Australian Government
Australian Institute of Health and Welfare

Authoritative information and statistics to promote better health and wellbeing

CANCER SERIES
Number 66

Cancer incidence projections

Australia, 2011 to 2020

Australian Institute of Health and Welfare
Canberra

Cat. no. CAN 62

The Australian Institute of Health and Welfare is a major national agency which provides reliable, regular and relevant information and statistics on Australia's health and welfare. The Institute's mission is *authoritative information and statistics to promote better health and wellbeing*.

This publication is part of the Australian Institute of Health and Welfare's Cancer Series. A complete list of the Institute's publications is available from the Institute's website <www.aihw.gov.au>.

ISBN 978-1-74249-282-7

Suggested citation

Australian Institute of Health and Welfare 2012. Cancer incidence projections: Australia, 2011 to 2020. Cancer Series no. 66. Cat. No. CAN 62. Canberra: AIHW.

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

Communications, Media and Marketing Unit

Australian Institute of Health and Welfare

GPO Box 570

Canberra ACT 2601

Tel: (02) 6244 1032

Email: info@aihw.gov.au

Published by the Australian Institute of Health and Welfare

Please note that there is the potential for minor revisions of data in this report. Please check the online version at <www.aihw.gov.au> for any amendments.

Contents

Acknowledgments

This report was prepared by staff in the Cancer and Screening Unit of the Australian Institute of Health and Welfare (AIHW). The main authors were Melissa Goodwin (senior analyst) and Dr Brett Davis (statistical analyst). Valuable contributions to research, editing, data checking and graphical representations were made by Theresa Negrello and Paul Leonard. The authors would like to thank AIHW staff who commented on earlier drafts of this report.

This project was carried out under the guidance of Chris Sturrock, Unit Head, and with epidemiological advice from Professor David Roder (Head, Centre for Cancer Control Research, Cancer Council South Australia), Dr Chris Stevenson (Head, Epidemiological Modelling Unit, Monash University) and Dr Tim Threlfell (Principal Medical Officer and Manager, Western Australian Cancer Registry). Statistical advice was provided by Allan Nicholls (AIHW statistical advisor). Advice from Teresa Dickinson (Head, Information and Statistics Group, AIHW) and Lisa McGlynn (Head, Health Group, AIHW) is gratefully acknowledged.

The support of the Australasian Association of Cancer Registries in providing data and reviewing the draft report is also gratefully acknowledged.

The report was produced under the National Centre for Monitoring Cancer (NCMC). The NCMC was established by the AIHW in 2009 with funding from the Australian Government. The NCMC monitors cancer rates and their impacts and outcomes, and identifies and addresses priority gaps and deficiencies in population-level cancer and related data to improve the evidence base. The advice of the Cancer Monitoring Advisory Group, chaired by Professor Jim Bishop, is also acknowledged.

Abbreviations

AACR	Australasian Association of Cancer Registries
ABS	Australian Bureau of Statistics
ACD	Australian Cancer Database
AIHW	Australian Institute of Health and Welfare
ARMA	Autoregressive moving average
ASR	Age-standardised rate
BMI	Body-mass index
CUP	Cancer of unknown primary site
DCIS	Ductal carcinoma in situ
DoHA	Australian Government Department of Health and Ageing
GORD	Gastro-oesophageal reflux disorder
HIV	Human immunodeficiency virus
HPV	Human papillomavirus
HRT	Hormone replacement therapy
IARC	International Agency for Research on Cancer
ICD-O-3	International Classification of Diseases for Oncology 3rd edition
ICD-10	International Classification of Diseases and Related Health Problems 10th edition
MBS	Medicare Benefits Schedule
NCSCH	National Cancer Statistics Clearing House
NBCSP	National Bowel Cancer Screening Program
NCMC	National Centre for Monitoring Cancer
OECD	Organisation for Economic Cooperation and Development
OLS	Ordinary least squares
PI	Prediction interval
PSA	Prostate-specific antigen
WCRF/AICR	World Cancer Research Fund/American Institute for Cancer Research
WHO	World Health Organization

Symbols

–	nil or rounded to zero
. .	not applicable
n.a.	not available
n.p.	not publishable because of small numbers, confidentiality or other concerns about the quality of the data

Summary

This report presents projections of cancer incidence in Australia for 2011 to 2020. The information is important for health service planning and resource allocation in the future. The projections are presented for males and females at the national level for all cancers combined, as well as the most commonly diagnosed cancers.

It is important to note that projections are not intended to function as exact forecasts, but to give an indication of what might be expected if the stated assumptions were to apply over the projected time frame.

Overall picture

The number of cases of cancer diagnosed in Australia is projected to rise over the next decade for both males and females and is expected to reach about 150,000 in 2020—an increase of almost 40% from 2007. Increases in the number of cases diagnosed are due primarily to the ageing and increasing population and are expected to be most evident in older populations.

Cancer incidence in males is highly influenced by prostate cancer, which accounts for about 30% of all cases. Assuming incidence of prostate cancer stabilises in the future, it is projected that the overall age-standardised rate of cancer in males will fall from 595 to about 568 cases per 100,000 males between 2007 and 2020. With the anticipated changes to the population, this equates to about 85,000 new cases expected to be diagnosed in 2020. Conversely, the overall age-standardised rate of cancer incidence in females is projected to rise from 394 to about 408 cases per 100,000 females between 2007 and 2020, which equates to about 65,000 new cases expected to be diagnosed in 2020.

While the total number of cases diagnosed is expected to rise for each of the cancers analysed in this report, changes in underlying incidence rates vary depending on existing trends for each cancer type. Table 1 presents an overview of the expected changes for each cancer type.

Which cancers will present the biggest burden in 2020?

For males, prostate cancer is expected to remain the most common cancer diagnosed in 2020 (25,300 cases), followed by bowel cancer and melanoma of the skin (about 10,800 cases each) and lung cancer (7,500 cases). For females, breast cancer is projected to continue to be the most common cancer diagnosed in 2020 (17,200 cases), followed by bowel cancer (9,200), melanoma (6,800) and lung cancer (6,100).

Which cancers are on the rise?

Age-standardised rates for liver cancer are projected to increase by 38% from 2007 to 2020 in males and 78% in females, while thyroid cancer rates are projected to increase by 33% in males and 62% in females. Increases are also expected in rates for melanoma (30% males; 18% females), testicular cancer (25%) and lung cancer in females (16%).

Which cancers are decreasing?

Age-standardised rates of stomach cancer are expected to fall by 25% from 2007 to 2020 for males and 20% for females. Additionally, bladder (19%), lung (15%) and pancreatic (14%) cancer rates for males are also projected to fall.

Data at a glance

Table 1: Selected cancers projected to be diagnosed in Australia in 2020

Males					Females				
Site/type	**Cases[a]**	**ASR[b]**	**PI (95%)[c]**	**Δ[d]**	**Site/type**	**Cases[a]**	**ASR[b]**	**PI (95%)[c]**	**Δ[d]**
Prostate	25,310	163.5	136.0–191.1	→	Breast	17,210	113.8	109.5–118.1	→
Bowel	10,800	71.4	68.9–74.0	→	Bowel	9,160	53.8	51.7–55.8	→
Melanoma	10,780	74.1	71.3–76.9	↗	Melanoma	6,790	45	43.0–47.0	↗
Lung	7,520	49.3	47.1–51.5	↘	Lung	6,120	36.2	34.6–37.8	↗
Non-Hodgkin lymphoma	3,470	23.5	22.2–24.8	↗	Uterine	2,830	17.6	16.6–18.6	→
Kidney	2,910	19.8	18.8–20.8	↗	Thyroid	2,660	19.8	18.0–21.6	↗
Bladder	2,040	13.4	12.4–14.4	↘	Non-Hodgkin lymphoma	2,480	15.4	14.7–16.1	→
Pancreatic	1,710	11.3	10.5–12.2	↘	Pancreatic	1,750	9.8	9.1–10.5	→
Liver	1,640	11	10.3–11.8	↗	Ovarian	1,640	10.2	9.4–11.0	→
Stomach	1,340	8.9	8.2–9.6	↘	Kidney	1,220	7.7	7.1–8.3	→
Oesophageal	1,270	8.5	7.9–9.1	→	Cervical	915	6.7	6.2–7.3	→
Brain	1,230	8.7	8.0–9.4	→	Brain	825	5.5	5.0–6.0	→
Testicular	1,020	8.5	7.9–9.0	↗	Liver	825	4.8	4.3–5.3	↗
Mesothelioma	945	6.2	5.5–6.9	→	Stomach	740	4.5	4.0–4.9	↘
Thyroid	775	5.7	5.1–6.2	↗	Bladder	755	3.8	3.4–4.2	→
Hodgkin lymphoma	330	2.8	2.4–3.2	→	Oesophageal	515	2.9	2.5–3.3	→
					Hodgkin lymphoma	295	2.5	2.2–2.8	→
					Mesothelioma	240	1.4	1.3–1.6	↗
All cancers[e]	**84,950**	**568.4**	**539.9–596.9**	→	**All cancers[e]**	**65,040**	**408.2**	**401.7–414.7**	↗

(a) Incidence counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5. This does not represent the level of certainty around projected counts.

(b) The projected rates were standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 population.

(c) 95% prediction intervals represent the range of uncertainty around the projected age-standardised rate.

(d) Changes are based on comparisons to 2007 ASRs. Rates are predicted to increase when the entire 95% prediction interval is above the 2007 95% confidence interval. Rates are predicted to decrease when the entire 95% prediction interval is below the 2007 95% confidence interval. No significant change is predicted where the 95% prediction interval and the 2007 95% confidence interval overlap.

(e) Includes cancers coded in ICD-10 as C00–C96, D45, D46, D47.1 and D47.3 with the exception of those C44 codes which indicate a basal or squamous cell carcinoma of the skin.

Source: AIHW Australian Cancer Database 2007 projected to 2020.

1 Introduction

> Prediction is very difficult, especially about the future.
>
> *Niels Bohr: Danish physicist (1885–1962)*

The number of new cases of cancer occurring in Australia each year, referred to as cancer incidence, is an important measure of the burden of cancer in Australia and has implications for resource allocation and planning. Investment decisions in cancer treatment facilities, workforce planning and evaluation of cancer policy rely not only on knowing how many cases of cancer were diagnosed in a given year, but on how many can be expected to be diagnosed in the future.

This report funded by the National Centre for Monitoring Cancer (NCMC) uses past trends to estimate cancer incidence in Australia from 2011 to 2020 for all cancers combined, as well as for a number of selected cancers. Key assumptions and an overview of the methodology used for calculating these projections are in Chapter 2. An examination of past trends and projected incidence counts, age-specific rates and age-standardised rates for each of the most commonly diagnosed cancers are provided in Chapter 3. Appendix B provides technical details of the models used in the projections methodology while Appendix C provides details of all data sources.

In addition, supplementary data are available as online Excel tables at <www.aihw.gov.au>. These tables contain detailed statistics, some of which are presented in summary form in the body of the report.

This report is intended as a companion publication to *Cancer in Australia: an overview, 2010,* which was the source of introductory statistics for each cancer. A copy of this publication is available on the Australian Institute of Health and Welfare (AIHW) website <www.aihw.gov.au>.

Background

In Australia, cancer (except basal cell and squamous cell carcinoma of skin) is a legally notifiable disease. Data relating to diagnosed cancers for individuals are collated by state and territory cancer registries through information provided by hospitals, pathology laboratories, radiotherapy centres, physicians and Registrars of Births, Deaths and Marriages in their respective jurisdiction. These data are supplied annually for national collation into the Australian Cancer Database (ACD) through the National Cancer Statistics Clearing House (NCSCH) and reported biennially in the joint AIHW and Australasian Association of Cancer Registries (AACR) publication *Cancer in Australia: an overview*. This complex process of collecting and collating national cancer incidence statistics means the most recent national cancer data generally lags 2–3 years behind the current year.

In 2003 the Cancer Strategies Group (a subcommittee of the National Health Priority Action Council) Data Subcommittee identified both current and future cancer incidence data as a critical gap in data available at the time to support the planning of cancer services. In 2005, in response to this identified gap, the AIHW published *Cancer incidence projections, Australia 2002 to 2011*, providing estimates of both current and future national cancer incidence based on incidence data from 1982 to 2001. This report has been widely used in policy development and research since its publication.

In 2007 the AACR recommended that short-term national projections to provide year-to-date estimates for incidence be included in the biennial publication of *Cancer in Australia: an overview*. Simple extrapolation of recent trend data was used to project trends from the past decade forward to the current year (usually only a span of 2–3 years) and applied to the current population. This short-term projection within known parameters means factors that may affect incidence rates in the projected period, such as changes in detection methods or introduction of screening programs, can be noted and, where possible, accounted for. The first such year-to-date estimates were included in *Cancer in Australia: an overview, 2008*, with the most recent estimates to 2010 included in *Cancer in Australia: an overview, 2010*.

In 2009, the NCMC was established to provide accessible, policy-related national information for evidence-based decisions across the cancer pathway. The information and data produced by the NCMC assists health professionals, policy makers, service planners, consumers and the general public to better understand national cancer trends and patterns. At the first meeting of the advisory group to the NCMC it was noted that while year-to-date estimates of current incidence were important measures of current policies and resource allocation, estimates of future cancer incidence using longer-term projections were required to provide data to support future cancer policies and longer-term planning of cancer services. Longer-term estimates of future cancer incidence were identified as a critical data gap, and a report of estimated cancer incidence for the next decade (2011–2020) was recommended as a key priority.

Terminology

Year-to-date estimates: simple extrapolation of recent trend data to the current year. This uses known parameters, such as current populations and knowledge of current practices in cancer detection.

Projections: longer-term extrapolation of recent trend data using unknown parameters, such as expected future populations.

Scope

In response to the gap identified by the NCMC, this report provides longer-term national projections of cancer incidence from 2011 to 2020. It does not present state or regional projections of cancer incidence—these are produced by individual state and territory cancer registries.

Cancer groups

Cancer groups in this report are consistent with the groupings in *Cancer in Australia: an overview, 2010* (AIHW & AACR 2010). These groups are defined according to the tenth version of the International Classification of Diseases and Related Health Problems (ICD-10) and are primarily based on cancer sites (for example, breast, lung and liver cancer) or, in the case of cancers of the blood and lymphatic systems, according to current understanding and histology (that is, cell types) of these cancers (namely lymphoid and myeloid cancers). Appendix Table A.1 lists the ICD-10 codes for cancer groupings used in this report.

This report presents national projections for the most commonly diagnosed cancers in 2007 for males and females with the following exceptions:

- Basal and squamous cell carcinomas of the skin (the most common forms of non-melanoma skin cancers) are not required to be reported to the cancer registries; hence, data on these cancers are not included in the ACD, and are therefore not included in this

report. These are the most common cancers diagnosed in Australia, but only rarely result in death (AIHW & CA 2008).

- Myeloid cancers: these are cancers that develop in the blood-forming cells of the bone marrow—the most common types are myeloid leukaemia and myelodysplastic syndrome. Myeloid cancers were formerly considered to be disorders of uncertain behaviour, but have only recently become recognised as cancers. Although these cancers accounted for 1,735 new cases in 2007, coverage in the ACD is only complete from 2003 onwards, which is not considered sufficient to enable accurate identification of trends.
- Cancers of unknown primary (CUP) site: this is a heterogeneous group of metastatic cancers (cancer that has spread) for which the primary cancer is unknown. While CUP was the seventh most commonly recorded cancer in 2007, incidence of such cancers is influenced by the stage of presentation of the cancers and the degree to which detailed investigation of the patient and the tumour is possible. While incidence of CUP is generally declining, this may represent an increase in the precision of diagnosis, which may be further improved by future techniques (Muir 1995) or earlier diagnosis of the primary cancer before it spreads. Given the future uncertainty in these factors, projections of CUP are unreliable.

Indigenous incidence projections

It is well established that Aboriginal and Torres Strait Islander people generally suffer more ill health than other Australians (AIHW 2010) and the number of Aboriginal and Torres Strait Islanders likely to be diagnosed with cancer in the future is important for providing adequate services to Indigenous Australians, and in informing policy, including closing the gap in health outcomes between Indigenous and non-Indigenous Australians.

However, while all state and territory cancer registries collect information on Indigenous status, in some jurisdictions the level of identification of Indigenous Australians is not considered to be sufficient to enable accurate estimates of incidence. Hence, reliable national data on the incidence of cancer for Indigenous Australians are not available and analyses of Indigenous cancer data from 1982 to 2007 must be restricted to Queensland, Western Australia, South Australia and the Northern Territory. While the majority (60%) of Australian Indigenous people live in these four jurisdictions (ABS 2009), the degree to which data for these jurisdictions are representative of data for all Indigenous people is unknown.

From 2003 to 2007, cancer in Indigenous Australians only accounted for 1% of all cancers diagnosed in Australia. The most common cancers diagnosed in Indigenous Australians (in those jurisdictions able to be analysed) were lung cancer (average of 71 cases per year), breast cancer in females (49) and bowel cancer (37) (AIHW & AACR 2010). These data are insufficient to develop a reliable projection model and would require specialised modelling techniques to provide projections generalisable to the whole Australian Indigenous population.

Mortality projections

This report is limited to projections of cancer incidence. Although modelling of future mortality due to cancer can be undertaken in a similar fashion to incidence, projections of cancer-related mortality are not included in this report as mortality is a function of both incidence and treatment; hence, modelling mortality independently of incidence rather than as a function of incidence and survival can produce meaningless results.

2 Methods

Introduction and disclaimer

These projections of cancer incidence are a mathematical extrapolation of past trends, assuming that the same trend will continue into the future, and are intended to illustrate future changes that might reasonably be expected to occur if the stated assumptions were to apply over the projection period. The projections are not forecasts and do not attempt to allow for future changes in cancer detection methods (such as the introduction of new screening programs), changes in cancer risk factors (such as the introduction of vaccination programs) or for non-demographic factors (such as major government policy decisions, economic factors, catastrophes, wars, epidemics or significant health treatment improvements) beyond the base years of the model which may affect future cancer incidence rates.

The nature of the projection method used and inherent fluctuations in both cancer trends and population dynamics mean that care should be taken when using and interpreting the projection results in this report. No liability will be accepted by the AIHW for any damages arising from decisions or actions based upon these cancer incidence projections.

Data sources and quality

Cancer data

National cancer incidence data from the ACD were used to develop the underlying model for projections. National statistics are currently available for all years from 1982 to 2007. Exceptions to this are:

- Incidence data for non-melanoma skin cancer (C44) excludes basal cell and squamous cell carcinomas and only covers the rarer kinds of non-melanoma skin cancer. These data are complete from 2001 onwards.
- Nationally consistent incidence data for polycythaemia vera (D45), myelodysplastic syndromes (D46) and other chronic myeloproliferative diseases (D47.1 and D47.3) are only available from 2003 to 2007.

Together these exceptions make up only a small fraction of all notifiable cancers and so are not expected to affect the overall projections.

Medicare Benefits Schedule (MBS) data were used in conjunction with cancer incidence data to inform trends in prostate cancer testing from 1992 to 2010.

More detailed information on the ACD and MBS data is in Appendix C.

Population data

Historic population data

Throughout this report, ABS Estimated Resident Population data by age and sex were used to calculate age-specific incidence rates for each cancer from 1982 to 2007. See Appendix C for further information on estimated resident populations.

Projected population data

For each of the age-sex-cancer models developed, projected rates were applied to the ABS's projected age-specific populations, *Population projections, Australia, 2006 to 2101 (*series B*)* to obtain projected incidence counts. Series B largely reflects current trends in fertility, life expectancy at birth and net overseas migration (ABS 2008).

Key assumptions

In producing projections to 2020, it was necessary to make a number of assumptions about trends in cancer incidence and demographic factors that must be considered in interpreting the results. These assumptions were made in consultation with the expert advisors appointed by the NCMC.

Underlying cancer rates

From 1982 (the first year for which national incidence data are available) to 2007, overall cancer incidence rates (age-standardised to the Australian 2001 population) have increased by about 1% per annum from 383 to 485 cases per 100,000 persons. However, this does not account for variations in specific cancers, or variations between the states and territories.

These projections are based on the following assumptions about underlying cancer rates:

1. Trends in age-sex-cancer specific incidence rates are nationally homogeneous.
2. The age effect will remain stable.
3. Past trends used to develop the model will continue to 2020.
4. The chosen model is an adequate representation of those trends.

These assumptions are discussed further below.

Assumption 1: National homogeneity

Although some differences are evident in incidence trends between state and territory jurisdictions (AIHW & AACR 2010), these projections are based on a national model developed using national incidence data. A national model is more sensitive to changes over time than models developed using individual jurisdictions, especially for the less populous jurisdictions. However, this approach assumes that incidence is homogenous across Australia; that is, cancer rates are spread evenly across all the Australian states and territories.

Assumption 2: Constant age effect

The modelled, and projected, incidence rates are calculated by aggregating rates for each of the 5-year age groups from 0–4 to 95 and over. The methodology assumes that each age group is homogenous and the trend for the 5-year age group is representative of the trends of each single year of age within that group. More specifically, we assume that the estimated trend, and the decision about its statistical significance, applies to each of the five single-year ages.

Assumption 3: Continuation of past trends

These projections assume continuation of the past trend into the future; however, incidence rates of specific cancers are affected by a number of factors, such as lifestyle changes, screening and early detection.

Variations in risk factors, such as smoking, consumption of alcohol, physical activity and obesity, are strongly associated with incidence rates of specific cancers. For example, decreases in smoking rates have led to decreases in lung cancer rates, while decreases in physical activity and increases in obesity are associated with increasing rates of some cancers, including uterine, oesophageal, kidney, gallbladder, pancreatic and breast cancer in post-menopausal women (IARC 2008; WCRF/AICR 2007).

Advancements in the sensitivity of early detection procedures and increases in screening practices also affect incidence rates. It is important to note this may sometimes lead to over-ascertainment of the disease—that is, pathological diagnosis of cancer which would not go on to cause symptoms or death—that cannot be determined at time of diagnosis.

Incorporation of these factors into projections requires specialised modelling techniques specific to each cancer and is beyond the scope of this publication. However, to aid interpretation, projections for specific cancers are presented with any available information that may have influenced past incidence rates, or may affect future incidence.

Assumption 4: Adequate model fitting

Extrapolation of a mathematical model to predict future incidence relies on two factors:

1. That the data collected are adequate for the production of accurate estimates of incidence time trends. This is true for those cancers with high annual case numbers; however, cancers with smaller case numbers, especially in particular age groups or for one sex, often yield parameters with wide statistical variation.
2. An appropriate model is chosen to describe both the historical data and expected future trend. All models are subject to uncertainties, and while a number of models will often fit the historic data well for short time periods, projections arising from these alternative models can be quite different.

An assessment of the model adequacy is provided in Appendix B.

Demographic factors

The Australian population has been increasing since 1982 and is expected to exceed 25 million by 2020. In Australia, cancer primarily occurs in people aged 65 and over, with about 50% of all people in this age group expected to be diagnosed with cancer in their remaining lifetime. Between 1982 and 2007 the proportion of the Australian population aged 65 and over increased from 10% to 13%, and is predicted to increase to about 17% by 2020 (Figure 1.1).

Population projections are based on assumptions of fertility, mortality and migration based on historical trends. Such trends may not reflect future effects on mortality through changing lifestyle factors, such as decreases in smoking or physical activity, or increases in obesity, or through advances in medical treatments.

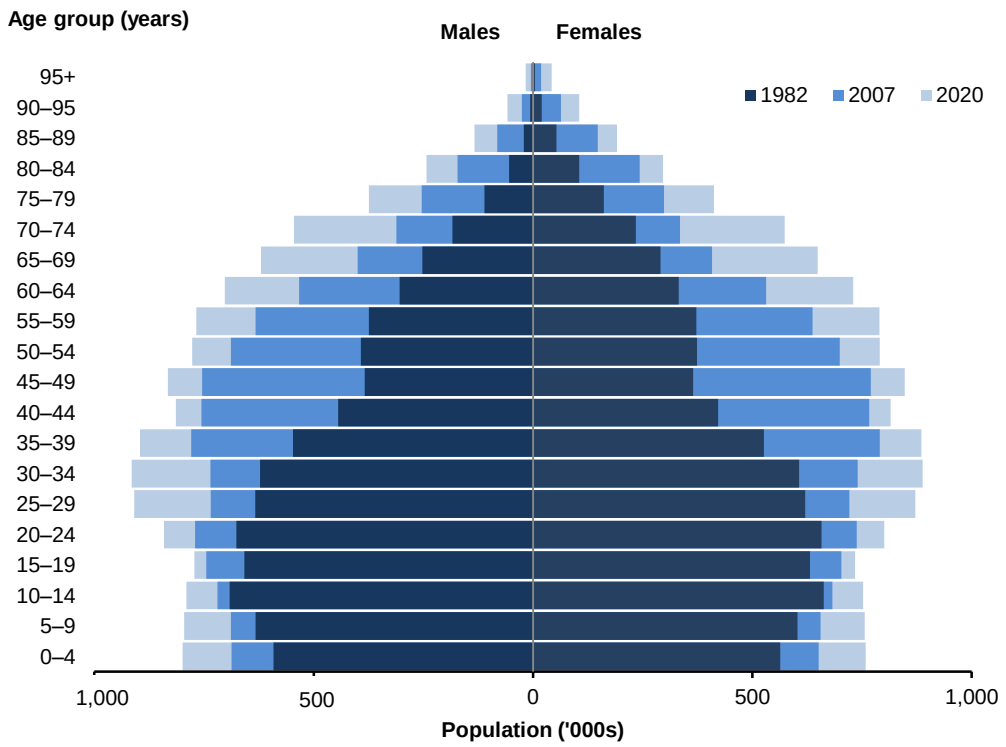


Figure 1.1: Australian population distribution, by age and sex: 1982, 2007, 2020

Model fitting and projection

There are a number of models that may be used to project cancer incidence. These range from a simple linear regression to provide a generalised picture of future trends in incidence, to elaborate mathematical modelling that attempts to account for changes in cancer incidence in response to changes in risk factors or detection methods, or even changes in incidence of other diseases, such as diabetes and cardiovascular disease, to accurately predict the number of expected new cases from each cancer.

One of the major challenges in undertaking cancer projections is developing a methodology to use with a large number of different cancer groupings at a national level to provide reasonable estimates. There is a general consensus among cancer statisticians that a relatively simple linear or log-linear Poisson model or ordinary least squares model of age-specific rates provides a good fit to the data while giving reasonably accurate predictions over a short to medium time span. The accepted (conservative) approach among statisticians preparing projections of this nature is to assume a linear model for increasing rates, and a log-linear model for decreasing rates to prevent projecting incidence rates below zero. It should be noted, however, that there is a fundamental assumption in this approach that the factors that affect cancer incidence (for example, risk factors, cancer detection) evolve in an approximately linear or log-linear way with time for each age group. This assumption holds as long as there are no major quantitative changes in any underlying factors, such as the introduction of a screening program.

Base data

The period used for fitting the data is a compromise between the accuracy of the model and the currency of the trends. If the period is too short, it does not provide enough data for accurate model estimates. If it is too long, there is a risk that the model is based on old trends that are no longer relevant to the current situation.

To determine the most recent national trend for each cancer, variations in trends in the age-standardised incidence rate of each cancer were analysed by fitting a piece-wise linear model to the age-standardised incidence rate from 1982 to 2007 using Joinpoint[1] software. Where statistically significant changes in the magnitude and/or direction of the trend were detected, the most recent trend was used as the base data.

Model

A national model was then developed using national incidence data for each cancer as follows:

- An ordinary least squares linear regression model was developed for each age-sex group using incidence rates from the most recent trend. Appendix A provides the observation window used for the most recent trend and modelling age groups for each cancer group.
- The significance of time as a predictor was determined at the 5% level by applying a two-tailed test to the slope coefficient. In age groups where the slope coefficient was not significant, the projection rate was set to the mean incidence rate over the most recent trend.
- Where a significant decreasing trend was detected, then it was assumed the incidence rate is decaying over time (but never reaching zero) and ordinary least squares linear regression model with a log transformation was used instead.
- For each of the age-sex-cancer models developed, projected rates were applied to the projected population data to obtain projected incidence counts. The predicted age-sex-cancer incidence counts were then summed to obtain national cancer-specific predicted incidence counts for males, females and persons. Age-standardised incidence rates for each cancer were calculated from the age-sex specific predicted counts.
- Prediction intervals to indicate the range of uncertainty around each projection were calculated. All estimated counts are rounded to the nearest 10. For counts less than 1,000, estimates are rounded to the nearest 5. Calculations of percentage and numeric change, proportions and rates are based on unrounded data.

A mathematical explanation of the model is provided in Appendix B, along with notes on model accuracy and validation.

[1] Joinpoint is statistical software for the analysis of trends using joinpoint models. See Appendix B for more information.

3 Projections by cancer site

This chapter presents an examination of past trends and projected incidence counts, age-specific rates and age-standardised rates for all cancers combined, as well as the most common cancers for males and females. For each projection, a set of graphs is provided to aid interpretation. These show:

- the change in the age-structure of the cancer over time
- observed and projected incidence rates for broad age groups 0–24, 25–44, 45–64, 65–84 and 85 and over
- observed and projected incidence counts
- observed, modelled and projected age-standardised rates.

More detailed data by 5-year age groups are available as a series of web-based tables at <www.aihw.gov.au>.

It is important to note that projections for cancer have a level of uncertainty around them, meaning there is a statistical range in which the true value is likely to lie. Each set of projections is therefore accompanied by prediction intervals to indicate the range of uncertainty around these figures.

It is also important to note that while projected incidence counts are presented to provide an indication of the future burden of each cancer, these counts are rounded to the nearest 10 for counts greater than 1,000 and the nearest 5 for counts less than 1,000. However, this rounding does not indicate the level of precision; this is provided by the associated prediction intervals.

All cancers combined (C00–C96, D45–D46, D47.1, D47.3)

Although cancer is a group of diseases, reporting and projecting the incidence of all cancers combined is a useful measure of the amount of illness due to cancer in a population. There are a number of methods for projecting total cancer incidence. The simplest is to treat all cancers combined as a single disease homogenous with respect to body site and model as such. More complex methods model each cancer group separately and combine them to form a single estimate. The first method has the advantage of greater statistical power and sensitivity to changes in age-specific trends, but there is the disadvantage that trends of the less common cancers are overshadowed by trends of the more common cancers. Conversely, while the second approach has the advantages of accounting for trends in less common cancers, it requires separate projections of more than 100 different diseases. This report uses the first method for projecting total cancer incidence, with a variation for males described below.

Males

Trends in age-standardised rates of cancer incidence in males have fluctuated over the 26 years of national data collection, making it difficult to project future incidence. The introduction of prostate-specific antigen (PSA) testing in the late 1980s was responsible for the detection of a large number of previously undiagnosed prostate cancers in the early to mid-1990s. As prostate cancer accounts for 25–30% of cancers diagnosed each year in males, this resulted in a peak in incidence rates of all cancers combined for males. Following this peak, incidence rates returned to trends similar to before PSA testing was introduced. Incidence data for 2002–2007 show a second noticeable increase in the detection of prostate cancer, which again has had an effect on the trend for all cancers combined for males. It is unclear whether this trend will continue or whether incidence rates will return to similar trends as they did in the late 1990s; however, analysis of trends in prostate cancer suggests that rates will steady (see separate section on prostate cancer). As a result, the projections for all cancers combined have been prepared by combining separate projections for prostate cancer with all other cancers combined.

Joinpoint analysis of age-standardised rates for all cancers combined excluding prostate cancer showed a slight, but statistically significant increasing trend from 1982 to 1994, followed by a slight, but statistically significant decreasing trend from 1994 to 2007. This most recent trend formed the observation window for developing the projections model for all cancers combined excluding prostate cancer. Analyses of trends in prostate cancer are described in the section on prostate cancer and two sets of projections (based on linear and logarithmic models) are presented. The logarithmic model of prostate cancer was used to develop the projections for all cancers combined presented here.

In 2007, there were more than 62,000 new cases of cancer diagnosed in males, which when age-standardised equated to about 595 cases per 100,000 males. While age-standardised rates of all cancers combined for males have risen since 1998, it is expected that they will steady at about 568 new cases per 100,000 males between 2011 and 2020. This is primarily due to the anticipated steadying of rates of prostate cancer incidence, coupled with decreasing rates in lung cancer in males, which accounts for a further 9–10% of cases. When taking into account expected changes in the population structure, this will translate into about 85,000 new cases expected to be diagnosed in 2020 (Tables 3.1a, Figure 3.1a). The accompanying prediction intervals indicate the range of uncertainty around the figures presented.

While overall rates are expected to steady, small decreases in rates are expected for males aged 25–44 and 65–84 and increases are expected for males aged 45–64 and 85 and over (Table 3.1b, Figure 3.1a).

