1 Introduction

Respiratory diseases are major causes of illness and mortality in Australia. They place great stress on the health care system and are a significant financial burden for the individual and the community. Acute respiratory infections such as influenza are still highly prevalent and claim a substantial number of lives each year. However, greater attention is now being paid to the prevalence, consequences and prevention of chronic respiratory diseases. This is illustrated by the naming of asthma as a National Health Priority Area, the only respiratory disease to be so named.

Chronic respiratory diseases are a diverse group of illnesses and conditions affecting the process of breathing and oxygen delivery. As a group, they involve many and varied causative pathways, symptoms and outcomes. They are highly prevalent in the community and constitute a significant health problem in Australia. Nevertheless, effective prevention of chronic respiratory diseases is possible because they have risk factors that are identifiable and avoidable. Tobacco smoking is by far the greatest risk factor for chronic obstructive pulmonary disease (COPD) and worsens some people's asthma.

Purpose and scope of the report

This report is a concise summary of the prevalence and consequences of a selection of chronic respiratory diseases affecting Australians. The report also contains an overview of chronic respiratory disease risk factors and prevention strategies. Some of the information on asthma contained in this report is available in the first national report (Australian Centre for Asthma Monitoring 2003), published by the Australian Institute of Health and Welfare (AIHW). Nevertheless, the present report provides a contrast between asthma and other chronic respiratory diseases, especially COPD. The information here will serve as a resource for anyone with an interest in the area, especially those who are developing policies and services to help reduce the burden of disease in Australia.

The report highlights the following diseases:

- COPD—a disease characterised by progressive development of airflow limitation that is not fully reversible. In most instances emphysema is the underlying condition, although people with COPD often also have chronic bronchitis.
- Asthma—a chronic inflammatory disorder of the airways characterised by reversible airflow obstruction and resulting in cough, wheeze, chest tightness and shortness of breath.
- Bronchiectasis—a chronic abnormal dilation and distortion of the bronchi characterised by persistent infection and cough.

- Pneumoconiosis—a non-cancerous reaction, and subsequent alteration, of the lungs to inhaled mineral or organic dusts or fibres.
- Chronic sinusitis—a chronic inflammation of the lining of one or more of the sinuses due to a recurring or long-lasting infection, or an allergy.
- Hay fever (allergic rhinitis)—an inflammation or irritation of the mucous membranes of the nose caused by an allergic reaction.

Lung cancer is excluded from this report because its treatment and management approaches are very different from other chronic respiratory diseases. Cystic fibrosis is also excluded as it is an inherited disease with few environmental factors (Loddenkemper et al. 2003).

Structure of the report

This report has seven chapters. Chapters 2 and 3 contain descriptions of COPD and asthma, their prevalence, and consequences for the health of Australians, including detailed information on aspects of each disease's contribution to mortality, disability, use of health care services and health system expenditure. These two chapters contain fact sheets highlighting aspects of the mortality and hospitalisation caused by each disease. The fact sheets also illustrate key tendencies and short-term trends. The short-term trends illustrated by the mortality fact sheets are complemented by graphs showing longer term trends.

Chapter 4 contains pertinent prevalence and consequence information on the other four chronic respiratory diseases. These diseases, though sometimes highly prevalent and potentially serious, do not cause as much mortality and disability as COPD and asthma and are therefore not covered in as much detail.

The following measures are used to profile the prevalence and consequences of the chronic respiratory diseases highlighted in this report:

- Prevalence—number or proportion of the population with the disease at a given time. The main source of prevalence data is the Australian Bureau of Statistics' 2001 National Health Survey.
- Mortality—number or rate of deaths caused by the disease over a specified time period, usually a calendar year. The source of mortality data is the AIHW's National Mortality Database.
- Activity restriction and disability—number or proportion of people with disability caused by the disease at a given time. The source of activity restriction and disability data is the Australian Bureau of Statistics' 2003 Survey of Disability, Ageing and Carers.
- Use of health care services—number, proportion or rate of general practitioner encounters, hospital separations and hospital procedures over a specified time period, usually a financial year. The source of general practitioner data is the AIHW's 2002–03 survey of general practice activity (BEACH survey); the source of hospital data is the AIHW's National Hospital Morbidity Database.

In this report, rates are expressed as either 'age-specific' (whereby the rate is limited to a specific age group) or 'age-standardised' (a statistical modification of the rate to conform to a particular age distribution, thereby minimising the effect of different age distributions in different populations). Information regarding the prevalence and consequence of some chronic respiratory diseases is incomplete. Furthermore, changes in disease classification and measurement affect the reliability of available information. It is therefore difficult to provide a full picture of the magnitude of the problem. Nevertheless, information has been compiled from existing sources to profile the six diseases that are the focus of this report (see Table 1).

Table 1: Data sources available for monitoring chronic respiratory diseases in Australia

Disease	Prevalence (up to 2001)	Mortality (up to 2003)	Disability (up to 2003)	GP encounters (up to 2002–03)	Hospital use (up to 2002–03)
COPD	(a)	✓	(a)	✓	✓
Asthma	✓	✓	✓	✓	✓
Bronchiectasis	not available	✓	not available	✓	✓
Pneumoconiosis	(b)	✓	not available	not available	✓
Chronic sinusitis	✓	✓	not available	✓	✓
Hay fever	✓	✓	not available	✓	✓

(a) Approximations based on data for emphysema and bronchitis.

(b) Approximation of incidence (new cases) based on National Occupational Health and Safety Commission data.

Chapter 5 contains a general discussion of the major risk factors for chronic respiratory diseases, focusing on diseases profiled in the report. The chapter highlights the role of tobacco smoking and environmental factors as risk factors common to several chronic respiratory diseases. Also discussed are known predisposing factors, such as inherited allergy.

Chapter 6 contains a general discussion of the prevention of chronic respiratory diseases in Australia. It includes a description of and evidence for the effectiveness of strategies known to reduce exposure to various risk factors, and interventions to prevent further worsening of the disease. The chapter concludes with a brief overview of the management of chronic respiratory diseases.

Chapter 7 contains the conclusions to the report.

2 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is a long-term lung disease marked by shortness of breath that initially occurs with exertion and becomes progressively worse over time. COPD is a major cause of mortality, illness and disability, making it a leading cause of disease burden in Australia. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines COPD as:

> a disease state characterised by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. (GOLD 2004, p. 6)

COPD most commonly arises from the gradual destruction of lung tissue due to the unopposed action of enzymes stimulated by inhaled irritants. This destruction of lung tissues, known as emphysema and mostly caused by tobacco smoking, makes the lungs floppy and less able to move air in and out, thereby limiting the ability of the lungs to exchange oxygen and carbon dioxide.

Some patients with COPD also have a persistent cough, producing a small amount of sputum each day. This condition, known as chronic bronchitis, is again caused mostly by tobacco smoking; hence, emphysema and chronic bronchitis often coexist. Some other diseases, such as chronic asthma, may also result in COPD by causing irreversible narrowing of the passages that allow air to move in and out of the lungs.

Tobacco smoking is overwhelmingly the strongest risk factor for COPD. The AIHW estimated that in 1998:

- about 70% of COPD in men was attributable to smoking
- about 60% of COPD in women was attributable to smoking
- about 90% of COPD among smokers (men and women) was attributable to smoking
- about 71% of deaths from COPD (74% for men and 65% for women) was attributable to smoking (AIHW: Ridolfo & Stevenson 2001).

Current smokers are up to 10 times more likely to have the disease than non-smokers. Passive smoking (exposure to environmental tobacco smoke) may also contribute to the development of COPD. Most, but not all, people with COPD have a history of tobacco smoking, suggesting that significant risks are associated with environmental and occupational factors. But only about 15% of smokers develop clinically recognised COPD (Kane & Graham 2004), suggesting that individual susceptibility plays a role in its development.

Environmental agents, including air pollutants, occupational dusts and chemicals, may contribute to the risk of developing COPD, either independently or in

conjunction with tobacco smoking. A small percentage of people with COPD have an inherited deficiency of the enzyme α-1-antitrypsin, which normally inhibits the action of destructive enzymes in the lungs.

There is no existing treatment that reverses the destruction of lung tissues underlying COPD. Cessation of smoking has been shown to slow the progression of the disease. Exercise-based rehabilitation programs improve the quality of life and exercise capacity of persons with COPD. For those with more severe forms of the disease, certain medications help improve exercise capacity and quality of life, and reduce the frequency of disease exacerbations.

Prevalence of chronic obstructive pulmonary disease

The prevalence of COPD in Australia is difficult to determine. First, there are major differences in how the disease is defined. Some definitions include asthmatic bronchitis while others include asthma (Rennard 1998). The only consensus is the inclusion of emphysema and chronic bronchitis. Second, the definition of COPD does not always match the available data, as many data sources do not distinguish between chronic and acute bronchitis. For example, the 2001 National Health Survey measured the self-reported prevalence of emphysema and of bronchitis as a long-term condition, but not of chronic bronchitis per se (estimates from the survey show a large prevalence of emphysema/bronchitis among children aged 0–14 years; a high proportion of these cases are likely to be acute bronchitis or other respiratory disorders). Therefore, the estimated prevalence of emphysema and bronchitis provided by the National Health Survey can only be used as a rough approximation of the prevalence of COPD in Australia.

Estimates from respondents' self-reports to the 2001 National Health Survey indicate that about 3.5% of the Australian population had emphysema or bronchitis (ABS 2002a). This compares to 3.0% in 1989 and 4.1% in 1995. Although these estimates would contain some cases of bronchitis that were not chronic in nature, there is still a likelihood that the true prevalence of COPD in Australia has been underestimated. This is because COPD is usually not diagnosed until it begins to restrict a person's lifestyle and is, by that time, moderately advanced. Estimates from the 2001 National Health Survey indicate that about 4.7% of the population aged 45–74 years had emphysema or bronchitis based on respondents' self-reports, with the prevalence rising to 10% by the age of 75 years and over (Figure 1). Relatively few people at younger ages report emphysema or bronchitis. The overall prevalence of COPD as estimated from the 2001 National Health Survey is slightly higher among females than males, although among the older age groups the reverse is true (ABS 2002a).

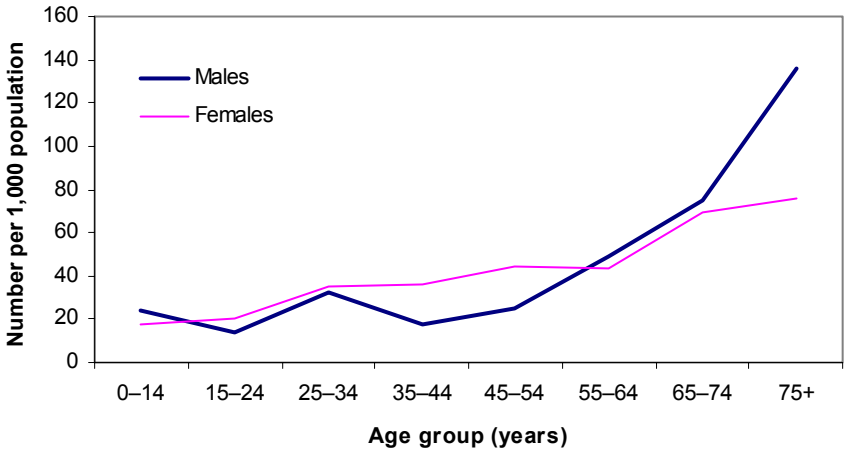


Figure 1: Prevalence of emphysema/bronchitis, by age and sex, 2001

Source: ABS 2001 National Health Survey.

Consequences of chronic obstructive pulmonary disease

The consequences of COPD for the health of the Australian population are best illustrated by the mortality attributed to it and by the degree of hospitalisation it causes (Box 1). COPD is a leading cause of death and hospitalisation in Australia. It was the underlying cause of 5,378 deaths in 2003 and led to over 53,000 hospital separations in 2002–03, with an average length of stay of 7.5 days.

Box 1: Consequences of COPD for the health of the Australian population

- *Mortality: COPD was the underlying cause of 5,378 deaths in 2003 (the 5th leading cause of death).*
- *Disability: About 12.3% of people reporting to have emphysema or bronchitis in 2003 had a severe or profound disability.*
- *General practice encounters: COPD is managed by general practitioners in less than 1% of encounters.*
- *Hospitalisation: COPD resulted in 53,566 hospital separations in 2002–03, with an average length of stay of 7.5 days.*
- *Health system expenditure: $433 million was spent on COPD in 2000–01.*

Mortality

COPD is a major cause of mortality, reflecting the end result of a progressive decline in lung function. It was the underlying cause of 5,378 deaths in 2003 (3,163 males and 2,215 females), representing 4.2% of all deaths in Australia that year.

Fact sheet 1 highlights aspects of COPD-associated mortality in 2003, in the previous year (2002), and in 1997. Two important facts about COPD mortality are that (1) the death rate is higher among males, with about 38 deaths per 100,000 males compared to about 19 deaths per 100,000 females in 2003, and (2) COPD deaths occur mostly among older people, in particular those aged 65 years and over. Appendix A shows the number of deaths attributed to COPD, as well as age-specific and age-standardised rates for deaths since 1997, by sex.

COPD is also listed commonly as a contributing or associated cause of death. In terms of how causes of death are registered in Australia, the condition that is believed to have initiated the train of events leading to death is referred to as the underlying cause. All other conditions listed on the death certificate—including the direct cause of death, other antecedents leading to the direct cause, and other conditions contributing to death—may be referred to as associated causes of death.

In addition to 5,378 times it was listed on death certificates as the underlying cause of death in 2003, COPD was listed 7,219 times as an associated cause of death. In almost half of the cases where COPD was listed as an associated cause, a circulatory disease was listed as the underlying cause of death. Besides chance, this may reflect the stress lung diseases can place on a compromised circulatory system, and vice versa, or the role of a common risk factor, tobacco smoking. About one in every eight deaths attributed to lung cancer in 2003 had COPD listed as an associated cause. Conversely, of the 5,378 deaths attributed to COPD, less than 100 had lung cancer listed as an associated cause of death.

Trends

The rate of male deaths from COPD increased markedly from the early 1950s until the early 1970s, but has been decreasing since the 1980s (Figure 2). In contrast, the female death rate has been increasing since the early 1960s, albeit it has plateaued in the last five years. Despite a marked decrease in the male death rate over the last two decades, it remains about double the female rate.

Reductions in male mortality from COPD probably reflect the decline in tobacco smoking rates in the last three decades and may also reflect better management of the disease. The increase in the female rate on the other hand could be due to the delayed effect of an increase in the proportion of female smokers since the late 1970s.

