Australian Government
Australian Institute of Health and Welfare

Authoritative information and statistics to promote better health and wellbeing

Allergic rhinitis ('hay fever') in Australia

November 2011

Australian Institute of Health and Welfare
Canberra
Cat. no. ACM 23

The Australian Institute of Health and Welfare is a major national agency that provides reliable, regular and relevant information and statistics on Australia's health and welfare. The Institute's mission is *authoritative information and statistics to promote better health and wellbeing*.

A complete list of the Institute's publications is available from the Institute's website <www.aihw.gov.au>.

ISBN 978-1-74249-228-5

Suggested citation

Australian Institute of Health and Welfare 2011. Allergic rhinitis ('hay fever') in Australia. Cat. no. ACM 23. Canberra: AIHW.

Any enquiries about or comments on this publication should be directed to:

Communications, Media and Marketing Unit

Australian Institute of Health and Welfare

GPO Box 570

Canberra ACT 2601

Tel: (02) 6244 1032

Email: info@aihw.gov.au

Published by the Australian Institute of Health and Welfare

Please note that there is the potential for minor revisions of data in this report. Please check the online version at <www.aihw.gov.au> for any amendments.

Contents

Acknowledgments ..... **iv**
Summary ..... **v**
Abbreviations ..... **vi**
1 Introduction **1**
1.1 Relationship to other conditions 2
1.2 Structure of report 3
2 What causes and worsens allergic rhinitis? **4**
2.1 Genetic factors 4
2.2 Age 4
2.3 Early life factors 5
2.4 Allergen exposure 5
2.5 Pollutants 7
2.6 Occupational exposures 10
3 Who has allergic rhinitis? **12**
3.1 Age and sex 12
3.2 Geography 14
3.3 Country of birth 16
4 How is allergic rhinitis managed? **18**
4.1 Use of medications for management of allergic rhinitis 18
4.2 Management of allergic rhinitis in general practice 27
4.3 Management of allergic rhinitis in hospital 29
Discussion **32**
Appendix A: Definitions and data sources for medication data **34**
A1 Regulatory control of medication supply 34
A2 Medication data 34
A3 Defined daily dose as a measure of drug utilisation 35
Glossary **36**
References **38**

Acknowledgments

This report was prepared by Eric Henry, Malcolm Gall, Katarzyna Krysiak, Alice Crisp, Tomoko Sugiura and Adrian Webster of the Primary Health and Respiratory and Musculoskeletal Monitoring Unit at the Australian Institute of Health and Welfare.

Preparation of this edition of *Allergic rhinitis ('hay fever') in Australia* was guided by members of the Asthma Advisory Group, chaired by Carol Armour. Members of the Asthma Advisory Group are Christine Jenkins, Alan James, Anne Chang, Graeme Maguire, Sean Walsh, Michael Abramson, Amanda Barnard, Peter Gibson and Paul Magnus.

This publication was funded by the Australian Government Department of Health and Ageing.

Summary

Hay fever is a common term referring to allergic rhinitis caused by seasonal exposure to pollen. Allergic rhinitis is a runny or blocked nose and/or sneezing and watery eyes, and is triggered by an allergic reaction. Allergic rhinitis can have many triggers and can occur seasonally or throughout the year.

Allergic rhinitis is one of the most common chronic respiratory conditions in Australia. Unlike many health conditions, allergic rhinitis is more common in those of working age than it is in the young and the elderly.

Allergic rhinitis can cause significant irritation and interference in a sufferer's daily activities, considerably reducing the quality of life.

Causes and exacerbating factors

The symptoms of allergic rhinitis are caused by an allergic reaction in the inner linings of the nose.

Common triggers of allergic rhinitis come from house dust, animal fur, pollens, fungal spores, air pollutants and occupational sources.

Prevalence

Based on self-reports from the 2007–08 National Health Survey, allergic rhinitis affects around 15% of the Australian population, or about 3.1 million people.

It is more commonly reported by females than males.

It is most commonly reported by those aged 25–44 years, and least commonly by those in the 0–14 and 65–74 year age groups.

The Australian Capital Territory and Western Australia have the highest rates of allergic rhinitis in Australia, and Queensland and New South Wales have the lowest.

Use of medications

The main medications used in the treatment of allergic rhinitis are intranasal corticosteroids (nasal sprays) and oral antihistamines.

According to IMS Health wholesale data, the amount of money paid by community pharmacies to wholesalers for these medications doubled between 2001 ($107.8 million) and 2010 ($226.8 million). Although not all of these medications would have been used for allergic rhinitis, it is likely to have accounted for a large proportion of this increase.

Management in hospital

Allergic rhinitis is not a common cause of hospitalisation. In 2008–09, hospitalisations for allergic rhinitis (as a principal diagnosis or additional diagnosis) represented around 0.02% of all hospitalisations.

Males are more likely to be hospitalised for allergic rhinitis than females, and children are more likely to be hospitalised than adults.

Other conditions that commonly occur together with allergic rhinitis

Conditions that commonly occur alongside allergic rhinitis include asthma, chronic sinusitis, otitis media (middle ear infection) and decreased quality of sleep.

Abbreviations

AIHW	Australian Institute of Health and Welfare
ARIA	Allergic Rhinitis and its Impact on Asthma
BEACH	Bettering the Evaluation and Care of Health (survey)
DDD	defined daily dose
GP	general practitioner
IAR	intermittent allergic rhinitis
NHS	National Health Survey
PBS	Pharmaceutical Benefits Scheme
PER	persistent allergic rhinitis
SUSMP	Standard for the Uniform Scheduling of Medicines and Poisons
VOC	volatile organic compound
WHOCC	World Health Organization Collaborating Centre for Drug Statistics Methodology

1 Introduction

Allergic rhinitis is a common chronic respiratory condition (Box 1.1). It is one of the most common chronic respiratory conditions in Australia, with 15% of the population reporting that they have allergic rhinitis (ABS 2009). It can affect sleep, mental and voluntary motor function, and participation in social activities. It frequently coexists with other allergic conditions such as allergic asthma and chronic sinusitis (Hu et al. 2008). These effects lead to a number of indirect and direct costs to Australians.

This chapter briefly outlines what allergic rhinitis is, why it is of concern in an Australian context and its relationship to other conditions.

Rhinitis is defined as an inflammation of the nose and is characterised by nasal symptoms including anterior or posterior rhinorrhoea (runny nose), sneezing, nasal blockage and/or itching of the nose (Bousquet et al. 2008).

Box 1.1: Classifying allergic rhinitis

The Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 report divides allergic rhinitis into two categories: intermittent allergic rhinitis (IAR) and persistent allergic rhinitis (PER).

IAR is defined by symptoms that are present for less than 4 days per week, or for less than 4 weeks at a time.

PER is defined by symptoms that are present for more than 4 days per week, and for more than 4 weeks at a time (Bousquet et al. 2001).

Before the 2008 ARIA report, allergic rhinitis was subdivided, based on time of exposure, into seasonal, perennial and occupational. Perennial allergic rhinitis was said to be most frequently caused by indoor allergens (allergy-causing substances) such as those from dust mites, moulds, insects and animal danders. Seasonal allergic rhinitis was said to be related to a wide variety of outdoor allergens such as pollens or moulds (Bousquet et al. 2008). This classification was not seen by ARIA as entirely satisfactory as:

- In certain areas pollens and moulds are perennial allergens.
- Symptoms of perennial allergic rhinitis may not always be present all year round.
- Most sufferers are sensitised to many different allergens and can therefore be exposed throughout the year.
- Climatic conditions modify the time and duration of the pollen season, making predictions difficult.
- Allergic rhinitis sufferers who travel may be exposed to sensitising allergens at different times of the year.
- Some sufferers sensitised to only a single pollen species have perennial symptoms.
- Symptoms do not necessarily occur strictly in conjunction with the allergen season.
- Nonspecific irritants can induce or aggravate symptoms (Bousquet et al. 2008).

For these reasons IAR and PER are not synonymous with ‘seasonal’ and ‘perennial’.

This change in classification affects this report as the research cited uses differing terminology depending on when it was conducted.

The severity of allergic rhinitis is classified as either mild or moderate/severe. With mild allergic rhinitis, there is no impairment of sleep, daily activities, leisure or sport, and school or work; and while symptoms are present, they are not troublesome. With moderate/severe allergic rhinitis, there are one or more of the following impairments:

- impairment of sleep
- impairment of daily activities, leisure or sport
- impairment of school or work
- troublesome symptoms (Bousquet et al. 2001).

Although there is no cure for allergic rhinitis, effective treatment is available.

1.1 Relationship to other conditions

Allergic rhinitis is closely linked to a number of other conditions, either as a contributing factor or through sharing risk and trigger factors.

Asthma

The relationship between allergic rhinitis and asthma has been investigated in recent epidemiological surveys, research studies and clinical trials (Bousquet et al. 2008).

Allergic rhinitis and asthma share many of their risk and trigger factors. In particular, atopy (a genetic tendency to develop allergic reactions) is a strong predisposing factor for developing allergic rhinitis and allergic asthma (allergic asthma accounts for around 60–85% of all asthma cases) (Bousquet et al. 2008).

Allergic rhinitis often precedes the development of asthma and the two conditions commonly coexist in patients (Thomsen et al. 2005). About 80% of asthmatics have rhinitis and between 15% and 30% of rhinitis patients have asthma (Bousquet et al. 2008). Both conditions are strongly associated with atopy.

In 2004–05, of the around 3.2 million Australians with allergic rhinitis and 2 million with asthma, about 700,000 had both conditions (AIHW 2010).

There is evidence that, in a patient who has both allergic rhinitis and asthma, the asthma symptoms are more difficult to control than in an asthma patient without allergic rhinitis (van den Berge et al. 2002).

One reason that has been suggested for the relationship between allergic rhinitis and asthma is the 'integrated airway hypothesis', which suggests that these conditions may be differing manifestations of the same inflammatory process within the airway (Box 1.2).

Box 1.2: The integrated airway hypothesis

Some investigators have referred to allergic rhinitis, asthma and chronic sinusitis as a 'united airways disease'. This suggests that these conditions may be differing manifestations of the same inflammatory process within the airway rather than fully separate diseases. Based on this hypothesis, the presence of upper airway symptoms (that is, symptoms affecting the nose, sinuses, larynx, pharynx or trachea) such as allergic rhinitis may worsen the natural course of lower airway disease (diseases of the lungs or bronchi) such as asthma (Meltzer et al. 2004).

Chronic sinusitis

In 2004–05, there were about 3.2 million Australians with self-reported allergic rhinitis and 1.8 million with self-reported chronic sinusitis. Around 800,000 people reported having both allergic rhinitis and chronic sinusitis, a combination often referred to as 'rhinosinusitus' (AIHW 2010).

As with asthma, allergic rhinitis and chronic sinusitis might be linked by the 'integrated airway hypothesis' (Box 1.2). Rhinitis causes swelling that obstructs the point where the sinuses normally drain into the nasal cavity.

Otitis media (middle ear infection)

Otitis media is an inflammatory disease of the lining of the middle ear. It is a significant problem among children. It is estimated that more than 80% of all children experience at least one episode by the age of 3 and that 40% will have three or more further episodes (Teele et al. 1989). Otitis media can cause ear pain and fever in the acute stage, and can lead to hearing loss if allowed to become chronic.

One study of children with a history of chronic or recurrent otitis media with effusion (sometimes referred to as 'glue ear') showed that 89% of them also suffered from allergic rhinitis (Alles et al. 2001). However, there is no established cause-and-effect relationship of rhinitis with recurrent otitis media or otitis media with effusion (Wallace et al. 2008).

Sleep disturbance

Decreased quality of sleep is often reported in patients with allergic rhinitis, particularly in patients with moderate/severe cases (Bousquet et al. 2001). Studies indicate that the nasal congestion associated with allergic rhinitis causes disrupted sleep and subsequent daytime drowsiness (Craig et al. 2004).

Although sleep apnoea has been associated with nasal disturbances (Rubinstein 1995), it is unclear whether allergic rhinitis is specifically associated with it (Bousquet et al. 2008).

1.2 Structure of report

This report aims to investigate the burden of allergic rhinitis in Australia by examining how prevalent it is and how it is managed by individuals and the health system, and by assessing its effects on other conditions. Included is information on what is known about risk and trigger factors for allergic rhinitis, medication use, general practice encounters, and the relationship of allergic rhinitis to other conditions.

A glossary is provided at the end of the publication to describe terms not in common use.

2 What causes and worsens allergic rhinitis?

The symptoms of allergic rhinitis are caused by an allergic reaction resulting in inflammation of the nasal mucosa.

Allergic reactions require sensitisation and exposure to an allergen. Sensitisation (see Glossary) to highly allergenic indoor allergens can occur in children younger than 2 years. Sensitisation to outdoor allergens usually occurs when a child is older than 3–5 years, and the average age at presentation is 9–10 years (Becker 2009).

The exact process of sensitisation is not entirely understood but, in order to be sensitised, an individual must be repeatedly exposed to an allergen, during which time the nasal mucosa becomes hyperresponsive to the allergen (Scarupa & Kaliner 2006).

Once an individual is sensitised, even nonspecific triggers or small amounts of the antigen can cause a rapid allergic response and severe symptoms (Becker 2009).

The process of sensitisation can occur at any age but it occurs most often in infants and children, and in people with a family history of allergy (Wang 2005).

Both genetic and environmental factors contribute to the onset (that is, sensitisation to an allergen) and development of allergic rhinitis.

A risk factor for allergic rhinitis may act in one or more of the following ways:

- increase the risk of an individual developing an allergic sensitisation
- induce a nasal response in someone with allergic rhinitis. This may be either through allergic or non-allergic mechanisms
- interact with the allergen to increase the individual's level of allergic reaction.

The following section describes some of the common risk factors for allergic rhinitis and, where known, explains how the factor affects the individual.

2.1 Genetic factors

The presence of certain genes increases a person's susceptibility to allergic rhinitis (Brasch-Andersen et al. 2006; Pinto et al. 2008_ENREF_53). In particular, genes associated with atopy have been shown to be risk factors for the condition (Brasch-Andersen et al. 2006).

Atopic individuals have an increased chance of developing not only allergic rhinitis, but also other allergic conditions such as asthma, dermatitis or eczema (World Allergy Organization 2003).

2.2 Age

In comparison with other forms of rhinitis, allergic rhinitis generally first presents at a younger age and frequent symptoms are evident by young adulthood. Non-allergenic forms of rhinitis, such as vasomotor, hormonal or irritant-induced rhinitis, are usually not evident until later in life (Quillen & Feller 2006; Settipane 2003).

