Chronic kidney disease in Australia

2005

The Australian Institute of Health and Welfare is Australia's national health and welfare statistics and information agency. The Institute's mission is *better health and wellbeing for Australians through better health and welfare statistics and information*.

Please note that as with all statistical reports there is the potential for minor revisions of data in *Chronic kidney disease in Australia, 2005* over its life. Please refer to the online version at <www.aihw.gov.au>.

Chronic kidney disease in Australia, 2005

October 2005

Australian Institute of Health and Welfare
Canberra

AIHW Cat. No. PHE 68

A complete list of the Institute's publications is available from the Business Promotion and Media Unit, Australian Institute of Health and Welfare, GPO Box 570, Canberra ACT 2601, or via the Institute's website <http://www.aihw.gov.au>.

ISBN 1 74024 508 3

Suggested citation

AIHW 2005. Chronic kidney disease in Australia, 2005. AIHW Cat. No. PHE 68. Canberra: AIHW.

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

Tracy Dixon
Australian Institute of Health and Welfare
GPO Box 570
Canberra ACT 2601

Phone: (02) 6244 1103

Published by Australian Institute of Health and Welfare

Printed by

Table of contents

List of tables

List of figures

Acknowledgments

This report was prepared and written by Bin Tong and Tracy Dixon.

The authors particularly wish to thank the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry for providing special data extractions on treated end-stage kidney disease and kidney replacement therapy used to produce this report. The assistance provided by Stephen McDonald and Victoria Shtangey from the ANZDATA Registry is greatly appreciated. The interpretation and reporting of these data are the responsibility of the authors and in no way should be seen as an official policy or interpretation of the Australia and New Zealand Dialysis and Transplant Registry.

The extensive input received from Dr Alan Cass (The George Institute for International Health), Dr Steve Chadban (Royal Prince Alfred Hospital), Dr Stephen McDonald (ANZDATA Registry) and Dr Timothy Mathew (Kidney Health Australia) is greatly appreciated.

The authors also wish to thank Garry Waller (National Centre for Classification in Health) for valuable assistance with developing the ICD-10 (International Classification of Diseases, 10th revision) coding list, and Dr Helena Britt and Dr Graeme Miller for assistance with ICPC-2 (International Classification of Primary Care, second edition) coding and use of Bettering the Evaluation and Care of Health (BEACH) survey data.

AIHW staff including Dr Fadwa Al-Yaman, Dr Kuldeep Bhatia, Nicola Bowman, Dr Ching Choi, Jenny Hargreaves, Robert van der Hoek, Dr Paul Magnus, Dr Paul Meyer, Lynelle Moon, and Dr Perri Timmins provided advice and comments on this report. Staff of the Australian Government Department of Health and Ageing, including Dr Joy Eshpeter and Bonnie Field, also provided helpful comments.

The health expenditure data used in this report was provided by John Goss and Nick Mann of the AIHW Summary Measures Unit.

The Australian Government Department of Health and Ageing provided funding to assist in the production of this report.

Abbreviations

ABS	Australian Bureau of Statistics
ACE	angiotensin converting enzyme
AIHW	Australian Institute of Health and Welfare
ANZDATA	Australia and New Zealand Dialysis and Transplant Registry
APD	automated peritoneal dialysis
AusDiab	Australian Diabetes, Obesity and Lifestyle study
BEACH	Bettering the Evaluation and Care of Health (survey)
BMI	body mass index
CAPD	continuous ambulatory peritoneal dialysis
CARI	Caring for Australians with Renal Impairment
CKD	chronic kidney disease
COPD	chronic obstructive pulmonary disease
ESKD	end-stage kidney disease
ESRD	end-stage renal disease
GFR	glomerular filtration rate
GP	general practitioner
HD	haemodialysis
ICD-10-AM	International Classification of Diseases, 10th Revision, Australian Modification
ICPC-2	International Classification of Primary Care, second edition
K/DOQI	(US) Kidney Disease Outcome Quality Initiative
MDRD	Modification of Diet in Renal Disease (formula)
NHS	National Health Survey
NKF	National Kidney Foundation of America
PD	peritoneal dialysis
PKD	polycystic kidney disease
URR	urea reduction ratio
USRDS	United States Renal Data System
WHO	World Health Organization

Executive summary

Chronic kidney disease (long-term and usually irreversible loss of kidney function) has impacts on quality of life, use of health services, health expenditure and mortality, but it is difficult to determine how many Australians are affected. Because of a lack of specific symptoms at the early stages, the diagnosis of chronic kidney disease is often delayed or missed. However, it may lead to serious illness and death from complications or comorbid conditions before it is even detected. In 2003, chronic kidney disease was recorded as the underlying cause of death in 2,431 cases and an associated cause of death in a further 9,217 cases.

In severe cases, a person's kidney function will deteriorate so much that it will no longer be sufficient to sustain their life. These people are said to have 'end-stage kidney disease', and require kidney replacement therapy—dialysis or a kidney transplant—to survive. At the end of 2003, a total of 13,625 people with end-stage kidney disease were reliant on kidney replacement therapy. The number of people receiving this treatment has more than tripled over the last 20 years, and is still growing. Care involving dialysis accounted for 11% of all hospital separations in 2003–04. Although relatively few people with chronic kidney disease require this treatment, the personal, social and economic costs relating to end-stage kidney disease make it an important public health issue.

A variety of factors, many of which are common in Australia, can increase the risk of developing chronic kidney disease. Some of these include diabetes, high blood pressure and smoking. Although it mainly affects the older population, chronic kidney disease can occur among people of any age. As there is no cure for this illness, reducing the burden of chronic kidney disease relies heavily on its prevention and management. With advanced technology and better management, the outcomes of treatment have improved, especially for people receiving kidney replacement therapy. However, not all aspects of prevention, early detection and management are covered by national programs.

Although chronic kidney disease has been a health issue for many years, it is only recently that a clear definition and conceptualisation of the disease has been developed. Many crucial issues relating to the disease remain unclear, and are under investigation and debate. There is a general lack of information on chronic kidney disease in Australia. There is no national monitoring system for chronic kidney disease, and regular information is collected and reported only for people receiving kidney replacement therapy.

Although the impacts of chronic kidney disease are substantial and the number of Australians at risk is increasing, chronic kidney disease is preventable and treatable in many cases, and there is great potential to reduce the burden of the disease.

Chronic kidney disease in Australia, 2005 is the first national report on this disease. The report compiles the latest information from a variety of data sources, and presents information on levels of kidney damage, reduced kidney function and end-stage kidney disease in the population, the factors that contribute to chronic kidney disease, and treatment and prevention programs. At times, limitations in knowledge and national information on chronic kidney disease have restricted the content and coverage of issues that are essential to a comprehensive understanding of the disease. Nevertheless, this report provides an opportunity to look at chronic kidney disease systematically, and also provides valuable baseline information for further monitoring of this disease in the future.

1 Introduction

Chronic kidney disease (CKD) is marked by long-term and usually irreversible loss of kidney function. It may have severe health outcomes. For people who develop end-stage kidney disease, treatment is expensive and requires intensive health services.

People with CKD are at risk of developing various complications and comorbidities, including cardiovascular diseases, respiratory system infections, bone and muscle problems, and anaemia. These problems can begin at a very early stage, even before CKD is detected, and the risks increase with the severity of CKD.

Many of the factors that increase a person's risk of developing CKD are common in Australia. With an ageing population and increasing prevalence of some risk factors, the number of Australians at risk of CKD is increasing. Nevertheless, effective prevention of CKD is possible as many of these risk factors are modifiable.

The kidneys

The kidneys are two bean-shaped organs located at the back of the abdomen. Each is about the size of a fist. They act as the body's filters, controlling the level of water and various chemicals, producing certain essential hormones, and clearing waste products from the blood. The waste products and any excess water are eliminated from the body through the bladder in the form of urine. When the kidneys do not work effectively, the body's chemical balance may be changed, essential bodily processes may be disrupted, and waste products may build up in the blood. This causes damage to the body's organs and systems, and may result in a range of serious complications.

How are kidney problems detected?

Initial evidence of kidney damage or reduction in kidney function can be detected through routine blood or urine testing. A blood test might find excess levels of waste products that are normally passed into the urine, or a urine test find blood or substances from the blood, that would normally not leak out of the kidneys. The most common indicators of kidney damage are proteins in the urine (proteinuria or albuminuria), blood in the urine (haematuria) and raised levels of urea or creatinine (a waste product of protein metabolism) in the blood.

Kidney function is measured by the glomerular filtration rate (GFR). The glomeruli are networks of blood vessels in the kidneys where the blood is filtered and waste products are removed. The glomerular filtration rate is a measurement of the amount of blood the kidneys clear of waste products in one minute.

Chronic kidney disease

The US Kidney Disease Outcome Quality Initiative (K/DOQI) has developed a definition and clinical guidelines for chronic kidney disease, which were published by the National Kidney Foundation of America (NKF) in 2002. This definition has been widely accepted by

the kidney community in Australia and around the world, and has been endorsed by Kidney Health Australia (Mathew & Johnson 2005).

According to the K/DOQI definition, the presence of chronic kidney disease should be established based on the occurrence of kidney damage and the level of kidney function, regardless of the specific diagnosis of diseases and conditions causing the damage. To be diagnosed with chronic kidney disease, a person must have evidence of kidney damage and/or reduced kidney function lasting for at least 3 months (Box 1.1).

Box 1.1: Diagnostic criteria for chronic kidney disease

In the clinical setting, a patient is diagnosed with CKD if he/she meets either of the following criteria:

1. *Kidney damage for 3 months or more, as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifest by either:*
   - *pathological abnormalities; or*
   - *markers of kidney damage, including abnormalities in the composition of the blood or urine, or abnormalities in imaging tests.*
2. *Glomerular filtration rate (GFR) <60 mL/min/1.73 m^2 for 3 months or more, with or without other markers of kidney damage.*

Source: Adapted from NKF 2002.

Severity of chronic kidney disease

The K/DOQI definition classifies chronic kidney disease into five stages of severity, based on evidence of kidney damage and the degree of kidney function reduction, classified by GFR (Box 1.2). Despite the diversity of causes of CKD, the functional changes and clinical manifestations are quite similar across the spectrum. Although the use of the word 'stage' implies progression of disease, it is important to note that treatment can slow, delay or prevent further kidney damage in many cases. The proportion of people with CKD who will progress from one stage to the next, and the rate of this progression, are unknown.

In some people with severe CKD, kidney function is insufficient to sustain life and death will follow in a short period (weeks to a month or two). These people are regarded as having 'end-stage kidney disease' (ESKD), and require dialysis or a kidney transplant to survive. People receiving dialysis or living with a kidney transplant are said to have 'treated ESKD'.

Development and course of chronic kidney disease

CKD is a complex disease. It can be caused by a variety of different diseases and conditions, and in most cases develops over a number of years. It is usually asymptomatic until the very late stages, but can lead to impairment of many different organs and body systems even from the very early stages. End-stage kidney disease requires specialised and expensive treatment.

A brief outline of the course of this disease (Figure 1.1) and its main features are provided below. These are discussed in more detail in the following chapters.

Box 1.2: Severity of chronic kidney disease

Stage 1: Kidney damage with GFR at least 90 mL/min/1.73 m²

People with stage 1 CKD have evidence of kidney damage (structural or functional abnormalities of the kidney), but without decreased GFR. There are usually no symptoms.

Stage 2: Kidney damage with GFR 60 to 89 mL/min/1.73 m²

People with stage 2 CKD have evidence of kidney damage with some reduction in GFR. Most patients at this stage have no symptoms. They usually have high blood pressure and may have laboratory abnormalities indicating dysfunction in other organs.

Stage 3: GFR 30 to 59 mL/min/1.73 m²*

People with stage 3 CKD have a significant reduction in GFR. They may or may not show other signs of kidney damage. Blood tests will show increased levels of urea and creatinine, and often there will be indications of dysfunction in other organs. Although patients may have symptoms, they often remain asymptomatic even though their kidney function may be reduced by as much as 70%.

Stage 4: GFR 15 to 29 mL/min/1.73 m²*

People with stage 4 CKD have severely reduced kidney function. Blood levels of urea and creatinine increase, and there is greater evidence of dysfunction in other organs. Patients usually have only mild symptoms.

Stage 5: GFR less than 15 mL/min/1.73 m²*

In most cases, stage 5 CKD is marked by a range of symptoms and laboratory abnormalities in several organ systems, which are collectively referred to as uraemia. Patients at this stage may need to be prepared for kidney replacement therapy (dialysis or transplant), which will be required when kidney function is no longer sufficient to sustain life.

** with or without evidence of kidney damage*

Source: Adapted from Obrador & Pereira 2002.

Complex causality and multiple risk factors

As with many other chronic diseases, the causes of CKD are complex and there are a number of factors that can increase risk. Kidney function can be damaged by a variety of causes, such as glomerulonephritis, polycystic kidney diseases, diabetes and high blood pressure. However, the pathways leading to CKD are not clear. CKD can also progress at different rates, depending on the underlying cause of the disease.

Although there is no information on the prevalence of different causes of CKD in general in Australia, there are data on the causes of end-stage kidney disease in people receiving kidney replacement therapy. In 2003, around 53% of cases of treated ESKD in Australia were caused by diseases occurring inside the kidneys and urinary tract. These include inflammation and infection of the kidneys, infections and blockages of the urinary tract, drug-induced kidney impairments, inherited kidney disorders and congenital malformations. More than 40% of cases resulted from complications of other diseases and conditions, mainly diabetes and high blood pressure. In around 7% of cases, the cause could not be determined (Excell & McDonald 2005b).

A number of factors contribute to the development and progress of CKD. Smoking and physical inactivity can significantly increase the risk of developing CKD. Poor nutrition and obesity also increase risk indirectly by influencing the development of biomedical risk factors such as Type 2 diabetes. Older people, people with a family history of CKD and Indigenous Australians also tend to have a greater risk of kidney damage (Chadban et al. 2003; Briganti et al. 2002; Hoy et al. 1998).

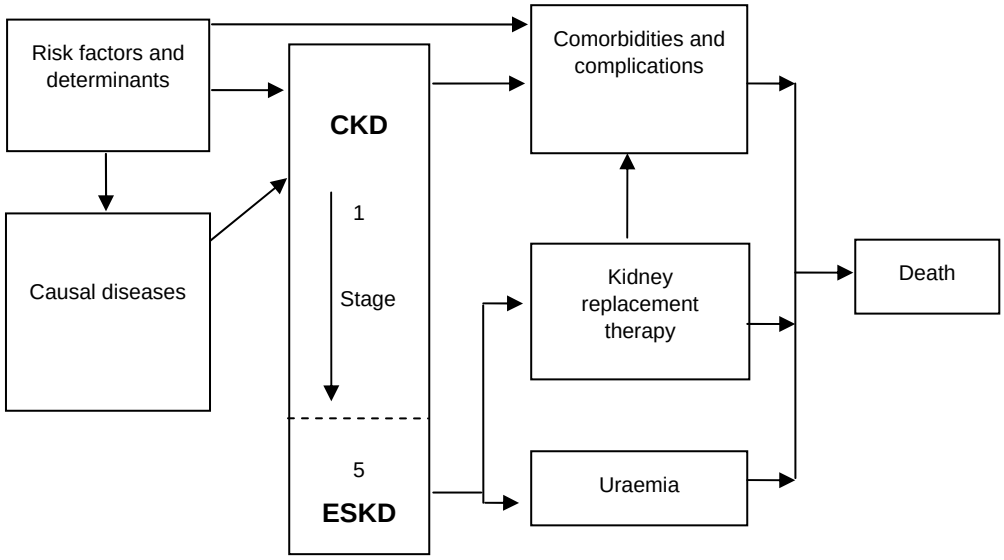


Figure 1.1: The course of chronic kidney disease

Asymptomatic nature of chronic kidney disease

In most cases of CKD, kidney function deteriorates over a period of years, but usually a person with CKD has no specific symptoms until the late stages. Initially the only signs of the disease are abnormal results of blood and urine tests (such as a raised blood urea and creatinine, or protein in the urine). A person can lose up to 85% of kidney function before even feeling sick. For this reason, the diagnosis of CKD is often delayed or missed.

Specialised treatment at end stage

In people with end-stage kidney disease, kidney function is no longer sufficient to sustain life. ESKD is one of the most severe outcomes of chronic kidney disease. Before the 1960s, death resulted very soon after reaching end-stage, but over the last 40 years there have been significant advances in our understanding of this condition and its treatment, especially kidney replacement therapy (dialysis and transplant). Since kidney replacement therapy became available and accessible, the lives of many people with ESKD have been prolonged. However, this treatment is still far from perfect. With kidney replacement therapy, the lives of patients with ESKD can be sustained, but the quality of their lives is much reduced and life expectancy is still shortened. Not all people with ESKD will agree to or be well enough to receive kidney replacement therapy.

Multi-organ impairment

ESKD is not the only health outcome of chronic kidney disease. CKD can affect all the organs and systems in the body. Although it is generally asymptomatic until the later stages, impairments of other organ systems due to poor kidney function occur and can be seen at the early stages, such as cardiovascular diseases, bone problems, anaemia and sleep apnoea (Johnson 2004). CKD has profound impacts on the circulatory system. There is substantial evidence that CKD is an independent risk factor for cardiovascular disease (Go et al. 2004).

The risks of damage to the circulatory system, as well as to other organs and systems, increase progressively with worsening kidney function. Many people with CKD do not develop ESKD or die from it directly, but die from complications of CKD, particularly heart disease and respiratory infections.

Prevention and management of chronic kidney disease

There is no cure for chronic kidney disease, but the disease is highly preventable and treatable, and progression can be slowed or stopped.

Although there are many factors that can increase the risk of developing CKD, there is strong evidence that many of these factors are modifiable and preventable. Although smoking reduces kidney function and increases the risk of kidney damage, these risks are diminished once smoking is stopped (Briganti et al. 2002; Pinto-Sietsma et al. 2000). A number of studies have shown that blood pressure lowering is associated with substantial slowing of the rate of decline in kidney function (Kshirsagar et al. 2000). In people with diabetes, tight control of blood sugar levels can retard progression of kidney damage and reduce vascular risk (DCCT 1993).

Once CKD has been diagnosed, appropriate therapeutic intervention has been demonstrated in a high-risk population to reduce the rate of progressive deterioration in kidney function and death by 20–50% (Hoy et al. 2003). For patients with advanced CKD, early referral to a nephrologist for consultation and treatment, and receiving care from a multidisciplinary team, have been found to significantly improve the outcomes of kidney replacement therapy and increase patients' life span (Curtis et al. 2005; Mendelssohn 2005).

Although CKD has many characteristics that are different from other chronic diseases, its occurrence and development largely interact with the onset and progress of certain of these diseases, such as diabetes and cardiovascular disease. Effective strategies for the prevention and management of CKD not only need to address kidney problems, but also need to tackle the problems of other related diseases and shared risk factors.

Although the burden posed by CKD is substantial, there is no national monitoring system for this disease. In the hope of stimulating discussion on this important topic, a brief outline of a possible national monitoring framework for CKD, including examples of potential indicators, is included in Appendix 3 of this report.

Purpose and structure of this report

This report compiles the latest information on CKD from a variety of data sources, with a focus on the modifiable and preventable risk factors associated with chronic kidney disease and the major causes of end-stage kidney disease in those receiving kidney replacement therapy. The aims of the report are:

1. to provide an overview of CKD and its impact on the Australian population and the health care system in Australia;
2. to describe the major risk factors for CKD and trends in recent years;
3. to examine trends in the incidence, prevalence and major causes of treated end-stage kidney disease; and
4. to review the current status of prevention and management of the disease.

The information provided in this report should serve as a vital resource for anyone with an interest in the area, especially those who are developing policies and providing services to help reduce the burden of CKD.

The report consists of six chapters.

- This introduction provides a definition of CKD, gives a brief overview of the major impacts of CKD, describes its major characteristics and outlines key issues in the CKD field.
- Chapter 2 documents detailed information on the burden of CKD. Aspects of its incidence and prevalence, impact on people's health and life, contribution to mortality, health service usage and health expenditure are described.
- Chapter 3 provides information on the risk factors for and causes of CKD.
- Chapter 4 examines the major causes of treated end-stage kidney disease. Information on trends in the incidence and prevalence of treated end-stage kidney disease due to these causes is given to highlight changes over recent years.
- Chapter 5 reviews the current situation with regard to the prevention and management of CKD in Australia. Information on strategies known to reduce exposure to risk factors and interventions to prevent further worsening of the disease is presented.
- Chapter 6 focuses on the burden of CKD among Aboriginal and Torres Strait Islander people. This population is disproportionately affected by CKD and particularly ESKD. Information on prevalence, incidence, health service use and mortality are presented and compared with the general population where possible.

The report also contains three appendix sections. An outline of methods, data sources and their limitations is included in Appendix 1, which also provides coding lists for CKD, using the International Classification of Diseases (ICD-10) and the International Classification of Primary Care (ICPC-2). Appendix 2 presents data relating to the Caring for Australians with Renal Impairment (CARI) guidelines on the adequacy of haemodialysis. Appendix 3 presents some information regarding the monitoring of CKD and related risk factors in Australia.

References

Briganti EM, Branley P, Chadban SJ et al. 2002. Smoking is associated with renal impairment and proteinuria in the normal population: the AusDiab kidney study. American Journal of Kidney Diseases 40:704–12.

Chadban SJ, Briganti EM, Kerr PG et al. 2003. Prevalence of kidney damage in Australian adults: the AusDiab kidney study. Journal of the American Society of Nephrology 14:S131–8.

Curtis BM, Ravani P, Malberti F et al. 2005. The short- and long-term impact of multi-disciplinary clinics in addition to standard nephrology care on patient outcomes. Nephrology Dialysis Transplantation 20:147–54.

DCCT (Diabetes Control and Complications Trial Research Group) 1993. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. New England Journal of Medicine 329:977–98.

Excell L & McDonald SP 2005. New patients commencing treatment in 2003. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide; Australia and New Zealand Dialysis and Transplant Registry, 7–14.

Go AS, Chertow GM, Fan D, McCulloch CE & Hsu C 2004. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. New England Journal of Medicine 351:13.

Hoy WE, Mathews JD, McCredie DA et al. 1998. The multidimensional nature of renal disease: rate and associations of albuminuria in an Australian Aboriginal community. Kidney International 54:1296–1304.

Hoy WE, Wang Z, Baker PR & Kelly AM 2003. Reduction in natural death and renal failure from a systematic screening and treatment program in an Australian Aboriginal community. Kidney International Suppl. 83:S66–73.

Johnson D 2004. Evidence-based guide to slowing the progression of early renal insufficiency. Internal Medicine Journal 34:50–7.

Mathew T & Johnson D 2005. Measurement of kidney function. Medical Observer, 29 July 2005, 29–31.

Mendelssohn DC 2005. Coping with the CKD epidemic: the promise of multidisciplinary team-based care. Nephrology, Dialysis, Transplantation 20:10–2.

NKF (National Kidney Foundation of America) 2002. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. American Journal of Kidney Disease 39 Suppl. 1:S1–266.

Obrador GT & Pereira BJG 2002. Systemic complications of chronic kidney disease. Pinpointing clinical manifestations and best management. Postgraduate Medicine 111(2):115–22.

Pinto-Sietsma SJ, Mulder J, Janssen WM, Hillege HL, de Zeeuw D & de Jong PE 2000. Smoking is related to albuminuria and abnormal renal function in nondiabetic persons. Annals of Internal Medicine 133:585–91.

2 The burden of chronic kidney disease

Highlights

- Up to 7.5% of Australians aged 25 years and over may have reduced kidney function.
- Although only a small proportion of people develop end-stage kidney disease, this accounts for the majority of the burden of CKD through use of hospital services, health expenditure and impacts on quality of life.
- In 2003, 13,625 Australians were receiving dialysis or living with a kidney transplant for end-stage kidney disease.
- In 2003–04, 11% of all hospital separations were for dialysis. The age-standardised hospital separation rate for same-day dialysis increased by 41% between 1998–99 and 2003–04.
- In 2003, CKD was recorded as the underlying cause of death in 2,431 cases. In a further 9,217 cases it was recorded as an associated cause of death.
- There is substantial evidence that CKD is an independent risk factor for cardiovascular disease, which is a common comorbidity and a major cause of death among people with CKD. In 2003, cardiovascular disease was recorded as the underlying cause of death in 44% of cases where CKD was an additional cause of death.

Introduction

Chronic kidney disease imposes a substantial burden on both communities and individuals. It mainly affects the older population, but can occur in people of any age. CKD usually develops without symptoms, and thus is often only detected at the very late stages. However, it begins to increase the risk of illness and death from other diseases and conditions from the very early stages.

CKD has profound impacts on wellbeing and quality of life, particularly at the final stage. Life expectancy is generally shortened. People with end-stage kidney disease require very expensive treatment and intensive health services.

This chapter provides an overview of the burden of CKD in Australia. The extent of the burden is illustrated through four components:

- the number of people who have CKD—described in terms of the prevalence of kidney damage or reduced kidney function, and the prevalence and incidence of treated ESKD;
- the impact of CKD on individuals—through description of its comorbidities and complications, effects on quality of life and disability, and mortality;
- the impact of CKD on health services—described in terms of use of GP and hospital services, and treatment for ESKD; and
- direct health expenditure on CKD.

Reduction in kidney function

How common is chronic kidney disease, what is the distribution of severity among those affected, and is CKD becoming more or less common over time? These questions are of interest to those developing strategies, allocating resources and planning services for preventing and managing CKD. The answers could also be useful in alerting primary care clinicians to the scope and need for early detection and intervention among their patients.

Unfortunately, current data sources provide limited answers. For example, the 2001 National Health Survey did not collect the biomedical data needed to examine the prevalence of CKD according to the US Kidney Disease Quality Outcome Initiative (K/DOQI) definition (see Box 1.1). Less than 0.5% of survey respondents self-reported having kidney disease as a long-term condition. This probably reflects the lack of symptoms in less severe CKD, so that many Australians would not be aware of any problems or have come to the notice of a doctor.

By contrast, the Australian Diabetes, Obesity and Lifestyle (AusDiab) study used biomedical measurements to explore the prevalence of CKD in its 1999–00 national survey of non-institutionalised Australians aged 25 years and over. Participants were tested for protein or blood in the urine (proteinuria and haematuria, respectively) as a sign of kidney damage and their GFR was also estimated as a measure of kidney function. The AusDiab Kidney Study investigators found that 11.2% of participants had a GFR of less than 60 mL/min/1.73m^2. A further 5.1% had protein or blood in the urine without significantly reduced kidney function (Chadban et al. 2003). If these conditions could be shown to have persisted for 3 months or longer, 16.3% of respondents would have met the K/DOQI criteria for CKD (Box 1.1).

However, important questions remain about these findings.

- The evidence of kidney problems in the study was based on a single measurement, or two measurements separated by a short interval, and so the K/DOQI criterion of 'three months or more' for CKD was not satisfied. Although it is reasonable to assume that many of the cases would show persistence, the proportion is not known.
- More importantly, GFR declines progressively with age (NKF 2002), so there is a question about the significance of the findings among older people. The K/DOQI criteria, although acknowledging that older people with reduced GFR may have normal kidney function for their age, make no allowance for age in defining CKD. As a result, if these criteria were applied literally to the AusDiab sample (assuming the kidney problems persisted for 3 months or more in all cases), over half of those aged 65 years and over would be described as having CKD (Table 2.1).

  But how many of these 'cases' of CKD in fact do not have disease but a normal age-related reduction in function that occurs slowly over time—a rate that is not accelerated consistent with CKD and that will not threaten their quality of life or life expectancy? The health implications of 'normal' age-related decline in kidney function are unclear (NKF 2002) and the issue remains a subject of intensive research.
- Two formulas are commonly used to calculate GFR: the MDRD (Modification of Diet in Renal Disease) formula and the Cockcroft-Gault formula. A US study has suggested that the MDRD formula provides a better estimate of GFR than the Cockcroft-Gault formula used to produce the prevalence statistics cited above, particularly in women and older people (Levey et al. 1999). The US National Kidney Foundation, the UK Renal Association and the Australasian Creatinine Consensus Working Group now recommend the use of the MDRD formula (NKF 2002; Joint Specialty Committee on

Renal Disease 2005; The Australasian Creatinine Consensus Working Group 2005), and the prevalence of CKD in the Australian population is currently being recalculated using this formula. Preliminary results suggest that the prevalence of significant reduction in kidney function (GFR less than 60 mL/min/1.73m^2) may be reduced from 11.2% to 7.5% (The Australasian Creatinine Consensus Working Group 2005). The final results of this re-investigation are not available at the time of writing.

Table 2.1: Prevalence of moderate or severe reduction in kidney function, by age and sex, 1999–00

Estimated kidney function		45–64 years	65 years and over	25 years and over
			(per cent)	
MDRD formula				
GFR <60 mL/min/1.73 m^2	**Persons**	**n.a.**	**n.a.**	**7.5**
Cockcroft-Gault formula				
GFR <60 mL/min/1.73 m^2	Males	1.8	51.8	9.3
	Females	3.2	57.2	13.0
	Persons	**2.5**	**54.8**	**11.2**
GFR 30–59 mL/min/1.73 m^2	Males	1.8	50.3	9.1
	Females	3.2	55.3	12.6
GFR <30 mL/min/1.73 m^2	Males	—	1.5	0.3
	Females	0	1.9	0.4

— Rounded to zero.

n.a. Not available.

Note: The prevalence of moderate or severe reduction in kidney function (as estimated using the Cockcroft-Gault formula) among people aged 25–44 years was 0.01%. Due to the small numbers, detailed data for this age group are not included in this table.

Sources: The Australasian Creatinine Consensus Working Group 2005; Chadban et al. 2003.

Despite these limitations, the AusDiab findings provide an indication of the number of people who might have CKD, and who are at increased long-term risk of developing end-stage kidney disease (ESKD). This is when a person's kidney function is no longer sufficient to sustain life, and dialysis or a kidney transplant is required. Although the ESKD population only represents a very small proportion of all people with CKD, a significant amount of the burden of CKD relates to the communities and individuals affected by ESKD. It is therefore very important to monitor the incidence and prevalence of ESKD so that adequate resources can be assigned to treat people with this illness.

Prevalence and incidence of treated end-stage kidney disease

People who are receiving dialysis or living with a kidney transplant are said to have 'treated ESKD'. Information on the incidence and prevalence of treated ESKD has been collected by the Australian and New Zealand Dialysis and Transplant (ANZDATA) Registry for the last 40 years. Data for the period 1981–2003 are presented below to illustrate recent trends in treated ESKD in Australia.

It should be noted that the incidence and prevalence of treated end-stage kidney disease will underestimate the incidence and prevalence of ESKD among the whole community, as not all people will accept or be suitable candidates for kidney replacement therapy. This is especially true for people in the older age groups. Information about the incidence and prevalence of ESKD among the general population is not currently available in Australia.

Prevalence of treated end-stage kidney disease

At the end of 2003, a total of 13,625 people (an age-standardised rate of 675 per million population) were being treated for ESKD in Australia. The prevalence rate increased rapidly with age to the 65–74 years age group, then declined. The prevalence of treated ESKD was higher in males (810 per million population) than in females (551 per million population) across all age groups.

Trends in prevalence

The number of people being treated for end-stage kidney disease has more than tripled over the last 20 years, from 3,181 patients in 1981 to 13,625 patients in 2003. Over this period, the prevalence rate of treated ESKD increased by 5.6% each year on average. However, the annual rate of change has gradually decreased from 7% (1981 to 1982) to 3% (2002 to 2003) (Figure 2.1). Although the rate of increase in the prevalence of ESKD has been slowing down in recent years, it is highly likely that the prevalence of ESKD will continue to increase in the near future. While a higher incidence of treated ESKD is a major factor in this increase, ageing population, improved management and new technologies have also contributed to these numbers by keeping people alive for longer.

Incidence of treated end-stage kidney disease

According to ANZDATA, 1,953 people (97 per million population) began kidney replacement therapy in 2003 (Excell & McDonald 2005a). The incidence rate in males (1,150 cases, 118 per million population) was higher than in females (803 cases, 77 per million population), and this pattern occurred across all age groups. The incidence rate increased progressively with age, peaking in the 65–74 years age group and then declining.

The average age at commencement of treatment in 2003 was 59.3 years, well above the average of 42.3 years 20 years earlier. This is predominantly due to increased numbers of new patients in the older age groups, rather than a decline in the number of young patients (see Figure 2.2).

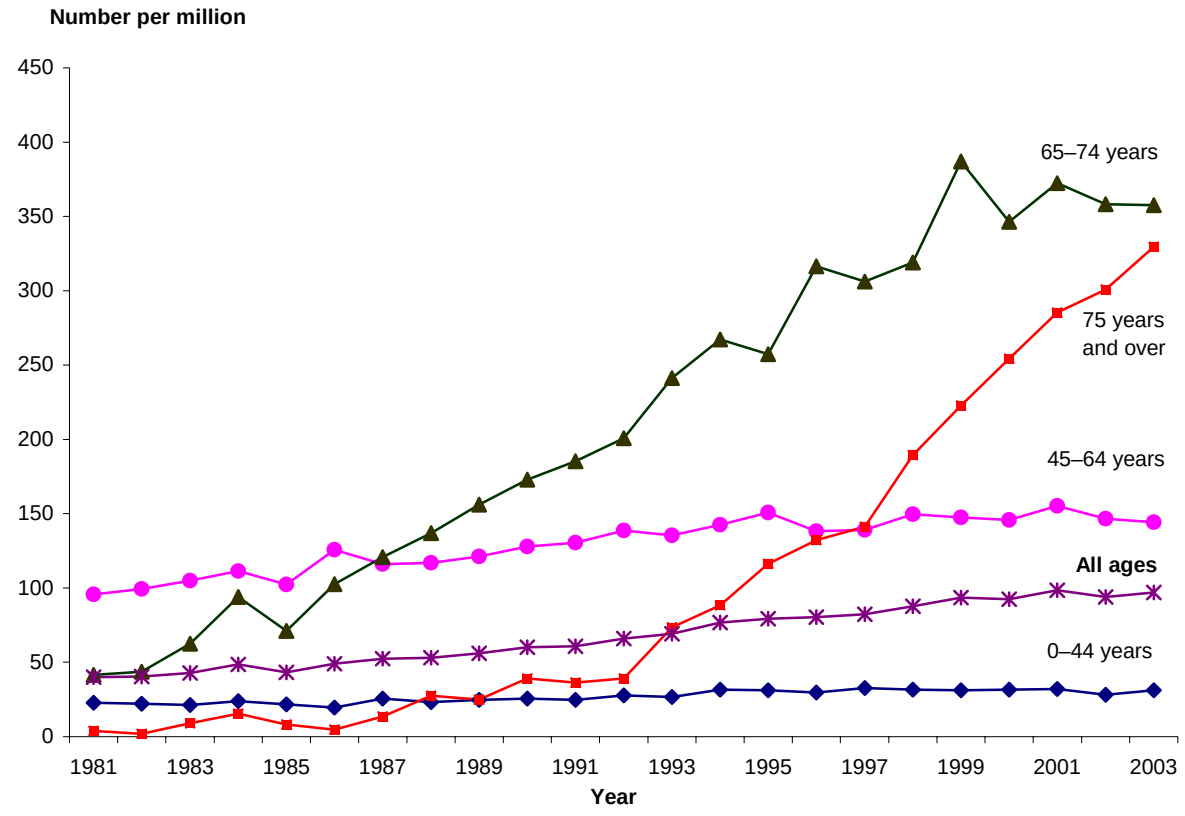
The higher incidence among males may be the result of gender differences in the causes of kidney damage and CKD. The most common causes of treated ESKD in Australia, such as glomerulonephritis, diabetes and high blood pressure, and risk factors such as smoking, are all more common among men than women.

Trends in incidence

Since the early 1980s, the age-standardised incidence rate of treated ESKD has more than doubled, from 40 per million population in 1981 to 97 per million population in 2003 (Figure 2.1). The changes in incidence rate vary between age groups. Between 1981 and 2003, the incidence of treated ESKD was stable in people below the age of 45 years, but increased among the middle and older age groups. For people aged 45–64 years the incidence increased by nearly 50%, from 96 to 144 per million population, while the incidence in those aged 65–74 years increased nearly ninefold (from 42 to 358 per million population). For the oldest age group (75 years and over), the incidence was low until 1992 but rapidly increased over the following decade to reach 330 per million population in 2003.

The reasons for the increasing incidence of treated ESKD among the middle-aged and older population are complex. The increasing prevalence of diabetes, high prevalence of high blood pressure in the past, and reduced cardiovascular mortality may all have contributed to

an increased incidence of ESKD in the community. At the same time, changes in acceptance policies for patients in the older age groups mean that more people in these age groups are being accepted into the kidney replacement therapy program. It is difficult to separate out the contribution each of these factors has made to the increasing number of people beginning kidney replacement therapy. These issues are discussed in more detail in Chapter 4 of this report.



Note: The 'all ages' rate has been age standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 2.1: Incidence of treated end-stage kidney disease, by age group, 1981 to 2003.

