2.7 Asthma

Asthma (ICD-9 code 493; ICD-10-AM codes J45, J46) is a chronic inflammatory disorder of the lung's air passages that makes them narrow in response to various triggers, leading to episodes of shortness of breath and wheezing. Asthma can begin at all ages, including the very young. The disease can start as a mild chronic cough and lead to mild or severe wheezing and sometimes even to respiratory arrest. Many people with asthma experience reduced quality of life and require a range of health services, from general practitioner (GP) care to emergency ward visits or hospital in-patient care.

Description

Signs and symptoms

The symptoms of asthma can vary greatly in frequency and severity, ranging from intermittent mild symptoms to an incapacitating and life-threatening disorder. Some individuals have an occasional episode (episodic asthma) that is mild and brief, but are otherwise symptom-free. Others have chronic asthma with mild coughing and wheezing much of the time, and occasional severe attacks after exposure to known environmental irritants, viral infections, exercise or non-specific irritants.

Disease severity and survival

The prognosis of individual asthma attacks is generally good. Spontaneous remission is fairly common in episodic asthma, particularly in children, but rare in chronic asthma. Seasonal fluctuations can occur in both types of asthma. People who tend to be allergic and have episodic asthma usually have more symptoms in spring and summer when they are more heavily exposed to triggers. By contrast, chronic asthmatics are usually worse off in winter months because of more frequent viral infections. There is occasionally a fatal outcome, especially if treatment is inadequate or delayed.

Co-morbidities

Asthma often occurs with other respiratory conditions, whose presence can influence the management of the disease. In allergic children and adolescents, asthma often coexists with conditions such as eczema, sinusitis and hay fever. Chronic obstructive pulmonary disease (COPD) commonly coexists with asthma among older people.

Fatigue is a common side effect of asthma. It is a normal response to breathing distress, lack of sleep or physical exertion. Asthma is known to disturb sleep, and one form of sleep disturbance, obstructive sleep apnoea, can itself cause asthma-like symptoms such as wheezing.

Also, there can sometimes be side effects associated with the long-term use of steroid medications prescribed to reduce inflammation in the lung's air passages. Some of these include osteoporosis, bruising easily, risk of cataract, heartburn and indigestion, and mood swings (Brostoff & Gamlin 1999).

Risk factors

Risk factors for asthma fall into two categories—factors that predispose a person to being asthmatic, and factors that can precipitate attacks or worsen their severity (see Box 2.7.1).

Factors that may increase the risk of asthma include a family history of the disease and being 'allergic'. The relationship of parental history to

asthma among children has been documented by many studies; it is estimated that having a parent with asthma nearly doubles the risk of asthma in the child (Peat 1996:10). Further, the marked rise in childhood asthma in Western societies, including Australia, over the past quarter-century is thought to reflect a basic shift in early-life environmental conditions. These, in turn, affect the direction of maturation of the young person's immune system, thereby influencing their disposition to asthma and other allergy-based disorders.

Box 2.7.1: Risk factors for asthma

Predisposing factors

Family history of asthma
Age
Excess weight

Environmental factors

Allergens
Exercise
Emotion
Viral infections
Tobacco smoke
Food
Chemicals
Drugs

Source: Crompton et al. 1999.

The symptoms of asthma can be triggered by a wide variety of agents in the environment, either acting alone or in conjunction with other irritants. The triggers that cause episodes of air passages narrowing and other asthma symptoms are specific to each individual. Known triggers include allergens, exercise, emotion, viral infections, exposure to tobacco smoke, food chemicals, cold air and weather changes.

Allergen exposure is currently recognised as an important risk factor for asthma in children (Peat 1996:9). House dust mites, pollens and animal fur are known asthma allergens (Newton et al. 1998). Australian homes are reported to have some of the highest levels of house dust mite. This is due mainly to environmental factors, such as housing being located in temperate coastal climates, that provide ideal conditions for breeding mites.

Tobacco smoking (including passive smoking) is another 'trigger' for asthma attacks. There is strong evidence supporting the association of exposure to environmental tobacco smoke (ETS) and asthma in childhood (age under 15). Exposure to ETS has been found to cause exacerbations of symptoms in children who have asthma (i.e. cough, wheeze, breathlessness and sputum) (WHO 1999:7). The association of ETS with childhood asthma is most consistent at high exposures (i.e. where mothers smoke more than 10 cigarettes a day), and the evidence is supportive of a causal relationship (NHMRC 1997:43). However, it is less clear whether exposure to lower levels of ETS increases the risk of asthma in children.

There is speculation that changes in diet in recent years, especially the higher use of processed foods, salt and polyunsaturated oils, are contributing factors to the increasing rate of childhood asthma (Peat 1996:9).

Prescription or non-prescription drugs can also trigger asthmatic episodes. The medications that may cause or exacerbate asthma symptoms include aspirin and non-steroid anti-inflammatory drugs. Allergic reactions and exacerbation of asthma have also been reported with the use of herbs such as echinacea (Newton et al. 1998).

Exposure to sensitising agents in the workplace is also known to trigger asthma, often referred to as occupational asthma. Known agents include wood dust, flour, industrial chemicals (isocyanates and epoxy resins), metal salts and laboratory animals. Extended exposure to these agents may increase the severity of asthma symptoms (AIHW 2000:91).

Impacts

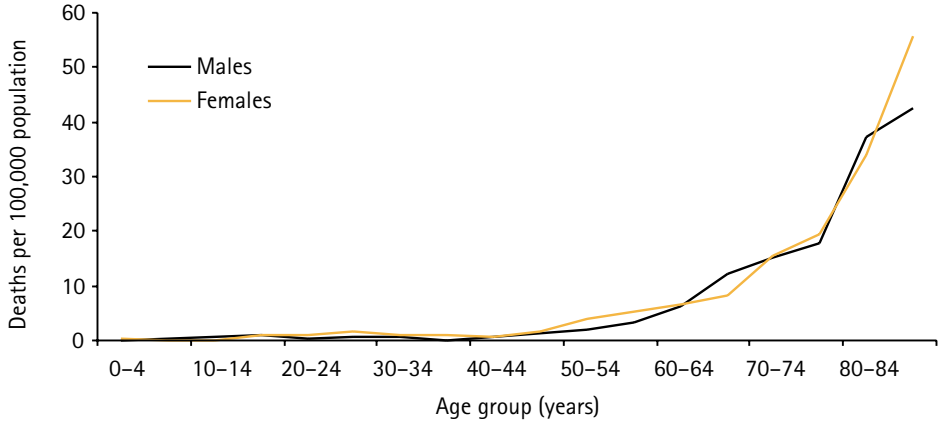
Deaths

In 1998, there were 701 deaths from asthma (or 0.5% of all deaths), with a death rate of 3.4 per 100,000. The female rate was higher than the male rate, 3.6 deaths per 100,000 females compared with 3.2 per 100,000 males.

Reported deaths from asthma generally increase with age in both males and females (Figure 2.7.1), although asthma mortality data are known to become less reliable among older people. Asthma is often difficult to distinguish from COPD in these age groups.