Table 3.1a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: all cancers combined

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	66,060	62,760	69,360	567.9	539.8	595.9
2012	68,100	64,680	71,530	568.0	539.9	596.1
2013	70,110	66,560	73,650	568.1	540.0	596.3
2014	72,110	68,440	75,790	568.2	540.0	596.4
2015	74,150	70,340	77,960	568.3	540.0	596.5
2016	76,240	72,280	80,200	568.3	540.0	596.6
2017	78,400	74,270	82,530	568.4	540.0	596.7
2018	80,540	76,240	84,840	568.4	540.0	596.8
2019	82,730	78,290	87,170	568.4	540.0	596.9
2020	84,950	80,370	89,530	568.4	539.9	596.9

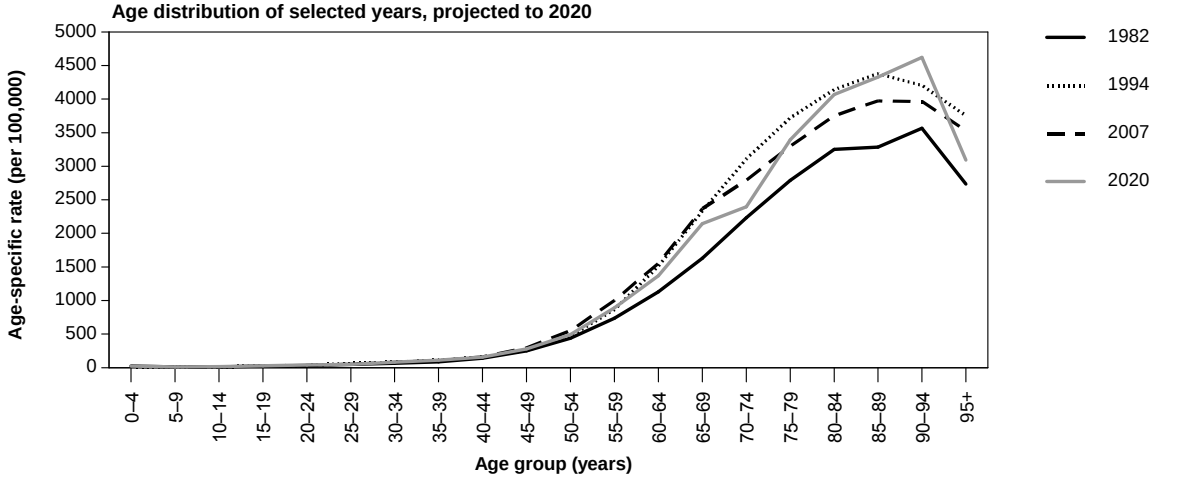
Table 3.1b: Projected number of new cases and age-specific rates, males, 2011–2020: all cancers combined

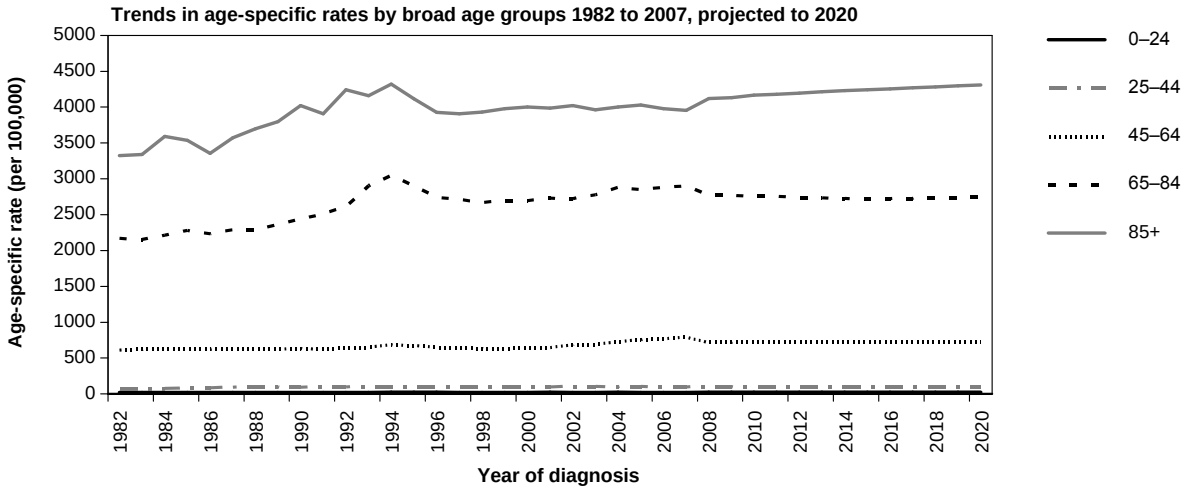
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	875	23.5	3,200	101.7	20,410	729.4	35,450	2758.5	6,120	4179.7
2012	885	23.6	3,240	101.6	20,530	728.9	36,950	2745.2	6,500	4196.3
2013	895	23.6	3,280	101.4	20,750	730.1	38,300	2735.3	6,880	4213.9
2014	900	23.6	3,310	101.0	21,030	731.1	39,630	2730.4	7,240	4230.3
2015	905	23.5	3,340	100.6	21,290	731.2	41,020	2726.1	7,600	4242.3
2016	910	23.5	3,360	100.0	21,590	731.1	42,450	2724.6	7,930	4255.6
2017	915	23.5	3,380	99.6	21,880	731.1	44,030	2729.5	8,190	4268.6
2018	920	23.4	3,410	99.3	22,130	731.6	45,620	2731.8	8,450	4282.8
2019	925	23.4	3,450	99.1	22,400	733.2	47,250	2738.3	8,710	4296.8
2020	930	23.3	3,500	99.1	22,620	734.5	48,930	2745.7	8,970	4310.7

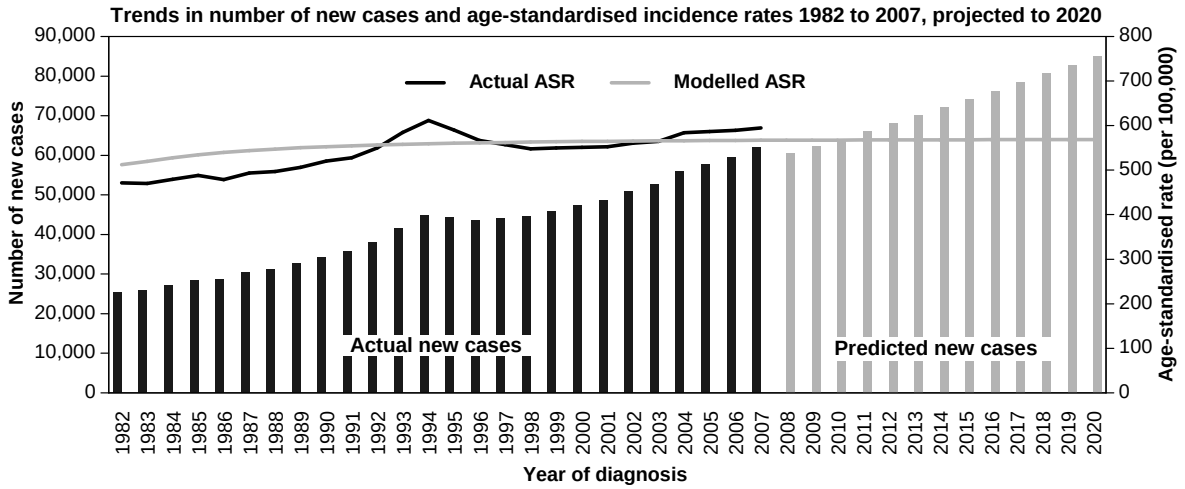
Notes

1. All cancers combined include ICD-10 codes C00–C96, D45, D46, D47.1 and D47.3. It excludes basal and squamous cell carcinomas.
2. Projected estimates are based on incidence data for all cancers excluding prostate cancer from 1994 to 2007, and logarithmic projection of prostate cancer from 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Rates are age-standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 population.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 population

(b) Age-standardised rates age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007)

Figure 3.1a: All cancers combined (C00–C96, D45, D46, D47.1, D47.3), males, Australia: actual 1982–2007 and projected to 2020

Females

In 2007, there were 46,000 cases of cancer diagnosed in females, which when age-standardised equated to about 394 cases per 100,000 females.

Unlike trends over time for males, trends in age-standardised rates of all cancer incidence in females are more stable. Joinpoint analysis of age-standardised rates for all cancers combined showed evidence of an increasing linear trend of about 0.01 cases per 100,000 females per year from 1982 to 1995. This slowed to an increase of only 0.002 cases per 100,000 females per year from 1995 to 2007.

Extrapolation of age-specific data from this most recent trend suggests that, overall, age-standardised rates of all cancers in females will increase slightly from an expected 403 new cases per 100,000 females in 2011 to 408 new cases in 2020. Increases in rates are expected in all major age categories 25 years and over, with changes in trend increasing with increasing age. This is expected to equate to about 65,000 new cancer cases diagnosed in 2020 (Tables 3.1c, 3.1d, Figure 3.1b).

Table 3.1c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: all cancers combined

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	52,080	51,370	52,790	402.6	397.1	408.0
2012	53,440	52,700	54,180	403.2	397.7	408.7
2013	54,800	54,030	55,570	403.8	398.2	409.5
2014	56,180	55,370	56,980	404.4	398.7	410.2
2015	57,580	56,740	58,430	405.1	399.2	410.9
2016	59,030	58,150	59,910	405.7	399.7	411.7
2017	60,500	59,580	61,430	406.3	400.2	412.4
2018	61,990	61,030	62,950	406.9	400.7	413.2
2019	63,510	62,500	64,510	407.6	401.2	413.9
2020	65,040	63,990	66,100	408.2	401.7	414.7

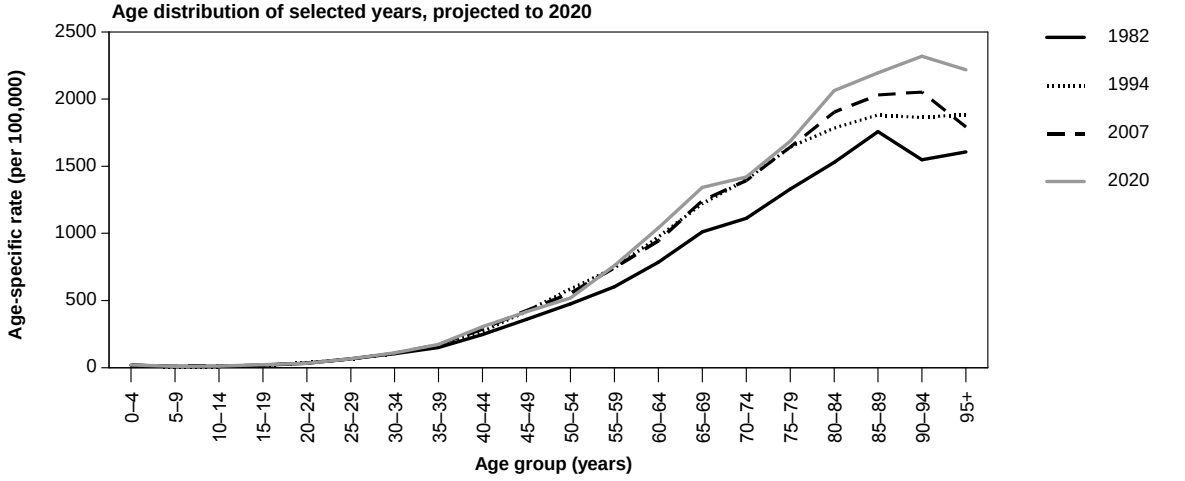
Table 3.1d: Projected number of new cases and age-specific rates, females, 2011–2020: all cancers combined

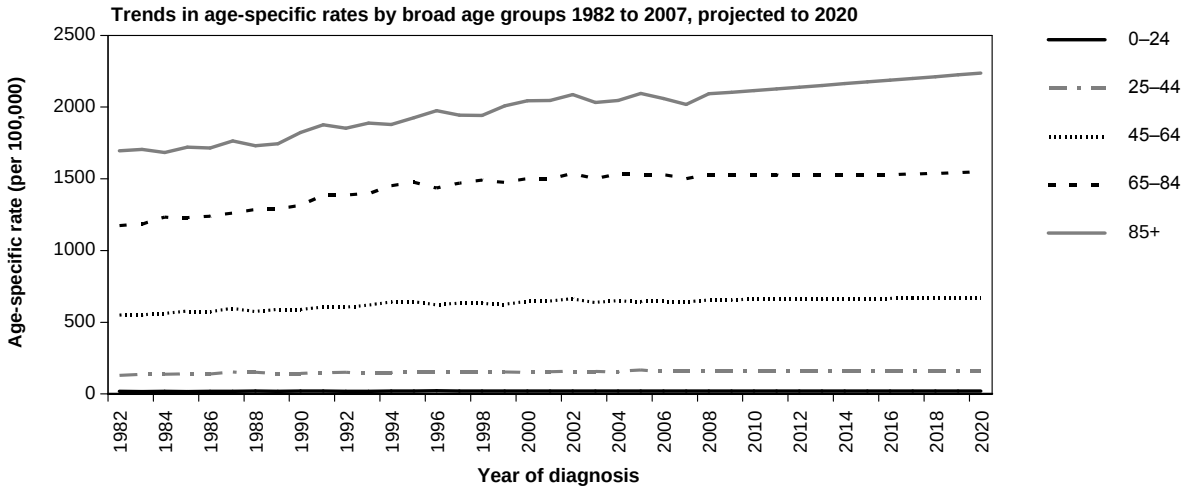
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	715	20.2	5,040	160.5	18,890	662.9	21,610	1528.8	5,830	2127.1
2012	720	20.1	5,120	161.2	19,060	663.4	22,470	1526.4	6,070	2139.2
2013	720	20.0	5,200	161.6	19,310	665.0	23,260	1525.5	6,310	2151.4
2014	725	20.0	5,250	161.6	19,600	666.3	24,070	1526.2	6,530	2163.7
2015	730	19.9	5,310	161.5	19,870	667.1	24,920	1527.3	6,760	2175.4
2016	730	19.8	5,330	160.9	20,190	667.6	25,820	1529.4	6,960	2187.3
2017	730	19.7	5,360	160.4	20,500	668.3	26,790	1534.2	7,110	2199.5
2018	735	19.6	5,420	160.3	20,760	669.4	27,820	1538.2	7,260	2212.0
2019	735	19.5	5,490	160.6	21,010	670.9	28,860	1543.6	7,410	2224.7
2020	740	19.4	5,570	161.1	21,230	672.5	29,920	1549.0	7,580	2237.4

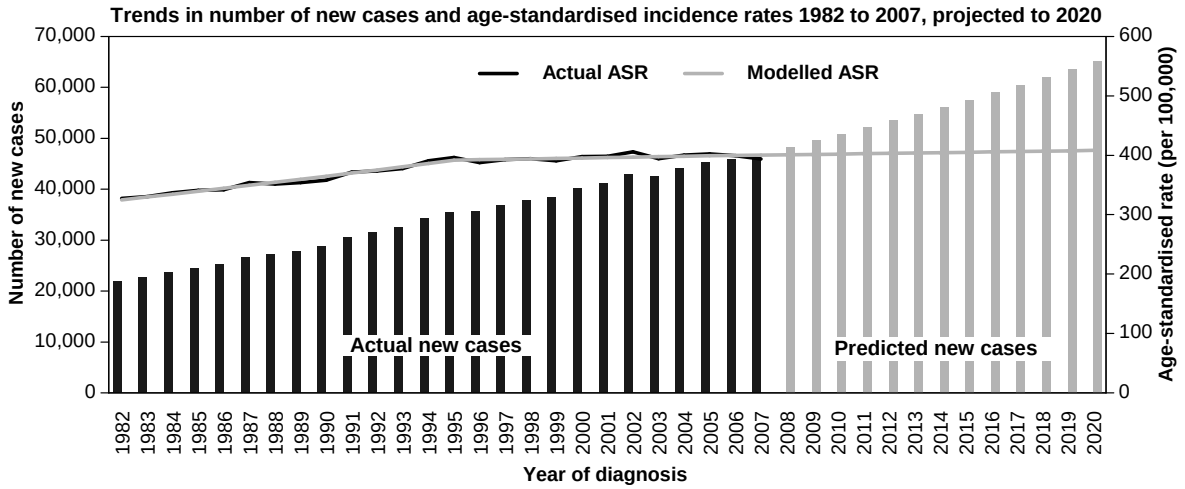
Notes

1. All cancers combined include ICD-10 codes C00–C96, D45, D46, D47.1 and D47.3. It excludes basal and squamous cell carcinomas.
2. Projected estimates are based on incidence data for 1995 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Rates are age-standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 population.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 population

(b) Age-standardised rates age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007)

Figure 3.1b: All cancers combined (C00–C96, D45, D46, D47.1, D47.3), females, Australia: actual 1982–2007 and projected to 2020

Bladder cancer (C67)

Bladder cancer accounted for 2.0% of all new cancer cases in Australia in 2007, making it the tenth most commonly diagnosed invasive cancer. It is much more common in males than females, with the risk of being diagnosed by age 85 being 1 in 44 for males and 1 in 154 in females. The incidence of bladder cancer increases rapidly with increasing age, with about 80% of people aged 65 or older at their first diagnosis.

The exact causes of bladder cancer are not known; however, factors that put some people at higher risk have been identified. Tobacco smoking is the largest risk factor, estimated to account for about 50% of cases (Freedman et al. 2011). Occupational exposure to certain chemicals used in the textile, petrochemical and rubber industries may also increase the risk of bladder cancer (Colt et al. 2004). Further, those with a family history of the disease are at higher risk (Pelucchi et al. 2006) as are those with diabetes mellitus (Larsson et al. 2006).

Distinguishing between malignant and non-malignant bladder tumours can be difficult, and classification and coding practices have changed over time resulting in more bladder tumours being categorised as in situ or uncertain—this makes trends in incidence rates difficult to interpret (Richards 2008). Individual cancer registries in Australia have attempted to retrospectively reclassify past cases; however, the extent of reclassification and uniformity in this practice is not known.

The age-standardised incidence rate of bladder cancer has been decreasing for both males and females since 1982; however, joinpoint analysis shows the rate of decline slowing from about the early 1990s for both males and females. Examination of the age-specific trends over time also shows a shift in the age of onset of bladder cancer in both males and females (figures 3.2a and 3.2b).While some of this decrease can be attributed to classification and coding changes, reductions in risk factors such as smoking and occupational exposure may also have played a role.

Males

Joinpoint analysis of the age-standardised incidence rates of bladder cancer for males showed a significant linear decreasing trend from 1982 to 1991 of about 0.8 cases per 100,000 males per year, followed by a lesser, but statistically significant decreasing trend of about 0.4 cases per 100,000 males per year from 1991 to 2007.

As this decreasing trend resembles a logarithmic decrease, extrapolation of age-specific trend from 1982–2007 using a logarithmic transformation indicates that bladder cancer incidence in males will continue to decrease to about 13 new cases expected to be diagnosed per 100,000 males in 2020; however, the expected increase in the ageing population will cause the actual number of cases to continue to rise slowly, with about 2,040 new cases expected to be diagnosed in 2020 (Table 3.2a). The largest decrease in rates is expected in males aged 65–84. Smaller decreases in rates are expected in males aged 45–64 and those aged 85 or over (Table 3.2b, Figure 3.2a).

Females

Joinpoint analysis of the age-standardised incidence rates of bladder cancer for females showed a similar, but less dramatic, trend to males. A significant linear decreasing trend of about 0.3 cases per 100,000 females per year was detected from 1982 to 1992, followed by a slight, but still statistically significant, decreasing trend of about 0.1 cases per 100,000 females per year from 1992 to 2007.

As this trend is also decreasing in a logarithmic fashion, extrapolation of the trend from 1982–2007 using a logarithmic transformation indicates that bladder cancer incidence in females is projected to decrease from 4.5 cases per 100,000 females in 2007 to about 3.8 in 2020. This will equate to about 755 new cases expected to be diagnosed in 2020 with the projected increase in the ageing population (Table 3.2c). Like males, the largest projected decline in the rate of bladder cancer will be in females aged 65–84 (Table 3.2d, Figure 3.2b).

Table 3.2a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: bladder cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,820	1,710	1,940	16.0	15.0	17.1
2012	1,850	1,730	1,970	15.7	14.7	16.8
2013	1,870	1,750	1,990	15.4	14.4	16.5
2014	1,890	1,770	2,020	15.1	14.1	16.1
2015	1,920	1,790	2,050	14.8	13.8	15.8
2016	1,940	1,810	2,070	14.5	13.5	15.5
2017	1,970	1,830	2,110	14.2	13.2	15.2
2018	1,990	1,850	2,140	14.0	12.9	15.0
2019	2,020	1,870	2,160	13.7	12.7	14.7
2020	2,040	1,890	2,190	13.4	12.4	14.4

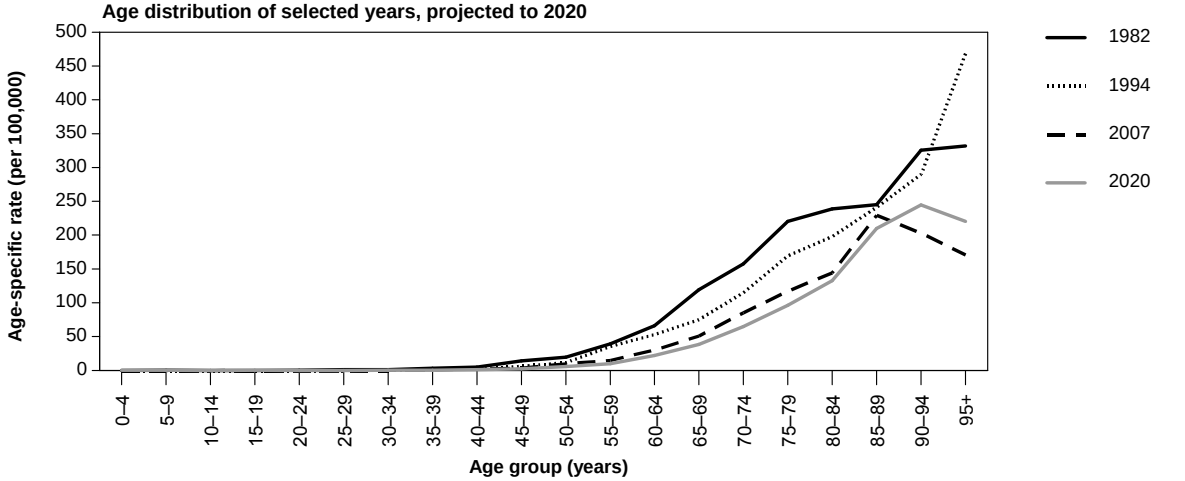
Table 3.2b: Projected number of new cases and age-specific rates, males, 2011–2020: bladder cancer

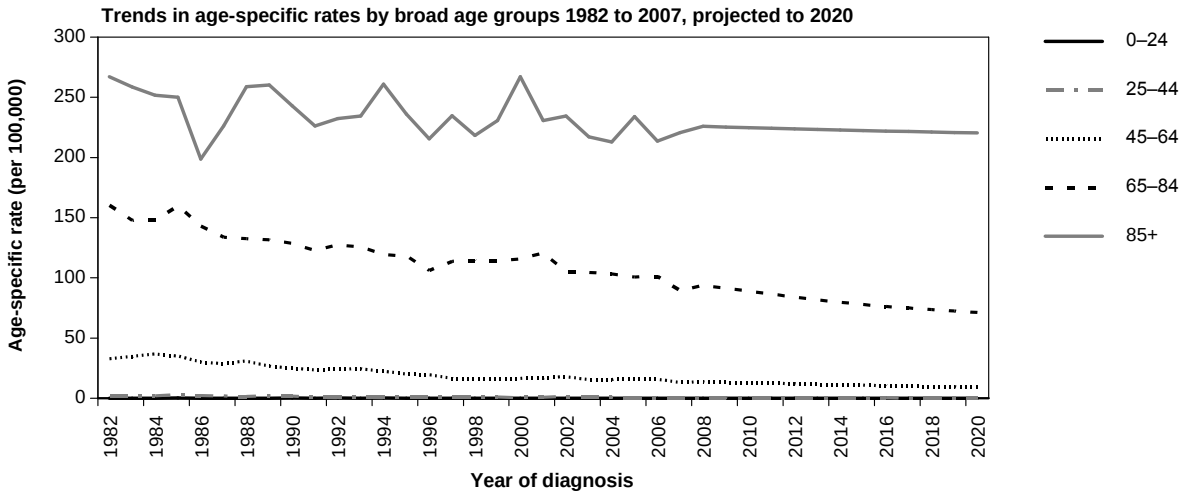
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.1	20	0.6	360	12.8	1,120	86.9	330	224.3
2012	0	0.1	20	0.6	345	12.3	1,130	84.3	345	223.9
2013	0	0.1	20	0.5	340	11.9	1,150	81.9	365	223.4
2014	0	0.1	15	0.5	330	11.6	1,160	79.9	380	223.0
2015	0	0.1	15	0.5	325	11.2	1,170	78.0	400	222.4
2016	0	0.1	15	0.5	320	10.8	1,190	76.4	415	222.0
2017	0	0.1	15	0.4	315	10.4	1,210	75.2	425	221.6
2018	0	0.1	15	0.4	305	10.1	1,230	73.9	435	221.2
2019	0	0.1	15	0.4	300	9.8	1,250	72.7	450	220.8
2020	0	0.1	15	0.4	295	9.5	1,270	71.5	460	220.4

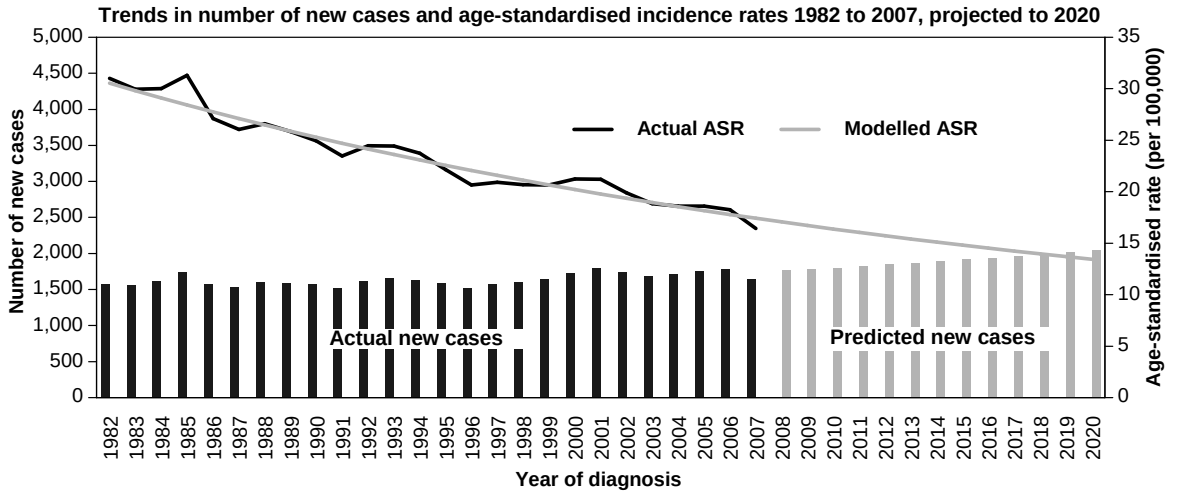
Notes

1. Bladder cancer includes ICD-10 code C67.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.2a: Bladder cancer (C67), males, Australia: actual 1982–2007 and projected to 2020

Table 3.2c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: bladder cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	645	575	715	4.4	3.9	4.8
2012	655	585	725	4.3	3.8	4.7
2013	665	595	740	4.2	3.8	4.7
2014	675	605	750	4.2	3.7	4.6
2015	690	615	765	4.1	3.7	4.5
2016	700	620	780	4.0	3.6	4.5
2017	715	635	795	4.0	3.5	4.4
2018	730	645	810	3.9	3.5	4.4
2019	740	655	830	3.9	3.4	4.3
2020	755	665	845	3.8	3.4	4.2

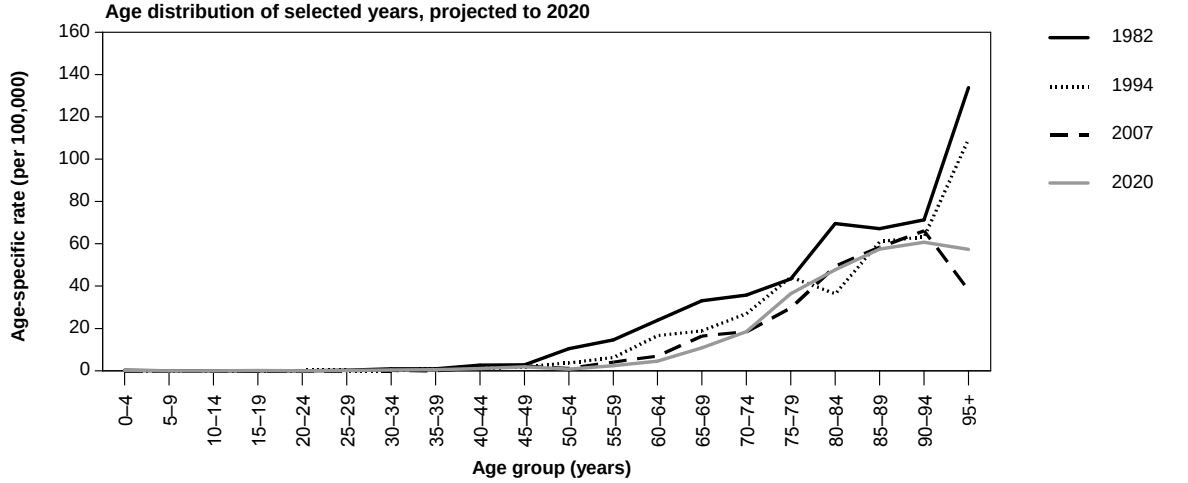
Table 3.2d: Projected number of new cases and age-specific rates, females, 2011–2020: bladder cancer

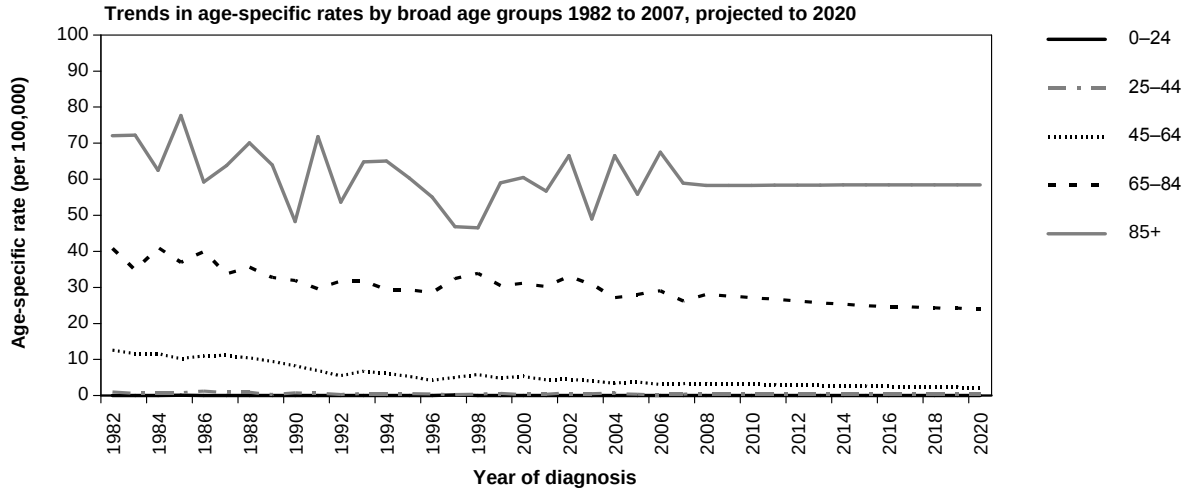
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	15	0.5	90	3.1	380	26.8	160	58.3
2012	0	0.0	15	0.5	85	3.0	385	26.3	165	58.4
2013	0	0.0	15	0.5	85	2.9	395	25.8	170	58.4
2014	0	0.0	15	0.5	85	2.8	400	25.4	175	58.4
2015	0	0.0	15	0.5	80	2.7	410	25.0	180	58.4
2016	0	0.0	15	0.5	80	2.6	420	24.7	185	58.4
2017	0	0.0	15	0.5	80	2.5	430	24.6	190	58.4
2018	0	0.0	15	0.5	75	2.5	440	24.4	190	58.4
2019	0	0.0	15	0.5	75	2.4	455	24.3	195	58.4
2020	0	0.0	15	0.5	75	2.3	470	24.2	200	58.4

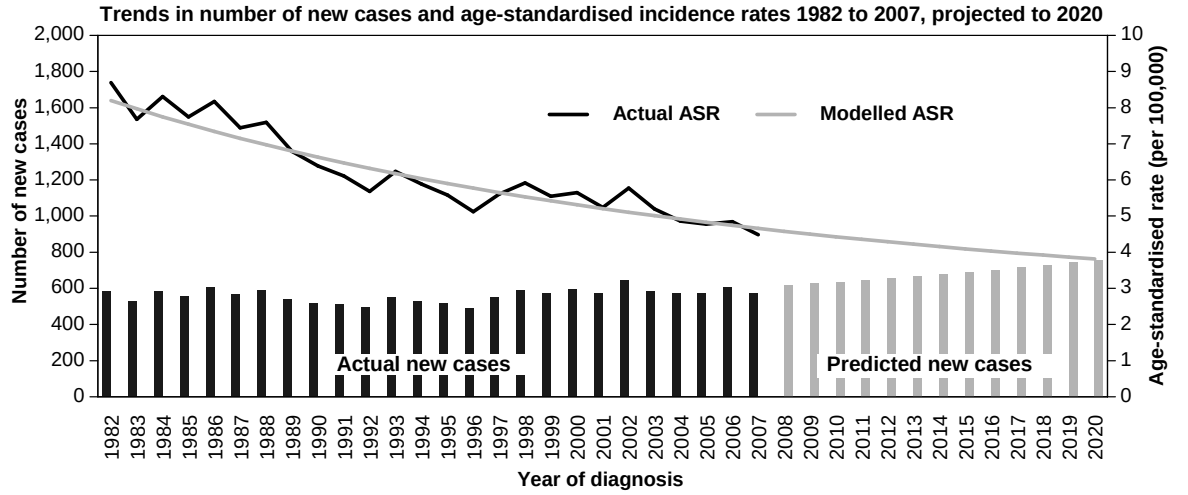
Notes

1. Bladder cancer includes ICD-10 code C67.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.2b: Bladder cancer (C67), females, Australia: actual 1982–2007 and projected to 2020

Bowel cancer (C18–C20)

Bowel cancer (which includes cancers of the colon, the rectosigmoid junction and the rectum) is also sometimes referred to as colorectal cancer. It is the second most common cancer diagnosed in males (after prostate cancer) and in females (after breast cancer) in Australia. Incidence of bowel cancer has been increasing each year since 1982, with 14,234 new cases diagnosed in 2007. Bowel cancer also accounts for 10% of all deaths from invasive cancers, with 4,047 deaths in 2007, making it the second most common cause of cancer-related death after lung cancer.

The National Bowel Cancer Screening Program (NBCSP) was introduced in mid-2006 to reduce both the incidence of and mortality due to bowel cancer. Phase 1 of the program (which ran from August 2006 to June 2008) offered screening to people aged 55 and 65—uptake of the screening test in Phase 1 was 39% for males and 47% for females (AIHW & DoHA 2008). Phase 2 (which began in July 2009) extended the program to also include people aged 50.

The introduction of screening is expected to lead to short-term increases in incidence rates due to the detection of previously undetected cancers in those being screened for the first time. However, as was observed for cervical cancer with the introduction of cervical screening, the long-term expectation is that incidence of bowel cancer in the age groups targeted for screening (currently those turning 50, 55, and 65) is likely to be reduced as pre-cancerous conditions are detected and treated before they progress to cancer.

The overall impact of the NBCSP on historical trends in bowel cancer incidence in this analysis is expected to be small due to the limited years of data available, limited ages screened during those years, and the low participation rates. The projections for bowel cancer in this section are based on extrapolation of the trends in incidence up to 2007 and do not attempt to model the impact of the NBCSP or other bowel screening campaigns on future incidence; however, their effects should be considered when interpreting these data.

Males

Joinpoint analysis of the age-standardised incidence rates of bowel cancer for males showed an increasing trend from 1982 to 1996, followed by a slight but statistically significant decreasing trend of about 0.3 cases per 100,000 males per year from 1996 to 2007.

Although extrapolation of the trend from 1996–2007 indicates that bowel cancer incidence in males will continue to decrease to about 71 cases diagnosed per 100,000 males in 2020, the expected increase in the ageing population causes the projected number of cases to continue to rise, with about 10,800 new cases expected to be diagnosed in 2020 (Table 3.3a).

The largest decrease in rates is expected in males aged 45–64 years, which is consistent with the National Health and Medical Research Council recommendation that people over 50 should participate in bowel screening (Australian Cancer Network 2005). Smaller decreases in rates are expected in males aged 85 and over (Table 3.3b, Figure 3.3a).

Females

Joinpoint analysis shows there has been no statistically significant change in the trend of the age-standardised incidence rates of bowel cancer for females from 1982 to 2007. Although rates have been increasing slightly at about 0.04 cases per 100,000 females per year, this is not statistically significant.

Extrapolation of age-specific trends from 1982 indicate that bowel cancer rates in females should remain constant at about 54 cases diagnosed per 100,000 females in 2020, equating to approximately 9,160 new cases (Table 3.3c). Similar to the projected incidence rate in males, there is expected to be a decline in the rate of bowel cancer in females aged 45–64, but it will not be as dramatic (Table 3.3d, Figure 3.3b).