COPD is closely related to lung cancer in terms of having the same major risk factor (tobacco smoking) and similar trends in death rates. Since World War II, COPD has caused about 200,000 deaths in Australia compared to about 230,000 deaths by lung cancer. The trends have also been similar, except that the COPD death rate has had a slightly higher and earlier peak and a slightly more rapid decline than the lung cancer death rate (see Figure 9 in Chapter 5).

Fact sheet 1: COPD mortality

Item	Males	Females	Persons
Number of deaths in 2003	3,163	2,215	5,378
… in 2002	3,327	2,270	5,597
… in 1997	3,608	2,229	5,837
Age-standardised death rate in 2003	37.6	18.5	26.1
… in 2002	40.7	19.6	28.0
… in 1997	52.3	22.3	34.0
Position in top 10 causes of death in 2003	5th	7th	5th
… in 2002	5th	7th	5th
… in 1997	4th	6th	5th
Average age at death (in years) in 2003	77.7	78.3	77.9
… in 2002	77.4	77.8	77.6
… in 1997	76.3	76.7	76.4
Percentage 65 years of age and over in 2003	91.3	90.0	90.7
… in 2002	91.4	90.6	91.1
… in 1997	90.8	89.5	90.3
Percentage in major cities[(a)] in 2003	58.1	62.2	59.8
… in 2002	58.0	64.7	60.7
… in 1997	58.8	65.5	61.4
Percentage in outer regional or remote communities[(b)] in 2003	16.1	12.5	14.6
… in 2002	16.7	11.7	14.6
… in 1997	15.8	12.3	14.5
Percentage Indigenous[(c)] in 2003	2.2	4.5	3.1
… in 2002	3.8	2.5	3.3
… in 1997[(d)]	2.5	3.8	3.0
Total potential years of life lost[(e)] in 2003	7,605	5,405	13,010
… in 2002	8,133	5,840	13,973
… in 1997	10,455	6,823	17,278
Potential years of life lost per death in 2003	2.4	2.4	2.4
… in 2002	2.4	2.6	2.5
… in 1997	2.9	3.1	3.0
Number of deaths in which COPD was an associated cause in 2003	4,642	2,577	7,219
… in 2002	4,455	2,371	6,826
… in 1997	4,750	2,346	7,096
Most common associated cause of COPD deaths in 2003	Pneumonia	Respiratory failure	Respiratory failure
… in 2002	Respiratory failure	Respiratory failure	Respiratory failure
… in 1997	Respiratory failure	Respiratory failure	Respiratory failure

Note: Rates are per 100,000 population and are age-standardised to the Australian population at 30 June 2001.
(a) Usual residence a major city. For reference against deaths from all causes, percentage in major cities in 2003 = 62.9, in 2002 = 63.0, and in 1997 = 63.9.
(b) Usual residence an outer regional or remote community. For reference against deaths from all causes, percentage in outer regional and remote communities in 2003, 2002, and 1997 = 13.2.
(c) Includes data from WA, SA, and NT only. For reference against deaths from all causes in these 3 states, percentage Indigenous in 2003 = 3.7, in 2002 = 3.9, and in 1997 = 4.0.
(d) Indigenous data for 1997 should be interpreted with caution.
(e) Potential years of life lost is an indicator of premature mortality based on an arbitrary upper age limit of 75 years.

Source: AIHW National Mortality Database.

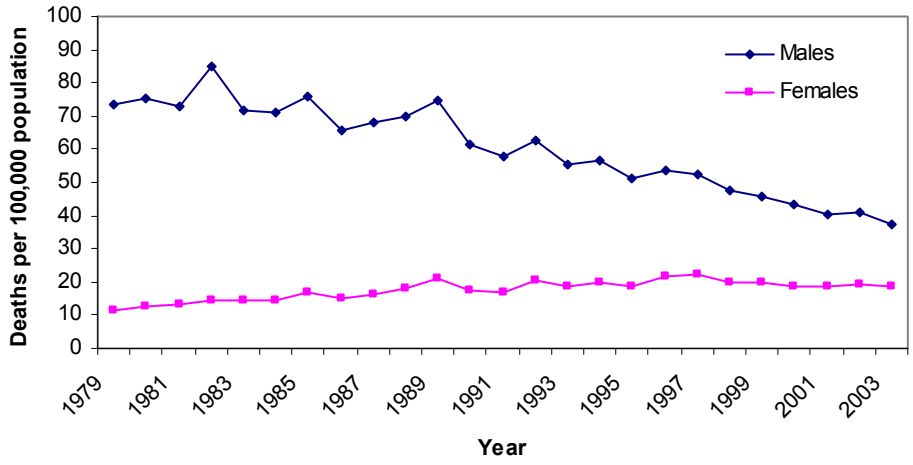


Figure 2: Trends in death rates for COPD, 1979–2003

Notes: Age-standardised to the Australian population at 30 June 2001; COPD classified according to ICD-9 codes 491, 492, 496 and ICD-10 codes J41 to J44; rates for 1979–96 have been adjusted by a factor of 0.93 to accommodate classification changes and be comparable with 1997–2003 rates.

Source: AIHW National Mortality Database.

Activity restriction and disability

The shortness of breath experienced by people with emphysema, bronchitis or other types of COPD can be quite disabling. It can interrupt daily activity, the ability to exercise and sleep patterns. Within seven to eight years of being diagnosed, most people with COPD become incapable of productive work.

Results from the 2003 Survey of Disability, Ageing and Carers indicate that about 34.1% of those reporting to have emphysema or bronchitis had a level of disability due to those diseases. Disability associated with emphysema or bronchitis was more than twice as common in males as in females. It was most prominent in the older age groups: about 68% of those reporting a level of disability associated with emphysema or bronchitis in 2003 were aged 65 years and over.

About 36.1% of people reporting a level of disability associated with bronchitis or emphysema had a severe or profound disability, that is, they sometimes or always needed personal assistance or supervision with one or more of the core activities. This represents about 12.3% of those reporting emphysema or bronchitis in the 2003 survey. 'Core activities' are defined as:

- self-care—bathing or showering, dressing, eating, using the toilet, and managing incontinence
- mobility—moving around at home and away from home, getting into or out of a bed or chair, and using public transport
- communication—understanding and being understood by others: strangers, family and friends.

Use of health care services

COPD is managed by general practitioners in less than 1% of encounters (AIHW: Britt et al. 2003). It is managed less often in general practice than asthma but accounts for considerably more hospitalisation, in terms of both the number of separations and the average length of time spent in hospital.

Hospitalisation data do not necessarily reflect the extent of a health problem in a community—they are more likely to reflect a combination of the nature, severity and prevalence of the disease as well as the availability of care (Loddenkemper et al. 2003). Hospital care may be required for COPD when symptoms increase and lead to increased disability, or become life threatening.

In 2002–03, COPD resulted in 53,566 hospital separations, with an average length of stay of 7.5 days. This is a significant increase in separations since 1997–98, but a reduction in the length of stay over the same period. Appendix A shows the number of separations with a principal diagnosis of COPD, as well as age-specific and age-standardised rates for separations since 1998–99, by sex.

Fact sheet 2 highlights aspects of hospitalisation caused by COPD in 2002–03, the previous year (2001–02), and in 1998–99. In 2002–03, people aged 65 years and over accounted for more than 75% of all COPD separations (that is, 41,836 separations). At these ages, the male hospital separation rate is much higher than the female rate. People in hospital with a principal diagnosis of COPD often receive allied health services, especially physiotherapy.

Health system expenditure

An analysis of health system expenditure (direct cost of health goods and services incurred by governments, non-government organisations and health service providers, see AIHW 2005b) shows that COPD accounted for $433 million in 2000–01.

Hospital use (admitted and non-admitted patients) was the largest expense, accounting for $273 million, or 63.1% of the total expenditure attributed to COPD. Pharmaceuticals (prescribed and over-the-counter) accounted for $85 million, or 19.6% of total expenditure. The greatest expense was associated with males aged 65–84 years: $163 million or 37.6% of total expenditure (see Table 2).

Table 2: Expenditure on COPD, by age and sex, 2000–01

	Expenditure ($ million)	
Age group (years)	**Males**	**Females**
0–24	6	6
25–54	15	20
55–64	38	29
65–74	81	57
75–84	82	56
85+	22	22
Total	**244**	**189**

Note: Numbers are rounded to the nearest million.

Source: AIHW Health System Expenditure Database.

Table 3 shows how the total expenditure attributed to COPD and the expenditure on COPD-related hospital use and pharmaceuticals compare with that of other major chronic diseases.

Table 3: Health expenditure in Australia for selected chronic diseases, 2000–01

	Expenditure ($ million)				
Disease	**Hospital use**	**Out of hospital[(a)]**	**Pharmaceuticals**	**Other[(b)]**	**Total**
Coronary heart disease	1,050	130	205	81	1,466
Osteoarthritis	567	125	148	344	1,184
Depression	240	276	340	251[(c)]	1,107
Stroke	360	27	31	476	894
Asthma	**170**	**110**	**370**	**43**	**693**
Type 2 diabetes	210	163	164	86	623
Kidney disease	451	12	7	13	483
COPD	**273**	**40**	**85**	**35**	**433**
Rheumatoid arthritis	68	36	47	95	246
Colorectal cancer	202	10	4	19	235
Osteoporosis	39	29	78	75	221
Lung cancer	107	5	8	16	136

(a) Includes general practitioners, imaging, pathology, etc.

(b) Includes aged care homes, allied health professionals, research.

(c) Includes community mental health.

Source: AIHW Health System Expenditure Database.

Fact sheet 2: COPD hospitalisation

Item	Males	Females	Persons
Number of hospital separations in 2002–03	30,532	23,034	53,566
… in 2001–02	29,631	21,989	51,620
… in 1998–99	27,703	19,232	46,935
Age-standardised hospital separation rate in 2002–03	344.3	210.2	265.7
… in 2001–02	343.6	205.7	262.6
… in 1998–99	349.2	194.0	258.3
Average length of stay (in days) in 2002–03	7.3	7.7	7.5
… in 2001–02	7.3	7.8	7.5
… in 1998–99	7.8	8.2	7.9
Average age at hospital separation (in years) in 2002–03	72.6	71.1	71.9
… in 2001–02	72.1	70.7	71.5
… in 1998–99	70.9	69.6	70.4
Percentage 65 years of age and over in 2002–03	80.8	74.6	78.1
… in 2001–02	79.6	74.0	77.2
… in 1998–99	78.1	72.4	75.8
Percentage under 5 years of age in 2002–03	0.1	0.2	0.1
… in 2001–02	0.2	0.2	0.2
… in 1998–99	0.3	0.4	0.3
Percentage of all hospital separations in 2002–03	1.0	0.6	0.8
… in 2001–02	1.0	0.6	0.8
… in 1998–99	1.0	0.6	0.8
Percentage Indigenous[(a)] in 2002–03	6.0	9.2	7.4
… in 2001–02	5.7	9.8	7.5
… in 1998–99	4.6	7.5	5.8
Most common procedure in 2002–03	Physiotherapy	Physiotherapy	Physiotherapy
… in 2001–02	Physiotherapy	Physiotherapy	Physiotherapy
… in 1998–99	Physiotherapy	Physiotherapy	Physiotherapy

Note: Rates are per 100,000 population and are age-standardised to the 2001 Australian population.

(a) Includes data from WA, SA, and NT only. For reference against separations for all conditions in these 3 states, percentage Indigenous in 2002–03 = 7.4, in 2001–02 = 7.2, and in 1998–99 = 7.0.

Source: AIHW National Hospital Morbidity Database.

3 Asthma

Asthma is a chronic disease marked by episodes of wheezing, chest tightness and shortness of breath associated with widespread narrowing of the airways within the lungs and obstruction of airflow. The underlying problem is chronic inflammation of the air passages, which also tend to overreact by narrowing too easily and too much in response to a wide range of 'triggers'. Common asthma triggers include:

- exercise
- viral infections
- allergens
- environmental irritants (including tobacco smoke, indoor and outdoor air pollutants, and occupational dusts and chemicals)
- food chemicals
- aspirin and other medications.

The symptoms of asthma are usually reversible, either spontaneously or with treatment. Many people with asthma, particularly those with more severe or persistent symptoms, are allergic to environmental allergens from dust mites, cockroaches, pollens, moulds and/or pets (especially cats and dogs). This may occur in association with eczema (particularly in young children) and hay fever.

A large proportion of asthma is developed in early childhood. For about 10% of the people who develop asthma in adulthood, the disease can be attributed to exposure to specific substances in the workplace. However, the cause of most cases of asthma is not known.

Asthma ranges in severity from mild, intermittent symptoms causing few problems for the individual to severe and persistent wheezing and shortness of breath, which severely impairs quality of life and may be life-threatening. Some young children with mild and occasional episodes of wheezing or cough, particularly those who are not allergic, have a self-limiting disease that resolves in later childhood.

Australia has a high prevalence of asthma, relative to other countries (Australian Centre for Asthma Monitoring 2003). The disease causes particular problems in children, for whom it is a frequent cause of visits to hospital emergency departments and admission to hospital, and older people, in whom the disease often overlaps with COPD. Asthma is also a common reason for visits to general practitioners and for use of medications. Hence, asthma is a substantial contributor to health care expenditure. However, it is not a major cause of death in Australia, with less than 320 deaths in 2003.

The fundamental causes of asthma remain unknown but many features of the disorder are becoming better understood. Asthma involves chronic inflammation of the airways, which makes them more sensitive (hyperresponsive) to allergens and

irritants. When stimulated, the smooth muscles of the airways contract, resulting in narrow airways and reduced airflow to lung tissues. This, along with blockage by airway wall secretions or fluid (oedema), causes symptoms such as wheezing, coughing, chest tightness and shortness of breath.

The major risk factors for asthma include a family history of asthma and a genetic predisposition for allergic reactions (atopy). Age and sex are also risk factors as males are more likely to have asthma until adolescence, after which asthma is more common among females. Environmental factors linked to the onset of asthma include maternal smoking during pregnancy, exposure to environmental tobacco smoke during infancy, exposure to sensitising agents and, less commonly, irritants in the workplace.

Prevalence of asthma

Estimates of the prevalence of asthma in Australia are usually made from self-report surveys. The 2001 National Health Survey asked respondents if they had been diagnosed with asthma and, if so, whether or not they still had asthma. According to responses to these questions, 11.6% of the Australian population was estimated to have asthma, including 13.9% of children aged under 18 years and 10.8% of adults aged 18 years and over (ABS 2002a). Age-specific prevalence rates for asthma in 2001 were highest in those aged less than 25 years (Figure 3). The National Health Survey time series indicates that the overall reported prevalence of asthma in Australia has risen from 7.8% in 1989–90 to 11.0% in 1995 and 11.6% in 2001.