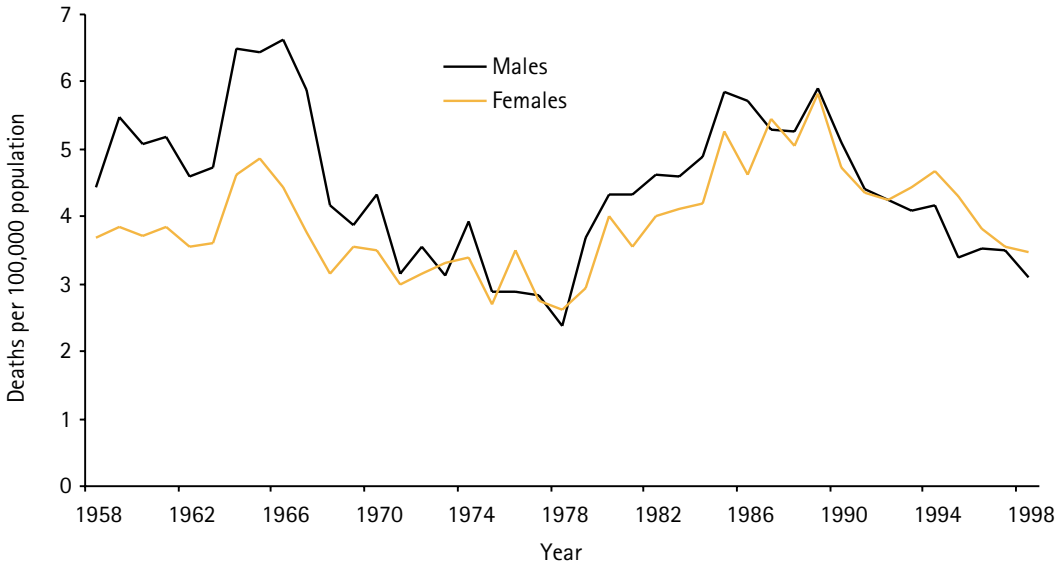
No clear trend has emerged in death rates for asthma over the last four decades. Following a decrease between 1968 and 1978, asthma death rates increased over the next decade. Since 1989, the death rates for asthma have been declining and have recently approached the rates of the 1970s (Figure 2.7.2).

Figure 2.7.1: Age-specific death rates for asthma, 1998



Source: AIHW National Mortality Database.

Figure 2.7.2: Death rates for asthma, 1958 to 1998



Source: AIHW National Mortality Database.

Table 2.7.1: Death rates for asthma in metropolitan, rural and remote areas, 1994–1998

Metropolitan		Rural			Remote		
Capital cities	Other	Large centres	Small centres	Other	Centres	Other	Australia
3.5	3.3	4.5*	3.5	4.6*	5.6*	4.3	3.8

** Significantly different from 'Capital cities' at the 5% level.*

Notes: 1. Rates are number of deaths per 100,000 persons, age-standardised to the Australian population at 30 June 1991.
2. Following RRMA (rural, remote and metropolitan area) classification.

Source: AIHW National Mortality Database.

Asthma mortality shows significant regional variation. Analysis of data for the period 1994–1998 reveals an association between death rates and increasing geographic remoteness (see Table 2.7.1). A major factor contributing to these differentials is the larger representation of the Indigenous population, with known higher death rates for asthma, in rural and remote areas. Other factors that contribute to these differentials are limited access to emergency and primary care and higher exposure to a variety of chemicals and pesticides.

Prevalence

The prevalence of asthma in Australia is one of the highest in the world, with more than two million Australians reporting the disease in 1995. Between 1989–90 and 1995, the self-reported prevalence of asthma increased from 85 per 1,000 to 113 per 1,000 (ABS 1997). Most of those reported asthma of mild to moderate severity, and were not at high risk of death from the disease.

Asthma is more prevalent in young people (aged under 25) than older age groups. In the 1995 ABS National Health Survey, it was most commonly reported as a recent or long-term condition by those aged 5–14 (192 per 1,000) and 15–24 (149 per 1,000). Among those aged 5–14, asthma was more common in males than in females, whereas it was more prevalent in females than males among those aged 15–24.

Also according to the 1995 ABS National Health Survey, smokers and ex-smokers both reported a higher prevalence of asthma (11%) than those who have never smoked (9%). Furthermore, young children living in households with one or more smokers reported a higher prevalence of asthma than children living in non-smoking households. Of children aged 0–4, 13% of those living with one or more smokers were reported to have asthma compared with 9% in non-smoking households. Similarly, asthma was reported to be more common in children aged 5–9 living with one or more smokers (22%) than in those from a non-smoking household (18%) (ABS 1997:8).

Asthma is more commonly reported among Indigenous than among non-Indigenous people across all age groups. It was the most commonly reported condition for Indigenous children and young adults: 17% of those aged 5 and under, 23% of those aged 5–14, and 20% of those aged 15–24. Although not the most common condition in older age groups, it was still reported by 16–17% of Indigenous adults aged 25 and over (ABS & AIHW 1999:92).

There is some geographic variation in the prevalence of asthma in Australia. The 1995 ABS National Health Survey found that Queensland had the highest (134 per 1,000) and Tasmania the lowest (103 per 1,000) prevalence of asthma (AIHW 2000:90).

Complications

Several different types of complications are associated with asthma attacks. An acute asthma attack may produce a pneumothorax, where air escapes from the lungs into the chest wall, compressing the lung. The symptoms are a

sudden worsening of breathing distress, accompanied by sharp chest pains and a rapid heart rate.

Air escaping out of the lungs and into the skin around the chest and neck is occasionally observed during an asthma attack (a condition known as subcutaneous emphysema). Collapse of part of the lung is a common complication of asthma, often leading to pneumonia.

Disability

Asthma can have a great impact on quality of life. People with asthma are reported to have lower scores than those without asthma when rated in relation to physical and social functioning, role limitations, bodily pain, vitality and general health, indicating a more negative health state. Episodes of asthma also lead to interruptions in schooling and work. According to the 1995 ABS National Health Survey, 12% of people with asthma reported taking days off work or school in the 2 weeks preceding the survey compared with 4% of the population without asthma (ABS 1997).

More than 171,000 persons in Australia reported asthma as a disabling condition in the 1998 ABS Survey of Disability, Ageing and Carers. The disability was mainly in the form of restriction of daily activities, including work and school participation. Another 131,000 persons reported experiencing specific activity limitations in regard to self-care, mobility and communication due to their asthma (AIHW 2000:93).

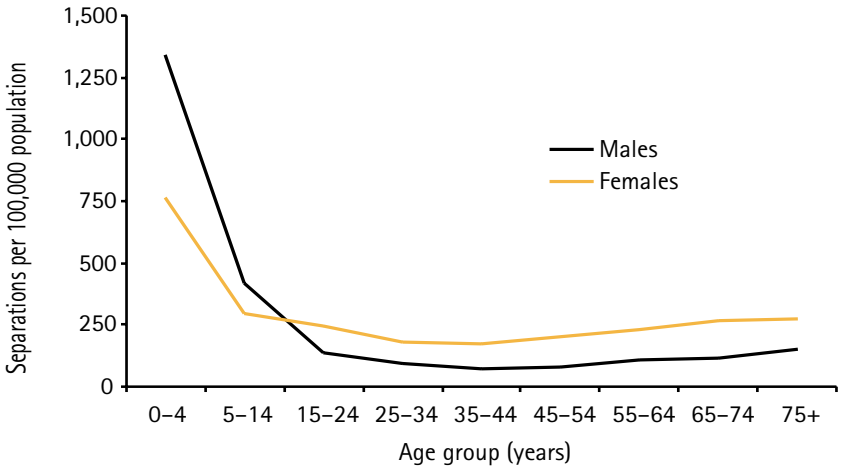
Use of health services

Asthma is the fifth most common problem managed in general practice. A survey of general practice activity in 1999–2000 reported that asthma was recorded on 3,363 occasions in the sample (at a rate of 32 per 1,000 encounters), accounting for 2.2% of all problems managed (AIHW: Britt et al. 2000:45). From 1998 to 2000, there were similar GP management rates for asthma in metropolitan and rural areas, with 31 problems per 1,000 encounters in metropolitan areas, 34 in large rural areas and 32 in small rural areas (AIHW: Britt et al. 2001).