Comorbidities and complications of chronic kidney disease

Chronic kidney disease can affect all the organs and systems in the body. The disturbances to the body's chemical balance and build-up of waste substances in the blood can have extensive functional consequences, leading to the development of complications. Most of these conditions develop early in the course of CKD and contribute substantially to its high morbidity and mortality. The exact path of development of most complications is unknown. CKD also shares a number of risk factors with other chronic diseases. For example, high blood pressure and diabetes increase the risk of cardiovascular disease, and smoking is linked to both cardiovascular and respiratory problems. For these reasons, CKD often coexists with one or more other diseases, adding to the burden of the disease on individuals and complicating their treatment.

Common problems among people with CKD in Australia include cardiovascular diseases (mainly high blood pressure, ischaemic heart disease and heart failure), respiratory diseases, infections, malnutrition, anaemia and cancers.

Cardiovascular disease

CKD is an independent and major risk factor for cardiovascular disease. High blood pressure (also called hypertension) almost invariably develops in people with CKD. In addition, reduced estimated kidney function has been found to independently increase the risk of cardiovascular disease events, including heart attack, angina, coronary artery disease, stroke and heart failure (Go et al. 2004). The risks of an event are greater in those with poorer kidney function. People with stage 5 CKD (GFR less than 15 mL/min/1.73m^2) are more than three times as likely to have a cardiovascular event as people without CKD (Go et al. 2004). In Australia, among hospitalisations that have CKD as an additional diagnosis, cardiovascular disease is the most frequent reason for overnight hospitalisation (Table 2.5). Similarly, in people who have CKD recorded as an associated cause of death, cardiovascular disease is the most common underlying cause of death (Table 2.7).

Cardiovascular disease is also the most common comorbidity and cause of death among people with treated end-stage kidney disease. Among new treated end-stage kidney disease patients in 2003, 39% had coronary artery disease, 25% had peripheral vascular disease and 14% had cerebrovascular disease when they began their kidney replacement therapy. Between 1991 and 2003, the proportion of new patients with coronary artery disease increased by 11%, peripheral vascular disease by 10% and cerebrovascular disease by 5%. In 2003, 40% of deaths among dialysis-dependent patients and 23% of deaths among kidney transplant patients in Australia were from cardiovascular disease (Excell & McDonald 2005b).

Although a history of high blood pressure and other shared risk factors may partly explain the association between CKD and cardiovascular disease, the increased risk is mostly attributed to the effects of reduced kidney function. This is associated with increased risk of inflammation and blood clotting, left ventricular hypertrophy (enlargement of the left chamber of the heart, which can lead to heart failure) and hardening of the arteries (Go et al. 2004).

Cancer

It is not known if there is an increased risk of cancer among CKD patients in the early stages of the disease, but it has been found that there is a higher risk of cancer among people who are receiving kidney replacement therapy. According to the ANZDATA Registry, the incidence rate for all types of cancer combined (excluding non-melanocytic skin cancer) was 1.7 times higher among dialysis patients and 3.1 times higher among kidney transplant patients than among the general population in 2003 (Chapman & Webster 2005). For dialysis patients the increased risk was seen for cancers of the respiratory system, bones and cartilage, urinary system, endocrine glands and central nervous system. For transplant patients, the risk was increased for most types of cancers.

The higher risk of cancer among people receiving treatment for end-stage kidney disease may be associated with the presence of chronic infections, a weakened immune system, treatment with immunosuppressive or cytotoxic drugs, nutritional deficiencies, and altered DNA repair (Maisonneuve et al. 1999).

Infections

Because reduced kidney function can impair the immune system, people with CKD have increased susceptibility to infections (Braunwald et al. 2001). The most common types of infections among people with CKD are respiratory infections (described below).

Dialysis-dependent patients are also at high risk of other types of infections. People receiving haemodialysis (filtering of the blood outside of the body by a machine) can develop serious infections, most of which are related to the access site (where the body is connected to the dialysis machine). People on peritoneal dialysis (filtering of the blood within the body) are also at high risk of developing inflammation of the lining of the abdomen (peritonitis).

Respiratory diseases

CKD shares some risk factors, such as smoking, with respiratory diseases. Further, people with CKD are prone to infection, as described above. Therefore, CKD and respiratory problems may often coexist. Respiratory diseases and infections, especially pneumonia and chronic obstructive pulmonary disease, have been found to be a common cause of overnight hospitalisation and one of the major causes of death among people with CKD. The ANZDATA Registry also reported that about 16% of new treated end-stage kidney disease patients in 2003 were found to have chronic lung disease, an increase of 5% from 1991.

Malnutrition

People with CKD have a risk of protein deficiency, inadequate caloric intake and electrolyte imbalance if their diet is not properly controlled as the kidney disease progresses. Weight loss and oedema (fluid retention causing swelling) may be signs of these problems. Malnutrition has a significant influence on patients' survival and is a strong predictor of patients' mortality and quality of life. In people receiving dialysis, malnutrition has been found to be associated with impaired pulmonary function, which may affect patient outcomes (Nascimento et al. 2004). There is no information on the nutritional status of people with CKD in Australia.

Other complications

Other common diseases and conditions associated with CKD include anaemia (too few red blood cells, leading to insufficient oxygen going to the tissues and organs), bone and muscle problems, disorders of the nervous system (neuropathy) and disorders of endocrine function (such as diabetes and thyroid problems) (Braunwald et al. 2001). There are also increased risks of sleep disorders, anxiety and depression among people with CKD, especially those on dialysis treatment. Information about the incidence and prevalence of these diseases among people with CKD in Australia is not currently available.

Disability and quality of life

CKD has a significant impact on patients' and their carers' lives, especially for those people whose life relies on dialysis treatment. People with CKD may have to take a number of different medications to control risk factors or other health problems, or to maintain the correct amounts of various vitamins, minerals and chemicals which are affected by their kidney problems. The complications of CKD may also impact on quality of life, and can cause disability. For example, heart disease and stroke are common causes of physical activity restrictions in Australians (AIHW 2004c). Problems such as disturbed sleep or insomnia can impact on the patient's physical and mental wellbeing as well as on that of their family.

Results from the Australian Diabetes, Obesity and Lifestyle study in 1999–00 showed that people with CKD reported significantly poorer physical functioning, general health and vitality than the general population, and were more likely to report difficulties performing their usual activities due to physical or emotional problems (Chow et al. 2003). Mental health in particular was a problem in younger people, while physical functioning was a greater issue for older people.

The Australian Bureau of Statistics' Disability, Ageing and Carers survey collects information on the presence and types of disability, and main disabling condition. In 2003, it was estimated that almost 8,000 Australians had a disability due to a kidney or urinary system disorder (excluding incontinence). It is not possible to determine how many of these people had chronic kidney disease.

Although CKD is usually symptomless until the later stages, people with CKD may experience a range of problems including fatigue, muscle cramps, nausea, itchy skin, urinary tract infections, headaches, and loss of appetite when nearing the end stage. These symptoms can affect the patient's mental state as well as their physical health. As kidney function worsens, physical and mental health may be increasingly affected. For people whose disease reaches end-stage, the diagnosis of ESKD is a significant event and may at first be met with denial. As with any serious health problem, people diagnosed with ESKD can feel angry, anxious, hopeless or depressed (Lew & Piraino 2005). They may 'grieve' for their lost health (Australian Kidney Foundation 2002). Depression is common among people being treated for ESKD, particularly those just beginning dialysis, and this can severely affect their quality of life and outcomes (Tossani et al. 2005). It may also affect their willingness to comply with their treatment, which can have serious consequences (Jindal et al. 2003). Family and friends, as well as dealing with their own feelings about the diagnosis, might also be affected by the patient's reactions, and relationships and friendships can become strained.

Impacts of dialysis

Dialysis is an artificial way of removing waste substances from the blood, a function usually performed by the kidneys. There are two main forms of dialysis: peritoneal dialysis, which occurs inside the body; and haemodialysis, which occurs outside the body. Which form is used depends on the patient's health, age and lifestyle. The choice may also be influenced by the availability of local resources. Starting dialysis means starting a new way of life that is beneficial yet also problematic. People's lives are prolonged by dialysis treatment, but may also be greatly altered by it. The different forms of dialysis have different effects on people's lives and quality of life.

In peritoneal dialysis, the dialysis solution is pumped into the abdomen and the blood is filtered through the peritoneal membrane. This is the lining of the abdominal cavity, which covers the organs such as the stomach, liver and intestines. The dialysis solution contains a type of sugar (usually glucose or dextrose) which draws the waste products and extra fluid out of the blood, through the peritoneal membrane and into the solution. After a few hours, the used solution, now containing the wastes and extra fluid, is drained out of the body and replaced with fresh solution. This process is called an exchange, and takes about 30 minutes. In between exchanges, the patient is free to continue their usual activities. Peritoneal dialysis can be performed either by the patient during the day, or automatically by a machine at night while the patient sleeps. This type of dialysis needs to be done every day, usually three or four times. As the necessary equipment is portable, peritoneal dialysis can be performed almost anywhere. The patient does not need to be in a hospital or clinic, and can usually manage the procedure without assistance.

In contrast, the lives of haemodialysis patients are to some extent 'fixed' by the need for the dialysis machine. Blood is diverted from the body to the machine, where it is filtered before being returned to the body. The patient is connected to the machine for around 4 hours per dialysis session, and during this time all their blood passes through the machine approximately six times. Haemodialysis is usually performed three times each week, and the patient cannot get up and move away from the machine during a session, though they can perform activities which do not require much movement such as sleeping, reading, talking, or using a computer. This type of dialysis can be done at home or in hospitals or clinics, however, the dialysis machine requires special plumbing and therefore the patient must limit their travel to places where dialysis facilities are available. In most cases the patient will need assistance connecting to the machine, and a partner, relative or friend can be trained to do this for home dialysis patients. The method and location of initial dialysis is selected based on the preference of a fully–informed patient.

People with ESKD have to constantly monitor and control their diet and fluids. ESKD reduces the appetite and so patients need to make sure to eat properly even when they don't feel like it. Complications of ESKD and the dialysis process may also bring further interruption to their lives (Polaschek 2003).

Dialysis treatment also has a profound effect on patients' families, friends and colleagues. Family members have to limit their own social life to provide support for the dialysis patient. The amount of care required also increases over time as the patient's health deteriorates. This may lead to resentment and distancing in the relationship (Polaschek 2003). However, various studies have shown that physical functioning, mental health and quality of life can be improved through appropriate intervention. Education, counselling, treatment with antidepressant medication, strong social and familial support and regular physical activity have all been found to have beneficial effects for dialysis patients (Koo et al. 2005; Tossani et al. 2005; Kouidi 2004; Levendoglu et al. 2004; Tsay & Hung 2004).

Health service usage

The treatment and care of people with CKD covers a variety of settings and types of care. Due to lack of information, many services consumed by people with CKD are impossible to identify. In this section, the focus is on the health services provided by general practitioners (GPs) and hospitals. Although it is acknowledged that specialists and allied health professionals would provide a substantial amount of services to people with CKD, particularly people receiving kidney replacement therapy, these services cannot be identified from available data sources. No data on these services are included in this section.

Visits to general practitioners

GPs are the source of first-contact care and they have a natural role in managing and monitoring CKD. Information about usage of GP services by people with CKD was drawn from the Bettering the Evaluation and Care of Health (BEACH) survey of general practice. This survey involves random samples of approximately 1,000 GPs per year, each of whom records the details of 100 consecutive patients' encounters (AIHW: Britt et al. 2005).

In 2003–04, CKD problems were managed at a rate of 3 per 1,000 GP encounters. This suggests that CKD was managed in approximately 290,000 Medicare-paid GP consultations in 2003–04. GP management of CKD problems becomes more common with age.

The most frequently managed CKD problem in 2003–04 was 'chronic kidney failure' (44% of CKD problems managed), followed by 'unspecified kidney failure' (19%) and glomerulonephritis/nephrosis (10%). Other problems such as hypertensive kidney disease and diabetic nephropathy were managed less frequently.

GPs manage these problems in a variety of ways: treating the problem without medication; prescribing/recommending medication; ordering imaging or pathology tests; and coordinating referrals. Management strategies reported by GPs in 2003–04 for CKD problems are shown in Table 2.2. Pathology tests were ordered for most problems, with electrolytes/urea/creatinine and a full blood count being the most common tests ordered. Medications were prescribed in around two-fifths of all problems managed. In 12% of cases the patient was referred to another health professional or health service provider.

Table 2.2: Management of chronic kidney disease by GPs, 2003–04

Type of management	% of CKD problems managed (n = 301)
Prescribed medications	41%
Ordered pathology tests	
Electrolytes/urea/creatinine (EUC)	17%
Full blood count	13%
Blood creatinine or albumin: creatinine ratio	3%
Urinary albumin	2%
Liver function	8%
Lipid profile or cholesterol/triglycerides	1%
Other	36%
Ordered imaging	
Ultrasound of kidney or renal tract	4%
Other	3%
Referral	
Specialist	2%
Urologist	6%
Hospital or clinic	1%
Other	2%

Note: Figures do not add to 100% as more than one form of management may be used for each problem.

Source: AIHW analysis of the BEACH GP survey.

Hospitalisation

People with CKD require a large amount of hospital services, particularly those people with end-stage kidney disease. In Australia, information about hospital services is available from the AIHW National Hospital Morbidity Database. In this database, the principal and any additional diagnoses are recorded for each hospital separation. The principal diagnosis is defined as 'the diagnosis established after study to be chiefly responsible for occasioning an episode of admitted patient care '(AIHW 2005a:333). An additional diagnosis is defined as 'a condition or complaint either coexisting with the principal diagnosis or arising during the episode of care' (AIHW 2005a:326).

The most frequent reason for using hospital services among people with CKD is day admission for dialysis. However, people with CKD also can be hospitalised for other reasons. Many of these are attributed to diseases associated with CKD, such as cardiovascular disease, and side effects of kidney replacement therapy. These cases are more likely to have CKD recorded as an additional diagnosis rather than as the principal diagnosis.

Chronic kidney disease as the principal diagnosis

In 2003–04 there were 784,925 hospital separations with a principal diagnosis of CKD (11.5% of all hospital separations). Among these, the major reason for hospitalisation was admission

for regular dialysis. There were 759,272 hospitalisations for dialysis in 2003–04 (96.7% of hospital separations with a principal diagnosis of CKD; 11% of all hospital separations). This figure represents multiple separations for a much smaller number of patients as each would have received dialysis about three times each week. It is not possible to determine the cause of end-stage kidney disease in people receiving dialysis from the hospital separations data.

Besides receiving dialysis, people with CKD also frequently use hospital services for the treatment of casual diseases, uraemia and complications of kidney replacement therapy. Common principal diagnoses in 2003–04 included kidney tubulo-interstitial diseases (6,312 separations), chronic kidney failure (4,983 separations) and diabetic nephropathy (4,075 separations) (Table 2.3).

Table 2.3: Hospital separations for chronic kidney disease, by principal diagnosis, 2003–04

Principal diagnosis	ICD-10-AM codes	Number of separations	Per cent of subtotal	Per cent of total
Diabetic nephropathy	E10.2, E11.2, E13.2, E14.2	4,075	15.9	0.5
Hypertensive kidney disease	I12, I13, I15.0, I15.1	639	2.5	0.1
Glomerular diseases	N00–N08	2,502	9.8	0.3
Kidney tubulo-interstitial diseases	N11, N12, N14–N16	6,312	24.6	0.8
Chronic kidney failure	N18	4,983	19.4	0.6
Unspecified kidney failure	N19, Z49.0	3,601	14.0	0.5
Other disorders of kidney and ureter	N25, N26, N27, N28, N39.1, N39.2	1,609	6.3	0.2
Congenital malformation of the kidney and ureter	Q60–Q63	1,132	4.4	0.1
Complications related to dialysis and kidney transplant	T82.4, T86.1	800	3.1	0.1
Subtotal		*25,653*	*100.0*	*3.3*
Dialysis	Z49.1, Z49.2	759,272		96.7
Total		**784,925**		**100.0**

Source: AIHW National Hospital Morbidity Database.

Bed days and length of stay

A total of 879,517 hospital bed days were occupied by people with a principal diagnosis of CKD in 2003–04, representing around 4% of the total bed days in that year. Of these bed days, 86% (758,611 days) were same-day separations for dialysis. After removal of same-day dialysis separations, the average length of stay in hospital for people with CKD in 2003–04 was 4.6 days, 35% longer than the average length of stay for all separations (3.4 days).

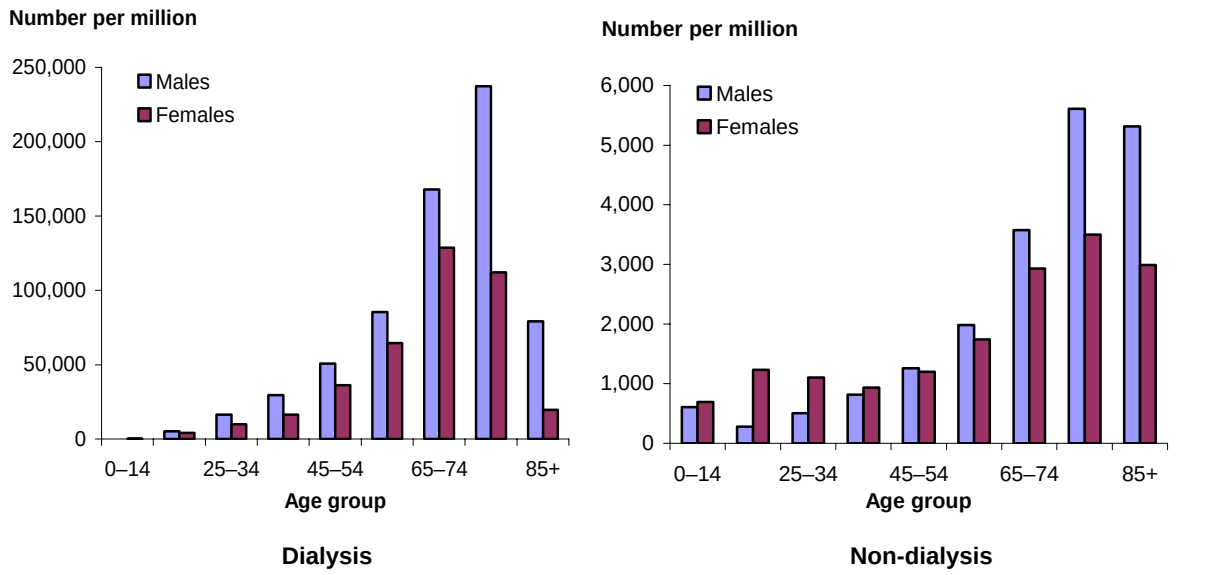
Sex and age

In 2003–04, males were more likely to be hospitalised with a principal diagnosis of CKD than females (457,655 and 327,269 separations, respectively). This pattern occurred across all age groups with the exception of those aged 5–19 years. Hospitalisation rates generally increased with age and peaked in the 75–79 years age group. Sixty-eight per cent of all CKD hospitalisations occurred in people aged 55 years and over.

The age and sex distribution of dialysis separations is quite different from that of separations for other CKD principal diagnoses (Figure 2.2). Separations for dialysis are more common in

males across all age groups, and the separation rate rises steeply with age. This reflects the prevalence of treated ESKD, and the higher proportions of transplants and peritoneal dialysis in the younger age groups (leading to less need for hospital-based dialysis).

In contrast, non-dialysis separations for CKD are more common among females than males in those aged less than 45 years, and the age-related increase in separation rates is much less steep. This is due to a much higher separation rate for tubulo-interstitial kidney diseases among females in the younger age groups.



Source: AIHW National Hospital Morbidity Database.

Figure 2.2: Hospital separations with a principal diagnosis of chronic kidney disease, 2003–04

Trends

Between 1998–99 and 2003–04, the age-standardised hospital separation rate for separations with a principal diagnosis of CKD increased by 39%, and the age-standardised number of bed days associated with these separations rose by 30% (Table 2.4). The average number of bed days per separation decreased by 6%. These changes can be mostly attributed to a substantial increase in the number of same-day separations for dialysis, from 478,658 in 1998–99 to 758,611 in 2003–04. This is a 41% increase in the same-day dialysis separation rate, from 26.3 to 37.1 per million population. When same-day separations for dialysis are excluded, the age-standardised hospital separation rate for CKD as principal diagnosis shows a 2% increase, with the age-standardised number of bed days and average bed days both showing decreases.

Table 2.4: Hospital separations with a principal diagnosis of chronic kidney disease, 1998–99 and 2003–04

	Separations with principal diagnosis of CKD			Excluding same-day dialysis		
	Separations	Bed days	Average bed days	Separations	Bed days	Average bed days
	(per 1,000 population)		(per separation)	(per 1,000 population)		(per separation)
1998–99	27.5	33.1	1.2	1.3	6.8	5.2
2003–04	38.4	43.0	1.1	1.3	5.9	4.6
% change	*39.3*	*30.0*	*–6.1*	*2.3*	*–12.8*	*–11.6*

Note: Figures for separations and bed days have been age standardised to the Australian population at 30 June 2001.

Source: AIHW National Hospital Morbidity Database.

Chronic kidney disease as an additional diagnosis

CKD is also commonly listed as an additional diagnosis. In 2003–04, CKD was listed as an additional diagnosis in 122,335 hospital separations. About 23% (28,139) of these separations were attributed to cardiovascular disease (Table 2.5). In comparison, only 6.6% of all hospital separations in 2003–04 had a principal diagnosis of cardiovascular disease. This highlights the strong association between cardiovascular disease and CKD. Other commonly recorded principal diagnoses in separations where CKD was an additional diagnosis were respiratory diseases (10,366 separations), other diseases of the genitourinary system (9,190 separations) and diabetes (6,311 separations). Each of these was also more likely to be recorded when CKD was an additional diagnosis than in general, again highlighting the complex interaction between CKD and these diseases.

Table 2.5: Hospital separations with an additional diagnosis of chronic kidney disease, by principal diagnosis, 2003–04

	Separations with CKD as an additional diagnosis		All separations	
Principal diagnosis (ICD-10-AM code)	**Number**	**Per cent**	**Number**	**Per cent**
Diseases of the circulatory system (I00–I99)[(a)]	28,139	23.0	448,220	6.6
Ischaemic heart disease (I20–I25)	9,379	7.7	164,226	2.4
Diseases of the respiratory system (J00–J99)	10,366	8.5	331,956	4.9
Pneumonia (J12–J18)	4,294	3.5	65,516	1.0
Endocrine, nutritional and metabolic diseases (E00–E89)[(b)]	9,838	8.0	107,076	1.6
Diabetes (E10–E14)[(b)]	6,311	5.2	56,207	0.8
Other diseases of the genitourinary system (N30–N99)	9,190	7.5	335,743	4.9
Diseases of the digestive system (K00–K93)	8,731	7.1	783,445	11.5
Symptoms, signs involving the circulatory and respiratory systems (R00–R09)	2,180	1.8	118,122	1.7
Complications of surgical and medical care, not elsewhere classified (T80–T88)	6,238	5.1	71,749	1.0
Neoplasms (C00–D48)	6,675	5.5	497,117	7.3
Care involving use of rehabilitation procedures (Z50.9)	5,899	4.8	130,209	1.9
Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism (D50–D89)	4,051	3.3	80,171	1.2
Infectious and parasitic diseases (A00–B99)	3,990	3.3	92,892	1.4
Diseases of the musculoskeletal system and connective tissue (M00–M99)	4,425	3.6	366,926	5.4
Other diseases and conditions	22,613	18.5	2,691,661	39.4
Total	**121,223**	**100**	**6,837,128**	**100**

(a) Excludes hypertensive kidney disease.

(b) Excludes diabetic nephropathy.

Source: AIHW National Hospital Morbidity Database.

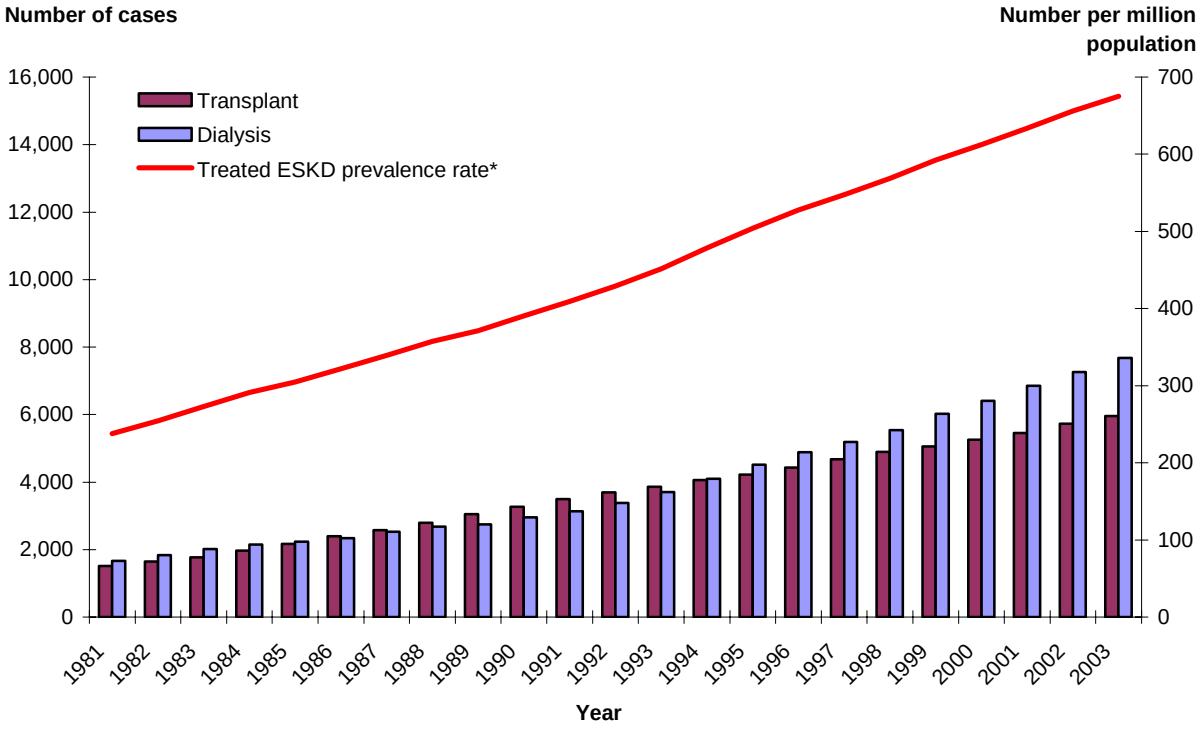
Treatment for end-stage kidney disease

People with end-stage kidney disease require kidney replacement therapy (dialysis or a kidney transplant) to sustain their life. As this treatment is very expensive and requires intensive health services, it poses a significant burden both financially and in terms of health service resources. If recent increases in the number of people receiving treatment for ESKD continue, this burden will also increase.

Using information from the ANZDATA Registry, the following section provides a profile of the kidney replacement therapy in Australia and details changes over the past 5 years.

Prevalence of kidney replacement therapy

At the end of 2003, a total of 13,625 people (675 per million population) were being treated for ESKD in Australia. This included 7,674 persons on dialysis and 5,951 persons with a functioning kidney transplant. Before 1994, about half of those receiving kidney replacement therapy were undergoing dialysis and the rest were living with a kidney transplant. Since then, the proportion of those on dialysis has increased rapidly (Figure 2.3). By 2003, 56% of patients registered with ANZDATA were undergoing dialysis and 44% had transplants. The higher average age of patients on kidney replacement therapy is one factor contributing to this shift, as older patients are less likely to opt for or be accepted for transplantation. Another factor is the low growth in the availability of donor organs (AIHW 2004a).



* Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

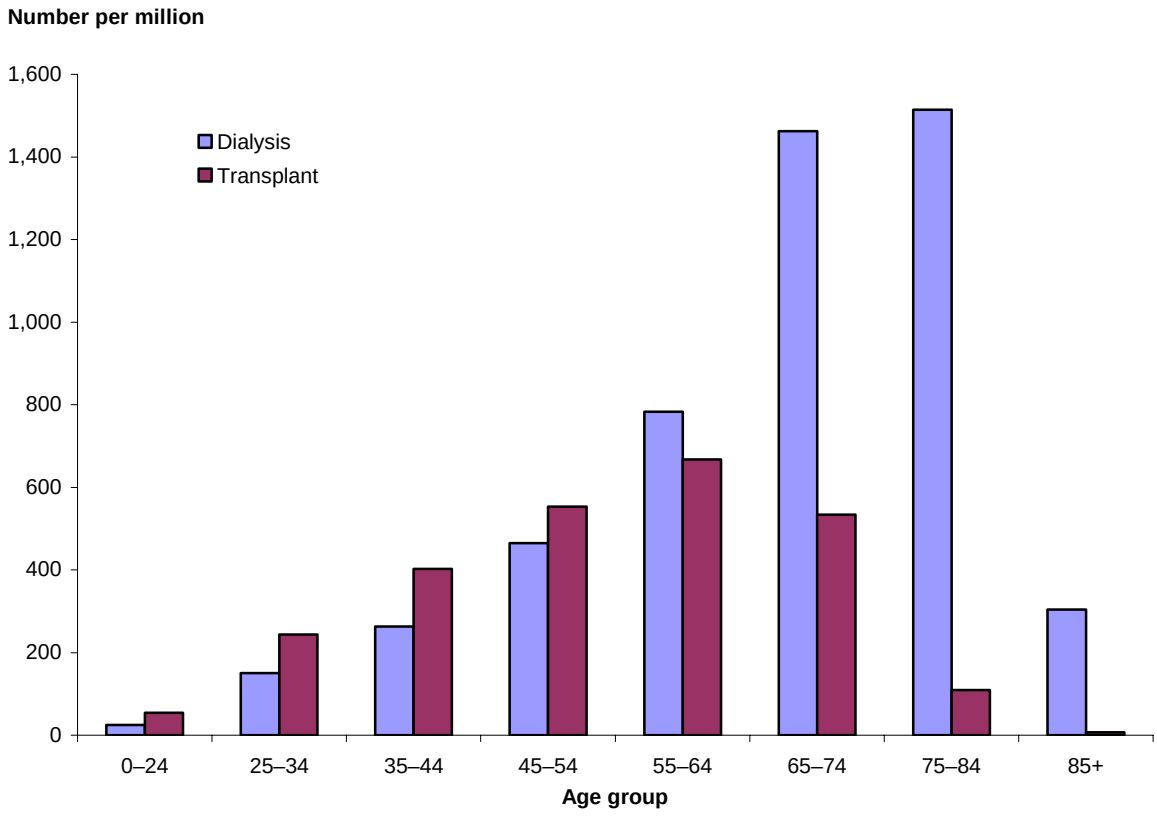
Figure 2.3: Number of treated end-stage kidney disease patients and age-standardised prevalence rate, 1981 to 2003

Dialysis

There were 7,674 people (386 per million population) receiving dialysis treatment at the end of 2003. The prevalence rate increased progressively with age, the highest rate being among people aged 75–84 years (1,514 per million population) (Figure 2.4).

The prevalence of dialysis in 2003 was an increase of 5% (409 more patients) from 2002, and 27% (1,653 more patients) from 1999. This increase occurred across all age groups among people aged 25 years and over. The annual rate of increase was higher among those aged 65–84 years (8%) and 85 years and over (14%).

The prevalence of dialysis varied between the states and territories. The highest prevalence rate in 2003 was 1,225 per million population in the Northern Territory, followed by Victoria (403 per million population), New South Wales (390 per million population), Western Australia (387 per million population), the Australian Capital Territory (358 per million population), South Australia (331 per million population) and Tasmania (312 per million population).



Source: AIHW analysis of ANZDATA Registry data.

Figure 2.4: Prevalence of functioning kidney transplants and dialysis, by age group, 2003

The demand for dialysis has been increasing consistently in Australia over the last 5 years. The growth rate of the dialysis pool is dependent on the entry of new patients and exits through death or transplantation. The incidence rate of end-stage kidney disease has increased steadily over the last 40 years. At the same time, dialysis-dependent patients are surviving longer as the technology and management improves. It also appears that the transplantation rate is unlikely to increase quickly to ease the pressure on demands for dialysis. All these factors suggest that the demand for dialysis treatment will continue to grow (Mathew 2005).

Methods and locations of dialysis

In 2003, about 76% (5,851) of dialysis-dependent patients were on haemodialysis, including 39% in satellite centres, 27% in hospital and 10% at home. About 24% (1,823) were on peritoneal dialysis, including 14% receiving continuous ambulatory peritoneal dialysis (CAPD) at home, 9% automated peritoneal dialysis (APD) at home and 0.4% CAPD or APD in hospital. Altogether, about 28% of dialysis patients received treatment in hospital, 33% at home and 39% in satellite centres.

Over the last 5 years, the number of patients increased for most methods of dialysis in all locations, except continuous ambulatory peritoneal dialysis (Table 2.6). However, the proportion of patients receiving peritoneal dialysis has been gradually decreasing, from 28% in 1999 to 24% in 2003. Although the proportion of patients receiving home haemodialysis is decreasing, the total proportion of patients receiving haemodialysis has been increasing, from 72% in 1999 to 76% in 2003. This increase was mainly due to increased haemodialysis in satellite centres (Excell & McDonald 2005c).

Table 2.6: Method and location of dialysis, 1999–2003

	1999	2000	2001	2002	2003
			(number)		
APD	264	390	501	612	726
CAPD	1,414	1,346	1,306	1,173	1,097
Total PD	*1,678*	*1,736*	*1,807*	*1,785*	*1,823*
Hospital HD	1,636	1,721	1,808	2,001	2,091
Home HD	706	742	773	777	772
Satellite HD	2,001	2,211	2,462	2,702	2,988
Total HD	*4,343*	*4,674*	*5,043*	*5,480*	*5,851*
Total	**6,021**	**6,410**	**6,850**	**7,265**	**7,674**
			(per cent)		
APD	4	6	7	8	9
CAPD	23	21	19	16	14
Total PD	*28*	*27*	*26*	*25*	*24*
Hospital HD	27	27	26	28	27
Home HD	12	12	11	11	10
Satellite HD	33	34	36	37	39
Total HD	*72*	*73*	*74*	*75*	*76*

APD automated peritoneal dialysis

CAPD continuous ambulatory peritoneal dialysis

HD haemodialysis

PD peritoneal dialysis

Source: AIHW analysis of ANZDATA Registry data.

Transplant

Transplantation of the human kidney is the most effective treatment for ESKD. Compared with dialysis, transplant has a significantly lower risk of comorbidities and mortality (McDonald & Russ 2002).

Number of functioning kidney transplants

Since 1963, there have been 14,068 transplant operations performed in Australia on 12,028 patients. If the first kidney transplanted fails, secondary or more transplants are possible, if suitable donors can be found. Of all kidney transplant operations performed, 85% were for primary transplant, and 15% were for secondary or further transplants. Of the total transplanted kidneys, 5,951 (300 per million population) were functioning at the end of 2003. The prevalence of functioning kidney transplants increased with age to 55–64 years, then decreased with age (Figure 2.4).

Young patients are more likely than older patients to have a kidney transplant. At the end of 2003, 83% of treated end-stage kidney disease patients aged 5–14 years had functioning kidney transplants. The proportion of patients with transplants decreased with age, to just 2% among patients aged 85 years and over.

The prevalence of functioning kidney transplants varied among the states and territories, being highest in South Australia and the Northern Territory (425 per million population), followed by Queensland (302 per million population), Victoria and Tasmania (291 per million population), New South Wales and the Australian Capital Territory (282 per million population) and Western Australia (267 per million population) (Excell et al. 2005).

Kidney transplantations in 2003

There were 543 kidney transplant operations in Australia in 2003, an age-standardised rate of 27 per million population. The annual transplant rate has increased by 13% since 1995 (when there were 455 operations, 24 per million population). While the operations were performed on patients with a wide age range (from 2.3 years to 77.6 years), the majority of recipients (93%) were aged under 65 years, with a median age of 44.8 years.

The transplantation rate in 2003 varied between the states and territories, from 38 per million population in South Australia and the Northern Territory to 18 per million population in Western Australia (Excell et al. 2005).

International comparison of treated end-stage kidney disease

Information on treated ESKD worldwide is only available for a small number of countries. International comparisons in this report are based on data published by the United States Renal Data System (USRDS 2004). This system has collected data on incidence, prevalence, modality of dialysis and kidney transplant from 31 countries.

