Allergy: an increasing problem

Classical rhinitis

Seasonal allergic rhinitis

Non-allergic rhinitis

Diagnosing rhinitis

Therapeutic principles

Rhinosinusitis and nasal polyps

Comorbidities and complications of allergic rhinitis

Future trends
Fast Facts: Rhinitis

Second edition

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Declaration of Independence
This book is as balanced and as practical as we can make it.
Ideas for improvement are always welcome: feedback@fastfacts.com

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Glossary of abbreviations

ACE inhibitor: angiotensin-converting enzyme inhibitor

ARIA: Allergic Rhinitis and its Impact on Asthma (an initiative of the World Health Organization and other groups)

ASA: acetylsalicylic acid (aspirin)

AU: allergy units

BU: biological units

EAACI: European Academy of Allergology and Clinical Immunology

eNO: exhaled nitric oxide

ENT: ear, nose and throat

FEV₁: forced expiratory volume in 1 minute

GM-CSF: granulocyte-macrophage colony-stimulating factor

ICAM: intercellular adhesion molecule

Ig: immunoglobulin

IL: interleukin

NARES: non-allergic rhinitis with eosinophilia

nNO: nasal nitric oxide

NO: nitric oxide

NSAID: non-steroidal anti-inflammatory drug

OME: otitis media with effusion

PCD: primary ciliary dyskinesia

RAST: radioallergosorbent test

SPT: skin-prick test

Th₁: T helper 1 (cells)

Th₂: T helper 2 (cells)

WHO: World Health Organization
Introduction

Allergic diseases have been described as a modern epidemic, with over 20% of the total population suffering from allergic rhinitis, asthma or atopic eczema. Of these diseases, allergic rhinitis is the most common and the most underdiagnosed and mistreated.

The effects of the symptoms of rhinitis, allergic or otherwise, on quality of life, particularly through sleep disturbance, are beginning to be appreciated. The long-term effects of poor school and examination performance are beginning to be investigated. The comorbidity of allergic rhinitis with asthma – the ‘one airway’ hypothesis – is generally accepted, but other relevant comorbidities involving the eyes, throat, ears and voice are still being elucidated. Studies suggest that accurate diagnosis of rhinitis and effective treatment can reduce other chest and ear, nose and throat problems.

Severely affected individuals are treated by specialists in allergy, lung medicine, pediatrics, otorhinolaryngology and dermatology. The majority of people, however, are seen by generalists or by specialists in other fields. We have written this short and practically oriented text on rhinitis, including not only allergic but other forms of rhinitis plus acute and chronic rhinosinusitis, nasal polyposis and comorbidities such as asthma, adenoid hypertrophy and otitis media with effusion, for these practitioners. We hope that it will enable them to optimize their treatment and to know when and to whom to refer those with difficult disease.
Allergy is common: 20–40% of the population of the industrialized world have a positive skin-prick test for allergens, and 15–20% will develop an atopic disease (see below). The prevalence of atopic diseases is highest among teenagers.

Allergy is common in very young children, though it can be difficult to discriminate it from recurrent viral upper airway disease. In Germany, 6% of children under 2 years old were found to be sensitized to grass and 3% were sensitized to house dust mite; in 40% of children, allergic rhinitis started before the age of 6 years, and 80% developed symptoms before the age of 20.

The risk of developing allergic disease is influenced by genetic and environmental factors (Figure 1.1).

**Genetic factors**

‘Atopy’ refers to a genetic predisposition to produce immunoglobulin E (IgE) in response to minute amounts of environmental protein allergens. Non-atopic individuals can produce IgE, but normally do so only transiently. In atopic individuals, the production continues and leads to various atopic disorders, such as:

- atopic dermatitis or eczema
- asthma
- allergic rhinitis.

A highly atopic person is affected early in life, developing atopic dermatitis soon after birth; asthma and allergic rhinitis develop subsequently. This is sometimes called the ‘atopic march’.

On the other hand, a person who becomes allergic to pollen in adolescence has a low degree of atopy and is less likely to be troubled by eczema. However, progression from seasonal rhinitis to a more persistent form and to rhinosinusitis and asthma is now increasingly recognized.

With one atopic parent, the risk of atopy in the child is doubled, with maternal influence being greater than paternal. If both parents are
atopic, the risk is quadrupled. Several genes are involved and children probably inherit predispositions: for atopic disease in general, for specific organ involvement and for disease severity. Thus, a child with one parent with hay fever is likely to be less severely affected than one whose parents have severe eczema and asthma.

Environmental exposure

A low concordance rate for atopy among monozygotic twins shows that genetic inheritance is not the sole arbiter of the atopic state. Birth immediately before a pollen season leads to a slightly increased risk of
The nose acts as an air conditioner that filters, warms and humidifies 10,000 liters of air daily. To accomplish this, it has a specialized structure that incorporates:

- a narrow slit-like orifice
- a large surface area
- mucus and cilia
- a turbulent airflow
- a bend into the nasopharynx (Figures 3.1 and 3.2).

The turbinate bones or conchae, of which there are two or three on each side, cause turbulent airflow so that particles are deposited and can be cleared by the mucociliary system. This moves them to the back of

**Figure 3.1** Lateral wall of nasal cavity. The arrow points to the internal ostium, and the colored area is the olfactory region. The openings from the naso-lacrimal duct and the paranasal sinuses are under the inferior, middle and superior turbinate.
the pharynx where they are swallowed. Many particles are deposited on the bend into the nasopharynx. Very few particles larger than 10 µm penetrate further than the nose; particles smaller than 2 µm do not tend to become deposited in the nose. Pollen grains are 20–30 µm in size and tend to cause allergic rhinitis, whereas mold spores, at 2 µm, tend to cause asthma rather than rhinitis. Water-soluble gases, such as sulfur dioxide, formaldehyde and ozone, are retained in the nose and can cause irritation of the nasal mucosa.

The width of the nasal passages is regulated by sympathetic nervous tone that acts on the venous sinusoids. Most individuals have a nasal cycle, with one nostril being more patent than the other for 2–4 hours at a time. This is controlled at brainstem level and is usually inapparent unless a degree of nasal obstruction is present.

Because of its filtration function, the nose is the site of more allergy-related symptoms and illnesses than any other organ.