In 1999–00, asthma was the fourth most common reason for admission to hospital. Asthma was the principal diagnosis in 47,008 hospital separations or 0.8% of all hospitalisations, with an average stay of 2.7 days. It accounted for 14% of all hospitalisations for diseases of the respiratory system.

Asthma is one of the most frequent reasons for hospitalisations among children. Males are hospitalised for asthma more often than females in the early years of life (Figure 2.7.3).

Figure 2.7.3: Age-specific hospital separation rates for asthma, 1999–00



Source: AIHW National Hospital Morbidity Database.

The hospital separation rate for asthma, like the death rate, shows regional variation, with rates increasing with geographic remoteness. Remote areas report the highest hospital separation rates, followed by the rural areas (Table 2.7.2).

Asthma is one of the most common reasons for visits to a casualty/emergency ward. Of respondents in the 1995 ABS National Health Survey reporting that they had visited a casualty/emergency ward, asthma was one of the most common reasons given, accounting for about 6% of all reasons.

In 1993–94, the health system costs for asthma were estimated to be $478 million. These include costs associated with the prevention, diagnosis and treatment of asthma (AIHW 2000). Pharmaceutical costs constituted a large proportion of the total cost for asthma ($199 million, approximately 42% of total costs). This is due to many asthma sufferers requiring prescription medication on a regular basis, which is evident with prescription medication accounting for four-fifths ($162 million) of the pharmaceutical costs for asthma.

Hospital services and pharmaceutical costs for asthma were concentrated in those aged 0–14 and 65 and over. The two groups accounted for 66% of the hospital services costs and 53% of the pharmaceutical costs for asthma.

The health system costs of females was higher than that of males, an overall difference of $27 million. The major source of the difference was hospital sector costs, where the cost for males was $42 million compared with $52 million for females.

Management

Asthma management may be divided into management of acute attacks and day-to-day long-term therapy. Drug therapy enables most patients to lead relatively normal lives with few adverse drug effects. Patients with mild asthma and infrequent episodes of wheezing may need therapy only intermittently when they have symptoms. Others, with more persistent symptoms, benefit from continuous treatment.

Two types of medications are used to treat asthma, quick relievers and long-term controllers. Quick relief medications prevent and help reduce the tightening of the muscles around the bronchial tubes. Since inflammation is a primary trigger factor of asthma, anti-inflammatory medications are often prescribed to maintain long-term control.

Asthma can be managed by effective education, regular self-use of devices that monitor lung function, identification of trigger factors, coordinating self-management with written

Table 2.7.2: Hospital separation rates for asthma in metropolitan, rural and remote areas 1996–1998

Metropolitan		Rural			Remote		
Capital cities	Other	Large centres	Small centres	Other	Centres	Other	Australia
3.0	2.3*	3.2*	3.7*	4.0*	5.4*	5.7*	3.2

**Significantly different from 'Capital cities' at the 5% level.*

Notes: 1. Rates are number of hospital separations per 100,000 persons, age-standardised to the Australian population at 30 June 1991.

2. Following RRMA (rural, remote and metropolitan area) classification.

Source: AIHW National Morbidity Database.

action plans and regular medical consultations. A healthy diet, regular exercise and not smoking are important in the successful management of asthma.

Prevention

There is a lack of knowledge currently on how to prevent people from becoming asthmatic, although control of environmental tobacco smoke and other air pollutants can reduce the frequency or severity of asthma attacks. Drug therapy can also do this for affected people, along with reducing exposure to other factors that trigger attacks.

Good prevention includes avoiding the well-known triggers. People with asthma are advised not to smoke tobacco or be exposed to tobacco smoke. Workplaces with excessive fine dusts of an organic nature (flour, sawdust, grain dust and proteins from animals), or with chemicals found in the manufacturing of plastics and resins, should also be avoided.

References

ABS 1997. 1995 National Health Survey: asthma and other respiratory conditions. ABS Cat. No. 4373.0. Canberra: ABS.

ABS & AIHW 1999. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples, 1999. ABS Cat. No. 4704.0. AIHW Cat. No. IHW 3. Canberra: ABS.

AIHW 2000. Australia's health 2000. AIHW Cat. No. AUS 19. Canberra: AIHW.

AIHW: Britt H, Miller G, Charles J et al. 2000. General practice activity in Australia 1999–2000. AIHW Cat. No. GEP 5. Canberra: AIHW.

AIHW: Britt H, Miller G & Valenti L 2001. 'It's different in the bush': a comparison of general practice activity in metropolitan and rural areas of Australia 1998–2000. AIHW Cat. No. GEP 6. Canberra: AIHW.

Brostoff J & Gamlin L 1999. Asthma—the complete guide. London: Bloomsbury.

Crompton G, Haslett C & Chilvers E 1999. Diseases of the respiratory system. In: Haslett C, Chilvers E Hunter J et al. (eds), Davidson's principles and practice of medicine, 18th edition. Edinburgh: Churchill Livingstone, 303–91.

Newton P, Flood J, Berry M et al. 1998. Environmental indicators for national state of the environment reporting—human settlements. Canberra: Environment Australia.

NHMRC 1997. The health effects of passive smoking. Canberra: NHMRC.

Peat J 1996. The epidemiology of asthma. Current Opinion in Pulmonary Medicine 2:7–15.

WHO 1999. Tobacco free initiative—international consultation on environmental tobacco smoke (ETS) and child health, 11–14 January 1999. Geneva: WHO.

2.8 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) (ICD-9 codes 491, 492, 496; ICD-10-AM codes J41–J44), is a long-term disease that causes continual and increasing shortness of breath. It is a major cause of mortality, illness and disability in Australia—the third leading cause of burden of disease following coronary heart disease and stroke. The single most important cause of COPD is tobacco smoking. Therefore the avoidance and cessation of smoking will greatly reduce the risk of the disease.

Description

COPD is a serious and disabling lung disease marked by progressive shortness of breath. Chronic bronchitis and emphysema are the two prominent forms of COPD, although some other lung diseases can be included under the heading of COPD.

Chronic bronchitis is the inflammation and thickening of the walls of the bronchial tubes (bronchi), which narrows the tubes. It often induces coughing spells, and the glands of the bronchial tubes produce excess mucus that leads to the coughing up of sputum.

In emphysema, the alveoli (air sacs) in the lungs are gradually destroyed by inflammation making it difficult for the lungs to convey oxygen to the blood stream and hence to the rest of the body. Also the bronchi become floppy and narrow, making it increasingly difficult to breathe.

