

5 > Osteoporosis

KEY POINTS

- Osteoporosis is a debilitating disease with major health impact through bone fractures. Fractures after minimal trauma are a hallmark of osteoporosis.
- Almost 300,000 Australians are reported to have osteoporosis. The disease is more common in females than males, and is mostly limited to the elderly.
- Around 64,000 hospital separations in Australia every year are for bone fractures in people aged 55 and above. A large proportion of these separations can be attributed to osteoporosis.
- The health costs for the treatment of osteoporotic fractures, associated complications and ongoing care are large and the burden is expected to rise as the population ages.
- A variety of risk factors contribute to the development of osteoporosis, many of which are preventable. Use of bisphosphonates/ appropriate exercise regimes and nutrition can reduce their impact.

Osteoporosis (or porous bones) is the loss of bone density and the deterioration of bone structure, which leads to fragile bones that are prone to fracture. The most common clinical presentation of osteoporosis is fracture after low energy trauma—when a healthy bone would not be expected to sustain fracture—particularly in the hip, spine and wrist. When a fracture occurs, osteoporosis has already been present for several years.

Fractures can impact upon a person's ability to walk unassisted and may lead to loss of independence. A hip fracture almost always requires hospitalisation and major surgery, and may even lead to death. Vertebral fractures can result in a loss of height, cause severe back pain and produce deformity. The health costs for the treatment of osteoporotic fractures, associated complications and ongoing care are large and the burden is expected to rise as the population ages (Wark 1996; AIHW 2002; WHO Scientific Group 2003).

The adverse health impact of osteoporosis can be reduced through early prevention and appropriate management (Seeman & Eisman 2004). In view of this prospect, osteoporosis has been chosen for focused attention in the National Health Priority Areas Initiative on Arthritis and Musculoskeletal Conditions (NAMSCAG 2004).

This chapter describes the health impact of osteoporosis in terms of:

- incidence and prevalence
- impairment and activity limitations
- health care service use, and
- fractures and mortality.

The nature of the problem and risk factors for osteoporosis are also reviewed.

Nature of the problem

Osteoporosis can be viewed at two different levels, namely bone porosity (or the loss of bone mass) and the adverse health outcome of bone fracture. The risk of fracture increases as the bone mass decreases (Riggs & Melton 1992). This inverse relationship between bone porosity and fracture, mediated by external trauma, needs to be regularly monitored.

Bone porosity

The human skeletal system acts as a repository for body minerals such as calcium and phosphorous. These minerals are deposited (bone formation) and absorbed from the bones (bone resorption) as part of healthy bone growth. The process, which continues throughout life, maintains skeletal homeostasis.

Bone production is at its peak during periods of accelerated growth in childhood and adolescence when more minerals are deposited in than absorbed from the bones. Total adult bone mass peaks around the age of 20 at which time the rates of bone formation and resorption are almost balanced (Abrams 2003).

Cortical bone loss starts occurring from about the age of 40–50, at an annual rate of around 0.3–0.5%. The cortical bone forms the hard outer layer, with the trabecular bone providing the inner architecture and strength of the bone. (The terms cortical and trabecular are derived from the Latin for *bark of a tree* and *beam or timber*, respectively). The rate of decline increases in females after menopause.

The diagnosis of osteoporosis can be based on bone mineral density (BMD). BMD is expressed in T-scores, which are standard deviations from the mean BMD in normal young people (Box 5.1). The relation between T-scores and the risk of fracture is computed for each site. For example, the risk of vertebral fractures increases more than two-fold for each unit T-score decrease in BMD (Cummings et al. 1995).

Box 5.1: Diagnostic criteria for osteoporosis using bone mineral density (BMD)

Measurement of BMD (densitometry) is a safe and effective method for diagnosing osteoporosis. Dual-energy x-ray absorptiometry (DEXA or DXA) is recommended and considered the 'gold standard' (Sambrook et al. 2002), but when DEXA is not available (e.g. in more remote areas) quantitative computerised tomography (QCT) is used. Three different categories—osteoporosis, osteopenia and normal bone density—are described:

- **Osteoporosis:** BMD more than 2.5 standard deviations below the mean bone mineral density in young normals (BMD T-score < -2.5)
- **Osteopenia (low bone mass):** BMD value between 1 and 2.5 standard deviations below the mean bone mineral density in young normals ($-2.5 < \text{BMD T-score} < -1$), and
- **Normal bone density:** T-score greater than -1 (at the same site and in the same sex).

Source: WH Source: WHO Scientific Group 1994

Fractures

Bone fracture is a serious outcome of osteoporosis, with long-term consequences of pain and disability. Virtually any bone can fracture if subjected to excessive force but osteoporotic bones fracture occurs with minimal trauma. Fractures sustained following falls from standing height or less are a hallmark of osteoporosis. A large proportion of fractures in people aged 55 or over is osteoporotic in nature.

- Most of the vertebral (spinal) fractures occur without symptoms; almost 70% are clinically undetected. These fractures are often associated with height loss, vertebral deformity (kyphosis) and vertebral compression. Activities such as lifting are a major cause of vertebral fractures.
- Non-vertebral fractures, on the other hand, are painful, associated with swelling and deformity. In particular, hip fractures are highly debilitating and even life-threatening—with almost 30% mortality occurring within 12 months (Woolf & Pfleger 2003). A large proportion of people with hip fractures do not regain their regular posture and mobility (Cumming et al. 1997). Falls are a major cause of these types of fractures.

Risk factors and markers

A range of risk factors contribute to the development of osteoporosis. In addition to age and gender, several environmental, constitutional and lifestyle factors have been associated with the condition. Genetic, hormonal and immunological factors also contribute to variation in bone mass (Gennari et al. 2005). Major known risk factors for osteoporosis are listed in Box 5.2.

Fractures are prominent markers and useful end points for monitoring osteoporosis. It is therefore also prudent to look at factors that increase the risk of fracture. This includes, in particular, the risk of falls and other external causes of injury (Schwartz et al. 2005). A history of fracture after minimal trauma also presages future fractures.

Ageing

Bones lose calcium with age, making them less dense. The hard outer shell of the bone thins out and holes in the honeycomb structure become larger. While they remain the same size on the outside, bones become thinner and weaker on the inside—particularly at the hip, wrist and spine. This decrease in bone mass affects both sexes, though the process accelerates after the age of 50 in females.

Box 5.2: Risk factors for bone loss and fracture**Biomedical and genetic**

- Ageing
- Being post-menopausal
- Family and population history
- Poor vitamin D status
- Low body weight (body mass index < 19 kg/m²)

Behavioural risk factors

- Low calcium intake (<500–850 mg/day)
- Physical inactivity, including long-term immobilisation
- Smoking (current)
- Excessive alcohol consumption

Other medical conditions and disability

- Corticosteroid use
- Reduced lifetime exposure to oestrogen (primary or secondary amenorrhoea or early natural or surgical menopause (<45 years))
- Rheumatoid arthritis
- Malabsorption syndromes, including chronic liver disease and inflammatory bowel disease
- Primary hyperparathyroidism
- Physical disability

Previous history

- Previous fracture after minimal trauma
- Vertebral deformity
- Loss of height and thoracic kyphosis (after radiographic confirmation of vertebral deformities)

Sources: Cumming et al. 1997; AIHW 2002; WHO Scientific Group 2003.

An ageing population will lead to a greater number of people with osteoporosis. In Australia, those aged 65 and over are about 13% of the population. Although this proportion is well below that in Japan (18%), Sweden (17%) and the United Kingdom (16%) it is likely to increase considerably over the next several decades (AIHW 2004a). This demographic shift would increase the overall number of Australians with osteoporosis and its adverse health outcomes.

Being post-menopausal

Low bone mass is linked to decreased levels of oestrogen—which plays a central role in maintaining and balancing bone mass—following menopause (Sowers & La Pietra 1995). The oestrogen is involved not only in regulating cortical and trabecular bone metabolism but also in retaining peak bone mass (Gennari et al. 2005).

With rare exceptions, all women have experienced menopause by age 55. Most women in Australia, therefore, will spend approximately 30 to 40 years in a post-menopausal state. Later age at menopause reduces the risk of osteoporosis. However, the translation of this biological staging into distribution of oestrogen levels and osteoporosis is not simple.