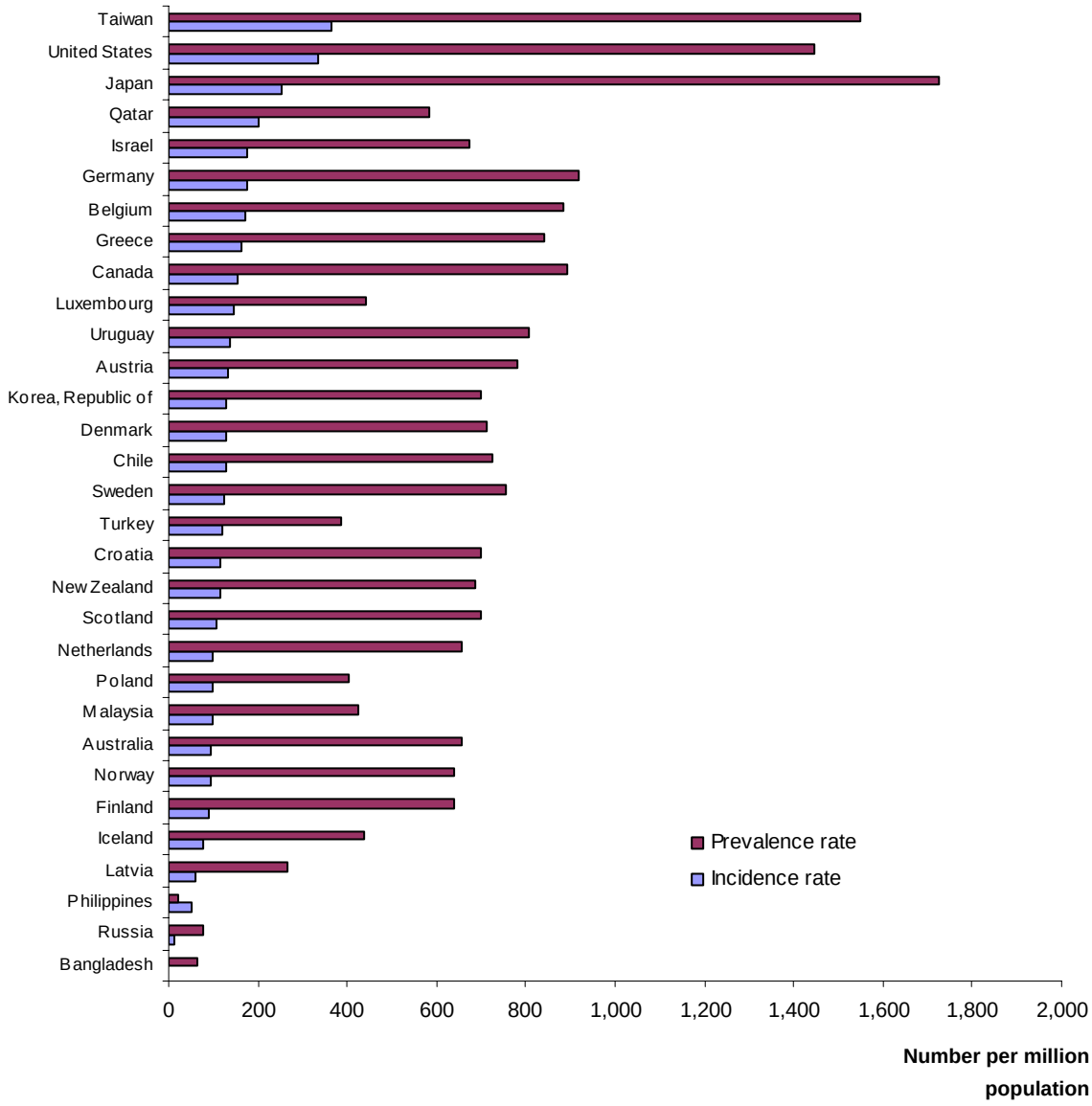
According to the data published by USRDS in 2004, the incidence and prevalence of treated end-stage kidney disease increased for most countries from 1998 to 2002, with the exception of Finland, Hungary, Russia and Sweden. In 2002, the highest incidence and prevalence rates occurred in the United States, Taiwan and Japan, and ranged from 1,446 to 1,726 per million population. The lowest rates were in the Philippines, Bangladesh and Russia, and ranged from 22 to 79 per million population (Figure 2.5).

Another marked change was the gradually increased percentage of new patients with diabetes, which happened in most of these countries.

Although treatment modalities for end-stage kidney disease varied widely from country to country, haemodialysis was the most common choice of treatment for most countries. There were significantly different functioning kidney rates and transplant rates among the countries compared. Both rates were highest in Norway and the US and lowest in Bangladesh.

In comparison, Australian incidence and prevalence rates of treated end-stage kidney disease ranked towards the lower end of these countries. Although haemodialysis was the major modality for dialysis, Australia had the second highest proportion of patients on peritoneal dialysis in the world. Both the functioning kidney rate and the transplant rate in Australia were ranked high middle of all countries, but were at the lower end when compared with those countries with similar economic conditions to Australia.

The incidence and prevalence of treated end-stage kidney disease can be affected by many factors. The differences in these rates not only reflect the true incidence and prevalence of end-stage kidney disease in these countries, but also can be much influenced by the availability and accessibility of kidney replacement therapy services provided by these countries. These are highly dependent on government infrastructure and economic conditions (USRDS 2003). Varying economic conditions may play a major role in the differences in incidence and prevalence of treated ESKD among these countries.



Source: USRDS 2004.

Figure 2.5: International comparison of incidence and prevalence of treated end-stage kidney disease, 2002

Mortality

CKD contributes significantly to mortality in Australia. It contributes to death in two ways: as the underlying cause of death (the condition that initiated the train of morbid events leading directly to death) or as an associated cause of death (a condition that gave rise to the underlying cause, or that contributed to the death but was not related to the disease or condition causing it) (ABS 2000).

In 2003, there were a total of 11,648 deaths in Australia where CKD was recorded as the underlying or an additional cause of death, with 6,272 deaths among males and 5,376 among females.

The contribution of CKD to deaths in Australia is described below through examining these underlying and additional causes. Note that there will be some overlap between the deaths presented here and deaths from other diseases published elsewhere. This is due to the inclusion of causes of death such as diabetic nephropathy and hypertensive renal disease, which are classified in other AIHW reports as deaths due to diabetes or cardiovascular disease, respectively.

Information from the ANZDATA Registry on deaths in people receiving treatment for ESKD is also presented.

Chronic kidney disease as the underlying cause of death

There were 2,431 deaths in 2003 where CKD was listed as the underlying cause of death (117 per million population, 2% of all deaths). CKD mortality rates were higher among males than among females across all age groups. The majority of deaths (87%) occurred among people aged 70 years and over.

Table 2.7: Chronic kidney disease as the underlying or an associated cause of death, by disease type, 2003

Type of chronic kidney disease	ICD-10 codes	Number as the underlying cause of death	Number as an associated cause of death	Ratio underlying: associated
Diabetic nephropathy	E10.2, E11.2, E12.2, E13.2, E14.2	91	61	1.4
Hypertensive kidney disease	I12, I13, I15.0, I15.1	477	214	2.2
Glomerular diseases	N00–N07	79	116	0.7
Kidney tubulo-interstitial diseases	N11, N12, N14, N15	93	82	1.1
Chronic kidney failure	N18	940	4,640	0.2
Unspecified kidney failure	N19	675	4,046	0.2
Congenital malformation of the kidney and ureter	Q60–Q63	41	50	0.8
Other disorders of the kidney and ureter	N25–N28, N39.1, N39.2, T82.4, T86.1	35	157	0.2
Total		**2,431**	**9,217**[(a)]	**0.3**

(a) Column will not add to total as more than one type of kidney disease may have been recorded.

Source: AIHW National Mortality Database.

Of these 2,431 deaths, 940 (39%) resulted from chronic kidney failure, with a further 675 (28%) attributed to unspecified kidney failure. Hypertensive kidney disease (477 deaths, 20%), kidney tubulo-interstitial diseases (93 deaths, 4%) and diabetic nephropathy (91 deaths, 4%) were also commonly recorded (Table 2.7).

Multiple cause of death data revealed that for most deaths with an underlying cause of CKD, one or more associated causes of death are recorded. Cardiovascular diseases are the most prominent associated causes and were listed in 58% of cases in 2003 (1,413 deaths). Other common associated causes include respiratory diseases (659 cases in 2003, 27%), acute kidney failure (341 cases, 14%), diabetes (214 cases, 9%), septicaemia (206 cases, 8%) and cancers (147 cases, 6%).

From 1997 to 2003, the mortality rate for CKD as the underlying cause of death decreased from 133 to 117 deaths per million population.

Chronic kidney disease as an associated cause of death

In 2003, CKD was listed on death certificates as an associated cause of death in 9,217 cases. As with the underlying cause of death, CKD is more commonly recorded as an associated cause of death among males, and mainly appears among people in the older age groups. Chronic and unspecified kidney failure are by far the most common CKD codes recorded as an associated cause of death (4,640 and 4,046 cases in 2003, respectively).

In cases where CKD is recorded as an associated cause of death, cardiovascular diseases are the most common underlying cause of death, with 4,045* such deaths in 2003 (44%). Other commonly recorded underlying causes of death include respiratory disease, cancers and diabetes (Table 2.8).

Between 1997 and 2003, the rate of deaths where CKD was recorded as an associated cause of death increased slightly from 449 to 456 deaths per million population.

CKD is nearly four times as likely to be recorded as an associated cause of death than as an underlying cause of death. There are several reasons that may contribute to this. In cases where uraemia (see Box 1.1) is the direct cause of death, CKD is likely to be recorded as the underlying cause of death. However, uraemia is not often directly responsible for death. From its early stages, CKD increases the risks of morbidity and mortality from a range of other diseases, such as cardiovascular disease and infections. Therefore, a substantial proportion of people with CKD may die from cardiovascular disease or other non-kidney causes before CKD reaches its end-stage (when uraemia occurs). When CKD does reach end-stage, kidney function is partly replaced through kidney replacement therapy. Treated ESKD patients do not generally die from uraemia, but die from complications of CKD or side effects of its treatment, unless they withdraw from kidney replacement therapy. These factors, along with the significant increase in the number of people receiving kidney replacement therapy, may have contributed to the different directions in the trends for CKD as an underlying or associated cause of death.

No causal relationship between the underlying and associated causes of death can be established from the AIHW National Mortality Database. However, some diseases, such as cardiovascular disease and diabetes, were much more likely to be recorded as the underlying cause of death in cases where CKD was recorded as an associated cause than in general (Table 2.8). These data highlight the associations between CKD and these diseases.

---

* This figure excludes deaths with an underlying cause of hypertensive kidney disease.

Table 2.8: Conditions for which chronic kidney disease was recorded as an associated cause of death, 2003

Underlying cause of death (ICD-10-AM code)	Deaths with CKD as an associated cause		All deaths	
	Number	Per cent	Number	Per cent
Cardiovascular diseases (I00–I99)[(a)]	4,045	44%	48,358	37%
Heart failure (I50)	*395*	*4%*	*2,432*	*2%*
Ischaemic heart diseases (I20–I25)	*2,445*	*27%*	*25,439*	*19%*
Cerebrovascular diseases (I60–I69)	*456*	*5%*	*12,240*	*9%*
Respiratory diseases (J00–J99)	822	9%	11,892	9%
COPD[(b)] (J41–J44)	*374*	*4%*	*5,434*	*4%*
Pneumonia (J12–J18)	*254*	*3%*	*3,501*	*3%*
Neoplasms (C00–D48)	1,695	18%	38,392	29%
Prostate cancer (C61)	*280*	*3%*	*2,842*	*2%*
Urinary tract cancers (C64–C68)	*211*	*2%*	*1,745*	*1%*
Diabetes[(c)] (E10–E14)	793	9%	3,298	2%
Septicaemia (A40–A41)	192	2%	1,079	1%
Other diseases	1,670	18%	26,842	20%
Total	**9,217**	**100%**	**132,292**	**100%**

(a) Excludes deaths with an underlying cause of hypertensive kidney disease.

(b) Chronic obstructive pulmonary disease.

(c) Excludes deaths with an underlying cause of diabetic nephropathy.

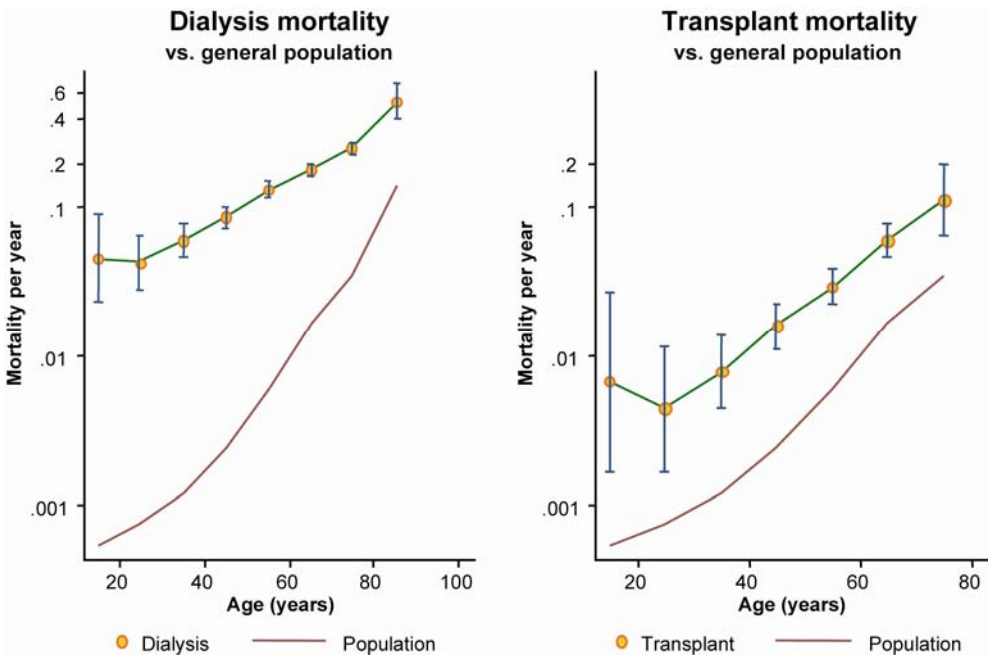
Source: AIHW National Mortality Database.

Deaths among people with treated end-stage kidney disease

According to the ANZDATA report, there were 1,260 deaths among people receiving treatment for end-stage kidney disease in 2003 (1,121 people who were dialysis dependent and 139 people who had received kidney transplants). The total number of deaths among these people in 2003 was more than five times higher than in 1981, when 230 deaths were recorded. This increase is in line with the higher prevalence of treated end-stage kidney disease and the increasing age of the patient population in recent years.

In 2003, the major causes of deaths among dialysis-dependent patients were cardiovascular diseases (40%), withdrawal from treatment (22%), infection (13%) and cancer (7%). Among kidney transplant patients, around 30% of deaths were due to cancers, followed by cardiovascular diseases (23%) and infections (17%) (Excell & McDonald 2005b).

Mortality rates among people with a functioning kidney transplant were much lower than those receiving dialysis treatment. However, the mortality rates in both groups were higher than in the general population (Figure 2.6).



Source: Excell & McDonald 2005b, p17. Reproduced with permission of the ANZDATA Registry.

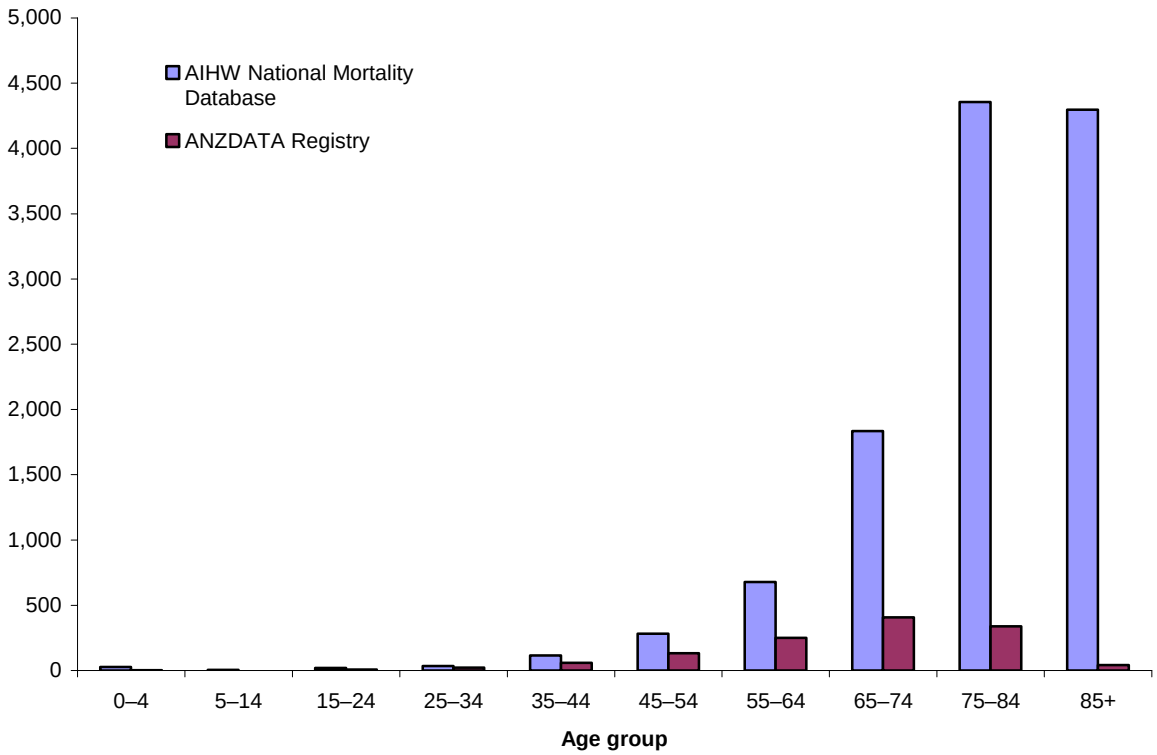
Figure 2.6: Mortality in people with treated end-stage kidney disease compared with the general population, 1998–2003

A comparison of chronic kidney disease mortality between the AIHW National Mortality Database and the ANZDATA Registry

The number of deaths where CKD was recorded as an underlying or associated cause in the AIHW National Mortality Database in 2003 was nearly 10 times the number of deaths recorded by the ANZDATA Registry among people with treated end-stage kidney disease (Figure 2.7). This implies that nine in ten people who died with CKD were not receiving kidney replacement therapy, although this does not necessarily mean that they required it.

Some of the excess deaths recorded in the AIHW National Mortality Database compared with ANZDATA may be attributed to deaths occurring in the early stages of CKD. These people therefore did not require kidney replacement therapy and were not recorded in the ANZDATA Registry. However a proportion may also be attributed to deaths of people who did not accept kidney replacement therapy, especially for people in the older age groups. The reasons for the lower acceptance of kidney replacement therapy among older Australians are not clear. It is thought that the serious comorbidity and significant disability that are highly prevalent in older people may influence decisions about suitability for treatment (Stewart et al. 2004).

The AIHW National Mortality Database does not distinguish between people who were or were not receiving kidney replacement therapy. Therefore, it is not possible to identify which people died during the early stages of CKD and determine what caused their death. Examination of the causes of death among people who died during the earlier stages of CKD is important in terms of developing strategies for prevention and management of the disease, and requires further investigation.



Notes

1. Data from AIHW National Mortality Database are deaths with an underlying or associated cause of CKD.
2. Data from ANZDATA Registry are deaths among people with treated end-stage kidney disease.

Sources: AIHW National Mortality Database; ANZDATA Registry.

Figure 2.7: Comparison of mortality, AIHW National Mortality Database and ANZDATA Registry, 2003

Health expenditure on chronic kidney disease

People with CKD use health services intensively to manage their disease. In particular, kidney replacement treatment by dialysis in its various forms is very expensive and needs to be repeated frequently to sustain patients' lives. Besides the direct expenditure on medical care and treatment, the indirect costs associated with other aspects of living with CKD are also numerous, such as the costs of travelling for treatment, the social and economic burden on carers and family, and lost wages and lost productivity due to illness.

This report provides data on direct health care expenditure for CKD—that is, money spent by governments, private health insurers, companies, households and individuals to prevent, diagnose and treat CKD. Due to lack of information, costs such as the time costs for carers or patients and work time lost due to CKD are not covered by this report. It should be noted that the data presented here are an estimate of direct expenditure on CKD itself. No attempt has been made to estimate expenditure due to the complications of CKD, or treatment for comorbid conditions in people with CKD. Complications and comorbidities may contribute substantially to overall expenditure and other costs for people with CKD. A detailed investigation of the economic burden of CKD has been commissioned by Kidney Health Australia.

The data on direct health expenditure in this report were drawn from the AIHW Disease Expenditure Database and additional analyses (AIHW 2005b). This database was compiled by allocating total recurrent health expenditure for 2000–01 to over 200 disease and injury categories, based on those used in the Australian burden of disease study (AIHW: Mathers et al. 1999). A detailed description of the methodology for the allocation of expenditure to disease is available in *Health system expenditure on disease and injury in Australia, 2000–01 (second edition)* (AIHW 2005b).

The disease categories used in the AIHW Disease Expenditure Database were based on burden of disease groupings. Since CKD is spread across several burden of disease groups it did not directly relate to a category in this database. Expenditure on CKD was estimated according to the diseases on the CKD coding list (Appendix 1) which involved taking various portions of the burden of disease groupings. Therefore parts of the expenditure on CKD overlap with burden of disease categories such as diabetes and so the results presented here are not directly comparable with those presented in the AIHW disease expenditure publications.

Total expenditure on chronic kidney disease

It is estimated that total recurrent health expenditure on CKD in 2000–01 was $647 million. This included:

- $397.2 million (61.4%) on admitted and non-admitted patient hospital services for dialysis
- $126.2 million (19.5%) on admitted and non-admitted patient hospital services for reasons other than dialysis
- $24.5 million (3.8%) on out-of-hospital medical services
- $83.1 million (12.8%) on pharmaceuticals (prescription, over-the-counter medications and highly specialised drugs)

- $5.7 million (0.9%) on research
- $2.8 million (0.4%) on services provided by other professionals
- $7.7 million (1.2%) on high-level residential aged care.

This expenditure of $647 million in 2000–01 is 1.3% of the total recurrent health expenditure of $50.2 billion that was able to be allocated by disease (AIHW 2005b).

Expenditure on dialysis services

The cost of haemodialysis in hospital or satellite centres was estimated to be $427 per separation (National Hospital Cost Data Collection). Dialysis is usually performed three times per week, therefore the total cost of dialysis in hospital was estimated to be $66,000 per person per year.

Estimation of the cost of home dialysis is difficult as there are no national data on dialysis performed outside of hospitals or satellite centres. A Victorian study estimated that home-based haemodialysis cost $9,891 less per person per year than hospital dialysis (Victorian Department of Human Services 2004). Applying this difference to the national data gives an estimated cost for home-based haemodialysis of over $56,000 per person per year. This study also estimated that both forms of peritoneal dialysis cost around $44,000 per person per year (Table 2.9). Total expenditure for home and peritoneal dialysis was estimated to be $116 million.

Table 2.9: Cost per year for different types of dialysis, 2000–01

Type of dialysis	Cost per patient per year ($)
Haemodialysis in hospital or satellite centre	66,072
Haemodialysis at home	56,181
Continuous ambulatory peritoneal dialysis	43,996
Automated peritoneal dialysis	43,996

Note: The estimate of expenditures on haemodialysis in hospital or satellite centre is an average of the hospital and satellite centre costs.

Sources: Victorian Department of Human Services 2004; National Hospital Cost Data Collection Cost Report Round 5 (2000–01).

Hospital services other than for dialysis

After dialysis services, other hospital services accounted for the largest portion of expenditure on CKD in 2000–01, around one-fifth of the total ($126 million). Of this, $99 million (79%) was for admitted patient services. A further $27 million was estimated to be spent on non-dialysis non-admitted patient services, but an unknown portion of this is also counted in out-of-hospital specialist expenditure.

Other expenditure relating to chronic kidney disease

Expenditure on CKD-related community pharmaceuticals in 2000–01 was estimated to be $9.9 million. This included $6.8 million on prescription medications and $3.1 million on over-the-counter medications.

Expenditure on highly specialised drugs for people with kidney transplants was estimated to be $73.1 million: $24.3 for immunosuppressive drugs and $48.8 million for haemopoietics.

(All highly specialised drugs are prescribed through hospitals, but are not included as part of hospital costs in Table 2.10. Pharmaceuticals used in hospitals for admitted patients with CKD are included with hospital costs in Table 2.10 as they cannot be identified separately.)

Out-of-hospital medical services for CKD include medical imaging (such as X-rays and ultrasound), pathology, visits to GPs and consultations with specialists outside of hospital. In 2000–01, direct health expenditure on out-of-hospital medical services relating to CKD was $24.5 million (Table 2.10). This included $5.4 million for GP consultations, $9.4 million for imaging and pathology and $9.7 million for specialist consultations. This $9.7 million includes the total expenditure through Medicare by nephrologists for out-of-hospital medical services ($8.3 million).

CKD-related expenditure on other professional services in 2000–01 (including those provided by allied health professionals, such as dietitians and psychologists in private practice) amounted to $2.8 million.

Table 2.10: Expenditure on chronic kidney disease, 2000–01, $million

		Type of expenditure			
Area of expenditure		**Dialysis[a]**	**Kidney transplant**	**Other CKD expenditure[b]**	**Total CKD**
Hospitals	Total	397.2	11.5	114.7	523.4
	Admitted patient[c]	*287.4*	*11.5*	*87.7*	*386.6*
	Non-admitted patients	*109.8*	*n.a.*	*27.0*	*136.8*
Aged care homes (high care component)		n.a.	n.a.	n.a.	7.7
Medical services (out-of-hospital)	Total	n.a.	n.a.	n.a.	24.5
	Unreferred attendances (GP)	*0.03*	*0.1*	*5.2*	*5.4*
	Imaging and pathology	*n.a.*	*n.a.*	*n.a.*	*9.4*
	Specialist	*n.a.*	*n.a.*	*n.a.*	*9.7*
Pharmaceuticals	Total	0.3	0.0	9.0	83.1
	Pharmaceuticals requiring a prescription	*0.3*	*0.6*	*5.9*	*6.8*
	Highly specialised drugs	*0.0*	*73.1*	*0.0*	*73.1*
	Over-the-counter	*0.0*	*0.0*	*3.1*	*3.1*
Other health professionals		0.0	0.0	2.8	2.8
Research		n.a.	n.a.	n.a.	5.7
Total expenditure		**n.a.**	**n.a.**	**n.a.**	**647.1**

(a) Includes haemodialysis in hospitals and satellite centres, peritoneal dialysis and expenditure on treatment of infection and inflammatory reactions due to peritoneal dialysis catheters.

(b) Includes an estimated CKD portion of expenditure on the burden of disease categories of diabetic nephropathy, hypertensive renal disease, genitourinary system disease and genitourinary system congenital malformations.

(c) Includes cost of private medical services delivered to admitted patients who are private.

n.a. Not available.

Note: Columns may not add to totals due to rounding.

Expenditure on high-level residential aged care services for people with CKD was estimated as $7.7 million for 2000–01. This estimate was based on Australian Bureau of Statistics data from 1998 which indicated that 0.29% of residents in residential aged care had as their main condition ICD-10 disorders N00 to N39. This proportion (with some adjustment for the relative costliness of different disorders) was applied to total expenditure for high-level

residential aged care. This is likely to be an overestimate of actual residential aged care expenditure for people with CKD as N00 to N39 includes disorders such as stress incontinence.

Direct health expenditure on CKD-related research in 2000–01 was estimated to be $6 million. (This was 43% of the research estimated to be for the genitourinary system. Genitourinary system research was estimated to be 1.1% of total health research based on data from the ABS 2000–01 survey of research and experimental development.)

The research expenditure is for research which supports the understanding of the causes, extent and impact of CKD, the development and evaluation of new and existing treatment methods, public health interventions, and a portion of general health services and epidemiological research.

References

ABS (Australian Bureau of Statistics) 2000. Cause of death, Australia 1999. Canberra: Australian Bureau of Statistics.

AIHW (Australian Institute of Health and Welfare) 2004a. Australia's health 2004. Canberra: AIHW.

AIHW 2004b. Australian hospital statistics 2002–03. AIHW Cat. No. HSE 32. Canberra: AIHW (Health Services Series No. 22).

AIHW 2004c. Disability and its relationship to health conditions and other factors. AIHW Cat. No. DIS 37. Canberra: AIHW (Disability Series).

AIHW 2005a. Australian hospital statistics 2003–04. AIHW Cat. No. HSE 37. Canberra: AIHW (Health Services Series No. 23).

AIHW 2005b. Health system expenditure on disease and injury in Australia, 2000–01. 2nd ed. AIHW Cat. No. HWE 28. Canberra: AIHW (Health and Welfare Expenditure Series No. 21).

AIHW: Britt H, Miller GC, Know S et al. 2005. General practice activity in Australia 2003–2004. AIHW Cat. No. GEP 16. Canberra: AIHW (General Practice Series No.16).

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

Australian Kidney Foundation 2002. Living with kidney failure. 6th ed. Sydney: Australian Kidney Foundation.

Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL & Jameson JL 2001. Harrison's principals of internal medicine. 15th ed. New York: McGraw-Hill.

Chadban SJ, Briganti EM, Kerr PG et al. 2003. Prevalence of kidney damage in Australian adults: the AusDiab kidney study. Journal of the American Society of Nephrology 14:S131–8.

Chapman J & Webster A 2005. Cancer report. In: Excell L & McDonald SP (eds). ANZDATA Registry 27th report. Australia and New Zealand Dialysis and Transplant Registry. Adelaide: ANZDATA, 99–103.

Chow FY, Briganti EM, Kerr PG, Chadban SJ, Zimmet PZ & Atkins RC 2003. Health-related quality of life in Australian adults with renal insufficiency: a population-based study. American Journal of Kidney Diseases 41(3):596–604.

Excell L, Chadban SJ & McDonald SP 2005. Transplantation. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Excell L & McDonald SP 2005a. Stock and flow. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry, 1–6.

Excell L & McDonald SP 2005b. Deaths. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry, 15–24.

Excell L & McDonald SP 2005c. Method and location of dialysis. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Go AS, Chertow GM, Fan D, McCulloch CE & Hsu C 2004. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. New England Journal of Medicine 351:13.

Jindal RM, Joseph JT, Morris MC, Santella RN & Baines LS 2003. Noncompliance after kidney transplantation: a systematic review. Transplantation Proceedings 35:2868–72.

Joint Specialty Committee on Renal Disease 2005. Chronic kidney disease in adults: UK guidelines for identification, management and referral. Viewed 29 August 2005, <http://www.renal.org/CKDguide/full/UKCKDfull.pdf>.

Koo JR, Yoon JY, Joo MH et al. 2005. Treatment of depression and effect of antidepression treatment on nutritional status in chronic hemodialysis patients. The American Journal of the Medical Sciences 329:1–5.

Kouidi E 2004. Health-related quality of life in end-stage renal disease patients: the effects of renal rehabilitation. Clinical Nephrology 61 Suppl. 1:S60–71.

Levendoglu F, Altintepe L, Okudan N et al. 2004. A twelve week exercise program improves the psychological status, quality of life and work capacity in hemodialysis patients. Journal of Nephrology 17:826–32.

Levey AS, Bosch JP, Lewis JB et al. 1999. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. Annals of Internal Medicine 130(6):461–70.

Lew SQ & Piraino B 2005. Quality of life and psychological issues in peritoneal dialysis patients. Seminars in Dialysis 18(2):119–23.

Maisonneuve P, Agodoa L & Gellert R 1999. Cancer in patients on dialysis for end-stage renal disease: an international collaborative study. The Lancet 354:93–8.

Mathew T 2005: Addressing the epidemic of chronic kidney disease in Australia. Nephrology (Carlton) 9 Suppl. 4:S109–12.

McDonald SP & Russ G 2002. Survival of recipients of cadaveric kidney transplants compared with those receiving dialysis treatment in Australia and New Zealand, 1999–2001. Nephrology Dialysis Transplantation 17:2212–9.

Nascimento MM, Qureshi AR, Stenvinkel P et al. 2004. Malnutrition and inflammation are associated with impaired pulmonary function in patients with chronic kidney disease. Nephrology Dialysis Transplantation 19:1823–8.

NKF (National Kidney Foundation of America) 2002. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. American Journal of Kidney Disease 39 Suppl. 1:S1–266.

Polaschek N 2003. The experience of living on dialysis: a literature review. Nephrology Nursing Journal (Pitman) 30(3):303.

Stewart JH, McCredie MR, Williams SM & McDonald SP 2004. Interpreting incidence trends for treated end-stage renal disease: implications for evaluating disease control in Australia. Nephrology 9:238–46.

The Australasian Creatinine Consensus Working Group 2005. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: a position statement. Medical Journal of Australia 183(3):138–41.

Tossani E, Cassano P & Fava M 2005. Depression and renal disease. Seminars in Dialysis 18(2):73–81.

Tsay SL & Hung LO 2004. Empowerment of patients with end-stage renal disease—a randomised controlled trial. International Journal of Nursing Studies 41:59–65.

USRDS (US Renal Data System) 2003. 2003 annual data report: atlas of end-stage renal disease in the United States. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases.

USRDS 2004. 2004 annual data report: atlas of end-stage renal disease in the United States. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases.

Victorian Department of Human Services 2004. Renal dialysis: a revised service model for Victoria. Melbourne: Department of Human Services.

3 Risk factors and causes of chronic kidney disease

Introduction

As described in Chapter 2, the burden posed by CKD in Australia is substantial. Assessing the prevalence of the risk factors for and causes of CKD in the population is useful in understanding underlying trends in disease incidence, as well as for predicting future trends in disease incidence, prevalence and mortality. Monitoring the prevalence and distribution of these risk factors and causes across the population can also provide insight into the success of health-related campaigns or the need to initiate health promotion interventions, and where and to whom these may need to be targeted.

However, the relationships between CKD, its risk factors and causes are very complex and not well understood. Although a number of risk factors and causes have been identified, little is known about the natural history of CKD or the rate of progression from exposure to these risk factors and causes to onset of CKD. The interactions between these factors and causes bring further difficulties to understanding the onset and development of CKD. There is a lack of data on the prevalence of most of the causes of CKD in Australia, and so it is not clear what proportion of CKD may be attributed to each cause. In some cases, the cause of CKD is not able to be identified.

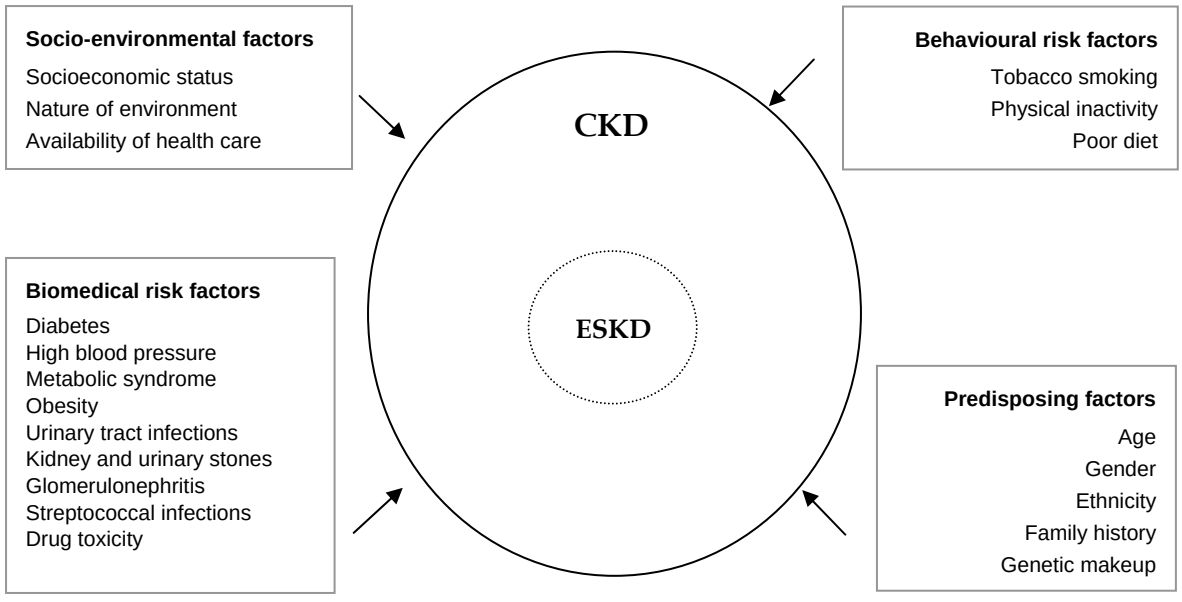


Figure 3.1: Risk factors and determinants of chronic kidney disease

The risk of developing CKD may be increased by several different types of factors in a variety of ways (Figure 3.1). Most of the biomedical risk factors are diseases and conditions that can directly initiate kidney damage. These include conditions both inside and outside the urinary system. The onset and development of CKD interacts with the onset and development of these biomedical factors. Poor management of these diseases and conditions in people with CKD can also accelerate kidney damage. In turn, CKD has similar impacts on other diseases (such as heart disease).

Behavioural and other biomedical risk factors tend to lead to CKD by increasing the risks of developing the diseases and conditions that cause kidney damage. Some of these risk factors, such as smoking and physical inactivity, have also been found to increase the risk of CKD independently.

