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.

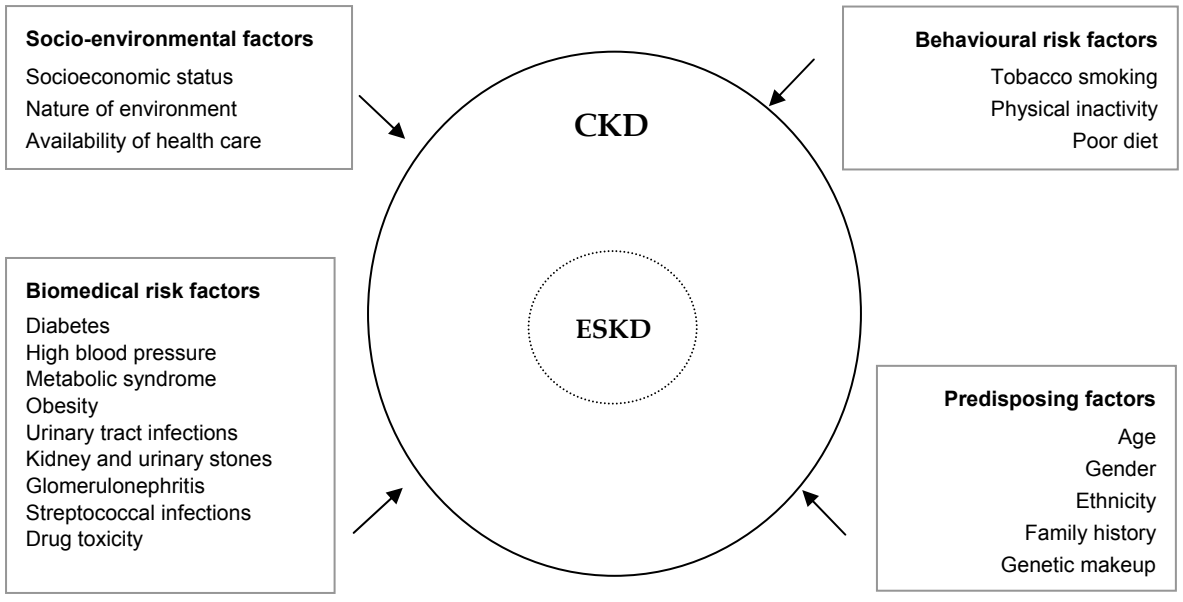


Figure 3.1: Risk factors and determinants of chronic kidney disease

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

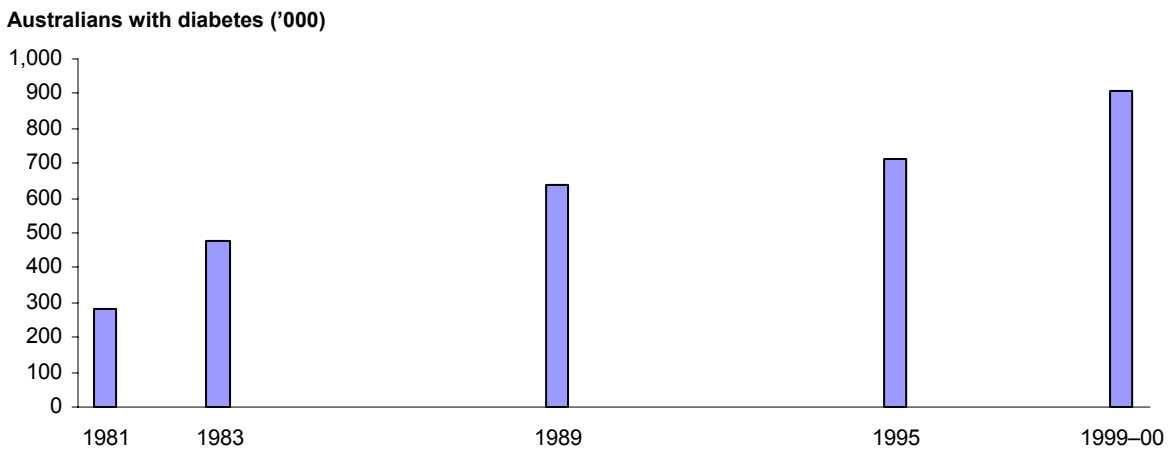
Diabetes mellitus is a long-term condition in which blood glucose levels are too high because the body produces little or no insulin, or cannot use insulin properly. Poorly managed diabetes may result in high blood sugar levels causing damage to the blood-filtering capillaries in the kidneys, a complication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