Each condition can occur on its own but chronic bronchitis and emphysema usually coexist in an individual.

Signs and symptoms

COPD develops gradually, with few symptoms in its early stages. The disease typically progresses over many years, from mild breathlessness on strenuous exertion to severe breathlessness on minimal exertion. The symptoms of the disease can vary among sufferers, but typical symptoms of COPD include breathlessness and a cough with sputum production and wheezing.

Because COPD and asthma share a number of symptoms and features, it is sometimes difficult to distinguish between the two diseases. This problem applies particularly in older people because COPD and asthma can both cause chest symptoms such as shortness of breath, coughing and wheezing. COPD sufferers are more likely than people with asthma to have a daily morning cough with sputum and persistent chest symptoms throughout the day.

Disease severity and survival

The progress of the disease and survival are most closely related to the progressive decline in lung function (Fletcher et al. 1976). In those with chronic bronchitis, the mild shortness of breath and wheezing in the early stages of the disease develops over time into a chronic cough with sputum. The cough then becomes more frequent and greater effort is required to move oxygen into and out of the lungs. Among those with emphysema the wheezing becomes more frequent and greater effort is needed to breathe sufficiently. In the advanced stages of the disease, the heart may be affected (NHLBI 1995).

COPD starts with a moderate decline in lung function before the age of 50 (Goldring et al. 1993:387). The progressive reduction in lung function caused by COPD is largely irreversible.

COPD has no cure and will usually shorten life. No drug therapies have been shown to improve survival by reducing the rate of decline in lung

function, although cessation of smoking does slow the progression of COPD.

Co-morbidities

People with COPD, particularly current or past tobacco smokers, often suffer from other related diseases and conditions. These may include concurrent conditions such as heart and blood vessel diseases, and various cancers.

Recurrent bouts of chest infections, pneumonia and allergies are usual among people with COPD, which complicate the symptoms and treatment of the disease.

It is common for COPD sufferers to experience depression and anxiety caused by the distressing symptoms of the disease, its prognosis and the limitations it imposes. Studies have shown that between 30% and 96% of people with COPD display anxiety, depression, panic, confusion or neurosis (ALF 2001:6).

Risk factors

Risk factors for COPD can be categorised first into predisposing factors, and second into environmental and behavioural factors (Box 2.8.1)

Heredity plays a role in predisposing some people to COPD. For example, the deficiency of the enzyme alpha-1-antitrypsin (a genetic trait) can lead to the development of emphysema. Airway hyper-responsiveness (over-reactive airways) is another genetically determined trait that contributes to the development of COPD.

Tobacco smoking is overwhelmingly the strongest risk factor for COPD. It has been estimated that about 77% of deaths from COPD are attributable to smoking (AIHW: Mathers et al. 1999). The majority of COPD sufferers have a long history of tobacco smoking. When inhaled, the smoke paralyses the microscopic hairs (cilia) lining the bronchial tubes. Irritants trapped in the mucus remain in the bronchial tubes and can inflame the bronchial membranes, eventually resulting in chronic obstruction.

Tobacco smoking also increases the severity of the disease. In smokers with COPD, lung function tends to deteriorate more rapidly than in non-smokers. If a smoker stops smoking before serious COPD develops, the rate at which the lung function declines from then on is equal to that for people without the disease. Unfortunately, because some lung damage cannot be reversed, lung function is unlikely to return completely to normal (NHLBI 1995).

Environmental agents, including air pollutants and occupational dusts and chemicals, may contribute to the risk of COPD, either independently or in addition to tobacco smoking (Goldring et al. 1993:389). There is some recent evidence that air pollution levels may be linked to hospital admissions for exacerbations of COPD, although the effect is small (Anderson et al. 1997). Several analyses from cities with high air pollution levels suggest that hospital admissions and mortality due to COPD can vary according to the level of certain pollutant chemicals (Bates & Sizto 1987).

COPD symptoms can be greatly aggravated by chest and viral infections such as the common cold, influenza and pneumonia.

Box 2.8.1: Risk factors for COPD

Predisposing factor

Heredity

Environmental and behavioural factors

Tobacco smoking

Pollution (in the workplace or elsewhere)

Chest and viral infections

Source: Crompton et al. 1999 .

Impacts

Deaths

There were 5,575 deaths in 1998 with COPD listed as the underlying cause of death. It was the fourth leading cause of death among males (40 per 100,000) and sixth most common cause of death among females (17 per 100,000).

COPD is not a significant contributor to mortality among young people. It affects mostly older people, reflecting largely the lifelong exposure to external risk factors, especially smoking. Death rates increase significantly among those aged 70 and over, in particular males (Figure 2.8.1).

In Australia, the death rate attributable to COPD increased steadily from the 1950s, peaking in the early 1970s. Since 1970 the male death rate for COPD has, in general, decreased, but the female death rate has increased steadily (Figure 2.8.2). The increase in the female rate could be the delayed outcome of an increase in the proportion of female smokers, from the late 1970s to mid-1980s (AIHW 2000:95).

Prevalence

The Australian Burden of Disease and Injury Study has estimated that in 1996 there were almost 300,000 people with COPD in Australia, with more than 20,000 new cases every year (AIHW: Mathers et al. 1999). The prevalence was higher in males compared with females: 1,940 per 100,000 males compared with 1,300 per 100,000 females in 1996.

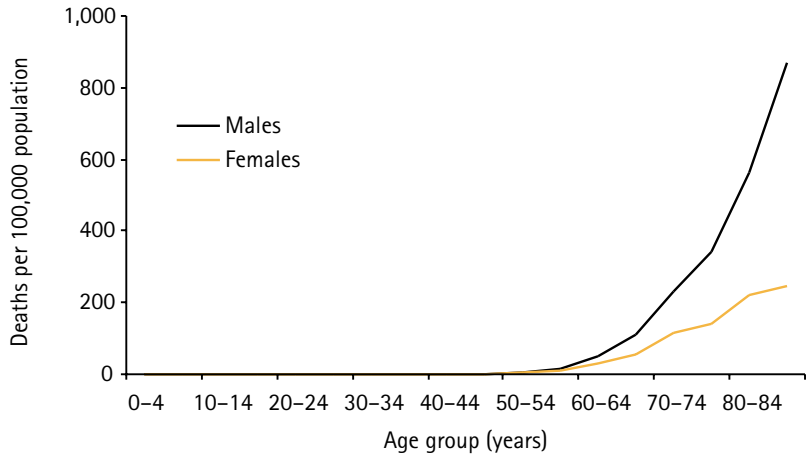
A different estimate put the prevalence much higher, estimating 474,000 cases of moderate to severe COPD (ALF 2001). This latter figure was derived by applying a population-based assessment model (used by the World Health Organization to estimate prevalence in developing countries) to Australian population data.

Obtaining comparable prevalence (and any other health-related data) for COPD is difficult. There are differences in the way the disease is defined, with the only consensus being to include chronic bronchitis and emphysema. Available data (often based on self-reports) are likely to underestimate the actual prevalence because COPD is often not diagnosed until it begins to impair a person's lifestyle and is moderately advanced.