Table 3.3a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: bowel cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	8,530	8,240	8,810	73.4	71.0	75.8
2012	8,770	8,480	9,070	73.1	70.7	75.6
2013	9,010	8,710	9,320	72.9	70.5	75.4
2014	9,250	8,930	9,570	72.7	70.2	75.2
2015	9,500	9,170	9,820	72.5	70.0	75.0
2016	9,750	9,400	10,090	72.3	69.8	74.8
2017	10,010	9,660	10,370	72.0	69.5	74.6
2018	10,270	9,910	10,640	71.8	69.3	74.4
2019	10,540	10,160	10,920	71.6	69.1	74.2
2020	10,800	10,410	11,200	71.4	68.9	74.0

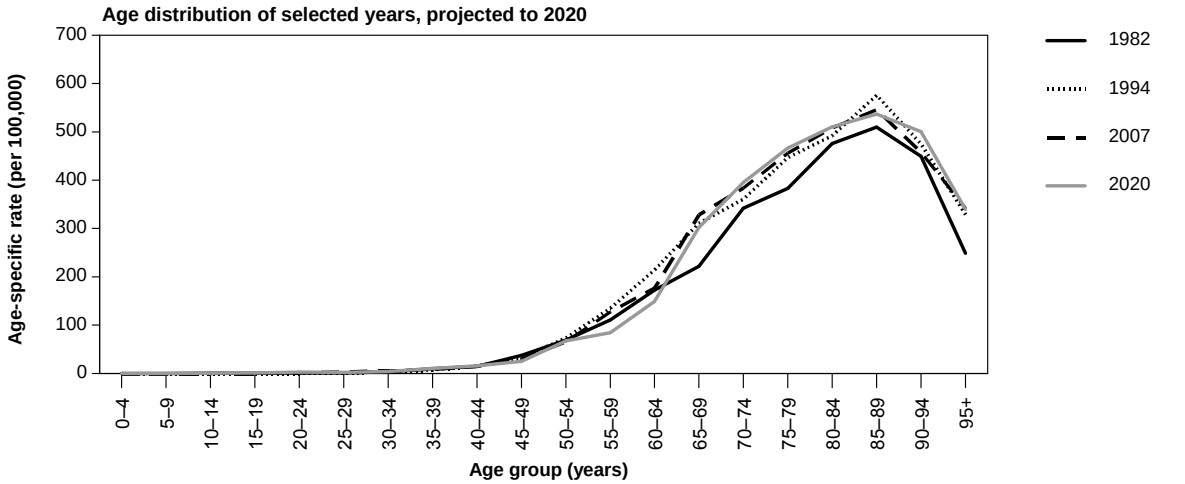
Table 3.3b: Projected number of new cases and age-specific rates, males, 2011–2020: bowel cancer

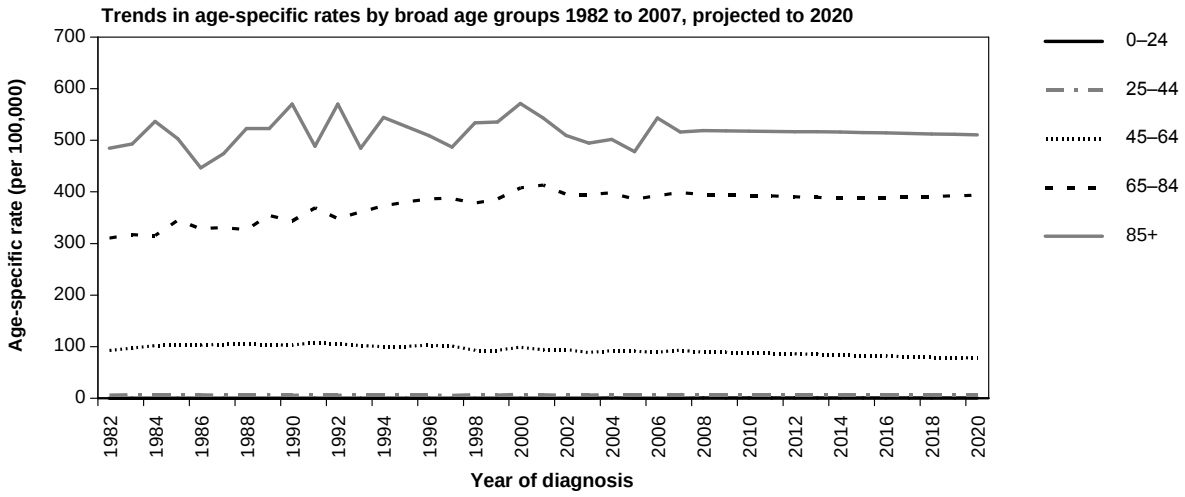
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	20	0.6	240	7.6	2,460	88.0	5,050	392.7	760	517.3
2012	25	0.6	245	7.7	2,440	86.8	5,260	390.9	800	516.8
2013	25	0.7	250	7.7	2,440	85.8	5,460	389.6	845	516.6
2014	25	0.7	250	7.7	2,440	84.8	5,650	389.3	885	516.2
2015	25	0.7	255	7.7	2,440	83.7	5,850	389.0	920	515.2
2016	30	0.7	255	7.6	2,440	82.6	6,060	389.2	960	514.3
2017	30	0.8	260	7.6	2,440	81.5	6,300	390.5	985	513.2
2018	30	0.8	265	7.6	2,430	80.4	6,540	391.4	1,010	512.3
2019	30	0.8	265	7.7	2,430	79.6	6,770	392.3	1,040	511.5
2020	35	0.8	275	7.7	2,430	78.8	7,010	393.4	1,060	510.7

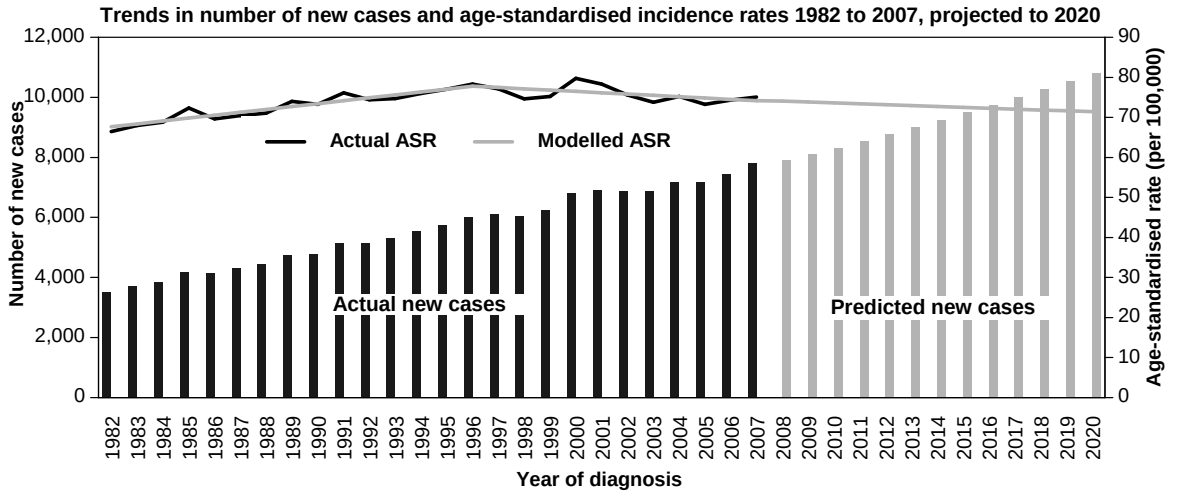
Notes

1. Bowel cancer includes ICD-10 codes C18–C20.
2. Projected estimates are based on incidence data for 1996 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.3a: Bowel cancer (C18–C20), males, Australia: actual 1982–2007 and projected to 2020

Table 3.3c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: bowel cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	7,110	6,850	7,380	53.1	51.1	55.1
2012	7,320	7,040	7,600	53.2	51.2	55.2
2013	7,520	7,240	7,810	53.2	51.2	55.2
2014	7,730	7,440	8,030	53.3	51.3	55.3
2015	7,950	7,650	8,260	53.4	51.3	55.4
2016	8,180	7,860	8,490	53.4	51.4	55.5
2017	8,410	8,090	8,740	53.5	51.5	55.6
2018	8,660	8,320	8,990	53.6	51.5	55.7
2019	8,900	8,560	9,240	53.7	51.6	55.8
2020	9,160	8,800	9,510	53.8	51.7	55.8

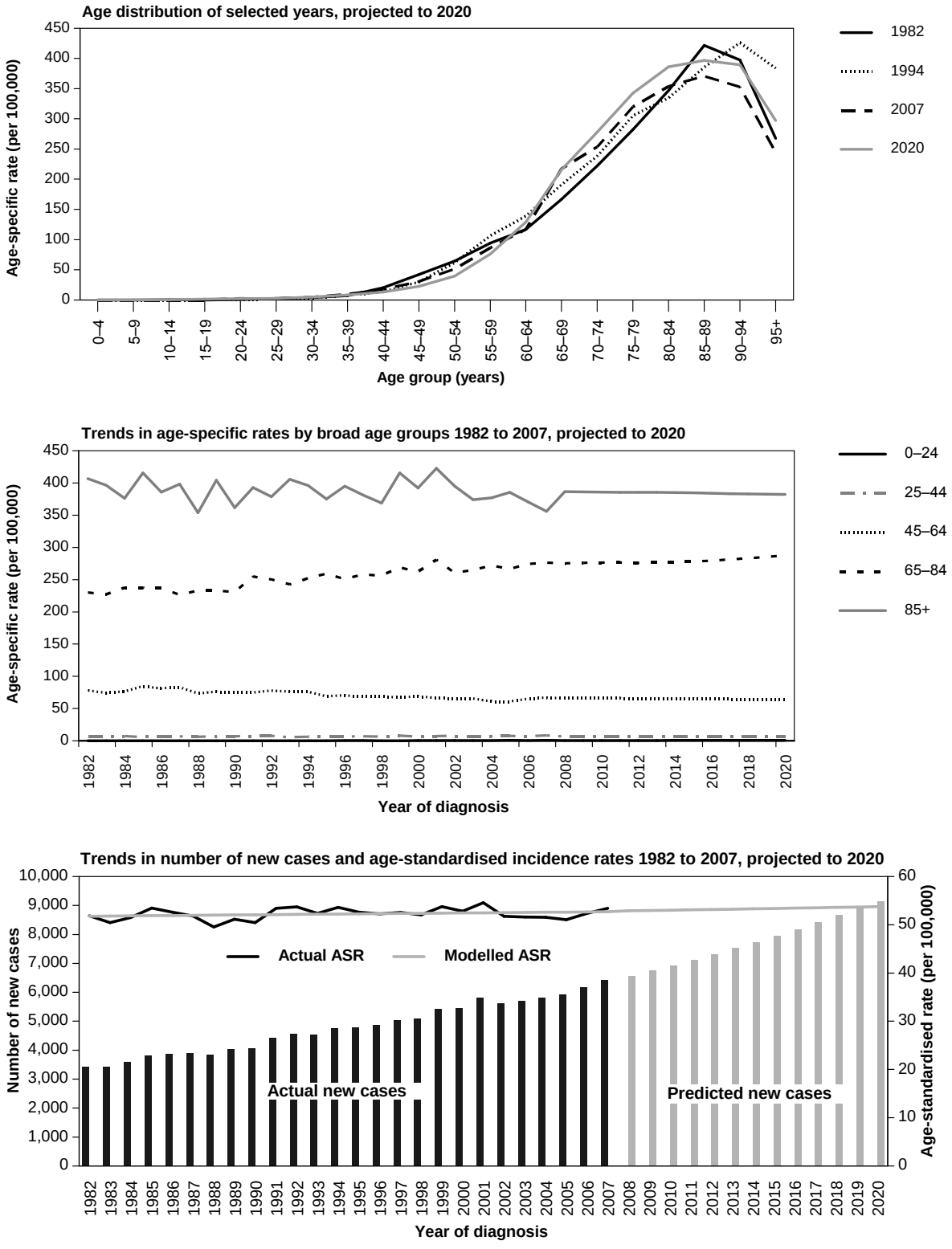
Table 3.3d: Projected number of new cases and age-specific rates, females, 2011–2020: bowel cancer

	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	20	0.6	225	7.2	1,900	66.6	3,910	276.8	1,060	385.4
2012	20	0.6	230	7.2	1,900	66.2	4,070	276.5	1,090	385.3
2013	25	0.6	230	7.2	1,920	66.1	4,220	276.7	1,130	385.2
2014	25	0.7	235	7.2	1,940	65.9	4,380	277.5	1,160	385.0
2015	25	0.7	235	7.1	1,960	65.7	4,540	278.3	1,190	384.5
2016	25	0.7	235	7.1	1,980	65.3	4,720	279.4	1,220	383.9
2017	25	0.7	235	7.0	2,000	65.1	4,920	281.5	1,240	383.4
2018	25	0.7	235	7.0	2,010	64.8	5,120	283.3	1,260	382.9
2019	30	0.7	240	7.0	2,030	64.7	5,330	285.3	1,270	382.4
2020	30	0.8	240	7.0	2,040	64.6	5,550	287.3	1,290	382.0

Notes

1. Bowel cancer includes ICD-10 codes C18–C20.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).



(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.3b: Bowel cancer (C18–C20), females, Australia: actual 1982–2007 and projected to 2020

Brain cancer (C71)

Brain cancer accounted for 1.4% of all new cancer cases in Australia in 2007 and is more common in males than females, with 1 in 101 males and 1 in 172 females developing the disease by the age of 85. While brain cancer only accounted for 2.8% of cancer deaths for all ages in Australia in 2007, it was the most common cause of cancer death for 15-29 year olds from 2003 to 2007, accounting for 14.2% of all cancer deaths in this age group (AIHW 2011a).

The exact causes of brain cancer are not known; however, factors that put some people at higher risk have been identified. Brain tumours are more common in people with certain inherited or genetic conditions, and those exposed to very high doses of therapeutic ionising radiation used in radiation therapy to treat cancer (Wrensch et al. 2002).

Concerns have been raised that radiofrequency electromagnetic fields emitted by mobile phones may also be a risk factor for brain cancer. Despite much research into the topic, current evidence is inconclusive as to whether mobile phones play a role in the aetiology of brain cancer (NCI 2011a). The International Agency for Research on Cancer (IARC) reviewed all the available evidence in 2011 and classified radiofrequency electromagnetic fields as a 'possible human carcinogen', but further research is recommended by the World Health Organization (WHO 2011). Multinational epidemiological research projects testing the relationship between mobile phone usage and cancer are being conducted to investigate the health effects of mobile phone use (ARPANSA 2011; COSMOS 2011).

The projections for brain cancer in this section are based on extrapolation of the trends in incidence from 1982 to 2007 and do not attempt to model changes in established or possible risk factors over time.

Males

Joinpoint analysis of age-standardised rates shows brain cancer in males to be increasing slightly, but significantly, at about 0.03 cases per 100,000 males per year since 1982. Extrapolation of age-specific trends from 1982 to 2007 indicates that rates are expected to increase for those aged 65–84, and 85 and over. Age-standardised rates are expected to reach 8.7 new cases per 100,000 males in 2020 which, taking into account expected changes to the population structure, will translate to an estimated 1,230 new cases diagnosed (tables 3.4a and 3.4b, Figure 3.4a).

Females

Joinpoint analysis of age-standardised rates show trends in brain cancer in females to be highly erratic, with three changes in the direction of the trend detected between 1982 and 2007. As a result, all available data were used to project age-specific trends, with the resultant projected age-standardised rate equivalent to the mean rate of 5.5 cases per 100,000 females.

Following this projection, it is expected that age-standardised rates for brain cancer in females will translate to an estimated 825 new cases diagnosed in 2020 (Table 3.4c). Rates are projected to remain constant in all age groups (Table 3.4d, Figure 3.4b).

Table 3.4a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: brain cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	975	900	1,050	8.5	7.8	9.1
2012	1,000	925	1,080	8.5	7.8	9.1
2013	1,030	950	1,110	8.5	7.9	9.2
2014	1,060	975	1,140	8.5	7.9	9.2
2015	1,080	1,000	1,170	8.6	7.9	9.2
2016	1,110	1,030	1,200	8.6	7.9	9.3
2017	1,140	1,050	1,230	8.6	8.0	9.3
2018	1,170	1,080	1,260	8.6	8.0	9.3
2019	1,200	1,110	1,290	8.7	8.0	9.4
2020	1,230	1,130	1,330	8.7	8.0	9.4

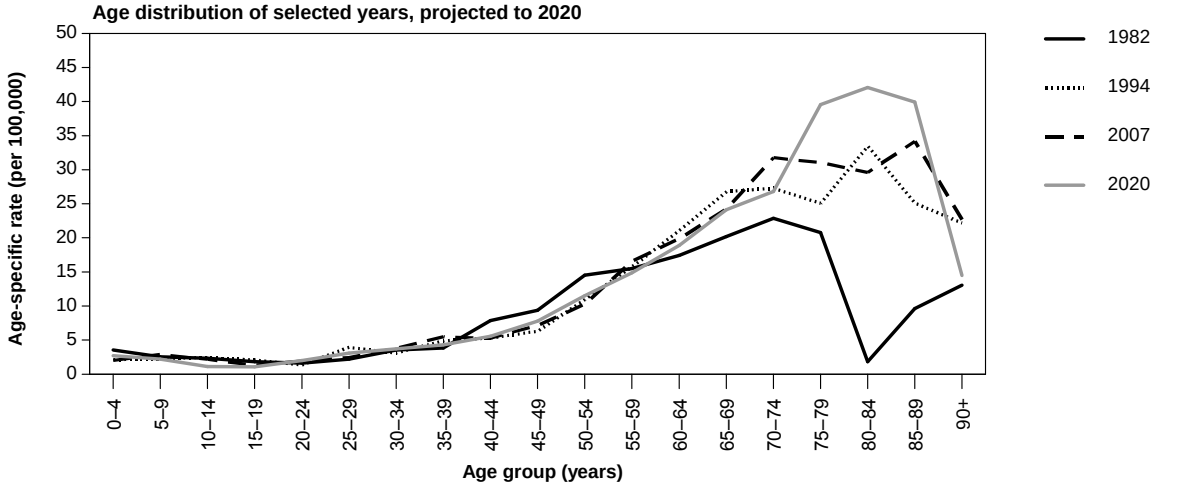
Table 3.4b: Projected number of new cases and age-specific rates, males, 2011–2020: brain cancer

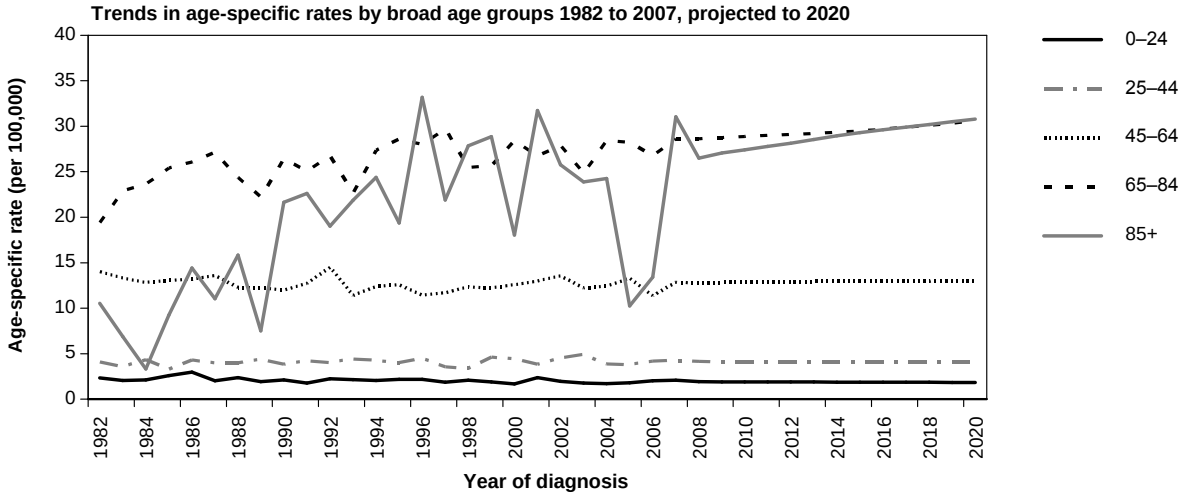
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	70	1.9	130	4.2	360	12.9	375	29.0	40	27.8
2012	70	1.9	135	4.2	365	12.9	390	29.1	45	28.2
2013	70	1.9	135	4.2	370	12.9	410	29.2	45	28.6
2014	70	1.9	135	4.1	375	13.0	425	29.4	50	28.9
2015	70	1.9	135	4.1	375	13.0	445	29.5	50	29.3
2016	75	1.9	140	4.1	385	13.0	460	29.7	55	29.6
2017	75	1.9	140	4.1	390	13.0	480	29.9	55	29.9
2018	75	1.9	140	4.1	395	13.0	505	30.1	60	30.2
2019	75	1.8	145	4.1	395	13.0	525	30.4	60	30.5
2020	75	1.8	145	4.1	400	13.0	545	30.6	65	30.8

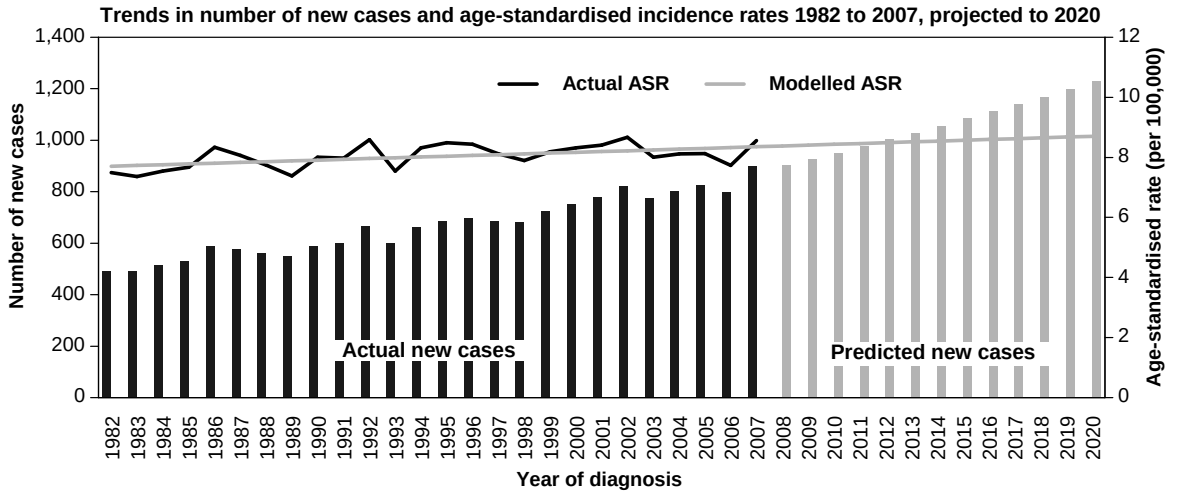
Notes

1. Brain cancer includes ICD-10 code C71.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.4a: Brain cancer (C71), males, Australia: actual 1982–2007 and projected to 2020

Table 3.4c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: brain cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	680	615	745	5.5	5.0	6.0
2012	695	625	760	5.5	5.0	6.0
2013	710	640	780	5.5	5.0	6.0
2014	725	655	795	5.5	5.0	6.0
2015	740	670	810	5.5	5.0	6.0
2016	755	680	830	5.5	5.0	6.0
2017	775	695	850	5.5	5.0	6.0
2018	790	710	865	5.5	5.0	6.0
2019	805	725	885	5.5	5.0	6.0
2020	825	740	905	5.5	5.0	6.0

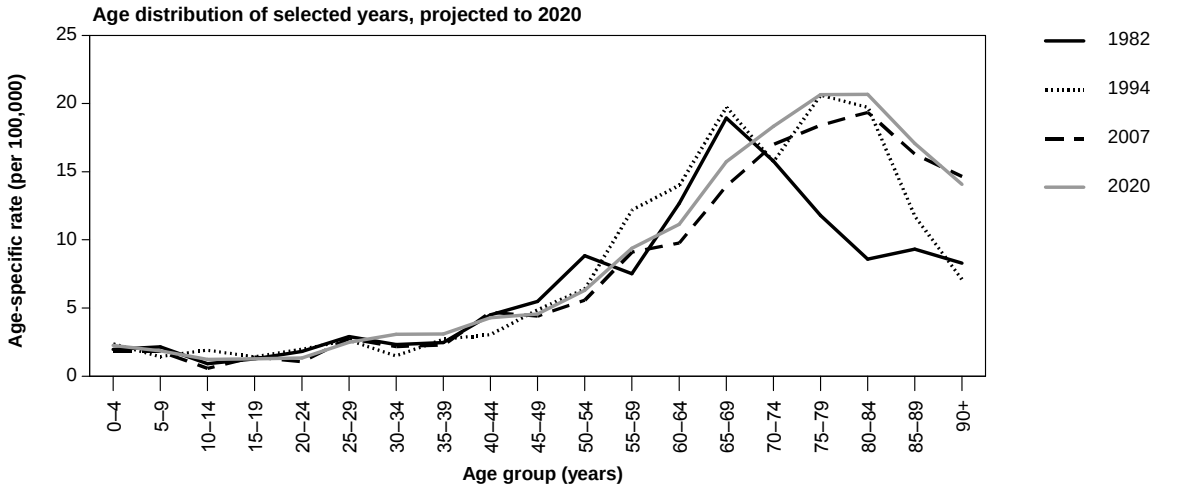
Table 3.4d: Projected number of new cases and age-specific rates, females, 2011–2020: brain cancer

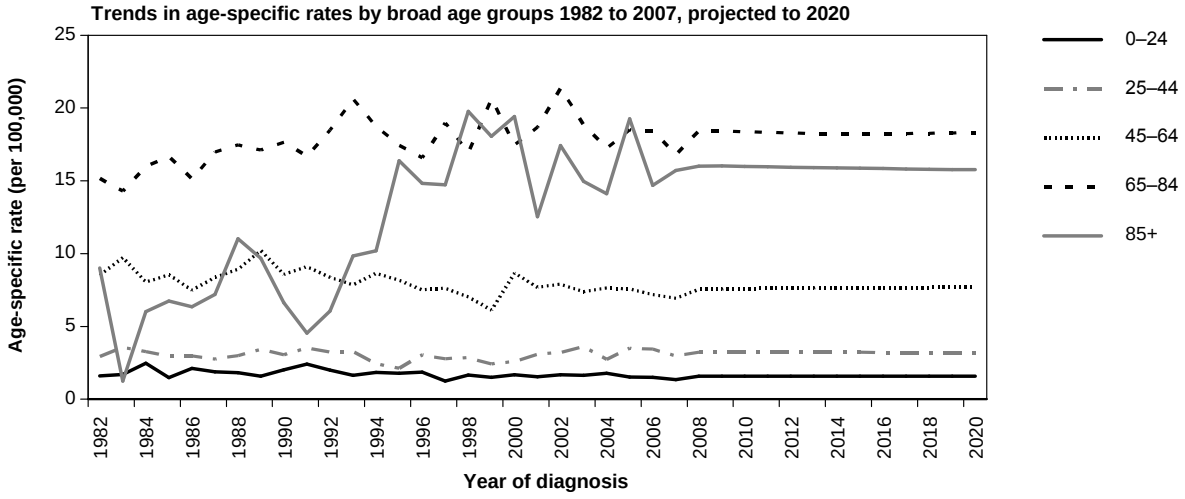
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	55	1.6	100	3.2	215	7.6	260	18.4	45	16.0
2012	55	1.6	105	3.2	220	7.6	270	18.3	45	15.9
2013	55	1.6	105	3.2	220	7.7	280	18.3	45	15.9
2014	60	1.6	105	3.2	225	7.7	290	18.2	50	15.9
2015	60	1.6	105	3.2	230	7.7	300	18.2	50	15.9
2016	60	1.6	105	3.2	230	7.7	310	18.2	50	15.8
2017	60	1.6	110	3.2	235	7.7	320	18.3	50	15.8
2018	60	1.6	110	3.2	240	7.7	330	18.3	50	15.8
2019	60	1.6	110	3.2	240	7.7	340	18.3	55	15.8
2020	60	1.6	110	3.2	245	7.7	355	18.3	55	15.8

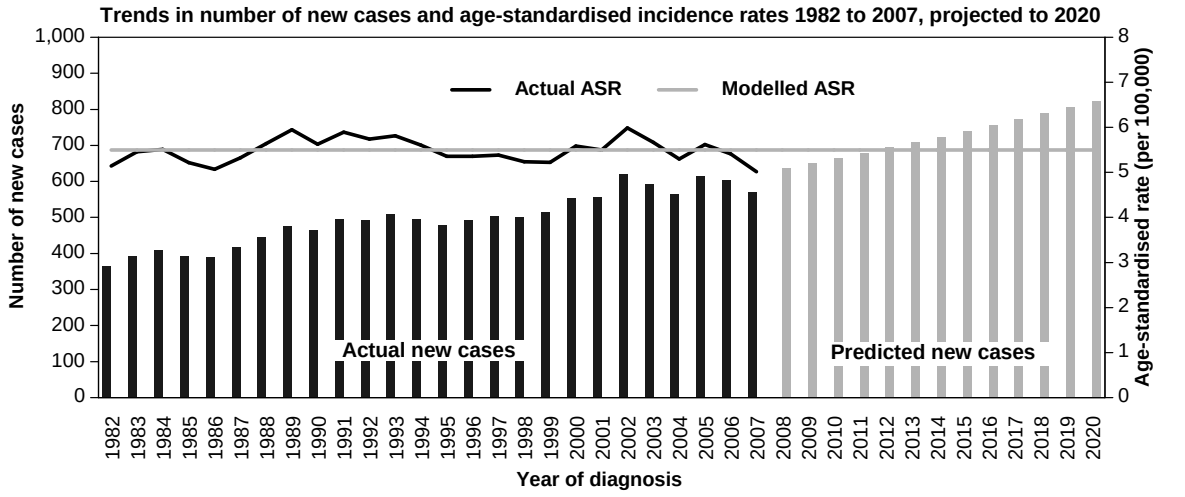
Notes

1. Brain cancer includes ICD-10 code C71.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.4b: Brain cancer (C71), females, Australia: actual 1982–2007 and projected to 2020

Breast cancer in females (C50)

Breast cancer is the most commonly diagnosed cancer in females with 12,567 cases diagnosed in 2007. Joinpoint analysis of the age-standardised rates shows a number of statistically significant variations in the incidence trends in females over time.

A sharply increasing rate of 4.3 cases per 100,000 females per year between 1990 and 1995 is most likely due to the detection of prevalent cases of breast cancer by mammographic screening. Organised mammographic screening programs for breast cancer in females were introduced in each state and territory between 1989 and 1994, offering mammographic screening every 2 years. The program is now known as BreastScreen Australia and targets women aged 50–69, although screening is also offered to women aged 40–49 and 70 or over. In 2008–2009, BreastScreen Australia screened approximately 1.3 million women (AIHW 2011b).

Between 1995 and 2002 age-standardised incidence rates slowed to a non-significant increase of 0.6 cases per 100,000 females per year; however, the most recent linear trend shows that rates have decreased on average by about 1.3 cases per 100,000 females each year from 2002 to 2007, although this trend shows only weak evidence of statistical significance.

This most recent change in trend may possibly be related to reduced use of long-term hormone replacement therapy (HRT). In 2002, results from the Million Women Study in the United Kingdom and the Women's Health Initiative in the United States indicated that long-term HRT was linked to an increased risk of breast cancer. Since that time, use of HRT has declined and a number of countries have reported a corresponding decline in incidence rates in post-menopausal women independent of screening, though the reasons for the decline are not yet fully understood (De et al. 2010) and the effect has not been fully studied in Australia.

Unfortunately, this most recent time series is not sufficient to build a model to project to 2020, and it is unclear whether this trend will continue. Use of a log-linear model shows the most recent log-linear trend to be a non-significant exponential decrease in the age-standardised rate of about 0.15% each year from 1995 onwards. Consequently, the observation window for the projection model of breast cancer in females was set at 1995 to 2007.

From extrapolation of age-specific trends over this period, it is estimated that age-standardised rates will remain constant at about 113–114 cases diagnosed per 100,000 females each year between 2011 and 2020. However, the expected changes to the population over this time means that the projected number of cases diagnosed will continue to increase from an estimated 14,290 in 2011 to 17,210 in 2020 (Table 3.5a). Examination of age-specific trends shows no changes expected in any of the broad age categories (Table 3.5b, Figure 3.5a).

It should be noted that no attempt has been made to model the effect of reduced use of HRT or future effects of developments in breast cancer screening (such as the introduction of digital mammography) on breast cancer incidence. These factors should be taken into consideration when interpreting these data.

Although generally associated with females, males can also develop breast cancer, however, it is far less common. While the number of males diagnosed with breast cancer each year has increased from 62 cases in 1982 to 103 cases in 2007, it is still rare, and the age-standardised rate has remained largely unchanged over the 26 years for which national data are available (AIHW & NBOCC 2009). Joinpoint analysis shows a non-significant increase of about

0.002 cases per 100,000 males (or 2 cases per 100 million males) each year. For this reason, projections of breast cancer in this report are limited to females.

Table 3.5a: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: breast cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	14,290	13,820	14,750	113.4	109.8	116.9
2012	14,610	14,130	15,100	113.4	109.7	117.0
2013	14,940	14,440	15,450	113.4	109.7	117.2
2014	15,270	14,740	15,800	113.5	109.7	117.3
2015	15,600	15,050	16,150	113.5	109.6	117.4
2016	15,930	15,360	16,510	113.6	109.6	117.5
2017	16,250	15,650	16,850	113.6	109.6	117.7
2018	16,570	15,950	17,200	113.7	109.5	117.8
2019	16,890	16,240	17,550	113.7	109.5	118.0
2020	17,210	16,530	17,890	113.8	109.5	118.1

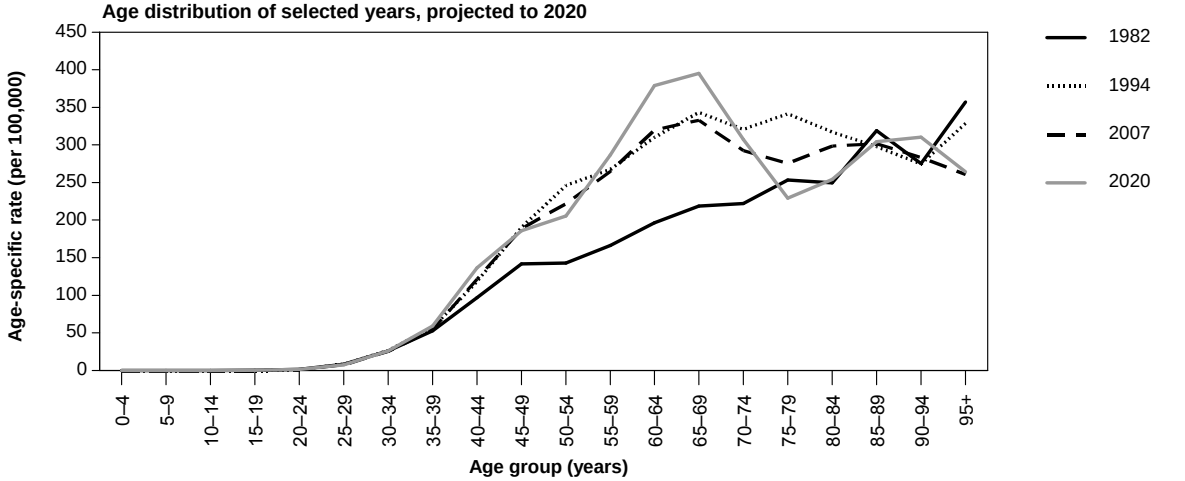
Table 3.5b: Projected number of new cases and age-specific rates, females, 2011–2020: breast cancer

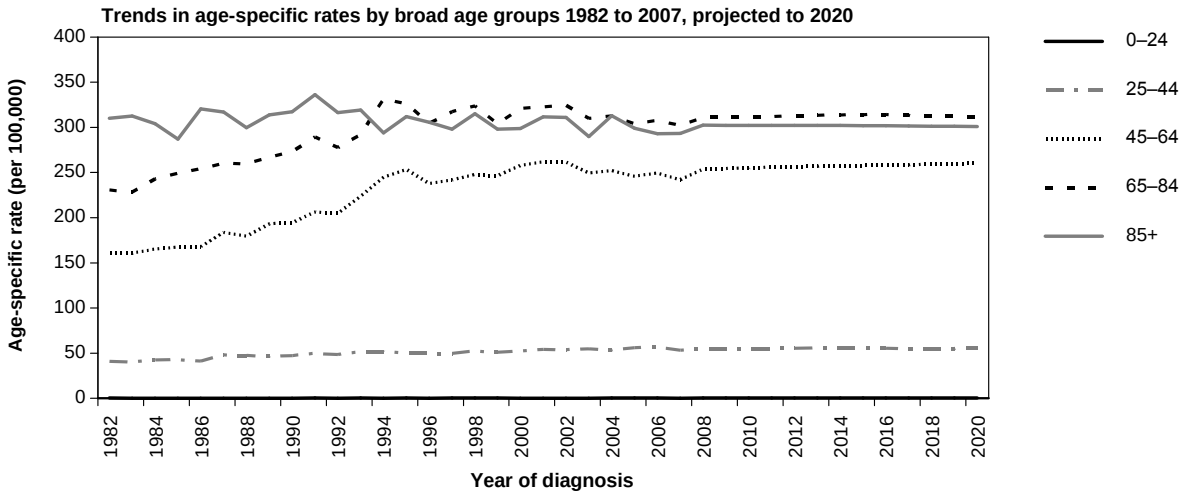
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	10	0.3	1,740	55.3	7,300	256.2	4,410	311.9	830	302.1
2012	10	0.3	1,770	55.7	7,370	256.5	4,610	312.8	860	302.1
2013	10	0.3	1,800	55.9	7,460	257.0	4,780	313.6	885	302.1
2014	10	0.3	1,820	56.0	7,580	257.5	4,950	313.9	910	302.1
2015	10	0.3	1,840	55.9	7,680	258.0	5,130	314.2	940	302.0
2016	10	0.3	1,840	55.5	7,810	258.3	5,310	314.4	960	301.7
2017	10	0.3	1,850	55.3	7,940	258.7	5,480	313.7	975	301.5
2018	10	0.3	1,870	55.3	8,040	259.2	5,670	313.3	990	301.4
2019	10	0.3	1,890	55.4	8,140	259.9	5,850	312.7	1,000	301.2
2020	10	0.3	1,930	55.7	8,230	260.5	6,030	312.0	1,020	301.0

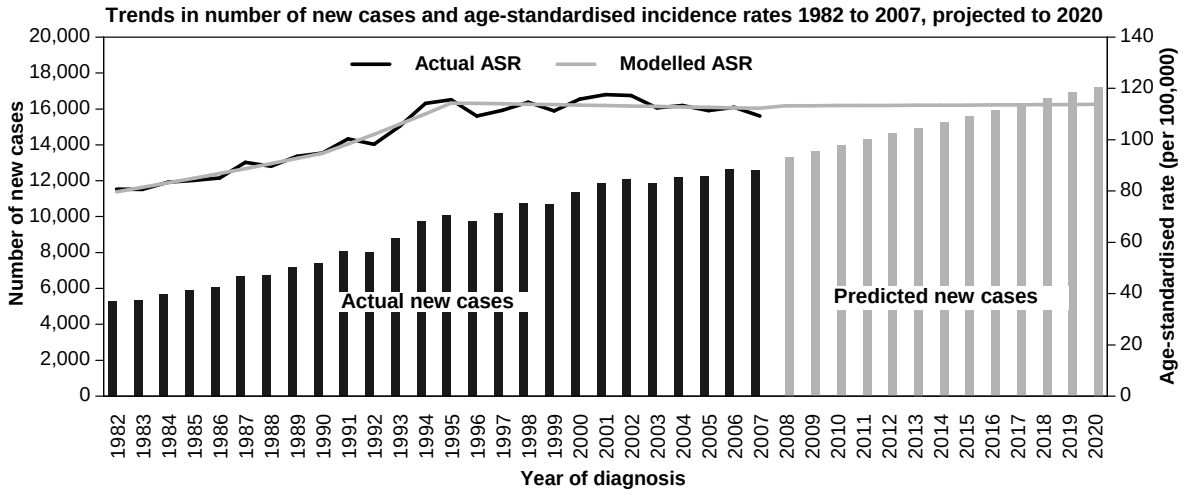
Notes

1. Breast cancer includes ICD-10 code C50.
2. Projected estimates are based on incidence data for 1995 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.5a: Breast cancer (C50), females, Australia: actual 1982–2007 and projected to 2020

Cervical cancer (C53)

Cervical cancer is the twelfth most common cancer diagnosed in Australian females and accounts for about 17% of all gynaecological cancers with 739 cases new cases (6.8 per 100,000 females age-standardised) diagnosed in 2007.