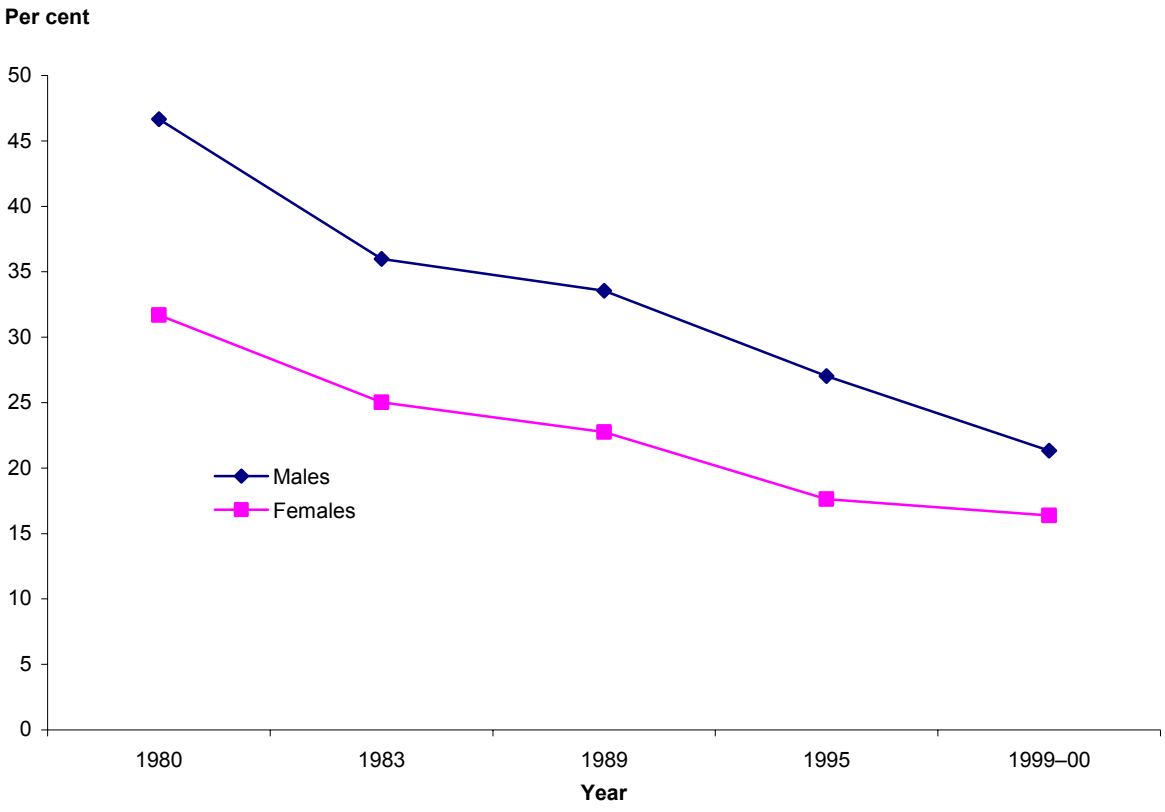
Table 3.2: Prevalence of high blood pressure, people aged 25 years and over, 1999–00