Complications

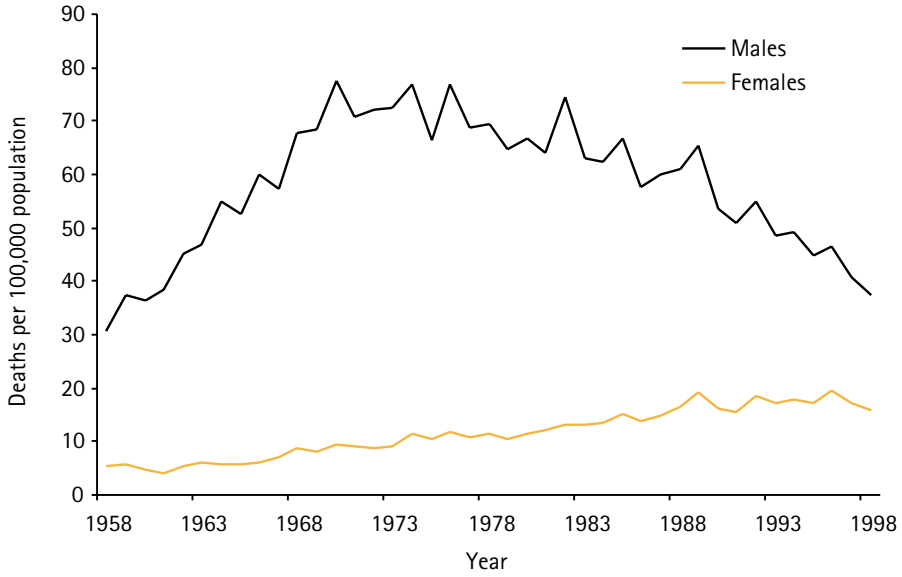
As COPD progressively inhibits normal lung function, other organ systems are threatened. Low blood oxygen (hypoxaemia) due to insufficient oxygen being absorbed into the

Figure 2.8.1: Age-specific death rates for COPD, 1998



Source: AIHW National Mortality Database.

Figure 2.8.2: Death rates for COPD, 1958 to 1998



Source: AIHW National Mortality Database.

blood, can lead to increased blood pressure in the lungs. This overtaxes the right side of the heart, which pumps blood to the lungs, leading to a form of heart failure known as cor pulmonale (NHLBI 1995).

When there is inadequate oxygen in the blood, the body increases production of oxygen-carrying red blood cells to compensate for the deficiency (secondary polycythemia). The excess number of red cells can thicken the blood so much that it clogs the small blood vessels, therefore restricting blood flow (NHLBI 1995).

Another complication of COPD is pneumothorax, which occurs when the lung develops air-filled 'blisters' (bullae) that puncture, allowing air to escape into the surrounding spaces (pleura). As the air accumulates in these spaces, it causes pressure on the lung and sudden severe pain on one side of the lung accompanied by shortness of breath (Crompton et al. 1999).

Osteoporosis may develop in some people with COPD, often caused by the long-term use of oral corticosteroids, a medication prescribed to reduce the inflammation of the air passages. Diabetes may also develop as a side effect of steroid use.

Disability

People with COPD typically experience increasing shortness of breath, which can be very disabling, for months or years before dying. As the disease advances, breathlessness may occur walking up stairs and eventually walking slowly along flat ground, and normal daily activities become more difficult. Often people have to sleep in a semi-sitting position because they are unable to breathe when they lie down. They often complain that they wake up during the night with a choking sensation, and need to sit up to cough (NHLBI 1995).

Most people with COPD are no longer capable of productive work within 7 to 8 years of initial diagnosis (Goldring et al. 1993).

Use of health services

In 1999–00, there were 48,583 hospital separations for COPD (0.8% of all separations). COPD accounted for 15% of all hospital

separations for diseases of the respiratory system. It contributed 377,407 bed days, with an average length of stay of 7.7 days.

The rate of hospital usage for COPD increases with age, particularly after the age of 45 reflecting largely the pattern noted in age-specific death rates. Males are hospitalised for COPD much more frequently than females, although the rates for the two sexes are very similar among those under 45 years of age (Figure 2.8.3).

The total health system costs of chronic bronchitis and emphysema (the two prominent COPD diseases) in 1993–94 were estimated to be $300 million (AIHW 2000), almost three times as much as for lung cancer ($107 million). The major costs were hospital services ($112 million or 37% of total costs), followed by pharmaceutical ($66 million, or 22%) and medical costs ($61 million, or 20%).

Management

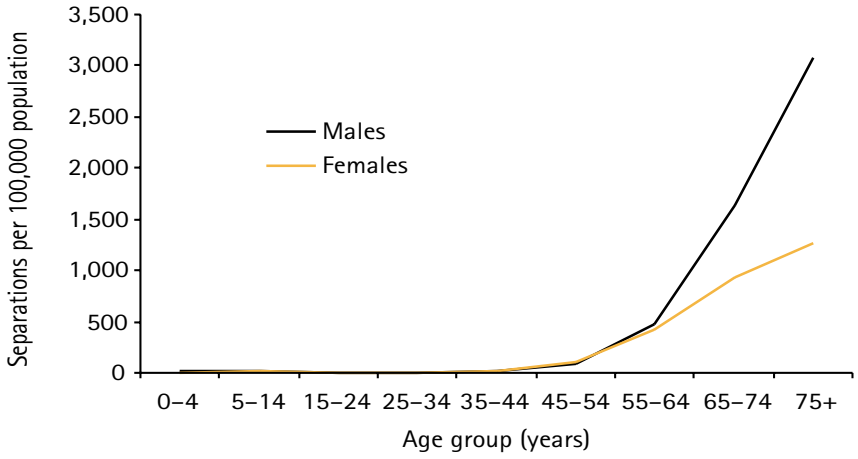
COPD cannot be cured, but therapy can often relieve symptoms and control potentially fatal exacerbations. It may also slow progression of the disease. Treatment must be directed at alleviating conditions that cause symptoms and excessive disability such as chest infections, asthmatic episodes and limitation of physical activity.

Depending on the severity of COPD, prescribed medications may include bronchodilators, which help open narrowed air passages; corticosteroids, which lessen inflammation of the air passage walls; and antibiotics, prescribed to fight infection. People with COPD may eventually require supplemental oxygen (NHLBI 1995).

For some people in the advanced stages of COPD, lung transplantation has been successful. In some sufferers it is beneficial to surgically remove the large air-filled 'blisters' (bullae) that form because of COPD (Crompton et al. 1999).

Since management of COPD is essentially about improving quality of life for the sufferer, the most effective intervention is for permanent cessation of smoking as this will slow the disease process (ALF 2001). The avoidance of work-related exposures to dusts and fumes, air pollution (including environmental tobacco

Figure 2.8.3: Age-specific hospital separation rates for COPD, 1999–00



Source: AIHW National Hospital Morbidity Database.

smoke), excessive heat and cold, and very high altitudes (such as flying, as this may cause hypoxaemia) is also recommended.

The control of complications caused by low blood oxygen, such as high blood pressure in the lungs (pulmonary hypertension) and cor pulmonale, is important in the management of COPD.

Respiratory infection in people with COPD should be treated immediately because it aggravates breathlessness and may lead to respiratory failure among advanced cases (Crompton et al. 1999). COPD sufferers should also refrain from intimate contact with those who have respiratory infections such as the common cold or influenza. Sufferers should consider vaccination against pneumonia and influenza (NHLBI 1995).

General exercise and maintenance of a healthy diet is considered necessary to successfully manage COPD. Also, consuming copious amounts of fluids will help keep the sputum loose so that it can be brought up by coughing.