Most CKD risk factors are common in Australia. According to the 2001 National Health Survey, nine in ten Australians aged 18 years and over reported having at least one of the following: overweight, physical inactivity, poor nutrition, tobacco smoking, high blood cholesterol, high blood pressure and diabetes. This corresponds to an estimated 13 million Australians affected (AIHW 2004). These risk factors rarely act alone or independently. They tend to coexist and to interact in their effects. The more risk factors a person has, the greater is his or her risk of developing CKD.

Socio-environmental and predisposing factors also influence the onset and progress of CKD. Older people, people with a family history of CKD, Indigenous Australians and people with low socioeconomic status tend to have increased susceptibility to kidney damage, regardless of what other risk factors they may have.

This chapter discusses the risk factors for and causes of CKD. Due to the complex relationships involved it is difficult to consider these issues separately. The chapter is therefore structured around three interrelated topics:

- biomedical factors causing kidney damage—covering diseases and conditions both inside and outside the urinary system;
- modifiable factors increasing risk of CKD—including the biomedical and behavioural risk factors that directly or indirectly increase the risk of CKD;
- other factors influencing CKD—socioeconomic status and age.

Biomedical factors causing kidney damage

Diabetes and diabetic nephropathy

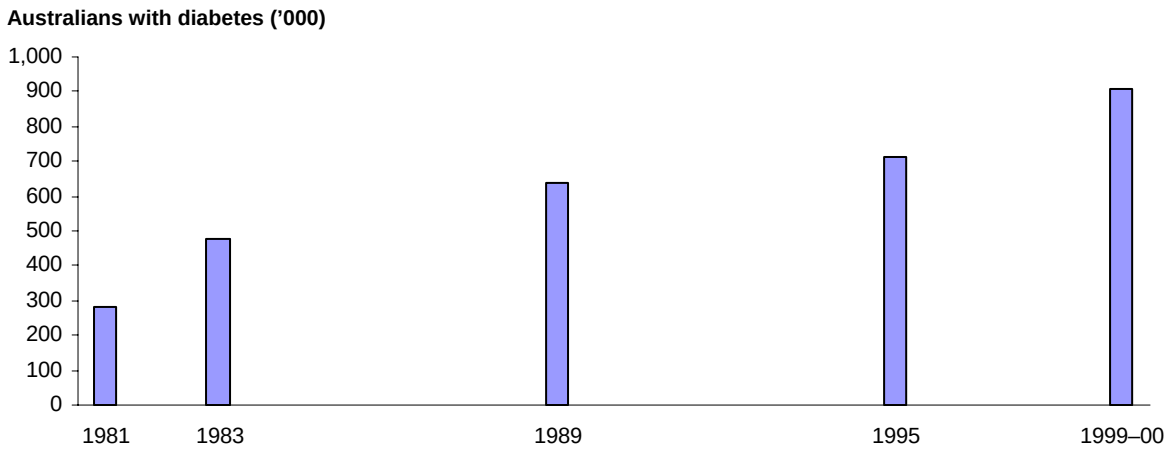
Diabetes mellitus is a long-term condition in which blood glucose levels are too high because the body produces little or no insulin, or cannot use insulin properly. Poorly managed diabetes may result in high blood sugar levels causing damage to the blood-filtering capillaries in the kidneys, a complication known as diabetic nephropathy.

Both genetic and environmental factors contribute to the onset of diabetes. Type 1 diabetes is believed to be caused by exposure to environmental triggers, possibly certain viruses or food toxins. The development of Type 2 diabetes is influenced largely by the presence of behavioural and biomedical risk factors including obesity, physical inactivity, and possibly poor nutrition in foetal and early infant life (AIHW 2002b). There is therefore potential to prevent or delay the onset of Type 2 diabetes in those at risk through modifying and controlling risk factors.

In recent years, diabetes has become one of the leading threats to the health of Australians. It is estimated that about 946,000 Australians aged 25 years and over (7.6% of that population) have diabetes, and the number of adults with the condition has trebled since 1981 (Figure 3.2). The growing epidemic of diabetes is mainly attributed to the recent rise in Type 2 diabetes, which contributes 85–90% of cases of diabetes in Australia (AIHW 2004).

Type 1 diabetes is one of the most serious and common chronic diseases of childhood, with about half of the people with Type 1 diabetes developing the disease before 18 years of age. According to self-reported data from the 2001 National Health Survey (NHS), about 0.5% of Australians (around 95,000 people) have Type 1 diabetes. Kidney damage generally takes 15 to 25 years to develop after the onset of diabetes. Due to the generally younger age of onset of Type 1 diabetes, people with this type of diabetes who develop progressive kidney damage tend to reach end-stage kidney disease at a relatively young age.

Type 2 diabetes is most common in those aged 40 years and over, though it may also occur in younger adults and even in children and adolescents. Self-reported information from the 2001 NHS shows that around 2.3% of Australians have Type 2 diabetes. However, Type 2 diabetes is known to be under-reported in self-report surveys, as it may be present without symptoms and therefore people may not be aware that they have the disease. Objectively measured data from the AusDiab study suggest that 7.2% of Australians aged 25 years or over (more than 900,000 people of this age) have Type 2 diabetes. Prevalence increases with age and is higher in males than females except in the oldest age group, where the prevalence is similar in both sexes (Table 3.1).



Note: Estimates based on self-reports (1983, 1989, 1995) have been adjusted for undiagnosed cases of diabetes based on the assumption that for every diagnosed case there exists an undiagnosed case (Guest et al. 1992; Dunstan et al. 2001).

Source: AIHW 2002b.

Figure 3.2: Australians aged 25 years and over with diabetes, 1981 to 1999–00

Kidney problems have been found to be highly prevalent among people with diabetes. Among participants in the AusDiab study, 27.6% of people with diabetes also had CKD, corresponding to around 250,000 Australians aged 25 years and over. The prevalence of CKD was three times as high in those with diabetes compared with those without (Chadban et al. 2003).

The burden of CKD relating to diabetes, particularly Type 2 diabetes, is likely to increase further as both the age of the population and prevalence of Type 2 diabetes are expected to rise (AIHW 2002b).

Table 3.1: Prevalence of Type 2 diabetes, people aged 25 years and over, 1999–00

Age group	Males	Females	Persons
		(per cent)	
25–34	0.1	0.1	0.1
35–44	2.4	1.8	2.1
45–54	5.8	5.4	5.6
55–64	16.5	9.5	13.0
65–74	20.4	15.4	17.7
75+	22.0	22.6	22.3
25+[(a)]	**8.3**	**6.5**	**7.3**

(a) Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of the 1999–00 AusDiab study.

Diabetic nephropathy

This is a severe complication of diabetes that results from high blood sugar levels damaging the blood-filtering capillaries in the kidneys. When the capillaries are damaged and the kidneys' filtering efficiency declines, blood proteins such as albumin leak into the urine (called albuminuria). In the early stages of diabetic nephropathy, small quantities of albumin leak into the urine (called microalbuminuria). As diabetic nephropathy progresses, the kidneys leak larger amounts of albumin (called macroalbuminuria or proteinuria).

Diabetic nephropathy can occur in both Type 1 and Type 2 diabetes, and generally takes 15 to 25 years to develop after the onset of diabetes. If not detected and well managed, it can progress rapidly and may result in end-stage kidney disease. In Australia the rapid increase in Type 2 diabetes is thought to have been a major contributor to the rising incidence of treated end-stage kidney disease in recent years (Stewart et al. 2004).

Diabetic nephropathy is often symptomless until late in the disease when therapeutic interventions are ineffective. However, it can be readily detected by urine testing for albumin, and tight control of blood glucose levels and treatment with angiotensin-converting enzyme (ACE) inhibitors can slow the progression of kidney damage (DCCT 1993).

High blood pressure and hypertensive kidney disease

High blood pressure (hypertension) is another major cause of CKD. Untreated high blood pressure can damage the blood vessels in the kidneys. The walls of these blood vessels become thick and the internal diameter narrowed, leading to reduced blood supply and decreased kidney function. This is called hypertensive kidney disease.

The level of blood pressure tends to increase with age. When people get older, they are more at risk of developing high blood pressure (Box 3.1). High blood pressure is significantly associated with obesity and high dietary salt intake. Other factors that can contribute to increased blood pressure are smoking and high alcohol consumption.

Box 3.1: Classification of high blood pressure

In this report, high blood pressure (also called hypertension) is defined as:

- *systolic blood pressure (SBP) greater than or equal to 140 mmHg; and /or*
- *diastolic blood pressure (DBP) greater than or equal to 90 mmHg; and/or*
- *receiving medication for high blood pressure.*

Source: WHO-ISH 1999.

High blood pressure is very common in Australia. In 1999–00, over 3.6 million Australians aged 25 years and over had high blood pressure or were on medication for this condition. The prevalence rate increased with age, and was higher among males than females (31% and 26%, respectively) (Table 3.2).

According to the AusDiab study, about 27.3% of participants with high blood pressure also had CKD, corresponding to nearly one million Australians aged 25 years and over. The prevalence of CKD was fivefold greater among participants with high blood pressure compared with those with normal blood pressure (Chadban et al. 2003).

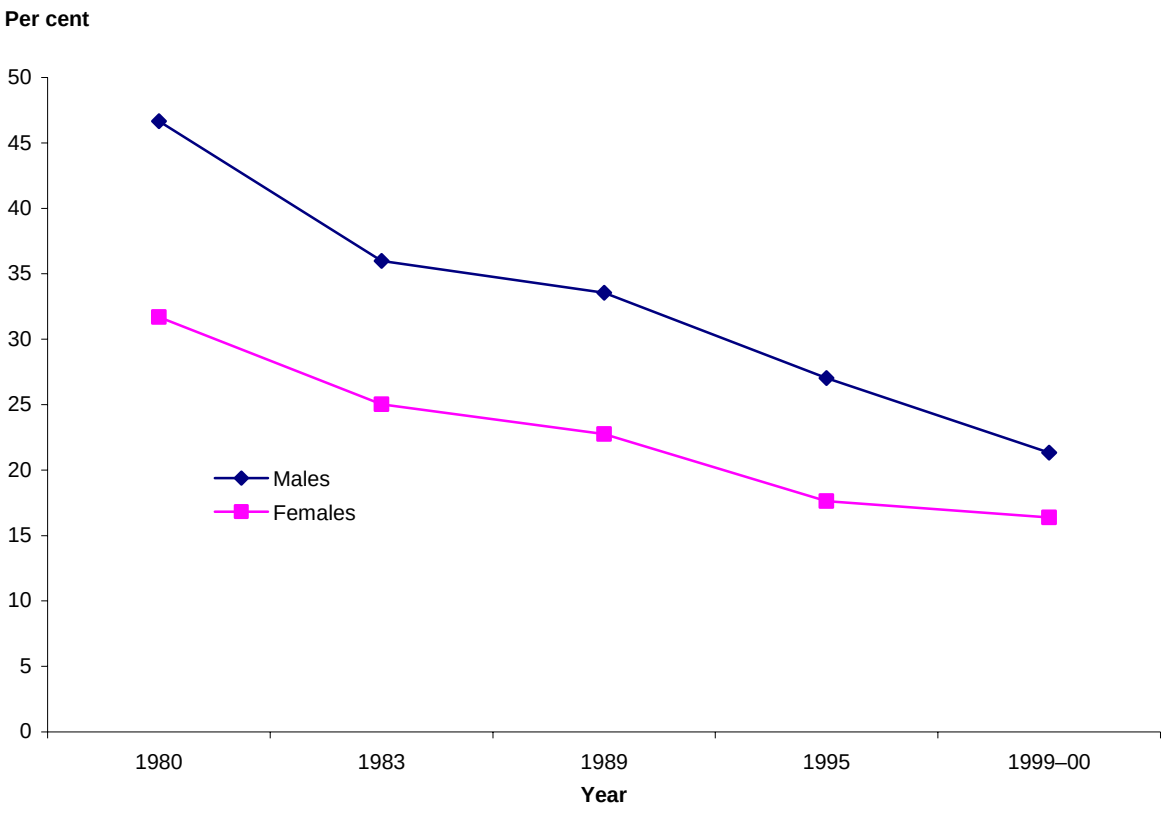
Table 3.2: Prevalence of high blood pressure, people aged 25 years and over, 1999–00

Age group	Males	Females	Persons
		(per cent)	
25–34	7.1	3.4	5.2
35–44	14.0	7.6	10.8
45–54	30.5	23.7	27.1
55–64	49.3	44.5	46.9
65–74	69.4	66.8	68.0
75+	78.8	74.6	76.3
25+[(a)]	**32.3**	**27.2**	**29.7**

(a) Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of 1999–00 AusDiab study.

Trend data for measured blood pressure are only available for people aged 25–64 years living in urban areas of Australia. Over the last two decades there have been large declines in the prevalence of high blood pressure (Figure 3.3). For men the prevalence has fallen steadily from 47% in 1980 to 21% in 1999–00. The rate for women has halved, from 32% in 1980 to 16% in 1999–00.



Notes

1. Age-standardised to the Australian population at 30 June 2001.
2. Urban areas only.

Sources: AIHW analysis of the 1980, 1983 and 1989 Risk Factor Prevalence Surveys, the 1995 National Nutrition Survey and the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab).

Figure 3.3: Trends in prevalence of hypertension, people aged 25–64 years, 1980 to 1999–00

Hypertensive kidney disease

The causes of hypertensive kidney disease are complex. Factors that have been implicated in its development include smoking, insulin resistance, excess salt intake, cocaine use, lead and cadmium exposure, and genetic factors (Tylicki et al. 2002).

Treatment involves controlling blood pressure through diet, exercise and use of blood-pressure-lowering drugs such as ACE inhibitors, and treating any kidney-related symptoms.

Glomerulonephritis

Glomerulonephritis is a group of kidney diseases characterised by inflammation of the glomeruli, which can lead to gradual, progressive destruction of the internal kidney structure. Outcomes for people with glomerulonephritis range from complete recovery to end-stage kidney disease, depending on the particular type of glomerulonephritis they have (Francis & Tomson 2004). The milder forms are relatively common and respond well to drug therapy, particularly when found in children. The more severe forms, although less common, often result in ESKD. In rapidly progressive cases, the end-stage may be reached within a few weeks to months, but more often this takes 10–20 years and there is opportunity for intervention to delay progress of the disease (Braunwald et al. 2001).

The causes of glomerulonephritis are complex and not completely understood. Many factors may contribute to its occurrence and development, such as autoimmunity (when a person's immune system reacts against their own body), cancer, structural abnormalities within the kidney, and infections (Chadban & Atkins 2005). There is also individual variability in susceptibility to glomerulonephritis, which is likely to have a genetic basis. Certain populations are at increased risk of glomerulonephritis. One Australian study has found that Australian Aboriginal children are at increased risk of infection-associated glomerulonephritis after streptococcal skin and throat infections (Streeton et al. 1995). A separate study found that this then increased the risk of kidney damage in later life (White et al. 2001).

The incidence and prevalence of glomerulonephritis in the general population in Australia are unknown. A study of all renal biopsies done in Victoria during 1995 and 1997 indicated that the incidence of biopsy-proven glomerulonephritis was 12.3 per 100,000 population (Briganti et al. 2000).

Tubulo-interstitial kidney diseases

These are diseases that affect the kidney tubules or interstitial tissue. The tubules are part of the filtering units of the kidneys, and are attached to the glomeruli. The interstitial tissue surrounds the tubules and glomeruli. Direct causes of tubulo-interstitial diseases include urinary reflux and toxic effects of certain drugs or heavy metals. Tubulo-interstitial diseases may also be secondary to other diseases such as cancers, infections, sickle-cell anaemia and systemic lupus erythematosus (an autoimmune disease which affects the body's tissues).

Analgesic nephropathy

Analgesic nephropathy is a type of toxic injury to the kidneys. It usually results from long-standing daily use of analgesics (pain killers), especially medications that contain phenacetin (Sandler et al. 1989). Constant use of analgesics can damage the internal structure

of the kidney. The impairment develops over years and gradually leads to irreversible kidney damage (analgesic nephropathy) and ESKD.

Reflux nephropathy

In reflux nephropathy, the kidneys are damaged by backward flow of urine into the kidney. Normally, urine is formed in the kidneys and flows through the ureters into the bladder, to be passed out of the body by the contraction of the bladder. Each ureter has a one-way valve where it enters the bladder, preventing urine from flowing back up the ureter. When these valves do not work properly, urine may flow backwards up the ureter toward the kidney when the bladder contracts. The kidney is exposed to the possibility of infection if the bladder is infected or the urine contains bacteria.

The abnormal structure or function of these valves is most often congenital, but it may also be associated with other conditions including recurrent urinary tract infection in early childhood, bladder infection, bladder stones, bladder outlet obstruction and abnormal ureters (Haslett et al. 1999:450–1).

Congenital kidney disorders

Congenital disorders are those that exist at birth. They may be inherited, or occur during foetal development. Development-related disorders that cause CKD include malformation, duplication or wrong positioning of the kidneys or ureter, and failure of the kidneys to develop completely or at all. Under-developed kidneys will have fewer glomeruli, and so will not be able to function as effectively. The kidneys will have to work harder than they should and kidney damage will occur.

The most common inherited kidney disorders are polycystic kidney diseases (PKD). In PKD, multiple cysts develop on the kidneys, which interfere with their ability to filter waste products from the blood, causing CKD.

The two major forms of polycystic kidney disease are distinguished by their patterns of inheritance. Adult PKD is a genetically dominant form, which means that if one parent has the disease, each child has a 50% chance of developing it. It has signs and symptoms that typically begin in adulthood, although cysts in the kidney are often present from childhood. Infantile PKD is a genetically recessive form, meaning that both parents need to carry the gene for the disease for the child to be at risk of developing it (though they have a 50% chance of becoming a carrier, able to pass the gene to their own children). This form is much rarer and often leads to death during early childhood. The signs and symptoms of infantile PKD are usually apparent at birth or in early infancy (Braunwald et al. 2001).

Kidney and urinary stones

Kidney and urinary stones (also called ‘calculi’) are hard, rock-like crystals of chemicals found in the urine. Normally, the urine contains chemicals which help to stop these stones forming, but sometimes there may not be enough of these, or there may be an excess of the stone-forming chemicals. Repeated urine infections, gout, certain inherited conditions and some medications can increase the risk of kidney or urinary stones (Braunwald et al. 2001). Indigenous Australians, people who are obese and those with a family history of stones are also at higher risk (Carson & Brewster 2003; Goldfarb et al. 2005; Taylor et al. 2005).

Stones may cause blockages of the urinary tract, which can damage the kidneys. They also increase the risk of infection, which can increase the risks of glomerulonephritis and reflux nephropathy.

Urinary tract infections

These infections may affect different parts of the urinary system. If only the urethra (the tube from the bladder to the outside) is infected, this is called 'urethritis'. If the infection extends up into the bladder, it is called 'cystitis'. Occasionally the infection spreads to the kidneys; this is called 'pyelonephritis' and may cause kidney damage.

Cystitis is the most common type of urinary tract infection. Women are much more likely to contract these infections than men as their urethra is much shorter, meaning the infection can easily reach the bladder. Infections in males are rare, except in older men with prostate problems (Braunwald et al. 2001).

Symptoms of urinary tract infections may include a burning sensation when passing urine, frequent desire to urinate, cloudy or bloody urine, and lower abdominal pain. Pyelonephritis may also cause fever, nausea and vomiting.

Streptococcal infections

Streptococci are a type of bacteria found commonly in humans and domestic animals. They are responsible for several different infections such as pharyngitis ('strep throat'), tonsillitis, scarlet fever, rheumatic fever, meningitis and various skin infections (Haslett et al. 1999). Streptococcal skin and throat infections can increase the risk of glomerulonephritis; this has been found to be a particular risk among Australian Aboriginal children (Streeton et al. 1995).

Modifiable factors increasing risk of chronic kidney disease

Metabolic syndrome

The metabolic syndrome is a cluster of biomedical risk factors, typically characterised by central obesity, insulin resistance, high blood pressure, and cholesterol or triglyceride abnormalities. The syndrome has been linked to increased risk of chronic kidney disease and kidney damage. Kurella et al. (2005) report a 24% higher risk of developing CKD in those with metabolic syndrome compared to those without, independent of the effects of diabetes and high blood pressure. Chen et al. (2004) found that adults with the metabolic syndrome are 2.6 times as likely to have chronic kidney disease as those without.

People with metabolic syndrome are three to six times as likely to develop Type 2 diabetes as those who do not have the syndrome (Dekker et al. 2005; Ford 2005). People with Type 2 diabetes who also have metabolic syndrome are more likely to develop kidney complications (Isomaa et al. 2001b). Thorn et al. (2005) report associations between poor blood sugar control, metabolic syndrome and diabetic nephropathy in people with Type 1 diabetes.

The definition of metabolic syndrome has been much debated, and several definitions have been proposed. The definitions are similar in the risk factors included but differ in the central component and the levels at which a person is considered to have each of the factors. The most recent definition (Box 3.2), published by the International Diabetes Federation in 2005, is intended to be easily applicable in clinical practice.

Box 3.2: Metabolic syndrome

For a person to be defined as having the metabolic syndrome they must have:

excess abdominal weight (waist circumference ≥94 cm for Caucasian men and ≥80 cm for Caucasian women – ethnicity-specific values apply for other groups)

plus any two of the following:

- *raised triglyceride level (≥1.7 mmol/L) or receiving treatment for raised triglycerides*
- *reduced HDL cholesterol (<1.03 mmol/L in males and <1.29 mmol/L in females) or receiving treatment for reduced HDL cholesterol*
- *raised blood pressure (systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg) or receiving treatment for previously diagnosed hypertension*
- *raised fasting plasma glucose (≥5.6 mmol/L) or previously diagnosed Type 2 diabetes.*

Source: Adapted from IDF 2005.

Accurate information on the prevalence of the metabolic syndrome is lacking in Australia. The 1999–00 AusDiab study collected objectively measured data on each of the factors needed to define metabolic syndrome, and detailed estimates of metabolic syndrome prevalence from this study are soon to be published. Initial results suggest that 29% of Australians aged 25 years and over may have metabolic syndrome (Zimmet et al. 2005).

Tobacco smoking

Tobacco smoking is a widespread behaviour with serious health consequences. It is the risk factor associated with the greatest burden of disease in Australia (AIHW: Mathers et al. 1999). Smoking is an independent risk factor for CKD. The risk of CKD can be increased directly through damaging kidney function, and also can be mediated through high blood pressure and other illnesses.

Smoking is associated with kidney damage in the healthy population. Men are particularly at risk from the effects of smoking on kidney function impairment. A recent Australian study showed that men who smoked were more than three times as likely as non-smokers to have reduced kidney function (Briganti et al. 2002). Smoking has also been found to increase the risk of kidney damage among people with primary kidney diseases, such as glomerulonephritis, polycystic kidney disease and diabetic nephropathy, and to accelerate the development and progress of these diseases (Stengel et al. 2000; Orth et al. 1998; Norden & Nyberg 1984).

Smoking in this report refers to the smoking of tobacco products, including packet cigarettes, roll-your-own cigarettes, pipes and cigars. 'Daily smokers' refers to those who smoke at least one cigarette per day, and 'occasional smokers' refers to those who smoke less often than daily.

Smoking is highly prevalent in Australia. According to the 2004 National Drug Strategy Household Survey, 2.9 million (17.4%) Australians aged 14 years and over were daily smokers, and an additional half million (3.2%) smoked occasionally (AIHW 2005).

Table 3.3: Prevalence of daily smoking, people aged 14 years and over, 2004

Age group	Males	Females	Persons
		(per cent)	
14–19	9.5	11.9	10.7
20–29	24.0	22.9	23.5
30–39	23.8	21.8	22.8
40–49	22.6	20.1	21.3
50–59	18.1	14.4	16.3
60+	11.0	7.1	8.9
14+	**18.6**	**16.3**	**17.4**

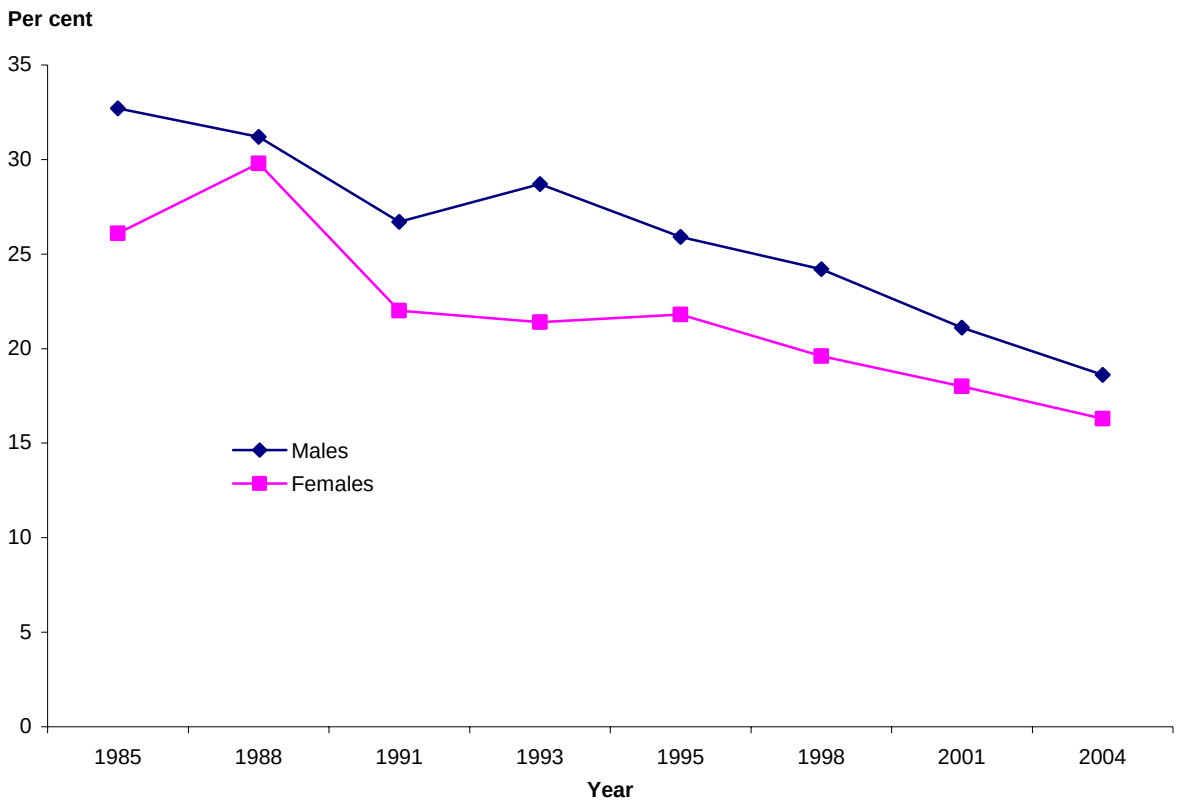
Source: National Drug Strategy Household Survey 2004.

Males were more likely to smoke than females (Table 3.3). Among people aged 14 years and over, 18.6% of males reported smoking daily, compared with 16.3% of females. A further 29.2% of males reported they were former smokers, compared with 23.6% of females.

The highest daily smoking rate occurred among people aged 20–29 years (23.5%). About 11% of young people (aged 14–19 years) were daily smokers. People aged 60 years or more recorded the lowest rates of daily smoking (8.9%).

Smoking rates in Australia have declined since the early 1970s. However, comparable prevalence data are only available from 1985 onwards. From 1985 to 2004, smoking rates declined by 35% among males and 31% among females (Figure 3.4).

Half of the people beginning kidney replacement therapy for end-stage kidney disease in 2003 had a history of smoking. Eleven per cent of all new patients were current smokers, and a further 39% were former smokers (Excell & McDonald 2005).



Note: Age-standardised to the Australian population at 30 June 2001.

Sources: National Campaign Against Drug Abuse Social Issues surveys 1985, 1988; National Campaign Against Drug Abuse Household surveys 1991, 1993; National Drug Strategy Household Surveys 1995, 1998, 2001, 2004.

Figure 3.4: Trends in smoking prevalence, people aged 14 years and over, 1985 to 2004

Insufficient physical activity

Insufficient physical activity is one of the most widespread of the established behavioural risk factors. Insufficient physical activity has been found to increase the risk of CKD, with the risk more than double among inactive people, compared to active people (Stengel et al. 2003). In addition, insufficient physical activity leads to greater risk of developing Type 2 diabetes and high blood pressure, two of the major causes of end-stage kidney disease.

The national physical activity guidelines for Australians recommend 'at least 30 minutes of moderate-intensity physical activity on most preferably all, days of the week' to achieve health benefits (DHAC 1999). This is generally interpreted as 30 minutes on at least five days of the week; a total of at least 150 minutes of moderate-intensity activity (for example brisk walking, swimming or cycling) each week. People who achieve this amount of activity in their leisure time are said to be 'sufficiently active' for health benefits.

In 2000, the National Physical Activity Survey found that more than half (54%) of Australians aged 18–75 years did not undertake physical activity at the levels recommended in the week before the survey. Around 15% of people were sedentary (did not do any moderate physical activity at all) in their leisure time during that week.

Between 1997 and 2000, the proportion of people who were not sufficiently active rose from 49% to 54% (Table 3.4). The increase occurred across all age groups with the exception of those aged 60–75 years (in whom activity levels remained fairly constant).

Table 3.4: Trends in insufficient physical activity, people aged 18–75 years, 1997 to 2000

Age group	1997	1999	2000
		(per cent)	
18–29	26.0	31.3	31.5
30–44	36.4	46.5	45.8
45–59	46.2	50.0	50.3
60–75	46.6	45.9	45.6
18–75	**49.4**	**55.3**	**54.2**

Notes

1. Based on self-reported data.
2. Insufficient physical activity is defined as achieving less than 150 minutes of leisure-time activity (including walking for transport) in the previous seven days.

Source: AIHW analysis of the National Physical Activity Surveys 1997, 1999, 2000.

Poor nutrition

Poor nutrition increases the risk of CKD through effects on other factors, such as blood glucose, blood pressure and body weight. The effect of nutrition can not be attributed to any one dietary component alone, but results from the combined effects of individual dietary factors and total energy intake.

The most recent information on dietary intake in Australia is from the 1995 ABS National Nutrition Survey. According to this survey, more than 50% of Australians did not have a healthy diet and did not achieve the dietary guidelines recommended by the National Health and Medical Research Council (NHMRC 1992). Among Australians aged 19 years and over, more than half consumed too much fat; two in three did not consume enough vegetables; four in five did not consume enough fruit; and more than half did not consume enough cereal foods (Table 3.5).

Table 3.5: Daily intake and comparison with recommended levels, adults aged 19 years and over, 1995

Food group	Average daily intake[(a)]	Recommended level[(a)]	Proportion of persons not meeting recommended level
Vegetables	259 g	300 g (minimum)	2 in 3
Fruit	144 g	300 g (minimum)	4 in 5
Fat	32.5%	30% (maximum)	2 in 3 males; 1 in 2 females
Saturated fat	12.5%	10% (maximum)	2 in 3

(a) For fat and saturated fat, 'daily intake' and 'recommended level' are reported as proportion of total energy intake.

Source: AIHW 2002a.

Obesity

People with excess weight, in particular obesity, are at high risk of developing CKD. The effect of excess weight is mainly mediated through high blood pressure and Type 2 diabetes.

The most recent estimates of levels of excess weight in the Australian population are from the 1999–00 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). The survey measured height, weight (which are used to calculate body mass index (BMI)) and waist circumference.

According to the AusDiab survey, 19% of males and 22% of females aged 25 years and over were obese, based on their BMI (see Box 3.3). An additional 48% of males and 30% of females were overweight but not obese. In both males and females, the proportion of people who were obese increased with age, peaking in the 55–64 years age group for both sexes.

Box 3.3: Body mass index

Body mass index, or BMI, is the most commonly used measure for classifying weight in population health surveys. It is calculated as a person's weight in kilograms divided by the square of their height in metres. In this report the following definitions are used:

Overweight	*BMI of 25 or more*
Overweight but not obese	*BMI of 25 to less than 30*
Obese	*BMI of 30 or more*

These definitions relate to adults (aged 18 years and over) only. A different classification, based on age and sex, is used in children and adolescents.

In Australian adults, obesity as measured by BMI has increased since the 1980s. The proportion of males aged 25–64 years who were obese increased from 9% in 1980 to 19% in 1995, before declining slightly to 16% in 1999–00. For females, the proportion obese increased from 8% to 19% between 1980 and 1995, but has since remained stable (Table 3.6).

Table 3.6: Trends in prevalence of obesity, people aged 25–64 years, 1980 to 1999–00

Year	Males	Females	Persons
		(per cent)	
1980	9.4	7.9	8.7
1983	8.9	10.4	9.7
1989	10.4	12.5	11.5
1995	19.6	19.2	19.4
1999–00	16.9	19.8	18.4

Notes
1. Age-standardised to the Australian population at 30 June 2001.
2. Urban areas only.
3. Based on BMI classification.

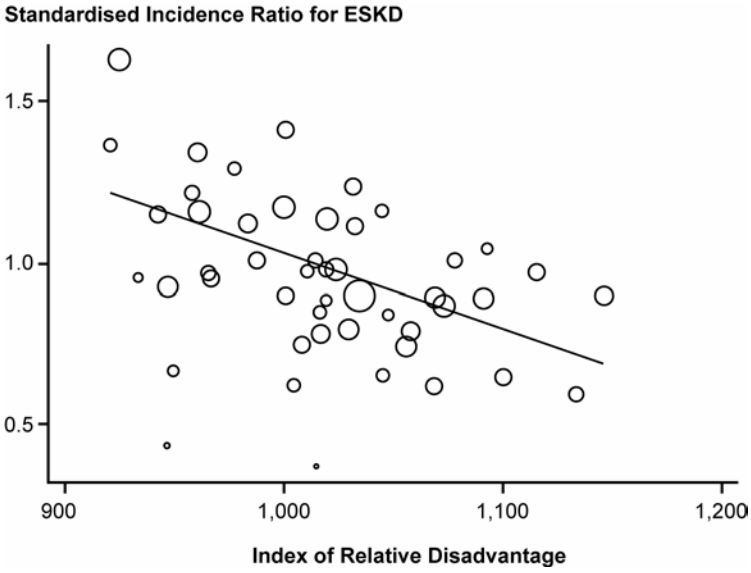
Source: AIHW analysis of the 1980, 1983 and 1989 Risk Factor Prevalence surveys, the 1995 National Nutrition Survey and the 1999–00 AusDiab study.

Other factors influencing chronic kidney disease

A number of socio-environmental and predisposing factors impact upon CKD. These include socioeconomic status, availability of health care, age, gender, ethnicity, family history and genetic factors. National information for most of these factors is lacking, however the issues of socioeconomic status and ageing are discussed below. These two factors are important as they contribute greatly to health inequalities and the number of people at risk of CKD, respectively.

Socioeconomic status

Lower socioeconomic status is associated with chronic kidney disease, particularly end-stage kidney disease, in Australia. Low socioeconomic status may influence the development of CKD and ESKD through its association with risk factors such as smoking, high blood pressure, diabetes and streptococcal skin or throat infections, as well as through reduced access to services for diagnosis and treatment (Chadban & Atkins 2005; Cass et al. 2002).



Notes

1. A lower score on the Index of Relative Disadvantage indicates greater disadvantage compared to areas with higher scores.
2. Circle size is proportional to area population.

Source: Cass et al. 2001. Reprinted with permission of the Australian and New Zealand Journal of Public Health.

Figure 3.5: Social disadvantage and variation in the incidence of treated end-stage kidney disease in Australian capital cities

Cass et al. (2001) have examined variation in the incidence of treated ESKD within Australian capital cities to explore the relationship between the incidence of treated ESKD and socioeconomic disadvantage. They found a significant correlation between the standardised incidence rate of treated ESKD and the Index of Relative Socioeconomic Disadvantage (Figure 3.5). The incidence rate was significantly higher in disadvantaged areas within each capital city, and the variation was up to threefold. In a separate study looking specifically at Indigenous Australians, Cass et al. (2002) found that the gradients in the incidence of treated ESKD with socioeconomic disadvantage were even steeper than in

the general population. The variation in the incidence of treated ESKD in this population was at least 30–fold.

Ageing

Ageing is accompanied by a substantial natural reduction in kidney function. Both the weight and volume of the kidneys may decrease (Mulder & Hillen 2001; McLachlan & Wasserman 1981). Some studies have also found that glomerular filtration rate (GFR) naturally declines with age after age 30; the GFR of people in their seventies is only one-half to two-thirds of that measured in young adults (Rowe et al. 1976). Although this level of kidney function is still sufficient under normal circumstances, these changes reduce the capacity of older people's kidneys to respond to physiological and pathological stresses. Therefore, older people are more vulnerable to kidney damage and CKD, especially those with other chronic illnesses, such as high blood pressure and diabetes.

Nearly 55% of AusDiab study participants aged 65 years and over had moderately or severely reduced kidney function (GFR <60 mL/min/1.73 m^2). Over 6% of people of this age had proteinuria and 5% had haematuria (Chadban et al. 2003). In comparison, only 0.01% of participants aged 25–44 years had moderately or severely reduced kidney function, 1% had proteinuria and 1% had haematuria.