Several studies indicate that oestrogen replacement prevents or greatly retards the loss of bone mass. According to one study, women younger than 75 on oestrogen therapy for seven years or more had higher bone mass than those who had never received oestrogen (Felson et al. 1993). A randomised controlled trial by the Women's Health Initiative, using fractures as clinical end points, has also reported positive outcomes, confirming the role of oestrogen in reducing osteoporosis (Writing Group for the Women's Health Initiative Investigators 2002).

In 2001, approximately 27% of Australian women aged 50–69 used hormone replacement therapy (HRT), which includes oestrogen, for relief of post-menopausal symptoms and prevention of osteoporosis. The HRT usage is highest in those aged 50–59 (Table 5.1).

Table 5.1: Use of hormone replacement therapy (HRT) by females, ages 18 and over, 2001

Age group (years)	Current usage rate (Per cent)	Time used (Per cent of current HRT users)		
		2–5 years	5–10 years	>10 years
18–29	0.4	21.7	n.a.	n.a.
30–39	0.9	43.9	11.5	n.a.
40–49	8.5	26.6	13.9	12.0
50–59	29.6	26.9	27.1	25.8
60–69	24.6	12.4	22.4	56.8
70+	6.5	8.3	25.4	54.7
Total	10.1	21.8	23.0	33.2

n.a. Not available.

Source: ABS 2002.

Almost one-third of women on HRT had received the therapy for more than a decade; the proportion was greater than one out of two females aged 60 and over. The numbers given in Table 5.1 however exclude females that currently do not use HRT but may have accessed the therapy in the past.

The benefits of HRT for osteoporosis notwithstanding, concerns have been raised about its potential risk for breast cancer and stroke and other problems. For longer term use and for women without severe symptoms the risks appear to outweigh the benefits. The use of HRT for the prevention of osteoporosis is therefore not recommended anymore (NHMRC 2005).

Family and population history

Osteoporosis often runs in families. Daughters of women with vertebral osteoporosis tend to have reduced bone mass. A maternal history of hip fracture doubles the risk of hip fracture for daughters as well as increasing the risk of vertebral deformities in sons (Cummings et al. 1995; Diaz et al. 1997). Those under the age of 50 are four times more likely to be told that they have osteoporosis if they had parents or siblings who had broken a bone (DHS 2002).

Genetic influences account for 70–85% of variation in bone mass density (Nuki et al. 1999). Several genes that maintain skeletal homeostasis have been identified (Eisman 1999). These include genes for cytokines, hormones, hormonal receptors and collagen. However, the identification of genetic pathways that lead to bone loss is confounded by various environmental interactions.

Osteoporosis also shows inter-population variation. Higher bone mass has been noted in the US black and Hispanic populations but the bone mass among Asians is similar to that in the white population (Cumming et al. 1997). Variation in hip fracture incidence has also been noted by race/ethnicity. While some of it is environmental in origin, genetic differences between populations contribute to this variation.

Poor vitamin D status

Vitamin D is an important hormone for the regulation of bone metabolism. There is some agreement that mild vitamin D deficiency stimulates parathyroid hormone secretion, which leads to hyperparathyroidism. Mainly synthesised through the skin from sunlight, vitamin D is often low in elderly or housebound people.

Vitamin D deficiency is common among elderly Australian citizens (Morris et al. 1999). It has been uncovered in women with hip fractures and in nursing home residents (Morris et al. 1984; Stein et al. 1996; Brock et al. 1997). The histological evidence in women with hip fractures has been confirmed by low serum levels of 25-hydroxy-vitamin D (Cummings et al. 1995).

Low body weight

Thinness or small frame is another risk factor for osteoporosis. This contrasts with overweight as a risk factor for osteoarthritis. There is some evidence that people with osteoarthritis do not commonly develop osteoporosis.

Low body weight is more frequent among females in higher age groups (AIHW 2004a). Almost 2.1% of females aged 65–74 reported low body weight during the 2001 National Health Survey. In comparison, a little over 1% of Australian males aged 65 and over had low body weight.

There has been an upward trend in body weight among Australians aged 75 and over (AIHW: Bennett et al. 2004). The proportion of females with low body weight in that age group has declined from 9.0% in 1989–90 to 5.8% in 2001. The decrease was higher among Australian males aged 75 and over (AIHW 2004a).

Dietary and behavioural factors

A variety of dietary and behavioural factors contribute to the development of osteoporosis. Good calcium intake is important in reducing the rate of post-menopausal bone loss. Lack of exposure to sunlight reduces vitamin D levels which, in turn, affect absorption of calcium. Limited weight-bearing activity and poor physical activity increase the susceptibility of bones to fracture. Risky health behaviours such as tobacco smoking and excessive alcohol use also contribute to osteoporosis.

Calcium intake

The metabolic pathways of bone formation and bone loss are strongly influenced by calcium intake. Low intake of calcium has been associated with bone loss. In turn, calcium and vitamin D supplementation has been shown to reduce non-vertebral fractures by up to 40% in elderly persons (Reginster 1995). A greater use of calcium and vitamin D is therefore recommended to prevent bone loss (Morris et al. 1999).

Tobacco smoking

Smokers are known to have lower bone mass than non-smokers. Smoking may impact upon the metabolism of hormones that affect bone strength. Although the level of smoking declines with age, a sizeable proportion of the older population at risk of developing osteoporosis are current smokers.

In the 2001 National Health Survey, 13% of people aged 55 and over reported that they currently smoke (12% were regular, daily smokers). More females than males were current smokers (14% and 12%, respectively). There has been a decline in tobacco smoking in both sexes in that age group over the last two decades (AIHW 2004a).

Alcohol abuse

Alcoholics tend to have a low bone mass but this may be attributed more to general nutritional deficiencies rather than to a specific alcohol effect (Cumming et al. 1997). In contrast, those drinking in moderation tend to have a higher bone mass.

Consumption of alcohol at levels considered to be risky for health is high among elderly Australians. In the 2001 NHS, 7.0% of Australian males aged 65–74 reported alcohol consumption at health risk levels; the proportion among Australian females in that age group was 8.0%. These proportions are higher than those reported during the 1995 NHS (AIHW 2004a).

Physical inactivity

Low levels of physical activity are associated with increased risk of osteoporosis and fractures. Physical activity is a determinant of peak bone mass. Appropriate physical activity can slow bone mineral loss, help maintain posture and improve overall fitness. Choosing the right exercises and performing them correctly can help treat and prevent osteoporosis. Weight-bearing physical activity, in particular, is important for maintaining bone mass (Forwood & Larsen 2000).

A large proportion of Australians aged 55 and over report 'sedentary/very low' (including no physical activity) and 'low' levels of physical activity (Table 5.2). There has been very little change in these proportions since 1989–90.

Table 5.2: Level of physical activity, ages 55 and over, 2001

Level of physical activity	Males		Females	
	Number '000	Per cent	Number '000	Per cent
Sedentary (including no exercise)	663.3	34.8	839.6	39.7
Low	625.2	32.8	769.8	36.4
Moderate	564.4	29.7	475.1	22.4
High	50.6	2.7	32.8	1.5
Total	1,903.5	100.0	2,117.3	100.0

Source: ABS 2002.

Other medical conditions

Several systemic illnesses affecting bone metabolism increase the risk for osteoporosis. These include malabsorption syndrome, chronic renal disease, metastatic cancer, thyrotoxicosis and rheumatoid arthritis.

Two metabolic disorders (in particular, hyperparathyroidism and hypogonadism) are associated with osteoporosis. Hyperparathyroidism involves excessive production of parathyroid hormone that increases blood calcium level by its reabsorption from bones. In hypogonadism, decreased or absent secretion of gonadal hormones causes increased loss of bone mass (O'Neill 1997). Loss of ovarian function, premature ovarian failure and amenorrhoea are also associated with bone loss.

Osteoporosis may also occur with certain drug treatments, in particular chronic corticosteroid use (Nuki et al. 1999). The fracture risk appears to be dependent on the dose of oral prednisolone: people receiving a dose of 7.5 mg per day or more are at higher risk of both vertebral and non-vertebral fractures (van Staa et al. 2000).