Prevention

Since smoking has such an important role in the initiation and progression of COPD, smoking cessation is the central preventive strategy. Exposure to environmental tobacco smoke (ETS) should also be avoided whenever possible. Research suggests that exposure to ETS can contribute to COPD if combined with other risk factors such as past smoking (NHLBI 1995).

In addition to smoking as the most common cause of COPD, dust from grain, cotton, wood, mining and other sources or irritant chemical gases may contribute to COPD. Exposure to these factors should therefore be minimised, especially in those who already have COPD, and this may require a change of occupation.

For COPD, the risks from various factors appear to be additive. Therefore it is important to identify the risk factors that occur concurrently such as smoking, exposure to dust particles in the workplace or other irritants, to ensure that the disease is prevented successfully.

References

AIHW 2000. Australia's health 2000. AIHW Cat. No. AUS 19. Canberra: AIHW.

AIHW: Mathers C, Vos T & Stevenson C 1999. The burden of disease and injury in Australia. AIHW Cat. No. PHE 17. Canberra: AIHW.

ALF (Australian Lung Foundation) 2001. Case statement: chronic obstructive pulmonary disease (COPD). Brisbane: ALF.

Anderson H, Spix C, Medina S et al. 1997. Air pollution and daily admissions for chronic obstructive pulmonary disease in six European cities: results from the APHEA project. European Respiratory Journal 10: 1064–71.

Bates D & Sizto R 1987. Air pollution and hospital admissions in Southern Ontario: the acid summer haze effect. Environment Research 79: 69–72.

Crompton G, Haslett C & Chilvers E 1999. Diseases of the respiratory system. In: Haslett C, Chilvers E, Hunter J et al. (eds), Davidson's principles and practice of medicine, 18th edition. Edinburgh: Churchill Livingstone, 303–91.

Fletcher C, Peto R, Tinker C et al. 1976. The natural history of chronic bronchitis and emphysema. Oxford: Oxford University Press.

Goldring J, James D & Anderson H 1993. Chronic lung disease. In: Brownson R, Remington P, David J (eds), Chronic disease epidemiology and control. Washington: American Public Health Association, 375–420.

NHLBI (National Heart, Lung, and Blood Institute) 1995. Chronic obstructive pulmonary disease. NIH Publication No. 95–2020. Bethesda: National Institutes of Health.

2.9 Chronic renal disease

Chronic renal (kidney) disease is a debilitating and irreversible disorder in which kidney functions become progressively worse. The disease contributes substantially to mortality and disability in Australia and is especially prominent in Indigenous populations.

Chronic renal disease contrasts with acute renal disease which occurs when kidney function suddenly slows or stops altogether. Acute renal disease may progress to a chronic stage and permanent loss of kidney function, but typically is reversible.

Mortality reported here for chronic renal disease is defined by the following ICD-9 codes: 403, 581–583, 585–587, 590–592, 593.2, 593.8 and 593.9; hospital separations are covered by the ICD-10-AM code Z49 'care involving dialysis'.

Description

Kidneys play a vital role in regulating the balance of water and certain substances such as sodium and potassium in the blood. They do this by acting as a filter of the blood, with excess and unwanted water and other materials being allowed to pass into the bladder as urine. The kidneys also help regulate blood pressure and production of red blood cells.

Renal disease can be caused by many conditions that damage the kidneys. These include an inflammatory process known as glomerulonephritis, diabetes, long-term high blood pressure and kidney infections. Although many kidney disorders may be successfully treated and therefore have no long-term consequence, some remain unresolved and become chronic.

Commonly, the loss of kidney function follows a path from renal insufficiency to renal failure, and progresses eventually to end-stage renal disease (ESRD). Various stages of renal disease are described in Box 2.9.1.

Box 2.9.1: Stages of renal disease

Renal insufficiency: *The initial phase of renal disease is renal insufficiency. There are typically no symptoms and it may go undetected to renal failure within months or over an extended period. Renal insufficiency is typically detected by abnormal levels of albumin and creatinine.*

Renal failure: *In this phase, there is a gradual loss of kidney functions; problems may include anaemia, and salt and fluid retention. Renal failure can be detected by abnormal albumin and creatinine levels or by tests showing lowered filtering capacity of the kidneys.*

End-stage renal disease (ESRD): *In this stage, there is a severe reduction in kidney function such that dialysis or kidney transplantation is necessary to maintain life. ESRD is irreversible and incurable with attendant high morbidity and mortality.*

Signs and symptoms

Initial signs of renal disease may include unintentional weight loss, nausea, vomiting, general ill feeling, fatigue, headache, frequent hiccups and generalised itching (pruritus). Further symptoms can include anaemia (a deficiency of circulating red blood cells or

haemoglobin), high blood pressure, excessive urine passing, escape of certain proteins in the urine and a build up of certain substances in the blood, all of which may lead to other complications. For example, high blood potassium levels may lead to abnormal heart rhythms, high uric acid levels to gout, and retention of phosphate and abnormal calcium levels to bone disease.

Co-morbidities

Common co-morbidities for renal disease include heart disease, peripheral vascular disease, high blood pressure and diabetes (see Box 2.9.2). Many of these conditions, such as high blood pressure and diabetes, not only coexist or are found additional to renal disease, but are also common complications due to impaired renal function.

During 1996–99, almost 40% of persons beginning treatment for ESRD were confirmed or suspected to also have coronary artery disease, 28% had peripheral vascular disease, 17% had chronic lung disease and 16% had cerebrovascular disease. Diabetes was present in 26% of new cases (Disney et al. 2000).

Box 2.9.2: Common co-morbidities for renal disease

Heart and vascular diseases
Chronic lung disease
Diabetes
High blood pressure

Source: Disney et al. 2000.

Risk factors

A number of factors contribute to the development and progression of chronic renal disease including predisposing, biomedical and lifestyle factors (Box 2.9.3). Many of these risk factors may exist simultaneously.

Box 2.9.3: Risk factors for renal disease

Predisposing factors
Heredity

Biomedical factors
Glomerulonephritis
Diabetes
High blood pressure

Lifestyle factors
Excess weight
Infections
Injury
Long-term use of analgesic compounds and related agents

Sources: Briganti et al. 1999; Disney et al. 2000.

Some kidney disorders are inherited or are congenital conditions. Polycystic kidney disease for example is an inherited condition in which many cysts form in the kidneys, slowly replacing the mass of the kidneys, and thus leading to kidney failure.

Glomerulonephritis, a group of kidney diseases caused by inflammation and gradual, progressive destruction of the internal kidney structures, is the leading cause of renal failure in Australia. Glomerulonephritis may be triggered in some persons by exposure to stimuli such as streptococcal infections.

Diabetes is the second most common cause of deterioration of the kidneys. Diabetes damages both the small and large blood vessels in the body, thus affecting kidney functions. Good control of blood sugar levels in diabetics can delay or avoid damage to kidney functions (AKF 2000).

High blood pressure, a controllable and reversible risk factor, may also lead to kidney impairment. Increased pressure in the arteries and vessels of the kidneys may cause them to

become narrowed and thickened, impairing the filtering ability of the vessels. In persons with diabetes, high blood pressure further increases the risk of damage to the blood vessels of the kidney.