Seasonal allergic rhinitis has a lower median onset age than perennial rhinitis. The median onset age for seasonal allergic rhinitis is 15 years compared with 20 years for perennial rhinitis (Sibbald & Rink 1991).

2.3 Early life factors

Exposure to tobacco smoke in early childhood may increase allergic sensitisation in some children (California Environmental Protection Agency: Air Resources Board 2005; Martinez et al. 1988). However, there has been no association found between exposure to maternal tobacco smoking during pregnancy and an increased risk of allergic rhinitis (Raherison et al. 2007).

After several studies noted that the risk of developing allergies is inversely related to the number of children in the family, a hypothesis (known as the 'hygiene hypothesis') was suggested in the late 1980s. According to this hypothesis, early exposure to infections with viruses and microbial organisms influences the developing immune system and reduces an individual's susceptibility to allergic diseases. The underlying mechanisms of the protective effects of multiple siblings in a family are still unclear.

Studies have shown that the risk of early onset allergic rhinitis decreases with increasing viral infections during childhood (Matheson et al. 2009). However, some other studies have demonstrated that some specific respiratory infections, such as whooping cough and respiratory syncytial virus (RSV), and some forms of gastroenteritis may increase allergic sensitisation (World Allergy Organization 2003).

Young maternal age (Sibbald & Strachan 1995), prematurity and low birthweight (Bousquet et al. 2008;) have all been inconsistently related to the risk of developing allergic rhinitis.

2.4 Allergen exposure

Allergens are antigens (see Glossary) that react with specific antibodies (see Glossary) to produce an allergic reaction in a susceptible individual.

Allergens originate from a wide range of insects, plants, fungi, mammals and occupational sources.

The most commonly implicated allergens in allergic rhinitis patients are inhalant allergens (that is, allergens that are breathed in) (Bousquet et al. 2008). Discussion of each of these follows.

House dust

House dust contains numerous organic and inorganic compounds, including:

- house dust mites
- hair
- smoke
- dirt
- fibres
- mould spores
- pollen grains
- insects

- mammalian dander (small scales from the skin or hair)
- secreta (for example, saliva) of insects, mites and pets
- excreta (for example, faeces) of insects, mites and pets.

House dust mites make up a large part of house dust allergens and are also abundant in mattresses, bed bases, pillows, carpets and upholstered furniture. Though house dust mites are present in the home all year round, their numbers usually peak during humid periods, when their growth is at its greatest (Kalra et al. 1992; Platts-Mills et al. 1987).

House dust mites have been associated with the sensitisation and persistence of allergic rhinitis (Matheson et al. 2005). Studies have shown that the higher number of mites in a household, the earlier sensitisation is likely to occur (Sporik et al. 1990).

There is some evidence that avoidance of dust mite allergen can help control allergic rhinitis. Some studies suggest that use of acaricides (substances that kill mites) and extensive bedroom-based environmental control programs may be of some benefit in reducing rhinitis symptoms (Sheikh et al. 2010). However, isolated use of bedding impermeable to house dust mite is unlikely to prove effective in controlling or reducing the symptoms of allergic rhinitis (Sheikh et al. 2010). Allergen avoidance is recommended as a strategy for dealing with allergic rhinitis, where possible and practical (Australian Society of Clinical Immunology and Allergy 2010a).

Pets

Household pets, particularly cats and dogs, are an important source of domestic allergens for allergic rhinitis sufferers. The major allergens produced by cats and dogs can be found in the animals' fur and in house dust, and can remain airborne for long periods (Luczynska et al. 1990). Exposure to these allergens has been linked to inducing and worsening symptoms of allergic rhinitis (Bollinger et al. 1996; Gordon 1997).

Research conclusions about the effect of early exposure to pets and the risk of allergic rhinitis are inconsistent.

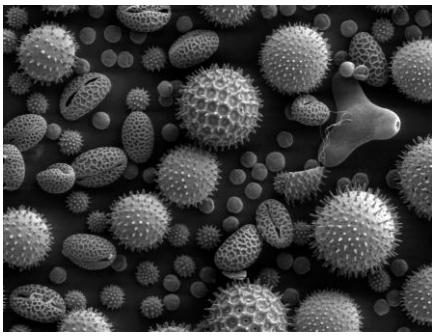
A 2007 study (Pohlabeln et al. 2007) confirms the findings of several earlier studies suggesting that children in a household with a dog developed fewer atopic diseases in early childhood than those without a dog. This effect was only observed in families without a history of atopic disorders. Other studies suggest that early exposure to dogs has no effect on the risk of allergic sensitisation or slightly increases it (Arshad et al. 2001; Remes et al. 2001).

A number of studies suggest that exposure to cats in early childhood may slightly decrease the risk of cat allergy (Pohlabeln et al. 2007).

Patients allergic to cats and dogs frequently display IgE reactivity (that is, hypersensitivity of membranes to environmental substances) against allergens from other animals such as rabbits, guinea pigs and hamsters (Boutin et al. 1988;).

Pollens

Pollens are small protein particles surrounded by an inner, cellulose-rich cell wall and a resistant outer wall. Pollens are produced by trees, grasses, flowers and other plants. The role of pollens is to fertilise the female flower to reproduce plant species. The nature and number of pollens in a particular environment varies with the vegetation, geography, temperature and climate.



Assorted pollens

Most people with allergic rhinitis are sensitised to many different pollen species (Pallasaho et al. 2006). The tiny, hardly visible pollens of wind-pollinated plants are the predominant triggers. Pollens of insect-pollinated plants (such as Australian wattles) are too heavy to remain airborne and pose little risk. The most troublesome pollens tend to be airborne pollens produced by Northern Hemisphere grasses, trees and flowering weed species (Australian Society of Clinical Immunology and Allergy 2010b).

The pollens to which sufferers are commonly allergic are:

- universally distributed grasses

  These generally pollinate at the end of spring and beginning of summer and are most frequently linked with allergic rhinitis sensitisation (Durham 2000). Improved pasture grasses are generally more allergenic than Australian native grasses (Australian Society of Clinical Immunology and Allergy 2010b).
- flowering plants

  A variety of flowering plants can cause allergies, including many species widely regarded as weeds. They vary widely in when and for how long they pollinate. Flowering plants to which allergic rhinitis sufferers are commonly sensitised include pellitory weed (also known as asthma weed) (*Parietaria judaica*), Patterson's curse (*Echium plantagineum*), ragweed (*Ambrosia*) and parthenium weed (*Parthenium hysterophorus*) (Australian Society of Clinical Immunology and Allergy 2008).
- trees

  Trees to which allergic rhinitis patients are commonly sensitised include the silver birch (*Betula pendula*), olive tree (*Olea europea*), English oak (*Quercus robur*) and Murray pine (or white cypress pine) (*Callitris glaucophylla*) (Australian Society of Clinical Immunology and Allergy 2008). Trees tend to pollinate at the end of winter and beginning of spring. This can vary with geography and from one year to another.

Fungal allergens

Fungi, such as moulds and yeasts, are organisms that lack chlorophyll (the green-coloured substance in plants, used in photosynthesis), leaves, true stems, and roots; reproduce by spores; and live on decaying matter or as parasites. They can release large amounts of allergenic spores into indoor and outdoor environments.

A wide variety of moulds is known to cause and provoke allergic rhinitis symptoms (Bousquet et al. 2008). They can be broadly classified into atmospheric (outdoor) moulds and domestic (indoor) moulds.

Outdoor moulds include *Alternaria*, *Cladosporium* and *Aspergillus.* They can be present in all conditions, with seasonal peaks in hot and humid conditions.

Indoor moulds are associated with dampness and are particularly abundant in bathrooms and kitchens.

The yeasts known to be most allergenic are *Candida albicans*, *Saccaromyces cerevistae* and *Sporobolmyces* (Bousquet et al. 2008). Yeasts can be found in foods and in the atmosphere.

2.5 Pollutants

Air pollution occurs when the air contains gases, dust or fumes in amounts that are considered harmful to the health or comfort of humans and animals or that could cause damage to plants and materials.

Air pollution arises from a range of sources both natural and caused by humans. Natural sources of pollutants include vegetation and dust storms, while industrial premises and road vehicles are examples of human-caused sources.

The pollutants directly emitted into the atmosphere, either from natural or human-caused sources, are known as *primary* pollutants. These may additionally undergo chemical reactions in the atmosphere, forming *secondary* pollutants (for example, ozone).

In recent years multiple studies have investigated the association between air pollutants, such as ozone, sulfur dioxide and tobacco smoke, and allergic rhinitis (Bousquet et al. 2008). Some of the studies have focused on distinguishing whether the pollutants are allergenic, and therefore specific to allergic rhinitis patients, or simply nasal irritants, inducing nasal symptoms in all individuals. Where it is known whether a particular pollutant is an allergen or a nasal irritant, it is discussed in descriptions of the individual pollutants below.

Results from one study examining the association between air pollution and daily consultations with general practitioners (GPs) for allergic rhinitis suggest that air pollution worsens allergic rhinitis symptoms. Sulfur dioxide (SO_2) and ozone (O_3) seemed particularly responsible and both appeared to contribute independently (Hajat et al. 2001). This study did not, however, address the issue of whether the pollutants affect allergic rhinitis patients through allergic mechanisms or not.

Some researchers have suggested that air pollution may worsen allergic rhinitis symptoms through its interaction with allergens. There have been studies that suggest that pollution from car traffic could interact with pollen grains or other allergen-bearing particles to increase their allergenicity (Ciccone et al. 1998; Weiland et al. 1994).

The French ISAAC (International Study on Allergies and Asthma in Childhood) did not find an association between long-term exposure to gaseous air pollutants and the prevalence of rhinitis (Ramadour et al. 2000).

In 2004, the World Health Organization published a report examining the relationship between air pollution and health. It concluded that, at present, there is insufficient evidence to determine whether increased air pollution exposure affects the prevalence of allergic diseases such as allergic rhinitis (WHO 2004).

Other studies have looked at the effect of individual pollutants. These are detailed below.

Nitrogen dioxide (NO_2)

Nitrogen dioxide is an odorous, brown, acidic gas. It is formed when nitric oxide (NO) is combined with oxygen in the atmosphere. Sources of nitric oxide include natural sources, industrial premises (in particular coal-fired power stations) and motor vehicles.

Short-term exposure to nitrogen dioxide has been shown to increase airway allergic inflammation and sensitivity to allergen exposure in subjects with mild allergic rhinitis (WHO 2004).

Carbon monoxide (CO)

Carbon monoxide is a colourless, odourless gas and is the most common pollutant by mass in the atmosphere. Carbon monoxide is formed when substances containing carbon are burned with an insufficient air supply. Major sources include industrial premises and motor vehicles.

Studies have shown that carbon monoxide can act as a nasal irritant (Shusterman D et al. 2003). Carbon monoxide is not allergenic and has no apparent involvement in allergic rhinitis beyond its role as a nasal irritant (Bousquet et al. 2008).

Ozone (O_3)

Ozone is a colourless, strongly oxidising gas found in both the upper and lower layers of the atmosphere. Ozone is a secondary pollutant formed in sunlight through reactions between oxides of nitrogen (NOx) and volatile organic compounds (VOCs).

Combustion processes (including motor vehicle engines, power stations, and bushfires) are major sources of nitrogen oxides and VOCs.

Long-term exposure to ozone was not found to increase the risk of developing allergic rhinitis in children (Zwick et al. 1991). Some studies have noted that ozone can act as a respiratory irritant for some people, exacerbating existing allergic rhinitis (Santamaria 2007).

Sulfur dioxide (SO_2)

Sulfur dioxide is a colourless, odorous gas. The major sources of sulfur dioxide include natural sources (for example, volcanic activity), burning of fossil fuels and smelting of mineral ores that contain sulfur.

High concentrations of sulfur dioxide have been associated with an increased risk of developing upper respiratory symptoms (von Mutius et al. 1995), and with increased symptoms in those with already developed allergic rhinitis (Arnedo-Pena et al. 2009; Hajat et al. 2001).

Particulate matter

Particulate matter consists of particles suspended in the air with a diameter in a specified size range. Particulate matter is a complex mixture of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil and dust particles (United States Environmental Protection Agency 2011).

The diameter of particulate matter is measured in micrometres, a micrometre being one millionth of a metre, or one thousandth of a millimetre. For comparison purposes, an average human hair is about 70 micrometres in diameter.

Particulate matter is classified as coarse (PM_{10}) when its diameter is larger than 2.5 micrometres and smaller than 10 micrometres (for example, construction debris and road dust); fine ($PM_{2.5}$) when its diameter is between 2.5 micrometres and 0.1 micrometres (for example, wood and tobacco smoke); or ultrafine (UFP) when its diameter is less than 0.1 micrometres (for example, products of fossil fuel combustion) (Bousquet et al. 2008).

The main sources of particulate matter include:

- combustion processes using coal or fossil fuels, such as power generation, industrial operations and motor vehicle fuels
- agricultural burning practices
- emissions from domestic solid fuel heaters and woodstoves.

Some studies found that increased exposure to coarse particulate matter (PM_{10}) increases upper respiratory symptoms, although this increase was not specific to allergic rhinitis.

2.6 Occupational exposures

Occupational agents can cause and exacerbate allergic rhinitis by both allergic and non-allergic mechanisms (Bousquet et al. 2008; Castano & Theriault 2006; Radon et al. 2008).

These irritants may be in different forms (for example, fumes, dust, vapours and gases) and of different types (for example, chlorine, ammonia, glutaraldehyde and wood dust) (Castano & Theriault 2006).

Common allergens in occupational environments

Allergens in the workplace can cause sensitisation and the development of allergic rhinitis in people who did not previously have the condition.

Natural rubber latex

Latex is a water-soluble protein obtained from the *Hevea brasiliensis* rubber tree. It is used to make many products including mattresses, gloves and toys. Latex sensitivity refers to an immunological response to either the protein in the latex or the chemicals used in the production of latex products.

Occupations vulnerable to latex sensitivity include toy manufacturers, medical personnel and people in the textile industry (AIHW 2008,AIHW 2010; Bousquet et al. 2006; Jaeger et al. 1992).

Bakery allergens

Cereals are an important but not the only potential source of allergens for bakers. The list of potential allergens includes those coming from mites that occur in stored cereal, and natural and supplementary enzymes that are used in the baking industry.

Like all other living material, the cells in cereal grains contain enzymes. These enzymes are important in baking but are not always found in optimum quantities naturally. Supplementary enzymes are often added to enhance baking. These supplementary enzymes usually come in the form of flour improver mix.