Physical disability

People with existing physical disabilities have a greater risk of developing osteoporosis because they are less likely to build and maintain bone mass through weight-bearing activities. They are also more likely to use medications that contribute to the loss of bone mass. Younger women with disability have been found to have seven times the rate of osteoporosis than those without disability (Nosek et al. 1997).

Previous history

A history of bone fracture after minimal trauma is a good marker of osteoporosis. Increased risk of a future fracture is associated with prior fracture(s) sustained at any site. A history of vertebral fractures increases the risk of further vertebral fractures five-fold compared with no such history (Lindsay et al. 2001). Vertebral fractures also indicate increased risk of future non-vertebral fractures.

Bone size and bone quality are also important in fracture risk. A longer hip axis length raises the risk of hip fracture, independent of BMD.

Incidence and prevalence

Measuring the incidence and prevalence of osteoporosis is notoriously hard, as the disease is usually not detected until a fracture occurs and the person has presented to a general practitioner (GP) or hospital Emergency Department. Direct estimation of osteoporosis incidence/prevalence is possible through bone densitometry at regular intervals but there are no national data based on this measurement. However, a variety of indirect epidemiological measures, based on falls and fractures, can be used to try to gauge the extent of the problem. These include minimal trauma fractures, hospital separations for fractures, and the diagnosis of osteoporosis after the event of fracture.

Incidence

No national data, based on bone densitometry or fractures, are available to estimate the incidence of osteoporosis. Some regional information on the incidence of osteoporotic fractures has been generated by three prospective studies (Jones et al. 1994b; Cooley & Jones 2001; Sanders et al. 1999b).

According to these studies, the incidence of minimal trauma fractures varies among males from 12 to 19 per 1,000 person years and among females from 19 to 32 per 1,000 person years. These estimates translate to between 51,000 and 73,000 new cases of osteoporotic fractures each year nationwide.

Prevalence

Information as to how many people in Australia currently have osteoporosis comes from two different sources, namely:

- distribution of bone density in the population, and
- self-reports from population health surveys.

There is, as expected, a significant difference in the prevalence of osteoporosis based on BMD distributions and self-reports. While BMD distribution provides a more objective view of the prevalence of osteoporosis, self-reports are more often than not based on the diagnosis of osteoporosis after a fracture is sustained. The two estimates therefore may not be comparable.

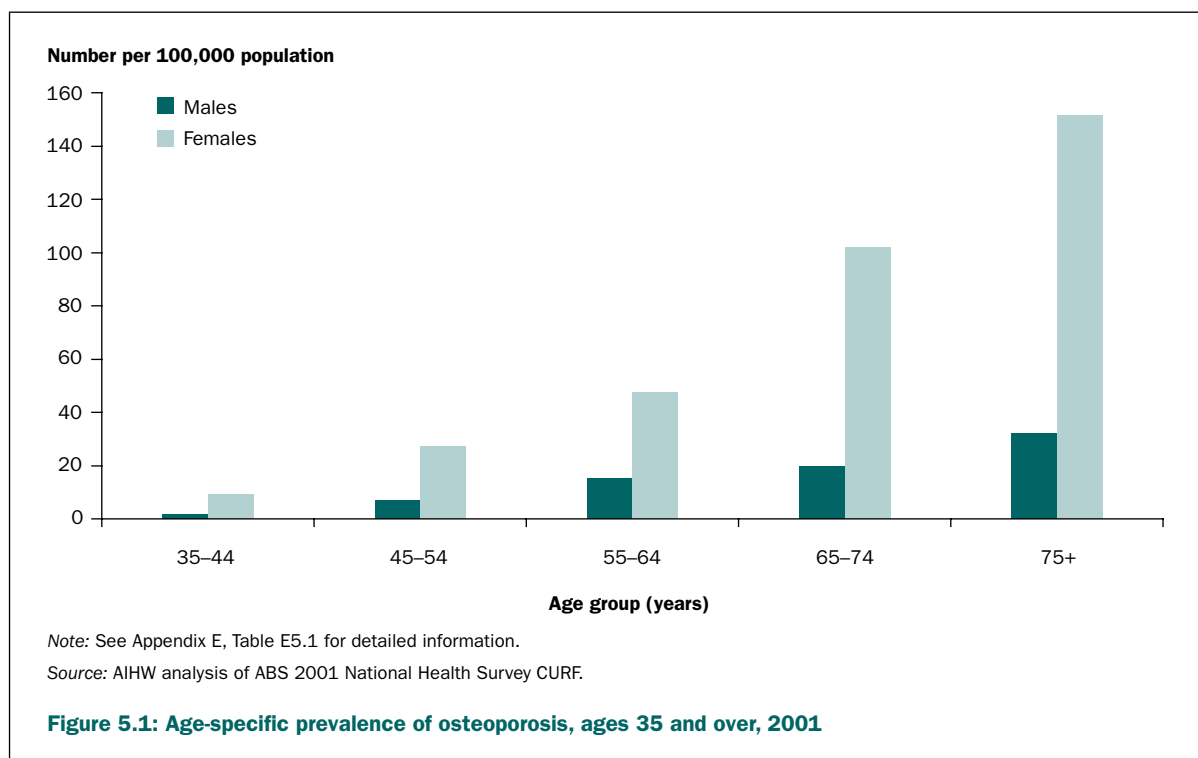
Bone densitometry

BMD is generally not measured at the population level, but rather for those considered at high risk. Regional studies such as the Dubbo Osteoporosis Epidemiology Study and Geelong Osteoporosis Study provide some insight into the age-specific prevalence of low BMD. No national data are available on the distribution of BMD in Australia.

The Geelong Osteoporosis Study, using WHO-approved cut-off levels for BMD, found the prevalence of osteoporosis among Australian females to be high, increasing from the age of 40–44. About 90 out of 1,000 females, aged 50–54, have low BMD (defining osteoporosis) in either the spine, femoral neck (hip) or mid-forearm. The proportion increases more than four-fold to 380 per 1,000 females aged 60–64, and to 560 per 1,000 females aged 65–69. Among those aged 80 or more, the prevalence rate rises to 870 per 1,000 females (Henry et al. 2000).

Self-reports

Estimates of the prevalence of osteoporosis, based on 2001 NHS self-reports, indicate that around 300,000 people, at a rate of 16 per 1,000 persons aged 15 and over, had osteoporosis (ABS 2002). As expected, there was a strong relationship of osteoporosis prevalence with age (Figure 5.1); almost 79% of these people were aged 55 and over. Females reported osteoporosis four times more often than males.



The prevalence of osteoporosis estimated from the 2003 Survey of Disability, Ageing and Carers (SDAC) is quite close to that obtained from the 2001 NHS at 17 per 1,000 persons aged 15 and over. This similarity in estimates from the two surveys is coincidental, as the enumeration of health conditions in SDAC is associated with the presence of disability (AIHW 2004b). Besides, the SDAC numbers include institutionalised people, among whom the condition is likely to be more prevalent, whereas the NHS estimates are community-based only.

The self-report estimates of osteoporosis from both 2001 NHS and 2003 SDAC are much lower than those obtained through the South Australian Omnibus Surveys, conducted between 1995 and 2001 (DHS 2002). These surveys indicate much higher prevalence of osteoporosis—at 48 per 1,000 persons aged 15 and over in 2001 (Table 5.3). The overall prevalence in that age group declined to 37 per 1,000 persons from 1995 to 2001. These South Australian rates are almost double the national prevalence rates, based on 2001 NHS, among people in that age group.

Self-reporting significantly underestimates the prevalence of osteoporosis because of the lack of knowledge about osteoporosis in the general community and the low rates of diagnosis of asymptomatic osteoporosis (Phillipov et al. 1998). However, the considerable differences in prevalence rates between the two national surveys and the South Australian Health Omnibus Surveys are more likely to be due to differing sampling strategies and population bases.

Table 5.3: Prevalence of osteoporosis in Australia, various years

Source	Method	Age group	Year	Prevalence rate (per 1,000 persons)		
				Males	Females	Persons
National Health Survey	Self-reports	15 years+	2001	5	26	16
Survey of Disability, Ageing and Carers	Self-reports	15 years+	2003	17
South Australian Health Omnibus Survey	Self-reports	15 years+	2001	48

.. Not applicable.

Sources: AIHW analysis of ABS 2001 National Health Survey CURF and ABS 2003 Survey of Disability, Ageing and Carers CURF; DHS 2002.