A number of state-based surveys, using different methods to identify people with asthma, have also been conducted over the past decade. The self-reported prevalence of asthma is highly subject to definitional issues and survey methods. Studies vary in the type of questions asked and the size and selection of the sample, making it difficult to compare findings. Nevertheless, it has been estimated that between 14% and 16% of children and 10% to 12% of adults have asthma as a diagnosed and continuing condition in Australia (Australian Centre for Asthma Monitoring 2003).

Although inconclusive, evidence is beginning to point to a peaking of the rise in prevalence among children (Australian Centre for Asthma Monitoring 2003). The increased prevalence experienced in the 1980s and early 1990s may have been the result of several factors, including higher incidence, heightened awareness of the condition and improved diagnosis.

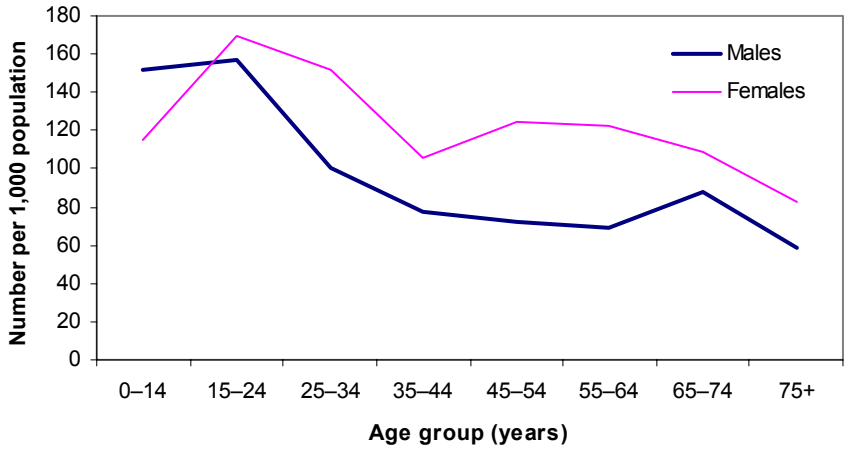


Figure 3: Prevalence of asthma, by age and sex, 2001

Source: ABS 2001 National Health Survey.

Consequences of asthma

The consequences of asthma in Australia are best illustrated by the observation that it is one of the major reasons for visiting a doctor, being the seventh most common problem managed by general practitioners in 2002–03, and is a major cause of health system expenditure (Box 2).

Box 2: Consequences of asthma for the health of the Australian population

- *Mortality: Asthma was the underlying cause of 314 deaths in 2003.*
- *General practice encounters: Asthma was managed in 2.7% of general practice encounters in 2002–03.*
- *Hospitalisation: Asthma was the principal diagnosis in 37,230 hospital separations in 2002–03, with an average length of stay of 2.5 days.*
- *Health system expenditure: $693 million was spent on asthma in 2000–01.*

Mortality

Asthma is not a common cause of death in Australia. In 2003, it was identified as the underlying cause of 314 deaths (108 males and 206 females). Appendix A shows the number of deaths attributed to asthma, as well as age-specific and age-standardised rates for asthma deaths since 1997, by sex. Fact sheet 3 highlights aspects of asthma mortality in 2003, in the previous year (2002), and in 1997.

Deaths attributed to asthma increase with age, with very few deaths in childhood. Despite high prevalence and hospital use, the death rates among children aged 0–4, 5–9 and 10–14 years were less than 1.0 death per 100,000 persons in 2003. The rate remains low during early and middle adult life but increases markedly after the age of 50 years, peaking at 32 deaths per 100,000 persons in the 85 years and over age group. However, deaths from asthma among older people are often complicated by the presence of COPD; hence, attributing the actual cause of death in this group may be problematic. For this reason, Fact sheet 3 shows the proportion of deaths among those aged 5–34 years, in whom the attribution of death to asthma is most certain (Australian Centre for Asthma Monitoring 2003). Nevertheless, those whose underlying cause of death was attributed to asthma died on average about 10 years younger than those whose underlying cause of death was attributed to COPD.

In addition to the 314 deaths in 2003 where asthma was registered as the underlying cause, asthma was registered 934 times as an associated cause of death (i.e., one of the other conditions listed on the death certificate, including the direct cause, conditions related to the direct cause, and conditions otherwise contributing to the death).

Trends

The asthma death rate increased from 1979, peaking at about 5.0 deaths per 100,000 persons in 1989 (Figure 4). Since 1989, the death rate has decreased markedly to under 2.0 deaths per 100,000 persons. At ages less than 55 years, the death rates were similar for both sexes.

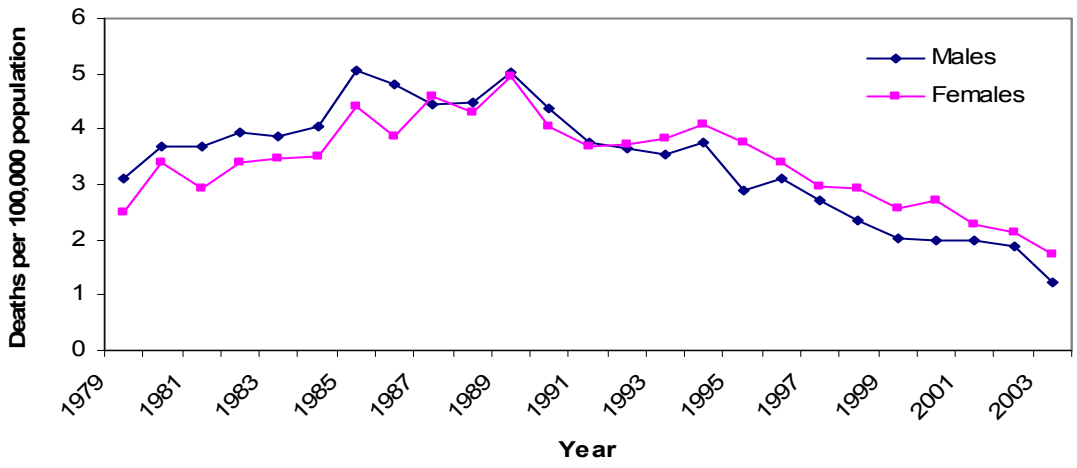


Figure 4: Trends in death rates for asthma, 1979–2003

Notes: Age-standardised to the Australian population at 30 June 2001; asthma classified according to ICD-9 code 493 and ICD-10 codes J45 and J46; rates for 1979–96 have been adjusted by a factor of 0.75 to accommodate classification changes and be comparable with 1997–2003 rates.

Source: AIHW National Mortality Database.

Fact sheet 3: Asthma mortality

Item	Males	Females	Persons
Number of deaths in 2003	108	206	314
… in 2002	158	239	397
… in 1997	207	292	499
Age-standardised death rate in 2003	1.2	1.8	1.5
… in 2002	1.9	2.1	2.0
… in 1997	2.7	3.0	2.9
Average age at death (in years) in 2003	61.5	71.6	68.1
… in 2002	66.4	68.8	67.8
… in 1997	61.8	68.8	65.9
Percentage 65 years of age and over in 2003	52.8	70.4	64.3
… in 2002	62.0	64.4	63.5
… in 1997	55.1	69.2	63.3
Percentage between 5 and 34 years of age in 2003	17.6	5.8	9.9
… in 2002	10.1	7.1	8.3
… in 1997	13.0	7.5	9.8
Percentage in major cities[(a)] in 2003	57.5	62.8	61.0
… in 2002	57.4	61.8	60.0
… in 1997	61.1	63.2	62.3
Percentage in outer regional or remote communities[(b)] in 2003	15.8	12.2	13.4
… in 2002	12.3	10.2	11.0
… in 1997	17.4	14.5	15.7
Percentage Indigenous[(c)] in 2003	7.7	9.8	9.3
… in 2002	10.3	10.5	10.4
… in 1997[(d)]	0	5.0	2.7
Total potential years of life lost[(e)] in 2003	1,820	1,815	3,635
… in 2002	2,025	2,550	4,575
… in 1997	3,250	3,105	6,355
Potential years of life lost per death in 2003	16.9	8.8	11.6
… in 2002	12.8	10.7	11.5
… in 1997	15.7	10.6	12.7
Number of deaths in which asthma was an associated cause in 2003	343	591	934
… in 2002	413	615	1,028
… in 1997	433	566	999
Most common associated cause of asthma deaths in 2003	Respiratory failure	Pneumonia	Pneumonia
… in 2002	Pneumonia	Respiratory failure	Pneumonia
… in 1997	Respiratory arrest	Respiratory failure	Respiratory failure

Note: Rates are per 100,000 population and are age-standardised to the Australian population at 30 June 2001.
(a) Usual residence a major city. For reference against deaths from all causes, percentage in major cities in 2003 = 62.9, in 2002 = 63.0, and in 1997 = 63.9.
(b) Usual residence an outer regional or remote community. For reference against deaths from all causes, percentage in outer regional and remote communities in 2003, 2002, and 1997 = 13.2.
(c) Includes data from WA, SA, and NT only. For reference against deaths from all causes in these 3 states, percentage Indigenous in 2003 = 3.7, in 2002 = 3.9, and in 1997 = 4.0.
(d) Indigenous data for 1997 should be interpreted with caution.
(e) Potential years of life lost is an indicator of premature mortality based on an arbitrary upper age limit of 75 years.

Source: AIHW National Mortality Database.

Activity restriction and disability

Asthma can result in time off work or school and some restriction of participation in physical and social activities. Results from the 2003 Survey of Disability, Ageing and Carers indicate that about 8.2% of those reporting to have asthma had a level of disability associated with asthma. Disability associated with asthma was reported about equally by males and females, although among males it was particularly marked in the younger age groups. Among people with asthma as the main disabling condition, about 24.8% had a severe or profound disability, meaning that they sometimes or always needed personal assistance or supervision with one or more of the core activities (self-care, mobility and communication). This group represents about 2.0% of those reporting asthma in the 2003 survey.

Use of health care services

Asthma is commonly managed in general practice. A survey of general practice activity indicates that asthma was managed in 2.7% of general practice encounters in 2002–03, making it the seventh most common problem managed (AIHW: Britt et al. 2003). General practitioners are usually the first point of contact for asthma management and also provide support and ongoing care. General practitioners may also review the asthma status of patients visiting for other conditions.

Acute or reactive management of asthma (for severe exacerbations or increased symptoms) often occurs in hospital emergency departments. However, it should be noted that the diagnosis of asthma is more problematic in very young children (0–4 years) and in adults aged over 50 years. In both these age groups, other breathing disorders may be difficult to distinguish from asthma. For this reason, hospital separation data in these age ranges should be treated with caution. Appendix A shows the number of separations with a principal diagnosis of asthma, as well as age-specific and age-standardised rates for separations since 1998–99, by sex. Fact sheet 4 highlights aspects of hospitalisation for asthma in 2002–03, the previous year (2001–02), and in 1998–99.

Asthma was the principal diagnosis in 37,230 hospital separations in 2002–03 (0.6% of all separations) with an average length of stay of 2.5 days. It accounted for about 12.5% of hospital separations for diseases of the respiratory system. The number of separations was about 16,000 fewer than that for COPD, and the average length of stay was one-third of that for COPD. However, the number of separations for asthma has decreased since 1997–98 (Fact sheet 4), in contrast to a rise in COPD separations over the same period (see Fact sheet 2).

Asthma is one of the most frequent reasons for hospitalisation among children aged 0–14 years, especially boys. The hospital separation rate for asthma is highest in early childhood (0–4 years), at 1,219 per 100,000 boys and 680 per 100,000 girls in 2002–03.

Fact sheet 4: Asthma hospitalisation

Item	Males	Females	Persons
Number of hospital separations in 2002–03	17,686	19,544	37,230
… in 2001–02	19,762	21,256	41,018
… in 1998–99	26,272	27,635	53,907
Age-standardised hospital separation rate in 2002–03	179.9	197.9	190.2
… in 2001–02	201.0	217.1	210.7
… in 1998–99	271.7	291.9	283.4
Average length of stay (in days) in 2002–03	2.0	2.9	2.5
… in 2001–02	2.0	3.0	2.5
… in 1998–99	2.2	3.2	2.7
Average age at hospital separation (in years) in 2002–03	16.4	32.2	24.7
… in 2001–02	16.8	31.9	24.6
… in 1998–99	15.8	29.4	22.8
Percentage 65 years of age and over in 2002–03	5.9	15.0	10.7
… in 2001–02	5.9	14.3	10.3
… in 1998–99	5.5	12.7	9.2
Percentage under 5 years of age in 2002–03	44.9	21.5	32.6
… in 2001–02	42.6	21.2	31.5
… in 1998–99	42.8	21.6	31.9
Percentage of all hospital separations in 2002–03	0.6	0.6	0.6
… in 2001–02	0.7	0.6	0.6
… in 1998–99	1.0	0.9	0.9
Percentage Indigenous[(a)] in 2002–03	7.5	11.8	9.7
… in 2001–02	6.6	10.8	8.8
… in 1998–99	7.6	9.8	8.7
Most common procedure in 2002–03	Physiotherapy	Physiotherapy	Physiotherapy
… in 2001–02	Physiotherapy	Physiotherapy	Physiotherapy
… in 1998–99	Respiratory medication administered by nebuliser	Physiotherapy	Physiotherapy

Note: Rates are per 100,000 population and are age-standardised to the 2001 Australian population.

(a) Includes data from WA, SA, and NT only. For reference against separations for all conditions in these 3 states, percentage Indigenous in 2002–03 = 7.4, in 2001–02 = 7.2, and in 1998–99 = 7.0.

Source: AIHW National Hospital Morbidity Database.

Health system expenditure

An analysis of health system expenditure (direct cost of health goods and services incurred by governments, non-government organisations and health service providers, see AIHW 2005b) shows that asthma accounted for $693 million in 2000–01. Pharmaceuticals (prescribed and over-the-counter) were the largest expense, accounting for $370 million, or 53.4% of the total expenditure attributed to asthma. Hospital use (admitted and non-admitted patients) accounted for $170 million, or 24.5% of total expenditure.

The greatest expense was associated with males aged 0–14 years: $133 million or 19% of total expenditure (see Table 4). Of note is that from the age of 15 years greater expense is associated with females than males.