A study in 2003 found that nasal symptoms were significantly more prevalent in an enzyme-sensitised group working in the baking industry compared with those sensitised to wheat flour alone. The authors suggested that sensitisation may be a consequence of repeated exposure to enzymes. They also suggested that the early airway responses in the form of allergic rhinitis may identify an at-risk group for occupational asthma (Elms et al. 2003).

Small mammals

It is estimated that between 10% and 30% of laboratory animal workers may develop occupational allergy to animal allergens (Aoyama et al. 1992). Allergy to rats and mice is the most common occupational problem, primarily because these animals are the most widely used in medical research (Bush et al. 1998).

Plants and flowers

Reports on occupational allergy to plants and flowers usually concern gardeners, greenhouse workers, florists and those involved in the manual preparation of plant extracts (Giavina-Bianchi et al. 1997; Swierczyniska-Machura et al. 2006).

Common chemical triggers in occupational environments

Triggers are irritants that cause adverse reactions in the airways, particularly localised inflammation, which can bring on or exacerbate the symptoms of allergic rhinitis.

More than 250 different chemicals have been identified as capable of inducing rhinitis. They typically do this by irritating the airways, producing localised inflammation, and triggering or aggravating symptoms in allergic rhinitis sufferers.

Chlorine

Chlorine is an oxidant (see Glossary) that is used in bleaching, disinfectants, water purification and swimming pool treatments. It is also a reagent used in the chemical industry.

Acute and chronic exposure to chlorine has been linked to aggravating nasal symptoms in allergic rhinitis patients (Leroyer et al. 1999; Shusterman DJ et al. 1998). According to recent studies, exposure to chlorine through recreational swimming does not increase the risk of allergic symptoms (Font-Ribera et al. 2009,Font-Ribera et al. 2011).

Formaldehyde

Formaldehyde is a VOC widely used in industry in the manufacture of resins, plastics, adhesives, paints and explosives; as a finisher for fabrics; and as a sterilising agent in medicine. At high concentrations it can act as a nasal irritant in people with allergic rhinitis (Dykewicz et al. 1991).

Isocyanates

Isocyanates are a family of highly reactive molecular chemical compounds. They are used in the manufacture of foams, fibres, coatings such as paints and varnishes, and elastomers, and are used in the motor vehicle industry, auto body repairs and building insulation materials.

One study examining the effect of exposure to isocyanates found that exposed workers reported significantly more rhinitis symptoms than did those not exposed (Sari-Minodier et al. 1999).

Acid anhydrides

Acid anhydrides are reactive organic chemicals, widely used in adhesives, coatings, fabric treatments, sealants, and dental and surgical appliances.

Organic acid anhydrides have been found to be both allergens and irritants to the airways (Nielsen et al. 1988).

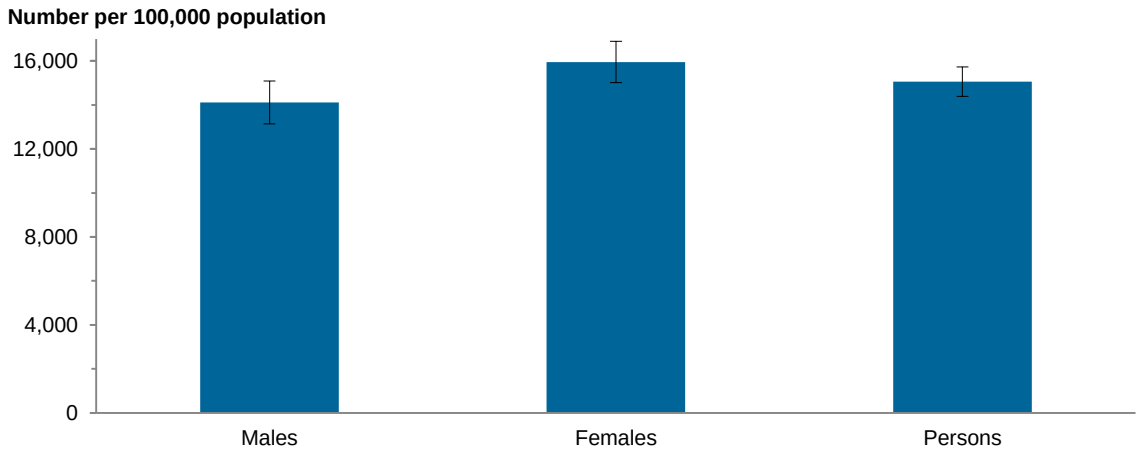
3 Who has allergic rhinitis?

Based on self-reports in the National Health Survey (NHS), about 3.1 million Australians (15.1% of the population) had allergic rhinitis as a long-term condition in 2007–08. This represents a slight drop from 2004–05, when 3.2 million people (16.1% of the population) reported allergic rhinitis as a long-term condition.

3.1 Age and sex

In 2007–08, an estimated 1.7 million females and 1.5 million males in Australia suffered from allergic rhinitis as a long term condition (ABS 2009).

Allergic rhinitis was slightly more common among females (16,000 per 100,000 population) than males (14,100 per 100,000 population) in the 2007–08 NHS. Females were 1.1 times as likely to report that they suffer from allergic rhinitis as males ($p<0.0001$).



Notes

1. Directly age standardised to the 2001 Australian population.
2. The thin vertical bars attached to the top of each column are 95% confidence intervals. We can be 95% confident that the true value is within the interval depicted.

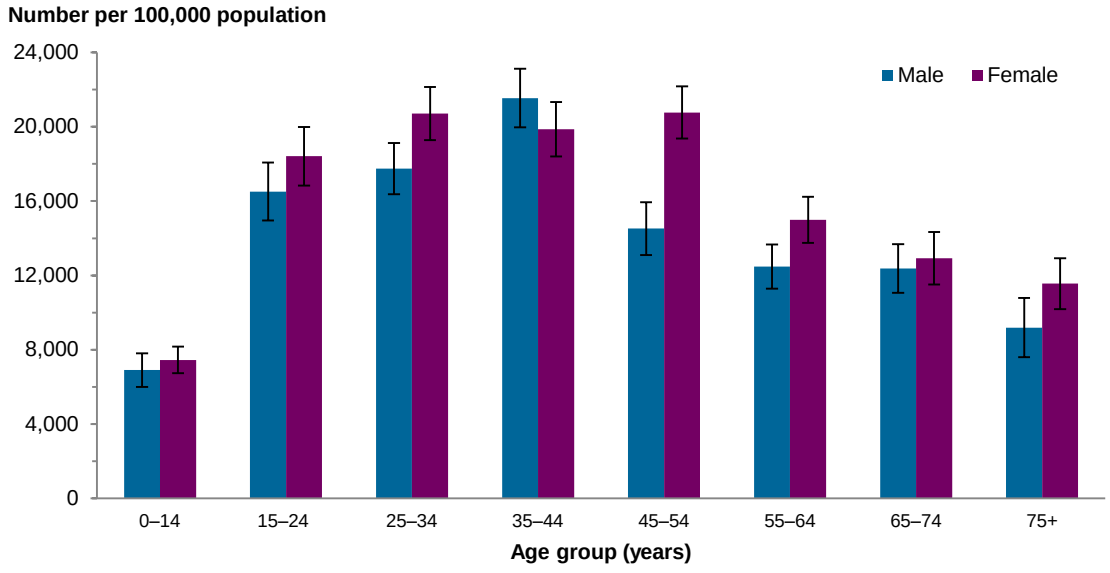
Source: ABS National Health Survey, 2007–08.

Figure 3.1: Australians with allergic rhinitis per 100,000 population, by sex, 2007–08

Allergic rhinitis was most common among those aged 35–44 (20,700 per 100,000 population), followed closely by those aged 25–34 (19,200 per 100,000 population).

Allergic rhinitis was least common among the 0–14 year age group (7,200 per 100,000 population) and those aged 75 years or older (10,500 per 100,000 population).

Allergic rhinitis was more common among females in all age groups except those aged 35–44 years.



Notes

1. Directly age standardised to the 2001 Australian population.
2. The thin vertical bars attached to the top of each column are 95% confidence intervals. We can be 95% confident that the true value is within the interval depicted.

Source: ABS National Health Survey, 2007–08.

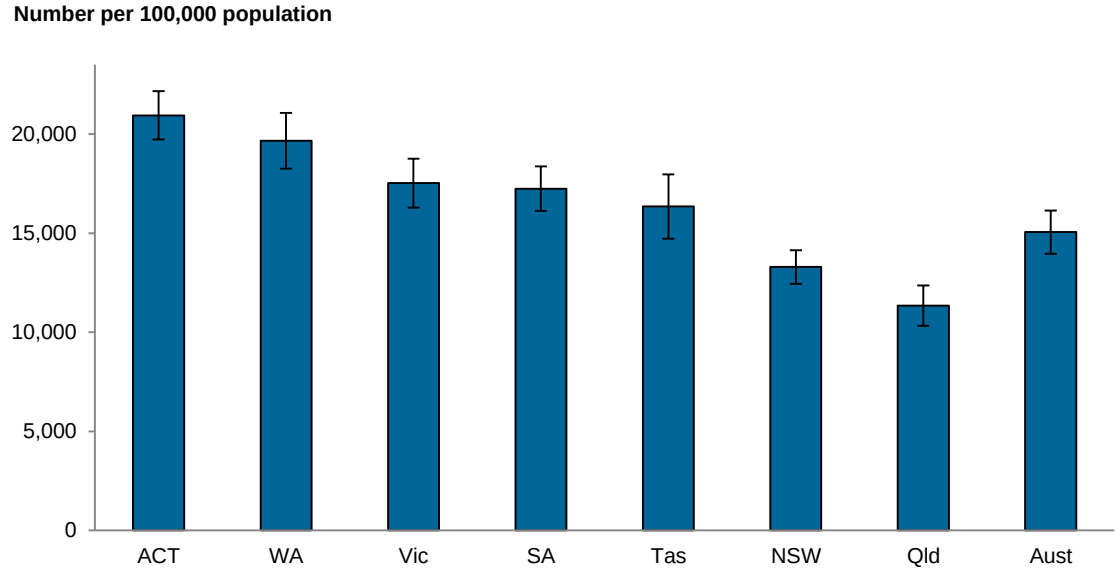
Figure 3.2: Australians with allergic rhinitis per 100,000 population, by age and sex, 2007–08

3.2 Geography

According to the 2007–08 NHS, allergic rhinitis rates were highest in the Australian Capital Territory, where 21,000 per 100,000 population reported suffering from long-term allergic rhinitis, and Western Australia, where the rate was 19,700 per 100,000 population.

The rates reported in Victoria (17,500 per 100,000 population) and South Australia (17,200 per 100,000 population) were also significantly higher than the rate for all of Australia (15,100 per 100,000 population).

The lowest rates occurred in Queensland (11,300 per 100,000 population) and New South Wales (13,300 per 100,000 population).



Notes

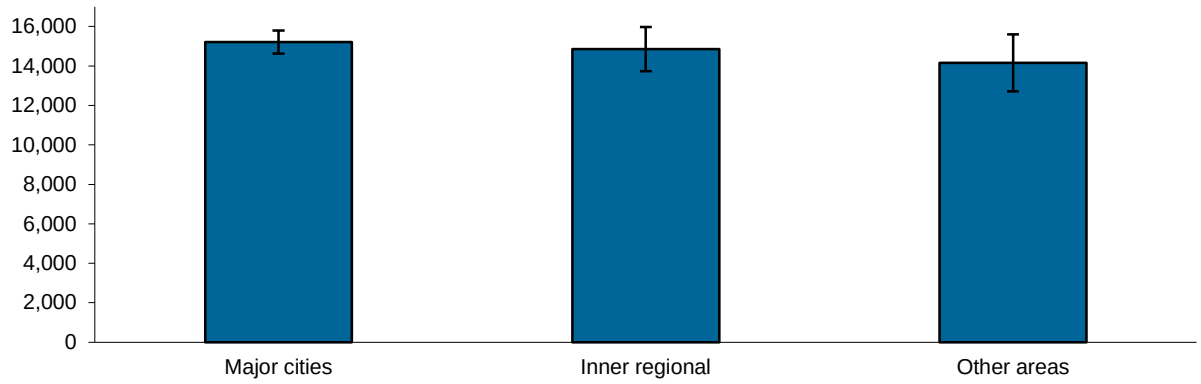
1. Directly age standardised to the Australian population as at June 2001.
2. Northern Territory was not included because the NHS sample size is too small for a reliable estimate.
3. The thin vertical bars attached to the top of each column are 95% confidence intervals. We can be 95% confident that the true value is within the interval depicted.

Source: ABS National Health Survey, 2007–08.

Figure 3.3: Australians with allergic rhinitis per 100,000 population, by state or territory, 2007–08

Reasons for these differences between states are not clear, but they may reflect regional differences in allergen exposure.

Despite the differences between the states, there was no significant difference in rates of allergic rhinitis between the *Major cities*, *Inner regional* and *Other* areas (Box 3.1).



Notes

1. Directly age standardised to the 2001 Australian population.
2. The thin vertical bars attached to the top of each column are 95% confidence intervals. We can be 95% confident that the true value is within the interval depicted.

Source: ABS National Health Survey, 2007–08.

Figure 3.4: Australians with allergic rhinitis per 100,000 population, by region, 2007–08

Box 3.1: Classifying remoteness of location

The Australian Standard Geographical Classification (ASGC) of Remoteness Areas identifies various regional areas (ABS 2011). The classification allocates one of five remoteness categories to collection districts depending on their distance from major service centres. Areas are classified as *Major cities*, *Inner regional*, *Outer regional*, *Remote* and *Very remote*.

Major cities includes most capital cities, as well as major urban areas such as Newcastle, Geelong and the Gold Coast. *Inner regional* includes towns and cities such as Hobart, Launceston, Mackay and Tamworth. *Outer regional* includes towns and cities such as Darwin, Whyalla, Cairns and Gunnedah. Examples of *Remote* include Alice Springs, Mount Isa and Esperance. *Very remote* represents much of central and western Australia and includes towns such as Tennant Creek, Longreach and Coober Pedy.