Although screening for cervical abnormalities has been available since the 1960s, a national organised screening program to detect women at risk of cervical cancer was introduced in Australia in the early 1990s that targets women between the ages of 20 and 69. This program is known as the National Cervical Screening Program.

Joinpoint analysis of age-standardised rates of cervical cancer showed statistically significant changes in the trends over time. Rates have been declining since 1982 (from when national data are available) as pre-cancerous conditions were detected and treated before they progressed to cancer. However, a significant drop in incidence rates between 1991 and 2001 is consistent with the introduction of an organised approach to cervical screening.

The most recent trend from 2001 to 2007 shows a slight but non-significant decrease in the age-standardised rate of about 0.07 cases per 100,000 females per year and is used as the observation window for calculating projections to 2020.

Extrapolation of this trend estimates that age-standardised incidence rates will remain constant at about 7 new cases diagnosed per 100,000 females between 2011 and 2020. Actual incidence is expected to increase from about 800 new cases in 2011 to 915 new cases in 2020 with the expected changes in the population (Table 3.6a).

Analysis of age-specific projections showed that incidence rates of cervical cancer in females aged 25–44 and 45–64 are expected to remain steady at 9.4 and 10.4 cases per 100,000 females, while rates for females aged 65–84 are expected to decline slowly to a similar rate (10.5 per 100,000 females) by 2020. Rates for females aged 85 and over are expected to remain constant at about 14 cases per 100,000 females (Table 3.6b, Figure 3.6a).

During the last decade there has been a greater understanding of the natural history of cervical cancer and it is now recognised that cervical cancer is a rare outcome of persistent infection with human papillomavirus (HPV). As a result of this new knowledge, Australia introduced the National HPV Vaccination Program in April 2007 to protect young Australian women against infection with HPV and further reduce cervical cancer incidence (AIHW & DoHA 2011).The effect of the HPV vaccination program on incidence of cervical cancer will not be evident for some time, and is not accounted for in these projections; however, the potential effects should be considered when interpreting these data.

Table 3.6a: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: cervical cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	800	740	865	6.8	6.3	7.4
2012	815	750	875	6.8	6.3	7.4
2013	825	760	890	6.8	6.2	7.3
2014	840	775	905	6.8	6.2	7.3
2015	850	785	920	6.8	6.2	7.3
2016	865	795	935	6.8	6.2	7.3
2017	880	810	945	6.8	6.2	7.3
2018	890	820	960	6.8	6.2	7.3
2019	905	835	975	6.7	6.2	7.3
2020	915	845	990	6.7	6.2	7.3

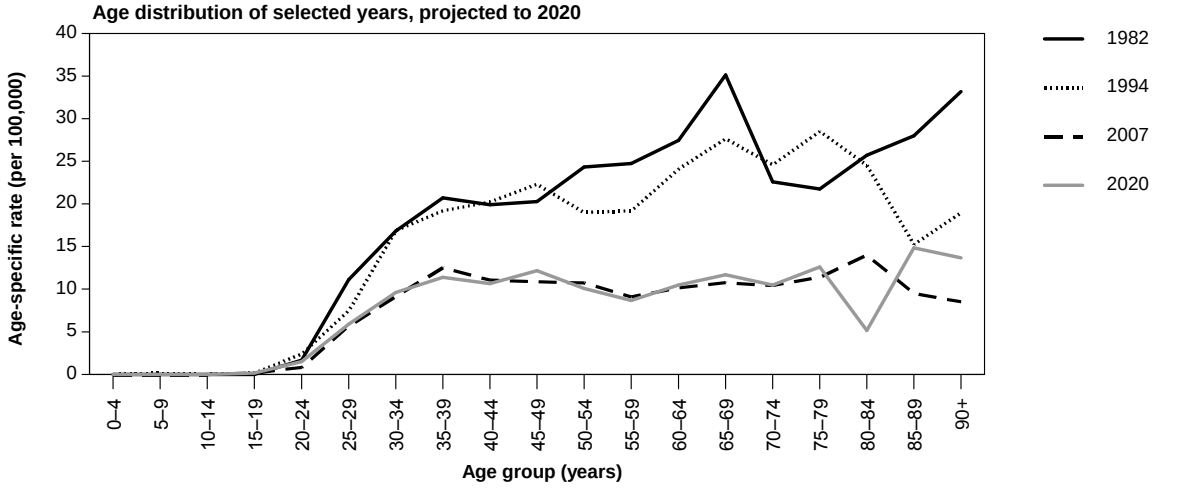
Table 3.6b: Projected number of new cases and age-specific rates, females, 2011–2020: cervical cancer

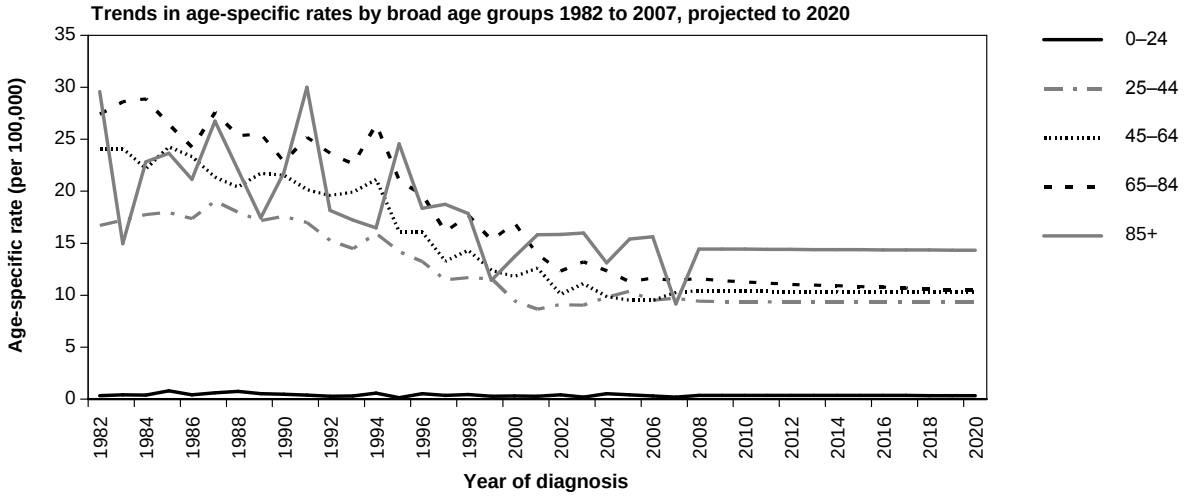
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	15	0.4	295	9.4	295	10.4	160	11.2	40	14.4
2012	15	0.4	300	9.4	300	10.4	165	11.1	40	14.4
2013	15	0.4	300	9.4	300	10.4	170	11.0	40	14.4
2014	15	0.4	305	9.4	305	10.4	170	10.9	45	14.4
2015	15	0.4	310	9.4	310	10.4	175	10.9	45	14.4
2016	15	0.4	310	9.4	315	10.4	180	10.8	45	14.4
2017	15	0.4	315	9.4	320	10.4	185	10.7	45	14.4
2018	15	0.4	315	9.4	320	10.4	190	10.6	45	14.3
2019	15	0.4	320	9.4	325	10.4	200	10.6	50	14.3
2020	15	0.3	325	9.4	330	10.4	205	10.5	50	14.3

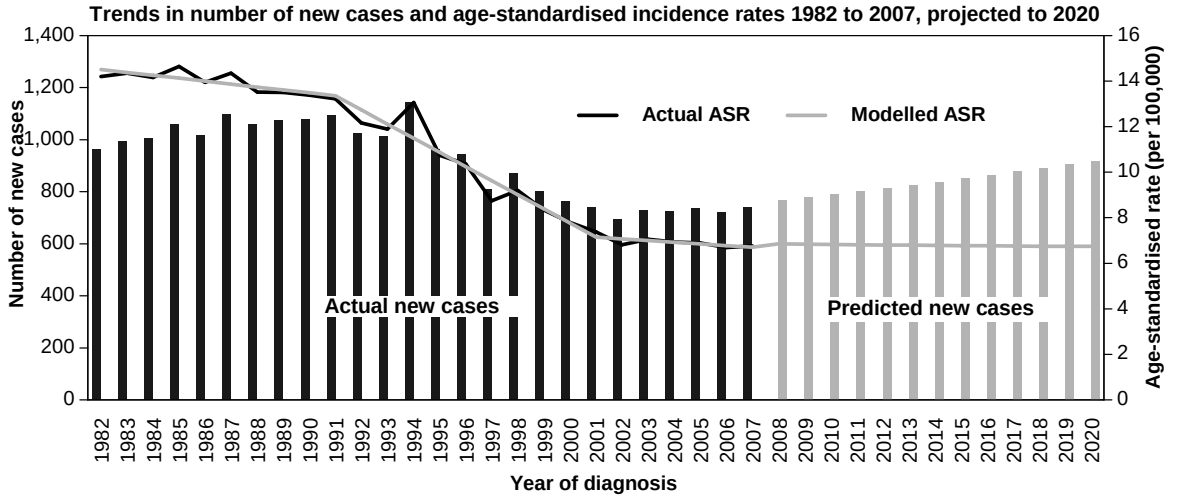
Notes

1. Cervical cancer includes ICD-10 code C53.
2. Projected estimates are based on incidence data for 2001 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.6a: Cervical cancer (C53), females, Australia: actual 1982–2007 and projected to 2020

Hodgkin lymphoma (C81)

Hodgkin lymphoma is a rare disease, accounting for 538 or 0.5% of new cases of cancer in 2007. Males are more likely to develop the disease, with the risk to the age of 85 being 1 in 425 males and 1 in 480 females. While most cancers are common among older age groups, Hodgkin lymphoma affects comparatively younger age groups with a mean age of diagnosis of 40.

The exact causes of Hodgkin lymphoma remain unknown; however, there is sufficient evidence of a causal association between Hodgkin lymphoma and infection with Epstein Barr virus and also with human immunodeficiency virus (HIV) (Jarrett 2002; Grulich et al. 2007b; Bouvard et al. 2009). Childhood environment has also been associated with increased risk of Hodgkin lymphoma. Some research has suggested this may be related to decreased or delayed exposure to a common infectious agent in childhood (Chang et al. 2004; Chatenoud et al. 2005). Another risk factor associated with the disease is a family history of blood or lymphatic cancers (Chang et al. 2005).

Males

Joinpoint analysis of age-standardised incidence rates in males showed a significant change in the trend of Hodgkin lymphoma. Rates decreased significantly from 1982 to 1993 at about 0.04 cases per 100,000 males per year; however, from 1993 to 2007, rates increased at about 0.04 cases per 100,000 males per year. Using data from the most recent trend to model projections, it is expected that age-standardised rates will continue to increase to about 2.8 new cases diagnosed per 100,000 males in 2020, equating to approximately 330 cases (Table 3.7a). Rates are expected to show a slight increase for age groups 25–44 and 65–84, and a small decrease for those aged 0–24 (Table 3.7b, Figure 3.7a).

Females

Joinpoint analysis of age-standardised rates of Hodgkin lymphoma in females showed a clear increasing trend from 1982 to 2007. Extrapolation of age-specific trends from 1982 onwards suggests that age-standardised rates will continue to increase to about 2.5 new cases diagnosed per 100,000 females in 2020, equating to approximately 295 cases (Table 3.7c). Rates are expected to increase in females aged 0–24, 25–44 and 45–64; rates for older women are expected to remain stable (Table 3.7d, Figure 3.7b).

Table 3.7a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: Hodgkin lymphoma

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	280	240	325	2.6	2.3	3.0
2012	285	245	330	2.7	2.3	3.0
2013	295	250	335	2.7	2.3	3.0
2014	300	255	340	2.7	2.3	3.1
2015	305	260	350	2.7	2.4	3.1
2016	310	265	355	2.7	2.4	3.1
2017	315	265	360	2.7	2.4	3.1
2018	320	270	365	2.8	2.4	3.1
2019	325	275	370	2.8	2.4	3.1
2020	330	280	380	2.8	2.4	3.2

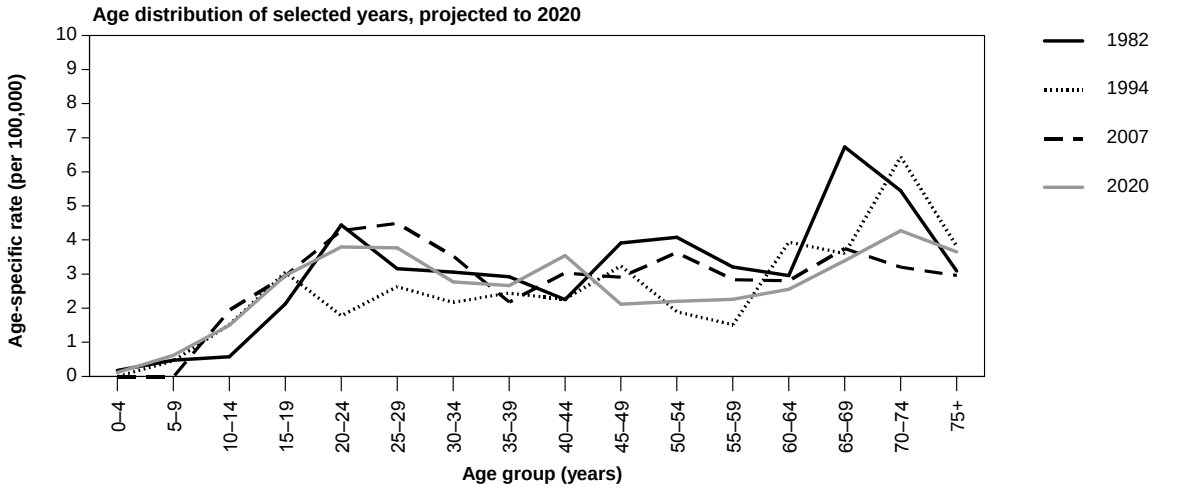
Table 3.7b: Projected number of new cases and age-specific rates, males, 2011–2020: non-Hodgkin lymphoma

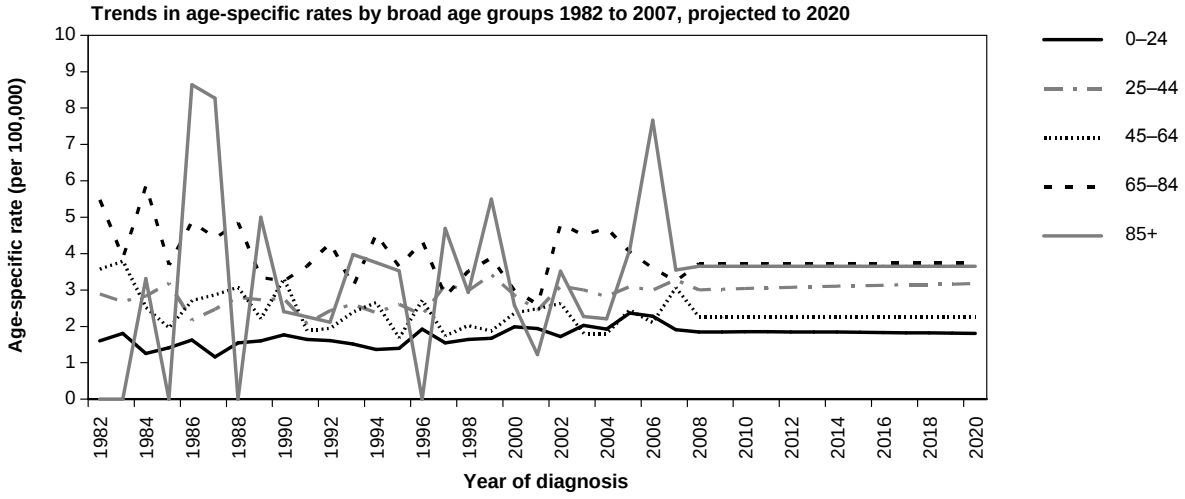
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	70	1.9	95	3.1	65	2.3	50	3.7	5	3.7
2012	70	1.9	100	3.1	65	2.3	50	3.7	5	3.7
2013	70	1.8	100	3.1	65	2.3	50	3.7	5	3.7
2014	70	1.8	100	3.1	65	2.3	55	3.7	5	3.7
2015	70	1.8	105	3.1	65	2.3	55	3.7	5	3.7
2016	70	1.8	105	3.1	65	2.3	60	3.7	5	3.7
2017	70	1.8	105	3.1	70	2.3	60	3.7	5	3.7
2018	70	1.8	110	3.2	70	2.3	65	3.7	5	3.7
2019	70	1.8	110	3.2	70	2.3	65	3.7	5	3.7
2020	70	1.8	110	3.2	70	2.3	65	3.8	10	3.7

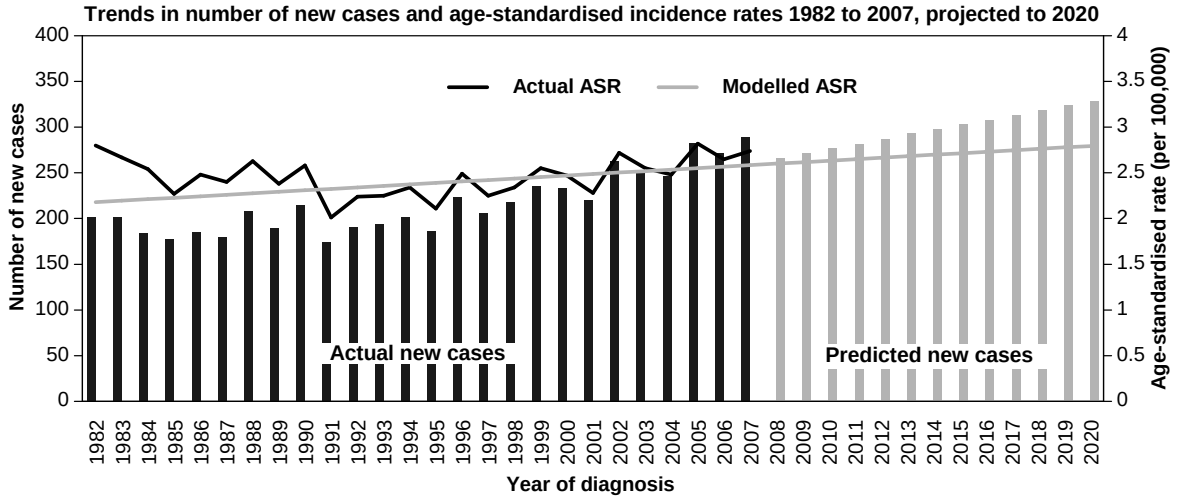
Notes

1. Hodgkin lymphoma includes ICD-10 code C81.
2. Projected estimates are based on incidence data for 1993 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.7a: Hodgkin lymphoma (C81), males, Australia: actual 1982–2007 and projected to 2020

Table 3.7c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: Hodgkin lymphoma

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	245	210	280	2.3	2.0	2.6
2012	250	215	285	2.3	2.0	2.6
2013	255	220	290	2.3	2.0	2.6
2014	260	225	295	2.4	2.1	2.7
2015	265	230	300	2.4	2.1	2.7
2016	270	235	310	2.4	2.1	2.7
2017	275	240	315	2.5	2.1	2.8
2018	280	245	320	2.5	2.2	2.8
2019	290	250	330	2.5	2.2	2.8
2020	295	255	335	2.5	2.2	2.8

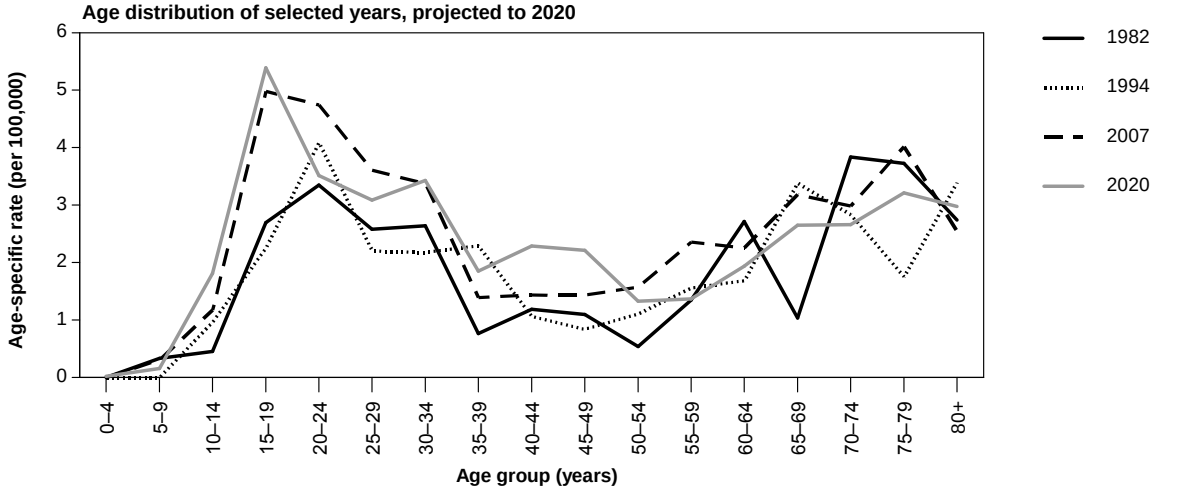
Table 3.7d: Projected number of new cases and age-specific rates, females, 2011–2020: Hodgkin lymphoma

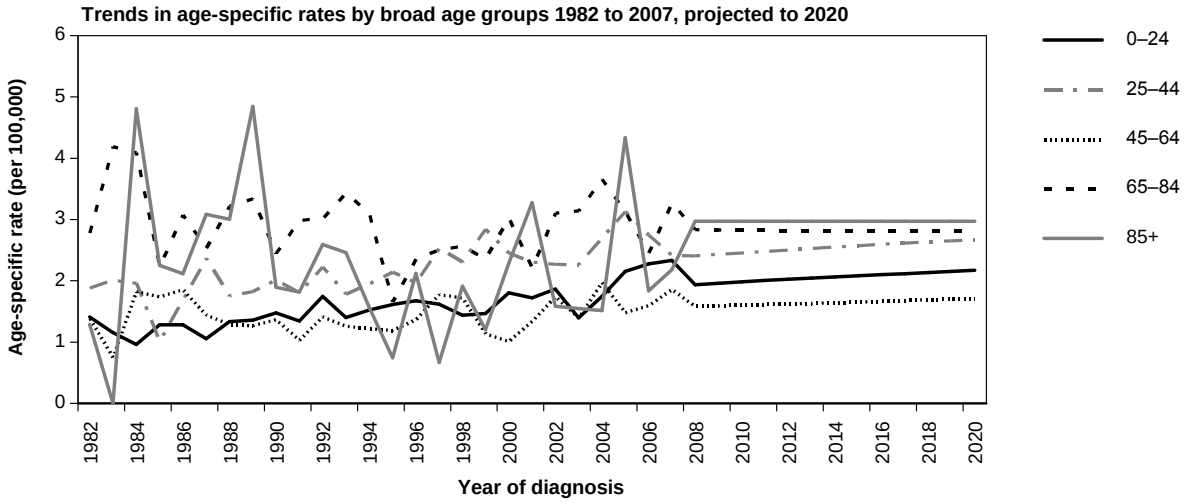
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	70	2.0	80	2.5	45	1.6	40	2.8	10	3.0
2012	70	2.0	80	2.5	45	1.6	40	2.8	10	3.0
2013	75	2.0	80	2.5	45	1.6	45	2.8	10	3.0
2014	75	2.1	85	2.6	50	1.6	45	2.8	10	3.0
2015	75	2.1	85	2.6	50	1.7	45	2.8	10	3.0
2016	75	2.1	85	2.6	50	1.7	50	2.8	10	3.0
2017	80	2.1	90	2.6	50	1.7	50	2.8	10	3.0
2018	80	2.1	90	2.6	55	1.7	50	2.8	10	3.0
2019	80	2.2	90	2.7	55	1.7	55	2.8	10	3.0
2020	85	2.2	90	2.7	55	1.7	55	2.8	10	3.0

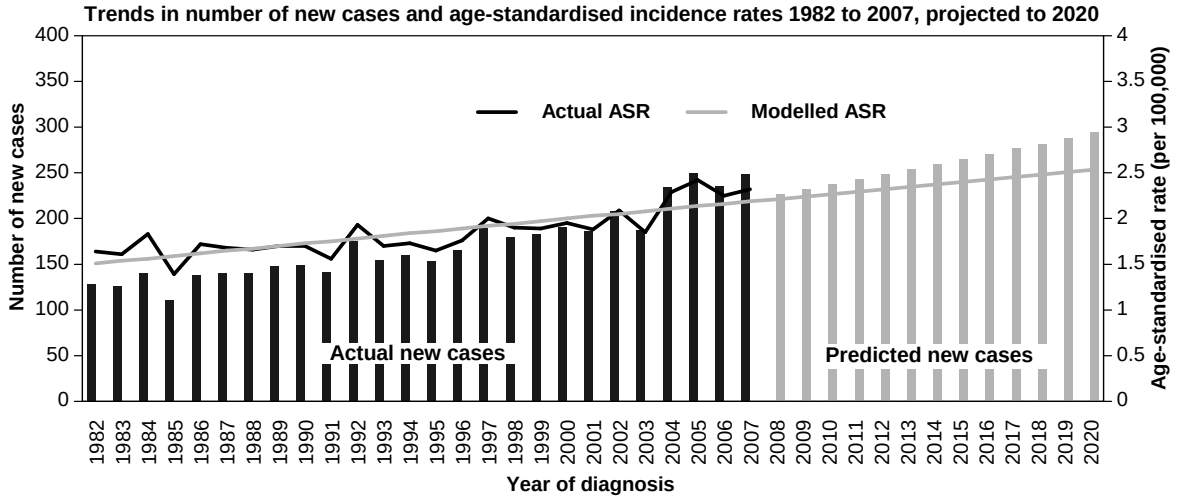
Notes

1. Hodgkin lymphoma includes ICD-10 code C81
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.7b: Hodgkin lymphoma (C81), females, Australia: actual 1982–2007 and projected to 2020

Kidney cancer (C64)

Kidney cancer accounted for 2.4% of all cancer diagnoses in 2007, making it the eighth most common cancer diagnosed in Australia. Usually only one kidney is affected, but in rare cases the cancer may develop in both kidneys. Kidney cancer is often diagnosed late in its progression, which usually leads to poorer outcomes.

There are many factors associated with the development of kidney cancer but the key risk factors include age, sex, cigarette smoking, high Body Mass Index (BMI) and genetic/medical conditions (IARC 2008). Kidney cancer is more common in older people, with the average age at diagnosis 63.5. The relationship between cigarette smoking and kidney cancer is dose-related, with heavy smokers 2 to 3 times more likely to develop the disease than those who have never smoked (Hunt et al. 2005; Scélo & Brennan 2007). Further, an estimated 25% of cases of kidney cancers result from being overweight or obese, particularly in women (Bergström et al. 2001; Scélo & Brennan 2007).

While the number of smoking-related kidney cancers is likely to have declined over time due to the decline in the smoking rate in Australia over the past three to five decades (OECD 2010), the number of age- and weight-related kidney cancers is likely to have increased due to an increasing number of Australians who are overweight or obese.

The increasing trend in both males and females has also been attributed to more sophisticated imaging techniques, which lead to the incidental detection of kidney cancers in both males and females as a consequence of investigating other kidney diseases (Masood et al. 2001; Hollingsworth et al. 2006).

Males

Joinpoint analysis of the age-standardised incidence rates of kidney cancer for males show a slight, but statistically significant increasing trend of about 0.3 cases per 100,000 males per year from 1982 to 2007.

Extrapolation of the trend from 1982 to 2007 suggests that age-standardised rates will increase to 19.8 new cases diagnosed per 100,000 males in 2020 which, when taking into account the expected changes to the population, will equate to an estimated 2,910 new cases (Table 3.8a). The greatest rate of increase is expected to be in males aged 65–84, and 85 and over (Table 3.8b, Figure 3.8a).

Females

Kidney cancer rates in females have similarly increased since 1982; however, joinpoint analysis showed a significant change in the trend with the rate changing from an increase of 0.2 cases per 100,000 females per year from 1982 to 1992 to an increase of less than 0.1 case per year from 1992 to 2007.

Extrapolation of the age-specific trends from 1992 onwards suggests that age-standardised rates will increase only slightly from 7.4 new cases per 100,000 females in 2007 to 7.7 in 2020 which, when taking into account the expected changes to the population, will equate to an estimated 1,220 new cases (Table 3.8c).

Unlike males, the greatest increase in rates is expected in females aged 45–64.