Impairments and activity limitations

Osteoporosis is a major cause of both acute and chronic disability (Fink et al. 2003). In its wake, people suffer the pain and disability of fracture that can lead to loss of independence and an early nursing home admission. Note that little, if any, impairment or activity restriction is attributed to osteoporosis until a bone fractures.

Functional and activity limitations due to osteoporotic fractures are highly variable in severity and chronicity, ranging from none to more than six months. The acute pain following fracture may last a few weeks as the bone heals, but in several cases the fracture may lead to long-term activity limitation. In some cases, the pain may also become chronic. A significant proportion of people with fractures require long-term care.

The impact of certain osteoporotic fractures may be severe, even profound. Almost half of those who fracture a hip will be permanently disabled and not regain their former independence (Johnell 1997). About 40% of people are unable to walk independently one year after hip fracture, about 60% have difficulty with at least one essential activity of daily living, and about 80% are limited in activities such as driving and shopping (Boonen et al. 2004). Substantial disability is also reported after fractures of thoracic vertebrae, the humerus, forearm, ankle and foot (Fink et al. 2003).

According to the SDAC, 58,600 persons reported osteoporosis as their main disabling condition in 2003 (see Chapter 2). Almost half of those with disability associated with osteoporosis (22,994 out of 51,133 persons) had a severe or profound core-activity limitation. These people needed assistance with one or more activities of daily living, such as self-care, mobility and communication.

The proportion of severe or profound core-activity restriction varies with the type of impairment and age. Incomplete use of feet or legs, incomplete use of arms or fingers, disfigurement or deformity and difficulty gripping or holding things—impairments that are prominent in people with osteoporosis—contribute greatly to core-activity restrictions. Almost one out of five people with disability associated with osteoporosis requires assistance more than three times a day (AIHW 2004b).

Current service use

The treatment for osteoporosis usually begins when the condition has weakened the bone to such an extent that a minimal trauma fracture is sustained. Most often, the condition is detected only when a person first presents to their general practitioner (GP) or hospital Emergency Department with a fracture.

Osteoporosis is not a common diagnosis in hospital separations; nonetheless, it is a large contributor to hospitalisation for a variety of treatments and procedures. Most prominent among these are fractures that require immediate attention. Innovations in surgical techniques and biomedical devices have greatly increased the treatment options available to people with fractures. Nonetheless, most of these procedures can be performed only in a hospital setting.

Treatment and management

The treatment options for established disease can be grouped into two classes: Class I agents that either impair bone resorption and/or reduce activation frequency and Class II agents that increase bone formation (Box 5.3).

Box 5.3: Potential treatments for established osteoporosis

Class I: Impair bone resorption and/or reduce activation frequencies

- Hormone replacement therapy (HRT)
- Calcitonin
- Bisphosphonates
- Anabolic steroids
- Calcium
- Vitamin D and metabolites

Source: Kanis et al. 2002.

Class II: Stimulate bone formation

- Intermittent parathyroid injections

Bisphosphonates, such as alendronate and risedronate, are safe and effective agents for the treatment and prevention of osteoporosis. They increase bone mass, and in patients with established osteoporosis reduce the risk of vertebral fractures. They also reduce the risk of hip and other non-vertebral fractures. In combination with oestrogen, bisphosphonates produce greater gains in bone mass; the greater benefit of combination therapy on fracture risk, however, is not clear (Watts 2001).

Calcium treatment, the second most commonly prescribed treatment/prevention modality, is efficacious in populations with low calcium intake. Calcium, as described earlier, is an essential nutrient for the prevention and treatment of osteoporosis. The treatment, however, does not completely arrest post-menopausal bone loss but it does slow the rate of decline by 30 to 50% (Reid 1996).

General practice visits

Osteoporosis accounted for only 0.6% of all problems managed by GPs during the 2003–04 BEACH sampling period. Nonetheless, treatment and management of a large proportion of cases usually begins with a visit to a GP. The person may have sustained an injury from a low fall, or be experiencing ongoing and unexpected pain after bracing themselves with their hands when they tripped.

A large proportion of GP encounters in relation to osteoporosis are for medication only. More than 96% of those who visit their GP with osteoporosis are prescribed medication. Other forms of GP management of osteoporosis included imaging tests, particularly densitometry and x-rays of the spine and thorax, and pathology tests for calcium levels and full blood counts.

Medications prescribed

Alendronate was the most common medication prescribed or advised by GPs, followed by calcium carbonate. Other commonly prescribed/recommended medications for osteoporosis by GPs are listed in Table 5.4.

Table 5.4: Commonly prescribed medications for osteoporosis by general practitioners, 2003–04

Type of medication	Prescriptions/medications	
	Number reported	Per cent
Alendronate	281	35.0
Calcium carbonate	132	16.5
Risedronate sodium	86	10.7
Raloxifene	52	6.5
Calcitriol (vitamin D analogue)	30	3.7
Nandrolone	26	3.2
Ergocalciferol (vitamin D analogue)	19	2.4
Paracetamol	15	1.9
Other medications	152	20.1
Total	793	100.0

Source: AIHW BEACH data.

GP visits provide a good opportunity to identify the patient as being in a high-risk category for osteoporosis, and to suggest preventive health behaviours such as increased calcium intake and regular exercise. The aim of primary care should be to prevent bone loss in order to decrease the risk of fractures. Today there are many therapeutic options, and safe and effective pharmacological treatments to reduce the risk of fracture (Reid 1996).

Specialist services

The most common referral for osteoporosis by GPs was to an endocrinologist. Other specialists seen by persons with osteoporosis included orthopaedic surgeons, rheumatologists, neurosurgeons and pain specialists.

Hospital use

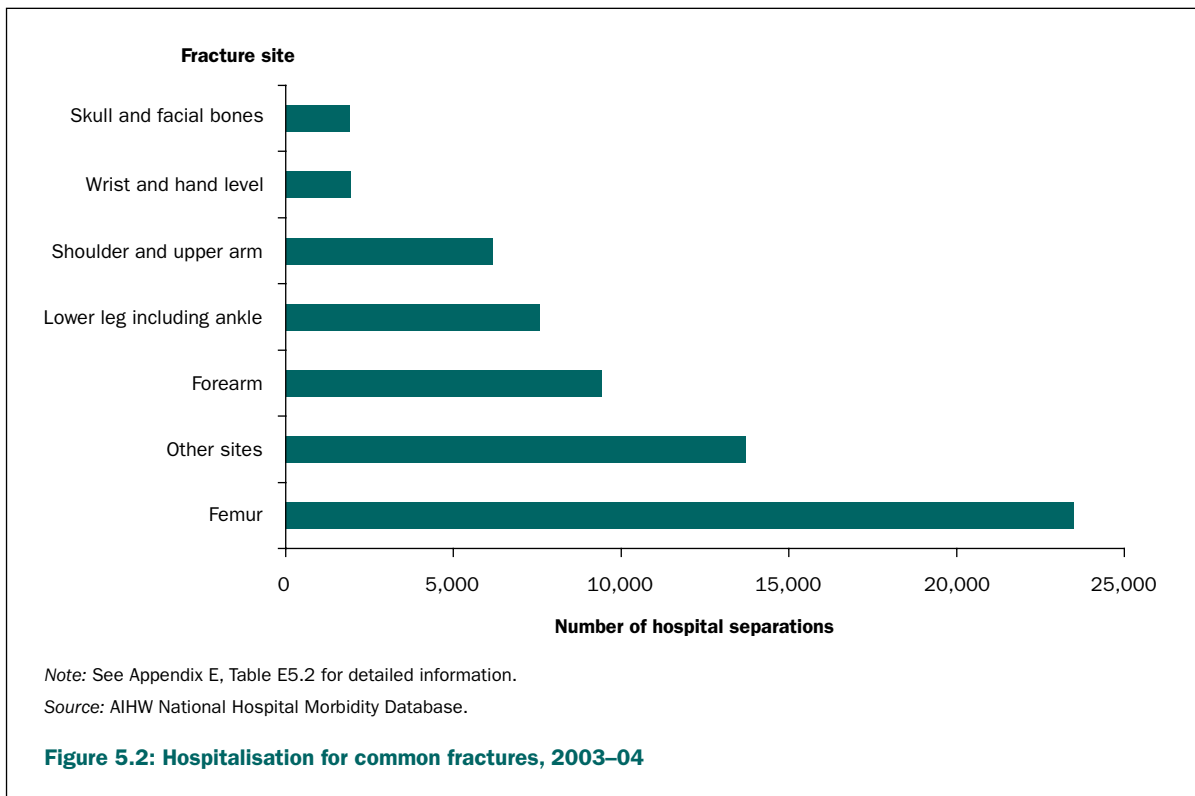
Osteoporosis is not a common principal diagnosis in hospital separations. However, if applicable, procedures are reported in relation to this particular diagnosis. It is more often listed as an additional diagnosis in relation to fractures. Two different measures are used to provide insight into the problem, namely separation rate per 1,000 persons and average length of stay (ALOS) in the hospital.