Table 4: Expenditure on asthma, by age and sex, 2000–01

	Expenditure ($ million)	
Age group (years)	**Males**	**Females**
0–14	133	94
15–24	36	43
25–54	71	124
55–64	24	46
65–74	27	37
75–84	16	26
85+	5	10
Total	**313**	**380**

Note: Numbers are rounded to the nearest million.

Source: AIHW Health System Expenditure Database.

Table 3 (Chapter 2) shows how the total expenditure attributed to asthma and the expenditure on asthma-related hospital use and pharmaceuticals compare with that of other major chronic diseases.

4 Other chronic respiratory diseases

This chapter provides brief profiles of four chronic respiratory diseases, namely bronchiectasis, pneumoconiosis, chronic sinusitis and hay fever. Bronchiectasis and pneumoconiosis are not common diseases and therefore do not cause much population morbidity and mortality. They are noteworthy, however, for the degree of discomfort they can cause an individual. Chronic sinusitis and hay fever, on the other hand, are highly prevalent and compromise the quality of life of many Australians.

Bronchiectasis

Bronchiectasis is an abnormal and irreversible dilation and distortion of the airways. The disorder is characterised by chronic cough with large amount of strong-smelling sputum, recurrent respiratory tract infections with slight to massive coughing up of blood, and chronic airflow obstruction.

Bronchiectasis is most often acquired as a result of a chronic or recurrent infection of the airways, especially in childhood, or from a severe bacterial infection such as pneumonia (associated with whooping cough or measles) or tuberculosis (Swartz 1998; Venes & Thomas 2001). It is also acquired in a number of other ways, including congenital abnormalities, ciliary dysfunction syndromes, or as a result of having a foreign body, such as a peanut, lodged in the airways.

Prevalence and consequences of bronchiectasis

The consequence of bronchiectasis is best illustrated by the average length of stay for people who are hospitalised with the disease. Bronchiectasis was the principal diagnosis in 3,120 hospital separations in 2002–03, with an average length of stay of 7.2 days. Although bronchiectasis results in far fewer hospital separations, the average length of stay compares with COPD (7.5 days).

Females are hospitalised for bronchiectasis at twice the male rate (age-standardised rate of 20.0 separations per 100,000 females in 2002–03 compared to 10.7 separations per 100,000 males). The hospital separation rate rises steeply after the age of 40 years, especially for females (Figure 5). People hospitalised with the principal diagnosis of bronchiectasis mostly receive a generalised allied health service, particularly from physiotherapists. Bronchiectasis was the underlying cause of death of 79 males and 158 females in 2003. Most of these deaths occurred in people aged 65 years and over, the average age of death being about 75 years. The reasons for sex differences in mortality and hospital separation rates are unclear.

There is little information available on the prevalence of bronchiectasis and the disability it causes. The survey of general practice activity reported few occasions in 2002–03 in which bronchiectasis was managed by general practitioners, accounting for an estimated 0.1% of encounters and 0.04% of all problems managed (AIHW: Britt et al. 2003).

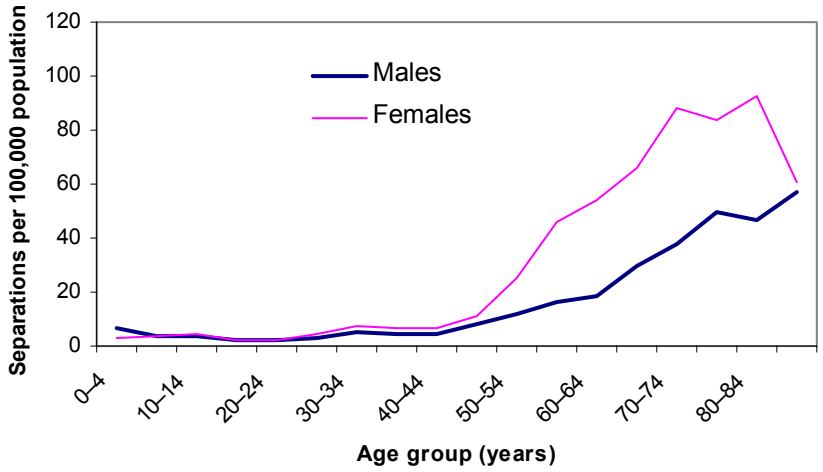


Figure 5: Hospital separation rates for bronchiectasis, by age and sex, 2002–03

Source: AIHW National Hospital Morbidity Database.

Pneumoconiosis

Pneumoconiosis is the non-cancerous reaction, and resultant structural alteration, of the lungs to inhaled mineral or organic dusts or fibres. It is almost totally attributable to exposure to dusts and fibres in the workplace. In certain occupations, prolonged exposure to dusts, fibres, fumes or other noxious substances can trigger an inflammatory response in lung tissues and may lead to diffuse scarring (pulmonary fibrosis). Coal workers' pneumoconiosis, asbestosis and silicosis are important types of pneumoconiosis. The main symptom of pneumoconiosis is breathlessness on exertion, although there are often no symptoms in the early stages. The disease can be highly disabling and shortens life expectancy.

Prevalence and consequences of pneumoconiosis

The overall morbidity due to pneumoconiosis in Australia is difficult to estimate. No national data are available on the number of people who have the disease, or on the activity restriction and disability of people suffering from pneumoconiosis. The number of general practice encounters for pneumoconiosis is also too small to be determined through surveys. The National Occupational Health and Safety Commission maintains a national workers' compensation statistics database, based

on the National Data Set for Compensation-based Statistics. This database contains information on new cases of compensable work-related injury and disease, thereby providing some estimation of the incidence of such diseases. Between 2001 and 2003, approximately 750 new cases of pneumoconiosis were reported.

Pneumoconiosis is not a large contributor to mortality in Australia. There were 92 deaths in 2003 where pneumoconiosis was identified as the underlying cause of death. All but two of these deaths were of males, probably reflecting the male-dominated nature of the occupations, such as mining and manufacturing, in which workers are mostly exposed to dusts and fibres. The average age at death by pneumoconiosis in 2003 was 77 years.

The pneumoconiosis death rate has fallen sharply since the early 1950s when the male age-standardised rate peaked at 3.9 deaths per 100,000 population. For the last quarter-century, the death rate has been less than or about one per 100,000 population (Figure 6). The sharp decline and eventual levelling of the death rate is probably due to decreased exposure to hazardous dusts and fibres in the workplace (as a result of legislation, and improved technology and occupational health practices) and to the increased time lag between exposure and death.

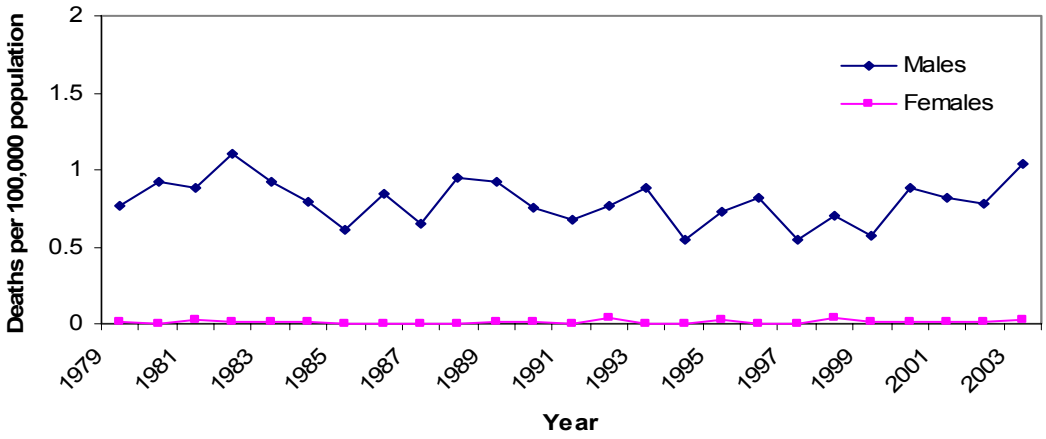


Figure 6: Trends in death rates for pneumoconiosis, 1979–2003

Note: Age-standardised to the Australian population at 30 June 2001; pneumoconiosis classified according to ICD-9 codes 500 to 505 and ICD-10 codes J60 to J64.

Source: AIHW National Mortality Database.

Chronic sinusitis

Sinusitis is the inflammation of the lining of one or more of the sinuses, which are the air-filled spaces within the bones around the nose. It results from an allergy, or a viral or bacterial infection. When sinusitis recurs frequently, or lasts for a prolonged period of time, it is classified as chronic. The typical symptoms of chronic sinusitis include pressure-like pain on the forehead, temples, cheeks, nose or around the eyes;

difficulty breathing through the nose; nasal drainage (thick yellow or yellow-green); and reduced sense of smell or taste.

Factors that may predispose an individual to developing chronic sinusitis include inadequate drainage of the sinuses, which may result from polyps; enlarged nose bone (turbinate); deviated septum; hay fever; and abscess in the upper jaw.

Prevalence and consequences of chronic sinusitis

Chronic sinusitis is highly prevalent in the community. Estimates from the National Health Survey indicate that, based on respondents' self-reports, over 2 million people in Australia (10.5% of the population) had chronic sinusitis in 2001. This makes chronic sinusitis one of the most frequently reported health conditions in Australia, comparable to asthma. The prevalence was higher among females (12.1%) than males (8.8%), peaking among those aged 45–54 years (Figure 7).

Very few deaths in Australia are attributed to chronic sinusitis, and there are no national data available on activity restriction or disability due to chronic sinusitis. In general practice, management of sinusitis includes both chronic and acute sinusitis. According to the survey of general practice activity, sinusitis was managed in 1.3% of general practice encounters in 2002–03 (AIHW: Britt et al. 2003), about half the rate of encounters for asthma.

Chronic sinusitis is often mild and self-limiting, but it can result in hospitalisation. In 2002–03, chronic sinusitis was the principal diagnosis for 10,617 hospital separations, with an average length of stay of 1.4 days. Hospital separation rates for chronic sinusitis increase with age before peaking at 60–64 years, after which the rate declines sharply.

Hospital procedures for chronic sinusitis include surgery, such as ethmoidectomy and maxillary antrostomy, and diagnostic procedures such as sinoscopy and nasendoscopy. Ethmoidectomy is the surgical drainage of the sinus cavity between the eyes and was performed on 5,212 occasions in 2002–03. Maxillary antrostomy is the surgical creation of a hole in the wall between the nasal passages and the sinus cavity in the cheekbone. It was performed on 5,608 occasions in 2002–03.

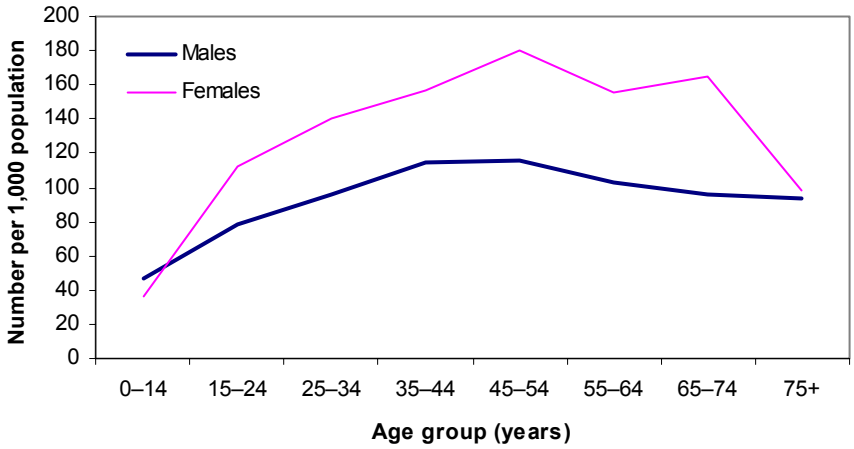


Figure 7: Prevalence of chronic sinusitis, by age and sex, 2001

Source: ABS 2001 National Health Survey.

Hay fever (allergic rhinitis)

Hay fever is an inflammation or irritation of the mucous membranes of the nose caused by an allergic reaction. The common allergens may include airborne particles of dust, pet dander and plant pollens. Untreated hay fever can have a significant impact on quality of life, mood, work performance, and other medical conditions including asthma and sinusitis.

The term 'hay fever' is often used to describe allergic rhinitis when it is caused by pollen. In this context, the disorder is also often referred to as seasonal allergic rhinitis to reflect the seasonality of the responsible pollens. In contrast, perennial allergic rhinitis may occur any time of the year and is usually due to other allergens such as house dust. In this report, hay fever is used synonymously with allergic rhinitis, both seasonal and perennial.

Hay fever results in a well-known combination of symptoms affecting the eyes and nasal passages. The common characteristics of the disease include nasal congestion, frequent sneezing, itchy and runny nose, itchy and watery eyes, and sore throat. The symptoms of hay fever occur in people who have a predisposition to allergies. That is, their immune system reacts to substances (allergens) that do not affect most people. This predisposition may be inherited or caused by sensitisation to a substance in the environment. The most common trigger for hay fever symptoms is pollen from various grasses, trees and flowers. House dust, dust mites, mould and animal dander are also common hay fever triggers.

Prevalence and consequences of hay fever

Hay fever is highly prevalent in Australia. Estimates from the National Health Survey indicate that, based on respondents' self-reports, about 2.9 million people (15.6% of the population) had hay fever as a long-term condition in 2001. This is more than the 2.2 million asthma sufferers estimated from that survey. Hay fever is slightly more common among females (16.0%) than males (15.2%), and in both sexes the prevalence is highest among those aged 25–34 years (Figure 8).

No national data are available on activity restriction or disability due to hay fever. In contrast to its high prevalence, it is not a common problem encountered in general practice—it was managed in less than 1% of general practice encounters in 2002–03 (AIHW: Britt et al. 2003). Hay fever and sinusitis often coexist and their diagnoses often overlap.

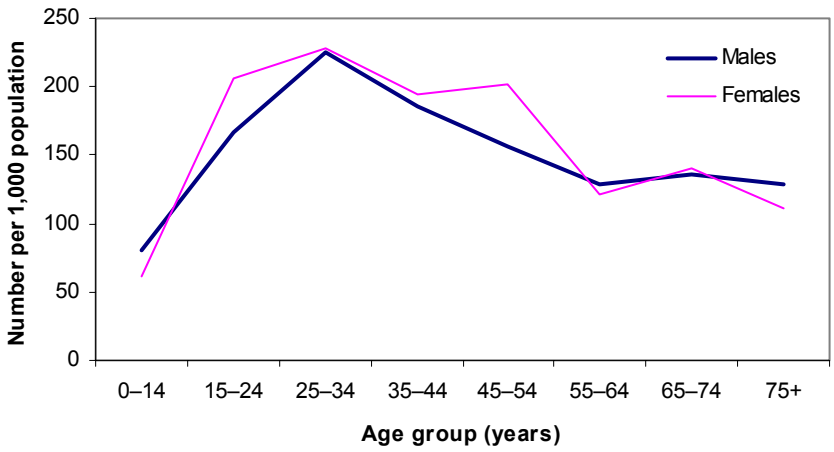


Figure 8: Prevalence of hay fever, by age and sex, 2001

Source: ABS 2001 National Health Survey.