The Australian population is ageing rapidly. According to the Australian Bureau of Statistics, there were an estimated 2.4 million Australians aged 65 years and over at 30 June 2001 (ABS 2002). This represents 22% growth since 1991, when there were 2.0 million Australians of this age. The ABS project that there will be more than 4.2 million people aged 65 years and over by 2021 (ABS 2000). This will increase the proportion of the population who are at high risk of chronic kidney disease.

References

ABS (Australian Bureau of Statistics) 2000. Population projections Australia 1999–2101. Cat. No. 3222.0. Canberra: ABS.

ABS 2002. Australian demographics statistics. Cat. No. 3101.0. Canberra: ABS.

AIHW (Australian Institute of Health and Welfare) 2002a. Chronic diseases and associated risk factors in Australia, 2001. AIHW Cat. No. PHE 33. Canberra: AIHW.

AIHW 2002b. Diabetes: Australian facts 2002. AIHW Cat. No. CVD 20. Canberra: AIHW (Diabetes Series No. 3).

AIHW 2004. Heart, stroke and vascular diseases—Australian facts 2004. AIHW Cat. No. CVD 27. Canberra: AIHW and National Heart Foundation of Australia (Cardiovascular Disease Series No. 22).

AIHW 2005. 2004 National drug strategy household survey: first results. AIHW Cat. No. PHE 57. Canberra: AIHW (Drug Statistics Series No. 13).

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

Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL & Jameson JL 2001. Harrison's principals of internal medicine. 15th ed. New York: McGraw-Hill.

Briganti EM, Branley P, Chadban SJ et al. 2002. Smoking is associated with renal impairment and proteinuria in the normal population: the AusDiab kidney study. American Journal of Kidney Diseases 40:704–12.

Briganti EM, McNeil J, Atkins R (eds) 2000. The epidemiology of diseases of the kidney and urinary tract: an Australian perspective. A report to the Board of the Australian Kidney Foundation. Melbourne: Monash University & Monash Medical Centre.

Carson PJ & Brewster DR 2003. Unique pattern of urinary tract calculi in Australian Aboriginal children. Journal of Paediatrics and Child Health 39:325–8.

Cass A, Cunningham J, Snelling P, Wang Z & Hoy W 2002. End-stage renal disease in Indigenous Australians: a disease of disadvantage. Ethnicity & Disease 12:373–8.

Cass A, Cunningham J, Wang Z & Hoy W 2001. Social disadvantage and variation in the incidence of end-stage renal disease in Australian capital cities. Australian and New Zealand Journal of Public Health 25(4):322–6.

Chadban SJ & Atkins RC 2005. Glomerulonephritis. Lancet 365:1797–806.

Chadban SJ, Briganti EM, Kerr PG et al. 2003. Prevalence of kidney damage in Australian adults: the AusDiab kidney study. Journal of the American Society of Nephrology 14 Suppl. 2:S131–8.

Chen J, Muntner P, Hamm LL et al. 2004. The metabolic syndrome and chronic kidney disease in US adults. Annals of Internal Medicine 140:167–74.

DCCT (Diabetes Control and Complications Trial Research Group) 1993. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. New England Journal of Medicine 329:977–98.

Dekker JM, Girman C, Rhodes T et al. 2005. Metabolic syndrome and 10-year cardiovascular disease risk in the Hoorn study. Circulation 112:666–73.

DHAC (Department of Health and Aged Care) 1999. National physical activity guidelines for Australians. Canberra: DHAC.

Dunstan D, Zimmet P, Welborn T et al. 2001. Diabesity and associated disorders in Australia, 2000: the accelerating epidemic. Final report of the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Melbourne: International Diabetes Institute.

Excell L & McDonald SP 2005. New patients commencing treatment in 2003. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry, 7–14.

Ford ES 2005. Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome. Diabetes Care 28:1769–78.

Francis RS & Tomson CR 2004. A GP guide to glomerulonephritis. Practitioner 248:848–55.

Goldfarb DS, Fischer ME, Keich Y & Goldberg J 2005. A twin study of genetic and dietary influences on nephrolithiasis: a report from the Vietnam Era Twin (VET) Registry. Kidney International 67:1053–61.

Guest C, O'Dea K, Hopper J, Navkervis A & Larkins R 1992. The prevalence of glucose intolerance in Aborigines and Europids of south-eastern Australia. Diabetes Research in Clinical Practice 15:227–35.

Haslett C, Chilvers ER, Hunter JAA & Boon NA (eds) 1999. Davidson's principles and practice of medicine. 18th ed. London: Churchill Livingstone.

IDF (International Diabetes Federation) 2005. The IDF consensus worldwide definition of the metabolic syndrome. Viewed 1 August 2005, <http://www.idf.org/home/index.cfm?node=1401>.

Isomaa B, Henricsson M, Almgren P, Tuomi T, Taskinen M-R & Groop L 2001. The metabolic syndrome influences the risk of chronic complications in patients with Type II diabetes. Diabetologia 44:1148–54.

Kurella M, Lo JC & Chertow GM 2005. Metabolic syndrome and the risk for chronic kidney disease among nondiabetic adults. Journal of the American Society of Nephrology 16:2134–40.

McLachlan MSF & Wasserman P 1981. Changes in size and distensibility of the aging kidney. The British Journal of Radiology 54:488–91.

Mulder WJ & Hillen HFP 2001. Renal function and renal disease in the elderly: part I. European Journal of Internal Medicine 12:86–97.

NHMRC (National Health and Medical Research Council) 1992. Dietary guidelines for Australians. Canberra: Australian Government Publishing Service.

Norden G & Nyberg G 1984. Smoking and diabetic nephropathy. Acta Medica Scandinavica 215:257–61.

Orth SR, Stockmann A, Conradt C et al. 1998. Smoking as a risk factor for end-stage renal failure in men with primary renal disease. Kidney International 54:926–31.

Rowe JW, Andres R, Tobin JD, Norris AH & Shock NW 1976. The effect of aging on creatinine clearance in men: a cross sectional and longitudinal study. Journal of Gerontology 31:155–63.

Sandler DP, Smith JC, Weinberg CR et al. 1989. Analgesic use and chronic renal disease. New England Journal of Medicine 320: 1238–43.

Stengel B, Couchoud C, Cenee S & Hemon D 2000. Age, blood pressure and smoking effects on chronic renal failure in primary glomerular nephropathies. Kidney International 57:2519–26.

Stengel B, Tarver-Carr ME, Powe NR, Eberhardt MS & Brancati FL 2003. Lifestyle factors, obesity and the risk of chronic kidney disease. Epidemiology 14(4):479–87.

Stewart JH, McCredie MR, Williams SM & McDonald SP 2004. Interpreting incidence trends for treated end-stage renal disease: implications for evaluating disease control in Australia. Nephrology 9:238–46.

Streeton CL, Hanna JN, Messer RD et al. 1995. An epidemic of acute post-streptococcal glomerulonephritis among Aboriginal children. Journal of Paediatrics and Child Health 74:63–73.

Taylor EN, Stampfer MJ & Curhan GC 2005. Obesity, weight gain, and the risk of kidney stones. Journal of the American Medical Association 293:455–62.

Thorn LM, Forsblom C, Fagerudd J et al. 2005. Metabolic syndrome in Type 1 diabetes. Diabetes Care 28:2019–24.

Tylicki L, Rutkowski B & Horl WH 2002. Multifactorial determination of hypertensive nephroangiosclerosis. Kidney & Blood Pressure Research 25:341–53.

White AV, Hoy WE & McCredie DA 2001. Childhood post-streptococcal glomerulonephritis as a risk factor for chronic renal disease in later life. Medical Journal of Australia 174:492–6.

WHO-ISH (World Health Organization International Society for Hypertension) 1999. 1999 guidelines for the management of hypertension. CVD Prevention 2(2):76–111.

Zimmet PZ, Alberti KGMM & Shaw JE 2005. Mainstreaming the metabolic syndrome: a definitive definition. Medical Journal of Australia 183(4):174–5.

4 Causes of treated end-stage kidney disease

Introduction

As the most severe level of kidney function reduction, end-stage kidney disease (ESKD) could be seen as the last opportunity for preventive intervention in chronic kidney disease (CKD). In Chapter 2 it was shown that dialysis for ESKD was by far the most common cause of hospitalisation for people with CKD, had profound impacts on quality of life, and accounted for a large proportion of expenditure on CKD. It is therefore important to understand and monitor trends in the causes of treated ESKD in order to develop and evaluate policies and strategies to prevent its development and reduce the demand for kidney replacement therapy.

The natural history of CKD, its causes and the rate of progression to each stage are not clearly understood. Therefore, the proportion of people with CKD who will develop ESKD, and which people are at greatest risk, are unclear (White et al. 2005). In general, CKD can progress at different rates, depending on the underlying cause of the disease. In some cases, progression of kidney damage is relatively swift and almost always results in end-stage kidney disease. In other cases, progression is relatively slow and can be slowed further through appropriate management. Therefore, the distribution of causes for ESKD will be very different to that for CKD. In most cases of CKD, early detection and appropriate management may prevent progression to end-stage kidney disease.

There is no information available on the incidence or prevalence of different causes of CKD in Australia. However, the ANZDATA Registry collects data on the causes of treated ESKD. Using the data provided by the ANZDATA Registry, this chapter provides detailed information on the different causes of treated ESKD in Australia. The chapter begins with a summary of the different causes, looking at their contribution to the incidence and prevalence of treated ESKD in 2003 and comparing trends since 1981. It then provides data on the incidence and prevalence of treated ESKD due to each major cause, and describes recent trends.

A few causes account for the majority of cases of treated ESKD in Australia. These include:

- glomerulonephritis;
- diabetic nephropathy;
- hypertensive kidney disease;
- analgesic nephropathy;
- reflux nephropathy; and
- polycystic kidney diseases.

Although there are other causes of ESKD, such as kidney stones and cancers, they account for relatively few cases and are not covered in detail in this report.

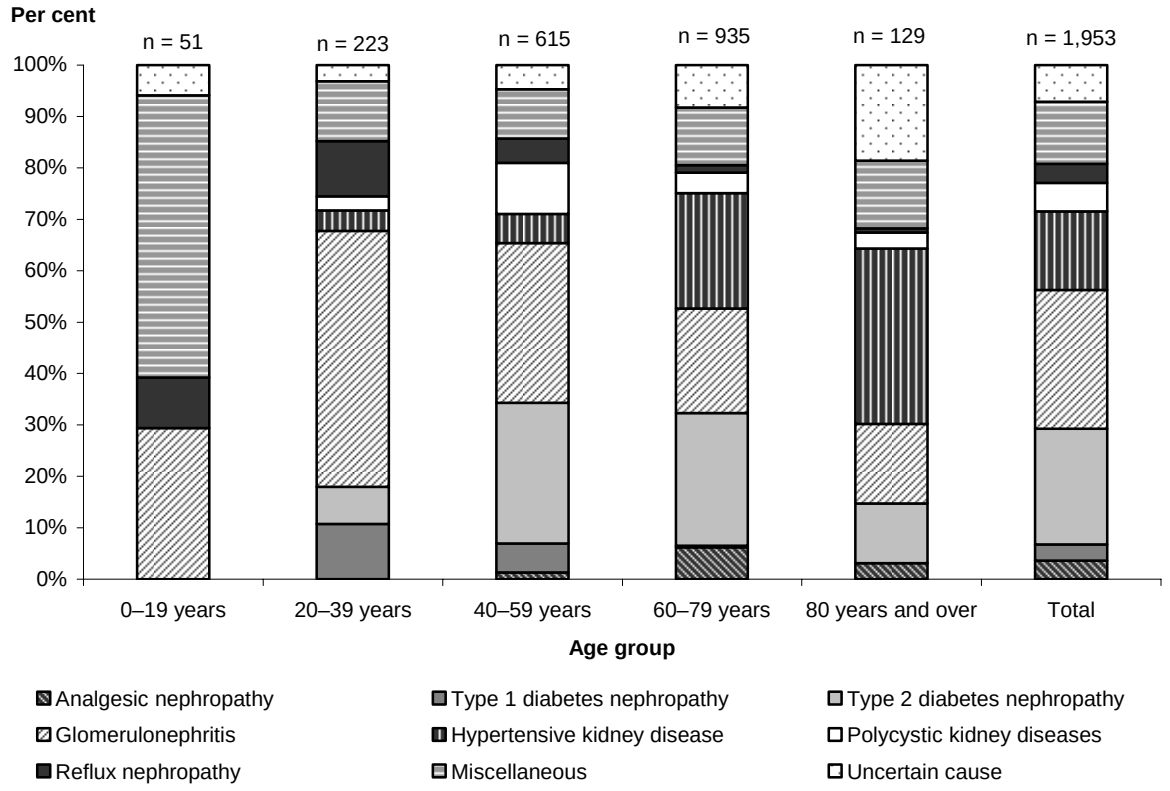
Major causes of treated end-stage kidney disease

The proportion of new and existing cases of treated ESKD attributed to different causes has changed over time, and differs across age groups and between males and females. Looking at the variation in treated ESKD from different causes across the population and how this changes over time can provide information to help target and refine policies and interventions, and to evaluate the success of prevention programs.

Incidence of treated end-stage kidney disease by cause

The major causes of new cases of treated ESKD in 2003 were glomerulonephritis (27%), diabetic nephropathy (26%) and hypertension (15%). These were followed by polycystic kidney disease (5%), analgesic nephropathy (4%) and reflux nephropathy (4%). Miscellaneous causes such as interstitial nephritis (an inflammation of the kidneys), cancers and congenital diseases accounted for a further 12%. There were about 7% of patients whose diagnoses were uncertain.

The causes of treated ESKD among new patients in 2003 varied with age (Figure 4.1). Glomerulonephritis was the major cause of new treated ESKD cases among people aged less than 40 years, accounting for 29% of new cases in 0–19-year-olds and half of all new cases in people aged 20–39 years. In people aged 40–79 years the most common cause was diabetic nephropathy, accounting for around 30% of new cases. One-third of new cases in people aged 80 years and over were due to hypertensive kidney disease.

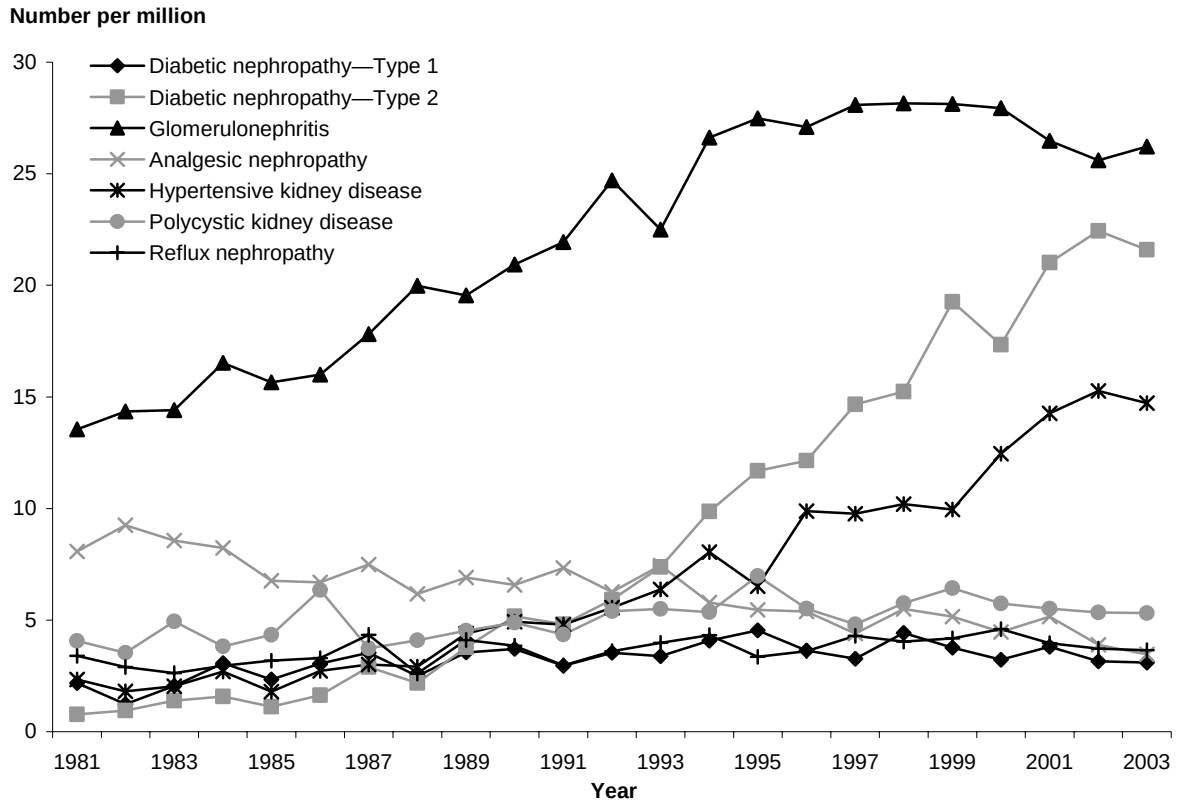


Source: AIHW analysis of ANZDATA Registry data.

Figure 4.1: Causes of new cases of treated end-stage kidney disease, by age group, 2003

Trends in incidence

Between 1981 and 2003, the incidence of treated ESKD increased for all causes except analgesic nephropathy (Figure 4.2). The incidence of treated ESKD caused by glomerulonephritis rose gradually until 1995 but appears to have stabilised and may be decreasing slightly. There have been large and rapid increases in the incidence of treated ESKD caused by hypertensive kidney disease and diabetic nephropathy in Type 2 diabetes since the early 1990s. The incidence of treated ESKD caused by diabetic nephropathy in Type 1 diabetes, reflux nephropathy and polycystic kidney disease have risen very slowly but gradually over the period.



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.2: Incidence of treated end-stage kidney disease, selected causes, 1981 to 2003

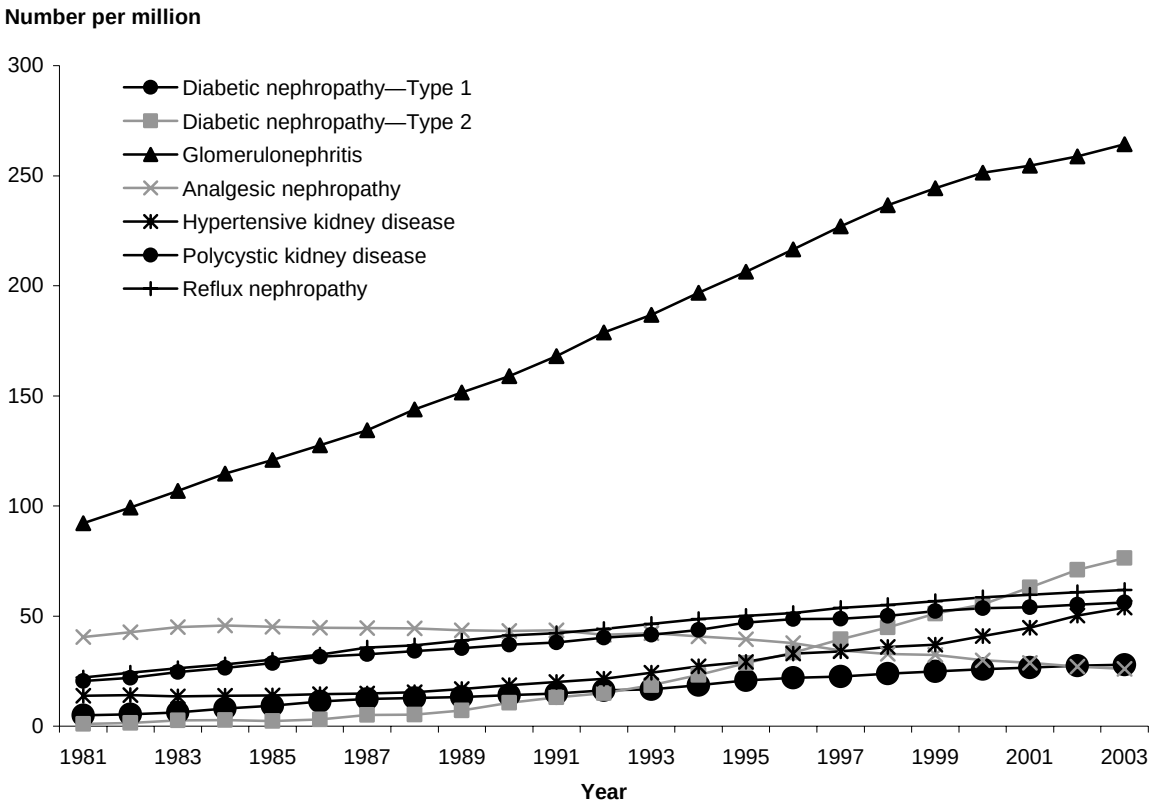
The increased incidence of treated ESKD in recent years can be attributed to a number of factors. The rising prevalence of diabetes and high prevalence of hypertension in the past may have led to increased incidence of CKD. At the same time, the significant reduction in mortality from cardiovascular disease may have resulted in a greater number of people surviving long enough to reach end-stage kidney disease. Also changing acceptance policies for older patients have led to increased numbers of older people being accepted for kidney replacement therapy. All these factors also interact with other factors such as demographic changes (ageing of the population and immigration of people at higher risk) and changes in coding and diagnostic criteria for ESKD (McDonald et al. 2005).

Prevalence of treated end-stage kidney disease by cause

The distribution of causes among the whole population who were receiving kidney replacement therapy in 2003 was similar to that of new patients. Of 13,625 patients, 39% had treated ESKD caused by glomerulonephritis, while diabetes and hypertension together accounted for 24% (Table 4.1).

Trends in prevalence

Compared with other causes, the prevalence of treated ESKD caused by glomerulonephritis is high. Although the incidence of ESKD due to this disease has been stable in recent years, the prevalence is still increasing slowly due to large numbers of ongoing cases. This reflects the high incidence rate of this disease in previous years and increasing survival of treated patients. In contrast, the total number of cases attributed to hypertensive kidney disease and nephropathy in Type 2 diabetes is lower than the number attributed to glomerulonephritis, but both prevalence and incidence of treated ESKD due to these causes have increased (Figure 4.3).



Note: Rates have been age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.3: Prevalence of treated end-stage kidney disease, selected causes, 1981 to 2003.

Table 4.1: Causes of end-stage kidney disease among all treated end-stage kidney disease patients, by age group, 2003

Cause	0–19 years	20–39 years	40–59 years	60–79 years	80 years and over	All ages
	Number of cases (per cent of age group)					
Analgesic nephropathy	0 (0%)	0 (0%)	38 (1%)	446 (9%)	41 (7%)	525 (4%)
Diabetic nephropathy	0 (0%)	208 (9%)	978 (18%)	875 (17%)	53 (10%)	2,114 (16%)
Glomerulonephritis	66 (23%)	1,087 (48%)	2,443 (45%)	1,619 (31%)	113 (20%)	5,328 (39%)
Hypertensive kidney disease	0 (0%)	38 (2%)	184 (3%)	685 (13%)	188 (34%)	1,095 (8%)
Polycystic kidney disease	0 (0%)	28 (1%)	544 (10%)	546 (11%)	28 (5%)	1,146 (8%)
Reflux nephropathy	29 (10%)	422 (19%)	591 (11%)	191 (4%)	4 (1%)	1,237 (9%)
Other causes	183 (64%)	395 (17%)	402 (7%)	429 (8%)	55 (10%)	1,464 (11%)
Uncertain cause	7 (2%)	81 (4%)	200 (4%)	357 (7%)	71 (13%)	716 (5%)
Total prevalent cases	**285**	**2,259**	**5,380**	**5,148**	**553**	**13,625**

Source: AIHW analysis of ANZDATA Registry data.

Glomerulonephritis

Incidence of treated end-stage kidney disease caused by glomerulonephritis

Glomerulonephritis accounted for 1,548 new patients (27%) who started kidney replacement therapy between 2001 and 2003, an incidence rate of 26 per million population. The incidence was higher among males than females and peaked among those aged 65–74 years (Table 4.2).

Table 4.2: Incidence of treated end-stage kidney disease caused by glomerulonephritis, 2001–2003

	0–24 years	25–44 years	45–54 years	55–64 years	65–74 years	75–84 years	85 years and over
	Number per million population						
Males	5	29	47	62	99	96	15
Females	5	18	27	33	47	40	7

Source: AIHW analysis of ANZDATA Registry data.

Trends in incidence

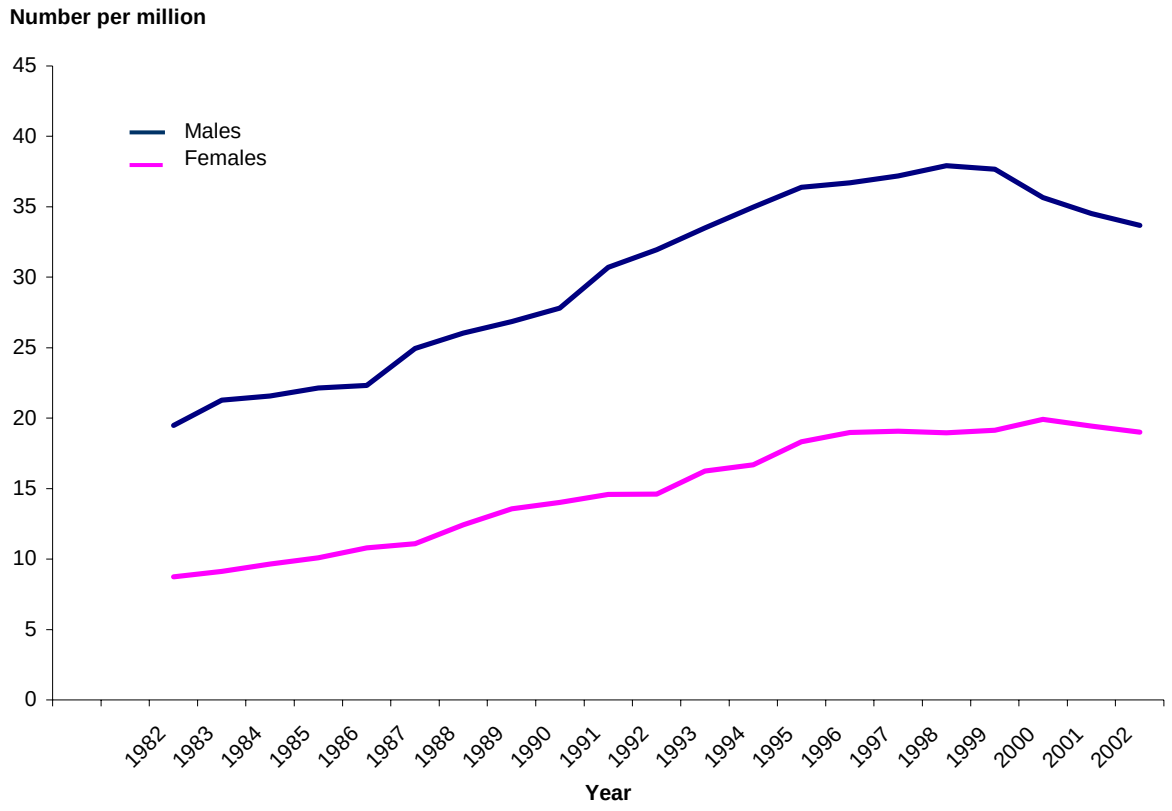
For males, the age-standardised incidence rate of treated ESKD due to glomerulonephritis increased substantially from 19 cases per million population in 1981 to 40 per million in 1999, then declined to 34 per million in 2003 (Figure 4.4). For females, the incidence rate increased from 8 cases per million population in 1982 to 20 per million in 1997, and has remained relatively steady around this rate since then.

Prevalence of treated end-stage kidney disease caused by glomerulonephritis

There were 5,328 people receiving kidney replacement therapy in 2003 for treated ESKD due to glomerulonephritis, an age-standardised prevalence rate of 264 per million population. There were almost twice as many males as females among these people, reflecting the much higher incidence rate among males.

Trends in prevalence

Although declining in incidence, the prevalence of treated ESKD due to glomerulonephritis is relatively high and still increasing slowly due to large numbers of ongoing cases. As with incidence, the prevalence rate in males is almost twice that in females (Figure 4.5).

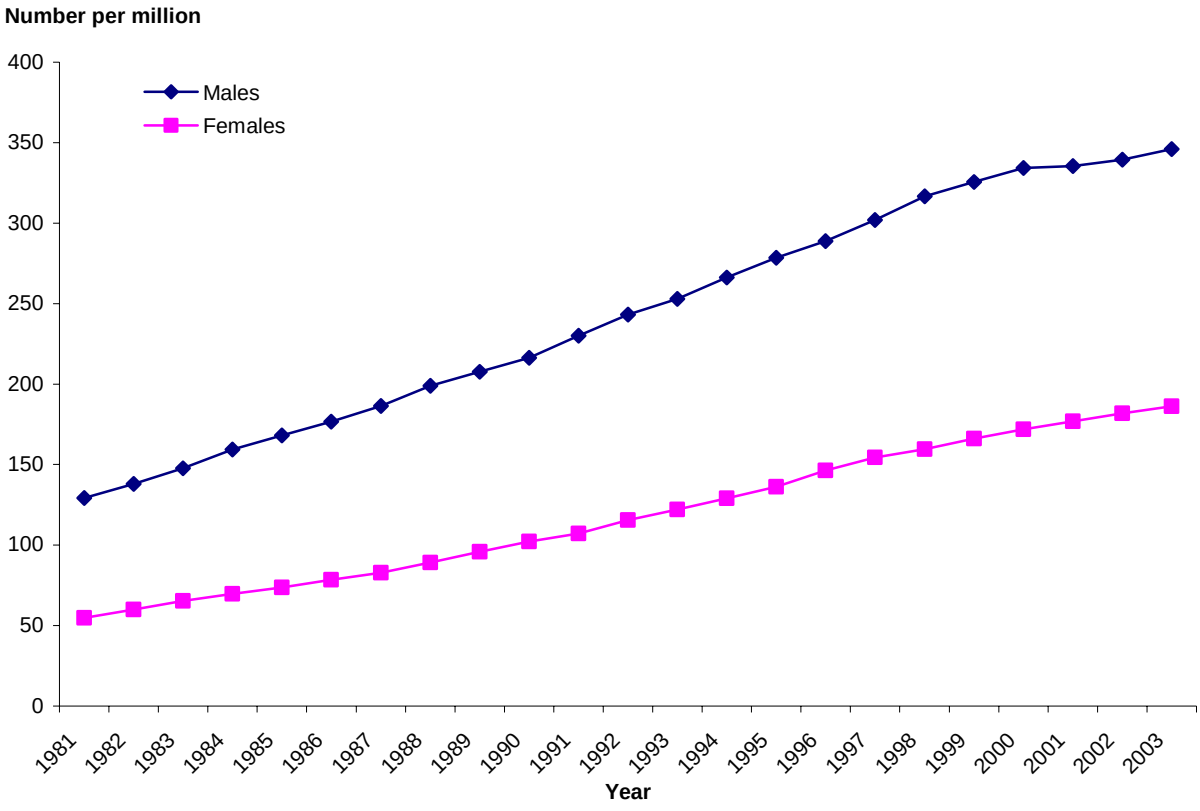


Notes

1. Age-standardised to the Australian population at 30 June 2001.
2. Data are three-year moving averages.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.4: Trends in the incidence of treated end-stage kidney disease due to glomerulonephritis, 1982 to 2002



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.5: Trends in the prevalence of treated end-stage kidney disease due to glomerulonephritis, 1981 to 2003

As there is no information available on the incidence or prevalence of glomerulonephritis in the general population, it is not certain if increasing incidence of this disease has contributed to the rising numbers of people with treated ESKD due to the disease. However, given the high incidence among patients in the older age groups (Table 4.3), the increased acceptance of older patients into the kidney replacement therapy program and improved survival rates are likely to have been factors in the rising prevalence rates of treated ESKD due to glomerulonephritis.

Diabetic nephropathy

Incidence of treated end-stage kidney disease caused by diabetic nephropathy

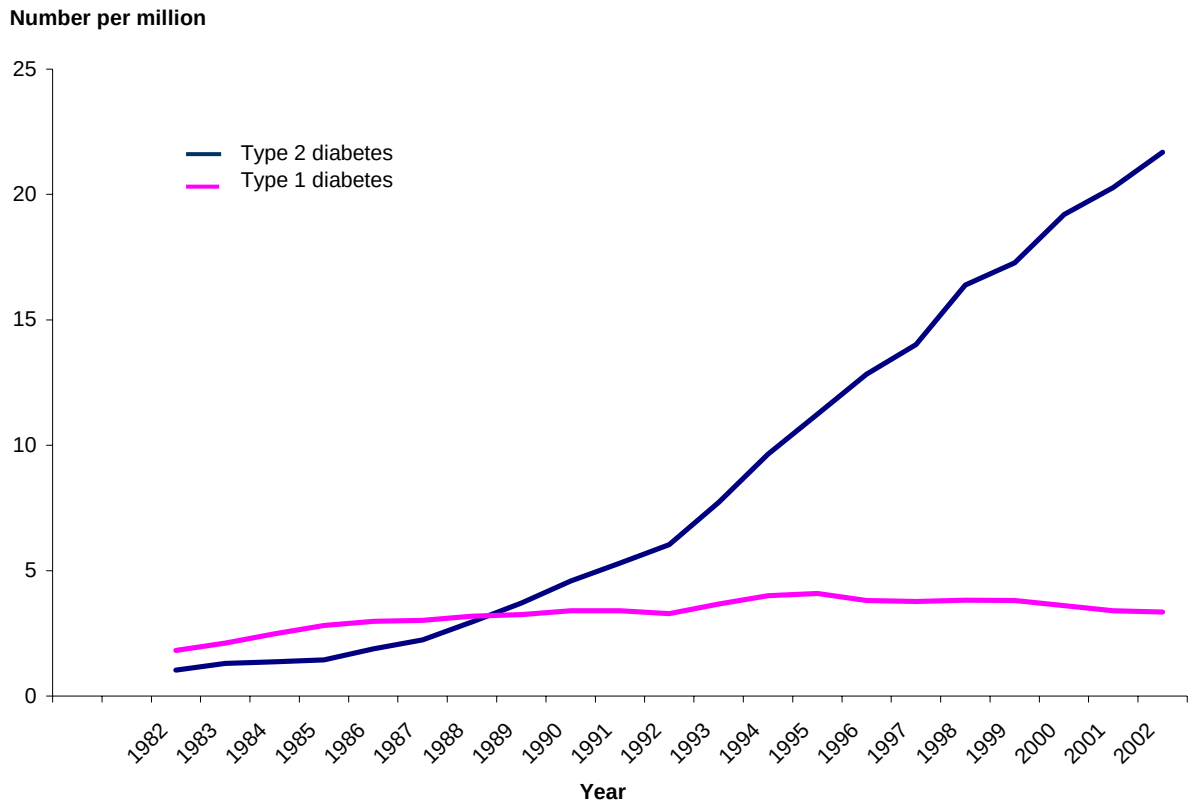
In line with the rising rate of diabetes, growing numbers of treated ESKD cases in Australia were caused by this illness. Between 2001 and 2003, 1,492 new treated ESKD cases (26%) registered in the ANZDATA Registry were attributed to diabetic nephropathy. The incidence of treated ESKD caused by nephropathy in Type 2 diabetes (1,294 cases, 22 per million population) was much higher than for Type 1 diabetes (198 cases, 3 per million population), reflecting the much higher rates of Type 2 diabetes in the Australian population.

Table 4.3: Incidence of treated end-stage kidney disease caused by diabetic nephropathy, 2001–2003

		0–24 years	25–44 years	45–54 years	55–64 years	65–74 years	75–84 years	85 years and over
		Number per million population						
Type 1 diabetic nephropathy	**Males**	0	7	8	4	1	0	0
	Females	0	7	4	2	0	1	0
Type 2 diabetic nephropathy	**Males**	0	6	40	85	121	72	8
	Females	0	5	26	53	75	39	2

Source: AIHW analysis of ANZDATA Registry data.

Consistent with the generally much younger age of onset of Type 1 diabetes, treated ESKD due to nephropathy in Type 1 diabetes tended to occur at younger ages compared with Type 2 diabetes. In 2001–03, the incidence peaked at 25–54 years of age for people with Type 1 diabetes and at 65–74 years of age for people with Type 2. For both types of diabetes the incidence of treated ESKD caused by nephropathy was higher among males than females (Table 4.3).



Notes

1. Age-standardised to the Australian population at 30 June 2001.
2. Data are three-year moving averages.

Source: AIHW analysis of ANZDATA Registry data.