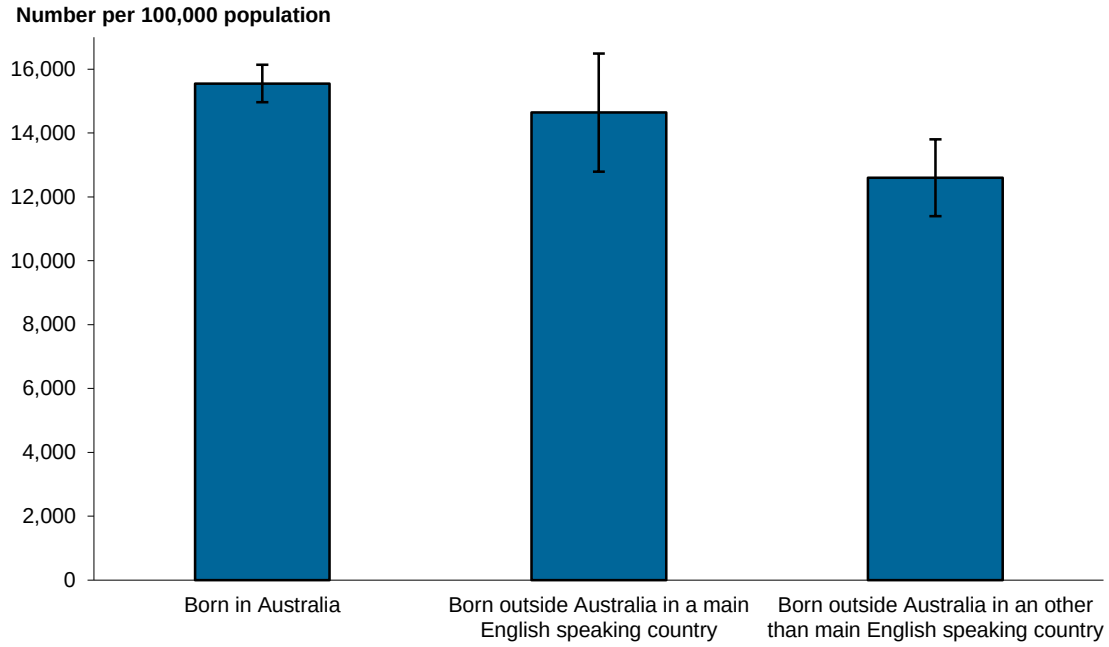
The NHS does not gather data from *Very remote* areas. The *Other* areas category used here only covers *Outer regional* and *Remote* areas.

In 2007–08, two-thirds of Australians (66%) lived in *Major cities*, with a smaller proportion in *Inner regional* areas (22%) and *Other* areas (12%; including *Outer regional*, *Remote* and *Very remote* areas).

3.3 Country of birth

The 2007–08 NHS suggests that people born in Australia have higher rates of allergic rhinitis than Australians born in a country other than a main English-speaking country. Of the population born in Australia, 15,600 per 100,000 reported allergic rhinitis as a long-term condition. The rate for those born outside Australia in a country other than a main English-speaking country (that is, countries other than New Zealand, Canada, United Kingdom, Ireland, United States of America or South Africa) was 12,600 per 100,000.

The rate of allergic rhinitis for those born in Australia was not very different from those born outside Australia in the main English-speaking countries.



Notes

1. Directly age standardised to the 2001 Australian population.
2. The thin vertical bars attached to the top of each column are 95% confidence intervals. We can be 95% confident that the true value is within the interval depicted.
3. Main English-speaking countries comprise the United Kingdom (England, Scotland, Wales, Northern Ireland), Republic of Ireland, New Zealand, Canada, United States of America and South Africa. The list of main English-speaking countries is not an attempt to classify countries on the basis of whether or not English is the predominant or official language of each country. It is a list of the main countries from which Australia receives, or has received, significant numbers of overseas settlers who are likely to speak English.

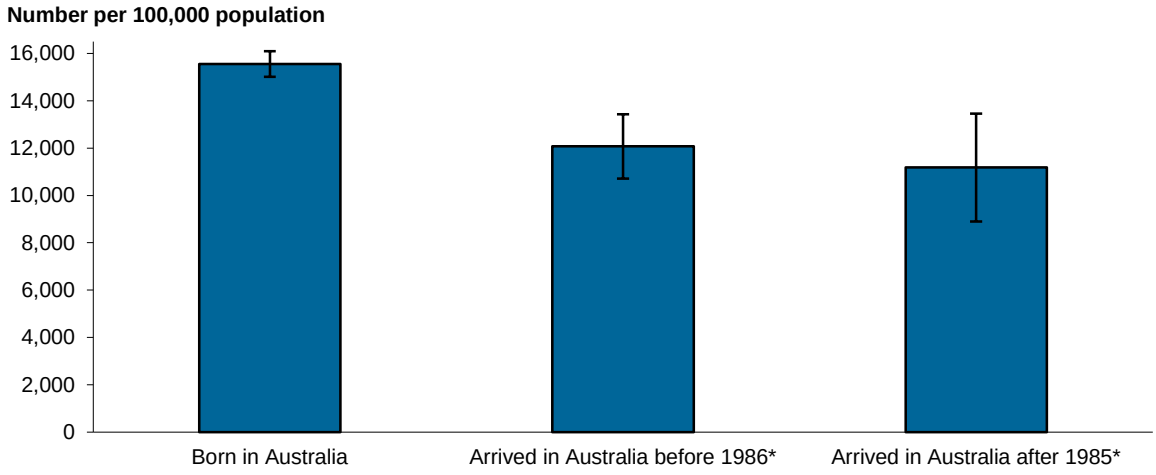
Source: ABS National Health Survey, 2007–08.

Figure 3.5: Persons with allergic rhinitis per 100,000 population, by country of birth, 2007–08

The prevalence of allergic rhinitis does not appear to be linked to length of residence in Australia. People born in Australia had higher rates of allergic rhinitis than those born overseas, regardless of year of arrival.

The rate of persons born in Australia reporting allergic rhinitis as a long-term condition was 15,600 per 100,000 population (CI 15,057–16,143).

For persons born overseas who arrived in Australia before 1986, the rate was 12,100 per 100,000 population (CI 10,743–13,457), and for those who arrived in 1986 or later it was 11,200 per 100,000 (CI 8,969–13,431).



* Includes both arrivals who were born in main English-speaking countries and arrivals who were not born in main English-speaking countries.

Notes

1. Directly age standardised to the Australian population
2. The thin vertical bars attached to the top of each column are 95% confidence intervals. We can be 95% confident that the true value is within the interval depicted.

Source: ABS National Health Survey, 2007–08.

Figure 3.6: Australians with allergic rhinitis per 100,000 population, by year of arrival, 2007–08

The lower rates of allergic rhinitis among migrants might reflect the prevalence rates in their country of origin or it might be explained by the healthy migrant effect, where, on average, immigrant populations are healthier than the native-born population of their host country.

There are several explanations for the immigrant population being healthier (Kennedy et al. 2006):

- healthier people tend to migrate
- health screening by recipient countries

healthy behaviour before migration. Over time, immigrants steadily adopt the health behaviours of the new country, leading to health outcomes that approach those of the native-born population. This is sometimes evident in health data but is not evident in this analysis.

4 How is allergic rhinitis managed?

Allergic rhinitis is managed in a number of ways in the community. Many sufferers self-manage their allergic rhinitis (perhaps in consultation with their pharmacy, as there are a range of treatments available over-the-counter (see Glossary) for allergic rhinitis).

Allergic rhinitis is a relatively common problem treated by GPs, although it is not one of the 30 most frequently managed. A 2002 survey of allergy sufferers suggested that nearly two-thirds of respondents did not consult their doctor about their current allergic rhinitis treatment (Walls et al. 2005).

Allergic rhinitis is seldom a cause of hospitalisation. Nevertheless, there are some hospitalisations recorded where allergic rhinitis is the principal diagnosis.

The goal of allergic rhinitis management is to achieve optimal symptom control. Therapeutic options include allergen avoidance, pharmacotherapy (such as intranasal corticosteroids or antihistamines), non-medicated treatments (such as saline douches or sprays) and immunotherapy (Australian Society of Clinical Immunology and Allergy 2010a).

Data indicate that allergic rhinitis sufferers who have a health professional, such as a pharmacist, guiding their management have better outcomes than patients who set their own goals for treating the disease.

4.1 Use of medications for management of allergic rhinitis

With a wide range of medications for management of allergic rhinitis now available from pharmacies without the need for doctors' prescription, many Australians self-medicate for this condition (Walls et al. 2005).

In mid-1999, the regulatory control of medicines used to treat allergic rhinitis shifted from that requiring a prescription to that requiring pharmacist advice only. This change began with intranasal corticosteroids. In 2001, the first non-sedating oral antihistamine also made this shift.

This section examines the supply patterns of medications for management of allergic rhinitis in the last 10 years.

Three focus areas of investigation were:

- changes in the availability of types of medication and the amount supplied
- change in the cost of these medications
- seasonal variation in the supplies of medications for allergic rhinitis.

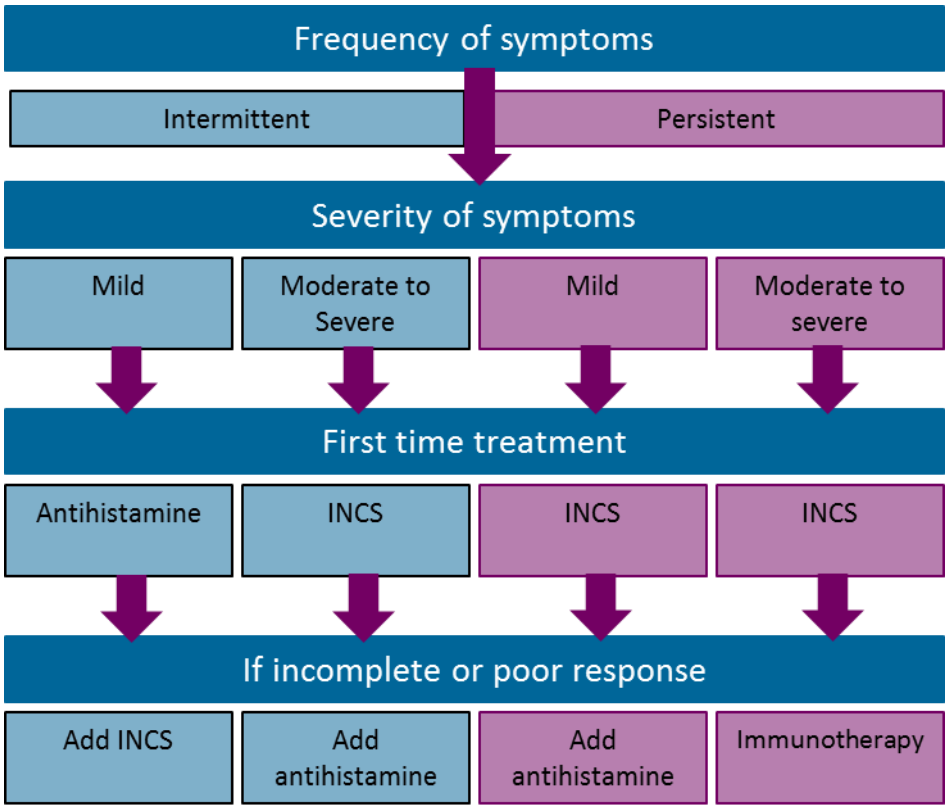
Use of medications in the treatment guidelines for allergic rhinitis

Intranasal corticosteroids (see Glossary) and oral antihistamines (see Glossary) are the major categories of medications used to treat allergic rhinitis. The recommended usage of these are outlined in the guidelines for allergic rhinitis (Figure 4.1) (Walls et al. 2005).

The treatment guidelines for allergic rhinitis differ depending on the subtypes of the condition.

For persistent allergic rhinitis and for moderate/severe intermittent allergic rhinitis, the guidelines recommend the use of intranasal corticosteroids (corticosteroids administered through the nose) as the first-line therapy, as these are more cost-effective than oral antihistamines for these conditions (Dykewicz et al. 1998). For patients with these types of allergic rhinitis whose symptoms are not adequately controlled by intranasal corticosteroids, additional use of oral antihistamines is recommended.

For mild intermittent allergic rhinitis, the use of antihistamines as the first-line therapy is recommended. For those whose symptoms are not adequately controlled by antihistamines, additional use of intranasal corticosteroids is recommended.



Note: INCS refers to intranasal corticosteroids.

Source: Adapted from Walls et al. 2005.

Figure 4.1: Guidelines for allergic rhinitis management

Source of medication data

IMS Health, a commercial market research company, collects pharmaceutical wholesale supply data. IMS Health data were used to explore the supply patterns of intranasal corticosteroids and oral antihistamines, as the data include prescription as well as non-prescription medicines.

IMS Health's wholesale data can be analysed separately for supplies to hospitals and community pharmacies. As allergic rhinitis is largely managed through self-care or in primary health-care services, the analysis here focuses on the medication supplies to community pharmacies.

It should be noted that intranasal corticosteroids and oral antihistamines are used in the treatment of conditions other than allergic rhinitis. There is no way to determine from these data the proportion of the medications supplied that were specifically used in the treatment of allergic rhinitis.

It should also be noted that these supply figures do not necessarily translate into usage. Because the IMS Health data only record the wholesale supply of drugs to community pharmacies, it is not known how much of the medications supplied were actually sold, nor how much of the medications sold were actually consumed.

See Appendix A2 for more detailed information about IMS Health data and the data extraction method used.

Costs of allergic rhinitis medication

The total wholesale cost to community pharmacies of wholesale intranasal corticosteroids and oral antihistamines (the main medications used in the treatment of allergic rhinitis) approximately doubled in the last 10 years. In 2001 the total wholesale cost of these medications was $107.8 million and in 2010 it was $226.8 million.

