons from a high-risk population. J Allergy Clin Immunol. 2016 Dec;138(6):1556-8. Full text (https://www.jacionline.org/article/S0091-6749(16)31142-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27751794?tool=bestpractice.bmj.com)

43. Gillam SJ, Jarman B, White P, et al. Ethnic differences in consultation rates in urban general practice. BMJ. 1989 Oct 14;299(6705):953-7. Full text (https://www.bmj.com/content/bmj/299/6705/953.full.pdf) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/2508951?tool=bestpractice.bmj.com)

44. American Academy of Allergy, Asthma & Immunology. Ten things physicians and patients should question. Choosing Wisely, an initiative of the ABIM Foundation. 2021 [internet publication]. Full text (https://web.archive.org/web/20230402083712/https://www.choosingwisely.org/societies/american-academy-of-allergy-asthma-immunology)

45. Greer FR, Sicherer SH, Burks AW, et al; American Academy of Pediatrics: Committee on Nutrition; Section on Allergy and Immunology. The effects of early nutritional interventions on the development of atopic disease in infants and children: the role of maternal dietary restriction, breastfeeding, hydrolyzed formulas, and timing of introduction of allergenic complementary foods. Pediatrics. 2019 Apr;143(4):e20190281. Full text (https://publications.aap.org/pediatrics/article/143/4/e20190281/37226/The-Effects-of-Early-Nutritional-Interventions-on) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30886111?tool=bestpractice.bmj.com)

46. Kramer MS, Kakuma R. Maternal dietary antigen avoidance during pregnancy or lactation, or both, for preventing or treating atopic disease in the child. Cochrane Database Syst Rev. 2012 Sep 12;(9):CD000133. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD000133.pub3/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22972039?tool=bestpractice.bmj.com)

47. Cuello-Garcia CA, Brożek JL, Fiocchi A, et al. Probiotics for the prevention of allergy: a systematic review and meta-analysis of randomized controlled trials. J Allergy Clin Immunol. 2015 Oct;136(4):952-61. Full text (https://www.jacionline.org/article/S0091-6749(15)00636-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26044853?tool=bestpractice.bmj.com)

48. Obbagy JE, English LK, Wong YP, et al. Complementary feeding and food allergy, atopic dermatitis/eczema, asthma, and allergic rhinitis: a systematic review. Am J Clin Nutr. 2019 Mar 1;109(suppl_7):890S-934S. Full text (https://academic.oup.com/ajcn/article/109/Supplement_1/890S/5456693) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30982864?tool=bestpractice.bmj.com)

49. Grabenhenrich LB, Keil T, Reich A, et al. Prediction and prevention of allergic rhinitis: a birth cohort study of 20 years. J Allergy Clin Immunol. 2015 Oct;136(4):932-40.e12. Full text (https://www.jacionline.org/article/S0091-6749(15)00492-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25976706?tool=bestpractice.bmj.com)

50. American Academy of Otolaryngology—Head and Neck Surgery Foundation. Ten things physicians and patients should question. Choosing Wisely, an initiative of the ABIM Foundation. 2021 [internet publication]. Full text (https://web.archive.org/web/20230209023649/https://www.choosingwisely.org/societies/american-academy-of-otolaryngology-head-neck-surgery-foundation)

51. Seidman MD, Gurgel RK, Lin SY; Guideline Otolaryngology Development Group. AAO-HNSF. Clinical practice guideline: allergic rhinitis. Otolaryngol Head Neck Surg. 2015 Feb;152(1 Suppl):S1-43. Full text (https://journals.sagepub.com/doi/full/10.1177/0194599814561600) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25644617?tool=bestpractice.bmj.com)

52. Scadding GK, Kariyawasam HH, Scadding G, et al. BSACI guideline for the diagnosis and management of allergic and non-allergic rhinitis (revised edition 2017). Clin Exp Allergy. 2017 July;47(7):856-89. Full text (https://onlinelibrary.wiley.com/doi/10.1111/cea.12953) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30239057?tool=bestpractice.bmj.com)

53. Wise SK, Lin SY, Toskala E, et al. International consensus statement on allergy and rhinology: allergic rhinitis. Int Forum Allergy Rhinol. 2018 Feb;8(2):108-352. Full text (https://onlinelibrary.wiley.com/doi/full/10.1002/alr.22073) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29438602?tool=bestpractice.bmj.com)