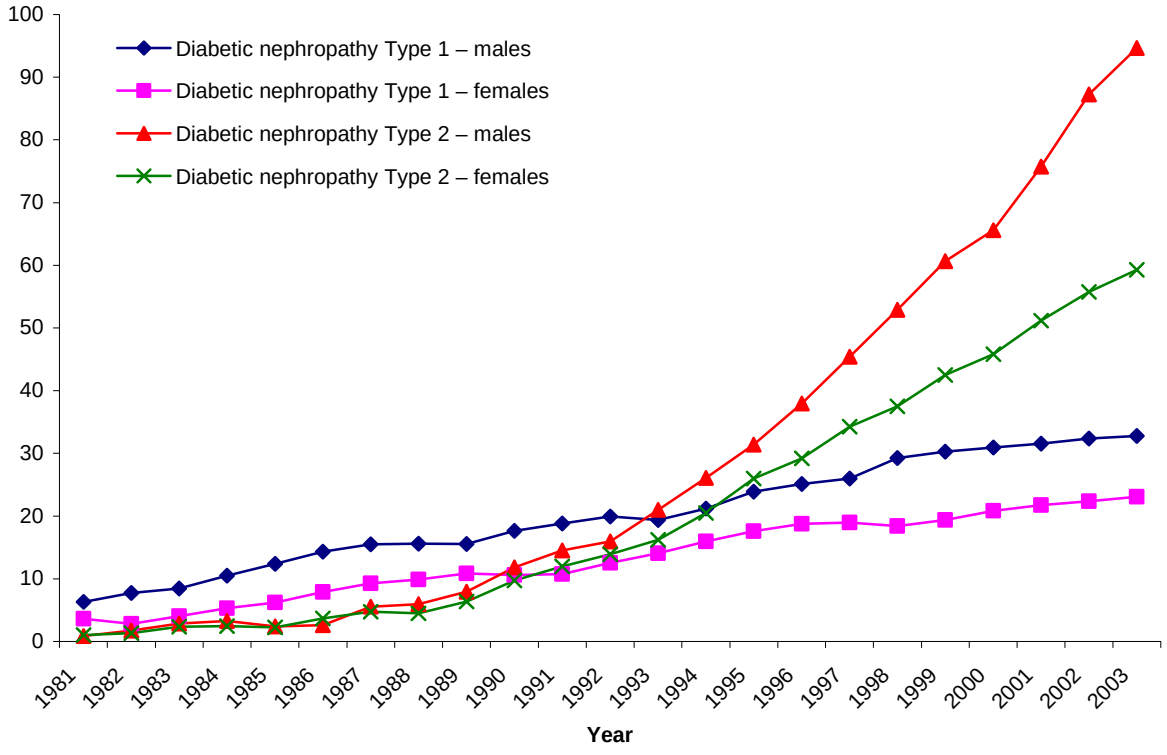
Figure 4.6: Trends in the incidence of treated end-stage kidney disease due to diabetic nephropathy, 1982 to 2002

Trends in incidence

From 1982 to 2002, the age-standardised incidence rate of treated ESKD caused by diabetic nephropathy in people with Type 1 diabetes increased slightly from 2 to 3 cases per million population. In the same period, the incidence rate in people with Type 2 diabetes increased from one case per million population to 22 cases per million population (Figure 4.6).

Prevalence of treated end-stage kidney disease caused by diabetic nephropathy

In 2003, 2,114 people (104 per million population) in the Australian kidney replacement therapy program had ESKD due to diabetic nephropathy. Among these, 557 (26%) had Type 1 diabetes and 1,556 (74%) had Type 2 diabetes. The prevalence rates among males were higher than among females for both types: 33 and 23 per million population respectively for Type 1 diabetes, and 95 and 59 per million population respectively for Type 2 diabetes. The prevalence rates for both types varied with age but, as with incidence, people with Type 1 diabetes tended to be younger. The prevalence was highest in the 35–44 years age group for people with Type 1 diabetes and the 65–74 years age group for people with Type 2 diabetes.



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.7: Trends in the prevalence of treated end-stage kidney disease due to diabetic nephropathy, 1981 to 2003

Trends in prevalence

Although the incidence of treated ESKD caused by diabetic nephropathy in people with Type 1 diabetes has increased only slightly since 1981, its prevalence has increased fivefold, from 6 cases to 33 cases per million population for males and from 4 to 23 cases per million population for females (Figure 4.7). This is mostly attributed to improved treatment keeping patients alive.

In the same period, the prevalence rate of treated ESKD caused by diabetic nephropathy in people with Type 2 diabetes increased sharply. From 1981 to 2003, it increased from 1 case per million to 95 cases per million for males and from 1 case per million to 59 cases per million for females (Figure 4.7). Improvements in treatment, the increasing acceptance of older patients into the kidney replacement therapy program and the rising prevalence of Type 2 diabetes are all believed to have contributed to this increase.

Hypertensive kidney disease

Incidence of treated end-stage kidney disease caused by hypertensive kidney disease

Hypertensive kidney disease accounted for 878 new cases of treated ESKD (15%) between 2001 and 2003, an age-standardised incidence rate of 15 per million population. The incidence rate was higher among males (572 cases, 21 per million population) than among females (306 cases, 10 per million population). The incidence of treated ESKD due to hypertensive kidney disease increased sharply with age, being highest in the 75–84 years age group for both sexes (Table 4.4).

Table 4.4: Incidence of treated end-stage kidney disease caused by hypertensive kidney disease, 2001–2003

	0–24 years	25–44 years	45–54 years	55–64 years	65–74 years	75–84 years	85 years and over
				Number per million population			
Males	0	2	8	27	101	209	54
Females	0	1	4	14	53	82	14

Source: AIHW analysis of ANZDATA Registry data.

Trends in incidence

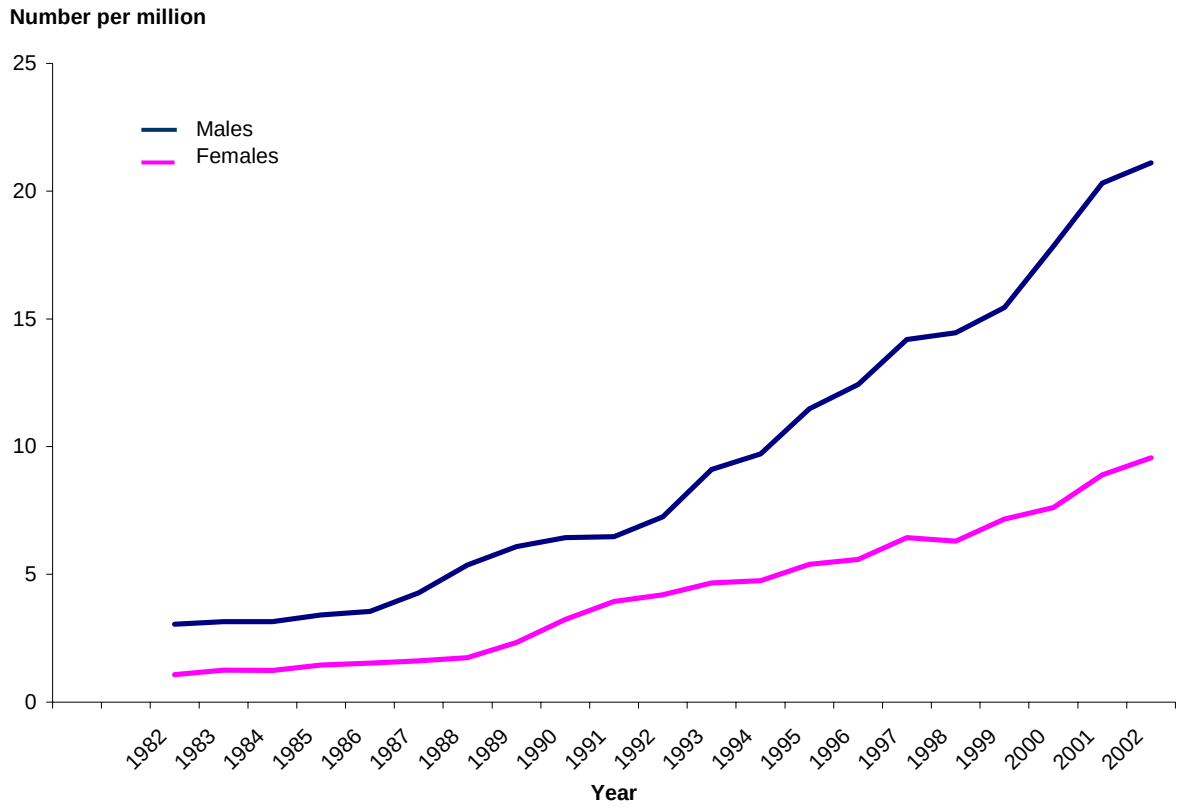
Between 1982 and 2002, the age-standardised incidence rate of treated ESKD due to hypertensive kidney disease increased by five times among males (from 4 to 20 cases per million population), and by more than ten times among females (from 1 to 11 cases per million population) (Figure 4.8).

Prevalence of treated end-stage kidney disease due to hypertensive kidney disease

In 2003, there were 1,095 people receiving treatment for ESKD due to hypertensive kidney disease. About twice as many males (714 cases, 76 per million population) as females (381 cases, 35 per million population) were receiving treatment. The highest prevalence occurred among those aged 75–84 years.

Trends in prevalence

In line with increased incidence of treated ESKD caused by hypertensive kidney disease, its prevalence rate also increased sharply between 1981 and 2003, from 14 to 54 cases per million population. Most of this increase has occurred over the past decade (Figure 4.9).



Notes

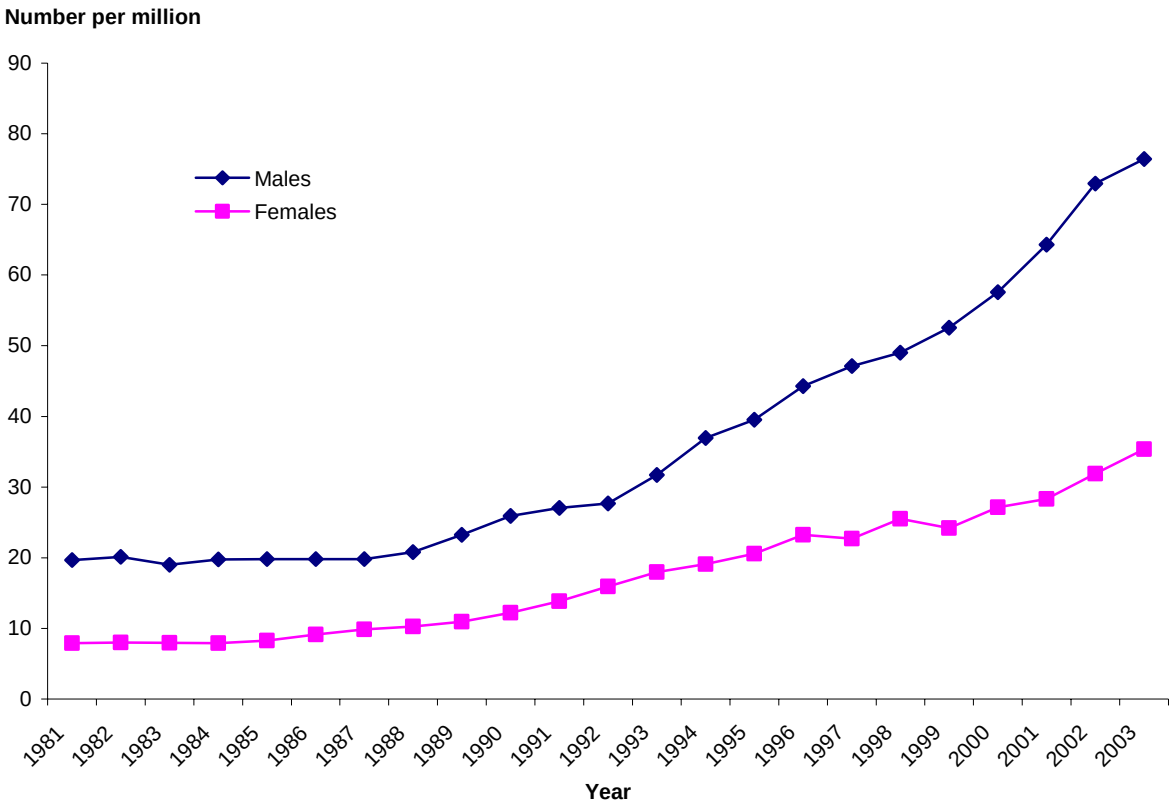
1. Age-standardised to the Australian population at 30 June 2001.
2. Data are three-year moving averages.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.8: Trends in the incidence of treated end-stage kidney disease due to hypertensive kidney disease, 1982 to 2002

There is an apparent incongruence between the decreasing prevalence of hypertension (Chapter 3, Figure 3.2) and the increasing incidence and prevalence of treated ESKD due to hypertensive kidney disease (Figures 4.9 and 4.10). The reasons for this are not clear, but there are several possible explanations. First, there is considerable time from the onset of hypertension to the development of hypertensive kidney complications. That is, the group of people who had hypertension 20 years ago are now suffering kidney complications of hypertension. It is possible that the decline in the prevalence of high blood pressure may

result in reduced incidence of treated ESKD due to hypertensive kidney disease in the future. Second, improvements in the management of cardiovascular diseases, especially improvements in intensive care, allow cardiovascular patients with hypertension to survive long enough to develop kidney complications. Third, the diagnosis of hypertensive kidney disease is usually confirmed through a kidney biopsy, but these are not performed in all cases. Therefore some misclassification is possible. Finally, people at older ages are now being accepted onto the kidney replacement therapy program. As the prevalence of hypertension increases with age, it might be expected that the number of patients with hypertensive kidney disease on the kidney replacement therapy program would increase. A combination of these factors may have contributed to the rising incidence and prevalence rates of treated ESKD due to hypertensive kidney disease.



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.9: Trends in the prevalence of treated end-stage kidney disease due to hypertensive kidney disease, 1981 to 2003

Analgesic nephropathy

Incidence of treated end-stage kidney disease caused by analgesic nephropathy

Between 2001 and 2003, 247 new treated ESKD cases (4%) in Australia were attributed to analgesic nephropathy, an incidence rate of 4 cases per million population. The incidence

was significantly higher among females (7 cases per million population) than males (1 case per million population). All cases over this period occurred among people aged 40 years and over, with the incidence rates being considerably higher among those aged 65–84 years (Table 4.5).

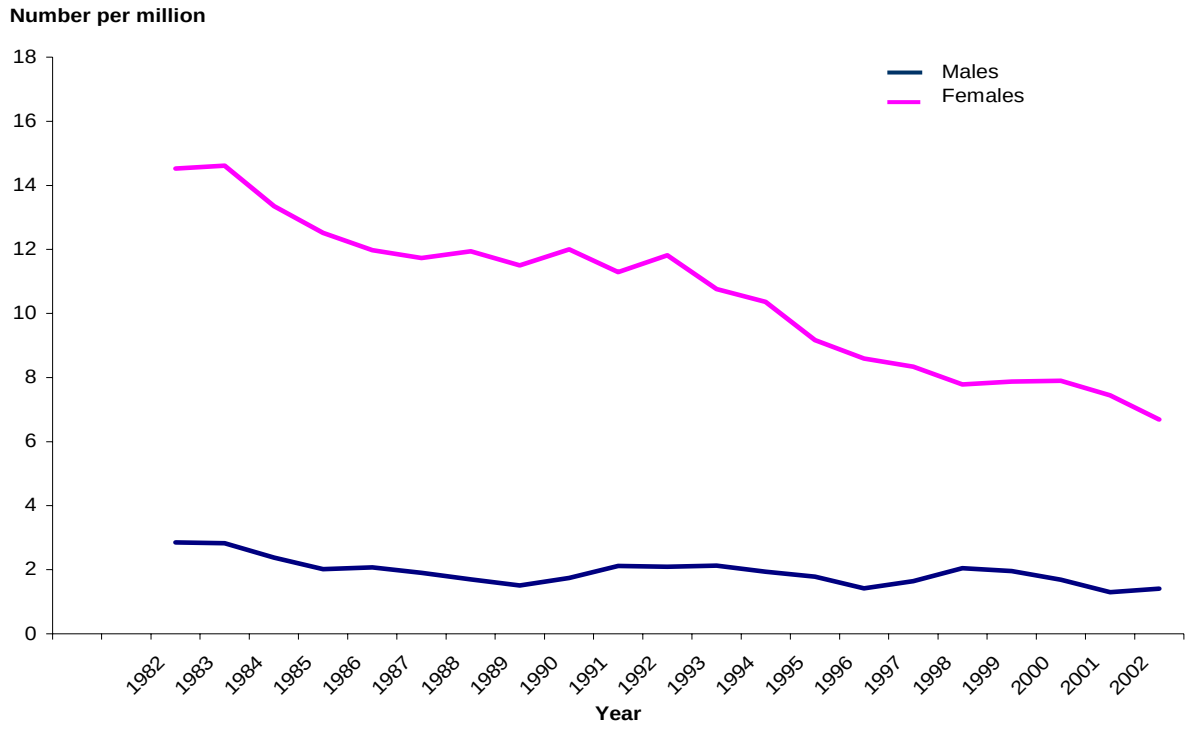
Table 4.5: Incidence of treated end-stage kidney disease caused by analgesic nephropathy, 2001–2003

	0–24 years	25–44 years	45–54 years	55–64 years	65–74 years	75–84 years	85 years and over
	Number per million population						
Males	0	0	0	1	10	11	0
Females	0	0	1	15	50	36	2

Source: AIHW analysis of ANZDATA Registry data.

Trends in incidence

The age-standardised incidence rate among females decreased by more than half over the last 20 years, from 14 cases per million population in 1982 to 6 cases per million population in 2002 (Figure 4.10). The incidence rate also decreased for males over this period, from 2 to 1 cases per million population. The decrease in incidence is attributed to the tighter controls on access to analgesics brought about by Commonwealth legislation passed in 1979. This is discussed in more detail in Chapter 5 of this report.



Notes

1. Age-standardised to the Australian population at 30 June 2001.
2. Data are three-year moving averages.

Source: AIHW analysis of ANZDATA Registry data.

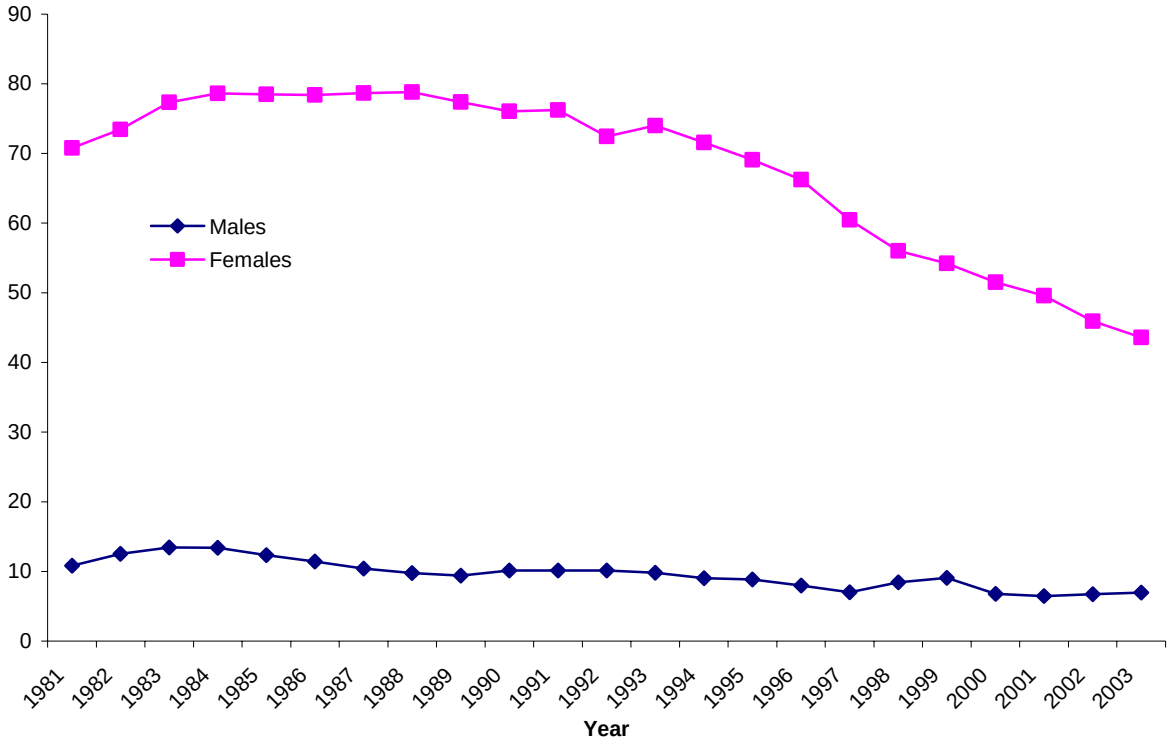
Figure 4.10: Trends in the incidence of treated end-stage kidney disease due to analgesic nephropathy, 1982 to 2002

Prevalence of treated end-stage kidney disease caused by analgesic nephropathy

There were 525 patients receiving kidney replacement therapy for ESKD due to analgesic nephropathy in 2003. The majority of these were females, with an age-standardised prevalence rate of 44 per million population compared with 7 per million population for males. All patients were 40 years of age or more.

Trends in prevalence

In line with the drop in incidence rates over the past two decades, the prevalence of treated ESKD due to analgesic nephropathy has also decreased (Figure 4.11). This decrease is apparent mainly among females, who account for the majority of cases. After a relatively stable period at around 78 per million population during the 1980s, the prevalence rate among females dropped steadily to 44 per million population in 2003. The prevalence rate among males also decreased slightly to 7 per million in 2003 after peaking at 13 per million in 1983.



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.11: Trends in the prevalence of treated end-stage kidney disease due to analgesic nephropathy, 1981 to 2003

Reflux nephropathy

Incidence of treated end-stage kidney disease caused by reflux nephropathy

Between 2001 and 2003, 223 new treated ESKD cases (4%) were caused by reflux nephropathy, at an age-standardised rate of 4 cases per million population. The age distribution of these new cases differed between males and females (Table 4.6). For males, the new cases occurred most frequently in younger adults, with the highest incidence rate in the 25–44 years age group. For females the incidence rate peaked in the 55–64 years age group. The reasons for the age difference between the sexes are not clear.

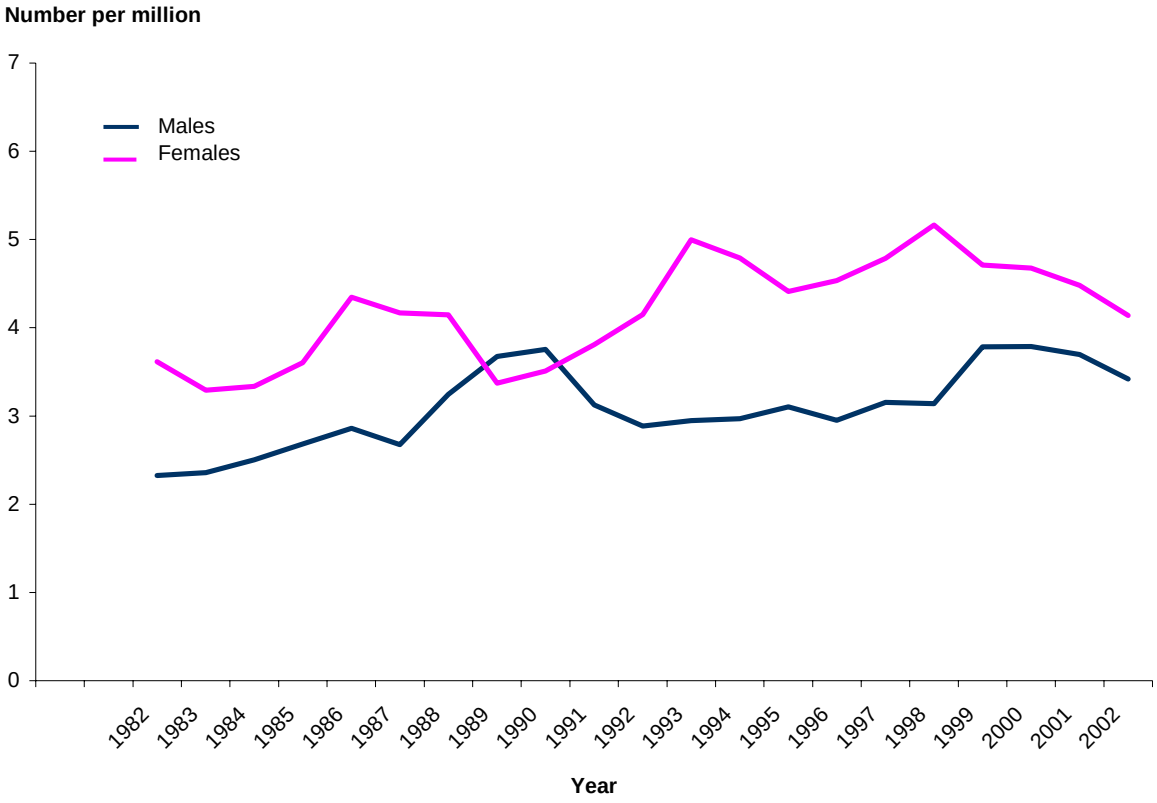
Table 4.6: Incidence of treated end-stage kidney disease caused by reflux nephropathy, 2001–2003

	0–24 years	25–44 years	45–54 years	55–64 years	65–74 years	75–84 years	85 years and over
				Number per million population			
Males	1	6	4	2	4	2	0
Females	1	5	6	7	5	3	0

Source: AIHW analysis of ANZDATA Registry data.

Trends in incidence

The age-standardised incidence rate increased slightly in both sexes between 1982 and 2002 (Figure 4.12). It increased from 2 to 4 cases per million for males and from around 3.5 to 4 cases per million for females. This may be partly due to improved diagnostic techniques (such as using ultrasound rather than X-ray) increasing the number of cases where reflux is able to be detected.



Notes

1. Age-standardised to the Australian population at 30 June 2001.
2. Data are three-year moving averages.

Source: AIHW analysis of ANZDATA Registry data.

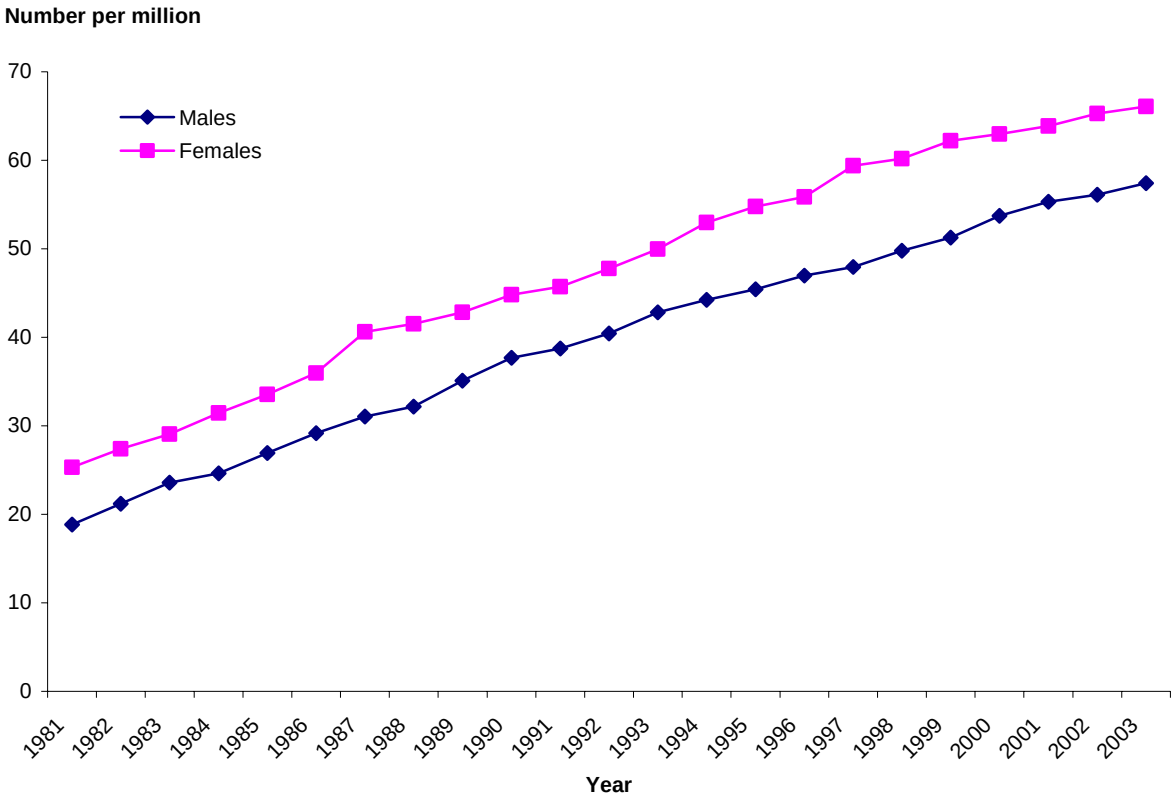
Figure 4.12: Trends in the incidence of treated end-stage kidney disease due to reflux nephropathy, 1982 to 2002

Prevalence of treated end-stage kidney disease caused by reflux nephropathy

There were 1,237 patients in the kidney replacement therapy program in 2003 who had treated ESKD due to reflux nephropathy, an age-standardised prevalence rate of 62 per million population. The prevalence of this condition was slightly higher among females than males, with 66 cases per million and 57 cases per million population, respectively. Prevalence was highest among males aged 35–44 years and females aged 45–54 years.

Trends in prevalence

The trends in prevalence have been similar between the sexes (Figure 4.13). From 1981 to 2003, the prevalence rate increased from 19 to 57 cases per million population for males and from 25 to 66 cases per million population for females.



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.13: Trends in the prevalence of treated end-stage kidney disease due to reflux nephropathy, 1981 to 2003

Polycystic kidney diseases

Incidence of treated end-stage kidney disease caused by polycystic kidney diseases

Between 2001 and 2003, 321 new treated ESKD cases (6%) were caused by PKD, at an incidence rate of 5 cases per million population. Most of these cases occurred among people aged 45 years and over (Table 4.7), and the condition was more common among males than females (incidence rates of 7 per million population and 4 per million population respectively).

Trends in incidence

Between 1981 and 2003, the incidence of treated ESKD due to PKD slowly but steadily increased in males (Figure 4.14). No clear trend was apparent for females.

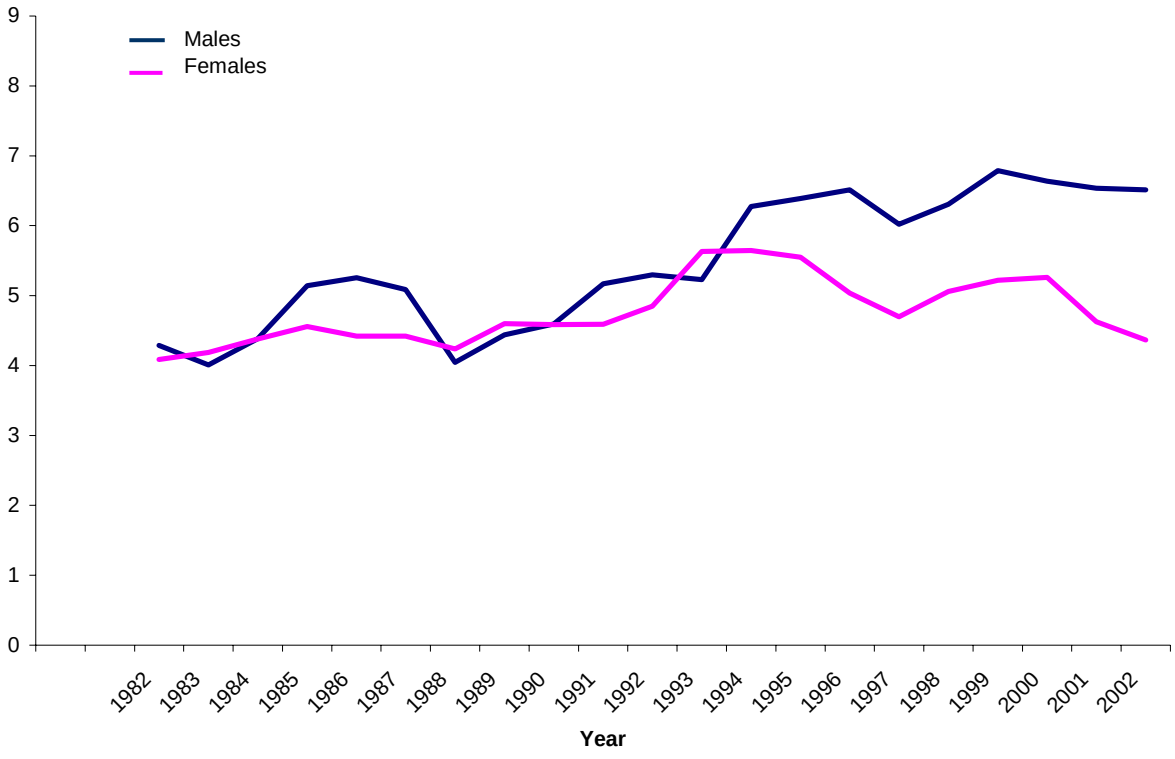
Table 4.7: Incidence of treated end-stage kidney disease caused by polycystic kidney diseases, 2001–2003

	0–24 years	25–44 years	45–54 years	55–64 years	65–74 years	75–84 years	85 years and over
	Number per million population						
Males	0	5	14	13	17	15	8
Females	0	2	11	14	8	9	2

Source: AIHW analysis of ANZDATA Registry data.

Prevalence of treated end-stage kidney disease caused by polycystic kidney diseases

In 2003, 1,146 people (62 cases per million population for males and 50 cases per million population for females) received kidney replacement therapy in Australia for ESKD due to PKD. Prevalence was highest among males aged 55–64 years (219 per million population) and females aged 65–74 years (217 per million population).



Notes

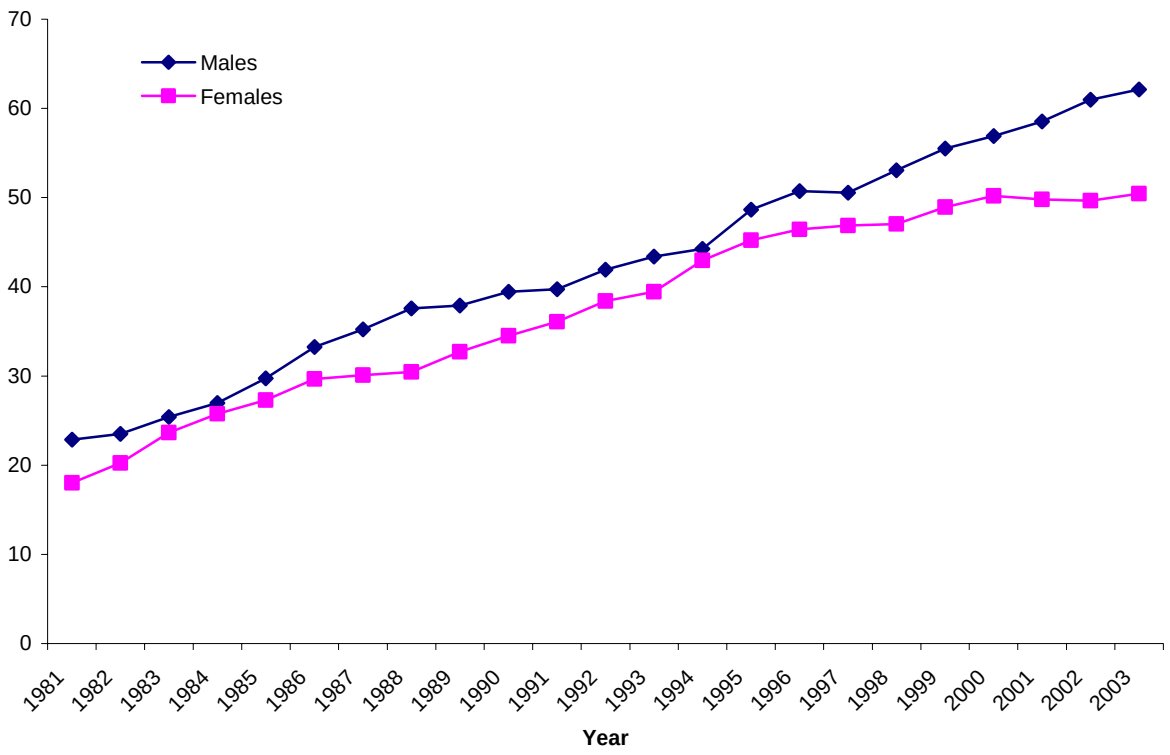
1. Age-standardised to the Australian population at 30 June 2001.
2. Data are three-year moving averages.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.14: Trends in the incidence of treated end-stage kidney disease due to polycystic kidney diseases, 1982 to 2002

Trends in prevalence

The prevalence rate of treated ESKD due to PKD gradually increased for both males and females from 1981, when it was 23 and 18 cases per million population, respectively (Figure 4.15). The prevalence was similar between males and females until 1998, but since then has been increasingly higher among males.



Note: Age-standardised to the Australian population at 30 June 2001.

Source: AIHW analysis of ANZDATA Registry data.

Figure 4.15: Trends in the prevalence of treated end-stage kidney disease due to polycystic kidney diseases, 1981 to 2003

References

McDonald SP, McCredie MR, Williams SM, Stewart JH 2005. Factors influencing reported rates of treated end-stage renal disease. Advances in Chronic Kidney Disease 12(1) 32-8.