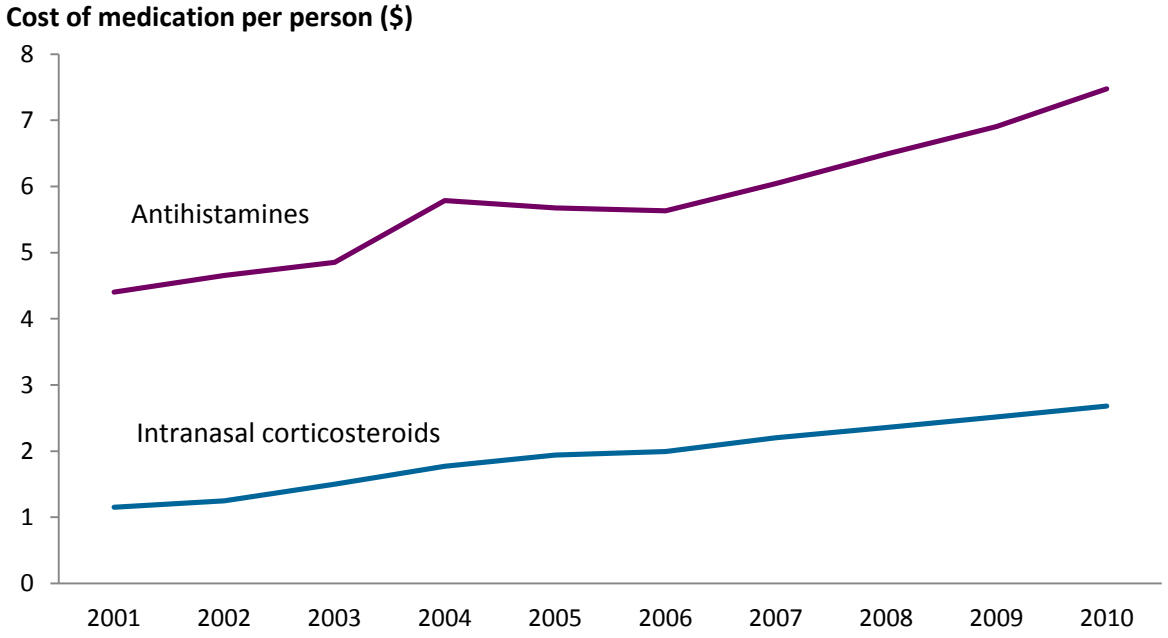
The total cost of oral antihistamines supplied by wholesalers to community pharmacies almost doubled, from $85.5 million in 2001 to $167.0 million in 2010.

The total wholesale cost of intranasal corticosteroids supplied by wholesalers to community pharmacies more than doubled, from $22.3 million in 2001 to $59.8 million in 2010.

The wholesale cost is summarised in terms of a cost per Australian in Figure 4.2. The wholesale cost per Australian represents the price paid by the pharmacy to the wholesaler, calculated on a per person basis for the year. The price paid by the customer to the pharmacy is likely to be significantly higher.

From 2001 to 2010 the wholesale cost of:

- oral antihistamines increased from $4.40 to $7.48 (1.7 times) per person
- intranasal corticosteroids increased from $1.15 to $2.68 (2.3 times) per person.



Note: Australian population as at December of each year was used to calculate the cost of medication per person.

Source: IMS Health.

Figure 4.2: Increase in wholesale cost of allergic rhinitis medications supplied to pharmacies by wholesalers, 2001–10

Medications used to manage allergic rhinitis, 2001–10

Table 4.1 summarises the intranasal corticosteroids and oral antihistamines supplied by wholesalers to community pharmacies in Australia from 2001 to 2010. Six intranasal corticosteroids and 13 oral antihistamine medications were available at some time during 2001 to 2010.

'Defined daily dose' (DDD) refers to the 'assumed average maintenance dose per day for a drug used for its main indication in adults' (WHOCC 2009), determined for each therapeutic substance by the World Health Organization Collaborating Centre for Drug Statistics Methodology (WHOCC). See Appendix A3 for more information about the DDD, the widely used unit of measuring drug utilisation.

For each medication, the relevant DDD was obtained from the website of the WHOCC (WHOCC 2010). The WHOCC-determined DDD for each medication is listed in Table 4.1.

Table 4.1: Allergic rhinitis medications supplied by wholesalers to community pharmacies, 2001–10

Intranasal corticosteroids		
	Medications included	**DDD (mcg)[a]**
	Beclometasone	400
	Budesonide[b]	200
	Fluticasone	200
	Fluticasone furoate	110
	Mometasone	200
	Triamcinolone acetonide	220
Oral antihistamines		
	Medications included	**DDD (mg)[c]**
	Alimemazine	30
	Azatadine	2
	Cetirizine	10
	Cyproheptadine	12
	Desloratadine	5
	Dexchlorpheniramine	6
	Diphenhydramine	150
	Fexofenadine	120
	Levocetirizine	5
	Loratadine	10
	Methdilazine	16
	Pheniramine	75
	Promethazine	25

(a) 'mcg' stands for microgram. A microgram is a unit of weight—one-millionth of a gram.

(b) The DDD for budesonide was 300 mcg until 2003, and was reduced to 200 mcg from 2004.

(c) 'mg' stands for milligram. A milligram is a unit of weight—one-thousandth of a gram.

Source: WHOCC 2010.

Supply patterns of allergic rhinitis medications by wholesalers to community pharmacies, 2001–2010

Number of available medications and products

In this section, *medication* refers to a specific chemical compound used to treat a medical condition. The same *medication* may be available as several different *products* (that is, sold under different proprietary names from different manufacturers).

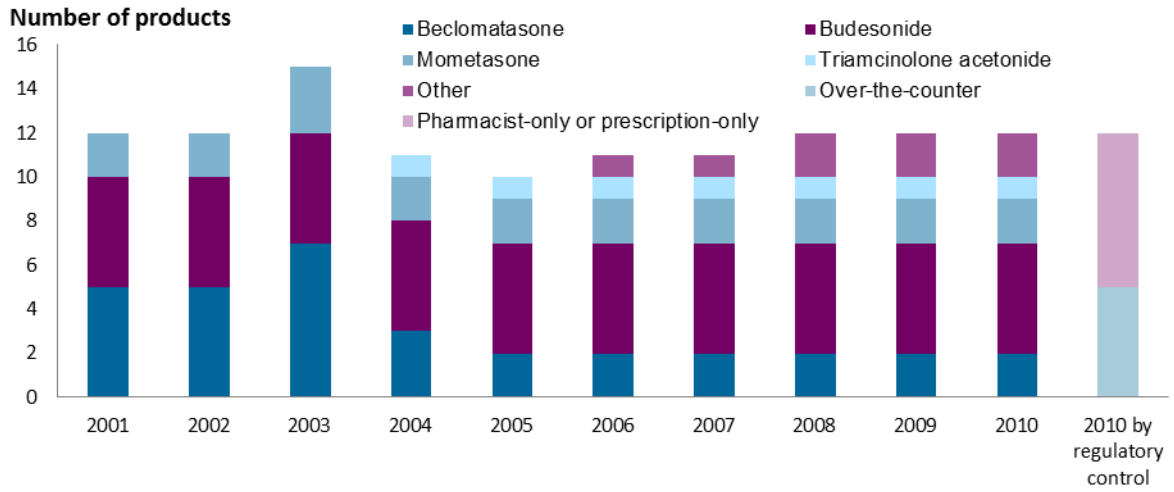
The number of intranasal corticosteroid *medications* available in Australia increased from 3 to 5 in 10 years, while the number of *products* (proprietary names) available from pharmacies fluctuated between 10 and 15 during this time (Figure 4.3). At the time of writing, no generic *products* for intranasal corticosteroids were available in Australia.

Of the 12 intranasal corticosteroid *products* available in 2010, five were over-the-counter *medications* and seven were either pharmacist-only or prescription-only *medications*.

While the number of oral antihistamine *medication*s remained steady, ranging from 9 to 11, the number of *products* almost tripled from 50 in 2001 to 147 in 2010 (the highest number of *products* available was 151 in 2009) (Figure 4.4).

The introduction of generic *products* for fexofenadine in 2005, loratadine in 2006 and cetirizine in 2007 largely accounts for this increase in the number of oral antihistamine *products* in the years from 2005 to 2007. The number of oral antihistamine *products* steadily increased thereafter to 2010.

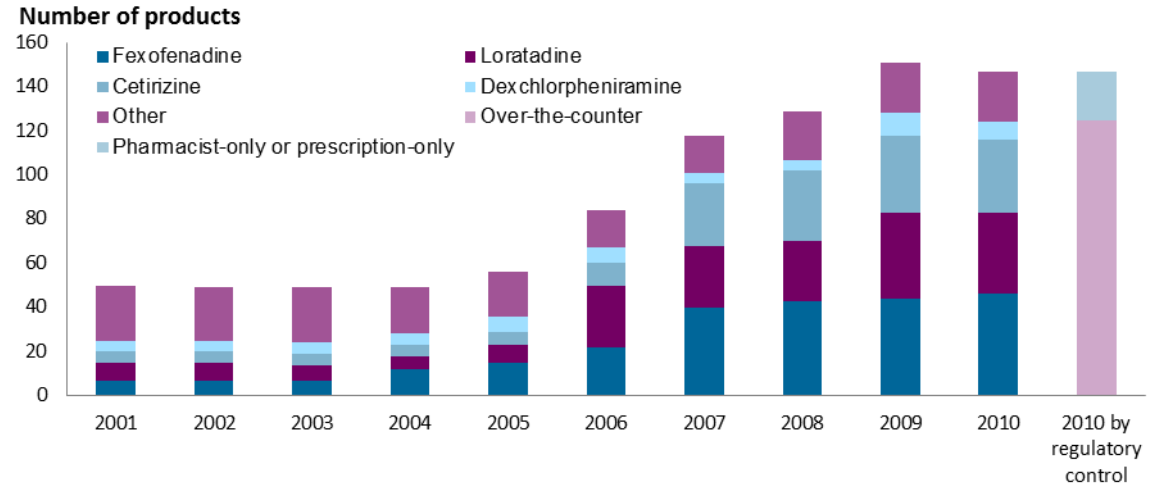
The majority of oral antihistamine *products* available in Australia in 2010 (125 out of 147) had over-the-counter status, making 9 out of 10 oral antihistamine *products* obtainable without consulting a pharmacist or medical practitioner.



Note: 'Other' intranasal corticosteroid medications include fluticasone and fluticasone furoate.

Source: IMS Health.

Figure 4.3: Number of available intranasal corticosteroid products supplied to community pharmacies by wholesalers, 2001–10, and type of regulatory control, 2010



Note: 'Other' intranasal corticosteroid medications include alimemazine, azatadine, cuproheptadine, desloratadine, diphenhydramine, levocetirizine, methdilazine, pheniramine and promethazine.

Source: IMS Health.

Figure 4.4: Number of antihistamine products supplied to community pharmacies by wholesalers, 2001–10, and type of regulatory control, 2010

Supply quantity of intranasal corticosteroids and oral antihistamines

In this section, medication supply for allergic rhinitis was measured in terms of DDD per 1,000 persons per day (DDD/1,000 persons/day) to enable comparison of supplies across time.

For each intranasal corticosteroid and oral antihistamine product, the DDD/1,000 persons/day was calculated by applying the following formula (AIHW: Australian Centre for Asthma Monitoring 2008):

$$DDD/1{,}000\ persons\ /\ day = \frac{N \times M \times Q \times 1{,}000}{DDD \times P \times D}$$

where:

N	=	total number of packs supplied to community pharmacies per year
M	=	mass of each dosage unit (for example, mg per spray)
Q	=	total number of dosage units supplied per pack
P	=	mid-year Australian population
D	=	number of days in the year for yearly analysis or number of days in the month for monthly analysis.

The supply amount for individual products in DDD/1,000 persons/day units obtained was aggregated separately for intranasal corticosteroids and oral antihistamines for each year. These are presented in Figure 4.4.

The amount of oral antihistamines supplied exceeded that of intranasal corticosteroids every year from 2001 to 2010. In 2010, about 3 times as many oral antihistamines (26.48 DDD/1,000 persons/day) were supplied than intranasal corticosteroids (7.8 DDD/1,000 persons/day).

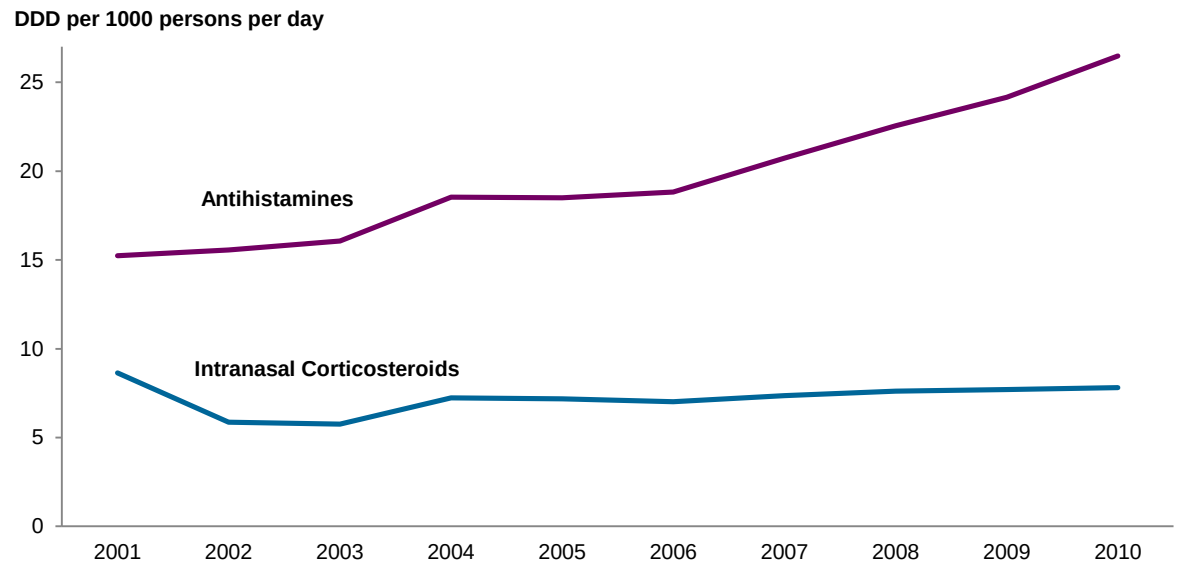
Since 2001 the supply of antihistamines increased steadily over 10 years, growing by about 75% from 15.24 to 26.48 DDD/1,000 persons/day (Figure 4.4).

The supply of intranasal corticosteroids, on the other hand, remained more constant over the same time period. Between 2004 and 2010, the amount supplied was steady at around 7.5 DDD/1,000 persons/day.

The supply pattern of intranasal corticosteroids from 2001 to 2003 was less straightforward. The supply of intranasal corticosteroids in 2001 was higher than any other year in the 10-year period examined. In 2001, there were 8.63 DDD of intranasal corticosteroids supplied per 1,000 persons per day. Almost 60% of this amount (5.16 DDD/1,000 persons/day) were supplied in the form of two products, both high-dose budesonide (100 mcg per dose) products containing 200 sprays.

These products were available only up to December 2001. Thus, the relatively higher supply of intranasal corticosteroids in 2001 might be attributable to the availability of these two products, each pack of which contained just over 66 DDD. Some patients might not have used up one pack of these intranasal corticosteroids in one season.

For the years 2002 onwards, intranasal corticosteroid products that contained 25 to 38 DDD became the most widely sold.



Source: IMS Health.