5 Risk factors

A risk factor increases the risk of a health disorder or other unwanted condition or event. A variety of risk factors contribute to the development of chronic respiratory diseases; several others are known to exacerbate an existing condition. Chronic respiratory diseases share a number of common risk factors, but the level of 'risk' varies according to the individual disease.

Tobacco smoke is the most important preventable risk factor for chronic respiratory diseases. In particular, smoking greatly increases the risk of developing COPD. For people with asthma and COPD, exposure to environmental tobacco smoke ('passive smoking') can aggravate the symptoms.

This chapter describes the major risk factors for chronic respiratory diseases: tobacco smoking, environmental factors (including food and drugs), infectious diseases and predisposing factors. The information provided is largely expository in nature and emphasises those risk factors that are likely to be the target of monitoring or preventive strategies. Predisposing factors are also described, to complete the picture of the gene–environment relationships underlying various chronic respiratory diseases.

Tobacco smoking

Tobacco smoking is well established as the single largest preventable cause of chronic respiratory diseases. It is the single most preventable cause of lung cancer and COPD, responsible for 80% to 90% of cases. Tobacco smoke contains thousands of chemicals, many of which interact. These chemicals produce a range of health effects, especially when combined with other genetic, behavioural and environmental factors. Tobacco smoke is particularly harmful to the respiratory system but also increases the risk of coronary heart disease, stroke and peripheral vascular disease as well as a range of cancers, including those that affect the mouth, oesophagus, kidney, pancreas and cervix (AIHW: Ridolfo & Stevenson 2001).

The majority of people with COPD have a long history of tobacco smoking. In Australia in 1998, smoking accounted for about 70% of COPD in men and about 60% of COPD in women (AIHW: Ridolfo & Stevenson 2001). Among smokers, about 90% of COPD was attributable to smoking (AIHW: Ridolfo & Stevenson 2001). Smoking accelerates the development of COPD and increases its severity.

The role of smoking in the onset of asthma is not clear. It has been suggested that adults who smoke are more sensitive to other agents known to induce asthma because they have higher total levels of immunoglobulin E (IgE), an antibody that plays a major role in allergic reactions. In general, smokers who have asthma tend to have more symptoms and worse asthma control (Siroux et al. 2000).

Mainstream and environmental tobacco smoke

Mainstream smoke is the smoke drawn through the cigarette by the smoker. It contains chemical substances such as nicotine, tar, carbon monoxide, carbon dioxide, sulphur dioxide and nitrogen dioxide. These substances can cause chronic cough, production of sputum, and emphysema. Mainstream smoke is the major cause of COPD.

Environmental tobacco smoke (ETS) is a combination of exhaled mainstream smoke and sidestream smoke (smoke from the burning end of a cigarette). It contains basically the same carcinogens and toxic agents that are inhaled directly by smokers, although the deposition of smoke in the respiratory system is quite different between those who inhale mainstream smoke and those who only inhale ETS due to the patterns of breathing and concentration of compounds (NHMRC 1997; Redhead & Rowberg 1995).

Environmental tobacco smoke can lead to serious health consequences for both adults and children. In adults, exposure to ETS can increase coughs, chest discomfort and sputum production, and decrease lung function (US Environmental Protection Agency 1999). About 8% of childhood asthma has been attributed to ETS (NHMRC 1997). Exposure to ETS can also exacerbate existing asthma in children and increase the chance of developing other chronic respiratory diseases and impaired lung function (US Environmental Protection Agency 1999).

Health benefits of quitting smoking

Smoking cessation brings great health benefits. The relative risks of developing chronic respiratory diseases—as well as lung cancer, coronary heart disease and stroke—decrease gradually after quitting. However, there will often be a degree of irreversible lung damage in smokers; lung function is unlikely to return to completely normal after quitting. Nevertheless, quitting usually has a beneficial effect on lung function as the rate of decline in lung function is slower among former smokers compared with current smokers (US Department of Health and Human Services 1990). Recent evidence shows that quitting before the age of 35 is of greater benefit than quitting at a later age, but there are substantial benefits regardless of age.

Prevalence of smoking in Australia

Latest information on the prevalence of tobacco smoking in Australia has been gathered by the 2001 National Health Survey (ABS 2002a) and the 2004 National Drug Strategy Household Survey (AIHW 2005a). In 2004, 17.4% of Australians aged 14 years and over were daily smokers (Table 5). Furthermore, about 26.4% of the population aged 14 years and over were ex-smokers while almost 53% claimed to have never smoked (Table 6).

The prevalence of smoking is higher among people aged between 20 and 49 years than among teenagers or the elderly, and is higher among males than females, except among those aged between 14 and 19 years (AIHW 2005a). Smoking is more common

among people from lower than from higher socioeconomic backgrounds and is more than twice as likely among Indigenous people than non-Indigenous people (ABS 2002a). These variations in tobacco smoking are reflected in the prevalence of chronic respiratory diseases (Glover et al. 2004).

Table 5: Prevalence of daily tobacco smoking in Australia, persons aged 14 years and over, 2004

	Per cent		
Age group	**Males**	**Females**	**Persons**
14–19 years	9.5	11.9	10.7
14 years and over	18.6	16.3	17.4

Source: AIHW (2005a).

Exposure to tobacco smoke in the home is common. In 2001, approximately 36% of Australians lived in a household where at least one person smoked (ABS 2002a). The exposure to ETS in the workplace is estimated to be equivalent to or greater than the exposure in the home (NHMRC 1997). All states and territories have now introduced or amended legislation on ETS and, consequently, exposure levels outside the home may be reduced.

Although smoking remains a serious public health problem, the prevalence of smoking in Australia has been declining steadily over the last three decades. Between 1995 and 2004 alone, the proportion of daily smokers aged 14 years and over has fallen by about 6.4 percentage points (Table 6). The steady decline in the proportion of daily smokers over the last decade appears to have been associated with a substantial rise in the proportion of ex-smokers between 1995 and 1998 followed by a steady rise in the proportion of people who have never smoked.

Table 6: Trends in tobacco smoking, persons aged 14 years and over, 1995 to 2004

	Per cent			
Smoking status	**1995**	**1998**	**2001**	**2004**
Daily smokers	23.8	21.8	19.5	17.4
Less-than-daily smokers	3.4	3.1	3.6	3.2
Ex-smokers	20.2	25.9	26.2	26.4
Never smoked	52.6	49.2	50.6	52.9

Source: AIHW (2005a).

Trends in tobacco consumption in Australia are closely reflected in respiratory disease mortality (Figure 9). The impact can be seen in both the increases and decline in COPD and lung cancer death rates among males, given a time lag of about 20 years between tobacco consumption and disease manifestation. Female death rates for these diseases have been much lower, because of lower smoking rates, but are yet to show sustained declines.

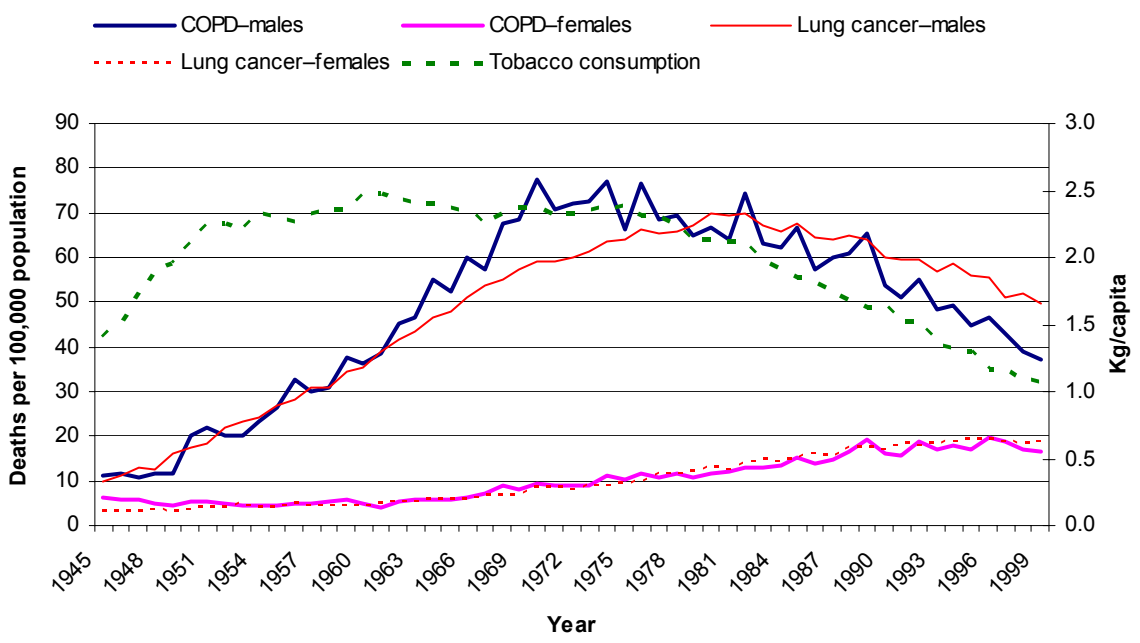


Figure 9: Tobacco consumption and death rates for COPD and lung cancer, 1945 to 1999

Notes: Death rates age-standardised to the Australian population at 30 June 1991; tobacco consumption is given in kilograms per capita; the basis for calculating consumption was changed in November 1999 from a weight to a per stick basis.

Sources: AIHW National Mortality Database and VicHealth Centre for Tobacco Control.

The death rate for COPD among males peaked about a decade before the lung cancer death rate. This probably reflects the respective lengths of time required for the diseases to develop and lead to death. Symptoms of obstructive lung disease often appear before cancer develops. On the other hand, the beneficial impact of quitting tobacco smoking appears to be more immediate for COPD than for lung cancer. People with relatively mild impairment of lung function generally show improvements in the first year after quitting smoking, whereas people who quit smoking take about 10 to 15 years to lower their risk of lung cancer to that of a non-smoker (Rennard & Daughton 1998).

Environmental factors

The role of environmental factors in chronic respiratory diseases is complex. It involves the interrelationship of numerous factors such as genes, underlying diseases, personal exposures, behaviour, dietary patterns, home environment, geographic location and weather patterns. Some environmental factors play a clear causal or sensitising role, such as pollen with hay fever. The role of environmental factors in the causation of asthma on the other hand remains largely uncertain. Nevertheless, a large number of environmental factors are known to exacerbate pre-existing respiratory conditions by inducing inflammation and/or acute broncho-constriction. They act as triggers for episodes of the disease once the individual has

been sensitised to an allergen or irritant. In asthma, triggers include exposure to allergens, indoor pollutants (for example, tobacco smoke), ambient air pollutants (for example, sulphur dioxide, nitrogen dioxide and inhalable particles), cold air, weather changes, physical exertion and extreme emotional expression.

The amount of damage caused by an environmental agent depends on several factors, including the degree of exposure, extent to which it penetrates the respiratory system, the length of time retained in the body, the rate at which it is cleared by the body's natural defences and individual characteristics of the person exposed to it.

Indoor allergens and air pollutants

Most people in developed countries, especially children, spend most of their time indoors. Each dwelling's internal environment is unique, depending on its location, age, design, constituent and content materials, and the number and activities of the inhabitants. Other factors, such as lifestyle, climate and proximity to pollution sources will affect how much pollution enters the dwelling from the outdoor (ambient) environment. Hence each house will contain a number of pollutants that, to varying degrees, may act as allergens, irritants or toxins.

Major indoor air pollutants include biologicals (dust mites, pet dander, cockroaches, fungi, bacteria), combustion by-products (nitrogen and sulphur compounds, fine inhalable particles) and volatile organic compounds (for example, formaldehyde). Once an individual's air passages are sensitised, exposure to inhalable indoor allergens may trigger or exacerbate chronic respiratory diseases, particularly asthma (Ng 2000).

House dust and dust mites

House dust is a cocktail of numerous organic and inorganic compounds, including hair, smoke, dirt, fibres, mould spores, pollen grains, insects, mites, mammalian danders (small scales from the skin or hair), and the secreta (for example, saliva) and excreta (for example, faeces) of insects, mites and pets. A number of these constituents can act as allergens for asthma and hay fever. However, the most likely sources of dust-induced allergy are the bodies, secreta and excreta of house dust mites.

Mites are tiny arachnids that feed on shed human skin cells. They are most often found in older houses with carpeted floors, as well as mattresses and soft furnishings (Mihrshahi et al. 2002). Allergy to house dust mite allergen (identified as the protein Der p 1) is a common risk factor for asthma in Australia and worldwide, particularly when the exposure occurs at an early age (Rutherford & Eigeland 2000).

Domestic pet allergens

The secreta, excreta and dander of domestic pets are allergens that can trigger asthma and hay fever episodes in sensitised people. Although sensitivity to dog allergen does occur, cat allergen (Fel d 1) is widely regarded as a particularly strong allergen

(Duffy et al. 1998). Cat allergen is produced in sebaceous glands and may be deposited on the epidermis and along hair shafts.

Cockroach allergen

Cockroach allergens (Bla g I and Bla g II) have been associated with airway inflammation (Papouchado et al. 2001). House dust may contain the allergens from at least two species of cockroaches found commonly in Australia, the American cockroach (*Periplaneta americana*) and the German cockroach (*Blatella germanica*). The allergens are found in cockroach droppings and saliva. These excretions and secretions are most concentrated in dust reservoirs in kitchens (Rutherford & Eigeland 2000).

Fungi and bacteria

Fungi, such as moulds and yeasts, are potential indoor airborne allergens. Moulds are mostly saprophytic fungi that live on foodstuffs and other organic matter. Studies of adults living in homes with reported dampness or mould growth show an increased risk of upper respiratory symptoms ranging from cough to shortness of breath (Dales et al. 1991). *Cladosporium* and *Penicillium* are fungi that have been associated with asthma (Dharmage et al. 2001).

Part of the wall of some bacteria is formed by endotoxins, which are shed when the bacterium dies or grows. Endotoxins bind with white blood cells and stimulate the release of nonantibody proteins (cytokines) that mediate an inflammatory response (Douwes et al. 2002).