In 2003–04, osteoporosis was, in itself, the principal diagnosis for only 6,892 separations (of people aged 55 and over), in both public and private hospitals. It was also listed as an additional diagnosis in 4,122 separations of people in that age group, with fracture as the principal diagnosis. The age-standardised separation rate for the latter diagnoses was less than one per 1,000 persons with an ALOS of 13.9 days.

Fracture-related separations and osteoporosis

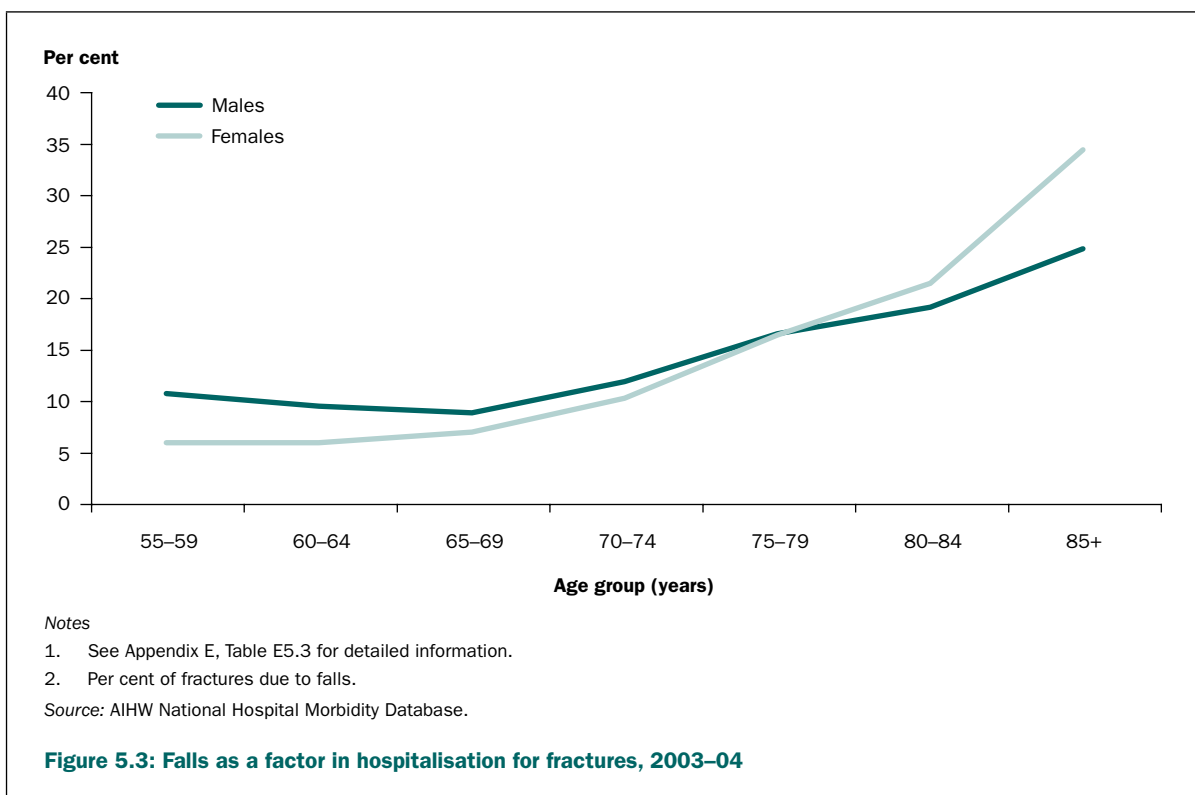
Not all hospital separations for fractures have osteoporosis as an additional diagnosis. It is therefore prudent to look at all hospital separations for fractures among those aged 55 and over. A large proportion of these fractures are likely to be due to osteoporosis.

- A total of 64,173 separations for fractures were recorded in 2003–04, a separation rate of 14 per 1,000 persons. More than half of the separations were of those aged 80 and over; the proportion was higher in females.
- Hip fracture (fracture of the femur) was the most common reason for hospitalisation, followed by fracture of the forearm and the lower leg (Figure 5.2). Hip fractures constituted more than 37% of all fracture separations among those aged 55 and over; the proportion increased to 55% among those aged 85 and over.
- The ALOS for separations in relation to these fractures was 8.0 days, but was higher for fracture of the neck of the humerus (11.1 days), fracture of the femur (12.8 days) and fracture of the pubis (13.4 days).



Falls and fractures

The role of falls in fractures that require hospitalisation shows much age- and sex-related variation (Figure 5.3). More than 70% of hospital separations for fractures among those aged 55 and over have falls listed as the external cause of injury. The proportion exceeds 80% among those aged 85 and over. The ratios have not changed much over the past five years.



Procedures

A large proportion of people hospitalised for osteoporotic fractures require some form of procedure. Of the 10 most frequently reported procedures performed on people with osteoporotic fractures, physiotherapy, occupational therapy and social work were the most common forms of generalised health intervention. Surgery of specific fracture sites was performed in 5% of cases.

Visits to other/allied health professionals

Allied health services are an integral component of the management of osteoporosis. According to the 2001 NHS, about 22% of people with osteoporosis had consulted an allied or other health professional within the previous two weeks of the survey. The allied or other health professionals most frequently consulted were chemists (6%), followed by physiotherapists/hydrotherapists, chiropodists/podiatrists, chiropractors and nurses, each accounting for 3% of the consultations.

Mortality

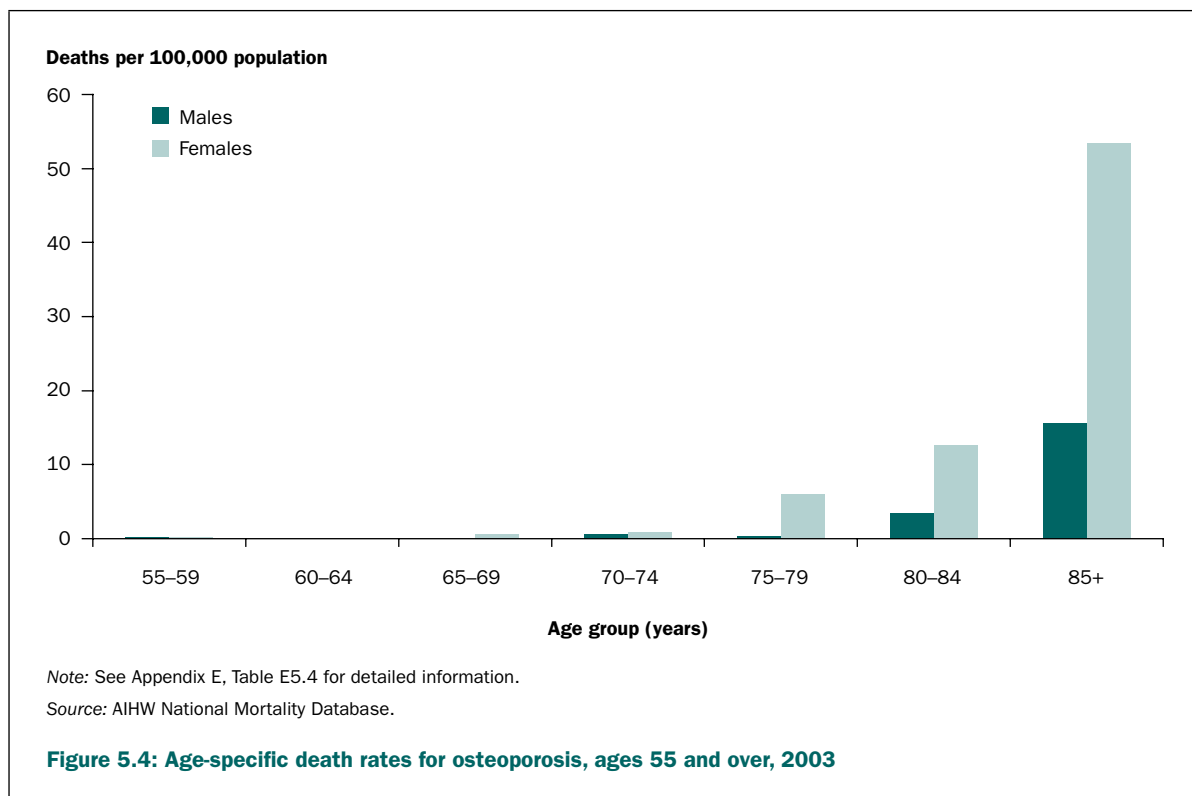
The contribution of osteoporosis to mortality mostly manifest in fractures, especially of the hip, vertebrae and wrist, and their sequelae (Sanders et al. 1999a). The mortality attribution for osteoporosis in other clinical forms is difficult.