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**

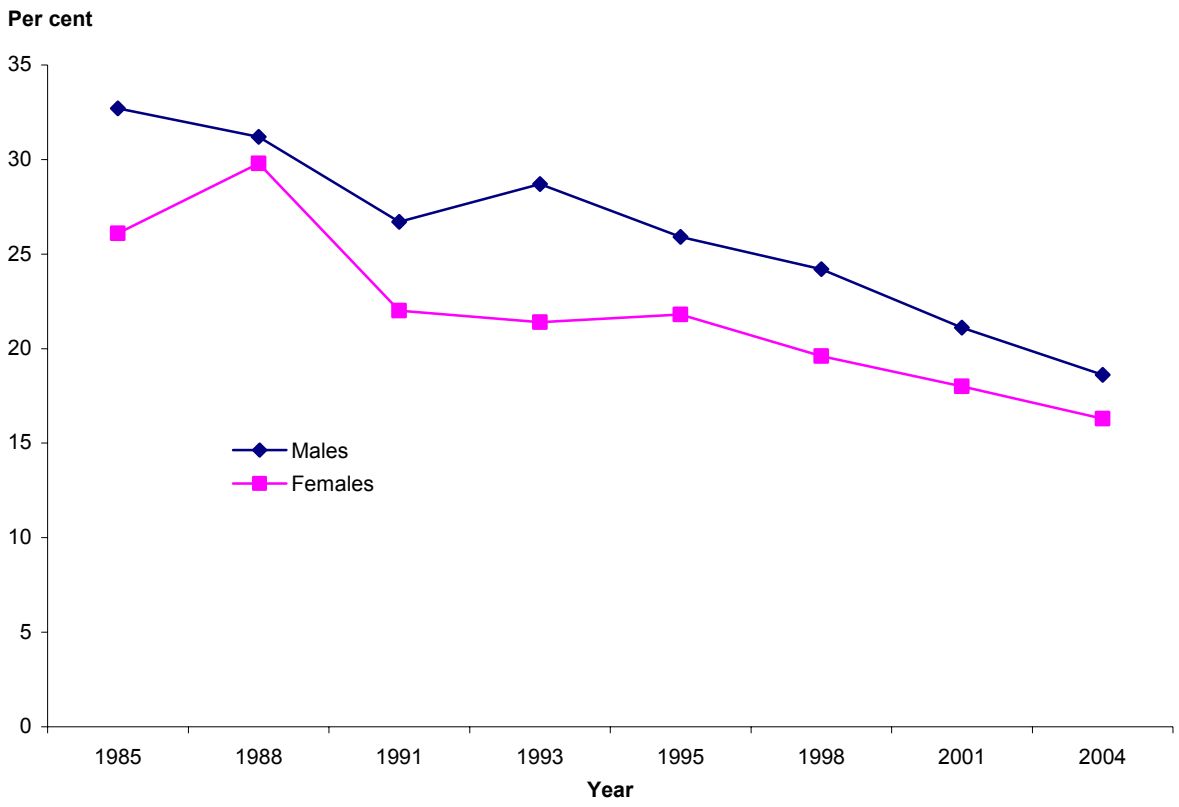
Source: National Drug Strategy Household Survey 2004.

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.

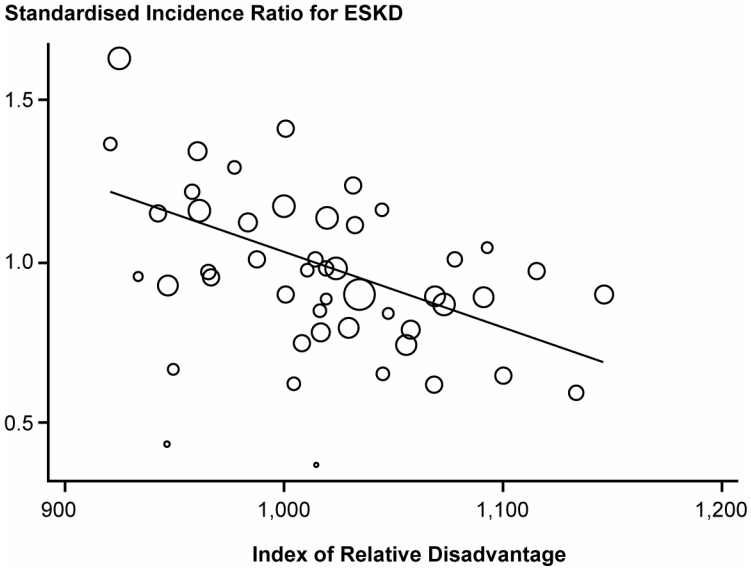
Source: AIHW analysis of the 1980, 1983 and 1989 Risk Factor Prevalence surveys, the 1995 National Nutrition Survey and the 1999–00 AusDiab study.

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.

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