White SL, Cass A, Atkins RC, Chadban SJ 2005. Chronic kidney disease in the general population. Advances in Chronic Kidney Disease 12:5–13.

5 Prevention and management of chronic kidney disease

Chronic kidney disease (CKD) is a serious and costly, but highly preventable and treatable, disease. Reducing the human and economic burden imposed by CKD relies heavily on preventing the onset and progression of the disease.

The Strategic Framework for Preventing Chronic Disease (NPHP 2001) takes a continuum of care approach to disease prevention. Intervention at various stages across the course of disease can prevent or delay progression to the next stage. The strategy identifies three broad levels of prevention: primary, secondary and tertiary.

Primary prevention measures aim to eliminate or reduce exposure to factors which cause ill health or disease. For CKD this involves strategies to reduce the incidence and prevalence of risk factors such as diabetes and high blood pressure, in order to reduce the number of people at risk of developing CKD. Secondary prevention consists of measures for early detection of disease to allow prompt and effective intervention to prevent the disease becoming established. Early detection and effective intervention in the early stages of kidney damage are essential to prevent or delay the development of CKD. Tertiary prevention strategies are focused on management of established disease to prevent progression and reduce or delay long-term complications, impairment or disability. Management of CKD aims to prevent or delay further kidney damage and loss of kidney function, and hence reduce the incidence and prevalence of ESKD and other complications. In those who do develop ESKD, good management during kidney replacement therapy not only reduces suffering and death, but also improves quality of life.

In Australia, considerable efforts have been made by government, health professionals and consumer groups to improve prevention and management of CKD. For example, successful use of public health strategies and legislative changes in Australia in 1979 have been credited with the significant decrease in incidence of treated end-stage kidney disease caused by analgesic nephropathy in the last decade. Also, improvements in management and techniques of kidney replacement therapy have led to longer survival of patients with end-stage kidney disease.

In 2000, Kidney Health Australia and the Australian and New Zealand Society of Nephrology developed the Caring for Australians with Renal Impairment (CARI) Guidelines to guide and improve the management of kidney disease in Australia (Box 5.1). The guidelines have been well accepted and have extensively influenced practice throughout Australia and New Zealand.

Although there have been successes in the past and consistent effort in recent years, the burden imposed by CKD in Australia has the potential to increase. This chapter provides an overview of the current situation with regard to the prevention and management of CKD in Australia. Where available, CARI guidelines are used against available information to illustrate the status of relevant practices. This overview provides information to inform future policies, strategies and interventions to help reduce the burden of CKD.

Risk reduction

There are some difficulties in preventing the population from developing CKD through reducing exposure to risk factors and causes of the disease, as these are not completely understood. However, there is strong evidence that many of the known risk factors that lead to developing CKD are modifiable. In Australia, widespread and consistent efforts have been made to promote healthy lifestyle in recent years. These efforts have led to a significant reduction in some risk factors, such as smoking and high blood pressure. However, not all risk factors have shown an improvement and there is potential for further gains in health through risk factor modification.

Several factors are involved in the reduction in risk of CKD in the community:

- Improving information and awareness of the disease;
- Reducing prevalence of behavioural risk factors;
- Reducing prevalence and improving control of biomedical risk factors; and
- Reducing exposure to external factors which increase risk.

Improving information and awareness of chronic kidney disease in the community

'One of the primary barriers to reducing the incidence of CKD in Australia is the limited public awareness of the preventable risk factors that contribute to its development and progress' (KHA 2004). Information regarding CKD risk reduction is available and accessible for most Australians. There are numerous government, non-government, pharmaceutical, and consumer web sites containing kidney disease information, and printed information is also available. In addition, an annual public awareness campaign (Kidney Awareness Week, or 'Wee Week') aims to improve awareness of kidney disease in the community and among health professionals.

However, national polling undertaken for the Pfizer Australia Health Report indicated that almost half of all respondents were not aware they could reduce their risk of developing kidney disease, and had little or no knowledge of how to keep their kidneys healthy (Pfizer Australia & Kidney Health Australia 2004). A recent stakeholder survey also found that less than 15% of respondents were aware that simple actions such as not smoking, following a healthy diet and having regular blood pressure checks could have benefits for the health of their kidneys (TNS Consultants 2004).

Reducing prevalence of behavioural risk factors

Recent progress in risk reduction with regard to behavioural factors is mixed. There has been a substantial reduction in the prevalence of tobacco smoking in the general population in Australia over the last 20 years, with the proportion of daily smokers aged 14 years and over nearly halving between 1985 and 2004. However, the proportion of people undertaking insufficient physical activity for health benefits has increased in recent years, and evidence suggests that the average Australian's diet is not as healthy as it could be. These two factors have played important roles in increasing the prevalence of obesity and diabetes in Australia, thereby indirectly contributing to the prevalence of CKD.

Reducing prevalence and improving control of biomedical risk factors

Of the biomedical risk factors for CKD, two of the most common and amenable to management are high blood pressure (hypertension) and diabetes. As progressive CKD can develop as complications of these conditions, good management is essential in reducing risk.

In people with established CKD, high blood pressure and poorly controlled blood glucose levels (among those who also have diabetes) may promote kidney function reduction and increase the risk of developing complications such as cardiovascular disease. These factors can therefore also be incorporated into tertiary prevention efforts.

Box 5.1: Caring for Australians with Renal Impairment (CARI) Guidelines

'Caring for Australians with Renal Impairment (CARI) Guidelines is a national evidence-based project that commenced in 1999 with funding from the pharmaceutical industry. The two bodies responsible for the CARI Guidelines are the Council of the Australian and New Zealand Society of Nephrology (ANZSN) and Kidney Health Australia (KHA).

The aim of the CARI Guidelines is to improve the health care and outcomes of paediatric and adult renal patients by helping clinicians and nurses to adhere to evidence-based medical practice as often as possible. It is anticipated that the guidelines will serve as both a valuable educational resource and a means of enhancing the quality, appropriateness, consistency and cost-effectiveness of renal health care.' (KHA 2005).

Existing guidelines cover the areas of chronic kidney disease, dialysis and transplantation. Each guideline comprises various subtopics and their recommendations. The first version of the guidelines was completed in 2000. It is intended that updating and revision of all guidelines will occur every 3 years, to ensure that the guideline contents are kept relatively up to date.

The detailed CARI Guidelines and associated evidence statements can be found on the internet at <http://www.kidney.org.au/cari/guidelines.php>.

Blood pressure control

The prevalence of high blood pressure among people aged 25–64 years has reduced in Australia in recent years. However, blood pressure control among those who have high blood pressure is not always ideal. According to the 1999–00 AusDiab study, among 3.6 million (28.6%) Australians aged 25 years and over with high blood pressure, 53.1% were untreated. Only 18.6% had blood pressure controlled in the normal range, and the remaining 81.4% had elevations in blood pressure to varied degrees (Table 5.1) (Briganti et al. 2003).

Table 5.1: Elevations in blood pressure among people with hypertension aged 25 years and over, 1999–00

Elevation in blood pressure	Untreated hypertension	Treated hypertension	Per cent of people with hypertension
Normal	0%	18.6%	18.6%
Mild	41.6%	16.7%	58.3%
Moderate	9.2%	8.3%	17.5%
Severe	2.3%	3.3%	5.6%
Total	**53.1%**	**46.9%**	**100%**

Source: Briganti et al. 2003.

For those people who already have CKD, a number of studies have shown that blood pressure lowering is associated with substantial lessening in kidney function decline. CARI Guidelines currently recommend the following targets for blood pressure (ANZSN & KHA 2001):

- ≤130/85 mmHg if proteinuria is <0.25 g/24 hours
- ≤130/80 mmHg if proteinuria is 0.25–1 g/24 hours
- ≤125/75 mmHg if proteinuria is >1 g/24 hours.

Information about blood pressure control and hypertension in people with CKD is not currently available in Australia.

Blood glucose control

There is evidence that tight control of blood glucose in people with diabetes can prevent or delay onset and slow the progression of kidney damage (DCCT 1993). The percentage of glycosylated haemoglobin in the blood (HbA1c level) is a measure of how well blood glucose has been controlled over the past three months. CARI Guidelines recommend that HbA1c be maintained at less than 7.5% for primary prevention of nephropathy and prevention of progression from microalbuminuria to overt nephropathy (ANZSN & KHA 2004a).

In the 1999–00 AusDiab study, the average HbA1c level among people with diabetes was 6.7%, with 78% of people meeting the CARI Guidelines HbA1c level of less than 7.5%. People with Type 1 diabetes were much less likely to have an HbA1c less than 7.5% than those with Type 2 diabetes (26% and 80% achieving this level, respectively).

Reducing exposure to external risks

In some cases, the toxic effects of certain chemicals or drugs may cause damage to the kidneys. These include certain antibiotics, heroin, and a number of drugs used in chemotherapy. Overuse of analgesics can also cause kidney damage.

The case of analgesic nephropathy is an example of successful disease control in Australia. Analgesic nephropathy is a type of toxic injury to the kidneys which may result from long-term daily use of analgesics. It was highly prevalent among the treated ESKD population in the past, accounting for nearly 20% of all new cases of treated ESKD in Australia in the early 1970s. From 1970 to 1983, the incidence rate of treated ESKD caused by analgesic nephropathy increased from 4 to 8 per million population. In 1979, legislative restriction on the advertising and over-the-counter sale of compound analgesics took place in Australia. This policy change has had a significant impact on the incidence of treated ESKD due to analgesic nephropathy (Briganti et al. 2000). The incidence was stable from 1984 to 1993, and then consistently decreased over the following decade to reach 4 per million population in 2003. Analgesic nephropathy is now one of the less commonly identified causes of treated ESKD in Australia.

Early detection

There is substantial evidence that early diagnosis and intervention provides the greatest opportunity for kidney function preservation. While there is currently no evidence to support mass screening of the general population by urine or blood testing, opportunistic screening is justified for selected high-risk groups (Johnson et al. 2004; Boulware et al. 2003).

CARI Guidelines recommend targeted screening of individuals at risk of developing kidney disease (ANZSN & KHA 2004b), including:

- patients with vascular disease, diabetes or hypertension;
- immediate relatives of patients with diabetes, hypertension or renal disease;
- Aboriginal and Torres Strait Islander Australians; and
- patients complaining of prostatic symptoms.

Methods of screening include checking blood pressure, performing urinalysis by microalbuminuria dipstick (or albumin/creatinine ratio) in people with diabetes and proteinuria dipstick in people without diabetes, and calculation of GFR (using serum creatinine).

There is currently no nationally coordinated, standardised screening program in Australia, and information on early detection among these high-risk groups is limited. Nevertheless, the results of the BEACH general practitioner survey provide some information on testing of kidney function in patients who were treated for vascular disease, diabetes and high blood pressure by GPs.

In 2002–03, high blood pressure was the most frequently managed problem in general practice, accounting for 9% of problems managed by GPs. GPs rarely referred patients with high blood pressure to other health professionals or services, suggesting that high blood pressure is mostly handled in general practice. Of these consultations for high blood pressure, around 13% had a pathology test ordered which could be used to detect kidney damage (see Chapter 2).

About 3% of all problems managed by GPs in 2002–03 related to diabetes. Tests that could be used to detect kidney damage were ordered in around 16% of these cases.

Besides hypertension and diabetes, other high-risk group individuals are patients with vascular diseases, including atherosclerosis, peripheral vascular disease, atrial fibrillation or flutter, cerebrovascular disease, coronary heart disease, heart failure and high blood cholesterol or lipid disorders. These diseases accounted for about 4% of problems managed by GPs in 2002–03. Tests that could be used to detect kidney damage were ordered in about 9% of these cases.

Management of early chronic kidney disease

Treatment of CKD may include use of angiotensin-converting enzyme (ACE) inhibitors, tight control of blood pressure and blood glucose, treatment of comorbidities, and patient education. The aims of management in patients with early stage CKD are delaying the progression of the disease, preventing onset of complications, providing early treatment for its comorbidities and preparing patients who are reaching the end stage for kidney replacement therapy.

In Australia, there is evidence that the aims of management of early CKD can be achieved in a high-risk population. In late 1995, a kidney and cardiovascular treatment program was introduced into the Tiwi community, which has a three- to five-fold increase in death rates and a recent annual incidence of end-stage kidney disease of 2,760 per million. People with confirmed hypertension, diabetes with microalbuminuria, or overt albuminuria received treatment including blood-pressure-lowering medication, attempts to control glucose and lipid levels, and health education. After nearly three and half years follow-up, there was a

50% reduction in end-stage kidney disease and natural death among the patients who had received treatment (Hoy et al. 2003).

It is not known if this treatment regime would benefit other patients who have CKD in Australia, as there are currently no national programs for CKD prevention. It is also impossible to evaluate outcomes of the treatment of CKD at early stages due to the absence of relevant information.

Management of pre-dialysis

Patients are classified as being at the pre-dialysis stage when they have severe CKD and are expected to progress to require dialysis or transplantation in the next 6–18 months (KHA 2004). Key patient needs at this stage include:

- appropriate selection of the preferred mode of therapy, and adequate preparation;
- timely initiation of treatment;
- availability of counselling, education, and rehabilitation throughout the process; and
- appropriate management of comorbid conditions (such as anaemia, high blood pressure and bone disease) and risk factors (such as blood lipids and nutrition) (KHA 2004).

Timely referral of patients to a specialist (nephrologist), pre-dialysis education and accessing a multidisciplinary health professional team are essential approaches to address these needs.

Timing of referral to nephrologist

The CARI Guidelines recommend that approximately one year is required to optimally prepare a patient and their family/carers for kidney replacement therapy (ANZSN & KHA 2004c). In Australia, referral to a nephrologist less than 90 days prior to the date of initiation of dialysis is regarded as a late referral. Late referral not only limits the patient's opportunity for selection of dialysis modality and for timely placement of an appropriate dialysis access, but also results in poorer patient survival and reduced likelihood of kidney transplant (Cass et al. 2003; Roderick et al. 2002).

The frequency of late referral in Australia has been between 25–30% in recent years. The reasons for such a high proportion of late referrals are not well studied. Several possible factors may influence the current situation, including:

- structural issues in the health system, such as distance from a dialysis facility and queues in outpatient clinics;
- lack of knowledge in physicians, and poor communication between GPs and nephrologists; and
- unavoidable factors, such as asymptomatic ESKD presenting at an advanced stage, or delay or refusal of the patient to seek medical care (KHA 2004).

Pre-dialysis education and training

Although various kidney units have developed pre-dialysis education programs, there are currently no national, standardised programs of pre-dialysis care and education available in Australia.

Results from a recent Australian survey suggested that patients who had been recently diagnosed as pre-dialysis had a low level of understanding as to what dialysis involved. They lacked knowledge of when they might expect to begin dialysis, how dialysis would affect them physically and how much of an impact it would have on their daily life (TNS 2004).

Availability of multidisciplinary health professional teams

There is evidence to suggest that patients who receive care from a multidisciplinary team (including nephrologists, dieticians, renal nurses, and social workers) have better outcomes from dialysis and survive longer (Curtis et al. 2005; Mendelssohn 2005). In Australia, there is some evidence to suggest that patients who obtain dialysis from a hospital unit have greater opportunities to access a multidisciplinary care team, compared with patients who attend satellite units or dialyse at home (Healthcare Management Advisors Pty Ltd 2004).

Management of end-stage kidney disease

When CKD reaches end-stage, kidney replacement therapy is the only currently available treatment to prolong life.

The number of people receiving kidney replacement therapy has more than tripled in Australia over the last 20 years. A greater number of older people are receiving treatment for ESKD, and the proportion of patients with comorbidities has increased. This has led to greater complexity in providing and managing kidney replacement therapy. However, despite these difficulties, advances in technology and better management have led to improvements in outcomes.

A profile of the general pattern of kidney replacement therapy in Australia is presented in Chapter 2. This section provides additional information on management of kidney replacement therapy, including adequacy of dialysis, transplant-related issues, and patient survival.

Adequacy of dialysis

Adequacy of haemodialysis

The outcome of haemodialysis may be influenced by many factors, particularly the urea reduction ratio (URR), nutritional status and vascular and infective comorbidities connected with dialysis.

Currently, no information has been collected nationally regarding the nutritional status and vascular and infective events during haemodialysis. However, data on some indicators of the technical aspects of haemodialysis, such as the URR, the dose of dialysis, vascular access, haemoglobin (Hb) concentration, calcium and phosphate products, are collected by the ANZDATA Registry. These indicators showed that up to March of 2004, most haemodialysis-dependent patients have received adequate treatment, and the adequacy of dialysis has improved over the last five years (Excell et al. 2005d; Kerr 2000). These data and corresponding CARI Guidelines (ANZSN & KHA 2000) are presented in Appendix 2.

Adequacy of peritoneal dialysis

Assessment of peritoneal dialysis adequacy involves various measurements including clinical assessment of wellbeing, physical measurements, small solute clearance, fluid removal and the impact of treatment on the individual's life. The results of such comprehensive measurements are not available nationally.

Peritonitis (inflammation of the peritoneum, the lining of the abdomen) is a major complication of peritoneal dialysis. Up to 31 March 2004, median peritonitis-free survival of peritoneal dialysis-dependent patients was 19.2 months, with 30% of patients completely free of peritonitis at 3 years (Excell et al. 2005c). This was an improvement from 1999, when the median peritonitis-free survival of peritoneal dialysis-dependent patients was 16.6 months, with 23% of patients completely free of peritonitis at 3 years (Collins 2000).

Transplant issues

Kidney donor sources

There are two sources of donor kidneys: living and deceased people. Kidneys from living donors can come from relatives (parents, sisters, brothers and adult children), or unrelated persons (usually the spouse, close friends or distant relatives). Kidneys can also come from a person who has been declared brain dead while their vital organs are being maintained on a breathing machine if they or their next of kin had agreed to donate their organs after death.

In 2003, 218 (40%) of 543 transplanted kidneys were from living donors, and 325 (60%) were from deceased donors. The number of transplanted kidneys from living donors has increased by 29% over the last 5 years (Excell et al. 2005b). This increase is largely attributed to the increasing number of unrelated living donors. This phenomenon can be traced to the development of more powerful anti-rejection drugs, which have reduced the importance of tissue matching in donor selection, thereby widening the possible pool of donors (International Association of Living Organ Donors 2002). In contrast, the increase in the number of kidneys from deceased donors appears relatively low, only 14% over the same period. The slow growth in deceased donor kidneys may be partly attributed to reduction in and increased survival following traffic accidents, and improvement of intensive care in the hospital.

Pre-emptive transplant

Kidney transplant prior to the initiation of dialysis is known as pre-emptive transplantation. Pre-emptive transplantation not only avoids pre-transplant dialysis and reduces the risk of morbidity and cost associated with dialysis, but also can increase the chance of survival.

In 2003, about 25% (50 cases) of all live donor transplantations were pre-emptive, accounting for 9% of all kidney transplantations (Excell et al. 2005b).

Waiting list for transplant

At 31 March 2004, 1,591 dialysis-dependent patients were waiting for a kidney transplant. This represents 21% of the total dialysis-dependent population. The majority of patients (94%) on the waiting list were under 65 years old, and 83% were waiting for their first transplant. The average waiting time for a kidney transplant was about 4 years.

Among the states and territories, the Australian Capital Territory had the longest waiting list, where 32% of dialysis-dependent patients were waiting for a kidney transplant. This was followed by New South Wales (28%), Victoria (21%), Western Australia (20%), Tasmania (18%), South Australia (14%), the Northern Territory (12%) and Queensland (9%) (Excell et al. 2005a). The reasons for the differences in the number of patients on the waiting list between the states are not clear, but could relate to different overall numbers of end-stage kidney disease patients, different age structures or suitability for transplant among these patients, variations in the number of deceased and living kidney donors, or other factors.

Survival

People receiving dialysis

The desired outcome of dialysis is to reduce morbidity and mortality and improve quality of life of people with ESKD. At present, there is no information available regarding morbidity of dialysis patients. According to the ANZDATA Registry, the survival of dialysis patients in Australia has improved across most age groups over the past 20 years in Australia. However, long-term survival rates for those aged 65 years and over are still low compared with people at younger ages. The survival rates at 1, 5 and 10 years after commencement of dialysis are shown in Table 5.2.

People with a kidney transplant

Quality of life and survival of kidney transplant patients are generally better than dialysis-dependent patients. The survival of kidney transplant patients has also improved over the past 20 years in Australia. The survival rates at 1, 5 and 10 years after transplantation are shown in Table 5.3.

Table 5.2: Trends in survival at 1, 5 and 10 years after commencement of dialysis, by age group and 5-year cohort

Age at commencement of dialysis	Years of survival	1981–85		1986–90		1991–95		1996–00		2001–03	
		Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)
		(Number)	(Per cent)	(Number)	(Per cent)	(Number)	(Per cent)	(Number)	(Per cent)	(Number)	(Per cent)
0–24 years	1	178	96 (± 0.01)	205	98 (± 0.01)	255	97 (± 0.01)	222	95 (±0.01)	98	98 (± 0.01)
	5	25	88 (± 0.03)	33	81 (± 0.05)	63	81 (± 0.03)	40	85 (± 0.03)		n.y.a.
	10	8	64 (± 0.11)	9	76 (± 0.07)	10	69 (± 0.06)		n.y.a.		n.y.a.
25–44 years	1	527	95 (± 0.01)	630	95 (± 0.01)	931	95 (± 0.07)	1,168	96 (± 0.01)	501	95 (± 0.01)
	5	106	70 (± 0.03)	139	68 (± 0.03)	307	71 (± 0.02)	304	73 (± 0.02)		n.y.a.
	10	25	42 (± 0.05)	47	46 (± 0.04)	48	48 (± 0.03)		n.y.a.		n.y.a.
45–64 years	1	1,083	87 (± 0.01)	1,430	89 (± 0.01)	2,006	90 (± 0.01)	2,441	89 (± 0.01)	1,298	89 (± 0.01)
	5	321	42 (± 0.02)	362	41 (± 0.01)	674	45 (± 0.01)	622	52 (± 0.01)		n.y.a.
	10	87	14 (± 0.01)	78	12 (± 0.01)	98	15 (± 0.01)		n.y.a.		n.y.a.
65–74 years	1	230	75 (± 0.02)	609	81 (± 0.01)	1,178	83 (± 0.01)	1,808	83 (± 0.01)	908	83 (± 0.01)
	5	86	28 (± 0.03)	185	25 (± 0.02)	393	29 (± 0.01)	416	33 (± 0.01)		n.y.a.
	10	9	3 (± 0.01)	27	4 (± 0.01)	47	6 (± 0.01)		n.y.a.		n.y.a.
75 years and over	1	16	68 (± 0.10)	60	76 (± 0.05)	223	76 (± 0.03)	732	78 (± 0.01)	602	77 (± 0.01)
	5	2	5 (± 0.04)	7	8 (± 0.03)	42	14 (± 0.02)	91	19 (± 0.01)		n.y.a.
	10	1	0	2	1 (± 0.01)	2	1 (± 0.01)		n.y.a.		n.y.a.

n.y.a. Not yet available.

Source: ANZDATA Registry.

Table 5.3: Trends in survival at 1, 5 and 10 years after kidney transplant, by age at transplant and 5-year cohort

		1981–85		1986–90		1991–95		1996–00		2001–03	
Age at transplant	Years of survival	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)	Patients at risk	Survival rate (std error)
		(Number)	(Per cent)	(Number)	(Per cent)	(Number)	(Per cent)	(Number)	(Per cent)	(Number)	(Per cent)
0–24 years	1	320	97 (±1.0)	328	98 (±0.8)	324	97 (±1.0)	320	99 (±0.6)	193	95 (±1.7)
	5	311	91 (±1.6)	320	92 (±1.5)	314	95 (±1.2)	313	94 (±1.5)		n.y.a.
	10	291	84 (±2.1)	299	87 (±1.9)	308	90 (±1.8)		n.y.a.		n.y.a.
25–44 years	1	656	94 (±1.0)	734	96 (±0.8)	738	97 (±0.6)	887	98 (±0.5)	590	99 (±0.3)
	5	613	82 (±1.5)	702	88 (±1.2)	715	91 (±1.1)	860	94 (±0.9)		n.y.a.
	10	539	70 (±1.8)	643	78 (±1.5)	668	79 (±1.6)		n.y.a.		n.y.a.
45–64 years	1	601	89 (±1.3)	795	88 (±1.1)	807	93 (±0.9)	925	94 (±0.8)	638	95 (±0.9)
	5	537	67 (±1.9)	703	75 (±1.5)	750	80 (±1.4)	870	84 (±1.3)		n.y.a.
	10	400	43 (±2.0)	597	52 (±1.8)	642	58 (±1.8)		n.y.a.		n.y.a.
65 years and over	1	2	50 (±35.4)	30	87 (±6.2)	59	88 (±4.2)	50	92 (±3.8)	76	94 (±3.0)
	5	1	0.0	26	73 (±8.1)	52	71 (±5.9)	46	73 (±6.7)		n.y.a.
	10		. .	22	37 (±8.8)	42	36 (±6.6)		n.y.a.		n.y.a.

. . Not applicable.

n.y.a. Not yet available.

Source: ANZDATA Registry.

References

ANZSN & KHA (Australian and New Zealand Society of Nephrology & Kidney Health Australia) 2000. The Caring for Australians with Renal Impairment (CARI) Guidelines (draft): Dialysis adequacy, in Dialysis guidelines. Adelaide: Australian and New Zealand Society of Nephrology and Kidney Health Australia. Viewed 8 June 2005, <http://www.kidney.org.au/cari/dialysis_adequacy_publ2000.php>.

ANZSN & KHA 2001. The Caring for Australians with Renal Impairment (CARI) Guidelines (draft): Blood pressure control-targets, in Prevention of progression of kidney disease. Adelaide: Australian and New Zealand Society of Nephrology and Kidney Health Australia. Viewed 8 June 2005, < http://www.kidney.org.au/cari/Part%202-46%20BP%20Control%20-%20targets.pdf>.

ANZSN & KHA 2004a. The Caring for Australians with Renal Impairment (CARI) Guidelines (draft): Glucose control and progression of diabetic nephropathy, in Prevention of progression of kidney disease. Adelaide: Australian and New Zealand Society of Nephrology and Kidney Health Australia. Viewed 8 June 2005, <http://www.kidney.org.au/cari/ckd_prevention_024.php>.

ANZSN & KHA 2004b. The Caring for Australians with Renal Impairment (CARI) Guidelines (draft): Early detection of patients with renal disease, in Prevention of progression of kidney disease. Adelaide: Australian and New Zealand Society of Nephrology and Kidney Health Australia. Viewed 8 June 2005, <http://www.kidney.org.au/cari/ckd_prevention_001.php>.

ANZSN & KHA 2004c. The Caring for Australians with Renal Impairment (CARI) Guidelines (draft): Early referral of pre-ESRD patients, in Prevention of progression of kidney disease. Adelaide: Australian and New Zealand Society of Nephrology and Kidney Health Australia. Viewed 8 June 2005, <http://www.kidney.org.au/cari/ckd_prevention_002.php>.

Boulware LE, Jaar BG, Tarver-Carr ME et al. 2003. Screening for proteinuria in US adults: a cost-effectiveness analysis. Journal of the American Medical Association 290:3114.

Briganti EM, McNeil J, Atkins R (eds) 2000. The epidemiology of diseases of the kidney and urinary tract: an Australian perspective. A report to the Board of the Australian Kidney Foundation. Melbourne: Monash University & Monash Medical Centre.

Briganti EM, Shaw JE, Chadban SJ et al. 2003. Untreated hypertension among Australian adults: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Medical Journal of America 179:135–9.

Cass A, Cunningham J, Snelling P et al. 2003. Late referral to a nephrologist reduces access to renal transplantation. American Journal of Kidney Diseases 42(5):1043–9.

Collins J 2000. Peritoneal dialysis. In: Disney APS (ed.). ANZDATA Registry report 2000. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Curtis BM, Ravani P, Malberti F et al. 2005. The short- and long-term impact of multi-disciplinary clinics in addition to standard nephrology care on patient outcomes. Nephrology Dialysis Transplantation 20(1):147–54.

DCCT (Diabetes Control and Complications Trial Research Group) 1993. The effect of intensive treatment of diabetes on the development and progression of long-term

complications in insulin-dependent diabetes mellitus. New England Journal of Medicine 329:977–98.

Excell L, Chadban SJ & McDonald SP 2005a. Transplant waiting list. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Excell L, Chadban SJ & McDonald SP 2005b. Transplantation. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Excell L, Johnson D & McDonald SP 2005c. Peritoneal dialysis. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Excell L, Kerr P & McDonald SP 2005d. Haemodialysis. In Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Healthcare Management Advisors Pty Ltd 2004. Improving and integrating care for patients on renal dialysis: a situation analysis. Melbourne: Victorian Department of Human Services.

Hoy WE, Wang Z, Baker PR & Kelly AM 2003. Reduction in natural death and renal failure from a systematic screening and treatment program in an Australian Aboriginal community. Kidney International Suppl. 83:S66–73.

International Association of Living Organ Donors 2002. Living kidney donation: history. International Association of Living Organ Donors, USA. Viewed 7 May 2005, <http://www.livingdonorsonline.org/kidney/kidney2.htm>.

Johnson CA, Levey AS, Coresh J et al. 2004. Clinical practice guidelines for chronic kidney disease in adults: part 1. Definition, disease stages, evaluation, treatment, and risk factors. American Family Physician 70:869–76.

Kerr P 2000. Haemodialysis. In: Disney APS (ed.). ANZDATA Registry report 2000. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

KHA (Kidney Health Australia) 2004. National service improvement frameworks: renal (CVD and Diabetes).

KHA 2005. The CARI guidelines. KHA, Melbourne. Viewed 22 August 2005, <http://www.kidney.org.au/?section=2&subsection=16>.

Mendelssohn DC 2005. Coping with the CKD epidemic: the promise of multidisciplinary team-based care. Nephrology Dialysis Transplantation 20(1):10–2.

NPHP (National Public Health Partnership) 2001. Preventing chronic disease: a strategic framework. Melbourne: NPHP.

Pfizer Australia and Kidney Health Australia 2004. Pfizer Australia Health Report. Issue no. 4. West Ryde, NSW: Pfizer Australia.

Roderick P, Jones C, Drey N et al 2002. Late referral for end-stage renal disease: a region-wide survey in the south west of England. Nephrology Dialysis Transplantation 17:1252–9.

TNS Consultants 2004. Stakeholders' needs assessment study: presentation of qualitative findings. Perth: TNS Consultants.

6 Chronic kidney disease in Aboriginal and Torres Strait Islander people

The poor health status and poor health outcomes among the Aboriginal and Torres Strait Islander population is a well known public health problem in Australia. Compared with other Australians, Indigenous Australians, in particular those in remote communities, have excessive chronic disease morbidity and mortality (ABS & AIHW 2003), and chronic kidney disease (CKD) is no exception to this. The impact of kidney disease, particularly ESKD, on Indigenous health was highlighted in an earlier report (ABS & AIHW 1999). Kidney damage, indicated by protein in the urine, is common among Indigenous Australians, and rates of treated end-stage kidney disease have been found in some communities to be up to 30 times the rates among other Australians (Spencer et al. 1998).

Although information on the incidence and prevalence of CKD among Indigenous Australians is not available at the national level, the heavy burden caused by CKD in this population is indicated by the high prevalence of CKD in certain communities, the high incidence and prevalence of treated ESKD, and the high hospitalisation and mortality rates associated with this disease among Indigenous Australians. This chapter outlines the available data relating to CKD in Indigenous Australians, and draws comparisons with other Australians where relevant. The particular challenges faced by Indigenous Australians in accessing health care for CKD are also discussed.

Prevalence of chronic kidney disease and its risk factors

Risk factors for CKD are highly prevalent among Aboriginal and Torres Strait Islander people (ABS & AIHW 2003). Tobacco smoking, poor nutrition, high blood pressure, alcohol abuse, obesity, diabetes, and preventable infections are common in many Aboriginal and Torres Strait Islander communities and have been associated with kidney impairment in this population (McDonald & Russ 2003). Results of the 2001 National Health Survey showed higher rates of diabetes, high blood pressure, smoking and obesity among Indigenous Australians compared with other Australians (ABS 2001). This, along with their poorer socioeconomic status and often remote location leading to poor access to health services, contributes to the increased rates of CKD and other chronic diseases among Indigenous Australians. In particular it is believed that the high incidence of streptococcal skin and throat infections among Indigenous Australians contributes to increased risk of glomerulonephritis (Chadban & Atkins 2005). Low birth weight is also common among Indigenous Australians, and there is evidence that this may be associated with greater risk for kidney disease, independent of other risk factors (Hoy et al. 1998).

Although no national data on CKD in Indigenous Australians are available, several studies have discovered high rates of CKD and indicators of kidney damage among Indigenous communities. McDonald et al. (2003) found that 12% of adults in a remote Aboriginal community in the Northern Territory had stage 3, 4 or 5 CKD and a further 36% had

evidence of reduced kidney function. Rates of treated ESKD in this community were previously known to be 15 times the national rate in the non-Indigenous population. However, although disease and risk factor prevalence are high among Indigenous Australians in general, wide variation is seen between different communities. Hoy et al. (2005) discovered marked variation in rates of smoking, alcohol use, hypertension, diabetes and kidney damage between three remote Aboriginal communities. This suggests that health interventions and preventive strategies for Indigenous Australians need to be adapted to address the different disease profiles of the various communities.

Incidence and prevalence of treated end-stage kidney disease

Between 2001 and 2003, 9% of new patients registered with ANZDATA in Australia identified as Indigenous Australians (514 people). This is a much higher proportion than Indigenous representation in the total population (2.4%). Although Indigenous identification in the ANZDATA Registry is based on hospital records, it is believed that this identification is more complete than in general hospital data, due to the heightened awareness of kidney disease among Indigenous Australians and the prolonged and repeated contact patients have with the hospital renal units (Cass et al. 2001).

The majority of new Indigenous patients between 2001 and 2003 were receiving dialysis, with only 52 transplants performed in Indigenous people (10% of new Indigenous patients). In contrast, 30% of new non-Indigenous patients during this period received kidney transplants. This partly reflects the lower number of donors compatible with Indigenous patients and the generally poorer health status leading to fewer Indigenous patients being suitable candidates for transplant, but these factors do not fully explain the low incidence of transplants in Indigenous Australians, nor their generally poorer survival following transplantation (McDonald 2004; Cass et al. 2003; Spencer et al. 1998). Similar inequalities in access to and survival following kidney transplants are seen in other indigenous populations such as New Zealand Maori, Native Americans and Aboriginal Canadians, but the reasons for these differences are not clear (Cass et al. 2004).

Indigenous Australians beginning kidney replacement therapy in 2001–03 were substantially younger than their non-Indigenous counterparts. Three-fifths (61%) of Indigenous patients were aged less than 55 years, with a median age of 52 years. In contrast, the median age of non-Indigenous patients was 64 years, with only one-third aged less than 55 years. There were more Indigenous females (284 cases, 55%) than males (230 cases, 45%) among these new patients, contrary to the overall pattern among the non-Indigenous treated end-stage kidney disease population (which was 61% male). This may be due to the higher rates of diabetes among Indigenous females.

Incidence rates of treated end-stage kidney disease in Indigenous Australians were higher than in other Australians. The difference was particularly marked at ages 45–54 years and 55–64 years (Table 5.1). Overall there were more than eight times as many Indigenous treated end-stage kidney disease patients as would be expected based on the incidence rates in non-Indigenous Australians.

Table 6.1: Standardised incidence ratios of treated end-stage kidney disease incidence in Indigenous compared with non-Indigenous Australians, 2001–2003

Age group	Number of Indigenous treated ESKD patients	Expected number of Indigenous treated ESKD patients[(a)]	Standardised incidence ratio[(b)]
0–34	40	16	2.47
35–44	92	10	9.67
45–54	182	10	17.57
55–64	138	10	14.00
65+	62	13	4.74
All ages	*514*	*59*	*8.72*

(a) Number of treated ESKD patients which would be expected in the Indigenous population if they experienced the same incidence rates as the non-Indigenous population.

(b) Ratio of the actual number of Indigenous treated ESKD patients compared to the expected number.

Source: AIHW analysis of ANZDATA Registry data.

The reasons for the high incidence of treated end-stage kidney disease among Indigenous Australians are not very clear, but are probably related to those factors which contribute to the increased risk of kidney impairment in this population, and lack of access to services for detection and treatment of CKD.

Comorbidities in treated end-stage kidney disease patients

Indigenous patients carry a high level of co-morbidities when they start kidney replacement therapy compared with non-Indigenous patients. This may not only result in poorer health status, decreased quality of life and poorer outcomes of dialysis, but may decrease eligibility for kidney transplant (McDonald & Russ 2003). Among new patients registered with ANZDATA between 2001 and 2003, Indigenous patients were more likely than non-Indigenous patients to have chronic lung disease and coronary artery disease, and much more likely to have diabetes (Table 6.2). Indigenous patients were also more likely than non-Indigenous patients to be current smokers and less likely to be former smokers.