Figure 4.5: Allergic rhinitis medication supplied to pharmacies by wholesalers, by defined daily dose (DDD) per 1,000 persons per day, 2001–10

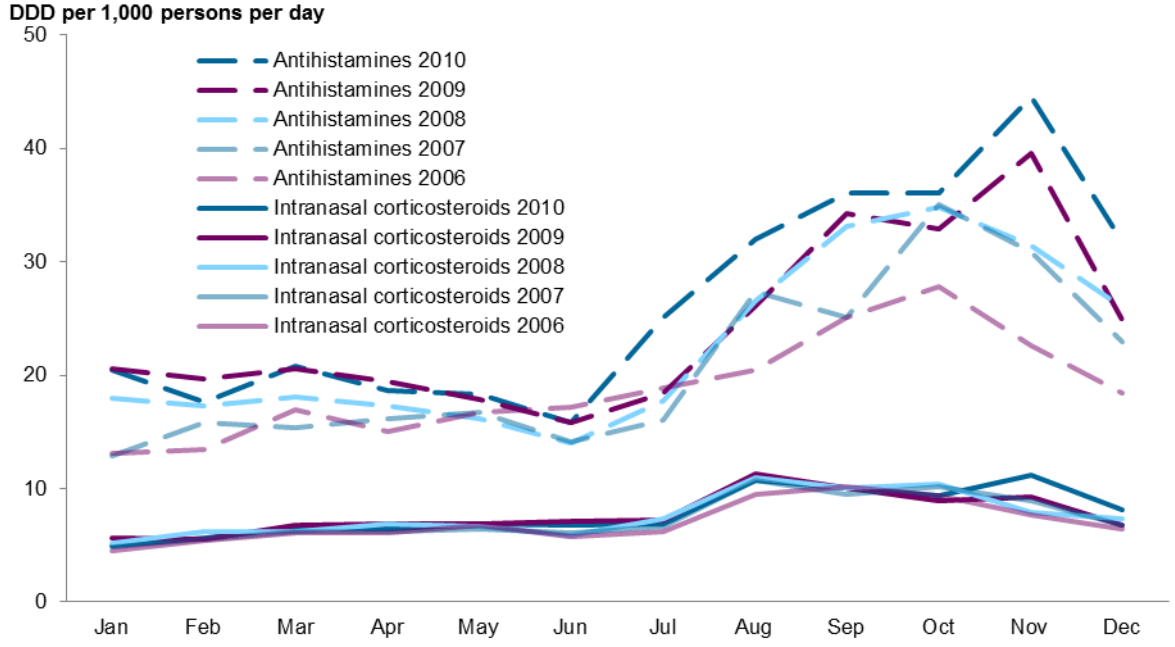
Seasonality in pharmacy supply pattern

The wholesale supply data from 2006 to 2010, summarised for each month, are presented in Figure 4.7.

Each year, the wholesale supplies of oral antihistamines started to increase around July and peaked around October–November.

The seasonality in wholesale supplies was not as marked for intranasal corticosteroids; however, the overall pattern of supply peak is similar to that of antihistamines. The monthly supplies of intranasal corticosteroids started to increase in August and peaked around November.

It is possible that the greater seasonality observed in the supply of oral antihistamines is related to the treatment guidelines. Oral antihistamines are recommended as the first-line therapy for mild intermittent allergic rhinitis. As mild intermittent allergic rhinitis is more likely to be seasonal than either persistent allergic rhinitis or moderate/severe intermittent allergic rhinitis, a peak in demand for oral antihistamines could be expected during the pollen season, with a corresponding peak in supply in the period leading up to this.



Source: IMS Health.

Figure 4.6: Allergic rhinitis medications supplied to pharmacies by wholesalers by month, 2008–10

4.2 Management of allergic rhinitis in general practice

In the management of allergic rhinitis, GPs provide diagnosis, prescription of medications (where required), advice on over-the-counter medications and patient education (Britt et al. 2009).

According to the Bettering the Evaluation and Care of Health (BEACH) survey of general practice activity, in 2007–08 allergic rhinitis was managed at a rate of 0.6 per 100 GP–patient encounters. The 2007–08 data have been used as it was the last period for which the most detailed data on allergic rhinitis were available in the BEACH annual report.

Allergic rhinitis made up 0.4% of all problems managed in general practice in 2007–08. This made it the 35th most frequently managed problem. In comparison, the most frequently managed problem in 2007–08 was hypertension, which made up 6.5% of total problems managed and was managed at a rate of 9.9 per 100 GP–patient encounters. The 30 most frequently managed problems accounted for just over 50% of all problems managed.

Patient education

Patient or carer education relating to the management of *rhinitis* is known to be very important. This education optimises treatment outcomes and maximises adherence. It should be noted, however, that these benefits have not been tested specifically in relation to *allergic rhinitis*, when looking at effectiveness, adherence and treatment efficacy (Bousquet et al. 2008).

A study conducted by Sheikh and colleagues (2007) found that a modest improvement in the disease-specific quality of life of patients with *perennial rhinitis* resulted from standardised allergy education given to primary health-care professionals.

Between 2002 and 2007, advice/education and counselling were not frequently given in general practice in Australia for allergic rhinitis, occurring at a rate of 5.5 per 100 allergic rhinitis problems managed (Fahridin & Britt 2008).

Comorbidities

Patients often have more than one problem managed during an encounter with a GP. Conditions that occur together are known as comorbidities. In 2006–07, comorbid conditions that were managed more frequently in encounters where allergic rhinitis was managed compared to all encounters were:

- asthma (6.0 per 100 encounters, compared with 2.3 per 100 for all encounters)
- sinusitis (2.7 per 100 encounters, compared with 1.4 per 100 for all encounters)
- conjunctivitis (1.0 per 100 encounters, compared with 0.7 per 100 for all encounters) (Fahridin & Britt 2008).

Referrals

In the period 2002–07, referrals from GPs for allergic rhinitis were most often to allergists (2.2 per 100 allergic rhinitis problems managed) and ear, nose and throat (ENT) surgeons (1.7 per 100 problems) (Fahridin & Britt 2008).

Time trends

The rate of general practice encounters for allergic rhinitis decreased between 1998–99 and 2007–08. In 1998–99, allergic rhinitis was managed at a rate of 1.0 per 100 encounters. By 2007–08, this had fallen to a rate of 0.6 per 100 encounters.

In 1998–99, allergic rhinitis represented 0.7% of all problems managed and 3.9% of respiratory problems managed. In 2007–08, these figures were 0.4% and 3.0% respectively (Henderson & Pan 2009).

In 1998–99, allergic rhinitis was the 29th most frequently managed problem. In 2007–08, it was the 35th most frequently managed problem (AIHW: Britt et al. 2008).

Prescription, recommendation and supply of medication

In the majority of cases, medications used for managing allergic rhinitis are administered orally or intranasally (within the nose) (Bousquet et al. 2008).

A Supplementary Analysis of Nominated Data study showed that in 2008–09, 71.3% of patients with allergic rhinitis were taking at least one allergic rhinitis medication. Nearly equal proportions were taking intranasal corticosteroids (38.0%) and antihistamines (37.2%) (AIHW Australian GP Statistics and Classification Centre 2009).

Between 2002 and 2007, there were 2,965 encounters recorded by the BEACH survey of general practice at which allergic rhinitis was managed. Based on these encounters:

- Medications were prescribed, advised for over-the-counter purchase or supplied by the GP at an overall rate of 107.6 per 100 allergic rhinitis problems. The rate reported here indicates that more than one drug could be prescribed, recommended or supplied per problem.
- Prescription of medication was most common, occurring at a rate of 64.9 per 100 allergic rhinitis problems.
- Over-the-counter medications were advised at a rate of 34.0 per 100 allergic rhinitis problems.
- Medication was supplied by the GP at a rate of 8.7 per 100 allergic rhinitis problems (Fahridin & Britt 2008).

The most common medications prescribed were intranasal corticosteroids, antihistamines and allergen treatment injections (see Glossary for definitions).

Allergen treatment injections are used during immunotherapy, which reduces sensitivity to allergens.

The six most common medications prescribed during the 2,965 encounters recorded by the BEACH survey of general practice activity (Fahridin & Britt 2008) between 2002 and 2007 were:

- budesonide topical nasal (an intranasal corticosteroid) (31.0% of prescribed medications for allergic rhinitis)
- mometasone nasal (an intranasal corticosteroid) (28.0%)
- loratadine (an antihistamine) (6.2%)
- beclomethasone nasal spray (an intranasal corticosteroid) (4.3%)
- cetirizine (an antihistamine) (4.0%)
- allergen treatment injections (2.9%).

4.3 Management of allergic rhinitis in hospital

Allergic rhinitis is not a common cause of hospitalisation. According to the Australian Institute of Health and Welfare's (AIHW) National Hospital Morbidity Database, in 2009–10 there were 1,523 hospitalisations with allergic rhinitis listed as either a principal diagnosis (the diagnosis describing the problem that was chiefly responsible for the patient's episode of care in hospital) or an additional diagnosis (a condition or complaint either coexisting with the principal diagnosis or arising during the episode of care in hospital, and relevant to the episode of care). This represents 0.02% of all hospitalisations in that year.

Allergic rhinitis was listed as the principal diagnosis in about a third (477) of these hospitalisations. In the remainder, allergic rhinitis was an additional diagnosis (1,046).

Age and sex

During the 5-year period from 2005–06 to 2009–10, there were 2,322 hospitalisations with a principal diagnosis of allergic rhinitis in Australia, according to the AIHW National Morbidity Database.

During this period, hospitalisations with a principal diagnosis of allergic rhinitis were more common in younger Australians compared with older Australians. Despite allergic rhinitis being most prevalent among those of working age, hospitalisations for allergic rhinitis were most likely to occur in those aged 14 years and under.

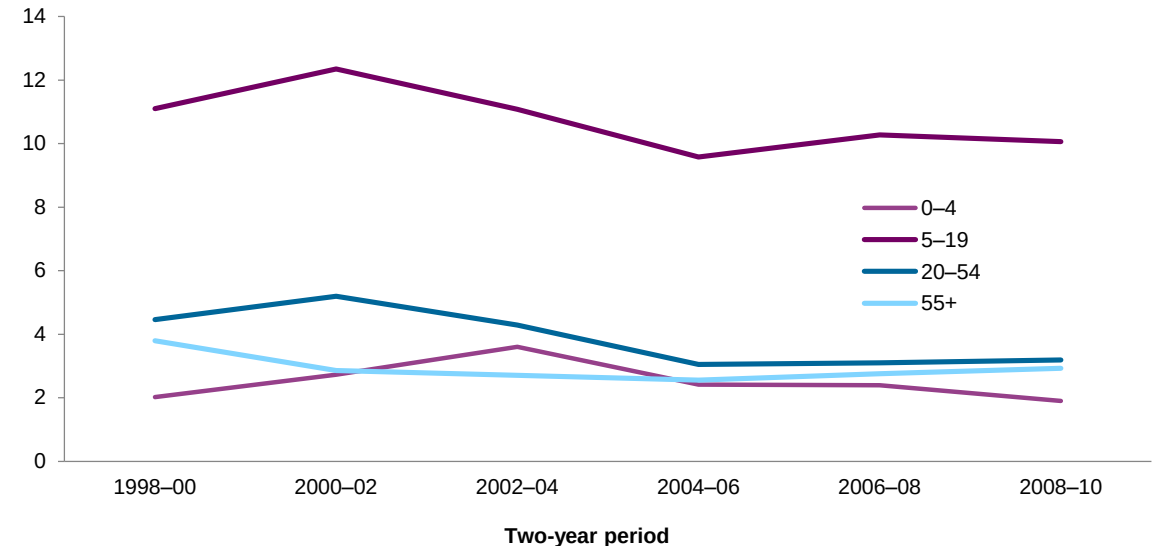
It was more common for males (12.7 per 100,000 population) to have a principal diagnosis of allergic rhinitis than females (9.1 per 100,000 population) during these 5 years.

Males aged 5–9 were 5 times more likely to be hospitalised for allergic rhinitis compared with females of the same age. Among those aged 10–14 years, males were twice as likely as females 10–14 years to have been hospitalised for allergic rhinitis.

Trends in hospitalisations

According to the AIHW National Hospital Morbidity Database, allergic rhinitis hospitalisation rates did not vary much over the 12 years from 1998–00 to 2008–10, apart from a peak around 2000–02, seen most clearly in the 5–19 years age group (Figure 4.7). There was no corresponding peak in all hospitalisations around this time.

The 5–19 years age group had a hospitalisation rate well above the other age groups for each of the years between 1998–00 and 2008–10.



Notes

1. The hospitalisation rate for each age group is age standardised to the Australian population as at June 2001.
2. Allergic rhinitis was classified according to the International Classification of Diseases, 10th Revision, Australian Modification (ICD-10-AM) codes J301–J304.
3. This represents the period from the introduction of the ICD-10-AM (1998–99) to the most recently available data (2008–09). The age groups were selected to highlight that hospitalisations for allergic rhinitis are most common in childhood and adolescence.

Source: AIHW National Hospital Morbidity database.

Figure 4.7: Hospitalisation rates where allergic rhinitis was the principal diagnosis, two year periods combined from 1998–00 to 2008–10

Comorbidities in patients admitted to hospital with allergic rhinitis

In 2009–10, 60% of patients admitted to hospital with a principal diagnosis of allergic rhinitis had at least one comorbid condition associated with their hospital stay, based on the AIHW Hospital Morbidity Database. The most common comorbidity was another respiratory condition. Around 44% of those hospitalised with a principal

diagnosis of allergic rhinitis had at least one other respiratory condition associated with their hospitalisation.

Among the comorbid respiratory conditions reported, the most common were those involving the nasal passages, such as nasal or other sinus polyps, hypertrophy of nasal turbinates (enlargement of the bones and tissue situated along the side wall of the nose), deviation of the nasal septum (the wall that divides the left and right nostrils) or rhinoliths

(stones that form in the nasal cavity). Combined, these conditions accounted for 79% of the comorbid respiratory conditions.

Other common comorbid respiratory conditions were hypertrophy of the adenoids (enlargement of the lymphatic tissue in the upper pharynx) (11% of comorbid respiratory conditions) and chronic sinusitis (11%).

Hospital procedures

The AIHW National Morbidity Database reveals that, in 2009–10, 88% of patients admitted to hospital with a principal diagnosis of allergic rhinitis received at least one hospital procedure.

Sinoscopy, or the examination of the sinuses through magnifying lenses, performed 121 times, was the most common procedure performed in hospital on those with a principal diagnosis of allergic rhinitis in 2009–10.