Excess body weight, especially when found in conjunction with both diabetes and/or high blood pressure, also increases the risk of renal disease, as do infections such as skin sores and scabies. Acute injury, such as extremely low blood pressure following trauma, complicated surgery, septic shock, haemorrhage, burns and associated dehydration, or other severe or complicated illness may lead to acute renal failure. In some cases this may not resolve, and therefore become chronic. Long-term use of various analgesic compounds and related agents are also causal to kidney harm and there is some evidence that smoking accelerates kidney disease.

Impacts

Information on the impact of chronic renal disease in Australia is currently limited, due primarily to lack of data. As noted earlier, kidney disease may go undetected for many years and therefore may not be fully reflected in incidence and prevalence data. Also, the high rate of complications associated with chronic renal disease may lead to deaths from kidney dysfunction to be coded to underlying causes of death other than the kidney impairment per se.

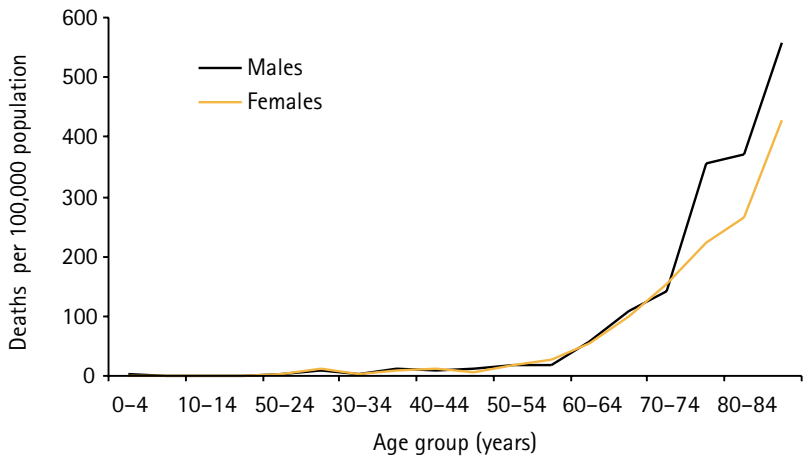
Deaths

Chronic renal disease is a significant contributor to mortality in Australia. In 1998, chronic renal disease accounted for 1,852 deaths (823 males and 1,029 females), with death rates of 9.7 and 7.6 per 100,000 males and females respectively.

The majority of deaths for chronic renal disease are in older age groups, with death rates for both sexes rising steeply after age 60. Although death rates for chronic renal disease are similar in both sexes until the age of 75, the male death rate begins to ascend above that of females at this point (Figure 2.9.1).

The greater burden of mortality in older age groups reflects both the long latency period of disease development and improvements in management. Persons treated by dialysis generally survive longer, albeit with disability.

Figure 2.9.1: Age-specific death rates for chronic renal disease, 1998



Source: AIHW National Mortality Database.

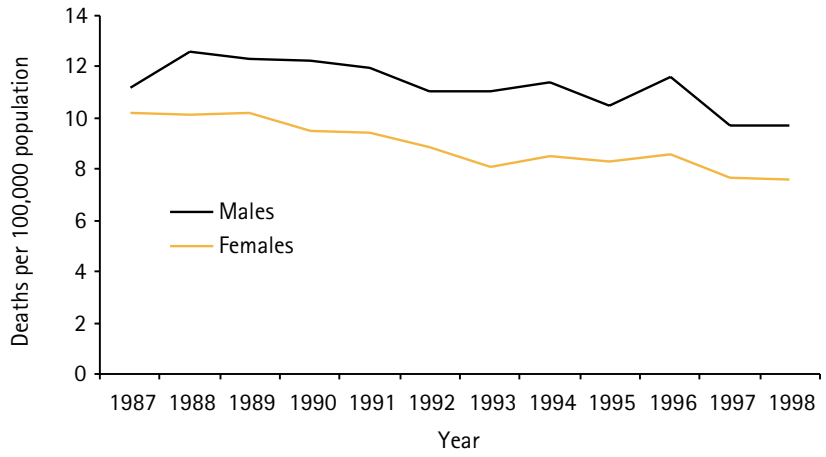
Deaths following chronic renal disease have decreased in both males and females in recent years (Figure 2.9.2). The decrease is most likely due to improved treatment and management extending the lifespan of persons with the disease.

Incidence

The incidence of chronic renal disease is difficult to determine. However, the Australian and New Zealand Dialysis and Transplant Registry (ANZDATA) provides a source of information to estimate incidence for ESRD through persons that begin dialysis or receive kidney transplants.

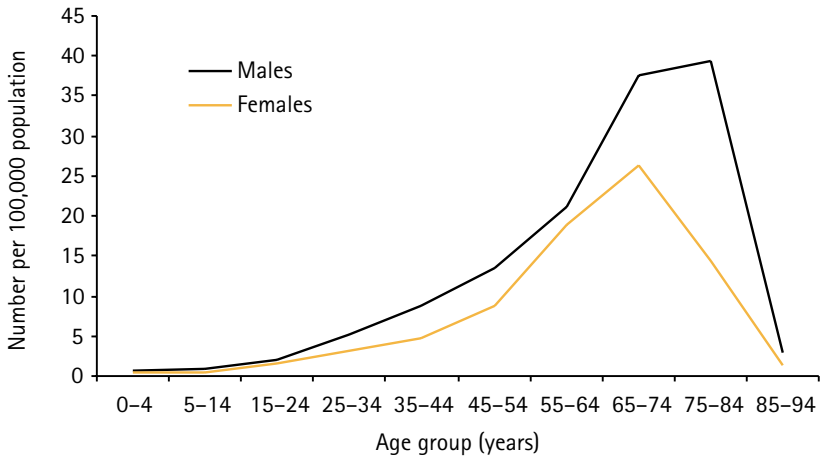
In 1998, a total of 1,589 persons began treatment for ESRD (Disney et al. 1999). Of these, 940 were males (59%) and 649 females (41%). The average age of new cases was 57 with the largest number in the 65–74 age group. Incidence peaked among males aged 75–84 (39 per 100,000) and among females aged 65–74 (26 per 100,000) (Figure 2.9.3).

Figure 2.9.2: Death rates for chronic renal disease, 1987 to 1998



Source: AIHW National Mortality Database.

Figure 2.9.3: Age-specific incidence of ESRD, 1998



Source: Disney et al. 1999.

The death rates for renal disease are decreasing, but the ESRD incidence is now on the rise. There has been a steady increase in new cases of ESRD in Australia, rising from 6.2 per 100,000 in 1992 to 8.5 per 100,000 in 1998.

Prevalence

By the end of 1998, a total of 10,403 persons were being treated for ESRD in Australia. These included 4,880 people with functioning transplants and 5,523 persons dependent on dialysis. The numbers, both of transplants and of persons dependent on dialysis, have increased steadily in the 10 years to 1998 (Figure 2.9.4).

The increase in the total number of people with functional transplants is due mainly to a marked improvement in the survival of kidney transplants. On the other hand, the increasing prevalence of persons reliant on dialysis is due in part to older patients beginning dialysis. In 1998, 38% of new patients were aged 65 and over, compared to 22% in 1989. A levelling off in the number of kidneys available for transplant in the 1990's has also contributed to the increase in persons undertaking dialysis (Disney et al. 2000).