Table 3.8a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: kidney cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	2,020	1,910	2,120	17.3	16.3	18.2
2012	2,110	2,000	2,210	17.5	16.6	18.4
2013	2,200	2,080	2,310	17.8	16.9	18.7
2014	2,290	2,170	2,410	18.1	17.2	19.0
2015	2,390	2,270	2,510	18.4	17.5	19.3
2016	2,490	2,360	2,610	18.7	17.7	19.6
2017	2,590	2,460	2,720	19.0	18.0	19.9
2018	2,690	2,560	2,830	19.3	18.3	20.2
2019	2,800	2,660	2,940	19.5	18.6	20.5
2020	2,910	2,760	3,060	19.8	18.8	20.8

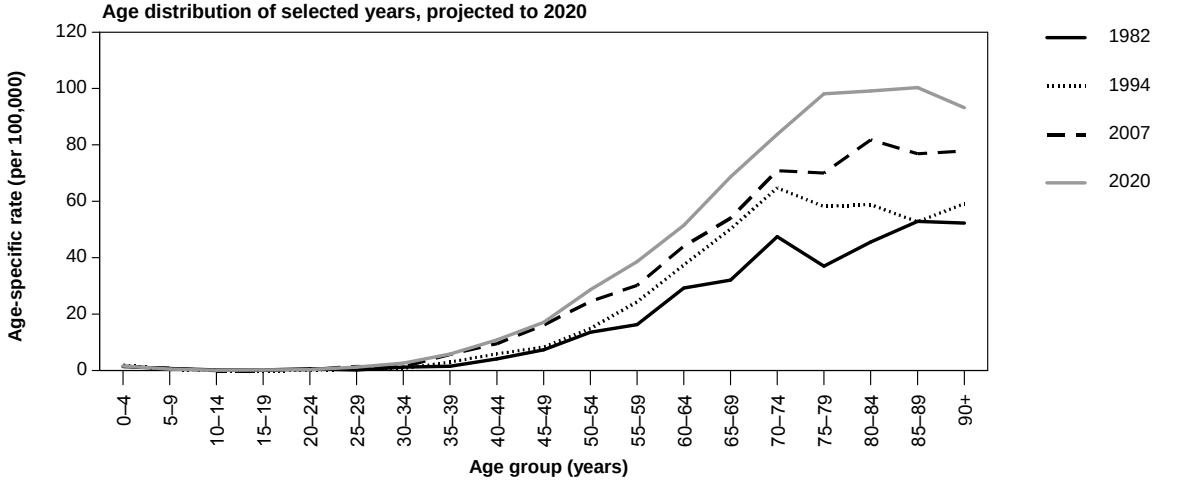
Table 3.8b: Projected number of new cases and age-specific rates, males, 2011–2020: kidney cancer

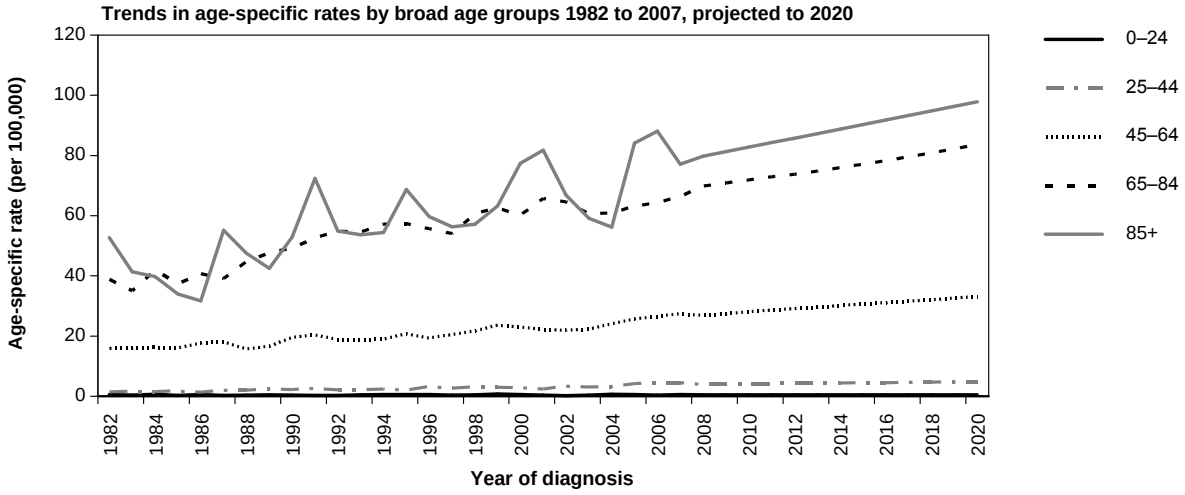
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	20	0.5	135	4.2	800	28.7	935	72.9	125	84.3
2012	20	0.5	140	4.3	820	29.1	995	73.8	135	85.8
2013	20	0.5	145	4.4	845	29.7	1,050	74.8	145	87.3
2014	20	0.5	145	4.5	870	30.2	1,100	76.0	150	88.8
2015	20	0.5	150	4.6	895	30.7	1,160	77.1	160	90.3
2016	20	0.5	155	4.6	920	31.1	1,220	78.3	170	91.8
2017	20	0.5	160	4.7	945	31.6	1,290	79.7	180	93.3
2018	20	0.5	165	4.7	970	32.1	1,350	81.0	185	94.8
2019	20	0.5	170	4.8	995	32.7	1,420	82.3	195	96.3
2020	20	0.5	175	4.9	1,020	33.2	1,490	83.6	205	97.8

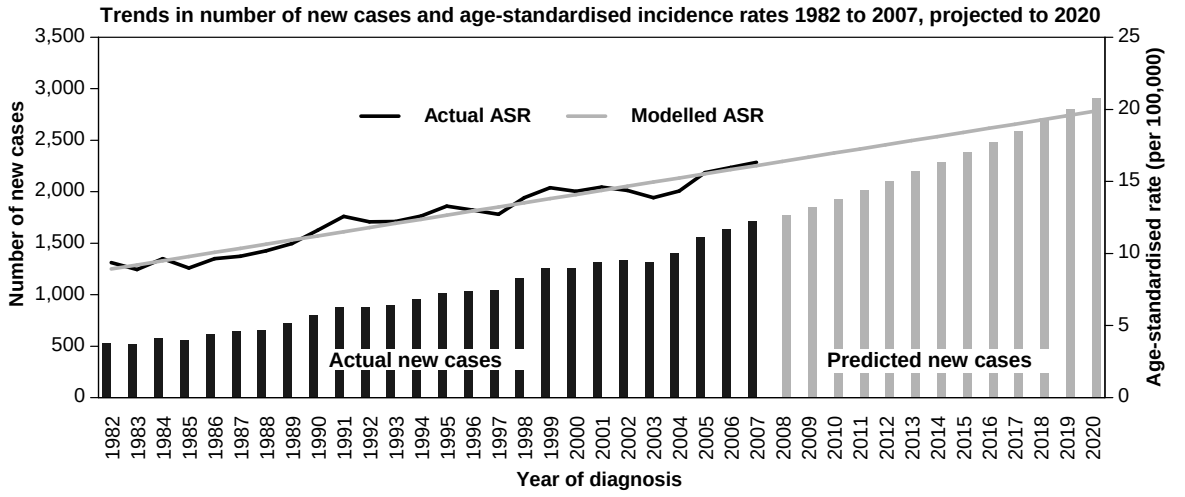
Notes

1. Kidney cancer includes ICD-10 code C64.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.8a: Kidney cancer (C64), males, Australia: actual 1982–2007 and projected to 2020

Table 3.8c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: kidney cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	950	875	1,030	7.4	6.8	8.0
2012	980	900	1,060	7.4	6.8	8.0
2013	1,010	930	1,090	7.5	6.9	8.1
2014	1,040	955	1,120	7.5	6.9	8.1
2015	1,070	980	1,150	7.5	6.9	8.1
2016	1,100	1,010	1,190	7.6	6.9	8.2
2017	1,130	1,040	1,220	7.6	7.0	8.2
2018	1,160	1,070	1,250	7.6	7.0	8.3
2019	1,190	1,100	1,290	7.7	7.0	8.3
2020	1,220	1,120	1,320	7.7	7.1	8.3

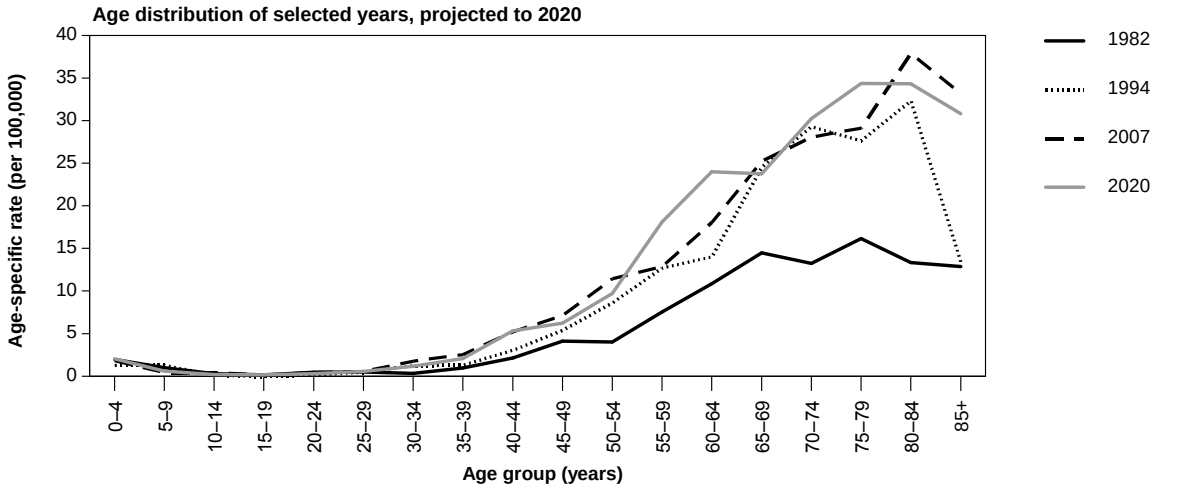
Table 3.8d: Projected number of new cases and age-specific rates, females, 2011–2020: kidney cancer

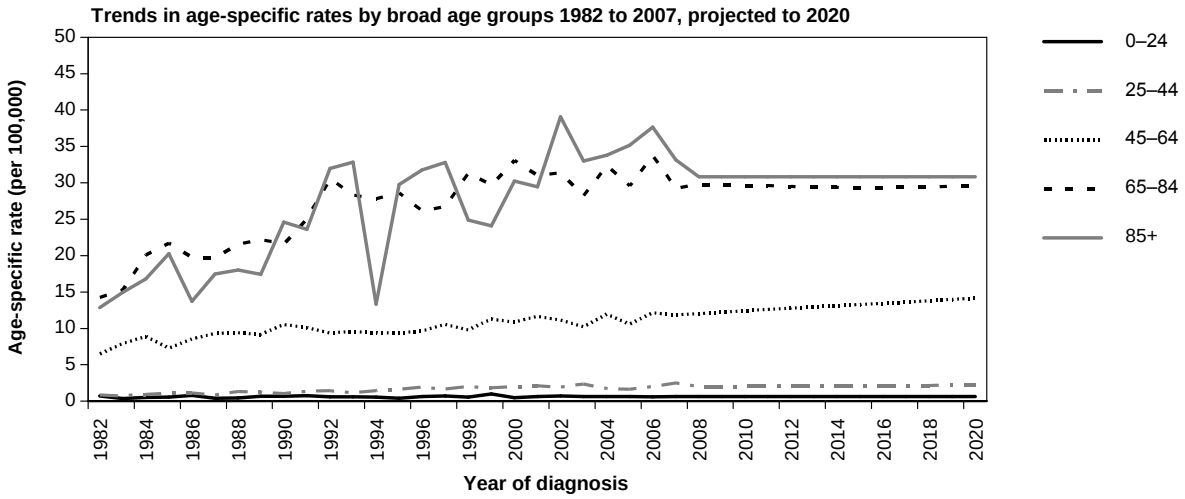
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	25	0.6	65	2.1	360	12.6	420	29.6	85	30.8
2012	25	0.6	70	2.1	365	12.8	435	29.5	85	30.8
2013	25	0.6	70	2.2	375	13.0	450	29.4	90	30.8
2014	25	0.6	70	2.2	385	13.1	465	29.4	95	30.8
2015	25	0.6	70	2.2	395	13.3	480	29.4	95	30.8
2016	25	0.6	70	2.2	405	13.5	495	29.4	100	30.8
2017	25	0.6	75	2.2	420	13.6	515	29.4	100	30.8
2018	25	0.6	75	2.2	430	13.8	535	29.5	100	30.8
2019	25	0.6	75	2.2	440	14.0	550	29.5	105	30.8
2020	25	0.6	75	2.2	445	14.2	570	29.6	105	30.8

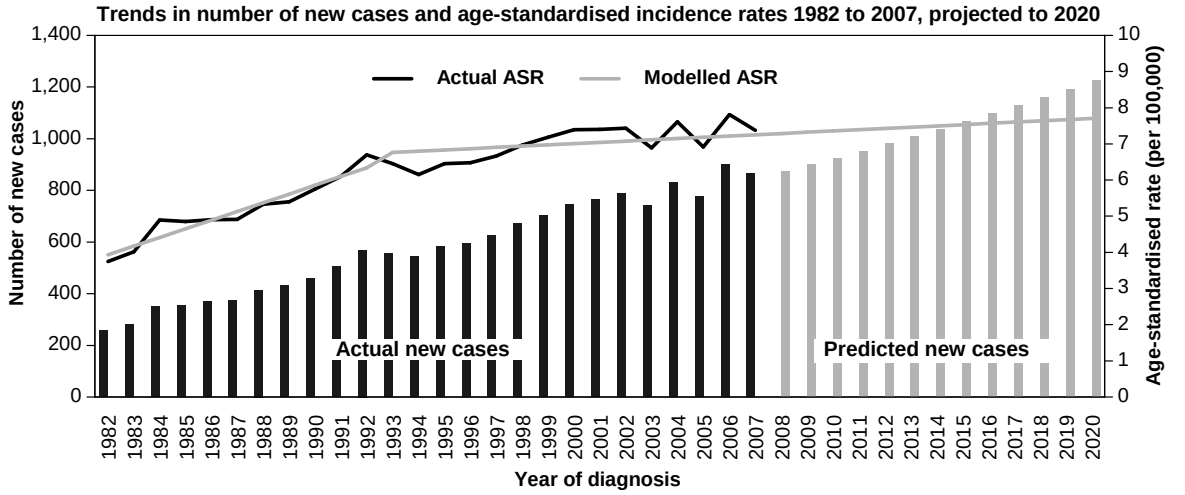
Notes

1. Kidney cancer includes ICD-10 code C64.
2. Projected estimates are based on incidence data for 1992 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.8b: Kidney cancer (C64), females, Australia: actual 1982–2007 and projected to 2020

Liver cancer (C22)

Liver cancer can be divided into two types—primary liver cancer (that is, cancer that originates in the liver) and secondary liver cancer (that is, cancer that develops elsewhere in the body and spreads to the liver). While secondary liver cancer is 20 times more common than primary liver cancer (Cancer Council NSW 2007), only primary cases are recorded in the ACD; hence, the discussion below and associated projections refer to primary liver cancer only.

Primary liver cancer was diagnosed in 1,169 people in Australia in 2007 and is nearly three times more common in males than females. Age-standardised incidence rates have been steadily increasing for both males and females since national data have been collected; however, this increasing trend is primarily in those people aged 65 and over.

A number of risk factors have been associated with liver cancer, including age, sex, being overweight (or having a high BMI) and alcoholic liver cirrhosis, but the most important risk factor is infection with hepatitis B and C virus (Ganem & Prince 2004).

Although the incidence of liver cancer is relatively low in Australia compared with other regions of the world (IARC 2008), it has increased throughout the past two decades. Analysis of incidence rates by area in New South Wales show liver cancer to be higher in areas that have a large overseas-born population, especially those from south-eastern and eastern Asia and middle and western Africa, where hepatitis B and C infections are endemic (Alam et al. 2009). It is important to note that these projections are based on projected population structures by age and sex—while population projections account for immigration no attempt has been made to model changes in incidence based on immigration trends by country of origin.

The Australian Government Department of Health and Ageing recommends immunisation against hepatitis B for all infants as part of the National Immunisation Program Schedule, as well as adult subgroups who have risk of contracting the disease (DoHA 2008). As this immunisation becomes more widespread it could affect the incidence of liver cancer in the future.

Males

Joinpoint analysis of age-standardised rates shows liver cancer in males to be increasing significantly at about 0.2 cases per 100,000 males per year since 1982. Extrapolation of age-specific trends from 1982 to 2007 indicate that rates are expected to continue to rise at a similar rate for those aged 65–84, and 85 and over, as well as a smaller increase in the rates for those aged 45–64. Age-standardised rates are expected to reach 11.0 per 100,000 males in 2020 which, taking into account expected changes to the population structure, will translate to an estimated 1,640 new cases diagnosed (tables 3.9a and 3.9b, Figure 3.9a).

Females

Joinpoint analysis of age-standardised incidence rates show the annual rate increase in liver cancer in females almost tripled from a small but significant increase of about 0.05 cases per 100,000 females per year from 1982 to 1996 to about 0.14 cases per 100,000 females per year from 1996 to 2007.

Following this most recent trend, it is expected that liver cancer in females will continue to increase to 4.8 new cases per 100,000 females by 2020 which, taking into account expected changes to the population structure, will translate to an estimated 825 new cases diagnosed

(Table 3.9c). The greatest increase in rates is expected to be in females aged 85 and over, where rates will approach that of males of the same age, followed by those aged 65–84 (Table 3.9c, Figure 3.9b).

Table 3.9a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: liver cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,040	965	1,120	9.0	8.3	9.6
2012	1,100	1,020	1,180	9.2	8.5	9.9
2013	1,160	1,070	1,250	9.4	8.7	10.1
2014	1,220	1,130	1,310	9.6	8.9	10.4
2015	1,280	1,190	1,380	9.9	9.2	10.6
2016	1,350	1,260	1,440	10.1	9.4	10.8
2017	1,420	1,320	1,520	10.3	9.6	11.1
2018	1,490	1,390	1,590	10.6	9.8	11.3
2019	1,560	1,460	1,670	10.8	10.1	11.6
2020	1,640	1,530	1,750	11.0	10.3	11.8

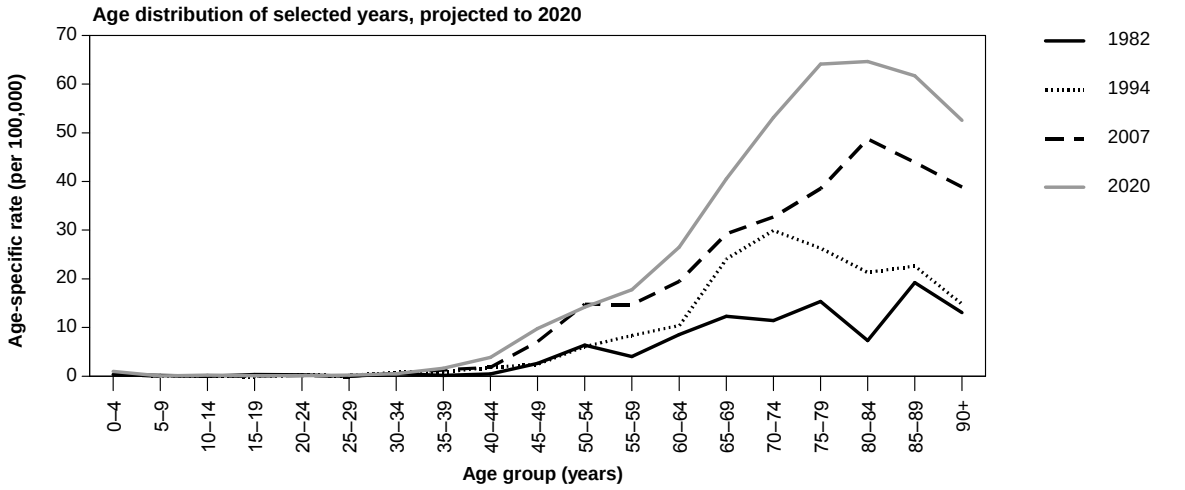
Table 3.9b: Projected number of new cases and age-specific rates, males, 2011–2020: liver cancer

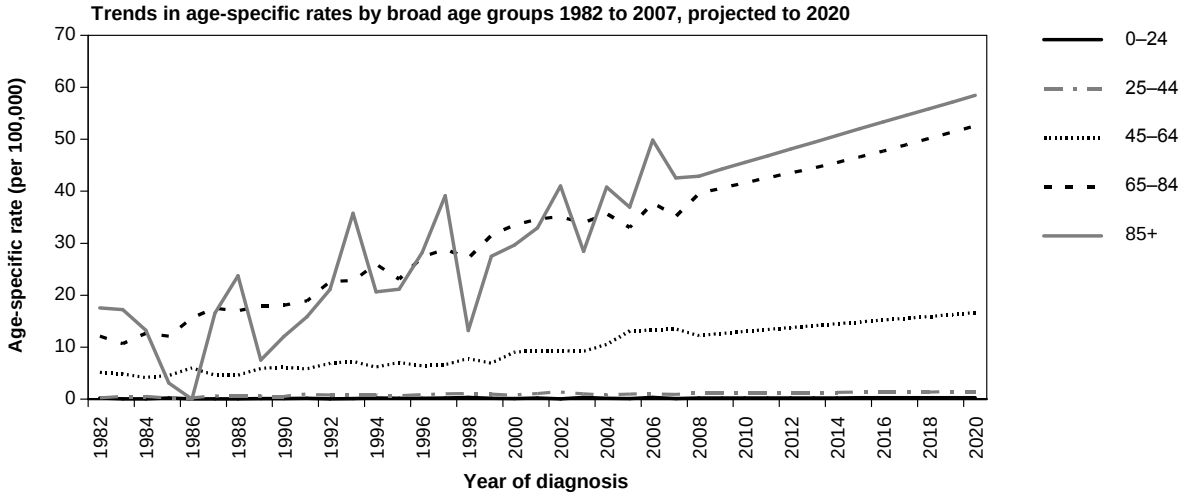
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	10	0.2	40	1.3	375	13.5	550	42.6	70	46.8
2012	10	0.3	40	1.3	390	13.8	585	43.5	75	48.1
2013	10	0.3	45	1.3	405	14.2	625	44.5	80	49.4
2014	10	0.3	45	1.4	420	14.5	660	45.6	85	50.7
2015	10	0.3	45	1.4	435	14.9	700	46.6	95	52.0
2016	10	0.3	45	1.4	450	15.2	745	47.7	100	53.3
2017	10	0.3	50	1.4	465	15.6	790	49.0	105	54.6
2018	10	0.3	50	1.4	480	15.9	840	50.2	110	55.9
2019	10	0.3	50	1.4	500	16.3	885	51.4	115	57.1
2020	10	0.3	50	1.5	515	16.7	940	52.6	120	58.4

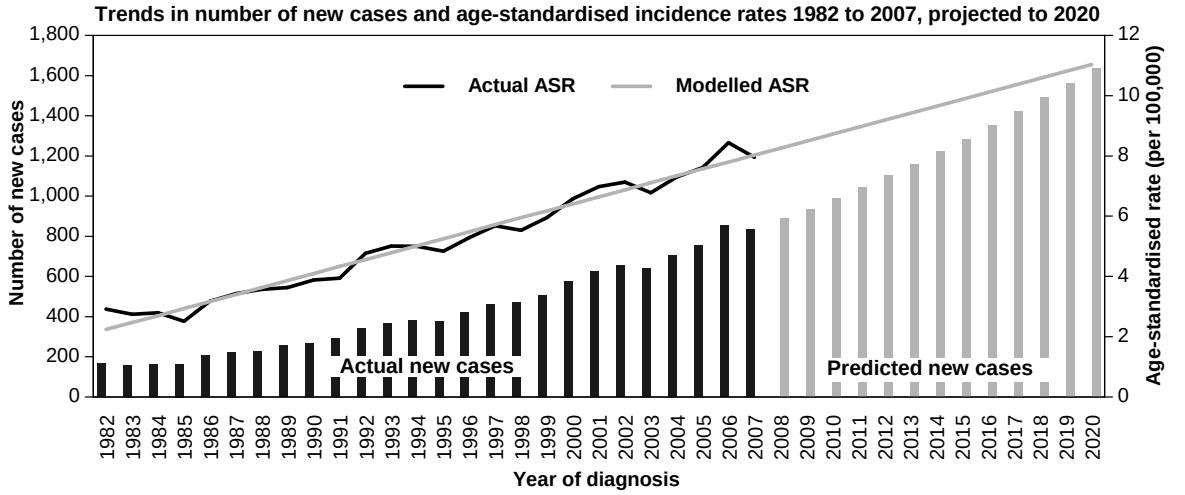
Notes

1. Liver cancer includes ICD-10 code C22.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.9a: Liver cancer (C22), males, Australia: actual 1982–2007 and projected to 2020

Table 3.9c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: liver cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	480	435	530	3.6	3.2	3.9
2012	510	460	565	3.7	3.3	4.1
2013	545	490	600	3.8	3.4	4.2
2014	580	520	635	4.0	3.6	4.4
2015	615	555	675	4.1	3.7	4.5
2016	650	585	715	4.2	3.8	4.7
2017	695	625	765	4.4	3.9	4.8
2018	735	660	810	4.5	4.0	5.0
2019	780	700	860	4.6	4.2	5.1
2020	825	740	910	4.8	4.3	5.3

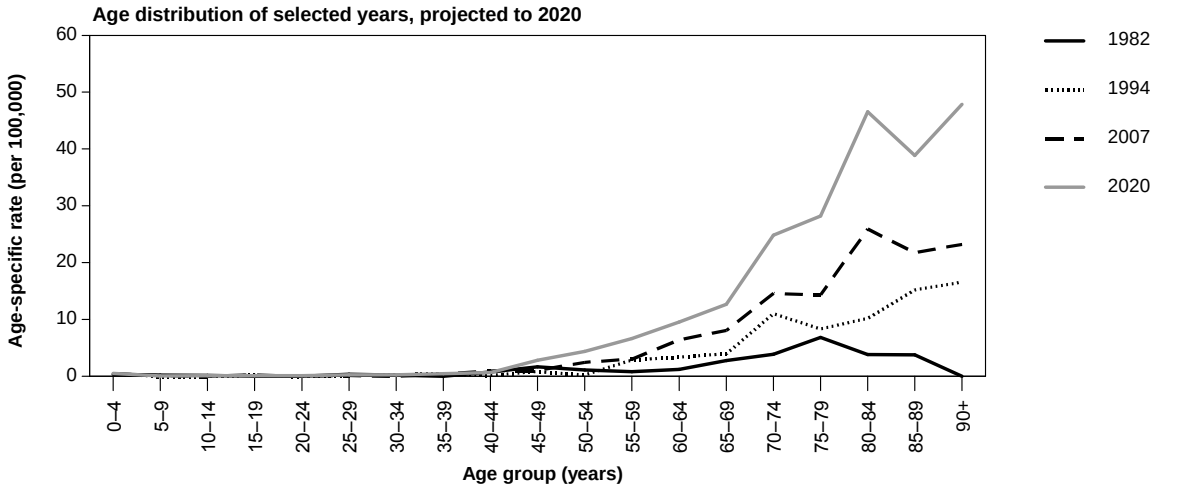
Table 3.9d: Projected number of new cases and age-specific rates, females, 2011–2020: liver cancer

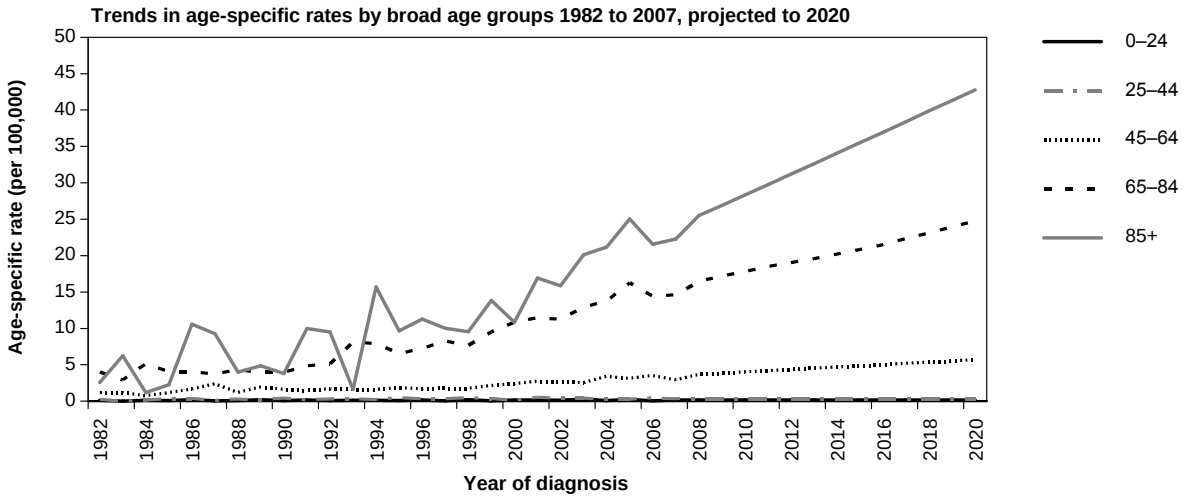
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	5	0.2	10	0.4	120	4.2	260	18.5	80	29.7
2012	5	0.2	10	0.4	125	4.4	280	19.0	90	31.2
2013	5	0.2	15	0.4	130	4.5	300	19.6	95	32.6
2014	5	0.2	15	0.4	140	4.7	320	20.2	105	34.1
2015	5	0.2	15	0.4	145	4.9	340	20.9	110	35.5
2016	5	0.2	15	0.4	150	5.0	365	21.5	120	37.0
2017	5	0.2	15	0.4	160	5.2	390	22.4	125	38.4
2018	5	0.2	15	0.4	165	5.4	420	23.2	130	39.9
2019	5	0.2	15	0.4	175	5.5	450	24.0	140	41.3
2020	5	0.2	15	0.4	180	5.7	480	24.8	145	42.8

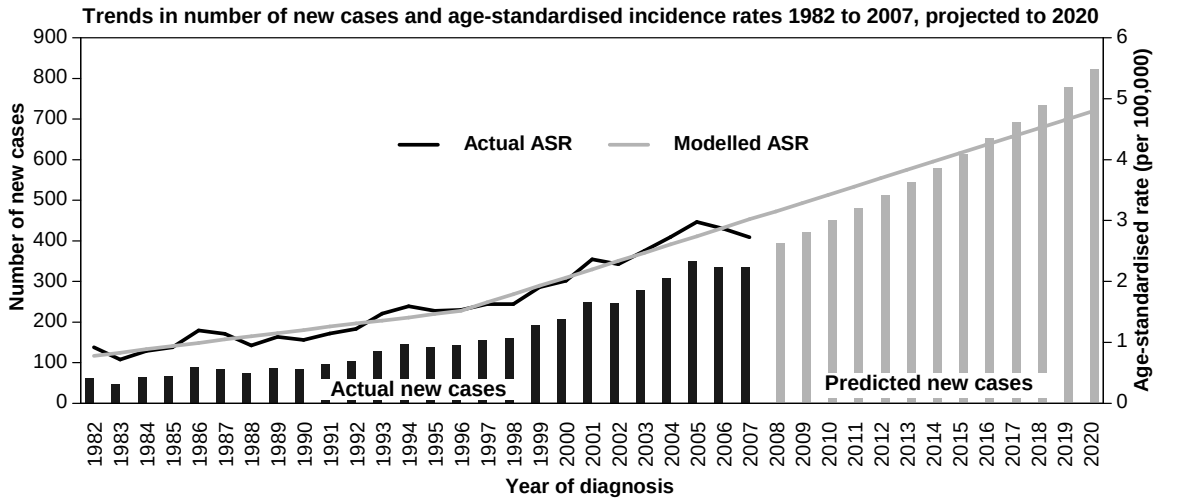
Notes

1. Liver cancer includes ICD-10 code C22.
2. Projected estimates are based on incidence data for 1996 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.9b: Liver cancer (C22), females, Australia: actual 1982–2007 and projected to 2020

Lung cancer (C33–C34)

Lung cancer is the fifth most commonly diagnosed invasive cancer in Australia and it causes more deaths than any other cancer in both males and females. Its high mortality rate results from both a high incidence rate and a very low survival rate. The poor survival outcome is due, at least partly, to the relatively high proportion of cases diagnosed at an advanced stage (AIHW & Cancer Australia 2011).

Tobacco smoking is the largest single risk factor for lung cancer in Australia, and is responsible for about 90% of lung cancers in males and 65% in females (AIHW: Ridolfo & Stevenson 2001). In Australia, the smoking rate among males has declined since the second half of the last century from an estimated 58% in 1964 to 18% in 2007. For females, the overall smoking rate increased slightly in the 1960s and 1970s, peaking in the mid-1970s at about 33%, but then declined to 15% in 2007 (OECD 2010). Overall, an estimated 20% of Australians aged 14 and over were current smokers in 2007(AIHW 2008).

In 2011, the Australian Government introduced a suite of measures to further reduce the number of Australians who smoke. In particular, the National Tobacco Campaign 2011 aims to reduce the proportion of adults who smoke on a daily basis from an estimated 16.6% in 2011 to 10% or less by 2018, while the *Break the Chain* component of this campaign aims to halve the proportion of Indigenous Australians who smoke, estimated to be 47% in 2011 (DoHA 2011a). In addition to these campaigns, legislation to require plain packaging of tobacco products to further reduce channels for tobacco advertising was introduced in 2011. These measures are expected to come into effect by December 2012.

The projections for lung cancer in this section are based on extrapolation of the trends in incidence up to 2007 and do not attempt to model the future impact of these interventions; however, their effect should be considered when interpreting these data.

Males

Joinpoint analysis of the age-standardised incidence rates of lung cancer for males shows a statistically significant decreasing trend of about 1.2 cases per 100,000 males per year from 1982 to 2007.

Following this trend, it is expected that lung cancer in males will continue to decrease to about 49 cases diagnosed per 100,000 males in 2020, equating to approximately 7,500 cases (Table 3.10a). The largest decrease in rates is expected in males aged 65–84. The rate for males aged 85 and over is expected to remain constant (Figure 3.10a).

Females

Unlike the decreasing trends for males, trends in the age-standardised rate of lung cancer for females have been increasing since 1982. Joinpoint analysis showed age-standardised rates increased at about 0.7 cases per 100,000 females from 1982 to 1990, but have slowed from 1990 to 2007, increasing at about only 0.4 cases per 100,000.

Following this most recent trend, it is expected that lung cancer in females will continue to rise slowly to about 36 new cases diagnosed per 100,000 females in 2020, equating to approximately 6,100 cases (Table 3.10c). The largest increase in rates is expected in females aged 85 and over, increasing from 156 cases per 100,000 females in 2007 to an estimated 214 cases per 100,000 in 2020. Large increases are also expected in females aged 65–84 (Table 3.10d, Figure 3.10b).

Table 3.10a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: lung cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	6,380	6,120	6,640	55.4	53.1	57.6
2012	6,500	6,230	6,770	54.6	52.4	56.9
2013	6,610	6,330	6,890	53.9	51.7	56.2
2014	6,720	6,440	7,010	53.2	51.0	55.5
2015	6,840	6,550	7,130	52.5	50.3	54.8
2016	6,960	6,660	7,270	51.9	49.6	54.1
2017	7,110	6,800	7,420	51.2	49.0	53.4
2018	7,250	6,930	7,570	50.6	48.4	52.8
2019	7,380	7,050	7,710	49.9	47.7	52.2
2020	7,520	7,180	7,860	49.3	47.1	51.5

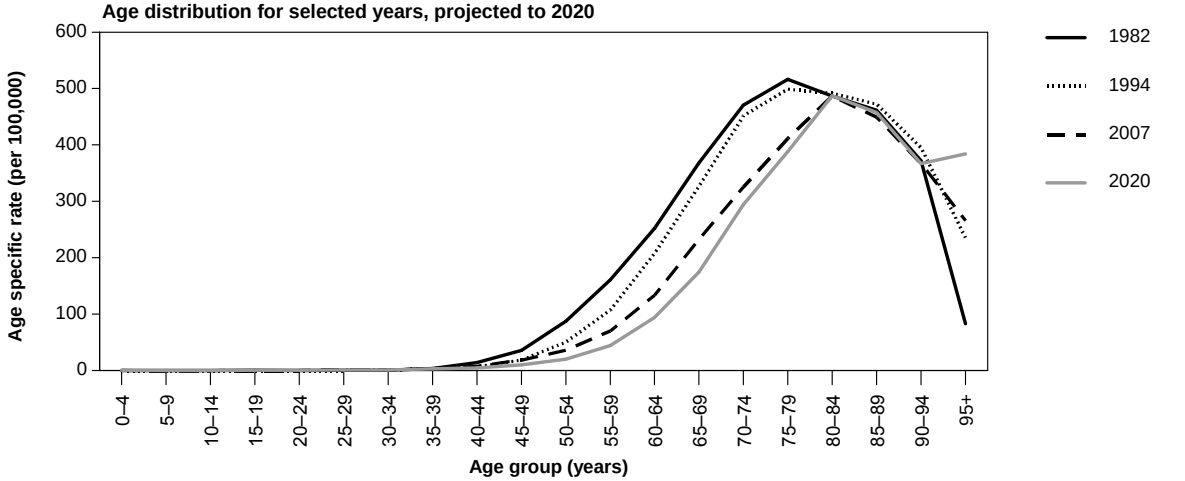
Table 3.10b: Projected number of new cases and age-specific rates, males, 2011–2020: lung cancer

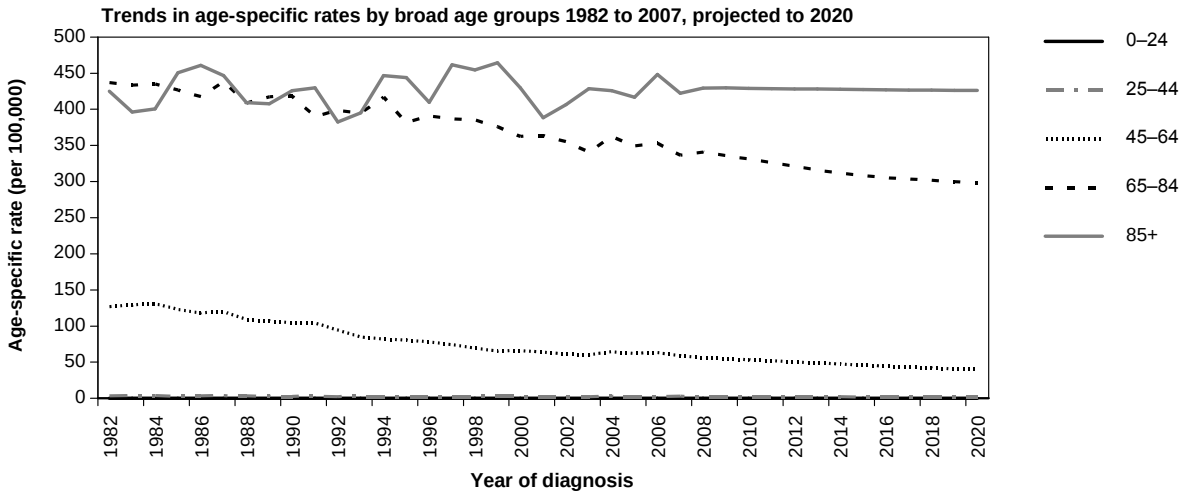
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	5	0.1	75	2.5	1,470	52.5	4,200	327.0	630	428.7
2012	5	0.1	75	2.4	1,430	50.8	4,320	321.3	665	428.2
2013	5	0.1	75	2.4	1,400	49.3	4,430	316.2	700	428.1
2014	5	0.1	75	2.3	1,380	47.9	4,530	312.3	730	427.9
2015	5	0.1	75	2.3	1,350	46.4	4,640	308.6	765	427.6
2016	5	0.1	75	2.2	1,330	45.0	4,760	305.5	795	427.2
2017	5	0.1	75	2.2	1,310	43.7	4,900	304.0	820	426.8
2018	5	0.1	75	2.2	1,280	42.4	5,040	301.9	840	426.4
2019	5	0.1	75	2.1	1,260	41.3	5,180	300.1	865	426.3
2020	5	0.1	75	2.1	1,240	40.1	5,320	298.5	885	426.2

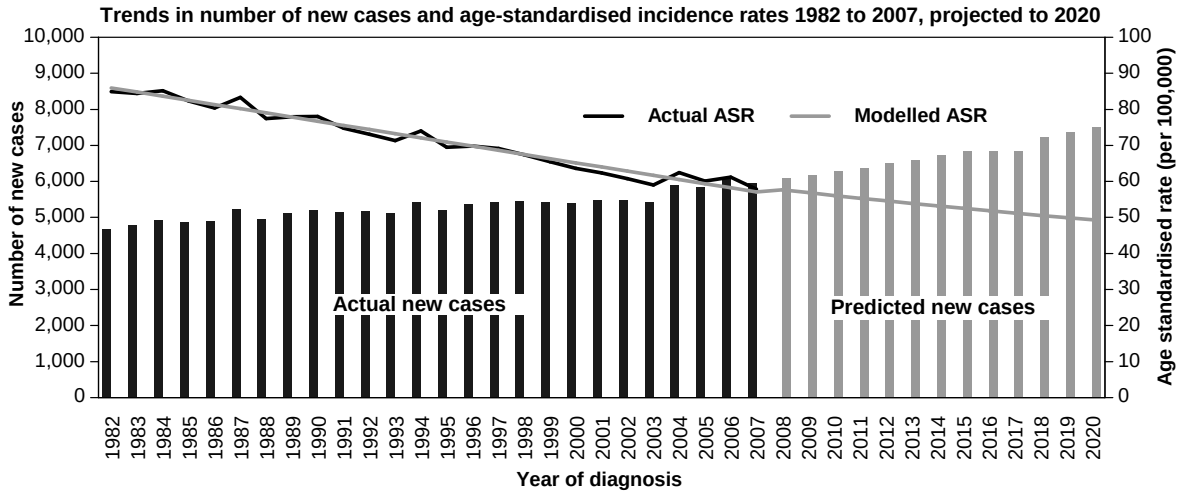
Notes

1. Lung cancer includes ICD-10 codes C33–C34.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.10a: Lung cancer (C33–C34), males, Australia: actual 1982–2007 and projected to 2020