Cauterisation, or diathermy, of nasal turbinates (bones situated along the side wall of the nose) was performed on 116 occasions in 2009–10 for hospitalisations where allergic rhinitis was the principal diagnosis, making it the equal second most common procedure. In this procedure, high-frequency electrical currents are used to seal a blood vessel in the nasal turbinates. This results in local tissue destruction. In allergic rhinitis sufferers this procedure can reduce the obstruction caused by rhinitis (Jones et al. 1985).

Intranasal maxillary antrostomy, performed 116 times in 2009–10, was the equal second most common procedure in hospital on those with a principal diagnosis of allergic rhinitis. It is the surgical creation of a hole in the wall between the nasal passages and the sinus cavity in one or both of the cheeks (maxillary sinuses), for the purposes of draining mucus more freely. The hole created is in addition to the natural drainage passage, which may be blocked or not operating efficiently in the patient requiring this procedure.

Discussion

In this report the authors have investigated the burden of allergic rhinitis in Australia by examining its common triggers, how prevalent it is and how it is managed, and by assessing its effect on other conditions.

Australia has a particularly high prevalence of allergic rhinitis by global standards (Bousquet et al. 2008). It is the most common chronic respiratory condition in Australia. It affects about 15% of the population, particularly those of working age. In 2007–08 allergic rhinitis was slightly more common among females than males, and was most common among Australians aged 25–44 years and least common among those aged 0–14 and 65–74.

Allergic rhinitis can affect sleep, cognitive and psychomotor function, and participation in social activities. It frequently coexists with other allergic conditions such as asthma and chronic sinusitis (Hu et al. 2008). These effects lead to a number of direct and indirect costs to Australians.

Both genetic and environmental factors contribute to the onset and development of allergic rhinitis. The main environmental factors are:

- inhaled airborne allergens originating from insects, mites, plants, fungi and mammals that are delivered from house dust, pets, pollens and indoor and outdoor fungi
- pollutants emanating from sources such as bushfires, vegetation, dust storms, industrial premises and road vehicles in the form of nitrogen dioxide, ozone and sulphur dioxide, and particulate matter, which can be coarse (such as road dust), fine (such as tobacco smoke) or ultrafine (such as residue of fossil fuel consumption)
- occupational exposures.

Based on self-reporting, in 2007–08 allergic rhinitis rates were highest in the Australian Capital Territory, Western Australia, Victoria and South Australia. New South Wales and Queensland had rates significantly lower than the overall rate for Australia.

Most Australian adults appear to self-medicate for allergic rhinitis. However, there is strong anecdotal evidence that pharmacies also play an important role in the management of allergic rhinitis. A 2002 survey of allergy sufferers revealed that nearly two-thirds of respondents did not consult their GP about their allergic rhinitis treatment (Walls et al. 2005).

The total wholesale cost of antihistamines almost doubled, from $85.5 million in 2001 to $167.0 million in 2010. In 2010, most of this medicine was available over-the-counter, and therefore much of this cost was borne by patients. In fact, the amount paid for by the patients is more than this wholesale figure.

The amount of oral antihistamines supplied to pharmacies exceeded that of intranasal corticosteroids every year from 2001 to 2010. It appears that this trend has strengthened over the 10-year period owing largely to increasing availability of over-the-counter oral antihistamines. In 2010, almost 3 times as many oral antihistamines (26.48 DDD/1,000 persons/day) were supplied than intranasal corticosteroids (7.8 DDD/1,000 persons/day).

The results showed that the use and wholesale cost of allergic rhinitis medication has increased.

Continued efforts to monitor the supply of medications to manage allergic rhinitis is critical in ensuring the quality use of medicine for this condition.

Allergic rhinitis is not a common cause of hospitalisation. Sinoscopy, cauterisation of the nasal turbinates (bones situated along the side wall of the nose) and intranasal maxillary antrostomy were three most common procedures performed on patients with allergic rhinitis in 2009–10.

The prevalence of allergic rhinitis in Australia is not reflected in GP visits or hospitalisations. This supports the notion that a large number of Australians are self-managing their allergic rhinitis. While it is known that pharmacies play an important role in the management of allergic rhinitis, the degree to which sufferers consult pharmacists regarding their self-management of their allergic rhinitis is unquantified.

In 2004–05 about 700,000 Australians had both allergic rhinitis and asthma. Similarly 800,000 had both allergic rhinitis and chronic sinusitis (AIHW 2010). There is evidence that in a patient who has both allergic rhinitis and asthma, the asthma symptoms are more difficult to control.

There are many questions about allergic rhinitis that were not answered within the scope of this report. They indicate areas for further investigation:

- To what extent is allergic rhinitis a self-managed condition in Australia?
- To what extent is the burden of health care for allergic rhinitis being placed on pharmacies rather than on general practice?
- What is the extent and relative share of prescribed and over-the-counter medications used to control the symptoms of allergic rhinitis?
- What is an up-to-date estimate of Australian national direct expenditure on allergic rhinitis?
- What is the geographical distribution of allergic rhinitis in Australia beyond state-based analysis?
- What are the qualitative effects of allergic rhinitis on daily activities such as work, school, sleep and recreation?

Appendix A: Definitions and data sources for medication data

A1 Regulatory control of medication supply

The Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) is the system used to regulate the availability of drugs and poisons in Australia. This system is produced by the Therapeutic Goods Administration of the Australian Government Department of Health and Ageing. Of the nine schedules included in the SUSMP, medicines are included in four schedules:

- Schedule 2 Pharmacy Medicine
- Schedule 3 Pharmacist Only Medicine
- Schedule 4 Prescription Only Medicine, or Prescription Animal Remedy
- Schedule 8 Controlled Drug.

The medicines included in each of the schedules are described as follows in the Poisons Standard 2011 (Department of Health and Aging and Therapeutic Goods Administration 2011):

> Schedule 2. Pharmacy Medicine — Substances, the safe use of which may require advice from a pharmacist and which should be available from a pharmacy or, where a pharmacy service is not available, from a licensed person.
>
> Schedule 3. Pharmacist Only Medicine — Substances, the safe use of which requires professional advice but which should be available to the public from a pharmacist without a prescription.
>
> Schedule 4. Prescription Only Medicine, or Prescription Animal Remedy — Substances, the use or supply of which should be by or on the order of persons permitted by State or Territory legislation to prescribe and should be available from a pharmacist on prescription.
>
> Schedule 8. Controlled Drug — Substances which should be available for use but require restriction of manufacture, supply, distribution, possession and use to reduce abuse, misuse and physical or psychological dependence.

Medicines used for management of allergic rhinitis are classified into Schedule 2, 3 or 4 with progression through these schedules signifying increasingly stricter controls.

A2 Medication data

IMS Health data includes both prescription and non-prescription medicines supplied to the hospitals and community pharmacies. IMS Health's wholesale data can be analysed separately for supplies to hospitals and pharmacies. As allergic rhinitis is largely managed through self-care or in primary health-care services, the analysis here focused on the supply of medications to community pharmacies.

While IMS Health collects data from most pharmaceutical wholesalers, data from two companies, namely Alphapharm and Arrow Pharmaceuticals, were not captured in their data (but note that the data from Arrow Pharmaceuticals are included in IMS Health data from February 2011). IMS Health estimates that Alphapharm and Arrow make up about 4%

of the cost of pharmaceutical supplies across all therapeutic areas at the national level. IMS Health uses statistical methods to project the sales of products from these companies in each therapeutic area, and these were also used in the analysis.

The Australian Government subsidises the cost of medicines through the Pharmaceutical Benefits Scheme (PBS) and the Repatriation Pharmaceutical Benefits Scheme (RPBS). As many allergic rhinitis medicines can be obtained from pharmacies without prescriptions, the PBS data were not suitable for examining supply of medications for this condition.

Data extraction for medicines used to manage allergic rhinitis

Intranasal corticosteroids and antihistamines are used also to treat allergies and not exclusively used to manage allergic rhinitis. IMS Health data being a wholesale data, no information about the purchasers of the medications are recorded.

Data items in IMS Health data

The wholesale supply data for intranasal corticoids and oral antihistamines from 2001 to 2010 were received from IMS Health. The data was supplied at the proprietary name (product) level, and each row of the data included:

- year of supply
- month of supply
- medication included
- number of packs sold
- wholesale dollar cost
- classification in the SUSMP as an over-the-counter or non–over-the-counter drug in 2010.

A3 Defined daily dose as a measure of drug utilisation

'Defined daily dose' (DDD), determined for each therapeutic substance by the WHOCC, refers to the 'assumed average maintenance dose per day for a drug used for its main indication in adults' (WHOCC 2009).

The DDD is not necessarily the recommended or prescribed daily dose for all consumers of the same medicine. Doses for individual patients often differ from the DDD as these depend on age, weight, ethnicity, genetic make-up and other individual characteristics that affect how medications are absorbed and eliminated in the body.

While the DDD does not provide the exact amount of medication consumption, the DDD is a widely used unit for estimating drug supply. This measure can be used to compare the supply of medicines that may come in a variety of dosage forms (for example, tablets, intranasal sprays, inhaler and liquid) and dosage strengths (the amount of medication contained in one unit of medication). The DDD was used to assess the supply patterns of intranasal corticosteroids and oral antihistamines.

Glossary

additional diagnosis	a condition or complaint either coexisting with the principal diagnosis or arising during the episode of care in hospital, and relevant to the episode of care
allergen	an antigen that reacts with specific antibodies to produce an allergic reaction in a susceptible individual
allergenic	having the potential to induce an allergic response
allergenicity	the property of being allergenic
allergic reaction	reactions of parts of an organism that occur because of contact with foreign substances or other organisms
allergic rhinitis	inflammation of the nasal mucosa caused by hypersensitivity to an allergen
allergy	a hypersensitivity disorder of the immune system in response to allergens
antibody	a protein used by the immune system to identify and neutralise foreign objects
antigen	a substance that, when introduced into the body, triggers the production of an antibody by the immune system
antihistamine	allergy medication that inhibits the body's reception of histamine
atopy	a genetic tendency to develop allergic reactions
comorbidity	when a person has two or more health problems at the same time
dander	particles of dry scales or fluff, shed from the skin, hair or feathers of animals, which may act as allergens
defined daily dose (DDD)	assumed average maintenance dose per day for a drug used for its main indication in adults (WHOCC 2009)
effusion	the escape of a fluid from its natural vessels into a body cavity
enzymes	proteins that accelerates specific metabolic processes of an organism
hay fever (seasonal rhinitis)	inflammation of the nasal mucosa when triggered by seasonal exposure to pollen
histamine	a product that is released by tissues during an allergic reaction and can trigger an inflammatory response
hormonal rhinitis	rhinitis caused by changes in hormones such as those occurring in pregnancy, menstruation, puberty or hypothyroidism
hyperresponsiveness	chronic inflammation in response to a variety of allergens, which have little or no effect on a non-allergic person
hypersensitivity	excessive reactions produced by the immune system to minimal stimuli
IgE antibodies	a class of antibody that has only been found in mammals (see *Immunoglobulin E*)

IgE reactivity	hypersensitivity of membranes to environmental substances associated with increased Immunoglobulin E (IgE) production
inflammation	local response to injury or infection, marked by local redness, heat, swelling and pain
immune system	a system of structures and processes within an organism that protects it against disease, by identifying and killing a wide variety of other organisms and foreign substances that it distinguishes from its own healthy cells and tissues
Immunoglobulin E (IgE)	a large protein used by the immune system to identify and neutralise foreign objects
inhalant allergens	inhaled airborne particles which may cause allergy symptoms
intranasal corticosteroids	a class of steroid hormones commonly used to to treat the nasal mucosa to control symptoms associated with sneezing, rhinorrhea (runny nose), itching and nasal congestion
irritant-induced rhinitis	non-allergic rhinitis due to an irritant
mucosa	a lubricating membrane lining an internal surface or an organ
nasal mucosa	moist tissues that line the inside of the nose
over-the-counter (OTC)	substances that can be purchased from community pharmacies without the need for a prescription from a health-care professional. This compares to prescription drugs, which may be sold only to consumers possessing a valid prescription. OTC medicines have ingredients that have been determined as safe and effective when used without the guidance of a health-care professional.
oxidant	a chemical compound that readily transfers oxygen atoms
perennial rhinitis	rhinitis where symptoms are present throughout the year
principal diagnosis	the diagnosis describing the problem that was chiefly responsible for the patient's episode of care in hospital
proteins	proteins are required for the structure, function, and regulation of an organism's cells tissues and organs. Each protein has unique functions.
reagent	a substance which, on account of the reactions it causes, is used in chemical analysis
rhinitis	inflammation of the nasal mucosa
rhinorrhea	excessive discharge from the nasal mucosa; commonly referred to as a 'runny nose'
sensitisation	refers to the process by which a cellular receptor becomes more likely to respond to a stimulus (more efficient). It follows progressive amplification of a response after repeated administrations of a stimulus.
vasomotor rhinitis	rhinitis due to abnormal nerve control of the blood vessels in the nose
volatile organic compound (VOC)	a substance containing carbon with a tendency to produce vapour at normal temperatures

References

ABS 2009. National Health Survey: Summary of Results, 2007-2008.

ABS 2011. Australian Standard Geographical Classification (ASGC) July 2010.

AIHW 2008. Occupational asthma in Australia. Canberra: AIHW.

AIHW 2010. Asthma, chronic obstructive pulmonary disease and other respiratory diseases in Australia. Cat. no. ACM 20. Canberra: AIHW.

AIHW Australian GP Statistics and Classification Centre 2009. SAND abstract number 126: Asthma and allergic rhinitis in general practice patients. December 2009. Viewed February 2011, <http://www.fmrc.org.au/Beach/Abstracts/126-Asthma_and_allergic_rhinitis.pdf>.

AIHW: Australian Centre for Asthma Monitoring 2008. Asthma in Australia 2008. Cat. no. ACM 14. Canberra: AIHW.

AIHW: Britt H, Miller G, Charles J, Henderson J, Bayram C, Harrison C et al. 2008. General practice activity in Australia 1998-99 to 2007-08: 10 year data tables. Cat. no. GEP 23. Canberra: AIHW.

Alles R, Parikh A, Hawk L, Darby Y, Romero J & Scadding G 2001. The prevalence of atopic disorders in children with chronic otitis media with effusion. Pediatr Allergy Immunol 12:102-6.

Aoyama K, Ueda A, Manda F, Matsushita T, Ueda T & Yamauchi C 1992. Allergy to laboratory animals: an epidemiological study. British Journal of Industrial Medicine 49:41-7.