Several authors have recommended the use of attributable fractions to map mortality due to osteoporosis (Harris et al. 1998; AIHW: Mathers et al. 1999). Data on multiple causes of death, available in Australia since 1997, provide some insight into the issue.

The contribution of osteoporosis to events leading to death may not be fully appreciated as osteoporosis is generally viewed as a non-fatal condition. It is not commonly listed as the underlying cause of death. Its listing as an associated cause of death has not been fully validated either.

Osteoporosis as the underlying cause of death

Osteoporosis was listed as the underlying cause of 180 deaths (23 male, 157 female) in 2003, at ages 55 and over, with an age-standardised death rate of 3.9 per 100,000 persons. The rate increased exponentially with age, rising an order of magnitude among those aged 85 and over (Figure 5.4).



Osteoporosis as an additional cause of death

Osteoporosis was listed as an additional cause for 1,303 deaths (235 male, 1,068 female) at ages 55 and over, mostly with another chronic disease listed as the underlying cause of death. Ischaemic heart disease, followed by chronic obstructive pulmonary disease (COPD), was the topmost underlying cause of death in these cases in 2003 (Table 5.5). Stroke, other heart diseases and dementia were other major underlying causes of death. It may be noted that these diseases accounted for more than 56% of all osteoporosis-related deaths.

Table 5.5: Osteoporosis as an additional cause of death, ages 55 and over, 2003

Underlying cause of death	Number of deaths	Per cent of deaths
Ischaemic heart disease	272	20.9
COPD	163	12.5
Stroke	142	10.9
Other heart diseases	117	9.0
Dementia and related disorders	56	4.3
Diabetes	38	2.9
Lung cancer	34	2.6
Other causes	481	36.9
Total	1,303	100.0

Source: AIHW National Mortality Database.

The relatively high ranking of COPD as an underlying cause of death in conjunction with osteoporosis underscores the role of common risk factors such as smoking in bone loss. It would also be related to immobility and muscle weakness in people with COPD and the use of corticosteroids. Osteoporotic fractures are common in advanced cases of COPD. These fractures may cause significant morbidity such as pain, worsened respiratory function, decreased mobility and mortality (Biskobing 2002).

Mortality associated with hip fractures

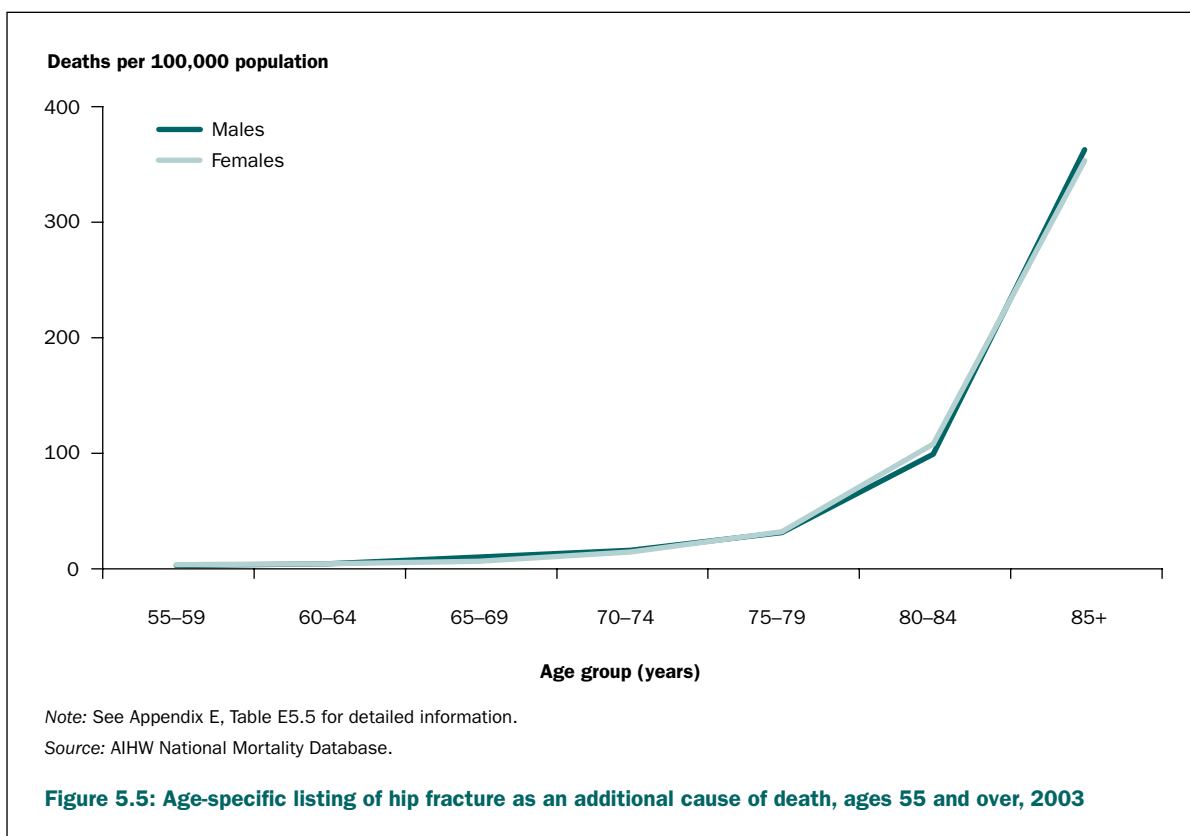
One of the largest causes of mortality due to osteoporosis is hip fracture. Mortality within 12 months of a hip fracture is estimated to be around 30%; the rates are higher in older populations (Woolf & Pfleger 2003). Time series of hip fracture mortality can be used as a proxy to monitor trends in mortality due to osteoporosis.

Hip fracture mortality is often due to blood clots, pneumonia or infection. Other sequelae of hip fractures, some of which are preventable, also contribute to the high death rate. It is, however, not clear how much of this mortality can be attributed to actual hip fracture and its sequelae and how much to the general poor health of many frail older Australians who suffer a hip fracture (Harris et al. 1998).

In Australia, hip fracture mortality within 12 months of the fracture has been estimated to be 23.8% (March et al. 1996), about five times greater than in an age-matched group who do not suffer hip fracture (DHFS 1997). Inpatient hip fracture mortality is estimated to be around 5% (Lord 1993; Boufous et al. 2004); the mortality is 22% within 12 months of fracture (Katelaris & Cumming 1996). However, not all hip fractures are due to osteoporosis (Jones et al. 1994a). The attributable fraction for osteoporosis in hip fracture has been estimated to be around 0.47 among those aged 65 and over (Seeley et al. 1995).

Hip fracture as an additional cause of death

Hip fracture is invariably listed as an additional rather than underlying cause of death. In 2003, it was an additional cause in 1,681 deaths at ages 55 or more. The sex ratio (female: male) in numbers was close to 2:1, but the age-specific rates did not differ very much at all between the two sexes (Figure 5.5).