54. Meltzer EO, Schatz M, Nathan R, et al. Reliability, validity, and responsiveness of the Rhinitis Control Assessment Test in patients with rhinitis. J Allergy Clin Immunol. 2013 Feb;131(2):379-86. Full text (https://www.jacionline.org/article/S0091-6749(12)01690-9/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23219170?tool=bestpractice.bmj.com)

55. Juniper EF, Thompson AK, Ferrie PJ, Roberts JN. Validation of the standardized version of the Rhinoconjunctivitis Quality of Life Questionnaire. J Allergy Clin Immunol. 1999 Aug;104(2 Pt 1):364-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/10452758?tool=bestpractice.bmj.com)

56. Juniper EF, Riis B, Juniper BA. Development and validation of an electronic version of the Rhinoconjunctivitis Quality of Life Questionnaire. Allergy. 2007 Sep;62(9):1091-3. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17521314?tool=bestpractice.bmj.com)

57. Bousquet J, Heinzerling L, Bachert C, et al. Practical guide to skin prick tests in allergy to aeroallergens. Allergy. 2012 Jan;67(1):18-24. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22050279?tool=bestpractice.bmj.com)

58. Hamizan AW, Rimmer J, Alvarado R, et al. Positive allergen reaction in allergic and nonallergic rhinitis: a systematic review. Int Forum Allergy Rhinol. 2017 Sep;7(9):868-77. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28727909?tool=bestpractice.bmj.com)

59. Blanc S, Bourrier T, Albertini M, et al. Dennie-Morgan fold plus dark circles: suspect atopy at first sight. J Pediatr. 2015 Jun;166(6):1541. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25890677?tool=bestpractice.bmj.com)

60. Lindberg S, Malm L. Comparison of allergic rhinitis and vasomotor rhinitis patients on the basis of a computer questionnaire. Allergy. 1993 Nov;48(8):602-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/8116859?tool=bestpractice.bmj.com)

61. Sur DKC, Plesa ML. Chronic nonallergic rhinitis. Am Fam Physician. 2018 Aug 1;98(3):171-6. Full text (https://www.aafp.org/afp/2018/0801/p171.html) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30215894?tool=bestpractice.bmj.com)

62. Bernstein JA, Brandt D, Ratner P, et al. Assessment of a rhinitis questionnaire in a seasonal allergic rhinitis population. Ann Allergy Asthma Immunol. 2008 May;100(5):512-3. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18517087?tool=bestpractice.bmj.com)

63. Meltzer EO, Hamilos DL, Hadley JA, et al. Rhinosinusitis: establishing definitions for clinical research and patient care. J Allergy Clin Immunol. 2004 Dec;114(6 Suppl):155-212. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15577865?tool=bestpractice.bmj.com)

64. Gurgel RK, Baroody FM, Damask CC, et al. Clinical practice guideline: immunotherapy for inhalant allergy. Otolaryngol Head Neck Surg. 2024 Mar;170 Suppl 1:S1-42. Full text (https://aao-hnsfjournals.onlinelibrary.wiley.com/doi/10.1002/ohn.648) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/38408152?tool=bestpractice.bmj.com)

65. Okubo K, Kurono Y, Ichimura K, et al. Japanese guidelines for allergic rhinitis 2020. Allergol Int. 2020 Jul;69(3):331-45. Full text (https://www.sciencedirect.com/science/article/pii/S1323893020300502) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32473790?tool=bestpractice.bmj.com)

66. Australasian Society of Clinical Immunology and Allergy (ASCIA). Australasian guidelines for allergic rhinitis 2020. 2022 [internet publication]. Full text (https://www.allergy.org.au/hp/papers)

67. Bousquet J, Schünemann HJ, Togias A, et al. Next-generation Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines for allergic rhinitis based on Grading of Recommendations Assessment, Development and Evaluation (GRADE) and real-world evidence. J Allergy Clin Immunol. 2020 Jan;145(1):70-80.e3. Full text (https://www.jacionline.org/article/S0091-6749(19)31187-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31627910?tool=bestpractice.bmj.com)

68. NHS Specialist Pharmacy Service. Which oral antihistamines are safe to use whilst breastfeeding? Apr 2020 [internet publication]. Full text (https://www.sps.nhs.uk/articles/which-oral-antihistamines-are-safe-to-use-whilst-breastfeeding)

69. Kok TH, Taitz LS, Bennett MJ, et al. Drowsiness due to clemastine transmitted in breast milk. Lancet. 1982 Apr 17;1(8277):914-5. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/6122135?tool=bestpractice.bmj.com)