Table 3.10c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: lung cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	4,290	4,110	4,470	32.4	31.1	33.8
2012	4,460	4,270	4,650	32.9	31.5	34.3
2013	4,640	4,450	4,840	33.3	31.9	34.7
2014	4,830	4,620	5,030	33.7	32.3	35.1
2015	5,020	4,810	5,240	34.1	32.7	35.6
2016	5,220	5,000	5,450	34.5	33.0	36.0
2017	5,440	5,200	5,670	34.9	33.4	36.5
2018	5,660	5,410	5,900	35.4	33.8	36.9
2019	5,890	5,630	6,140	35.8	34.2	37.3
2020	6,120	5,850	6,390	36.2	34.6	37.8

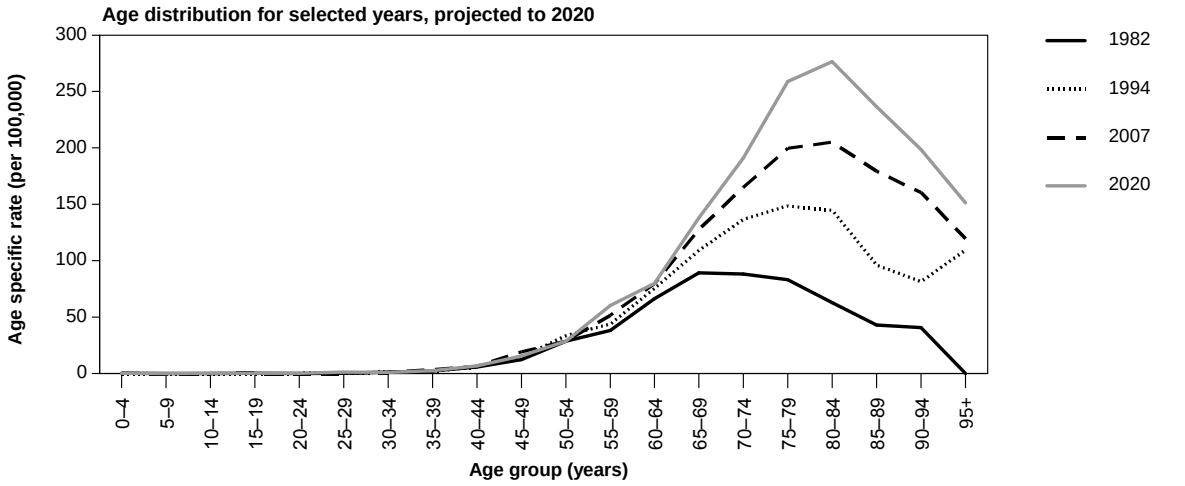
Table 3.10d: Projected number of new cases and age-specific rates, females, 2011–2020: lung cancer

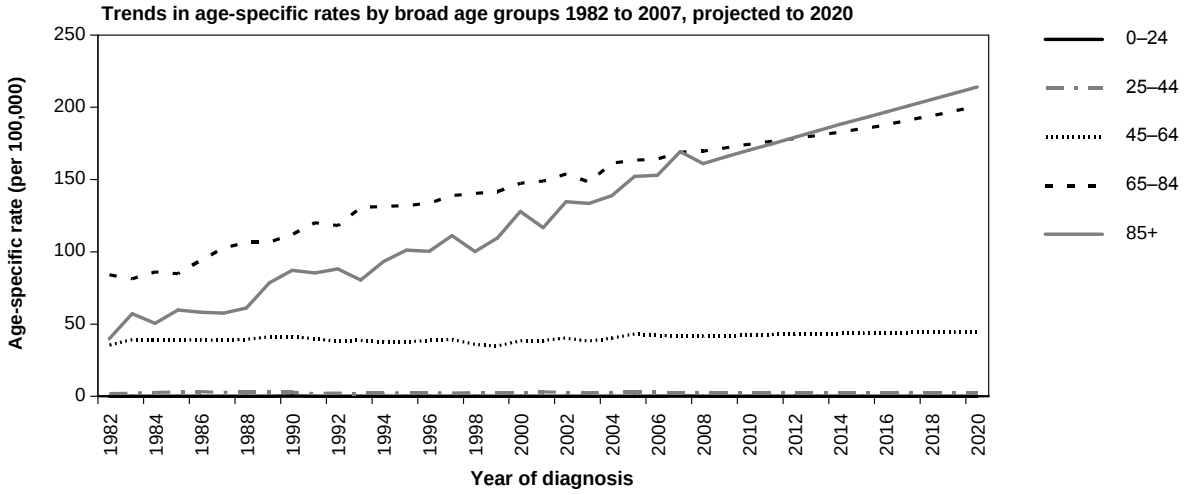
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	5	0.1	85	2.8	1,220	42.8	2,500	176.7	480	174.7
2012	5	0.1	90	2.8	1,240	43.0	2,630	178.5	510	179.1
2013	5	0.1	90	2.8	1,260	43.3	2,750	180.5	540	183.7
2014	5	0.1	90	2.8	1,280	43.5	2,880	182.9	570	188.2
2015	5	0.1	90	2.8	1,300	43.8	3,020	185.3	600	192.5
2016	5	0.1	90	2.8	1,330	43.9	3,170	187.9	625	196.8
2017	5	0.1	90	2.8	1,350	44.1	3,340	191.2	650	201.1
2018	5	0.1	95	2.7	1,380	44.3	3,510	194.2	675	205.4
2019	5	0.1	95	2.7	1,400	44.6	3,690	197.5	700	209.8
2020	5	0.1	95	2.8	1,420	44.9	3,880	200.8	725	214.2

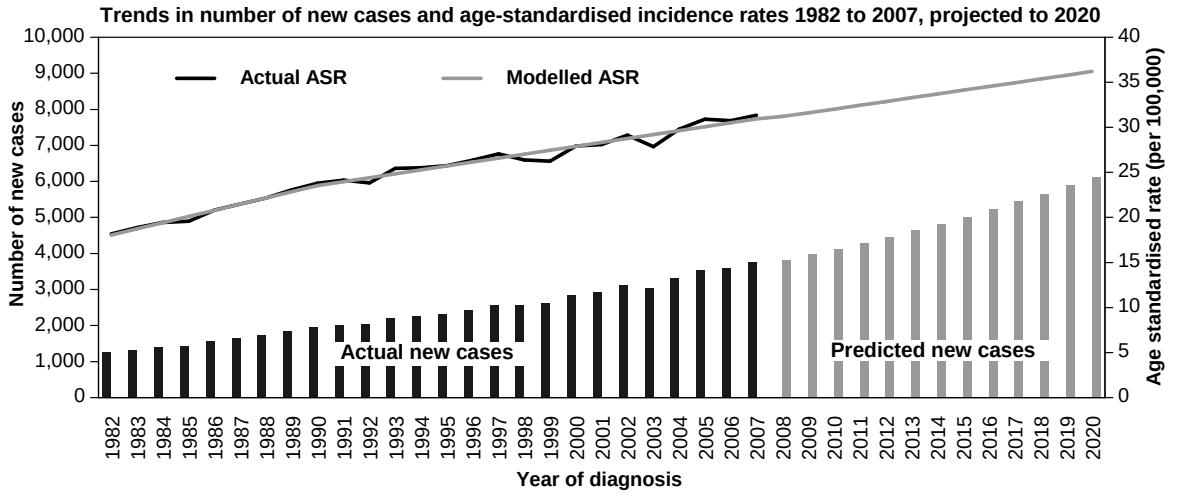
Notes

1. Lung cancer includes ICD-10 codes C33–C34.
2. Projected estimates are based on incidence data for 1990 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.10b: Lung cancer (C33–C34), females, Australia: actual 1982–2007 and projected to 2020

Melanoma of the skin (C43)

Melanoma of the skin is the third most commonly diagnosed cancer in males (after prostate and bowel cancer) and females (after breast and bowel cancer). Incidence has increased by 151% in males and 46% in females between 1982 and 2007, though it is unknown what proportion of this increase is due to an increase in the underlying disease, and how much is due to improved detection methods.

There are a number of risk factors for melanoma (Tucker & Goldstein 2003); however, exposure to ultra-violet (UV) radiation, from both sunlight (Armstrong & Kricker 2001) and solariums (Cust et al. 2010), is the major cause of melanoma in Australia.

Skin cancer prevention programs to reduce UV exposure from sunlight have operated in all Australian states and territories since the early 1980s, and joinpoint analysis of the age-standardised rates for both males and females show a significant slowing in the upward trend of the late 1980s. To continue to educate the Australian public about the seriousness of skin cancer and increase the awareness and adoption of sun protection behaviours, the Australian Government Department of Health and Ageing implemented the National Skin Cancer Awareness Campaign. This campaign ran from 2007 to 2010, with a particular focus in 2009–2010 targeting teenagers aged 14–17 (DoHA 2011b).

Artificial UV tanning devices (such as solaria and sunbeds) also emit UV radiation known to cause cancer and the World Health Organization does not recommend the use of such devices for cosmetic reasons (WHO 2010). In Australia, the solarium industry is regulated on a state by state basis. National standards for the solarium industry (AS/NZS 2635) were revised in 2009, and while the standard is voluntary, most states and territories in Australia now have legislation and regulations to govern the operation of solariums, using the revised standard as guidance (Makin & Dobbinson 2009).

The projections for melanoma of the skin in this section are based on extrapolation of the trends in incidence up to and including 2007 and do not attempt to model the future impact of the National Skin Cancer Awareness Campaign, or changes in solarium use, but their effects should be considered when interpreting these data.

Males

Melanoma has continued to increase in males since 1982; however, joinpoint analysis of age-standardised rates showed a significant change in the trend with the increase in rates slowing from 3 cases per 100,000 males each year from 1982 to 1988 to about 0.9 from 1988 onwards.

Following this most recent trend, it is expected that melanoma in males will continue to increase to about 74 cases diagnosed per 100,000 males in 2020, equating to approximately 10,780 cases (Table 3.11a). The largest increase in rates is expected to occur in males aged 65 and over (Table 3.11b, Figure 3.11a).

Females

The trend in melanoma of the skin in females showed a similar change to that in males. Joinpoint analysis of age-standardised incidence rates showed that between 1982 and 1987 the incidence of melanoma of the skin increased at about 1.6 cases per 100,000 females per year. After this, the increase slowed to 0.3 cases per 100,000 females per year.

Following this trend, it is expected that melanoma in females will continue to increase slowly to about 45 new cases diagnosed per 100,000 females in 2020, equating to approximately

6,790 cases (Table 3.11c). The largest increase in rates is expected in females aged 65 and over (Table 3.11d, Figure 3.11b).

Table 3.11a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: melanoma of the skin

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	7,570	7,280	7,850	65.5	63.1	67.9
2012	7,880	7,590	8,170	66.5	64.0	68.9
2013	8,200	7,900	8,510	67.4	64.9	69.9
2014	8,540	8,220	8,850	68.4	65.8	70.9
2015	8,880	8,550	9,210	69.3	66.7	71.9
2016	9,230	8,890	9,580	70.3	67.6	72.9
2017	9,610	9,250	9,970	71.2	68.6	73.9
2018	9,990	9,620	10,360	72.2	69.5	74.9
2019	10,380	9,990	10,770	73.1	70.4	75.9
2020	10,780	10,370	11,190	74.1	71.3	76.9

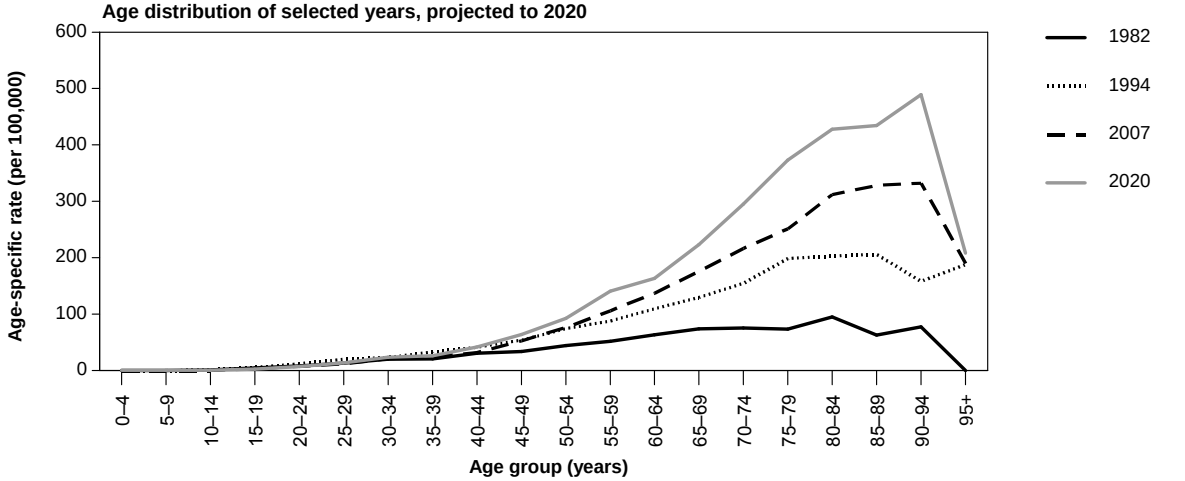
Table 3.11b: Projected number of new cases and age-specific rates, males, 2011–2020: melanoma of the skin

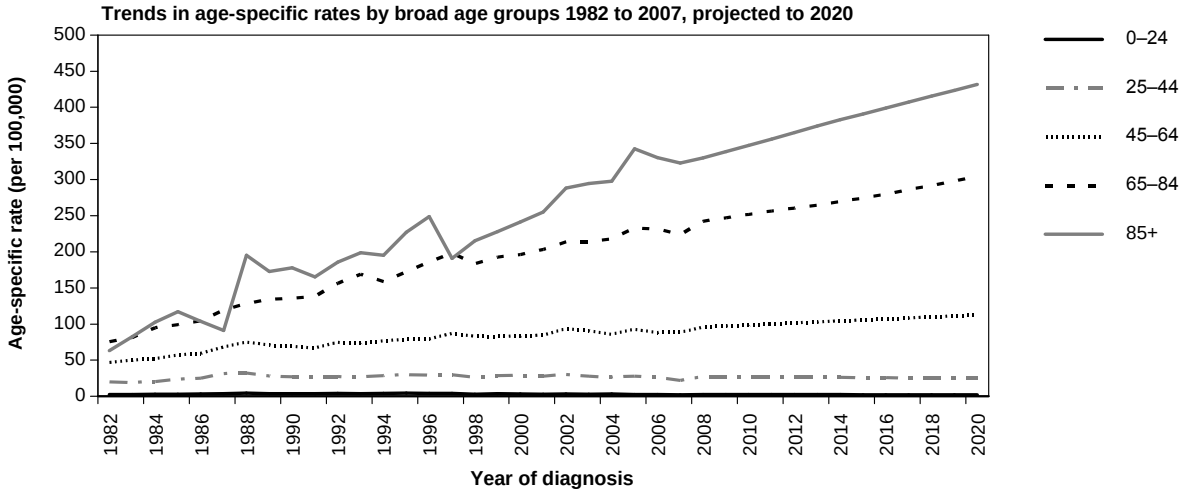
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	95	2.5	845	26.8	2,810	100.4	3,300	256.6	520	356.2
2012	90	2.4	855	26.7	2,870	101.7	3,510	260.5	565	365.0
2013	90	2.4	860	26.6	2,930	103.2	3,710	264.8	610	374.0
2014	90	2.3	870	26.5	3,010	104.6	3,910	269.7	655	382.8
2015	85	2.3	875	26.3	3,080	105.9	4,130	274.6	700	390.8
2016	85	2.2	875	26.1	3,170	107.2	4,360	279.9	745	399.0
2017	85	2.1	880	25.9	3,250	108.5	4,620	286.1	780	407.1
2018	80	2.1	885	25.8	3,330	109.9	4,880	292.0	820	415.4
2019	80	2.0	895	25.7	3,400	111.4	5,140	298.1	860	423.6
2020	80	2.0	905	25.6	3,480	112.8	5,420	304.3	900	431.7

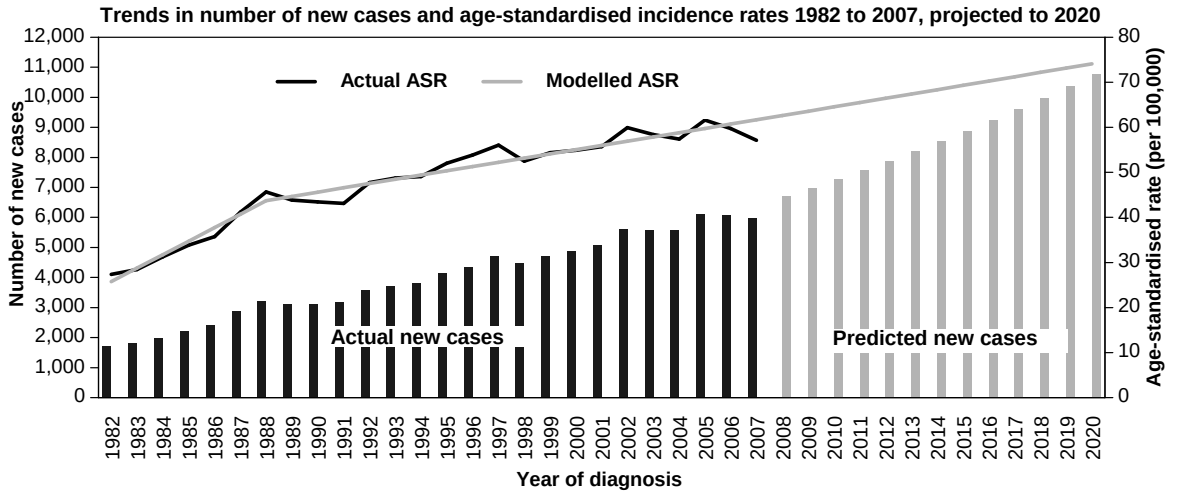
Notes

1. Melanoma of the skin includes ICD-10 code C43.
2. Projected estimates are based on incidence data for 1988 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.11a: Melanoma of the skin (C43), males, Australia: actual 1982–2007 and projected to 2020

Table 3.11c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: melanoma of the skin

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	5,200	4,990	5,420	41.9	40.1	43.7
2012	5,360	5,140	5,590	42.2	40.4	44.0
2013	5,530	5,300	5,760	42.6	40.8	44.4
2014	5,700	5,450	5,940	42.9	41.1	44.8
2015	5,870	5,620	6,120	43.3	41.4	45.1
2016	6,040	5,790	6,300	43.6	41.7	45.5
2017	6,220	5,960	6,490	44.0	42.1	45.9
2018	6,410	6,130	6,680	44.3	42.4	46.2
2019	6,600	6,310	6,880	44.6	42.7	46.6
2020	6,790	6,500	7,080	45.0	43.0	47.0

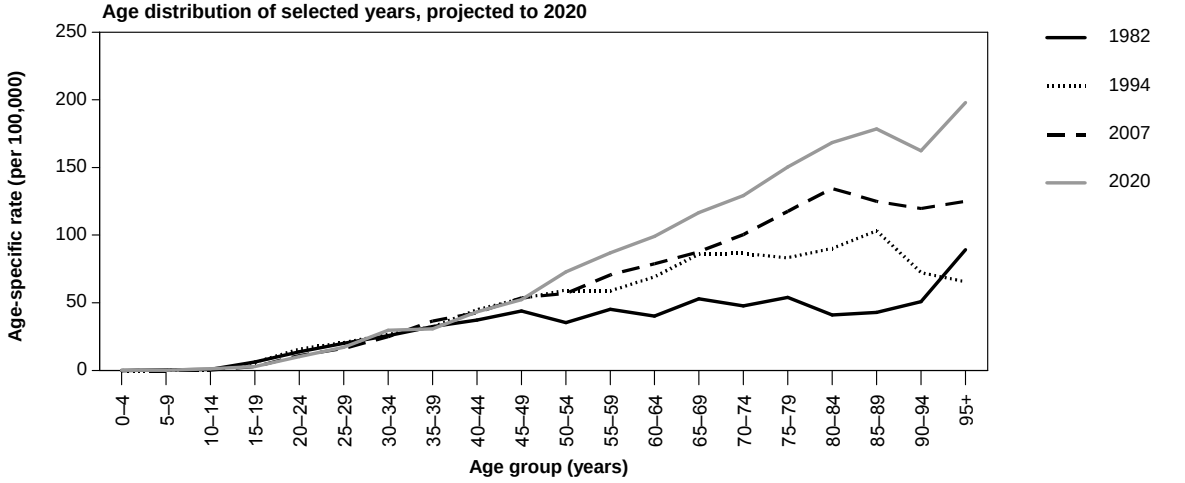
Table 3.11d: Projected number of new cases and age-specific rates, females, 2011–2020: melanoma of the skin

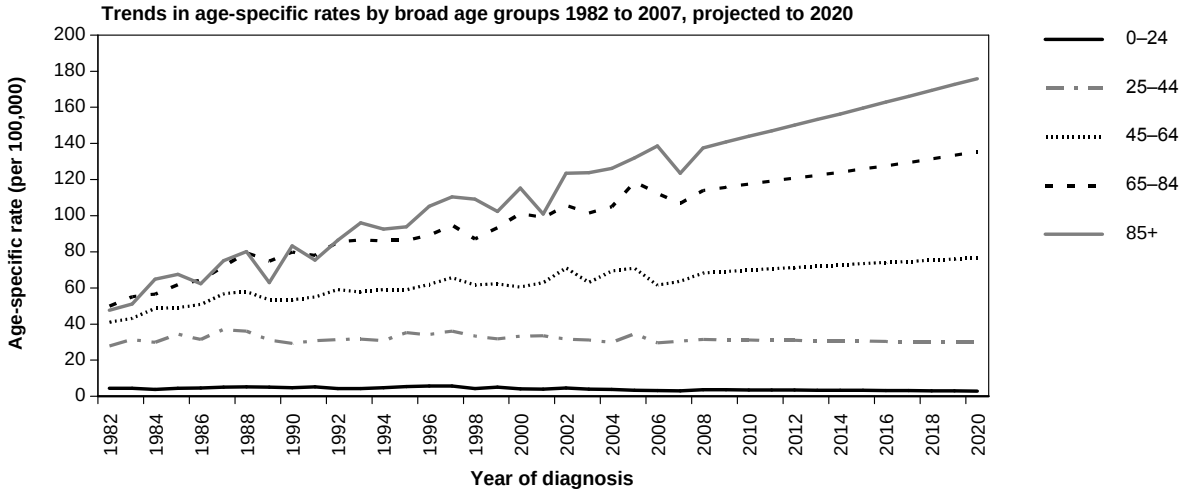
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	125	3.5	975	31.1	2,010	70.6	1,690	119.2	405	147.1
2012	125	3.5	985	31.1	2,050	71.3	1,780	120.8	425	150.1
2013	125	3.4	995	31.0	2,090	72.1	1,870	122.4	450	153.2
2014	120	3.4	1,000	30.9	2,140	72.8	1,960	124.1	470	156.4
2015	120	3.3	1,010	30.7	2,190	73.5	2,050	125.8	495	159.6
2016	120	3.2	1,010	30.5	2,240	74.1	2,150	127.6	520	162.8
2017	115	3.1	1,020	30.4	2,290	74.7	2,260	129.6	535	166.1
2018	115	3.1	1,020	30.2	2,340	75.4	2,380	131.5	555	169.3
2019	115	3.0	1,030	30.1	2,380	76.1	2,500	133.5	575	172.6
2020	110	2.9	1,040	30.0	2,430	76.8	2,620	135.5	595	175.9

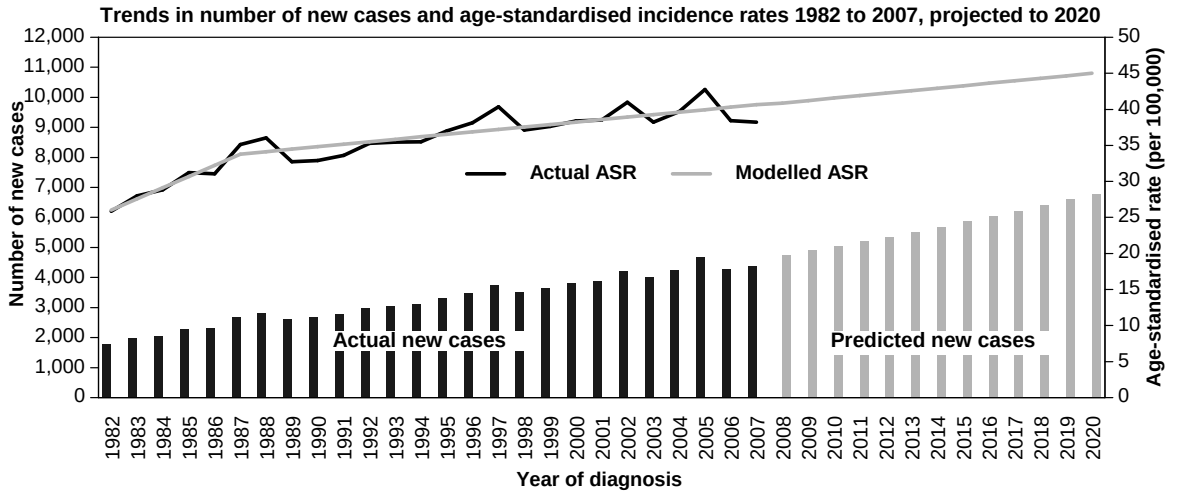
Notes

1. Melanoma of the skin includes ICD-10 code C43.
2. Projected estimates are based on incidence data for 1987 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.11b: Melanoma of the skin (C43), females, Australia: actual 1982–2007 and projected to 2020

Mesothelioma (C45)

Mesothelioma is a rare type of cancer that affects the membrane that covers and protects the internal organs of the body, including the lungs, heart and abdominal organs. It is much more common in males than females, with the age-standardised incidence rate in males six times higher than females (5.4 per 100,000 males and 0.9 per 100,000 females). While incidence of this cancer is low (there were 660 new cases diagnosed in Australia in 2007), Australia has one of the highest reported annual crude incidence rates in the world (Bianchi & Bianchi 2007).

Pleural mesothelioma (a cancer that affects the lining of the lungs) is the most commonly diagnosed type of mesothelioma. Pleural mesothelioma is sometimes incorrectly referred to as lung cancer; however, pleural mesothelioma is not regarded as a cancer of the lung in the current version of the international coding standards for cancer. Peritoneal mesothelioma (affecting the abdominal lining), pericardial mesothelioma (lining of the heart) and mesothelioma of the linings of reproductive organs are much less common diagnoses. The projections presented here are for all forms of mesothelioma combined.

Mesothelioma is almost always caused by exposure to asbestos—a mineral used in some building materials. Mesothelioma usually occurs 20–40 years after exposure to asbestos, although not all who are exposed get the disease. Asbestos was mined in Australia in the early part of the 20th century, ceasing in 1966 in response to the identification of the asbestos-mesothelioma link. Since then, the use of asbestos products has been regulated (Safe Work Australia 2010). Occupational exposure in male-dominated industries is the reason that mesothelioma is more common in males than females. The Australian Mesothelioma Registry collects all notifications of new cases of mesothelioma from state and territory cancer registries as well as detailed information on the past exposure to asbestos of mesothelioma patients. This information will be made available to the government, policy advisors, researchers and the public to inform policy and management of this disease (Australian Mesothelioma Registry 2011).

Because asbestos exposure in the workplace and the general environment is now minimised, incidence of new cases of mesothelioma in the population is expected to decline over time. However, due to the long latency between asbestos exposure and disease onset, it is expected that peak incidence in those previously exposed to asbestos will occur about 2014 (Clements 2007a, Clements 2007b). It is important to note that the projections for mesothelioma in this section are based on simple extrapolation of the trends in incidence from 1982 to 2007 and do not attempt to model changes in exposure over time; more detailed projections to 2060 that incorporate potential asbestos exposure are available in the work by Clements et al.

Males

Mesothelioma has continued to increase in males since 1982; however, joinpoint analysis of age-standardised rates showed a significant change in the trend with the increase in rates slowing from 0.2 cases per 100,000 males per year from 1982–1994 to about 0.06 cases per 100,000 from 1994 onwards. Using this most recent trend to model projections, it is expected that age-standardised rates of mesothelioma in males will continue to increase to about 6.2 cases diagnosed per 100,000 males in 2020, equating to approximately 945 cases (Table 3.12a), with the largest increase in rates expected in males aged 85 and over (Table 3.12b, Figure 3.12a).

Females

Although rates of mesothelioma are much lower in females than males, age-standardised rates have been increasing steadily since 1982. Extrapolation of trends from 1982 onwards suggests that age-standardised rates of mesothelioma in females will continue to increase slowly to about 1.4 new cases diagnosed per 100,000 females in 2020, equating to about 240 new cases diagnosed in that year (Table 3.12c). This increase is expected to predominantly occur in females aged 65 and over (Table 3.12d, Figure 3.12b).

Table 3.12a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: mesothelioma

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	650	585	715	5.6	5.1	6.2
2012	680	610	745	5.7	5.1	6.3
2013	705	635	780	5.8	5.2	6.4
2014	735	660	810	5.8	5.2	6.4
2015	765	690	845	5.9	5.3	6.5
2016	800	715	880	6.0	5.3	6.6
2017	835	745	920	6.0	5.4	6.7
2018	870	780	960	6.1	5.4	6.7
2019	905	810	1,000	6.2	5.5	6.8
2020	945	845	1,050	6.2	5.5	6.9

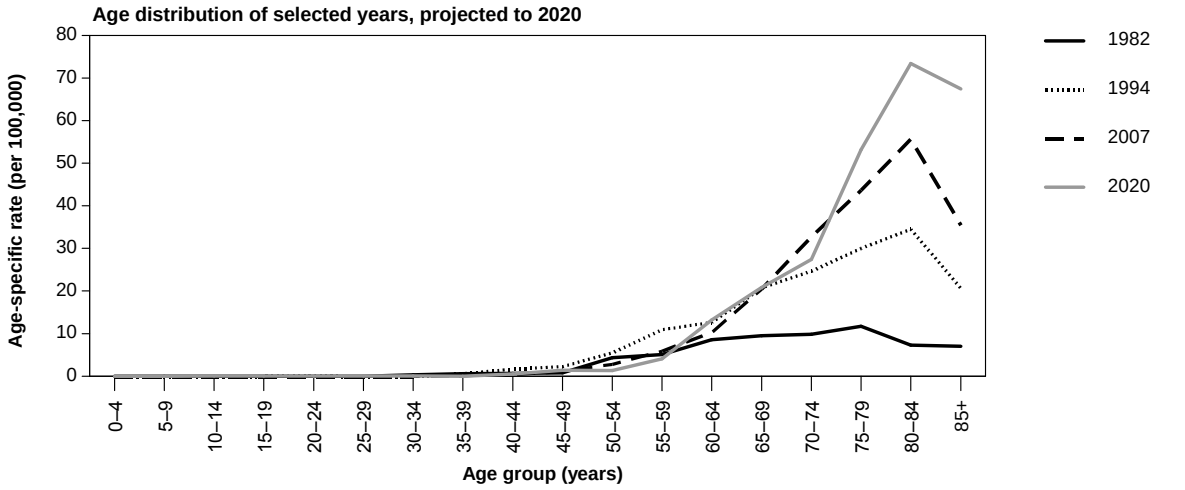
Table 3.12b: Projected number of new cases and age-specific rates, males, 2011–2020: mesothelioma

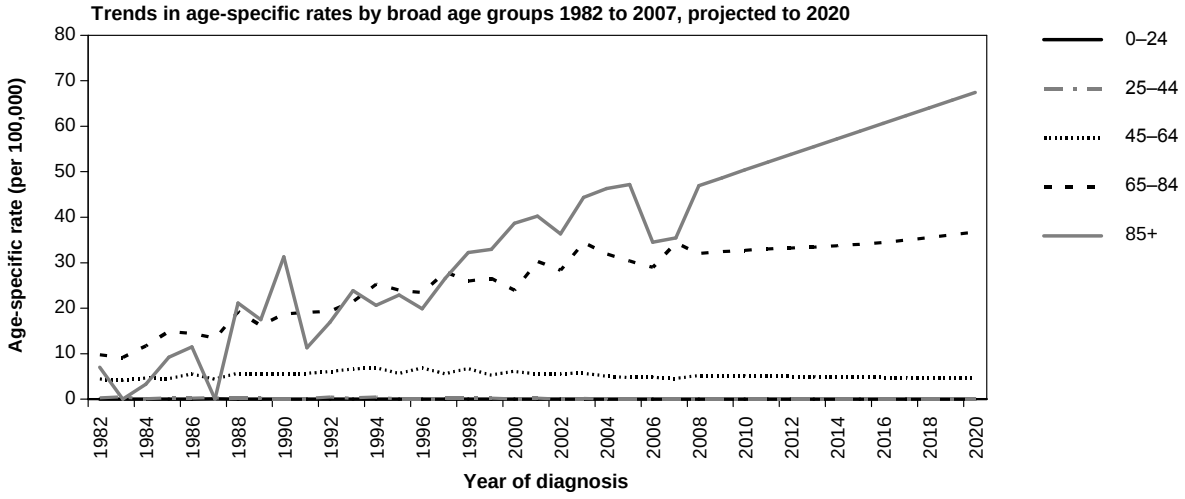
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	5	0.1	145	5.1	425	33.1	75	52.1
2012	0	0.0	5	0.1	140	5.1	445	33.2	85	53.8
2013	0	0.0	5	0.1	140	5.0	470	33.4	90	55.5
2014	0	0.0	5	0.1	140	4.9	490	33.8	100	57.2
2015	0	0.0	5	0.1	140	4.9	515	34.1	105	58.9
2016	0	0.0	5	0.1	145	4.8	535	34.5	115	60.6
2017	0	0.0	5	0.1	145	4.8	565	35.0	120	62.3
2018	0	0.0	5	0.1	145	4.8	595	35.5	125	64.0
2019	0	0.0	5	0.1	145	4.7	625	36.1	135	65.7
2020	0	0.0	5	0.1	145	4.7	655	36.8	140	67.4

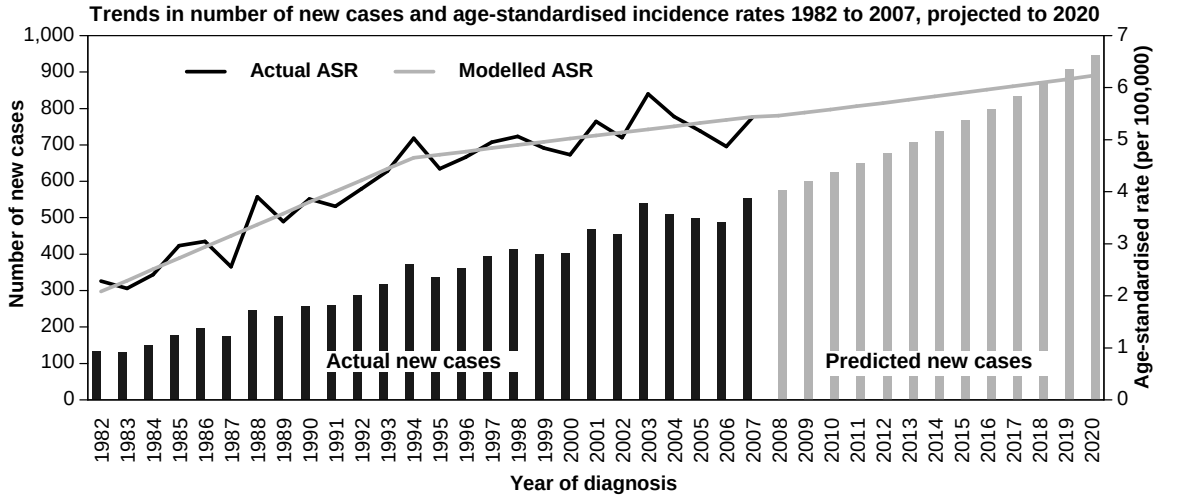
Notes

1. Mesothelioma includes ICD-10 codes C45.
2. Projected estimates are based on incidence data for 1994 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.12a: Mesothelioma (C45), males, Australia: actual 1982–2007 and projected to 2020