Combustion by-products

Cooking and heating activities in the home can produce inhalable particles (different-sized solid particles and liquid droplets) as well as gases such as sulphur dioxide (SO_2) and nitrogen dioxide (NO_2) (Australian State of the Environment Committee 2001; Denison et al. 2001; Katsouyanni 2003). Exposure to these pollutants can produce acute effects such as irritation of the airways, triggering of asthma episodes and aggravation of bronchitis (Denison et al. 2001; Katsouyanni 2003; Kunzli et al. 2000). At high concentrations, prolonged exposure may lead to tissue damage in the airways and chronic lung disease (Katsouyanni 2003).

There is some evidence that exposure to NO_2 may exacerbate virus-induced asthma attacks. The exact mechanism is uncertain, but one empirically supported suggestion is that exposure to NO_2 prior to infection increases the susceptibility of cells in the respiratory tract to damage from respiratory viruses, while concurrent exposure (especially to rhinoviruses) exacerbates virus-induced inflammation (Chauhan et al. 2003).

Volatile organic compounds

Hazardous air pollutants, or air toxics, is the broad term applied to a large number of chemicals, including the toxic ingredients of inhalable particles and tobacco smoke. Some of these chemicals may exacerbate asthma because, once sensitised, individuals can respond to very low concentrations. Hazardous air pollutants include volatile

organic compounds (VOCs), which are a group of compounds that are liquid at room temperature but emit a potentially harmful vapour.

Formaldehyde is a common VOC found in building materials, furnishings and fabrics in Australia. Airborne formaldehyde acts as an irritant to the upper and lower respiratory tract. Symptoms are usually temporary, but severe reactions may be associated with hypersensitivity and asthma exacerbation (Golding & Christensen 2000).

Outdoor allergens and air pollutants

Many of the allergens and irritants encountered indoors are also present outdoors. Moulds such as *Cladosporium* are found outdoors as well as indoors. Indoor air pollutants such as SO_2, NO_2 and inhalable particles are also encountered outside. The relative concentrations of these pollutants depend on many factors, including the weather, pollution source and human activities while outdoors. In the case of inhalable particles, the pollution source also influences the size and chemical constitution of the particles. Other allergens, such as pollen, are most likely encountered outdoors.

Pollen

Pollen is the term commonly applied to the microspores of seed-producing plants. Pollen from trees, grasses and weeds can trigger episodes of hay fever and asthma. Hay fever that occurs year-round due to exposure to allergens such as dust mites and pet dander is referred to as perennial hay fever. Seasonal hay fever is the hay fever that occurs as a response to the seasonal release of particular types of pollen. In Australia, most seasonal hay fever occurs in spring and early summer (Allergy Net Australia 2001). In spring, most hay fever is due to imported exotic grasses such as perennial ryegrass and couch (Bermuda) grass, rather than to native plants. Pollens are usually too large to progress beyond the nasal passages and upper airways. Fragments of pollen, however, may progress deeper into the lung.

Ambient air pollution

Epidemiological studies have shown that long-term exposure to outdoor, or ambient, air pollution has an adverse effect on lung function (Dockery & Brunekreef 1996). Studies in Australia have shown a relationship between air pollution and admissions to hospital for asthma and COPD (for example, Denison et al. 2001; Morgan et al. 1998).

Ambient air pollution consists of a broad range of chemical compounds as well as coarse and fine inhalable particles. The finer inhalable particles, which are considered most harmful, originate from combustion sources such as motor vehicle exhaust (especially from diesel fuel), smoke from bush fires and home heating, and emissions from industry, whereas the larger particles include wind-blown dust and emissions from mining activities (Australian State of the Environment Committee 2001).

The combustion of fossil fuels in energy production and manufacturing industries produces SO_2 and NO_2 as by-products (Australian State of the Environment

Committee 2001). NO_2 is also produced from motor vehicle exhaust. Ozone (O_3) is a secondary pollutant that is formed by the reactions of ultraviolet sunlight and primary pollutants, such as NO_2 and VOCs (Australian State of the Environment Committee 2001). It is a major component of photochemical smog and can cause bronchoconstriction.

Occupational irritants

Occupational irritants include fumes, vapours, gases, biological enzymes, dusts, fibres and high molecular weight antigens (for example, certain particles from textiles, grains, wood or latex). Inhaling occupational irritants causes or exacerbates chronic respiratory diseases such as asthma, hay fever and COPD, especially among smokers (Beckett 2000; Hnizdo et al. 2002).

It has been estimated that 5–15% of adult-onset asthma among the working population is due to occupational exposure (Beckett 2000; Blanc & Toren 1999; Newman-Taylor 2002). There are more than 250 substances found in the workplace that may contribute to the onset or exacerbation of occupational asthma. It may occur in direct response to an irritant such as chlorine gas, bleach and strong acids or it may be due to long-term sensitisation to substances such as aldehydes, animal proteins, dust from woods and latex.

The field of occupational and environmental lung disease has dealt traditionally with chronic diseases caused by very high levels of exposure to substances that affect virtually all workers to a similar degree (Singh & Davis 2002). Characteristic symptoms, signs and radiographic abnormalities allow the disease to be readily recognised. This approach served well for conditions such as coal workers' pneumoconiosis, pneumoconiosis due to asbestos and other mineral fibres, and pneumoconiosis due to dust containing silica. Current thinking in occupational and environmental lung diseases involves consideration of low levels of exposure to complex mixtures of materials that produce non-specific or intermittent symptoms in a subgroup of exposed individuals (Singh & Davis 2002). The risk presented by genetic susceptibility and concurrent tobacco smoking is that lower levels of exposure may be required to produce symptoms. Hence people who smoke or have a history of allergies are more likely to develop asthma or hay fever, even if their exposure to occupational irritants is relatively low.

Weather

There is a phenomenon referred to as 'thunderstorm asthma' whereby hospital emergency departments experience epidemics of asthma exacerbations following severe storms. One theory is that moisture causes pollen grains to break apart and release inhalable starch granules that may lead to an allergic response in sensitised individuals. A more likely explanation is that pollen and other inhalable allergens are carried ahead of thunderstorms by gusting winds caused by the outflow of cold air from the storm (Marks et al. 2001; Wallis et al. 1996). The weather also exerts an important effect by exacerbating and prolonging periods of air pollution.

Food intolerance and allergy

Food intolerance involves a reaction to food chemicals that may trigger asthma; these include sulphites (added to foods such as wine and sauces as a preservative), tartrazine (food colours) and salicylates (material made in all plants to fend off soil bacteria and pests). The evidence for monosodium glutamate (MSG) as a trigger for asthma is inconclusive (Food Standards Australia New Zealand 2003). Allergy to certain foods also occurs but is relatively uncommon.

Other dietary factors

A number of dietary factors may play a role in inflammatory reactions and airway hyperconstriction that characterise chronic respiratory diseases such as asthma (Romieu 2002). These factors include a deficiency in antioxidant vitamins, a lower consumption of omega-3 fatty acids, low magnesium intake, and higher consumption of omega-6 oils, processed foods and salt.

Medication

Respiratory symptoms can be caused or exacerbated by a number of pharmaceuticals, as well as complementary and alternative medicines. This can be due to an allergic reaction, chemical intolerance or the pharmacological action of the substance. The use of certain types of complementary and alternative medicine has been implicated in triggering asthma through allergic reactions. For example, both royal jelly and echinacea have been linked to allergy-induced asthma (Leung et al. 1997; Mullins & Heddle 2002).

An Australian study into the prevalence of aspirin-intolerant asthma found that respiratory symptoms triggered by aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs) were present in 10–11% of people with asthma and 2.5% of people without asthma (Vally et al. 2002). Many people with asthma who are sensitive to aspirin also have chronic rhinosinusitus and nasal polyposis; this is known as the aspirin triad. Persons with this condition often have asthma that is more difficult to treat. Beta blockers (antagonists) are the main pharmacological trigger of bronchoconstriction in people with asthma.

Infectious diseases and comorbidities

Viral respiratory tract infections can trigger asthma episodes, especially in children. Bacterial infection of the lower airways may stimulate chronic inflammation and may influence the interval between exacerbations (Fein & Fein 2000; Hunter & King 2001). Most acute exacerbations of COPD are attributable to viral or bacterial respiratory tract infections.

Studies in the United States have found that about 75% of adults with asthma have gastroesophageal reflux (GER). However, the relationship between asthma and GER is uncertain—it may be an inducer, a trigger or a comorbidity (Khoshoo et al. 2003). Nevertheless, it has been observed that children with both asthma and GER can

show significant improvement in their asthma following anti-GER treatment (Khoshoo et al. 2003). People with severe GER are also at significant risk of developing bronchiectasis.

There is a high prevalence of heart disease among people with chronic respiratory diseases, especially those with COPD, due mainly to cigarette smoking acting as a common risk factor for both diseases. The respiratory disease may affect the heart condition and vice versa. That is, a coexisting heart condition may exacerbate an existing chronic respiratory disease and accelerate the decrease in lung function. On the other hand, an acute respiratory infection in someone with severe COPD may lead to right ventricular enlargement or failure, known as cor pulmonale or right-sided heart failure (Fishman 1998).

Predisposing factors

Allergy is an immune response to a foreign antigen that results in inflammation and often dysfunction of the organ involved. It is generally accepted that chronic respiratory disorders such as hay fever and asthma are to some extent inherited allergies. For instance, the best-established risk factor for developing hay fever is having a history of allergy in the immediate family. The heritability of hay fever, asthma and, to a lesser extent, COPD observed in family and twin studies is probably underlined by the genetic regulation of inflammatory responses and anti-inflammatory protective mechanisms (Walter et al. 2000). Key genes of innate immunity may vary and interact with other genes or environmental antigens (allergens) leading to allergy and inflammation diseases such as hay fever and asthma (Baldini et al. 2002).

Atopy

Atopy is the propensity to produce abnormal amounts of IgE in response to exposure to allergens. Twin and family studies suggest that it is at least partly an inherited condition (Koeppen-Schomerus et al. 2001; Sandford et al. 1996). The child of two atopic parents has a 75% chance of being atopic, whereas if only one parent is atopic there is a 50% chance of being affected. An inherited tendency to be allergic increases the likelihood that an individual will become hypersensitive to allergens such as pollen or mould and exhibit symptoms of asthma or hay fever.

Atopy is considered the strongest identifiable predisposing factor for the development of asthma. However, at least with respect to asthma, the allergic response may not be the primary cause but rather a secondary inducer or trigger of episodes of the disorder. Depending on how asthma is defined, only a minority of atopic individuals (about 25–30%) develop asthma (Holt et al. 1999), whereas up to 85% of asthmatics are atopic (Kemp & Kemp 2001). It is not completely understood why some atopic individuals exposed to an allergen exhibit symptoms of asthma while others exposed to the same allergen exhibit other symptoms and may never develop asthma.

α-1-antitrypsin deficiency

A deficiency of the enzyme α-1-antitrypsin (AAT) (also called alpha-1-protease inhibitor deficiency) results in lack of inhibition of the enzyme neutrophil elastase. When the lungs do not have sufficient amounts of AAT, neutrophil elastase is free to destroy lung tissue. As a result, the lungs lose some of their ability to expand and contract (elasticity). The role of AAT deficiency in the development of COPD is well documented. On rare occasions it is associated with emphysema even in non-smokers. However, AAT deficiency accounts for only about 1% of COPD cases (Anto et al. 2001). Furthermore, even among individuals with similar smoking histories, AAT-deficient individuals exhibit wide variability in lung function impairment (Silverman 2001).

AAT deficiency is also a predisposing factor for bronchiectasis. Bronchiectasis that develops from congenital or hereditary disorders, such as cystic fibrosis, represents a minority of cases and is believed to develop in early childhood rather than being present at birth (Fishman et al. 1998).

Sex and ethnicity

Asthma and hay fever tend to be more common in boys than girls until adolescence (ABS 2002a). Some researchers suggest that asthma is less common in girls because of the earlier growth of lung functioning and airway calibre. But this suggestion has been refuted by findings that asthma slows the rate of growth of airway calibre, regardless of sex, rather than being a consequence of reduced or differential lung function growth (Xuan et al. 2000).

COPD tends to be more prevalent among males than females. This is often attributed to higher rates of smoking among males and to the occupational factors to which males are more often exposed. However, it has been found that COPD in females is underdiagnosed by primary-care physicians, and that the apparent sex difference is reduced if spirometry is used for diagnosis (Chapman et al. 2001).

Data from the 2001 National Health Survey indicate that asthma is more prevalent among people from an English-speaking background than a non-English-speaking background (ABS 2002a) and among Indigenous Australians than non-Indigenous Australians (ABS 2002b). Some researchers (for example, Aligne et al. 2000) suggest that such differences in prevalence do not point to asthma being an ethnically-linked genetic disease. Rather, the differences are probably associated with socioeconomic factors and varying exposure to environmental factors, such as dust, tobacco smoke and diet.

Other risk factors

Besides genetic and environmental risk factors, a number of behavioural and psychosocial factors may also increase the risk of developing or exacerbating chronic respiratory diseases, especially asthma.

Athletes who train outdoors are repeatedly exposed to ambient air pollution year-round, to cold air during winter, and to many pollen allergens in spring and summer (Helenius & Haahtela 2000). Physical exertion involved in exercising is also a common trigger of acute episodes of asthma. The exact mechanism involved in exercise-induced asthma is not fully known, although it is believed that heat exchange and/or moisture loss are involved (Lacroix 1999). That is, the drying and/or cooling of the airways leads to changes in the airway mucosa, limitation of the airflow and possibly the release of inflammatory mediators.

Overweight and obesity have been associated with chronic respiratory diseases. Although the evidence is inconclusive, higher body mass index has been linked to asthma (Guerra et al. 2002; Romieu 2002).

6 Prevention of chronic respiratory diseases

Chronic respiratory diseases are often incurable and involve irreversible damage to lung function. However, through knowledge of risk factors and by action at the individual, health provider and government levels, chronic respiratory diseases can be prevented. Effective prevention and management of respiratory diseases reduce the need for visits to physicians and hospitalisation, prolongs life, and enables patients to enjoy normal activities as much as possible.