70. US Food and Drug Administration. FDA drug safety communication: FDA requires Boxed Warning about serious mental health side effects for asthma and allergy drug montelukast (Singulair); advises restricting use for allergic rhinitis. Mar 2020 [internet publication]. Full text (https://www.fda.gov/drugs/drug-safety-and-availability/fda-requires-boxed-warning-about-serious-mental-health-side-effects-asthma-and-allergy-drug)

71. Australasian Society of Clinical Immunology and Allergy. Allergen minimisation. Mar 2019 [internet publication]. Full text (https://www.allergy.org.au/patients/allergy-treatments/allergen-minimisation)

72. Sheikh A, Hurwitz B, Nurmatov U, et al. House dust mite avoidance measures for perennial allergic rhinitis. Cochrane Database Syst Rev. 2010 Jul 7;(7):CD001563. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD001563.pub3/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20614426?tool=bestpractice.bmj.com)

73. Vredegoor DW, Willemse T, Chapman MD, et al. Can f 1 levels in hair and homes of different dog breeds: lack of evidence to describe any dog breed as hypoallergenic. J Allergy Clin Immunol. 2012 Oct;130(4):904-9.e7. Full text (https://www.jacionline.org/article/S0091-6749(12)00793-2/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22728082?tool=bestpractice.bmj.com)

74. Liem O, Kessen K, de Groot H. Hypoallergenic animals, fact or myth? [in Dutch]. Ned Tijdschr Geneeskd. 2019 Dec 31;164:D4298. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32186820?tool=bestpractice.bmj.com)

75. Nageotte C, Park M, Havstad S, et al. Duration of airborne Fel d 1 reduction after cat washing. J Allergy Clin Immunol. 2006 Aug;118(2):521-2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/16890781?tool=bestpractice.bmj.com)

76. Klucka CV, Ownby DR, Green J, et al. Cat shedding of Fel d I is not reduced by washings, Allerpet-C spray, or acepromazine. J Allergy Clin Immunol. 1995 Jun;95(6):1164-71. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/7797784?tool=bestpractice.bmj.com)

77. Sulser C, Schulz G, Wagner P, et al. Can the use of HEPA cleaners in homes of asthmatic children and adolescents sensitized to cat and dog allergens decrease bronchial hyperresponsiveness and allergen contents in solid dust? Int Arch Allergy Immunol. 2009;148(1):23-30. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18716400?tool=bestpractice.bmj.com)

78. Wood RA, Johnson EF, Van Natta ML, et al. A placebo-controlled trial of a HEPA air cleaner in the treatment of cat allergy. Am J Respir Crit Care Med. 1998 Jul;158(1):115-20. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/9655716?tool=bestpractice.bmj.com)

79. Huss K, Adkinson NF Jr, Eggleston PA, et al. House dust mite and cockroach exposure are strong risk factors for positive allergy skin test responses in the Childhood Asthma Management Program. J Allergy Clin Immunol. 2001 Jan;107(1):48-54. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/11149990?tool=bestpractice.bmj.com)

80. Wood RA, Eggleston PA, Rand C, et al. Cockroach allergen abatement with extermination and sodium hypochlorite cleaning in inner-city homes. Ann Allergy Asthma Immunol. 2001 Jul;87(1):60-4. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/11476465?tool=bestpractice.bmj.com)

81. Bousquet J, Devillier P, Arnavielhe S, et al. Treatment of allergic rhinitis using mobile technology with real-world data: the MASK observational pilot study. Allergy. 2018 Sep;73(9):1763-74. Full text (https://onlinelibrary.wiley.com/doi/10.1111/all.13406) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29336067?tool=bestpractice.bmj.com)

82. Xiao J, Wu WX, Ye YY, et al. A network meta-analysis of randomized controlled trials focusing on different allergic rhinitis medications. Am J Ther. 2016 Nov/Dec;23(6):e1568-78. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25867532?tool=bestpractice.bmj.com)

83. New Zealand Medicines and Medical Devices Safety Authority (Medsafe). Children and sedating antihistamines. Mar 2013 [internet publication]. Full text (https://www.medsafe.govt.nz/profs/PUArticles/Mar2013ChildrenAndSedatingAntihistamines.htm)

84. Blaiss MS. Evolving paradigm in the management of allergic rhinitis-associated ocular symptoms: role of intranasal corticosteroids. Curr Med Res Opin. 2008 Mar;24(3):821-36. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18257976?tool=bestpractice.bmj.com)