Exposure to an unspecified factor was the predominant underlying cause of death (32.9%) in cases where hip fracture was listed as an additional cause of death. This suggests a high degree of uncertainty in ascertaining hip fracture mortality. Ischaemic heart disease, stroke and dementia were the other major underlying causes of death, reflecting advanced age and probably poor pre-fracture health in many cases (Table 5.6).

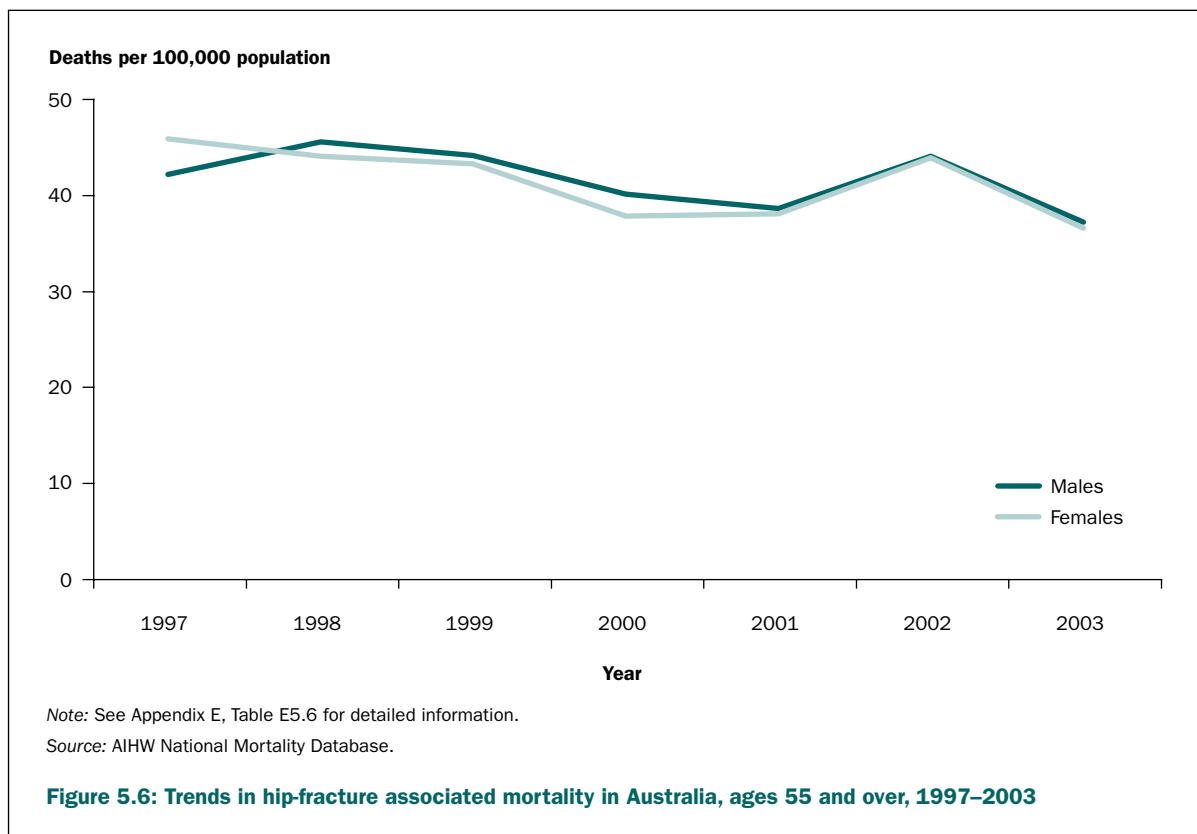
Table 5.6: Hip fracture as an additional cause of death, ages 55 and over, 2003

Underlying cause of death	Number of deaths	Per cent of deaths
Exposure to an unspecified factor	553	32.9
Ischaemic heart disease	293	17.4
Stroke	105	6.2
Other heart diseases	101	6.0
Dementia and related disorders	72	4.3
COPD	49	2.9
Lung cancer	27	1.6
Other causes	481	28.6
Total	1,681	100.0

Source: AIHW National Mortality Database.

Hip fracture as an additional cause of death has shown limited variation in listing over the period for which data on multiple causes of death are available in Australia. The listing rate varied between 36.6 and 44.5 per 100,000 persons, ages 55 and over, between 1997 and 2003 (Figure 5.6).

Not all hip fracture mortality described above can be attributed to osteoporosis. The proportion varies with the type of hip fracture (intracapsular, intertrochanteric or subtrochanteric) but is taken on average to be 0.47 (Seeley et al. 1995). By applying this aetiological fraction, the number of hip fracture deaths attributed to osteoporosis stands at 790.



Inpatient deaths

Additional insight into mortality in relation to the osteoporosis/hip fracture axis can be gleaned from hospital separations data. Since persons with hip fracture are invariably hospitalised, hip fracture inpatient death rate can be measured accurately.

A total of 5,236 inpatient deaths occurred in Australian public and private hospitals over the period 1997–98 to 2002–03 with hip fractures as the principal diagnosis. There were 63,598 separations for hip fracture during that five-year period (excluding transfers to other hospitals), with an inpatient death rate of 81.9 per 1,000 separations. The rate increased from 77.3 to 84.7 per 1,000 separations during the five-year period.

Not all inpatient hip fracture mortality can be attributed to osteoporosis. By applying the aetiological fraction for osteoporosis in hip fractures (0.47; Seeley et al. 1995), the annual average of inpatient osteoporotic hip fracture deaths in Australia is about 492.

This number is clearly an underestimate of hip fracture mortality attributable to osteoporosis. A more reasonable period within which to attribute deaths due to hip fracture is 12 months.

Attribution of hip fracture mortality

Another insight into osteoporotic hip fracture mortality can be obtained by using death within 12 months as the cut-off point. There were, on average, 12,792 valid hip fracture separations between 1997–98 and 2002–03 in public and private hospitals every year. Using the Katelaris and Cumming (1996) estimate of the 12-months hip fracture mortality (22%), and the Seeley et al. (1995) estimate of osteoporotic fraction of hip fractures (47%), the number of deaths attributed to osteoporotic hip fracture is estimated to be 1,323.

References

- Abrams SA 2003. Normal acquisition and loss of bone mass. *Hormone Research* 60 (Supplement 3): 71-6.
- ABS (Australian Bureau of Statistics) 2002. 2001 National Health Survey: summary of results. ABS Cat. No. 4364.0. Canberra: ABS.
- AIHW (Australian Institute of Health and Welfare) 2002. Chronic diseases and associated risk factors in Australia, 2001. AIHW Cat. No. PHE 33. Canberra: AIHW.
- AIHW 2004a. Australia's health 2004. AIHW Cat. No. AUS 44. Canberra: AIHW.
- AIHW 2004b. Disability and its relationship to health conditions and other factors. AIHW Cat. No. DIS 37. Canberra: AIHW.
- AIHW: Bennett S, Magnus P & Gibson D 2004. Obesity trends in older Australians. AIHW Cat. No. AUS 42. Canberra: AIHW.
- AIHW: Mathers C, Vos T & Stevenson C 1999. The burden of disease and injury in Australia. AIHW Cat. No. PHE 17. Canberra: AIHW.
- Bhalla AK 1993. Osteoporosis and osteomalacia. In: Maddison PJ, Isenberg DA, Woo P et al. (eds). *Oxford textbook of rheumatology*. Oxford: Oxford University Press, 1005-24.
- Biskobing DM 2002. COPD and osteoporosis. *Chest* 121:609-20.
- Boonen S, Rizzoli R, Meunier PJ et al. 2004. The need for clinical guidance in the use of calcium and vitamin D in the management of osteoporosis: a consensus report. *Osteoporosis International* 15:511-9.
- Boufous S, Finch CF & Lord SR 2004. Incidence of hip fracture in New South Wales: are our efforts having an effect? *Medical Journal of Australia* 180:623-6.
- Brock K, Reid J & Fraser D 1997. Effect of type of accommodation on vitamin D status of the elderly in Australia. In Norman AW, Bouillon TM & Thomasset M (eds). *Vitamin D: chemistry, biology and clinical applications of the steroid hormone*. Riverside: University of California, 885-6.
- Cooley H & Jones G 2001. A population-based study of fracture incidence in southern Tasmania: lifetime fracture risk and evidence for geographic variations within the same country. *Osteoporosis International* 12:124-30.
- Cumming RG, Nevitt MC & Cummings SR 1997. Epidemiology of hip fractures. *Epidemiological Reviews* 19:244-56.
- Cummings S, Nevitt M, Browner W et al. 1995. Risk factors for hip fracture in white women. *New England Journal of Medicine* 332:767-73.
- DHFS (Commonwealth Department of Health and Family Services) 1997. Consensus panel: the prevention and management of osteoporosis. *Medical Journal of Australia* 167:S1-S16.
- DHS (Department of Human Services) 2002. Osteoporosis in South Australia. Centre for Population Studies in Epidemiology Brief Report No. 2002-17. Adelaide: DHS.
- Diaz MN, O'Neill TW & Silman AJ 1997. The influence of family history of hip fracture on the risk of vertebral deformity in men and women: the European Vertebral Osteoporosis Study. *Bone* 20:145-9.
- Eisman JA 1999. Genetics of osteoporosis. *Endocrinology Reviews* 20:788-804.
- Felson DT, Zhang Y, Hannan MT et al. 1993. The effect of postmenopausal estrogen therapy on bone density in elderly women. *New England Journal of Medicine* 329:1141-6.
- Fink HA, Ensrud KE, Nelson DB et al. 2003. Disability after clinical fracture in postmenopausal women with low bone density: the fracture intervention trial (FIT). *Osteoporosis International* 14:69-76.
- Forwood MR & Larsen JA 2000. Exercise recommendations for osteoporosis: a position statement of Australian and New Zealand Bone and Mineral Society. *Australian Family Physician* 29:761-4.
- Gennari L, Merlotti V, De Paola V et al. 2005. Estrogen receptor gene polymorphisms and genetics of osteoporosis: a HuGE review. *American Journal of Epidemiology* 161:307-20.