Arnedo-Pena A, Garcia-Marcos L, Carvajal Uruena I, Busquets Monge R, Morales Suarez-Varela M, Miner Canflanca I et al. 2009. Air pollution and recent symptoms of asthma, allergic rhinitis, and atopic eczema in schoolchildren aged between 6 and 7 years. Arch Bronconeumol 45:224-9.

Arshad S, Tariq S, Matthews S & Hakim E 2001. Sensitization to common allergens and its association with allergic disorders at age 4 years: a whole population birth cohort study. Pediatrics 108:E33.

Australian Society of Clinical Immunology and Allergy 2008. Guide to common allergic pollen. February 2011. Viewed March 2011, <www.allergy.org.au/content/category/3/48/241/>.

Australian Society of Clinical Immunology and Allergy 2010a. Allergic rhinitis (hay fever) checklist. Viewed March 2011, <http://www.allergy.org.au/images/stories/aer/infobulletins/2010pdf/ascia_allergic_rhinitis_checklist_2010.pdf>.

Australian Society of Clinical Immunology and Allergy 2010b. Pollen allergy. 01 June 2010. Viewed February 2011, <http://www.allergy.org.au/content/view/132/131/>.

Becker J 2009. Allergic Rhinitis. Viewed April 2010, <http://emedicine.medscape.com/article/889259-overview>.

Bollinger M, Eggleston P, Flanagan E & Wood R 1996. Cat antigen in homes with and without cats may induce allergic symptoms. J Allergy Clin Immunol 97:907-14.

Bousquet J, Cauwenberge PV & Khaltaev N 2001. Allergic rhinitis and its impact on asthma. Journal of Allergy and Clinical Immunology 108:S147-S334.

Bousquet J, Flahault A, Vanden-Plas O, Ameille J, Duron J & Pecquet C 2006. Natural rubber latex allergy among health care workers: a systematic review of the evidence. Journal of Allergy and Clinical Immunology 118:447-54.

Bousquet J, Khaltaev N, Cruz A, Denburg J, Fokkens W & Togias A 2008. Allergic Rhinitis and its Impact on Asthma(ARIA) 2008 update (in collaboration with the World Health Organization, GA(2)LEN and AllerGen). Allergy 63:8-160.

Boutin Y, Hebert H, Vrancken E & Mourad W 1988. Allergenicity and cross-reactivity of cat and dog allergenic extracts. Clin Allergy 18:287-93.

Brasch-Andersen C, Haagerup A, Borglum A, Vestbo J & Kruse T 2006. Highly significant linkage to chromosome 3q13.31 for rhinitis and related allergic diseases. J Med Genet 43:e10.

Britt H, Miller G, Charles J, Henderson J, Bayram C, Valenti L et al. 2009. General practice activity in Australia 1999-00 to 2008-09: changes over time data reference tables. Vol. 26, (Ed. AIHW). Canberra.

Bush RK, Wood RA & Eggleston PA 1998. Laboratory animal allergy. Journal of Allergy and Clinical Immunology 102:99-112.

California Environmental Protection Agency: Air Resources Board 2005. Proposed identification of environmental tobacco smoke as a toxic air contaminant.

Castano R & Theriault G 2006. Defining and classifying occupational rhinitis. Journal of Laryngology and Otology 120:812-7.

Ciccone G, Forastiere F & Agabitit N 1998. Road traffic and adverse respiratory effects in children. Occupational and Environmental Medicine 55:771-8.

Craig TJ, McCann JL, Gurevich F & Davies MJ 2004. The correlation between allergic rhinitis and sleep disturbance. J Allergy Clin Immunol 114:S139-45.

Department of Health and Aging and Therapeutic Goods Administration 2011. Poisons Standard 2011.

Durham S 2000. ABC of allergies. London: BMJ Books.

Dykewicz M, Fineman S, Skoner D, Nicklas R, Lee R, Blessing-Moore J et al. 1998. Diagnosis and management of rhinitis: complete guidelines of the Joint Task Force on Practice Parameters in Allergy, Asthma and Immunology. American Academy of Allergy, Asthma, and Immunology. Ann Allergy Asthma Immunol 81:478-518.

Dykewicz M, Patterson R, Cugell DW, Harris KE & Wu AF 1991. Serum IgE and IgG to formaldehyde-human serum albumin: lack of relation to gaseous formaladehyde exposure and symptoms. Journal of Allergy and Clinical Immunology 87:48-57.

Elms J, Fishwisk D, Walker J, Rawbone R, Jerey P & Grin P 2003. Prevalence of sensitisation to cellulase and xylanase in bakery workers. Occupational and Environmental Medicine 60:802-4.

Fahridin S & Britt H 2008. Allergic rhinitis. Aust Fam Physician 37:203.

Font-Ribera L, Kogevinas M, Zock JP, Nieuwenhuijsen MJ, Heederik D & Villanueva CM 2009. Swimming pool attendance and risk of asthma and allergic symptoms in children. Eur Respir J 34:1304-10.

Font-Ribera L, Villanueva CM, Nieuwenhuijsen MJ, Zock JP, Kogevinas M & Henderson J 2011. Swimming pool attendance, asthma, allergies, and lung function in the avon longitudinal study of parents and children cohort. Am J Respir Crit Care Med 183:582-8.

Giavina-Bianchi PF, Jr., Castro FF, Machado ML & Duarte AJ 1997. Occupational respiratory allergic disease induced by Passiflora alata and Rhamnus purshiana. Ann Allergy Asthma Immunol 79:449-54.

Gordon S 1997. Allergy to furred animals. Clin Exp Allergy 27:479-81.

Hajat S, Haines A, Atkinson R, Bremner S, Anderson H & Emberlin J 2001. Association between air pollution and daily consultations with general practitioners for allergic rhinitis in London, United Kingdom. Am J Epidemiol 153:704-14.

Henderson J & Pan Y 2009. Respiratory problems. In: Britt H & Miller G (eds). General practice in Australia, health priorities and policies 1998 to 2008. Canberra: AIHW.

Hu W, Katelaris C & Kemp A 2008. Allergic rhinitis - practical management strategies. Aust Fam Physician 37:214-20.

Jaeger D, Kleinhans D, Czuppon AB & Baur X 1992. Latex-specific proteins causing immediate-type cutaneous nasal, bronchial, and systemic reactions. Journal of Allergy and Clinical Immunology 89:759-68.

Jones AS, Lancer JM, Moir AA & Stevens JC 1985. The effect of submucosal diathermy to the inferior turbinates on nasal resistance to airflow in allergic and vasomotor rhinitis. Clin Otolaryngol Allied Sci 10:249-52.

Kalra S, Crank P, Hepworth J, Pickering C & Woodcock A 1992. Absence of seasonal variation in concentrations of the house dust mite allergen Der p1 in south Manchester homes. Thorax 47:928-31.

Kennedy S, Mcdonald JT & Biddel N 2006. The Healthy Immigrant Effect and Immigrant Selection: Evidence from Four Countries, SEDAP Research Paper No. 164. A program for research on social and economic dimensions of an aging population (SEDAP).

Leroyer C, Malo JL, Girard D, Dufour JG & Gautrin D 1999. Chronic rhinitis in workers at risk of reactive airways dysfunction syndrome due to exposure to chlorine. Occup Environ Med 56:334-8.

Luczynska C, Li Y, Chapman M & Platts-Mills T 1990. Airborne concentrations and particle size distribution of allergen derived from domestic cats (Felis domesticus). Measurements using cascade impactor, liquid impinger, and a two-site monoclonal antibody assay for Fel d I. Am Rev Respir Dis 141:361-7.

Martinez F, Antognoni G, Macri F, Bonci E, Midulla F, Castro GD et al. 1988. Parental smoking enhances bronchial responsiveness in nine-year-old children. Am Rev Respir Dis 138:518-23.

Matheson M, Abramson M, Dharmage S, Forbes A, Raven J, Thien F et al. 2005. Changes in indoor allergen and fungal levels predict changes in asthma activity among young adults. Clin Exp Allergy 35:907-13.

Matheson M, Walters E, Simpson J, Wharton C, Ponsonby A, Johns D et al. 2009. Relevance of the hygiene hypothesis to early vs. late onset allergic rhinitis. Clin Exp Allergy 39:370-8.

Meltzer E, Szwarcberg J & Pill M 2004. Allergic rhinitis, asthma, and rhinosinusitis: diseases of the integrated airway. J Manag Care Pharm 10:310-7.

Nielsen J, Welinder H, Schutz A & Skerfing S 1988. Specific antibodies against phthalic anhydride in occupationally exposed subjects. Journal of Allergy and Clinical Immunology 82:126-32.

Pallasaho P, Ronmark E, Haahtela T, Sovijarvi AR & Lundback B 2006. Degree and clinical relevance of sensitization to common allergens among adults: a population study in Helsinki, Finland. Clin Exp Allergy 36:503-9.

Pinto J, Hayes M, Schneider D, Naclerio R & Ober C 2008. A genomewide screen for chronic rhinosinusitis genes identifies a locus on chromosome 7q. Laryngoscope 118:2067-72.

Platts-Mills T, Hayden M, Chapman M & Wilkins S 1987. Seasonal variation in dust mite and grass-pollen allergens in dust from the houses of patients with asthma. J Allergy Clin Immunol 79:781-91.

Pohlabeln H, Jacobs S & Bohmann J 2007. Exposure to pets and the risk of allergic symptoms during the first 2 years of life. J Investig Allergol Clin Immunol 17:302-8.

Quillen D & Feller D 2006. Diagnosing rhinitis: allergic vs. nonallergic. Am Fam Physician 73:1583-90.

Radon K, Gerhardinger U, Schulze A, Zock J, Norback D, Toren K et al. 2008. Occupation and adult onset of rhinitis in the general population. Occupational and Environmental Medicine 65:38-43.

Raherison C, Penard-Morand C, Moreau D, Caillaud D, Charpin D, Kopfersmitt C et al. 2007. In utero and childhood exposure to parental tobacco smoke, and allergies in schoolchildren. Respir Med 101:107-17.

Ramadour M, Burel C, Lanteaume A, Vervloet D, Charpin D, Brisse F et al. 2000. Prevalence of asthma and rhinitis in relation to long-term exposure to gaseous air pollutants. Allergy 55:1163-9.

Remes S, Castro-Rodriguez J, Holberg C, Martinez F & Wright A 2001. Dog exposure in infancy decreases the subsequent risk of frequent wheeze but not of atopy. J Allergy Clin Immunol 108:509-15.

Rubinstein I 1995. Nasal inflammation in patients with obstructive sleep apnea. Laryngoscope 105:175-7.

Santamaria A 2007. Human health basis for proposed revisions to ozone standard. Houston, Texas: ENVIRON International Corporation.

Sari-Minodier I, Charpin D, Signouret M, Poyen D & Vervloet D 1999. Prevalence of self-reported respiratory symptoms in workers exposed to iso-cyanates. Journal of Occupational and Environmental Medicine 41:582-8.

Scarupa M & Kaliner M 2006. Rhinitis. Feburary 2011. Viewed March 2011, <http://www.worldallergy.org/professional/allergic_diseases_center/rhinitis/rhinitis_indepth.php>.

Settipane R 2003. Rhinitis: a dose of epidemiological reality. Allergy Asthma Proc 24:147-54.

Sheikh A, Hurwitz B, Nurmatov U & van Schayck CP 2010. House dust mite avoidance measures for perennial allergic rhinitis. Cochrane Database Syst Rev:CD001563.

Shusterman D, Murphy M & Balmes J 1998. Subjects with seasonal allergic rhinitis and nonrhinitic subjects react differentially to nasal provocation with chlorine gas. J Allergy Clin Immunol 101:732-40.

Shusterman D, Murphy M & Balmes J 2003. Differences in nasal irritant sensitivity by age, gender, and allergic rhinitis status. International Archives of Occupational and Environmental Health 76:577-83.

Sibbald B & Rink E 1991. Epidemiology of seasonal and perennial rhinitis: clinical presentation and medical history. Thorax 46:895-901.

Sibbald B & Strachan D 1995. Epidemiology of rhinitis. In: Busse W & Holgate S (eds). Asthma and rhinitis. London: Blackwell Scientific, 32-43.

Sporik R, Holgate S, Platts-Mills T & Cogswell J 1990. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. A prospective study. N Engl J Med 323:502-7.

Swierczyniska-Machura D, Krakowiak A & Palczynski C 2006. [Occupational allergy caused by ornamental plants]. Med Pr 57:359-64.

Teele DW, Klein JO & Rosner B 1989. Epidemiology of otitis media during the first seven years of life in children in greater Boston: a prospective, cohort study. J Infect Dis 160:83-94.

Thomsen S, Ulrik C, Kyvik K, Larsen K, Skadhauge L, Steffensen I et al. 2005. The incidence of asthma in young adults. Chest 127:1928-34.

United States Environmental Protection Agency 2011. Particulate Matter. March 2011. Viewed June 2011, <http://www.epa.gov/airquality/particlepollution/>.

van den Berge M, Kerstjens HA & Postma DS 2002. Provocation with adenosine 5'-monophosphate as a marker of inflammation in asthma, allergic rhinitis and chronic obstructive pulmonary disease. Clin Exp Allergy 32:824-30.

von Mutius E, Sherrill DL, Fritzsch C, Martinez FD & Lebowitz MD 1995. Air pollution and upper respiratory symptoms in children from East Germany. Eur Respir J 8:723-8.

Wallace DV, Dykewicz MS, Bernstein DI, Blessing-Moore J, Cox L, Khan DA et al. 2008. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol 122:S1-84.

Walls R, Heddle R, Tang M, Basger B, Solley G & Yeo G 2005. Optimising the management of allergic rhinitis: an Australian perspective. Med J Aust 182:28-33.

Wang D 2005. Risk factors of allergic rhinitis: genetic or environmental? Ther Clin Risk Manag 1:115-23.

Weiland SK, Mundt KA, Ruckmann A & Keil U 1994. Self-reported wheezing and allergic rhinitis in children and traffic density on street of residence. Annals of Epidemiology 4:243-7.

WHO 2004. Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide.

WHOCC 2009. Definition and general considerations. Viewed 15 July 2011, <http://www.whocc.no/ddd/definition_and_general_considera/>.

WHOCC 2010. ATC/DDD index 2011. Viewed 15 July 2011, <http://www.whocc.no/atc_ddd_index>.

World Allergy Organization 2003. IgE in Clinical Allergy and Allergy Diagnosis.

Zwick H, Popp W, Wagner C, Reiser K, Schmoger J, Bock A et al. 1991. Effects of ozone on the respiratory health, allergic sensitization, and cellular immune system in children. Am Rev Respir Dis 144:1075-9.