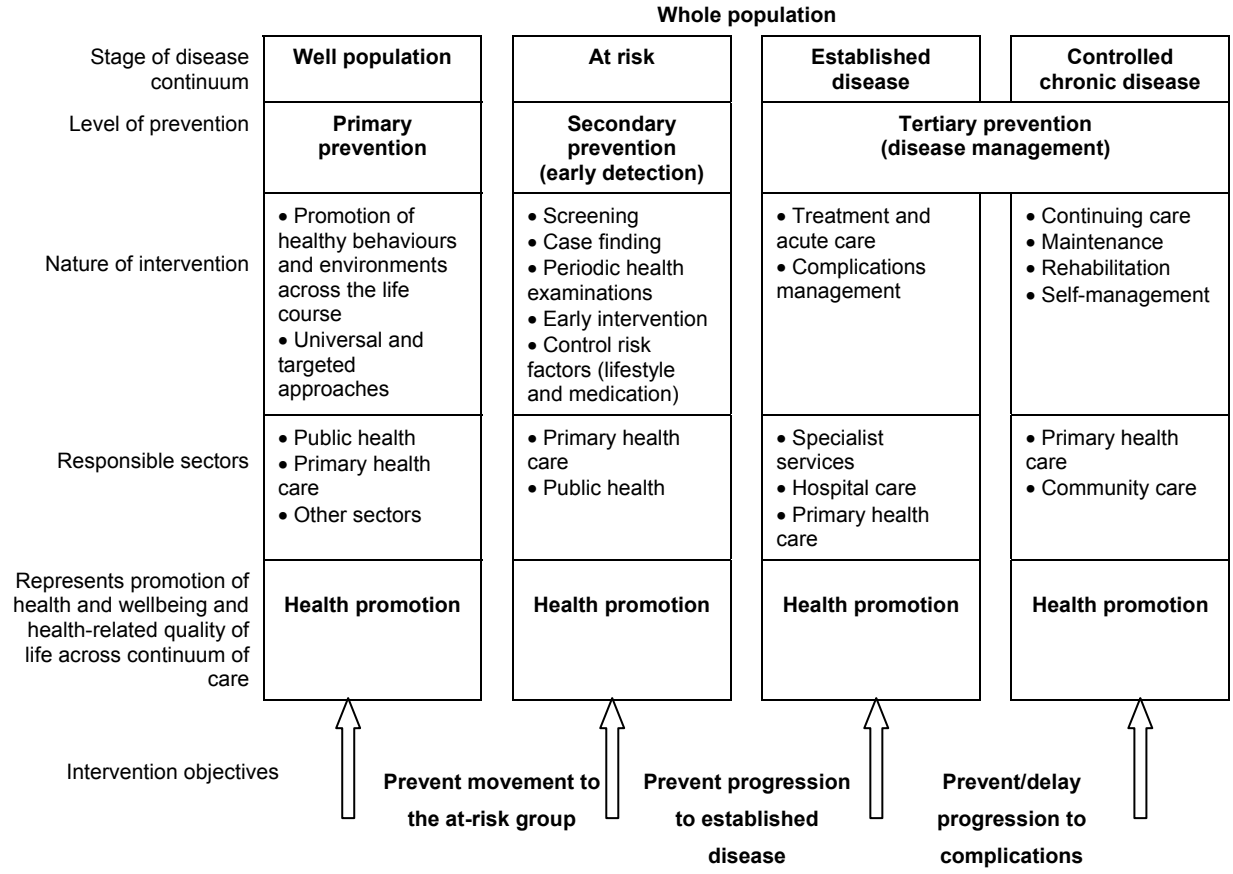
Prevention strategies

Canada's National Asthma Control Task Force (2000) has noted an historical emphasis on the treatment of asthma rather than on its prevention. This emphasis is partly due to a lack of knowledge on how to prevent the onset of asthma. Nevertheless, the incidence and consequences of asthma and other chronic respiratory diseases can be reduced through strategies that promote education, identification and avoidance of risk factors, regular monitoring of symptoms and lung function, and by combining regular medical advice with self-management.

The National Public Health Partnership has devised a strategic framework for preventing chronic diseases (National Public Health Partnership 2001). Key components of the framework include a 'cluster' of specified risk and protective factors and preventable conditions, models of joined-up action, components of a comprehensive strategy, and strategic management requirements. The initial cluster of preventable chronic diseases includes chronic lung diseases (COPD and asthma), while the initial cluster of risk and protective factors includes behavioural factors (for example, tobacco smoking) and early life factors (for example, childhood infections) that are relevant to chronic respiratory diseases. The cluster also contains relevant nonmodifiable factors, such as age, sex, ethnicity and genetic makeup. Other risk factors in the cluster include socioenvironmental determinants, such as socioeconomic status, working conditions and environmental health.

One of the 'models of joined-up action' under the strategic framework is a life course, or 'whole of life', approach to chronic disease prevention. For example, tobacco control, immunisation and environmental health are promoted throughout an individual's life course, but with greater emphasis at the most appropriate stages of life and within the most appropriate settings. This approach involves four target populations along the life course (mothers and infants, younger people, adults, older people/elderly) that become the focal points for strategic intervention by health promotion and protection strategies such as tobacco control, immunisation and environmental health (National Public Health Partnership 2001).

The strategic framework integrates the whole-of-life approach with a 'whole of health system' approach to form a comprehensive model of chronic disease prevention and control (Figure 10) that can be applied to chronic respiratory diseases. A key feature of the model is that it links disease prevention and management by outlining four stages of the disease continuum (well population, at-risk population, established disease and controlled chronic disease) and three levels of prevention (primary, secondary and tertiary). The purpose of primary prevention is to reduce the incidence of disease by preventing susceptible people moving from the well population to the at-risk population. Through early detection and effective intervention, the purpose of secondary prevention is to prevent members of the at-risk population progressing to established disease. The purpose of tertiary prevention is to prevent or delay complications and disability through the effective management of established disease.



Each stage requires critical assessment of workforce requirements, resource allocation, data requirements, evidence base for intervention (including cost effectiveness), quality measures, guidelines and standards, monitoring and evaluation, roles and responsibilities (Commonwealth/state, public/private), equity impact, consumer involvement, etc.

Figure 10: A comprehensive model of chronic disease prevention and control

Source: Adapted with permission from National Public Health Partnership (2001).

The model of chronic disease prevention illustrated in Figure 10 is reflected in the long-term goals of the Australian Lung Foundation's COPD strategy (McKenzie et al. 2003). These goals include primary prevention of smoking commencement, early detection of disease in at-risk groups (especially smokers), improving rates of smoking cessation, and management of stable disease and prevention of exacerbations.

Avoiding, reducing or eliminating exposure to risk factors is a major component of primary, secondary and tertiary prevention interventions for chronic respiratory diseases. Interventions aimed at reducing exposure to tobacco smoking, allergens, other air pollutants, occupational irritants and infections are discussed in the following sections.

Tobacco smoking

Controlling tobacco use is a major primary preventive strategy for reducing the burden of chronic respiratory diseases. This is because cigarette smoking is the single most important risk factor for developing COPD, while passive exposure to tobacco smoke (environmental tobacco smoke) also contributes to the risk and may trigger asthma.

Strategies for minimising and controlling the effects of tobacco smoking focus on (1) the avoidance of smoking commencement among current non-smokers, (2) the cessation of smoking among current smokers, and (3) the avoidance of exposure to environmental tobacco smoke in public spaces and facilities and in the workplace. Smokers can seek professional counselling from their physician or state and national information services. Nicotine replacement therapy (nicotine transdermal patches, nicotine gum, nicotine lozenges or a nicotine inhaler) is frequently used to help people quit.

Smoking cessation strategies recommended by the United States Centers for Disease Control and Prevention include improving access to quit lines, providing insurance coverage of smoking cessation services, and increasing the involvement of health care providers and systems in the delivery of cessation advice and services (Centers for Disease Control and Prevention 2003).

As part of the 'Reduce Risk Factors' component of their COPD management program, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) has a strategy for health care providers to help patients quit smoking (GOLD 2004). This strategy (the '5 As') consists of:

- **Ask:** identify all tobacco users among patients
- **Advise:** strongly urge all tobacco users to quit
- **Assess:** determine willingness to quit
- **Assist:** aid the patient to quit
- **Arrange:** schedule follow-up contact.

GOLD also has a strategy for smoking prevention, including the promotion of clear, consistent and repeated non-smoking messages; encouragement of smoke-free homes; and promotion of legislation to establish smoke-free schools, public facilities and spaces, and work environments (GOLD 2004).

The Australian Lung Foundation's COPD strategy includes the primary prevention of smoking, improving rates of smoking cessation and early detection of airflow limitation in smokers (McKenzie at al. 2003). The Thoracic Society of Australia and New Zealand and the Australian Lung Foundation's guidelines for patient care (COPDX) are:

- **C**onfirm diagnosis and confirm severity
- **O**ptimise function
- **P**revent deterioration
- **D**evelop support network and self-management plan
- e**X**acerbations—manage appropriately (McKenzie at al. 2003).

A major part of the 'Prevent deterioration' component of the COPDX plan is risk factor reduction, especially smoking cessation.

The National Tobacco Strategy 1999 to 2002–03 was the first of the National Drug Action Plans developed under the National Drug Strategic Framework. The aim of the strategy was to reduce Australians' exposure to tobacco in all its forms by preventing the commencement of smoking and reducing the number of people who smoke. Miller and Wood (2002) reviewed the evidence of smoking cessation interventions. They looked at behavioural interventions (self-help intervention, minimal and intensive clinical interventions, individual behavioural counselling, supportive group sessions and aversion therapy), pharmacological aids (nicotine replacement therapy, antidepressants), and interventions such as acupuncture and hypnotherapy. Table 7 lists some of Miller and Wood's conclusions based on good empirical evidence; that is, evidence from multiple well-designed and salient randomised controlled trials from which a consistent pattern of findings has been established.

Stewart and Huang (2004) conducted a review of public health interventions for asthma on behalf of Asthma Victoria. The review included interventions for smoking commencement and cessation. They concluded that the most effective school-based smoking commencement interventions are peer-led interventions underpinned by social influence resistance. They also concluded that mass media interventions for preventing smoking in adolescents are most successful when combined with other school-based or community-based education interventions. Community-based education interventions alone show little evidence of preventing smoking in adolescents.

With respect to interventions by health professionals for smoking cessation, Stewart and Huang (2004) concluded that advice from general practitioners has a small but statistically significant effect on cessation rates. Individual counselling from specialists also assists smoking cessation. These conclusions concur with those of

Miller and Wood (2002). Relapse back to smoking is a common finding. McKenzie at al. (2003) concluded that relapse may be prevented by combining counselling with nicotine replacement therapy.

Table 7: Evidence for smoking cessation interventions

Intervention	Conclusion/recommendation
Self-help interventions (e.g., printed leaflets, manuals, tapes, videos, etc.)	
	Generic self-help materials alone are of small benefit compared to no intervention.
	Tailoring materials to the characteristics of individual smokers improves effectiveness.
	Adding follow-up telephone calls improves effectiveness of self-help materials.
Minimal clinical interventions ('5 As')	
	(Steps 2 and 4: Advising and Assisting) brief cessation advice to smokers from doctors delivered opportunistically during routine consultations has a modest effect size but substantial potential public health impact.
Intensive clinical intervention	
	There is a strong dose–response relationship between the number of sessions and abstinence rates.
	Individual counselling, group and proactive telephone counselling are effective methods of increasing long-term quit rates.
Individual behavioural counselling	
Intensive intervention by a usual carer (doctor, nurse)	Follow-up visits with their doctor significantly increases cessation rate of smokers at six months or more compared to no follow-up.
Counselling by a smoking cessation specialist	Individual counselling is more effective in achieving sustained smoking cessation than brief advice, usual care or provision of self-help materials.
Proactive telephone counselling	Proactive telephone counselling is effective in increasing cessation rates when used as a sole intervention modality or when augmenting programs initiated in hospital settings.
	Repeated telephone support for up to 12 weeks is more effective than a single telephone counselling session.
Supportive group session	
	Group behaviour therapy is more effective in achieving sustained smoking cessation than self-help and other less intensive interventions.
Aversion therapy	
	There is no evidence of benefit from aversion methods other than 'rapid smoking' techniques (for which there is some evidence of benefit).
Nicotine replacement therapy (NRT)	
	Nicotine gum, nicotine transdermal patch, nicotine nasal spray and nicotine inhaler all increase quit rates at five to 12 months approximately twofold compared with placebo and regardless of the setting (there is some evidence that combinations of different forms of NRT are more effective than one form alone).
	NRT is effective on its own but there are added benefits of combination with behavioural intervention.
Antidepressants	
Bupropion	The combination of Bupropion and nicotine patch is more effective than nicotine patch alone.
Acupuncture	
	There is no evidence of a specific effect of acupuncture in smoking cessation other than as a placebo effect.

Source: Miller and Wood (2002).

Allergens

Whether to prevent the development of asthma or acute exacerbations, a common preventive measure is to minimise exposure to indoor allergens and irritants such as house dust mites, pet dander, cockroaches, moulds and environmental tobacco smoke. This is particularly pertinent to young children and those with a family history of asthma or allergy. To this end, the individual can take a number of simple steps such as having allergies identified by a skin prick test. Regular cleaning and adequate ventilation of living spaces and, as far as is practical, eliminating carpets, curtains, and other dust collecting fittings and furniture can help create a low-humidity and dust-free home. Workers in environments that contain respiratory irritants can wear protective devices and have their lung functioning monitored by spirometry tests. Exposure to outdoor allergens, such as pollen, can be reduced by monitoring pollen counts and storm warnings in the media. However, evidence for the effectiveness of such allergen-avoidance interventions is not clear.

Stewart and Huang (2004) found no evidence for effective primary prevention of exposure to house dust mites. The evidence for effective secondary and tertiary prevention was better but not conclusive. The Centers for Disease Control and Prevention in the United States have also reviewed the literature on asthma interventions. With respect to house dust mites, studies in their review found that mite-impermeable mattress and pillow covers, and other anti-mite measures, could reduce asthma symptoms (Centers for Disease Control and Prevention 2004). Although evidence for a relationship between cat allergens and asthma symptoms is strong, there is a lack of evidence of effective interventions to reduce exposure (Stewart & Huang 2004). Avoiding exposure to mould by minimising humidity in homes appears to be reasonably effective in reducing the frequency of asthma symptoms (Stewart & Huang 2004).

Air pollution

Air pollutants such as inhalable particles, SO_2 and NO_2 have been shown to exacerbate, rather than cause, chronic respiratory diseases. Therefore, interventions aimed at air pollutants other than environmental tobacco smoke tend to be of the nature of secondary and tertiary prevention.

Environmental protection measures to reduce emissions of various air pollutants and legislation to restrict the level of lead in petrol have reduced levels of outdoor air pollution over the last decade. National Environment Protection Measures, enforced by the National Environment Protection Council, have set standards for maximum concentrations of certain ambient air pollutants and air toxics. Under motor vehicle emission standards set by Australian Design Rules under the Commonwealth's *Motor Vehicle Standards Act 1989*, motor vehicles manufactured after 1986 must operate on unleaded fuel and have antipollution devices (catalytic converters) fitted.