Table 3.12c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: mesothelioma

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	150	135	170	1.1	1.0	1.3
2012	160	140	175	1.2	1.0	1.3
2013	165	150	185	1.2	1.1	1.4
2014	175	155	195	1.2	1.1	1.4
2015	185	165	205	1.3	1.1	1.4
2016	195	175	215	1.3	1.2	1.4
2017	205	185	225	1.3	1.2	1.5
2018	215	195	240	1.4	1.2	1.5
2019	225	205	250	1.4	1.3	1.5
2020	240	215	260	1.4	1.3	1.6

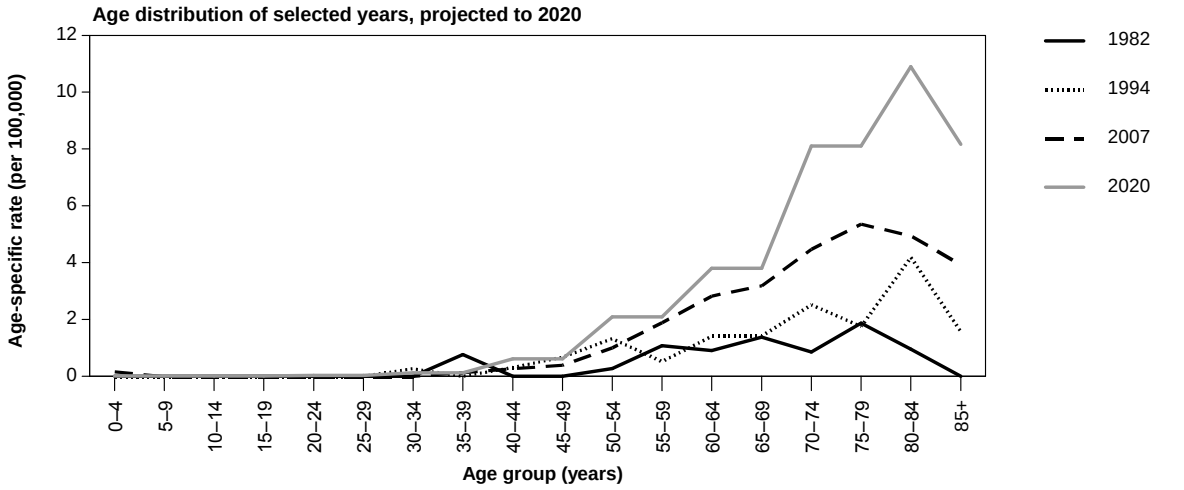
Table 3.12d: Projected number of new cases and age-specific rates, females, 2011–2020: mesothelioma

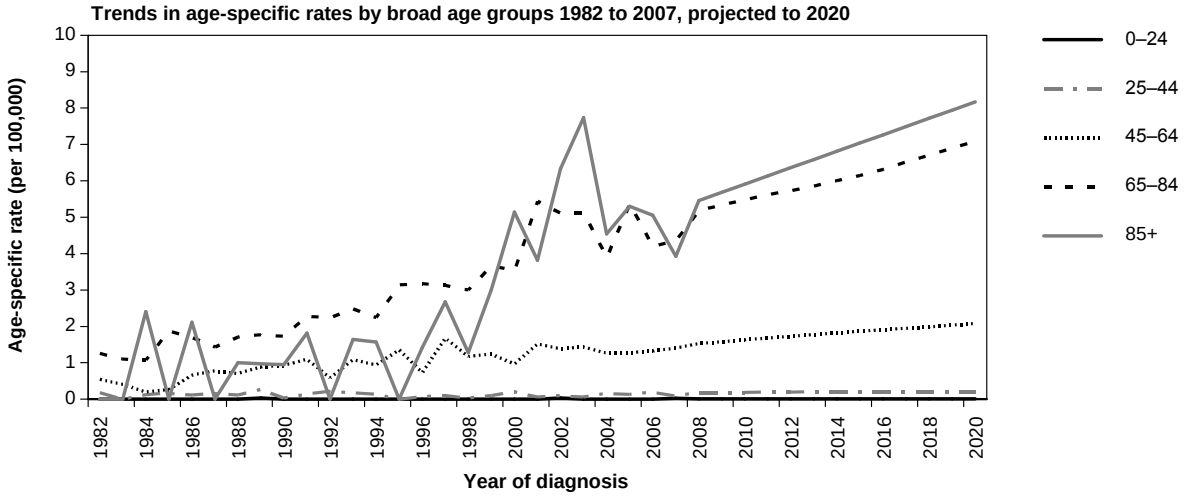
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	5	0.2	50	1.7	80	5.6	15	6.1
2012	0	0.0	5	0.2	50	1.7	85	5.7	20	6.4
2013	0	0.0	5	0.2	50	1.8	90	5.9	20	6.6
2014	0	0.0	5	0.2	55	1.8	95	6.0	20	6.8
2015	0	0.0	5	0.2	55	1.9	100	6.2	20	7.0
2016	0	0.0	5	0.2	60	1.9	105	6.3	25	7.3
2017	0	0.0	5	0.2	60	1.9	115	6.5	25	7.5
2018	0	0.0	5	0.2	60	2.0	120	6.7	25	7.7
2019	0	0.0	5	0.2	65	2.0	130	6.9	25	7.9
2020	0	0.0	5	0.2	65	2.1	135	7.1	30	8.2

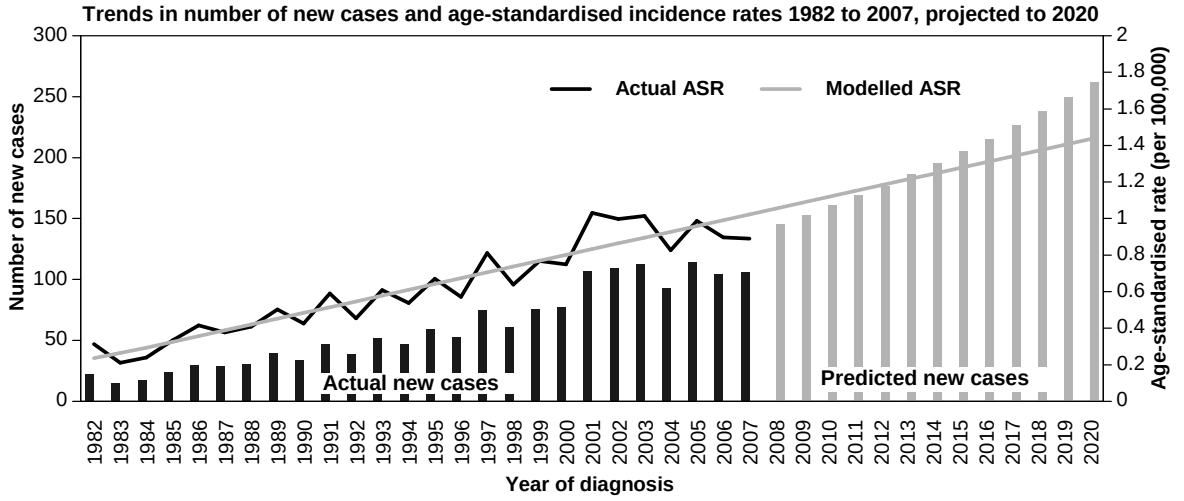
Notes

1. Mesothelioma includes ICD-10 code C45.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.12b: Mesothelioma (C45), females, Australia: actual 1982–2007 and projected to 2020

Non-Hodgkin lymphoma (C82–C85)

In 2007, non-Hodgkin lymphoma accounted for 4,025 (3.7%) of new cases and 1,319 (3.3%) cases of cancer death in Australia. It is more common in males than females, with 1 in 36 males and 1 in 51 females developing the disease by the age of 85. The chance of developing non-Hodgkin lymphoma increases with age, with the average age of diagnosis being 65.

The causes of non-Hodgkin lymphoma are not clear. Immune deficiency is one of the strongest known risk factors, with the risk increasing with the degree of immune deficiency. Viruses linked to increased risk of non-Hodgkin lymphoma include hepatitis C, human immunodeficiency (HIV) and Epstein-Barr (Grulich et al. 2007a; De Sanjose et al. 2008; IARC 2008). Bacterial infection is also an associated risk factor of non-Hodgkin lymphoma, specifically the stomach ulcer-causing *Helicobacter pylori* (Evens & Chiu 2008). The reasons for higher incidence rates of non-Hodgkin lymphoma in males compared with females are not known, but may include occupational and industrial exposures (Lahti et al. 2008).

Diagnosis of non-Hodgkin lymphoma has improved over time, and these improvements may at least partly be responsible for the observed increasing trends in incidence (Devesa & Fears 1992). As well as improved diagnosis, the underlying incidence rate in the population is increasing. Research investigating reasons for the increase is extensive, but remains inconclusive (Müller et al. 2005; IARC 2008).

Males

Non-Hodgkin lymphoma rates in males have increased since 1982. From 1982 to 1992 the rate increased by 0.6 cases per 100,000 males. After this time, the increasing rate slowed to about 0.1 cases per 100,000 males per year. While the increase in incidence of HIV/AIDS among young and middle-aged men during the 1980s is considered to be partially responsible for the increase in incidence rates up to 1992 (Devesa & Fears 1992), factors responsible for this change in incidence rates are not fully understood.

Using data from the most recent trend to model projections, it is expected that age-standardised rates of non-Hodgkin lymphoma in males will continue to increase to about 23.5 new cases diagnosed per 100,000 males in 2020, equating to approximately 3,470 cases (Table 3.13a). The largest increase in rates is expected in males aged 65–84 (Table 3.13b, Figure 3.13a).

Females

Joinpoint analysis of age-standardised incidence rates of non-Hodgkin lymphoma in females showed a similar, although much lower, trend to males. Between 1982 and 1997, rates increased significantly at about 1.3 cases per 100,000 females. From 1997 to 2007 rates have remained stable, with a non-significant increase of 0.03 cases per 100,000 females. Extrapolation of age-specific trends from 1997 onwards suggests that age-standardised rates in females will stay steady at about 15.4 new cases diagnosed per 100,000 females, equating to approximately 2,480 new cases in 2020 (Table 3.13c). A small increase in rates is expected in females aged 65–84 (Table 3.13d, Figure 3.13b).

Table 3.13a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: non-Hodgkin lymphoma

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	2,600	2,480	2,730	22.4	21.3	23.5
2012	2,690	2,560	2,820	22.5	21.4	23.6
2013	2,780	2,640	2,920	22.7	21.5	23.8
2014	2,870	2,720	3,010	22.8	21.6	23.9
2015	2,960	2,810	3,110	22.9	21.7	24.1
2016	3,060	2,890	3,220	23.0	21.8	24.2
2017	3,160	2,990	3,330	23.1	21.9	24.4
2018	3,260	3,080	3,440	23.3	22.0	24.5
2019	3,360	3,180	3,550	23.4	22.1	24.6
2020	3,470	3,270	3,660	23.5	22.2	24.8

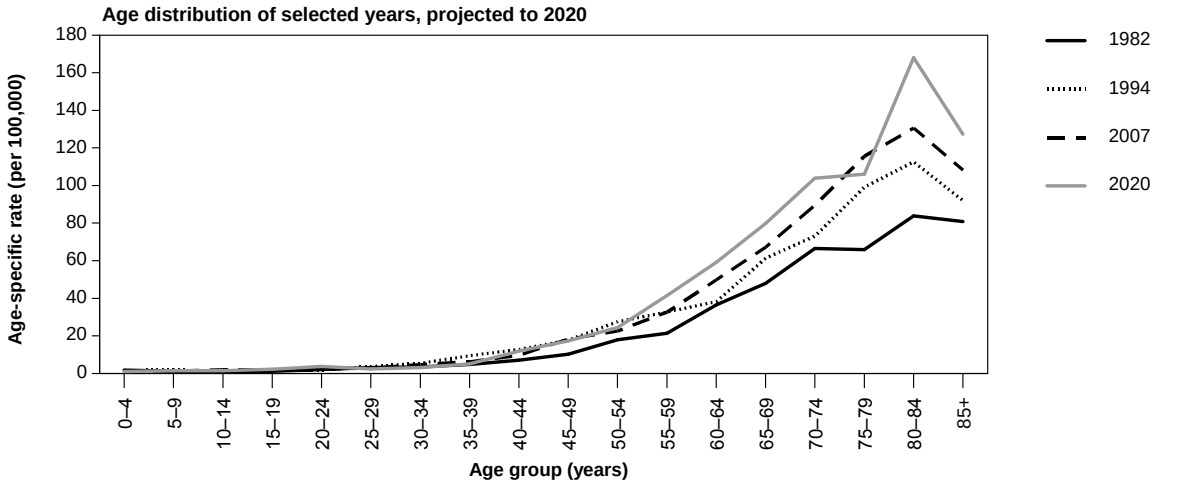
Table 3.13b: Projected number of new cases and age-specific rates, males, 2011–2020: non-Hodgkin lymphoma

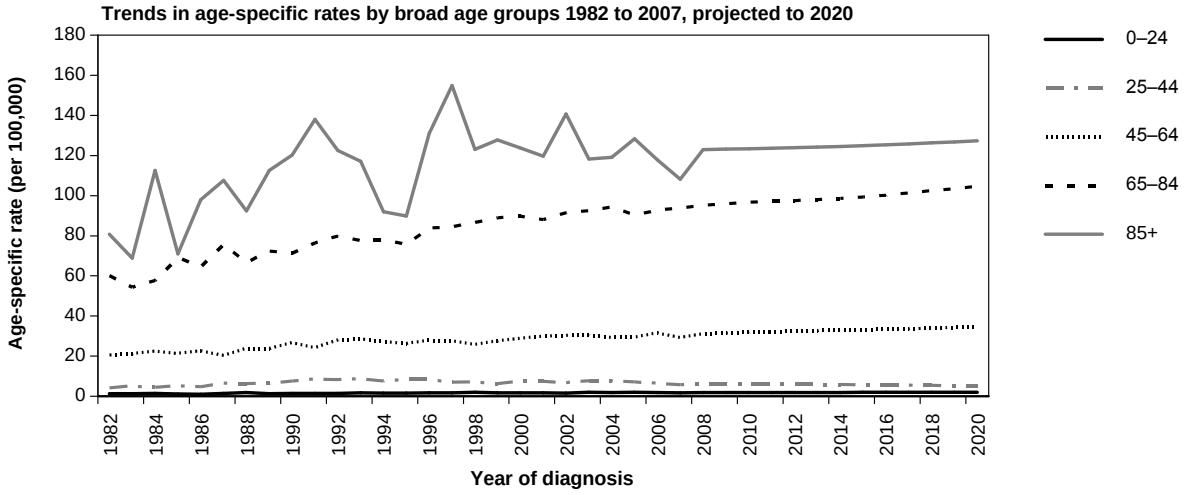
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	70	1.9	200	6.3	905	32.3	1,250	97.3	180	123.7
2012	75	1.9	200	6.2	915	32.5	1,310	97.6	190	124.0
2013	75	1.9	200	6.1	930	32.8	1,370	98.0	205	124.2
2014	75	2.0	195	6.0	950	33.0	1,430	98.7	215	124.5
2015	75	2.0	195	5.9	970	33.3	1,500	99.4	225	124.9
2016	75	2.0	195	5.8	990	33.5	1,560	100.2	235	125.3
2017	80	2.0	195	5.7	1,010	33.7	1,640	101.4	240	125.8
2018	80	2.0	190	5.6	1,030	34.0	1,710	102.4	250	126.3
2019	80	2.0	190	5.5	1,050	34.3	1,790	103.5	255	126.8
2020	80	2.0	190	5.4	1,070	34.6	1,870	104.6	265	127.3

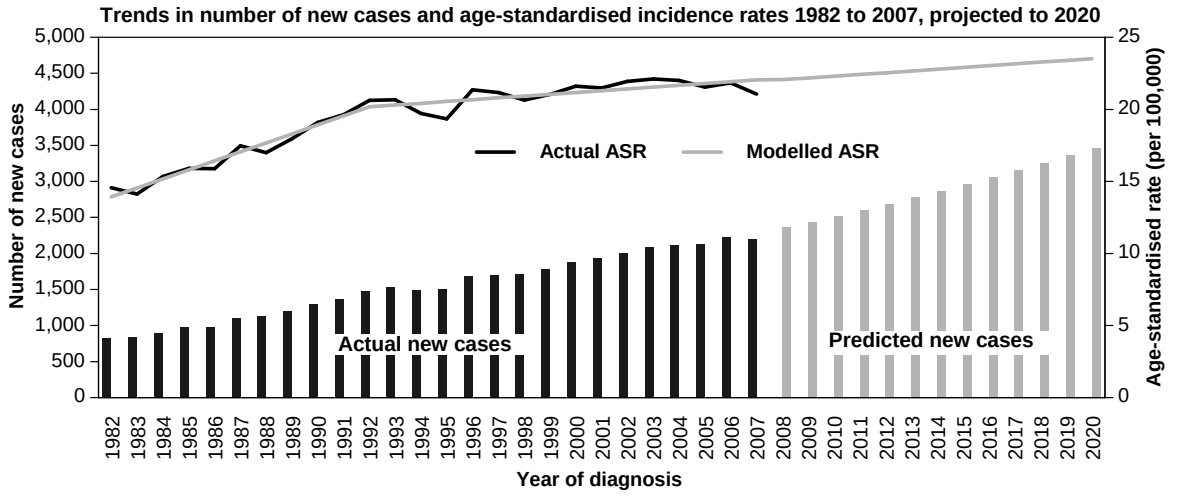
Notes

1. Non-Hodgkin lymphoma includes ICD-10 codes C82–C85.
2. Projected estimates are based on incidence data for 1992 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.13a: Non-Hodgkin lymphoma (C82–C85), males, Australia: actual 1982–2007 and projected to 2020

Table 3.13c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: non-Hodgkin lymphoma

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	2,010	1,920	2,100	15.4	14.7	16.1
2012	2,060	1,960	2,150	15.4	14.7	16.1
2013	2,110	2,010	2,200	15.4	14.7	16.1
2014	2,160	2,060	2,250	15.4	14.7	16.1
2015	2,210	2,110	2,310	15.4	14.7	16.1
2016	2,260	2,160	2,360	15.4	14.7	16.1
2017	2,320	2,210	2,420	15.4	14.7	16.1
2018	2,370	2,270	2,480	15.4	14.7	16.1
2019	2,430	2,320	2,540	15.4	14.7	16.1
2020	2,480	2,370	2,600	15.4	14.7	16.1

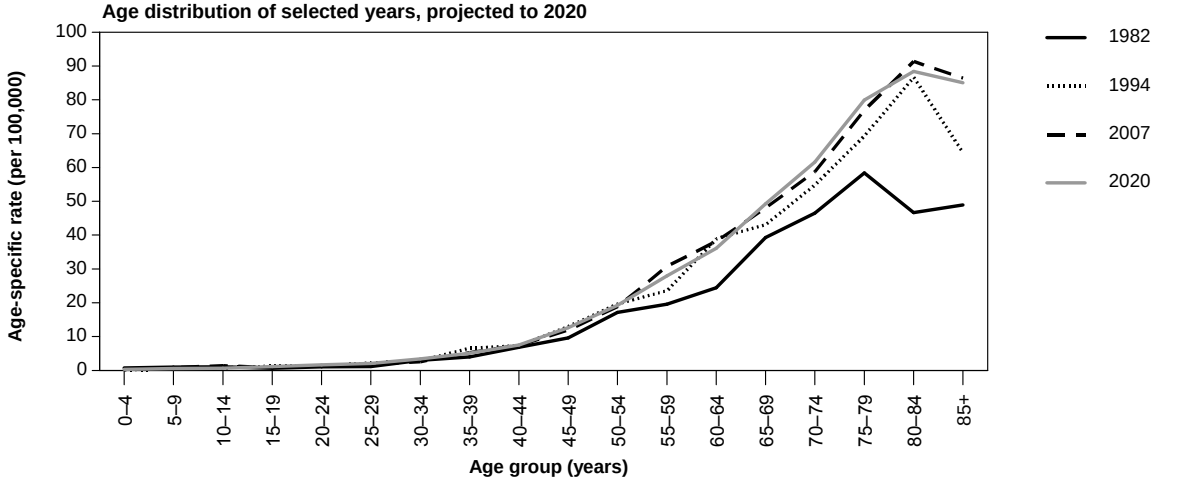
Table 3.13d: Projected number of new cases and age-specific rates, females, 2011–2020: non-Hodgkin lymphoma

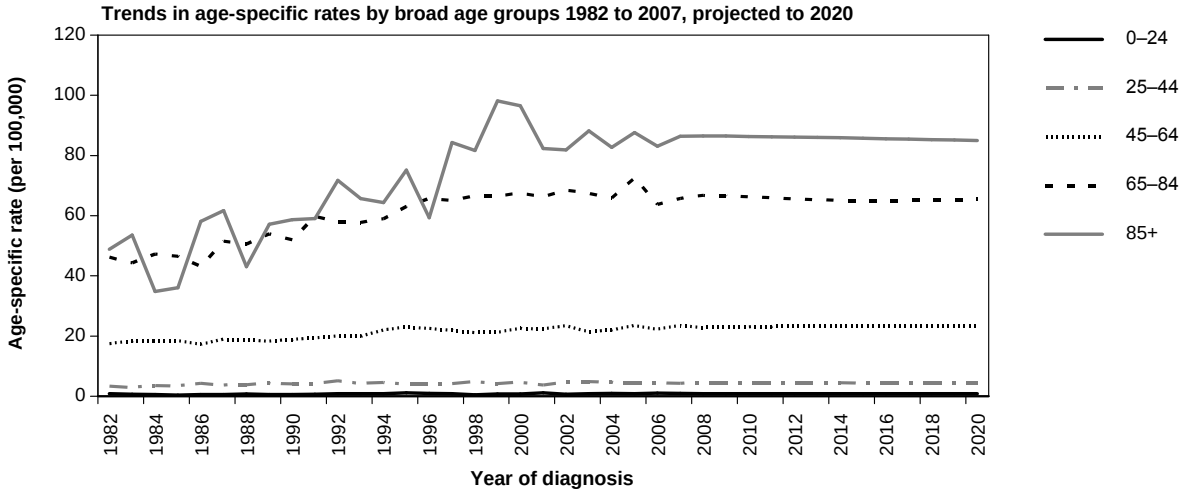
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	30	0.9	140	4.5	660	23.2	935	66.1	235	86.2
2012	30	0.9	145	4.5	670	23.3	965	65.7	245	86.1
2013	30	0.9	145	4.5	675	23.3	1,000	65.4	250	86.0
2014	30	0.9	145	4.5	690	23.4	1,030	65.2	260	85.9
2015	35	0.9	150	4.5	695	23.4	1,060	65.1	265	85.8
2016	35	0.9	150	4.5	710	23.4	1,100	65.0	270	85.6
2017	35	0.9	150	4.5	720	23.4	1,140	65.1	275	85.4
2018	35	0.9	150	4.5	725	23.4	1,180	65.2	280	85.3
2019	35	0.9	150	4.5	735	23.5	1,220	65.3	285	85.1
2020	35	0.9	155	4.5	745	23.6	1,270	65.5	290	85.0

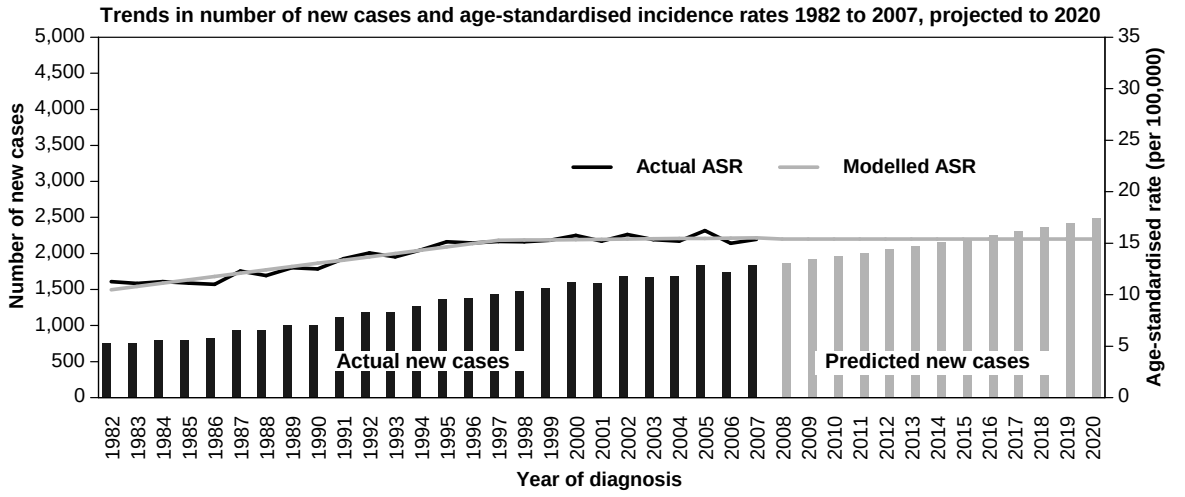
Notes

1. Non-Hodgkin lymphoma includes ICD-10 code C82–C85.
2. Projected estimates are based on incidence data for 1997 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.13b: Non-Hodgkin lymphoma (C82–C85), females, Australia: actual 1982–2007 and projected to 2020

Oesophageal cancer (C15)

There were 1,264 new cases of oesophageal cancer diagnosed in Australia in 2007 which represents 1.2% of new cancer cases. Males are three times more likely to develop oesophageal cancer than females. The likelihood of having oesophageal cancer increases with increasing age.

Risk factors for oesophageal cancer differ according to the different histological types of this cancer. Cigarette smoking, high BMI and obesity, low intake of fresh fruit and vegetables and gastro-oesophageal reflux disorder (GORD) are responsible for an estimated 79% of oesophageal adenocarcinoma cases. There is also convincing evidence that consumption of alcoholic drinks increase the risk of oesophageal adenocarcinoma (WCRF/AICR 2007). Cigarette smoking, excess alcohol consumption and low fruit and vegetable intake are responsible for an estimated 89% of squamous cell carcinoma cases (Engel et al. 2003). The effect of smoking is more pronounced for squamous cell carcinoma than adenocarcinoma, with current-smokers estimated to have four times the risk for oesophageal adenocarcinoma and nine times the risk for oesophageal squamous cell carcinoma compared with non-smokers (Freedman et al. 2007).

Males

In males, there has been a gradual increasing trend in age-standardised incidence rates of oesophageal cancer since 1982, the first year for which national incidence data are available. Rates increased significantly at about 0.2 cases per 100,000 males per year between 1982 and 1994. From 1994, there has been no significant increase in the age-standardised rate, which remains steady at about 8.3 cases per 100,000 males.

Projecting age-specific rates for 1994 to 2007, it is expected that age-standardised rates of oesophageal cancer in males will remain steady at about 8.4 cases per 100,000 males, equating to approximately 1,270 new cases in 2020 (Table 3.14a). Age-specific rates are expected to remain steady in all major age groups (Table 3.14b, Figure 3.14a).

Females

Like males, age-standardised rates of oesophageal cancer in females increased from 1982 to 1994, though the increase was slight and non-significant. From 1994, age-standardised rates have decreased significantly. Use of a log-linear joinpoint model (as the trend is decreasing) shows the most recent log-linear trend to be a significant exponential decrease in the age-standardised rate of about 1.8% each year from 1994 onward—that is about 6 cases per 100,000 females. Consequently, the observation window for the projection model of oesophageal cancer in females was set at 1994 to 2007.

Extrapolation of age-specific trends from 1994 onwards suggest that age-standardised rates of oesophageal cancer in females will continue to decrease slowly to about 2.9 new cases diagnosed per 100,000 females in 2020, equating to approximately 515 new cases (Table 3.14c). This decrease is expected to predominantly occur in females aged 65–84 (Table 3.14d, Figure 3.14b).

Table 3.14a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: oesophageal cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	975	905	1,050	8.4	7.8	9.0
2012	1,010	935	1,080	8.4	7.8	9.0
2013	1,040	965	1,110	8.4	7.8	9.0
2014	1,070	995	1,140	8.4	7.8	9.0
2015	1,100	1,020	1,180	8.4	7.8	9.0
2016	1,130	1,050	1,210	8.4	7.8	9.0
2017	1,170	1,090	1,250	8.4	7.8	9.0
2018	1,200	1,120	1,290	8.4	7.8	9.0
2019	1,240	1,150	1,320	8.5	7.9	9.0
2020	1,270	1,180	1,360	8.5	7.9	9.1

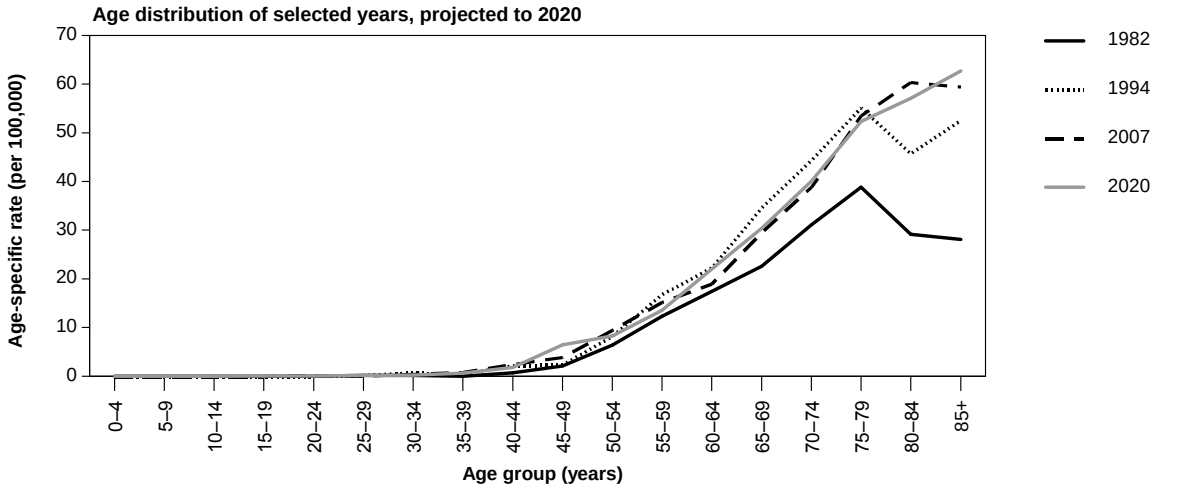
Table 3.14b: Projected number of new cases and age-specific rates, males, 2011–2020: oesophageal cancer

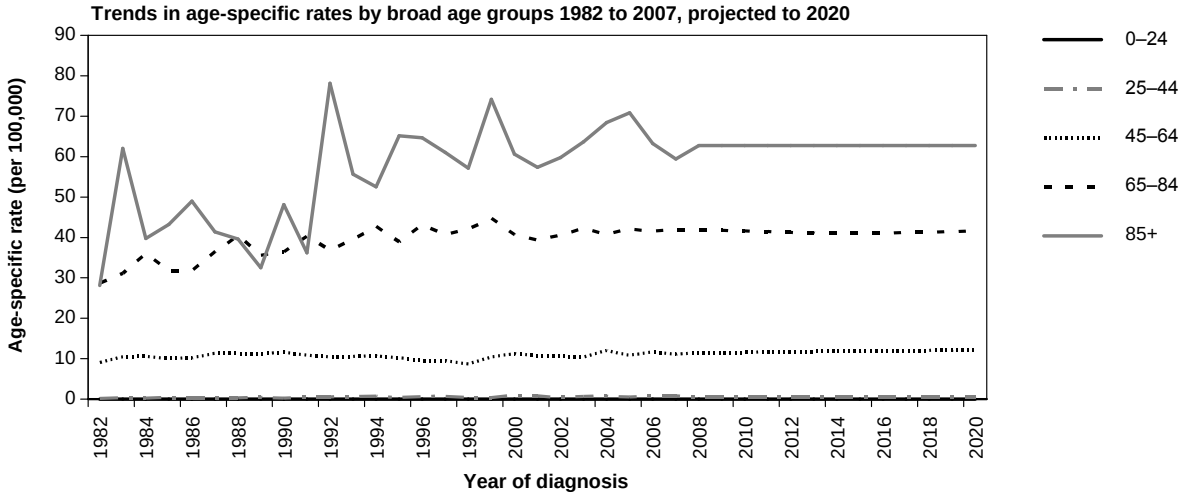
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	20	0.7	325	11.7	535	41.6	90	62.7
2012	0	0.0	20	0.7	330	11.7	555	41.3	95	62.7
2013	0	0.0	20	0.7	335	11.8	575	41.2	100	62.7
2014	0	0.0	25	0.7	340	11.9	595	41.1	105	62.7
2015	0	0.0	25	0.7	345	11.9	620	41.1	110	62.7
2016	0	0.0	25	0.7	355	12.0	640	41.1	115	62.7
2017	0	0.0	25	0.7	360	12.0	665	41.2	120	62.7
2018	0	0.0	25	0.7	365	12.1	690	41.3	125	62.7
2019	0	0.0	25	0.7	370	12.1	715	41.4	125	62.7
2020	0	0.0	25	0.7	375	12.2	740	41.6	130	62.7

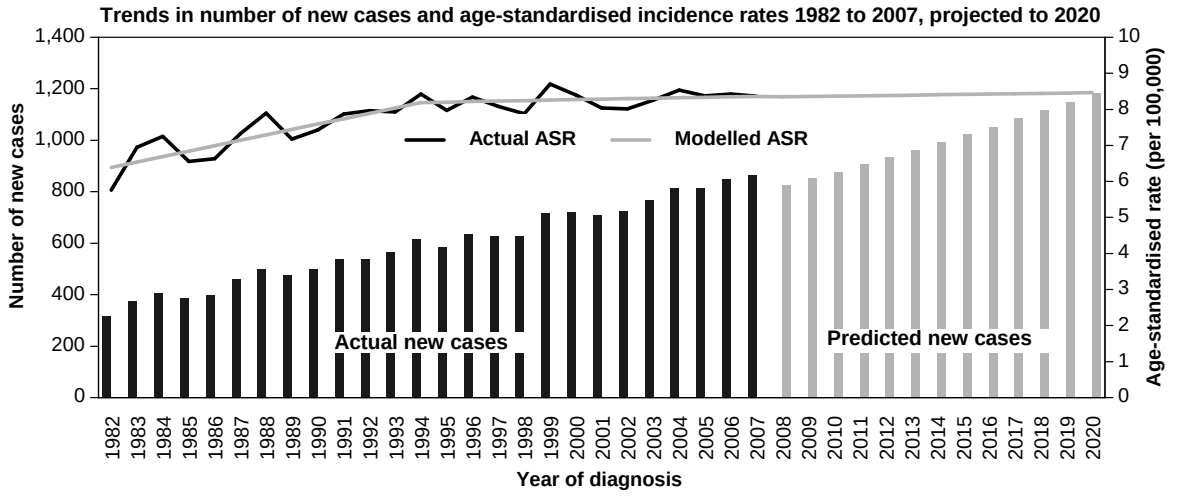
Notes

1. Oesophageal cancer includes ICD-10 code C15.
2. Projected estimates are based on incidence data for 1994 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.14a: Oesophageal cancer (C15), males, Australia: actual 1982–2007 and projected to 2020

Table 3.14c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: oesophageal cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	435	385	485	3.1	2.7	3.5
2012	445	390	495	3.1	2.7	3.4
2013	450	400	505	3.0	2.7	3.4
2014	460	405	515	3.0	2.6	3.4
2015	465	410	525	3.0	2.6	3.4
2016	475	420	530	3.0	2.6	3.3
2017	485	425	545	2.9	2.6	3.3
2018	495	435	555	2.9	2.6	3.3
2019	505	440	565	2.9	2.5	3.3
2020	515	450	575	2.9	2.5	3.3