85. Naclerio R. Intranasal corticosteroids reduce ocular symptoms associated with allergic rhinitis. Otolaryngol Head Neck Surg. 2008 Feb;138(2):129-39. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18241703?tool=bestpractice.bmj.com)

86. Dhami S, Nurmatov U, Arasi S, et al. Allergen immunotherapy for allergic rhinoconjunctivitis: a systematic review and meta-analysis. Allergy. 2017 Nov;72(11):1597-631. Full text (https://onlinelibrary.wiley.com/doi/full/10.1111/all.13201) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28493631?tool=bestpractice.bmj.com)

87. Radulovic S, Calderon MA, Wilson D, et al. Sublingual immunotherapy for allergic rhinitis. Cochrane Database Syst Rev. 2010 Dec 8;(12):CD002893. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD002893.pub2/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21154351?tool=bestpractice.bmj.com)

88. Jacobsen L, Niggemann B, Dreborg S, et al. Specific immunotherapy has long-term preventive effect of seasonal and perennial asthma: 10-year follow-up on the PAT study. Allergy. 2007 Aug;62(8):943-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17620073?tool=bestpractice.bmj.com)

89. Durham SR, Emminger W, Kapp A, et al. SQ-standardized sublingual grass immunotherapy: confirmation of disease modification 2 years after 3 years of treatment in a randomized trial. J Allergy Clin Immunol. 2012 Mar;129(3):717-25.e5. Full text (https://www.jacionline.org/article/

S0091-6749(11)02942-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22285278?tool=bestpractice.bmj.com)

90. Dretzke J, Meadows A, Novielli N, et al. Subcutaneous and sublingual immunotherapy for seasonal allergic rhinitis: a systematic review and indirect comparison. J Allergy Clin Immunol. 2013 May;131(5):1361-6. Full text (https://www.jacionline.org/article/S0091-6749(13)00323-0/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23557834?tool=bestpractice.bmj.com)

91. Lin SY, Erekosima N, Kim JM, et al. Sublingual immunotherapy for the treatment of allergic rhinoconjunctivitis and asthma: a systematic review. JAMA. 2013 Mar 27;309(12):1278-88. Full text (https://jamanetwork.com/journals/jama/fullarticle/1672214) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23532243?tool=bestpractice.bmj.com)

92. Durham SR, Creticos PS, Nelson HS, et al. Treatment effect of sublingual immunotherapy tablets and pharmacotherapies for seasonal and perennial allergic rhinitis: pooled analyses. J Allergy Clin Immunol. 2016 Oct;138(4):1081-8. Full text (https://www.jacionline.org/article/S0091-6749(16)30614-5/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27527264?tool=bestpractice.bmj.com)

93. Di Bona D, Plaia A, Leto-Barone MS, et al. Efficacy of subcutaneous and sublingual immunotherapy with grass allergens for seasonal allergic rhinitis: a meta-analysis-based comparison. J Allergy Clin Immunol. 2012 Nov;130(5):1097-107.e2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23021885?tool=bestpractice.bmj.com)

94. Canonica GW, Cox L, Pawankar R, et al. Sublingual immunotherapy: World Allergy Organization position paper 2013 update. World Allergy Organ J. 2014 Mar 28;7(1):6. Full text (https://www.worldallergyorganizationjournal.org/article/S1939-4551(19)30238-8/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24679069?tool=bestpractice.bmj.com)

95. Calderon MA, Eichel A, Makatsori M, et al. Comparability of subcutaneous and sublingual immunotherapy outcomes in allergic rhinitis clinical trials. Curr Opin Allergy Clin Immunol. 2012 Jun;12(3):249-56. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22499145?tool=bestpractice.bmj.com)

96. Demoly P, Emminger W, Rehm D, et al. Effective treatment of house dust mite-induced allergic rhinitis with 2 doses of the SQ HDM SLIT-tablet: results from a randomized double-blind, placebo-controlled phase III trial. J Allergy Clin Immunol. 2016 Feb;137(2):444-51.e8. Full text (https://www.jacionline.org/article/S0091-6749%2815%2900935-5/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26292778?tool=bestpractice.bmj.com)

97. Blaiss M, Maloney J, Nolte H, et al. Efficacy and safety of timothy grass allergy immunotherapy tablets in North American children and adolescents. J Allergy Clin Immunol. 2011 Jan;127(1):64-71, 71.e1-4. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21211642?tool=bestpractice.bmj.com)