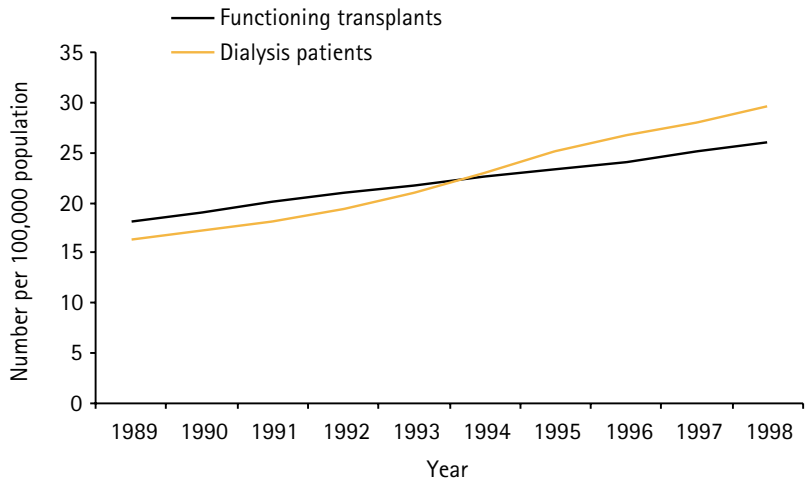
Chronic renal disease in Indigenous populations

There are large disparities in the burden of chronic renal disease between Indigenous and non-Indigenous Australians. In the Northern Territory, epidemic levels of renal failure are reported in the Indigenous population (Spencer et al. 1998).

In 1997–99, there were 78 deaths in Western Australia, South Australia and the Northern Territory of Indigenous persons, where renal failure was the underlying cause (identification of Indigenous persons in death records in other jurisdictions is not of sufficient quality for reporting). Indigenous persons whose deaths were due to renal failure are typically of relatively younger ages compared to non-Indigenous deaths (ABS 2001: 91).

Currently, new cases of ESRD in Indigenous populations are reported to be doubling every 4 years (Spencer et al. 1998). In 1998, 131 Indigenous Australians began dialysis or

Figure 2.9.4: Trends in the numbers of functioning transplants and dialysis patients, 1989 to 1998



Source: Disney et al. 1999.

received kidney transplants, accounting for about 8% of all new cases (Disney et al. 1999). In the Northern Territory, Indigenous persons accounted for 74% of new ESRD cases, yet account for only 28% of the Territory's population.

As at December 1999, there were 535 Indigenous persons with ESRD, 5% of all those registered with ANZDATA. Of these, 434 were dependent on dialysis and a further 101 were living with functioning transplants.

A large proportion of cases of kidney disease in Indigenous persons have diabetes as the underlying cause. In 1998, diabetic nephropathy contributed 42% of the Indigenous dialysis group, compared with 22% in the total dialysis groups (Disney et al. 1999).

Complications

Persons with chronic renal disease are at increased risk for severe anaemia, which reduces the blood oxygen carrying capability. Another common complication is an increased susceptibility to infection. Bone disorders such as the serious condition of renal osteodystrophy can also occur as a result of renal failure.

Box 2.9.4: Common complications of renal disease

Anaemia
Infections
Bone disorders
Heart failure
High blood pressure
Transplantation-related complications

Source: Disney et al. 2000.

There are also a range of complications following dialysis and kidney transplantation. These include heart failure, high blood pressure, abnormal bleeding, and liver, blood and other problems associated with transplant rejection.

Use of health services

Chronic renal disease is a long-term, debilitating, costly and life-threatening disease that requires intensive management, and therefore places a disproportionately large burden on the health system. Persons with chronic renal disease, in particular those who have progressed to ESRD, account for a large proportion of the use of health services. For example, in 1999–00 'care involving dialysis' was the single greatest reason for hospital separations; there were 535,396 separations recorded for this particular diagnosis, almost 10% of all separations.

In 1993–94, the direct costs of kidney diseases in Australia were an estimated $81 million, 0.25% of total health system costs. This included approximately $46 million in hospital costs, $6.6 million in GP and specialist costs, and $1.5 million in pharmaceutical costs (AIHW: Mathers et al. 1998). A further $135 million was attributable to unspecified treatments and procedural costs, including the cost of dialysis.

Treatment and management

In the early stages of renal disease, treatment and management focus on delaying progression to ESRD and controlling complications. The most important interventions to slow progression are good blood pressure and ACE inhibitors. Other management involves controlling blood glucose levels, low-protein

diets and maintaining healthy levels of cholesterol in the blood, all in combination with medication. Management is aimed at reducing strain on the urinary system by minimising the volume of urine produced and preventing a large load of waste products.

Diet is important in delaying the progression of established renal disease. Recommended diets for persons with kidney disease are usually low in sodium, low in potassium and protein, and fluid controlled. This reduces the load on the kidneys, thus preventing toxic build up of waste products in the blood.

Once progression to ESRD has occurred, two major treatment options are available to sustain life, dialysis and kidney transplantation. Dialysis is a method of removing excess water and waste products from the blood when the kidneys no longer can. Dialysis is typically combined with a diet to reduce the wastes that build up in the blood and to maintain nutritious elements. Transplantation involves the surgical replacement of a patient's kidney with one from a donor.

Dialysis aids the survival of patients with ESRD but the greatest chance for long-term survival lies in transplantation. In recent years there have been improvements in the survival of persons with kidney transplants. Of people who had a kidney transplant in 1983, 58% had a functioning transplant 5 years later, increasing to 73% of those who had a transplant in 1993 (AIHW 2000:106).

Prevention

Prevention of renal disease may be facilitated by a number of lifestyle and medical choices. Lifestyle factors include weight and diet control; medical factors include control of biomedical risk factors for renal disease such as high blood pressure.

Some preventive factors aim to avoid the onset of primary kidney disease while others aim to slow the progression of renal damage. These include careful control of glucose and blood pressure levels in persons with diabetes, and blood pressure control in persons with high blood pressure. Diets high in fresh fruit and vegetables, and with adequate fluid intake ease the burden on the kidneys.

References

ABS & AIHW 2001. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples. ABS Cat. No. 4704.0. AIHW Cat. No. IHW 6. Canberra: ABS & AIHW.

AIHW 2000. Australia's health 2000. AIHW Cat. No. AUS 19. Canberra: AIHW.

AIHW: Mathers C, Penm R, Carter R et al. 1998. Health system costs of diseases and injury in Australia 1993–94. AIHW Cat. No. HWE 5. Canberra: AIHW.

AKF (Australian Kidney Foundation) 2000. Living with kidney failure. Available from Internet: URL: http://www.kidney.org.au (cited 20 June 2001).

Briganti E, McNeil J & Atkins R (eds) 1999. The epidemiology of diseases of the kidney and urinary tract: an Australian perspective. Victoria: Australian Kidney Foundation.

Disney A, Sheil A, Collins J et al. (eds) 1999. ANZDATA Registry report 1999: the twenty-second report. Adelaide: ANZDATA.

Disney A, Collins J, Kerr P et al. (eds) 2000. ANZDATA Registry report 2000: the twenty-third report. Adelaide: ANZDATA.

Spencer J, Silva D, Snelling P et al. 1998. An epidemic of renal failure among Australian Aboriginals. Medical Journal of Australia 168:537–41.