Table 6.2: Comorbidities among new end-stage kidney disease patients, by Indigenous status, 2001–2003

	Indigenous patients (N = 514)	Non-Indigenous patients (N = 5,248)
Chronic lung disease[(a)]	19%	14%
Coronary artery disease[(a)]	43%	39%
Peripheral vascular disease[(a)]	33%	25%
Cerebrovascular disease[(a)]	14%	15%
Diabetes	78%	32%
Smoking status		
Current smoker	28%	10%
Former smoker	35%	41%
Never smoked	38%	49%

(a) Known or suspected to have the condition.

Source: Excell & McDonald 2005.

Trends in incidence and prevalence

From 1991 to 2003, although the incidence of treated end-stage kidney disease in Australia increased steadily for both the Indigenous and non-Indigenous populations, the increase was much faster in the Indigenous population. In 1991, the incidence rate of treated end-stage kidney disease among Indigenous people was 274 per million population, 4.8 times that in non-Indigenous people. By 2003, the incidence rate of treated end-stage kidney disease in Indigenous people was 7.8 times that in non-Indigenous people, at 696 per million population. This represents an average annual increase of 13% per year among the Indigenous population compared with 5% per year among the non-Indigenous population. This rapid increase may reflect both real growth in the incidence of ESKD among Indigenous people and the increasing availability and acceptability of kidney replacement therapy to Indigenous communities.

As kidney replacement therapy became more widely available and ESKD patients from remote Indigenous communities were able to be treated, the prevalence of treated end-stage kidney disease among Indigenous Australians also increased rapidly. Between 1991 and 2003 there was an average annual increase in prevalence of 20% per year, from 228 Indigenous patients (1,060 per million population) in 1991 to 882 Indigenous patients (3,573 per million population) in 2003. In comparison, over this period there was an average 5% annual increase in treated end-stage kidney disease prevalence among other Australians, from 6,400 cases (401 per million population) in 1991 to 12,743 cases (639 per million population) in 2003.

Cass et al. (1999) found variations in the incidence of treated ESKD between Aboriginal Australians in New South Wales and the Northern Territory. While the incidence among Aboriginal Australians in the Northern Territory was high (800 per million population in 1996–97) and had increased rapidly over the period 1988–89 to 1996–97, incidence among Aboriginal Australians in New South Wales was relatively low (111 per million population in 1996–97) and showed no increase over the same period. It was suggested that the less profound socioeconomic disadvantage and better access to care of Aboriginal Australians in New South Wales contributed to the lower incidence rates in that state (Cass et al. 1999).

Health care for Aboriginal and Torres Strait Islander people with chronic kidney disease

Aboriginal and Torres Strait Islander people, particularly those in remote communities, face many barriers to access to health care. These barriers impact on all stages of CKD, affecting the prevalence and management of risk factors and diseases associated with CKD, detection of kidney problems, management of CKD and prevention of disease progression. Treatment for ESKD is a particular problem for Australians living in remote communities. The distance to the nearest health facility with the necessary equipment for dialysis may be hundreds or even thousands of kilometres, and the cost of travel and accommodation may be prohibitive. In addition, the cultural importance of family and place to Indigenous Australians means that the thought of leaving their home and moving to a hospital or satellite dialysis facility which is too far away for them to return home easily or for family and friends to visit is distressing and frightening to many patients (Willis 1995).

Several programs are in place to provide better health services to remote communities. A number of city-based units in Western Australia and the Northern Territory provide support for peritoneal dialysis in remote communities. The Remote Area Dialysis Programme,

operating out of the Royal Perth Hospital, allows ESKD patients in remote parts of Western Australia to receive haemodialysis in their own community, either at home or in a community facility. Individuals, communities, governments and the Aboriginal Community Controlled Health Service have established satellite dialysis facilities in remote locations, such as Broome and Tennant Creek.

In the Tiwi Islands, a systematic treatment program involving medication to reduce blood pressure, improvement of blood glucose and blood lipid control, and health education was found to significantly reduce risk factor levels, progression of CKD and death from kidney and cardiovascular diseases (Hoy et al. 2000). The program was found to be cost-effective, with over $3 million worth of dialysis saved (Baker et al. 2005).

Hospitalisation associated with chronic kidney disease

In 2003–04, Indigenous identification in hospital separation records was considered of acceptable quality for use only in the Northern Territory, South Australia and Western Australia. Indigenous Australians were almost 19 times as likely to be hospitalised with CKD recorded compared with other Australians in these areas (Table 6.3). In the three jurisdictions there were 48,913 hospital separations among Indigenous Australians where CKD was the principal diagnosis. This represented 47% of all hospital separations for Indigenous Australians in these areas. Almost all of these separations (98%) were attributed to routine dialysis. In comparison, separations where CKD was the principal diagnosis accounted for 11% of all hospital separations among other Australians in these jurisdictions.

There were 52,914 bed days associated with these Indigenous CKD hospital separations. After removal of day-stay dialysis (47,975 separations), the average length of stay in hospital for Indigenous people with CKD as the principal diagnosis in these three jurisdictions was 5.3 days. This was almost 20% higher than the average length of stay for non-Indigenous people with a principal diagnosis of CKD (Table 6.3).

Table 6.3: Hospital separations with a principal diagnosis of chronic kidney disease, by Indigenous status, 2003–04

	Indigenous Australians		Other Australians		
	Number of separations	Average length of stay (days)	Number of separations	Average length of stay (days)	Standardised separation ratio[(a)]
Separations with principal diagnosis of CKD					
Males	18,727	1.1	80,877	1.1	12.1
Females	30,186	1.1	51,107	1.1	29.7
Persons	48,913	1.1	131,984	1.1	18.9
Excluding same-day dialysis					
Males	320	5.2	1,894	4.8	6.9
Females	618	5.3	2,372	4.2	7.4
Persons	938	5.3	4,266	4.5	7.2

(a) Calculated as the number of separations in Indigenous Australians divided by the number of separations that would be expected if Indigenous Australians experienced the same hospital separation rates as other Australians.

Note: Includes data for South Australia, Western Australia and the Northern Territory only.

Source: AIHW National Hospital Morbidity Database.

In contrast to other Australians, there were more separations where CKD was the principal diagnosis among Indigenous females than males in South Australia, Western Australia and the Northern Territory combined in 2003–04 (Table 6.3). Indigenous Australian females were almost 30 times as likely as other Australian females to be hospitalised with a principal diagnosis of CKD in these jurisdictions, with Indigenous Australian males being 12 times as likely to be hospitalised with a principal diagnosis of CKD as other Australian males. When day admissions for dialysis are excluded, both sexes are around seven times as likely as other Australians to be hospitalised with a principal diagnosis of CKD.

Deaths associated with chronic kidney disease

Between 2001 and 2003, Indigenous identification in mortality records was considered of acceptable quality for use in the Northern Territory, South Australia, Western Australia and Queensland. In these four jurisdictions during this period, Indigenous Australians were almost eight times as likely as other Australians to have died from CKD. CKD was recorded as the underlying cause of death in 154 of the 4,474 deaths identified in Indigenous Australians (3% of all deaths among Indigenous Australians in these areas). In a further 474 cases, CKD was recorded as an associated cause of death only (11% of all Indigenous deaths). In comparison, there were 138,668 deaths among other Australians in these four jurisdictions between 2001 and 2003, with CKD recorded as the underlying cause in 2,381 cases and as an additional cause in a further 8,821 cases (2% and 6% of deaths among other Australians, respectively).

Deaths from CKD occurred at younger ages among Indigenous Australians than among other Australians. In deaths with an underlying cause of CKD, the average age at death among Indigenous Australians was 55 years for males and 61 years for females, compared with 79 years and 82 years among other Australian males and females, respectively. On average, approximately 26 years of life were lost per death for Indigenous Australians, compared with around 9 years per death for other Australians.

References

ABS (Australian Bureau of Statistics) 2001. National health survey: Aboriginal and Torres Strait Islander results, Australia 2001. ABS Cat. No. 4715.0. Canberra: ABS.

ABS & AIHW (Australian Bureau of Statistics & Australian Institute of Health and Welfare) 1999. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples. ABS Cat. No. 4704.0 and AIHW Cat. No. IHW 3. Canberra: ABS & AIHW.

ABS & AIHW 2003. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples. ABS Cat. No. 4704.0 and AIHW Cat. No. IHW 11. Canberra: ABS & AIHW.

Baker PR, Hoy WE & Thomas RE 2005. Cost-effectiveness analysis of a kidney and cardiovascular disease treatment program in an Australian Aboriginal population. Advances in Chronic Kidney Disease 12:22–31.

Cass A, Cunningham J, Snelling P, Wang Z & Hoy W 2003. Renal transplantation for Indigenous Australians: identifying the barriers to equitable access. Ethnicity & Health 8(2):111–19.

Cass A, Cunningham J, Wang Z & Hoy W 2001. Regional variation in the incidence of end-stage renal disease in Indigenous Australians. Medical Journal of Australia 175:24–7.

Cass A, Devitt J, Preece C et al. 2004. Barriers to access by Indigenous Australians to kidney transplantation: the IMPAKT study. Nephrology 9 Suppl. 4:S144–6.

Cass A, Gillin AG & Horvath JS 1999. End-stage renal disease in Aboriginals in New South Wales: a very different picture to the Northern Territory. Medical Journal of Australia 171:407–10.

Chadban SJ & Atkins RC 2005. Glomerulonephritis. Lancet 365:1797–806.

Excell L & McDonald S (eds) 2005. Appendix II. Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) report 2004. Adelaide: ANZDATA.

Hoy WE, Baker PR, Kelly AM & Wang Z 2000. Reducing premature death and renal failure in Australian Aboriginals. A community-based cardiovascular and renal protective program. Medical Journal of Australia 174:201–2.

Hoy W, Kondalsamy-Chennakesavan S, Scheppingen J & Sharma S 2005. Kidney and related chronic disease profiles and risk factors in three remote Australian Aboriginal communities. Advances in Chronic Kidney Disease 12(1):64–70.

Hoy WE, Rees M, Kile E, Mathews JD, McCredie DA, Pugsley DJ & Wang Z 1998. Low birthweight and renal disease in Australian Aborigines. Lancet 352(9143):1826–7.

McDonald S 2004. Indigenous transplant outcomes in Australia: what the ANZDATA Registry tells us. Nephrology 9 Suppl. 4:S138–43.

McDonald SP, Maguire GP & Hoy WE 2003. Renal function and cardiovascular risk markers in a remote Australian Aboriginal community. Nephrology, Dialysis, Transplantation 18:1555–61.

McDonald SP & Russ GR 2003. Burden of end-stage renal disease among Indigenous peoples in Australia and New Zealand. Kidney International 63 Suppl. 83:S123–7.

Spencer JL, Silva DT, Snelling P & Hoy WE 1998. An epidemic of renal failure among Australian Aboriginals. Medical Journal of Australia 168:537–41.

Willis J 1995. Fatal attraction: do high technology treatments for end-stage renal disease benefit Aboriginal people in Central Australia? Australian Journal of Public Health 19:603–9.

Appendix 1. Identification of people with chronic kidney disease; statistical methods; and data sources

Identification of people with chronic kidney disease

Chronic kidney disease has long been recognised as a health problem. However, 'chronic kidney disease' is not used as a medical term in the WHO International Classification of Diseases (ICD), nor is it generally used as a diagnosis in clinical settings. For these reasons, it is impossible to identify CKD patients directly in most existing databases, where the data are collected based on doctors' diagnoses and classified using the ICD system. It is also not possible to identify CKD patients through assessing their kidney function, as most databases do not contain pathology information.

To overcome this barrier, we developed a coding list for chronic kidney disease (Table A1). This coding list contains the primary kidney diseases listed in the International Classification of Diseases version 10 (ICD-10) that are known to cause chronic kidney disease. People can be assumed to have CKD if they have any diagnosis of these primary kidney diseases.

Australian general practice data are classified according to the International Classification of Primary Care, second edition (ICPC-2) (WICC 1997). We developed a list of ICPC-2 codes and (where necessary) more specific ICPC-2 PLUS terms for CKD by considering the chronic disease list developed from ICPC-2 by O'Halloran et al. (2004) and considering all ICPC-2 codes that mapped to the selected ICD-10 codes (Table A2).

The coding lists have been discussed among several nephrologists, experts on disease classification and the researchers who prepared this report. They appear to cover CKD accurately and comprehensively. However, it is not possible to fully identify people with CKD using this method. The coding lists herein only include those diseases that are known to cause CKD. Once people are diagnosed with one of these diseases, they can be identified as having CKD without obtaining further evidence from pathology information. However, some other diseases or conditions, such as calculus of the kidney and ureter, do not always result in CKD. In these cases CKD can not be identified without pathological evidence to indicate that there is kidney damage and/or reduced kidney function. Because administrative databases such as the AIHW National Hospital Morbidity Database do not contain pathology information, these cases have not been identified as CKD in this report, unless they also recorded a diagnosis of one of the diseases contained in the CKD coding list. This may lead to some underestimation of the true burden of CKD in Australia.

Table A1: ICD-10 coding list for chronic kidney disease

ICD-10 code	Description
B52.0^	Plasmodium malariae malaria with nephropathy
D59.3^	Haemolytic-uraemic syndrome
E10.2	Insulin-dependent diabetes mellitus with renal complication
E11.2	Non-insulin-dependent diabetes mellitus with renal complication
E12.2	Malnutrition-related diabetes mellitus with renal complication
E13.2	Other specified diabetes mellitus with renal complication
E14.2	Unspecified diabetes mellitus with renal complication
E85.1^	Neuropathic heredofamilial amyloidosis
I12	Hypertensive renal disease
I13	Hypertensive heart and renal disease
I15.0	Renovascular hypertension
I15.1	Hypertension secondary to other renal disorders
N00	Acute nephritic syndrome
N01	Rapidly progressive nephritic syndrome
N02	Recurrent and persistent haematuria
N03	Chronic nephritic syndrome
N04	Nephrotic syndrome
N05	Unspecified nephritic syndrome
N06	Isolated proteinuria with specified morphological lesion
N07	Hereditary nephropathy, not elsewhere classified
N08*	Glomerular disorders in diseases classified elsewhere
N11	Chronic tubulo-interstitial nephritis
N12	Tubulo-interstitial nephritis, not specified as acute or chronic
N14	Drug- and heavy-metal-induced tubulo-interstitial and tubular conditions
N15	Other renal tubulo-interstitial diseases
N16*	Renal tubulo-interstitial disorders in diseases classified elsewhere
N18	Chronic renal failure
N19	Unspecified renal failure
N25	Disorders resulting from impaired renal tubular function
N26	Unspecified contracted kidney
N27	Small kidney of unknown cause
N28	Other disorders of kidney and ureter, not elsewhere classified
N39.1	Persistent proteinuria, unspecified
N39.2	Orthostatic proteinuria, unspecified
Q60	Renal agenesis and other reduction defects of kidney
Q61	Cystic kidney disease
Q62	Congenital obstructive defects of renal pelvis and congenital malformation of ureter
Q63	Other congenital malformations of kidney
T82.4	Mechanical complication of vascular dialysis catheter
T86.1	Kidney transplant failure and rejection
Z49*	Care involving dialysis
Z94.0*	Kidney transplant status
Z99.2*	Dialysis status

^ These codes are to be used for identification in mortality data only.

* These codes are to be used for identification in hospital morbidity data only.

Table A2: ICPC-2 and ICPC-2 PLUS coding list for chronic kidney disease

ICPC-2 code	ICPC-2 PLUS code	ICPC-2/ICPC-2 PLUS label
	K87002	Hypertension; renal disease
	K87003	Hypertension; nephropathy
	K87006	Hypertension; cardiorenal
	U28001	Kidney transplant
	U59001	Dialysis; kidney (renal)
	U59007	Dialysis; peritoneal
	U59008	Haemodialysis
	U59009	Dialysis; CAPD
	U85001	Polycystic kidney
	U85003	Duplex kidney
	U85004	Congenital anomaly; urological
	U85005	Congenital anomaly; kidney
U88	(all)	Glomerulonephritis/nephrosis
	U99002	Cyst; renal
	U99016	Uraemia
	U99020	Hypertrophic; kidney
	U99021	Hydronephrosis
	U99022	Insufficiency; renal
	U99023	Failure; renal; chronic
	U99024	Necrosis; renal; papillary
	U99028	Stenosis; artery; renal
	U99030	Failure; renal; not otherwise stated

References

O'Halloran J, Miller GC & Britt H 2004. Defining chronic conditions for primary care with ICPC-2. Family Practice 21(4):381–6.

WICC (Classification Committee of the World Organization of Family Doctors) 1997. ICPC-2: International Classification of Primary Care. Oxford: Oxford University Press.

International Classification of Diseases (ICD)

The International Classification of Diseases (ICD) is used to classify diseases and other health problems recorded on many types of health and vital records including death certificates and hospital records. In addition to enabling the storage and retrieval of diagnostic information for clinical and epidemiological purposes, these records also provide the basis for the compilation of national mortality and morbidity statistics by WHO member states.

ICD was created in the 1850s. The first edition, known as the International List of Causes of Death, was adopted by the International Statistical Institute in 1893. WHO took over the responsibility for the ICD at its creation in 1948 when the sixth revision, which included causes of morbidity for the first time, was published. ICD-10 was endorsed by the Forty-third World Health Assembly in May 1990 and came into use in WHO member states as from 1994. The classification is the latest version in the ICD series.

The ICD has become the international standard diagnostic classification for all general epidemiological and many health management purposes. These include the analysis of the general health situation of population groups and monitoring of the incidence and prevalence of diseases and other health problems in relation to other variables such as the characteristics and circumstances of the individuals affected.

International Classification of Primary Care, second edition (ICPC-2)

The International Classification of Primary Care, second edition (ICPC-2) is used as a classification for primary care or general practice wherever applicable.

ICPC-2 classifies patient data and clinical activity in the domains of general/family practice and primary care, taking into account the frequency distribution of problems seen in these domains. It allows classification of the patient's reason for encounter, the problems/diagnoses managed, interventions, and the ordering of these data in an episode of care structure.

It has a biaxial structure and consists of 17 chapters, each divided into seven components dealing with symptoms and complaints (comp. 1), diagnostic, screening and preventive procedures (comp. 2), medication, treatment and procedures (comp. 3), test results (comp. 4), administrative (comp. 5), referrals and other reasons for encounter (comp. 6) and diseases (comp. 7).

Statistical methods

Age standardisation

This is a method of removing the influence of age when comparing populations with different age structures. Age-standardised rates in this report use direct age-standardisation. The directly age-standardised rate is the weighted sum of age-specific (five-year age group) rates, where the weighting factor is the corresponding age-specific standard population. For this report, the Australian estimated residential population as at 30 June 2001 was used as the standard population. The same population was used for males and females to allow valid comparison of age-standardised rates between the sexes.

Direct age standardisation

Direct age standardisation is the most common method of age standardisation, and is used in this report for prevalence, incidence, hospitalisations and deaths data. This method is generally used when the population under study is large and the age-specific rates are reliable. The calculation of direct age-standardised rates comprises three steps:

Step 1: Calculate the age-specific rate for each age group.

Step 2: Calculate the expected number of cases in each age group by multiplying the age-specific rate by the corresponding standard population for each age group.

Step 3: Sum the expected number of cases in each age group and divide this sum by the total of the standard population to give the age-standardised rate.

In interpreting age-standardised rates, some issues need to be taken into consideration:

- The age-standardised rate is for comparison purposes only. The magnitude of an age-standardised rate has no intrinsic value since it is only an index measure. Therefore an age-standardised rate is not a substitute for age-specific rates.
- An age-standardised rate is not only influenced by the frequency of the underlying diseases, but is also dependent on the differences between the age structure of the population of interest and the standard population selected. Therefore, the results of comparisons based on age-standardised rates may not only reflect the difference in the frequency of the diseases compared, but also will be partly dependent on the standard population used. However, since the standard population used in this report is the total Australian population in 2001, the age distribution closely reflects that of the current Australian population. The results of comparison based on these age-standardised rates are valid.

Indirect age standardisation

In situations where populations are small or where there is some uncertainty about the stability of age-specific rates, indirect standardisation is used. This effectively removes the influence of different age structures, but does not provide a measure of incidence or prevalence in terms of a rate. Rather, the summary measure is a ratio of the number of observed cases compared to the number that would be expected if the age-specific rates of

the standard population applied in the population under study. Calculation of these ratios comprises the following steps:

Step 1: Calculate the age-specific rates for each age group in the standard population.

Step 2: Apply these age-specific rates to the number of people in each age group of the population under study, and sum these to derive the total expected number of cases in that population.

Step 3: Sum the observed cases in the population under study and divide this number by the expected number derived in step 2. This is the standardised incidence/prevalence ratio (SIR or SPR). Standardised mortality or morbidity ratios (SMRs) can be calculated similarly.

An SIR/SMR of 1 indicates the same number of observed cases as were expected, suggesting rates in the two populations are similar. An SIR greater than 1 indicates more cases observed than were expected, suggesting rates in the population under study are higher than in the standard population.

In this report, the indirect method is used in Chapter 6 when comparing Indigenous and other Australians.

Moving averages

Moving averages are used for smoothing of trend data, to even out the small seasonal or cyclic variations which occur from one time point to the next so that the underlying trend can be clearly seen. In this report, three-year moving averages are calculated to show trends in the age-standardised incidence rates of end-stage kidney disease. To calculate each moving average observation, the age-standardised rates for three consecutive years are combined and divided by three. This average is then used as the value for the middle year of the three points used to calculate the average.

Age-specific rates

Age-specific rates were calculated by dividing the number of events (such as deaths, disease cases or hospital separations) occurring in each specified age group by the estimated resident population for the corresponding age group. The rates are expressed as events per 1,000 or per million population.

Prevalence

Prevalence refers to the number or proportion (of cases, instances, etc.) present in a population at a given time.

Incidence

Incidence refers to the number of new cases (of a disease, condition or event) occurring during a given period.

Data sources

Most of the information on mortality, health services use and health expenditure in this report is drawn from administrative databases, such as the AIHW National Mortality Database and the AIHW National Hospital Morbidity Database. In recent years, administrative databases have been increasingly used for statistical analysis by health officials and academics, both at the national and international level. The data in these databases were collected systematically and regularly with broad population coverage. However, because the data are based on doctors' diagnoses, diseases that are likely to be under-diagnosed in the clinical setting, such as chronic kidney disease and diabetes, are also likely to be under-represented in these databases. Therefore it is likely that the burden of chronic kidney disease calculated from these databases will be an underestimate.

The administrative databases and other major data sources used in this report are briefly described below.

Administrative data sources

AIHW Disease Expenditure Database contains information on direct health expenditure in 2000–01 for around 200 different disease and injury categories. Estimates are available by age group, sex and area of expenditure—hospitals, high-level residential aged care, medical services, other professional services, pharmaceuticals and research. Capital expenditures, expenditure on community health (except community mental health), public health programs (except cancer screening), health administration and health aids and appliances were not allocated by disease group.

AIHW National Hospital Morbidity Database contains demographic, diagnostic, procedural and duration of stay information on episodes of care for patients admitted to hospital. The data collection is maintained by the AIHW using data supplied by state and territory health authorities. The database is episode-based and it is not possible to count patients individually. In this report, disease data relate to the principal diagnosis reported for hospitalisations unless otherwise specified. Data presented in this report were extracted in July 2005.

AIHW National Mortality Database contains information on the cause of death supplied by the medical practitioner certifying the death or by a coroner. Registration of deaths is the responsibility of the state and territory registrars of Births, Deaths and Marriages. Registrars provide the information to the ABS for coding of cause of death and the encoded data are then provided to AIHW. In this report, unless otherwise specified, death data relate only to the underlying cause of death. Data presented in this report were extracted in June 2005.

Register data sources

Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) collects information to monitor dialysis and transplant treatments from all renal units in Australia and New Zealand on all patients receiving kidney replacement therapy where the intention to treat is long term. Cases of acute kidney failure are excluded. The Registry is coordinated within the Queen Elizabeth Hospital (South Australia) and is funded by the Australian Government Department of Health and Ageing.

Survey data sources

Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (1999–00), conducted by the International Diabetes Institute, was designed to provide estimates of the prevalence of diagnosed and undiagnosed diabetes and self-reported chronic conditions such as heart disease and high blood pressure. It also provided national measurements of blood pressure, blood lipids, blood glucose, body fat, height and weight, and waist and hip circumference, as well as self-reported information on diet, smoking, alcohol consumption, physical activity, and general health and wellbeing. The study collected information in urban and non-urban areas in all states and the Northern Territory and sampled over 20,000 people aged 25 years and above, of whom more than 11,000 underwent a physical examination.

BEACH (Bettering the Evaluation and Care of Health) Survey of General Practice, an ongoing national survey looking at aspects of general practice in Australia, is conducted by the General Practice Statistics and Classification Unit (an AIHW collaborating unit within the Family Medicine Research Centre, University of Sydney). BEACH began in April 1998 and involves a random sample of approximately 1,000 general practitioners per year, each of whom records details regarding 100 consecutive patient encounters.

National Drug Strategy Household Survey (2004) was conducted between July and November 2004 and includes data on almost 30,000 Australians aged 12 years and older. This was the eighth survey in a series that began in 1985. Respondents were asked about their knowledge of drugs, their attitudes towards drugs, their drug consumption histories and related behaviours.

National Health Survey (2001), conducted by the ABS, included around 26,900 people of all ages. Collection occurred between February and November 2001 across urban and rural areas of Australia. Non-private dwellings (e.g. hospitals, nursing homes, hotels and boarding houses) were excluded. The survey collected information on long-term health conditions, use of health services, and health risk factors and behaviours.

National Nutrition Survey (1995), conducted by the ABS, was the largest and most comprehensive Australian survey of food and nutrient intake, dietary habits and body measurements. The survey collected information from a subsample of respondents from the 1995 National Health Survey, approximately 13,800 people from urban and rural areas of Australia. The National Nutrition Survey was conducted over a 13-month period from February 1995 to March 1996.

National Physical Activity Surveys (1997, 1999 and 2000). The 2000 survey was conducted to give an assessment of physical activity patterns and knowledge of the benefits of physical activity among adult Australians after the Olympics in Sydney (September 2000). The survey collected information from a national sample of 3,590 people aged 18–75 years during November and December 2000. This survey follows on from the 1997 (the Active Australia Baseline survey) and 1999 National Physical Activity Surveys. The 1997 survey collected information from a national sample of 4,821 people in November and December 1997. The 1999 survey collected information from a national sample of 3,841 people in November and December 1999.

Risk Factor Prevalence studies (1980, 1983 and 1989), a series of surveys conducted by the National Heart Foundation of Australia, were designed to obtain national information on biomedical and behavioural risk factors in Australia and to monitor trends over time. The studies collected information from a sample of around 22,000 adults living in capital cities of Australia (Canberra and Darwin were not included in the 1980 and 1983 surveys), between May/June and December of the survey year.

Appendix 2. Adequacy of haemodialysis

Table A3: Adequacy of haemodialysis, 2000 and 2004

Indicator	CARI guideline	Achievement at 31 March 2000	Achievement at 31 March 2004
Urea reduction ratio (URR)	The target URR should be equal or over 65%.	79% of haemodialysis-dependent patients achieved this target.	87% of haemodialysis-dependent patients achieved this target. This is an 8% increase over 5 years.
Frequency	Three times per week.	97% of patients dialysed three times per week.	93% of patients dialysed three times per week. This is a 4% decrease over 5 years.
Duration	Minimum 4 hours for each treatment session.	92% of patients were dialysing for 4 hours or longer for each treatment session. The median weekly treatment period was 12 hours; range 4–26 hours.	91% of patients were dialysing for 4 hours or longer for each treatment session. The median weekly treatment period was 12 hours; range 3–50 hours.
Membranes	High flux membranes were recommended for patients expecting prolonged dialysis (more than 5 years).	About 8% of patients received dialysis with high flux membranes.	36% of patients received dialysis with high flux membranes. This is a 28% increase over 5 years.
Blood flow rate	No guideline existing.	65% of patients were prescribed a blood flow rate of 300 mL/min or higher.	76% of patients were prescribed a blood flow rate of 300 mL/min or higher.
Vascular access	Creation of a native arteriovenous fistula is paramount.	Data not available.	39% of patients whose treatment began between 1 October 2003 and 31 March 2004 have a native haemodialysis access. This is a 30% increase from 2000.
Haemoglobin (Hb) concentration	The minimum recommended Hb concentration in chronic dialysis patients is 110 g/L.	Data not available.	66% of patients were at or above the minimum recommended Hb concentration at 31 March 2004.
Calcium x phosphate product	Serum albumin-corrected calcium x phosphate product should not exceed 5.8 mmol/L. The ideal target is less than 4.2 mmol/L.	Data not available.	87% of patients had calcium x phosphate product level less than 5.8 mmol/L. 60% of patients had calcium x phosphate product level less than 4.2 mmol/L.

Source: Excell L, Marshall M & McDonald S 2005. Haemodialysis. In: Excell L & McDonald SP (eds). ANZDATA Registry report 2004. Adelaide: ANZDATA, 35–52.

Appendix 3. Potential chronic kidney disease indicators and monitoring framework

Health indicators are tools that that can turn complicated data into relevant and easily understood information. They are measurements that are indicative of the impacts of diseases on communities and also reflect the result of efforts both of health service provision and intervention. Such information helps policy makers and others identify trends and patterns of the diseases, provides evidence for decision making and supports evaluations of progress towards addressing health challenges. It can also be used to highlight areas for possible intervention action. These indicators can be used for the regular surveillance and monitoring of the occurrence and development of diseases. They underpin strategies aimed at prevention and management of diseases and their risk factors.

Although national monitoring systems and health indicators have been developed for a number of chronic diseases, these do not yet exist for CKD. This appendix contains a set of potential health indicators and a monitoring framework for chronic kidney disease. They are presented with the hope of stimulating further development of this important issue.

Potential chronic kidney disease indicators

1 Disease incidence and prevalence

1.1 Prevalence rates for chronic kidney disease in:
- general population
- Indigenous population
- people from culturally and linguistically diverse backgrounds.

1.2 Incidence and prevalence rates for treated end-stage kidney disease in:
- general population
- Indigenous population
- people from culturally and linguistically diverse backgrounds.

2 Risk factors for chronic kidney disease and associated complications

2.1 Prevalence rates for obesity (as measured by BMI) in the general population.

2.2 Prevalence rates for diabetes in the general population.

2.3 Prevalence rates for smoking among people with chronic kidney disease and in the general population.

2.4 Prevalence rates for physical inactivity among people with chronic kidney disease and in the general population.

2.5 Prevalence rates for high blood pressure among people with chronic kidney disease and in the general population:

- ≤140 mmHg systolic and/or 90 mmHg diastolic and/or receiving treatment for high blood pressure in the general population
- ≤130 mmHg systolic and/or 85 mmHg diastolic and/or receiving treatment for high blood pressure among people with chronic kidney disease.

3 Chronic kidney disease comorbidities

3.1 Proportion of people with chronic kidney disease who have diabetes.

3.2 Proportion of people with chronic kidney disease who have hypertension.

3.3 Proportion of people with treated end-stage kidney disease who have diabetes.

3.4 Proportion of people with treated end-stage kidney disease who have coronary artery disease.

3.5 Proportion of people with treated end-stage kidney disease who have peripheral vascular disease.

3.6 Proportion of people with treated end-stage kidney disease who have cerebrovascular diseases.

4 Hospital separations for chronic kidney disease

4.1 Hospital separation rates for chronic kidney disease as the principal diagnosis and as an additional diagnosis in:

- general population
- Indigenous population.

4.2 Hospital separation rates for care involving dialysis in:

- general population
- Indigenous population.

4.3 Hospital separation rates for:

- cardiovascular disease as the principal diagnosis and chronic kidney disease as an additional diagnosis
- cardiovascular disease as an additional diagnosis and chronic kidney disease as the principal diagnosis.

5 Mortality

5.1 Death rates for chronic kidney disease as underlying or associated cause of death in:

- general population
- Indigenous population.

5.2 Death rates for:

- cardiovascular disease as the underlying cause of death with chronic kidney disease as an associated cause of death
- cardiovascular disease as an associated cause of death with chronic kidney disease as the underlying cause of death.

5.3 Death rates among people with treated end-stage kidney disease.

6 Screening

6.1 Proportion of people with chronic kidney disease who have annual:

- blood pressure measurement
- urinalysis: microalbuminuria dipstick (or albumin/creatinine ratio) in people with diabetes and proteinuria dipstick in people without diabetes
- GFR measurement (calculated using serum creatinine).

7 Management of kidney replacement therapy

7.1 Management of dialysis:

- prevalence of treated end-stage kidney disease patients receiving dialysis treatment
- proportion of dialysis patients receiving haemodialysis
- proportion of dialysis patients receiving peritoneal dialysis
- 1, 5 and 10 year survival of dialysis-dependent patients.

7.2 Management of kidney transplant:

- incidence rate for kidney transplant
- prevalence rate of functioning kidney transplant
- 1, 5 and 10 year survival rates of grafts
- 1, 5 and 10 year survival rates of patients
- proportion of cadaveric and live donors
- proportion of treated end-stage kidney disease patients on kidney transplant waiting list
- average waiting time for kidney transplant.

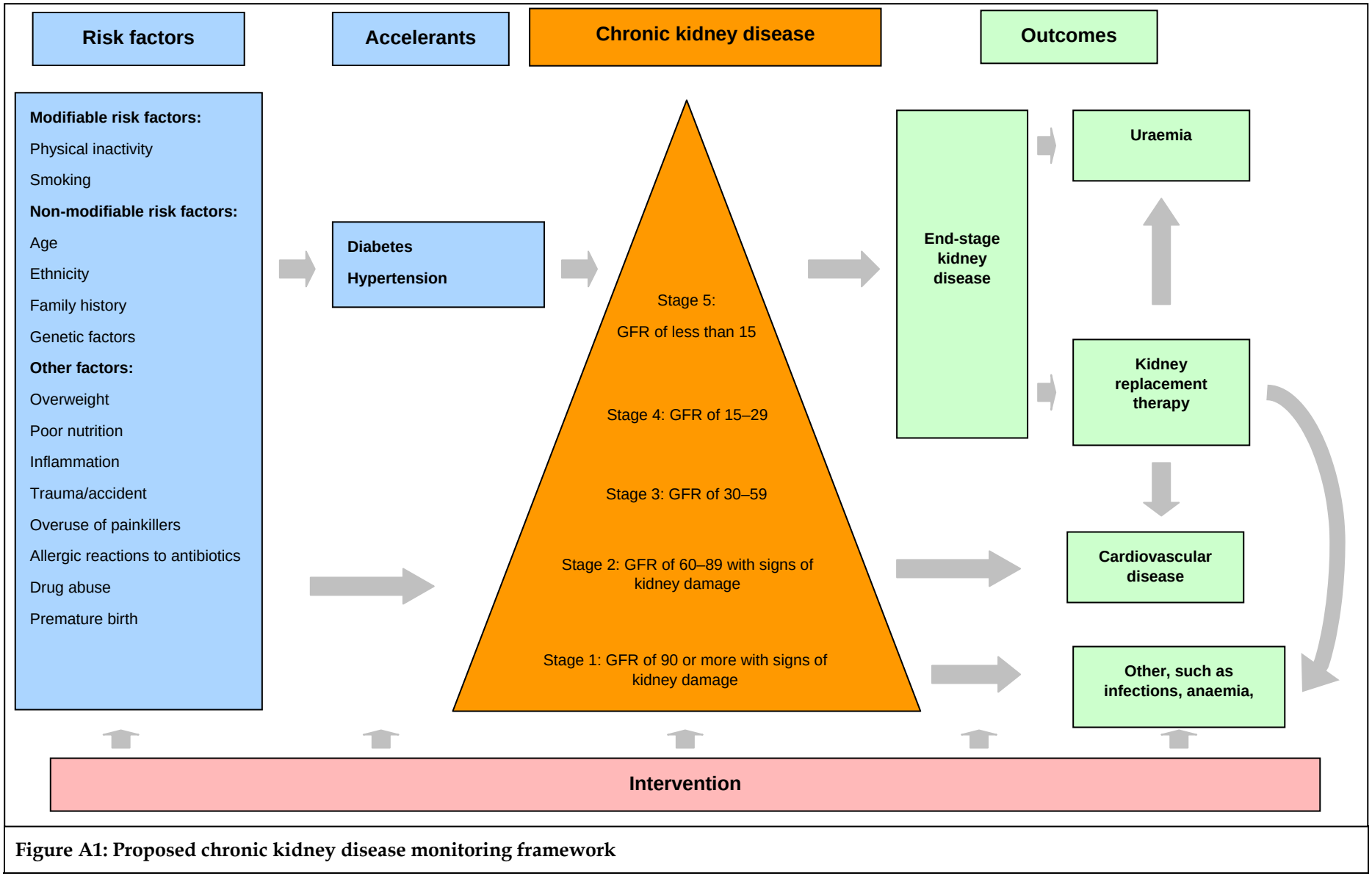


Figure A1: Proposed chronic kidney disease monitoring framework