98. Durham SR; GT-08 investigators. Sustained effects of grass pollen AIT. Allergy. 2011 Jul;66 (Suppl 95):50-2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21668855?tool=bestpractice.bmj.com)

99. Nelson HS, Nolte H, Creticos P, et al. Efficacy and safety of timothy grass allergy immunotherapy tablet treatment in North American adults. J Allergy Clin Immunol. 2011 Jan;127(1):72-80, 80.e1-2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21211643?tool=bestpractice.bmj.com)

100. Creticos PS, Esch RE, Couroux P, et al. Randomized, double-blind, placebo-controlled trial of standardized ragweed sublingual-liquid immunotherapy for allergic rhinoconjunctivitis. J Allergy Clin Immunol. 2014 Mar;133(3):751-8. Full text (https://www.jacionline.org/article/S0091-6749(13)01702-8/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24332263?tool=bestpractice.bmj.com)

101. Durham SR, Emminger W, Kapp A, et al. Long-term clinical efficacy in grass pollen-induced rhinoconjunctivitis after treatment with SQ-standardized grass allergy immunotherapy tablet. J Allergy Clin Immunol. 2010 Jan;125(1):131-8.e1-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20109743?tool=bestpractice.bmj.com)

102. Walker SM, Pajno GB, Lima MT, et al. Grass pollen immunotherapy for seasonal rhinitis and asthma: a randomized, controlled trial. J Allergy Clin Immunol. 2001 Jan;107(1):87-93. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/11149996?tool=bestpractice.bmj.com)

103. Nacaroglu HT, Erdem SB, Sumer O, et al. Local and systemic reactions to subcutaneous allergen immunotherapy: ten years' experience in a pediatric clinic. Ann Allergy Asthma Immunol. 2016 Apr;116(4):349-53. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26905639?tool=bestpractice.bmj.com)

104. Gur Cetinkaya P, Kahveci M, Esenboğa S, et al. Systemic and large local reactions during subcutaneous grass pollen immunotherapy in children. Pediatr Allergy Immunol. 2020 Aug;31(6):643-50. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32320504?tool=bestpractice.bmj.com)

105. Li JT. Immunotherapy for allergic rhinitis. Immunol Allergy Clin North Am. 2000 May;20(2):383-400. Full text (https://www.immunology.theclinics.com/article/S0889-8561(05)70154-5/fulltext)

106. Bernstein DI, Wanner M, Borish L, et al. Twelve-year survey of fatal reactions to allergen injections and skin testing: 1990-2001. J Allergy Clin Immunol. 2004 Jun;113(6):1129-36. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15208595?tool=bestpractice.bmj.com)

107. Greenhawt M, Oppenheimer J, Nelson M, et al. Sublingual immunotherapy: a focused allergen immunotherapy practice parameter update. Ann Allergy Asthma Immunol. 2017 Mar;118(3):276-82.e2. Full text (https://www.aaaai.org/Aaaai/media/Media-Library-PDFs/Allergist%20Resources/Statements%20and%20Practice%20Parameters/Sublingual-Immunotherapy-2017.pdf) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28284533?tool=bestpractice.bmj.com)

108. Roberts G, Pfaar O, Akdis CA, et al. EAACI Guidelines on allergen immunotherapy: allergic rhinoconjunctivitis. Allergy. 2018 Apr;73(4):765-98. Full text (https://onlinelibrary.wiley.com/doi/full/10.1111/all.13317) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28940458?tool=bestpractice.bmj.com)

109. Linna O, Kokkonen J, Lukin M. A 10-year prognosis for childhood allergic rhinitis. Acta Paediatr. 1992 Feb;81(2):100-2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/1515750?tool=bestpractice.bmj.com)

110. Greisner WA III, Settipane RJ, Settipane GA. Natural history of hay fever: a 23-year follow-up of college students. Allergy Asthma Proc. 1998 Sep-Oct;19(5):271-5. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/9801740?tool=bestpractice.bmj.com)

111. Al-Shaikhly T, Cox A, Nowak-Wegrzyn A, et al. An International Delphi consensus on the management of pollen-food allergy syndrome: a work group report of the AAAAI adverse reactions to foods committee. J Allergy Clin Immunol Pract. 2024 Dec;12(12):3242-9. Full text (https://www.jaci-inpractice.org/article/S2213-2198(24)01069-9/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/39488768?tool=bestpractice.bmj.com)

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This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

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numerals < 1: 0.25

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