At the individual level, outdoor air pollution can be avoided by monitoring public announcements of air quality and by remaining indoors during high pollution episodes. The National Health and Medical Research Council has set guidelines for

indoor air quality but lacks the powers to enforce them. Legislation, such as New South Wales' *Smoke-free Environment Act 2000*, has reduced the risk of exposure to environmental tobacco smoke in most public places.

Levels of air pollution in the home can be reduced by adequate ventilation. Exposure to NO_2 can be reduced by not using unflued heaters, while exposure to particulate matter from wood smoke can be reduced by not relying on wood for heating, or ensuring proper operation and ventilation of wood heaters.

Occupational exposure

Exposure to irritants in the workplace accounts for 5–15% of adult-onset asthma (Blanc & Toren 1999; Newman-Taylor 2002). Although Stewart and Huang (2004) suggest that there are no well-conducted studies of the effectiveness of primary prevention of occupational asthma, workplace exposures can be minimised by occupational health and safety regulations, and adequate occupational hygiene measures.

Infections

Infections such as influenza and pneumonia can cause many complications for people with chronic respiratory diseases, including cough, tiredness and muscle aches, and exacerbation of COPD. Consequently, influenza and pneumococcal vaccination is part of the 'Prevent deterioration' stage of the COPDX plan. Vaccination against influenza reduces the risk of hospitalisation and death associated with COPD (McKenzie at al. 2003). Vaccination against pneumonia is generally worthwhile for people with reduced respiratory function. Respiratory infections among young children can be prevented by vaccinations and effective infection control in day care.

To a large extent, the primary prevention of chronic respiratory disease incidence and the secondary prevention of chronic respiratory disease consequences is possible. However, for those who progress to symptoms, the goal becomes the prevention of complications and disability (tertiary prevention) through effective disease management.

Management of chronic respiratory diseases

The management of chronic respiratory diseases has shifted from reliance on pharmacological treatment to a range of interventions that include patient education, self-management of exacerbations and pulmonary rehabilitation. For example, the New South Wales Department of Health (2003) has compiled a chronic respiratory diseases management strategy that reflects the new approach to integrated, coordinated and more patient–focused care. The components of the strategy are:

- placing patients at the centre of care
- fostering an integrated and coordinated approach across the continuum of care

- developing standards of care
- fostering clinical governance
- fostering more timely and effective treatment in a community setting
- streamlining admission and discharge planning processes and practices
- educating patients.

In general, the management options in relation to the progression of a chronic respiratory disease include:

1. Disease management plan with a physician
2. Pulmonary function tests
3. Pulmonary rehabilitation
4. Nutrition intervention
5. Other allied health professionals
6. Drug treatments
7. Devices to assist breathing
8. Oxygen therapy
9. Surgical procedures.

Brief information on each of the options is given below.

1. Disease management plan with a physician

Regular review of the disease with a physician aims to optimise lung function, minimise respiratory decline, improve quality of life and provide health education. Formal disease management plans have been developed by the Thoracic Society of Australia and New Zealand for asthma and in conjunction with the Australian Lung Foundation for COPD.

The Asthma Management Plan was developed in 1990 to assist health professionals and patients with the management of asthma. Since its inception, the National Asthma Council has refined the plan and encourages its use by physicians and people with asthma. The aims of the Asthma Management Plan are to prevent the occurrence of asthma attacks, identify trigger factors, minimise the symptoms of asthma, maintain lung function and minimise the side effects from medication (National Asthma Council 2002). A key aspect of the Asthma Management Plan is the development of an action plan tailored to the needs of the patient to help them recognise worsening of symptoms and deterioration in their health.

The Australian Government-funded Asthma 3+ Visit Plan Practitioner Incentive Program is based on self-management education, written action plans, self-monitoring and regular medical review. Between the program's inception in late 2001 and the end of June 2003, more than 55,000 Asthma 3+ Visit Plans had been completed (Australian Centre for Asthma Monitoring 2003).

During 2002, the Thoracic Society of Australia and New Zealand and the Australian Lung Foundation released COPD management guidelines and a handbook (Thoracic Society of Australia and New Zealand & Australian Lung Foundation 2002). Many recommendations in the handbook are based on recent international guidelines outlined by the World Health Organization-endorsed global initiative for the diagnosis, management and prevention of chronic obstructive lung disease (referred to as GOLD), amended for treatment practices and regulations in Australia and New Zealand. The handbook was prepared for health professionals and others closely involved in the management of COPD patients and contains the COPDX plan for patient care.

2. Pulmonary function tests

Pulmonary (or respiratory or lung) function tests measure the function of the lungs without the need to physically examine the lungs themselves. They assist in the diagnosis of suspected respiratory diseases and also in the planning of treatments and in the decision to continue, discontinue or change treatments. Most pulmonary function tests involve relatively simple breathing tasks. The various types of pulmonary function tests include mechanical function tests (spirometry, static lung volumes and capacities, inhalation challenge), gas exchange tests (carbon monoxide diffusing capacity, arterial blood gases analysis) and exercise stress tests.

From 1994 to 2002, the number of health insurance claims for lung function tests has remained stable, although there is a trend for fewer claims for spirometry performed in the doctor's surgery and more claims for laboratory-based tests (Australian Centre for Asthma Monitoring 2003).

3. Pulmonary rehabilitation

The Australian Lung Foundation actively promotes pulmonary rehabilitation as an integral part in the treatment of chronic respiratory diseases (Australian Lung Foundation 1998). Pulmonary rehabilitation is a process in which the patient and their family works in a team environment with a range of physicians and other health professionals (physiotherapists, pharmacists, psychologists, occupational therapists, dietitians etc.) to meet the patient's specific physical, emotional and social needs, and to improve quality of life. The members of the team, the role they play and the time they spend with patients can vary considerably between programs.

Pulmonary rehabilitation is used widely for people with chronic respiratory diseases such as emphysema, chronic bronchitis, asthma, bronchiectasis, and occupational and environmental lung disease. The aim of rehabilitation is to help patients lead a satisfying life and restore their lung function to their highest possible capacity.

4. Nutrition intervention

Chronic respiratory disease drastically increases the metabolic rate of the body and has multiple consequences on the health of patients. The normal caloric requirement

for breathing of about 50–70 Kcal per day may rise up to 700 Kcal per day simply from the excess work that is required to continue breathing with a chronic respiratory disease (Manaker & Burke 1996). Thus, malnutrition is a major problem in chronic respiratory disease, especially when patients limit their energy intake. The poor nutritional status can lead to anorexia and muscle wasting, which in turn negatively affects pulmonary function. Nutrition intervention aims to prevent or reverse malnutrition, without worsening the disease process, and improve respiratory function, thereby reducing morbidity and delaying mortality (Harmon-Weiss 2002).

5. Other allied health professionals

As highlighted in the fact sheets in chapters 2 and 3, people hospitalised with COPD and asthma commonly encounter allied health professionals, particularly physiotherapists. Expenditure data for these diseases, particularly asthma, also suggest frequent encounters with pharmacists.

6. Drug treatments

Chronic respiratory diseases can usually be managed effectively through a combination of medications (GOLD 2004; National Asthma Council 2002), although, in the case of COPD, medication cannot prevent long-term decline in lung function (McKenzie at al. 2003). Each class of medicines works in a different way.

Bronchodilators open the bronchial airways to relieve symptoms of bronchoconstriction in people with asthma or COPD. They are usually administered by a puffer (pressurised metered dose inhaler). Long-acting bronchodilators can be used to provide sustained relief for people with asthma or moderate to severe COPD (McKenzie et al. 2003). Corticosteroids, also known as 'preventers', reduce inflammation and swelling of lung tissue. They are the main preventive medication for asthma (National Asthma Council 2002) and may be used for severe COPD with frequent exacerbations (McKenzie at al. 2003). Antibiotics are used to treat bacterial infections, thereby avoiding exacerbations that may be caused by the infection (GOLD 2004).

7. Devices to assist breathing

Several devices are available to assist the breathing of people with chronic respiratory diseases. These include inhalation devices such as the press-and-breathe pressurised metered dose inhaler (MDI), the most commonly used device. The medication is dissolved or suspended in a propellant under pressure. When activated, a valve system releases a metered volume of drug and propellant.

Nebulisers use oxygen, compressed air or ultrasonic power to break up solutions of medication into droplets for inhalation. The aerosol is administered through a mask or a mouthpiece. Nebulisers tend to be more expensive than MDIs and require a

power source and regular maintenance. They are only recommended for people with severe COPD or asthma (GOLD 2004; National Asthma Council 2002).

Ventilators, also referred to as respirators, assist patients to breathe when they are limited in their capacity to do so on their own.

8. Oxygen therapy

Oxygen therapy is the provision of supplemental oxygen. It is often used for COPD patients with long-standing low blood oxygen levels. Occasionally, oxygen is supplied in emergencies to patients who have had severe acute life-threatening attacks of asthma (Australian Lung Foundation 2002). The Australian Lung Foundation estimated that almost 22,000 Australians were on home oxygen therapy in 2001 (Crockett et al. 2002).

9. Surgical procedures

On rare occasions, when other treatments are not effective, surgery may be indicated for symptom relief for people with chronic respiratory diseases, particularly COPD (McKenzie at al. 2003). Possible operations include:

- lung volume reduction surgery (removal of parts of the lung with severe and localised emphysema damage)
- bullectomy (surgical removal of one or more bullae, which is an airspace that has formed due to lung tissue destruction)
- lung transplantation.

Lung volume reduction surgery is not common, possibly reflecting the high-risk nature of the procedure. It was performed just 189 times in the three years between mid-2000 and mid-2003.

Lung transplantation is also relatively rare due to a lack of donor organs and the requirement for potential recipients to be free of other major diseases. Not including combined heart and lung transplants, there were 252 lung transplants between mid-2000 and mid-2003.

Self-management

In addition to the above-mentioned strategies, one of the more popular strategies in health care management in recent years has been 'self-management'. The strategy aims to provide patients with as many tools as possible to help them cope with their disease. It is also about forming a partnership between the patient and health care providers. The Australian Lung Foundation (2000) recommends six steps towards self-management:

- Engage in activities that protect and promote health; for example, do a pulmonary rehabilitation course, participate in a regular exercise program, attend a patient support/education group.

- Monitor and manage symptoms and signs of illness; for example, monitor shortness of breath or sputum colour, visit a physician sooner rather than later when changes occur.
- Be aware of and manage the impact of illness on functioning, emotions and relationships to reduce the impact they have on your emotional state and on relationships.
- Adhere to treatment regimens.
- Maintain regular and open relationship with health professionals.
- Know and understand the disease and the range of treatment options to allow for a more active involvement in the decision making process.

The self-management strategy focuses on the cause of the disease rather than the consequences, thereby bringing prevention and management strategies together.

7 Conclusions

The chronic respiratory disease story is a mix of good news and caution. The good news emanating from improved disease prevention and management is balanced by the caution that, as the population ages and the role and nature of environmental factors change over time, chronic respiratory diseases are likely to have significant consequences for the health of many Australians.

The purpose of this report was to provide a concise summary of the prevalence and consequences of a selection of chronic respiratory diseases affecting Australians. The report also contains an overview of disease risk factors and prevention strategies. The report is focused on two major chronic respiratory diseases, COPD and asthma. These two diseases are very prevalent in Australia and result in substantial financial burden on the community and the individual. In the case of COPD, the greatest health system expenditure involves hospitalisation and is associated mostly with those aged over 65 years. In the case of asthma, the greatest health system expenditure involves medication and is associated mostly with those aged under 65 years.

The fact sheets in chapters 2 and 3 provide a contrast between mortality attributed to COPD and asthma. COPD causes many deaths each year. The number of deaths attributed to COPD is comparable to some of the major causes of death, particularly cancers such as lung cancer, prostate cancer and colorectal cancer. In addition, COPD may also cause prolonged disability. Far fewer deaths are attributed each year to asthma. However, for at least the previous few years, the average age of the relatively few asthma deaths tends to be about 10 years younger than the average COPD death.

The fact sheets also show interesting contrasts between aspects of hospitalisation due to COPD and asthma. For example, the average age of a hospital separation in 2002–03 was about 72 years for COPD and 25 years for asthma, while the average length of stay for COPD was three times that for asthma. The respective trends in the rate of hospitalisation in recent years are also of interest.

The message from this report extends beyond the prevalence and consequence of the selected diseases. To a large extent, the effective prevention of the onset and consequences of chronic respiratory diseases is possible. This is because many of their risk factors can be identified and avoided, or at least managed. Tobacco smoking is by far the greatest risk factor for COPD. It also exacerbates symptoms in people with asthma and is a key risk factor for other major diseases, such as cardiovascular disease and lung cancer. Exposure to environmental tobacco smoke, particularly in childhood, may lead to the development of asthma or exacerbate symptoms among those with asthma. Tobacco smoke has therefore received much prominence in public health research and policy.

Tobacco consumption has decreased markedly over the past three decades. Although it is difficult to be certain, a great deal of this decline is probably a result of public health programs. Allowing for the lag between smoking commencement and disease onset, the impact of the declining tobacco consumption is showing in the steadily declining number of deaths attributed to COPD. However, the number of hospital separations is steadily rising. More precise data is needed in coming years to indicate the impact of declining tobacco consumption on COPD incidence and prevalence.

Other risk factors are known to trigger episodes of asthma and exacerbate COPD. Some of these risk factors, such as outdoor air pollution and acute respiratory infections, can and have been addressed through public health policies; others, such as exposure to allergens and irritants in the home, and genetic predispositions, are largely personal matters that are not easily resolved through public health policies. The exact cause of asthma remains unknown, and the prevalence of the disease among children rose during the 1980s and early 1990s (Australian Centre for Asthma Monitoring 2003). In recent years, however, there has been a trend for fewer deaths and fewer hospital admissions. At least part of this trend may be due to interventions aimed at the environmental triggers of asthma. However, the evidence for the effectiveness of interventions aimed at triggers such as house dust mites is mixed.

The set of diseases covered by this report is not exhaustive, but the authors believe it is adequate to illustrate the commonalities and differences of chronic respiratory diseases with respect to their consequences, risk factors and prevention. Hay fever and chronic bronchitis are not great contributors to mortality or disability in Australia, but they are very prevalent conditions. There are far fewer people with pneumoconiosis or bronchiectasis, but to the individual they are significant conditions that can result in long stays in hospital and death. Similar reports on diseases such as lung cancer, cystic fibrosis and sleep-related disorders are required to provide a more comprehensive picture.