Harris AH, Cumming R, Watts J et al. 1998. The burden of illness and the cost of osteoporosis in Australia. Centre for Health Program Evaluation Technical Report no. 8. Melbourne: Monash University.

Henry MJ, Paco JA, Nicholson GC et al. 2000. Prevalence of osteoporosis in Australian women (Geelong Osteoporosis Study). *Journal of Clinical Densitometry* 3:261–8.

Johnell O 1997. The socioeconomic burden of fractures: today and in the 21st century. *American Journal of Medicine* 103(2A):20S–25S; discussion 25S–26S.

Jones G, Nguyen T, Sambrook P et al. 1994a. Progressive loss of bone in the femoral neck in elderly people: longitudinal findings from the Dubbo Osteoporosis Epidemiology Study. *British Medical Journal* 39:691–5.

Jones G, Nguyen T, Sambrook P et al. 1994b. Symptomatic fracture incidence in elderly men and women: the Dubbo Osteoporosis Epidemiology Study (DOES). *Osteoporosis International* 4:277–82.

Kanis JA, Brazier JE, Stevenson M et al. 2002. Treatment of established osteoporosis: a systematic review and cost–utility analysis. *Health Technology Assessment* 6(29): 19–60.

Kateleris AG & Cumming RG 1996. Health status before and mortality after hip fracture. *American Journal of Public Health* 86(4):557–60.

Lindsay R, Silverman S, Cooper C et al. 2001. Risk of new vertebral fracture in the year following a fracture. *Journal of the American Medical Association* 285:320–3.

Lord SR 1993. Hip fractures: changing patterns in hospital bed use between 1979 and 1990. *Australian and New Zealand Journal of Surgery* 63:352–5.

March L, Chamberlain A, Cameron I et al. 1996. Prevention, treatment and rehabilitation of fractured neck of femur. Report from the Northern Sydney Area Health Service Fractured Neck of Femur Health Outcomes Project. Cited 5 August 2005. <<http://www.mja.com.au/public/issues/iprs2/march/fnof.pdf>>.

Morris HA, Need AG, Burr M et al. 1984. Vitamin D and femoral neck fractures in elderly South Australian women. *Medical Journal of Australia* 140:519–21.

Morris HA, Need AG & Nordin BGC 1999. The hip fracture threat (editorial). *Medical Journal of Australia* 170:459–60.

NAMSCAG (National Arthritis and Musculoskeletal Conditions Advisory Group) 2004. Evidence to support the National Action Plan for osteoarthritis, rheumatoid arthritis and osteoporosis: opportunities to improve health–related quality of life and reduce the burden of disease and disability. Canberra: DoHA at <<http://www.nhpac.gov.au>>.

NHMRC (National Health and Medical Research Council) 2005. Should I use Hormone Replacement Therapy. Canberra; NHMRC.

Nosek MA, Howland CA, Rintal DH et al. 1997. National study of women with physical disabilities: Final Report. Houston, TX: Center for Research on Women with Disabilities.

Nuki G, Ralston S & Luqmani R 1999. Diseases of the connective tissues, joints and bones. In: Haslett C, Chivers E, Hunter J et al. (eds). *Davidson's principles and practice of medicine*, 18th ed. Edinburgh: Churchill Livingstone, 801–76.

O'Neill S 1997. Osteoporosis guidelines for general practitioners. *Australian Family Physician* 26(1):1181–96.

Phillipov G, Phillips PJ, Leach G et al. 1998. Public perceptions and self–reported prevalence of osteoporosis in South Australia. *Osteoporosis International* 8(6):552–6.

Reginster JY 1995. Treatment of bone in elderly subjects: calcium, vitamin D, fluor, bisphosphonates, calcitonin. *Hormone Research* 43:83–8.

Reid IR 1996. Therapy of osteoporosis: calcium, vitamin D and exercise. *American Journal of Medical Science* 312:278–86.

Riggs BL & Melton LJ III 1992. The prevention and treatment of osteoporosis. *New England Journal of Medicine* 327:620–7.

- Sambrook PN, Seeman E, Phillips SR et al. 2002. Preventing osteoporosis: outcomes of the Australian Fracture Prevention Summit. Co-sponsored by Osteoporosis Australia and the National Prescribing Service. *Medical Journal of Australia* 176(Supplement 8):1-16.
- Sanders KM, Nicholson GC, Ugoni AM et al. 1999a. Health burden of hip and other fractures in Australia beyond 2000: projections based on the Geelong Osteoporosis Study. *Medical Journal of Australia* 170:467-70.
- Sanders KM, Seeman E, Ugoni AM et al. 1999b. Age and gender specific rates of fractures in Australia: a population-based study. *Osteoporosis International* 10:240-7.
- Schwartz AV, Nevitt MC, Brown BW et al. 2005. Increased falling as a risk factor for fracture among older women: the study of osteoporotic fractures. *American Journal of Epidemiology* 161:180-5.
- Seeley DG, Browner WS, Nevitt MC et al. 1995. Almost all fractures are osteoporotic. *Journal of Bone and Mineral Research* 10:S468.
- Seeman E & Eisman JA 2004. *MJA practice essentials 7: treatment of osteoporosis: why, whom, when and how to treat.* *Medical Journal of Australia* 180:298-303.
- Sowers MR & La Pietra MT 1995. Menopause: its epidemiology and potential association with chronic diseases. *Epidemiological Reviews* 17:287-302.
- Stein MS, Scherer SC, Walton SL et al. 1996. Risk factors for secondary hyperparathyroidism in a nursing home population. *Clinical Endocrinology* 44:375-83.
- van Staa T, Leufkens H, Abenham L et al. 2000. Use of oral corticosteroids and risk of fractures. *Journal of Bone and Mineral Research* 15:993-1000.
- Wark JD 1996. Osteoporosis: the emerging epidemic. *Medical Journal of Australia* 164:327-8.
- Watts NB 2001. Treatment of osteoporosis with bisphosphonates. *Rheumatic Disease Clinics of North America* 27:197-214.
- WHO (World Health Organization) Scientific Group 1994. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. WHO Technical Report Series No. 843. Geneva: WHO.
- WHO Scientific Group 2003. The burden of musculoskeletal conditions at the start of the new millennium. WHO Technical Report Series no. 919. Geneva: WHO.
- Wolf AD & Pflieger B 2003. Burden of major musculoskeletal conditions. *Bulletin of the World Health Organization* 81(9):646-56.
- Writing Group for the Women's Health Initiative Investigators 2002. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *Journal of the American Medical Association* 288:321-33.