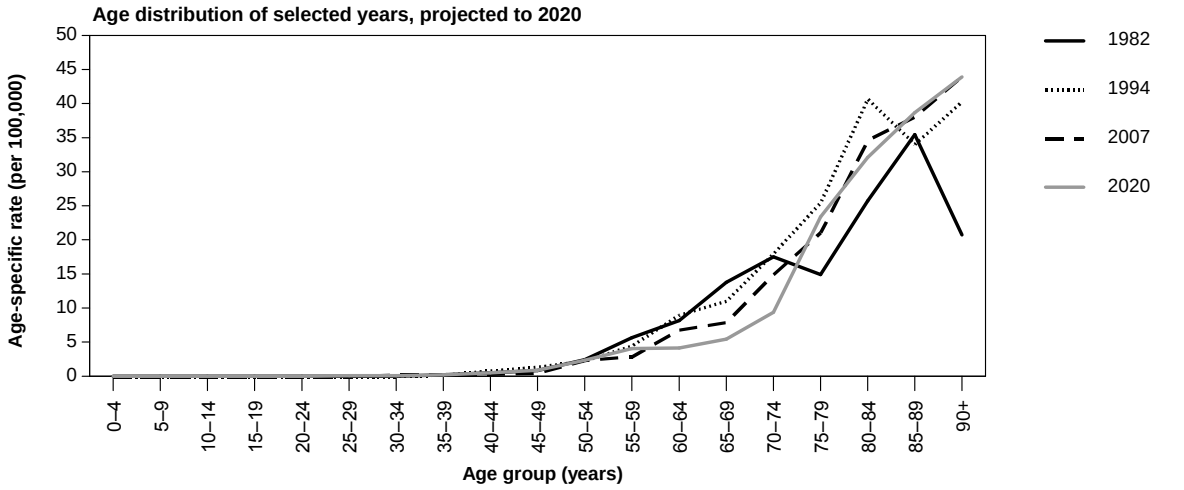
Table 3.14d: Projected number of new cases and age-specific rates, females, 2011–2020: oesophageal cancer

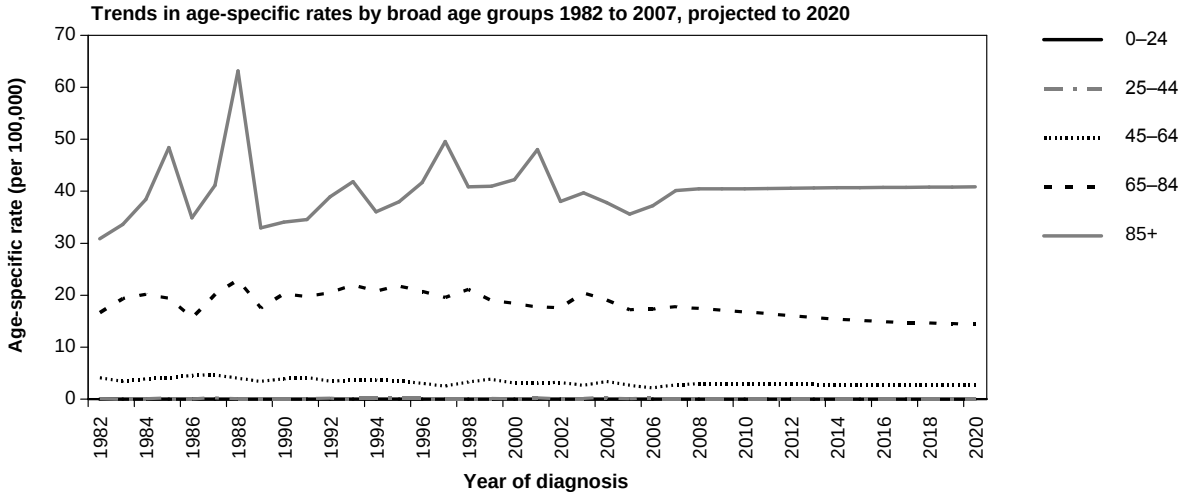
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	5	0.2	85	3.0	235	16.5	110	40.5
2012	0	0.0	5	0.2	85	2.9	235	16.1	115	40.6
2013	0	0.0	5	0.2	85	2.9	240	15.8	120	40.6
2014	0	0.0	5	0.2	85	2.9	245	15.5	125	40.7
2015	0	0.0	5	0.2	85	2.9	250	15.2	125	40.7
2016	0	0.0	5	0.2	85	2.9	250	15.0	130	40.7
2017	0	0.0	5	0.2	85	2.8	260	14.8	130	40.8
2018	0	0.0	5	0.2	85	2.8	265	14.7	135	40.8
2019	0	0.0	5	0.2	85	2.8	275	14.6	135	40.8
2020	0	0.0	5	0.2	90	2.8	280	14.5	140	40.8

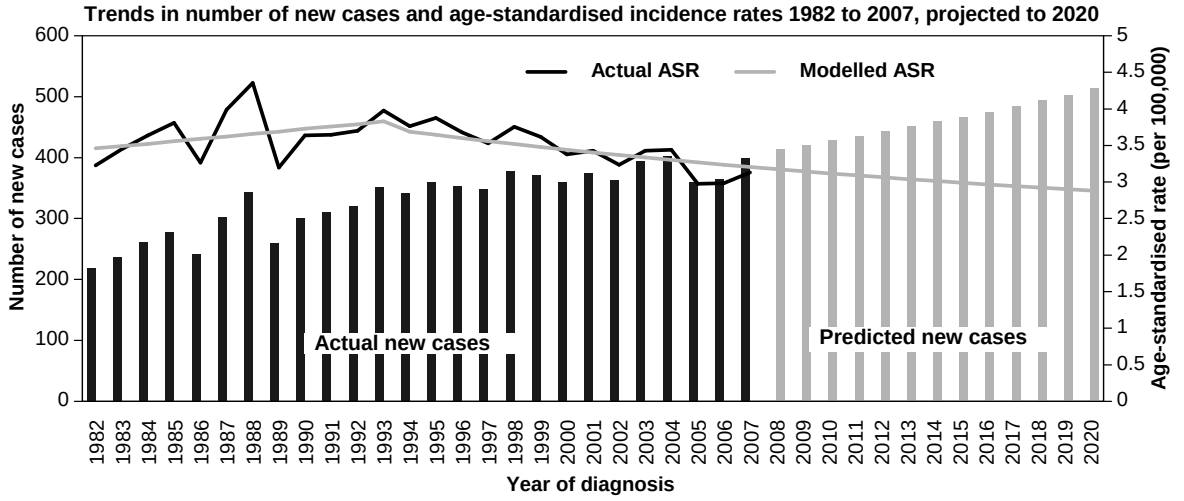
Notes

1. Oesophageal cancer includes ICD-10 code C15.
2. Projected estimates are based on incidence data for 1994 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.14b: Oesophageal cancer (C15), females, Australia: actual 1982–2007 and projected to 2020

Ovarian cancer (C56)

Ovarian cancer is the ninth most common cancer in Australian women with 1,266 new cases diagnosed in 2007. Unlike breast and cervical cancer, no effective tests are available for population-based screening and it is often diagnosed at a stage where the cancer has spread beyond the ovary. While prognoses for women diagnosed with ovarian cancer are improving, there were 64 deaths for every 100 cases diagnosed in 2007, making it the seventh most common cause of cancer-related deaths in Australian women (AIHW & NBOCC 2010).

Decreases in ovarian cancer incidence in younger women have been detected in those countries where use of oral contraceptive is widespread (La Vecchia 2006). Studies have shown that use of oral contraception may provide long-term protection against ovarian cancer (La Vecchia 2006; Calle et al. 2008).

Joinpoint analyses of age-standardised rates of ovarian cancer in Australia showed a slight, but non-significant increase of 0.01 cases per 100,000 females per year from 1982 to 1992. After this time, there has been a statistically significant decline in age-standardised rates of ovarian cancer of 0.1 cases per 100,000 females per year.

Extrapolation of this trend estimates that age-standardised incidence rates will continue to decline from 10.7 new cases diagnosed per 100,000 females in 2011 to 10.2 in 2020. Actual incidence is expected to increase from an estimated 1,390 new cases in 2011 to 1,640 new cases in 2020 with the expected changes in the population (Table 3.15a).

Analysis of age-specific projections showed that incidence rates of ovarian cancer in younger females (aged between 0–24 and 25–44) are expected to remain steady at 0.7 and 3.6 cases per 100,000 females respectively, while rates for females aged 45–64 and 65–84 are expected to decline. Rates for females aged 85 and over are expected to increase from about 56 cases per 100,000 females in 2011 to 60 in 2020 (Table 3.15b, Figure 3.15a).

Table 3.15a: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: ovarian cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,390	1,290	1,490	10.7	10.0	11.4
2012	1,420	1,310	1,520	10.6	9.9	11.4
2013	1,440	1,340	1,550	10.6	9.8	11.3
2014	1,470	1,360	1,580	10.5	9.8	11.3
2015	1,500	1,390	1,610	10.5	9.7	11.2
2016	1,520	1,410	1,640	10.4	9.7	11.2
2017	1,550	1,440	1,670	10.4	9.6	11.1
2018	1,580	1,460	1,700	10.3	9.5	11.1
2019	1,610	1,480	1,730	10.2	9.5	11.0
2020	1,640	1,510	1,760	10.2	9.4	11.0

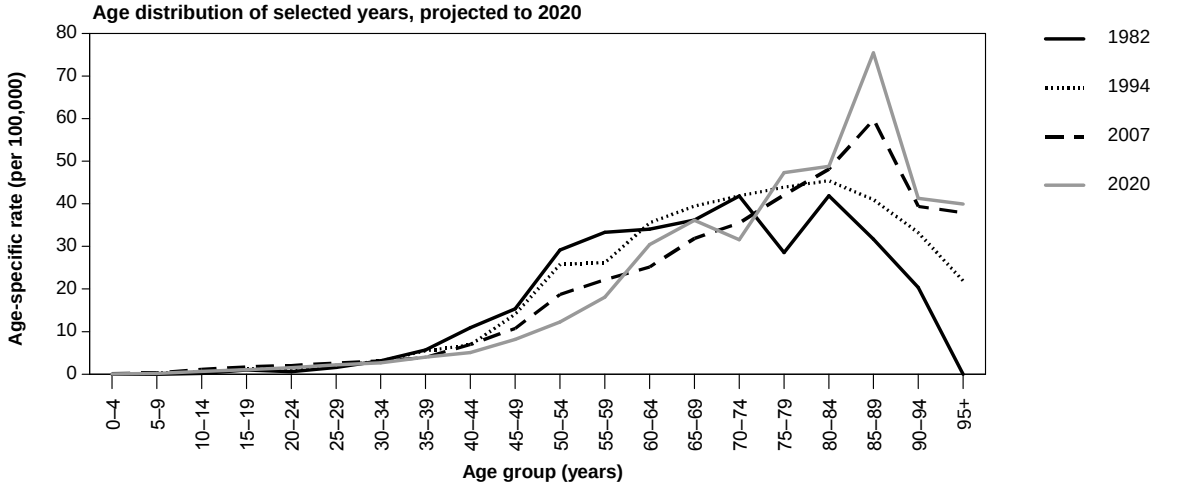
Table 3.15b: Projected number of new cases and age-specific rates, females, 2011–2020: ovarian cancer

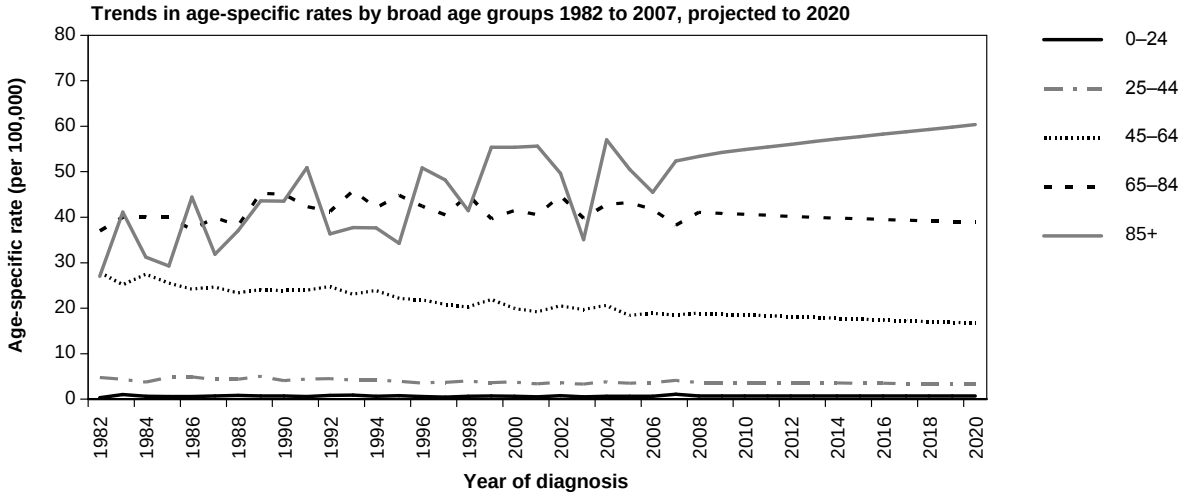
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	25	0.7	115	3.6	525	18.5	570	40.5	150	55.5
2012	25	0.7	115	3.6	525	18.2	595	40.3	160	56.0
2013	25	0.7	115	3.6	525	18.1	610	40.1	165	56.6
2014	25	0.7	115	3.6	525	17.9	630	39.9	175	57.2
2015	25	0.7	115	3.6	525	17.7	650	39.7	180	57.8
2016	25	0.7	115	3.5	530	17.5	670	39.6	185	58.3
2017	25	0.7	115	3.5	530	17.3	690	39.4	190	58.8
2018	25	0.7	120	3.5	530	17.1	710	39.3	195	59.3
2019	25	0.7	120	3.5	530	17.0	735	39.2	200	59.8
2020	25	0.7	120	3.5	530	16.8	755	39.1	205	60.4

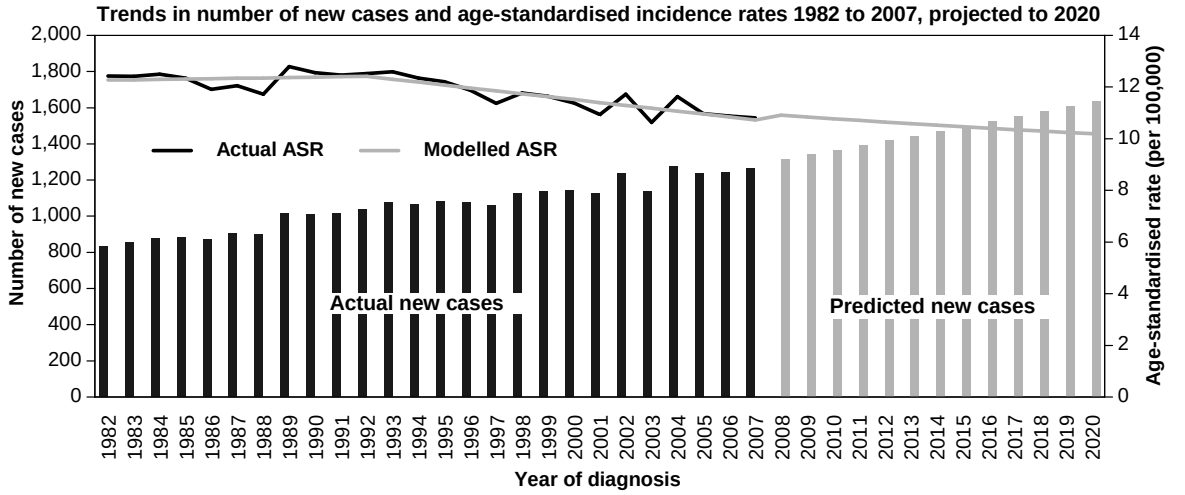
Notes

1. Ovarian cancer includes ICD-10 code C56.
2. Projected estimates are based on incidence data for 1992 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.15a: Ovarian cancer (C56), females, Australia: actual 1982–2007 and projected to 2020

Pancreatic cancer (C25)

Pancreatic cancer accounted for 2.3% of new cases of cancer in 2007, making it the ninth most common cancer diagnosis overall. The risk of pancreatic cancer increases with age, with the average age of diagnosis 71.6 years. Despite advances in cancer treatment and improved survival, outcomes remain poor. Pancreatic cancer was the sixth most common cause of cancer death in both males and females in 2007 and had the lowest 5-year relative survival rate of any of the major cancer sites.

The early stages of this cancer do not usually produce symptoms, so it is generally advanced when it is diagnosed. Poor outcomes associated with pancreatic cancer are primarily due to its presentation at an advanced stage, the aggressive behaviour of the cancer and the lack of effective treatments, especially for localised disease (Creighton et al. 2010).

Cigarette smoking is strongly associated with incidence such that up to 20–25% of pancreatic cancers are attributable to smoking (Iodice et al. 2008). Further, genetics and family history are associated with increased risk of pancreatic cancer with an estimated 5–10% due to hereditary syndromes or diseases (Hassan et al. 2007; Lochan et al. 2008). High BMI, particularly abdominal fatness, also increases the risk of pancreatic cancer (WCRF/AICR 2007). Diabetes, chronic cirrhosis, pancreatitis and prior cholecystectomy are also associated with increased risk of the disease (Huxley et al. 2005).

Males

Joinpoint analysis of age-standardised rates showed a significant change in the trend between 1982 and 2007. From 1982 to 2002, age-standardised rates were decreasing slightly, but significantly, at about 0.06 cases per 100,000 males per year. However, from 2002 to 2007, age-standardised incidence rates increased significantly at about 0.3 cases per 100,000 males per year.

A literature review revealed no documented explanation for this change, and this most recent trend is insufficient to build a projections model. As a result, projections for pancreatic cancer in males are modelled on all data from 1982 to 2007. Using these data, it is expected that age-standardised rates of pancreatic cancer in males will continue at an average of about 11.3 cases diagnosed per 100,000 males in 2020, equating to approximately 1,710 cases (Table 3.16a). Rates are expected to remain at the mean rate in all age groups (Table 3.16b, Figure 3.16a).

Females

Unlike males, rates of pancreatic cancer in females have increased steadily from 1982 to 2007 at about 0.5 cases per 100,000 females per year. Extrapolation of age-specific trends from 1982 onwards suggests that age-standardised rates will continue to increase slowly to about 9.8 new cases diagnosed per 100,000 females in 2020, equating to approximately 1,750 new cases (Table 3.16c). This increase is expected to predominantly occur in females aged 85 and over (Table 3.16d, Figure 3.16b).

Table 3.16a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: pancreatic cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,330	1,230	1,430	11.4	10.6	12.2
2012	1,370	1,260	1,470	11.4	10.5	12.2
2013	1,410	1,300	1,510	11.4	10.5	12.2
2014	1,450	1,340	1,560	11.4	10.5	12.2
2015	1,490	1,380	1,600	11.4	10.5	12.2
2016	1,530	1,420	1,650	11.3	10.5	12.2
2017	1,580	1,460	1,700	11.3	10.5	12.2
2018	1,620	1,500	1,750	11.3	10.5	12.2
2019	1,670	1,540	1,790	11.3	10.5	12.2
2020	1,710	1,590	1,840	11.3	10.5	12.2

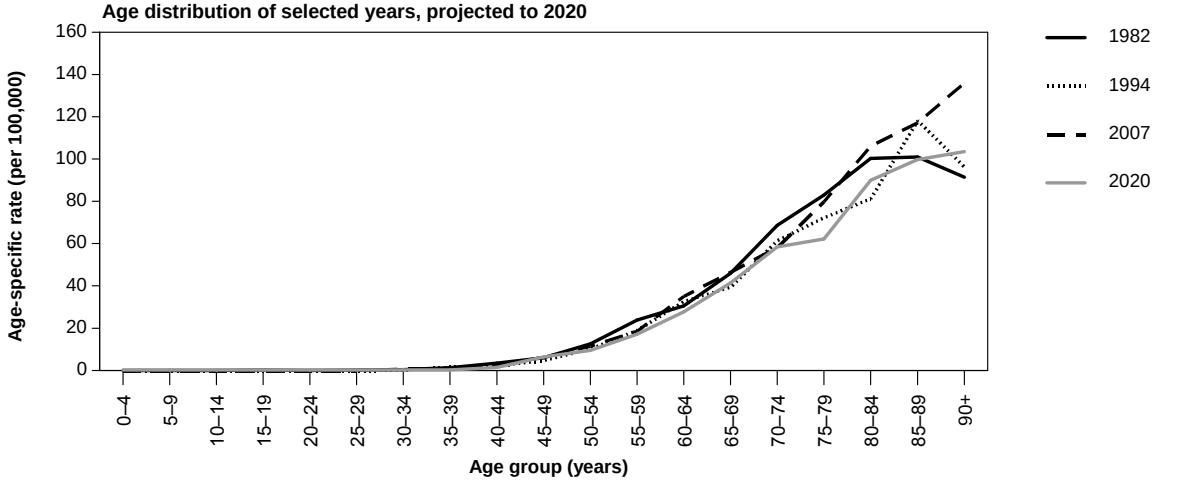
Table 3.16b: Projected number of new cases and age-specific rates, males, 2011–2020: pancreatic cancer

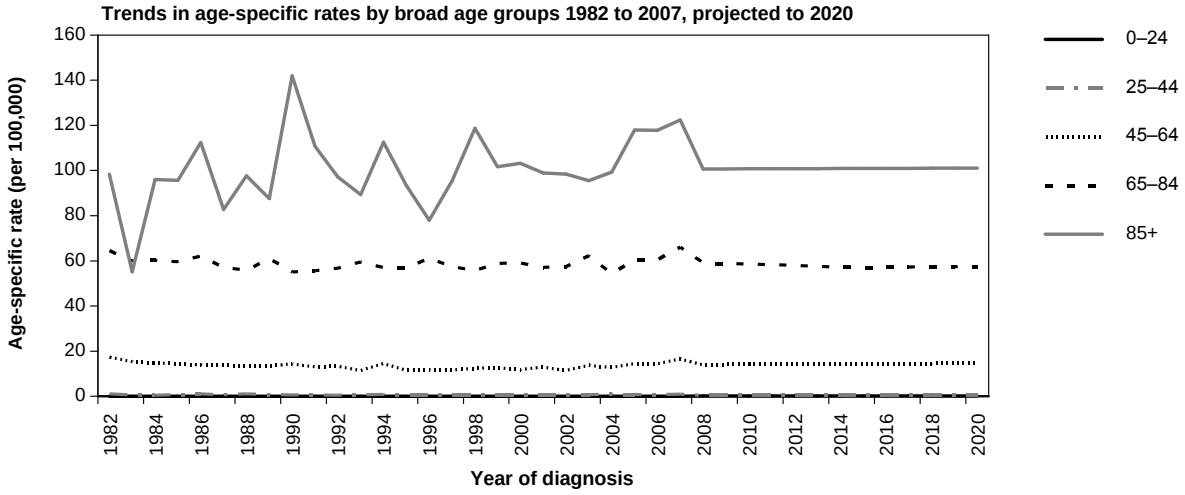
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	5	0.2	15	0.6	400	14.4	750	58.5	150	100.8
2012	5	0.2	20	0.6	405	14.4	780	58.0	155	100.9
2013	5	0.2	20	0.5	410	14.4	805	57.6	165	100.9
2014	5	0.2	20	0.5	415	14.5	835	57.4	175	100.9
2015	5	0.2	20	0.5	420	14.5	860	57.3	180	100.9
2016	5	0.2	15	0.5	430	14.5	890	57.2	190	101.0
2017	5	0.2	15	0.5	435	14.6	925	57.3	195	101.0
2018	5	0.2	15	0.5	440	14.6	960	57.4	200	101.0
2019	5	0.2	15	0.5	450	14.7	990	57.5	205	101.1
2020	5	0.2	15	0.5	455	14.7	1,030	57.6	210	101.1

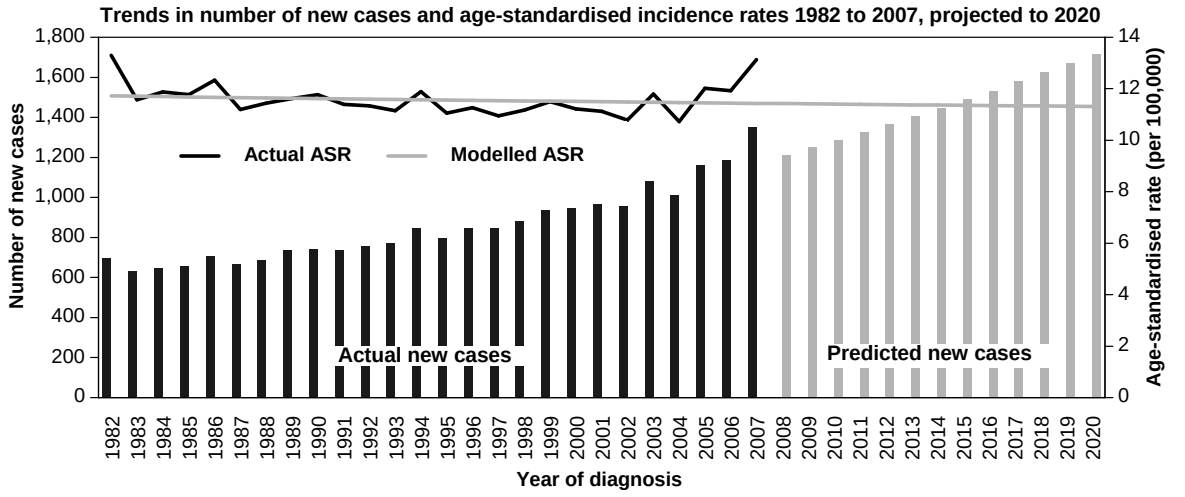
Notes

1. Pancreatic cancer includes ICD-10 code C25.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.16a: Pancreatic cancer (C25), males, Australia: actual 1982–2007 and projected to 2020

Table 3.16c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: pancreatic cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,310	1,210	1,400	9.4	8.7	10.1
2012	1,350	1,250	1,440	9.4	8.8	10.1
2013	1,390	1,300	1,490	9.5	8.8	10.2
2014	1,440	1,340	1,540	9.5	8.9	10.2
2015	1,490	1,380	1,590	9.6	8.9	10.3
2016	1,530	1,430	1,640	9.6	9.0	10.3
2017	1,590	1,470	1,700	9.7	9.0	10.4
2018	1,640	1,520	1,750	9.7	9.0	10.4
2019	1,690	1,570	1,810	9.8	9.1	10.5
2020	1,750	1,620	1,870	9.8	9.1	10.5

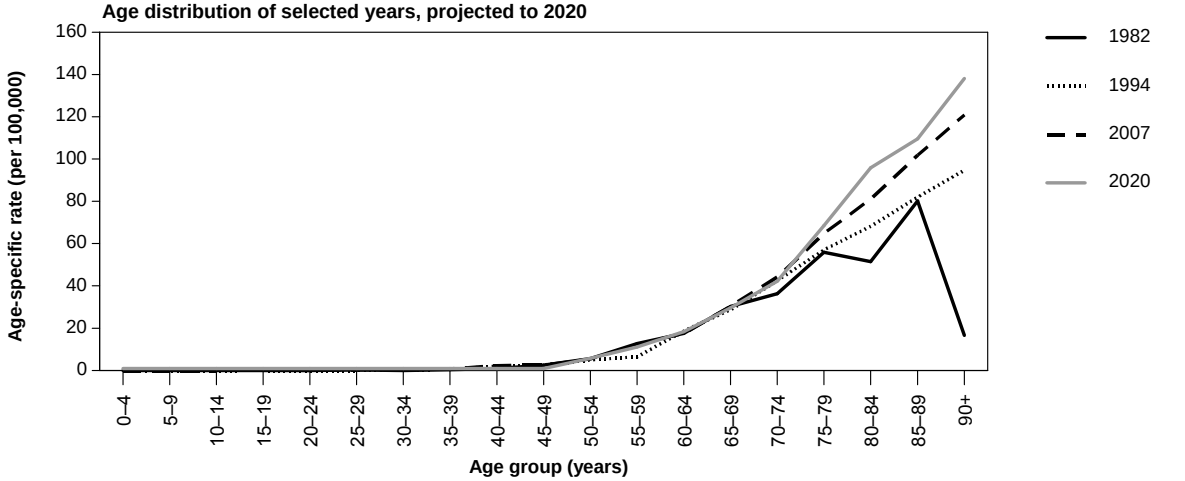
Table 3.16d: Projected number of new cases and age-specific rates, females, 2011–2020: pancreatic cancer

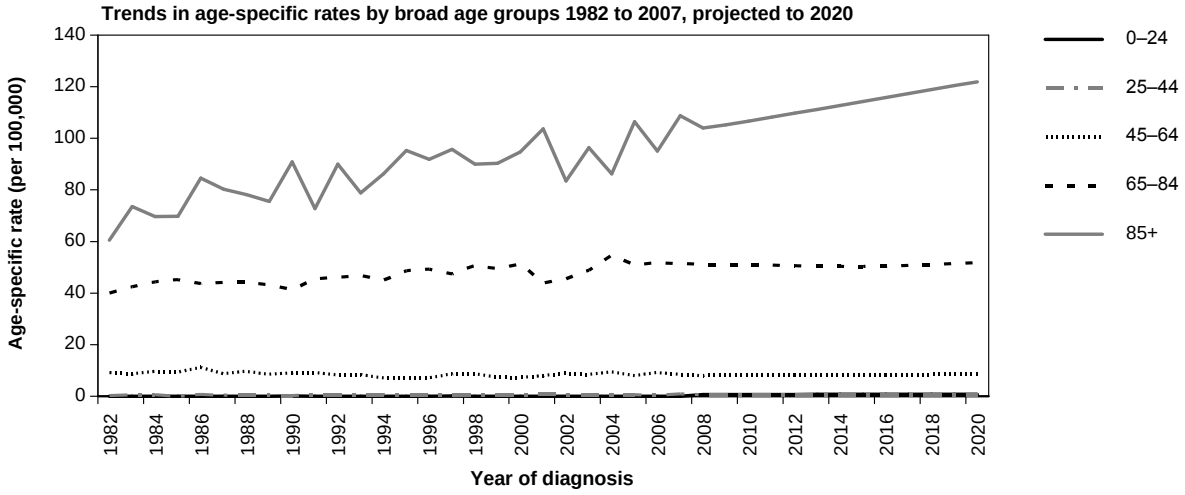
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	25	0.7	25	0.7	240	8.4	720	51.0	295	108.2
2012	25	0.7	25	0.7	240	8.4	745	50.7	310	109.7
2013	25	0.8	25	0.8	245	8.4	770	50.5	325	111.2
2014	30	0.8	25	0.8	250	8.5	795	50.4	340	112.7
2015	30	0.8	25	0.8	255	8.5	825	50.4	355	114.2
2016	30	0.8	25	0.8	255	8.5	850	50.5	370	115.8
2017	30	0.8	25	0.8	260	8.5	885	50.8	380	117.3
2018	30	0.8	30	0.8	265	8.5	925	51.1	390	118.9
2019	30	0.8	30	0.8	270	8.6	960	51.4	400	120.4
2020	30	0.8	30	0.8	275	8.6	1,000	51.8	415	121.9

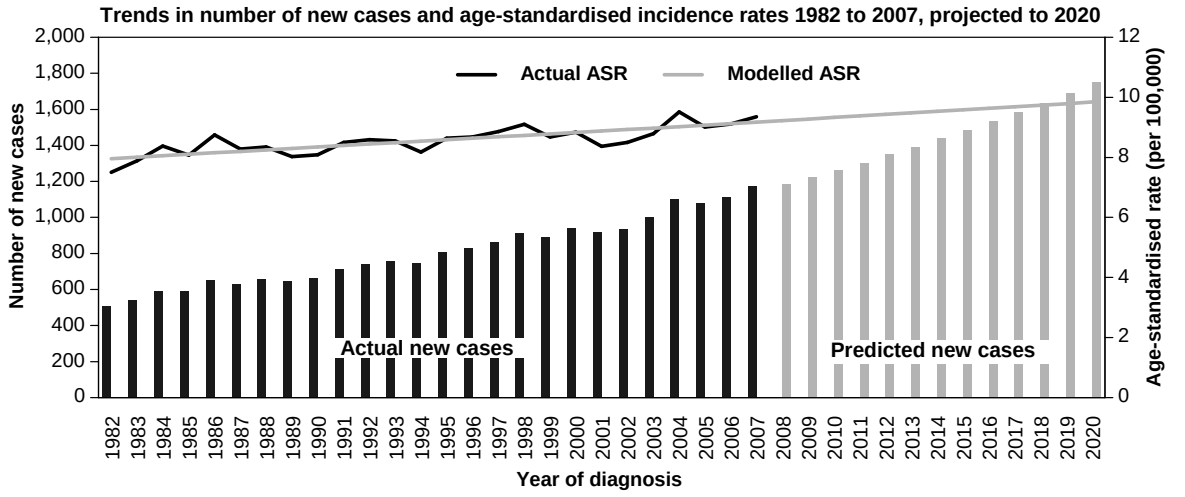
Notes

1. Pancreatic cancer includes ICD-10 code C25.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.16b: Pancreatic cancer (C25), females, Australia: actual 1982–2007 and projected to 2020

Prostate cancer (C61)

Prostate cancer accounts for about 30% of all new cases of cancer diagnosed in males (excluding non-melanoma skin cancers), and is the second most common cause of cancer-related death in males after lung cancer. With 19,403 new cases diagnosed in Australia in 2007, prostate cancer is a major public health concern.

Age-standardised incidence rates of prostate cancer in Australia have undergone a number of fluctuations since 1982. Prostate-specific antigen (PSA) testing first became available in 1987 and was listed in the Medicare Benefits Schedule in 1989. Sharp increases in the age-standardised incidence rate of prostate cancer began to appear in the early 1990s, with a peak of 184 cases per 100,000 males in 1994. This peak is attributed more to early detection, rather than elevated risk, and reflects the large pool of undiagnosed cases that were identified using the PSA test (and subsequent biopsy) that likely would have remained undiagnosed until symptoms emerged, or never diagnosed because of mortality from another condition (AIHW 2007).

After 1994 the age-standardised rate declined to 128 cases per 100,000 males in 1998, then increased slowly to 135 per 100,000 in 2002. Since 2002 there has been a further rapid increase in the number of new cases diagnosed, with rates climbing to 183 per 100,000 in 2007. This second rise in incidence is probably a result of changes in diagnostic procedures, including lowering the investigation threshold which results in more men being sent for biopsy, and increasing the number of core biopsies taken (Smith et al. 2008). It is unclear whether this latest upward trend will continue or stabilise at the current rate, or even return to lower values, as was the case in the late 1990s.

Examination of international prostate cancer incidence shows a variety of trends and rates with spikes similar to the Australian trends observed in other countries with a similar uptake of PSA testing (IARC 2008).

The fluctuating nature of prostate incidence over the past 26 years makes it difficult to project into the future with any degree of certainty. To gain further insights into the future trends, the AIHW undertook an analysis to further investigate the relationship between incidence rates and PSA testing between 2002 and 2007. This analysis showed a quantifiable relationship that could be applied to known rates of PSA testing to infer prostate cancer incidence from 2008–2010. The results are detailed in Appendix J of *Cancer in Australia: an overview, 2010* and showed an expected downturn in incidence in 2009 and 2010 (AIHW & AACR 2010). These inferred incidence counts were used to extend the base data to 2010.

To develop a model suitable for projecting to 2020, a series of models were fitted to the actual data from 1982 to 2007 and the inferred data to 2010. It should be noted that a well-fitting descriptive model may not provide a good predictive model, and the selection of the most appropriate model depends on the underlying assumptions. If it is assumed that the general upward trend over the past 26 years is indicative of increasing trends in underlying disease, then a linear model may be appropriate to project to 2020. However, if it is assumed that each of the peaks are due to increased uptake of PSA tests, or changes in diagnostic practices, which the evidence suggests, then these fluctuations represent prevalent but undiagnosed cases and it is likely the underlying trend will stabilise similar to, but higher than, the trends from 1982 to 1998 and 1998 to 2002. In this case, a logarithmic model provides a more suitable model for projecting to 2020.

As a result, this report presents two sets of projections based on these different assumptions.

Linear trend

Linear extrapolation of data from 1982 to 2007, supplemented by PSA testing data from 2008 to 2010, suggests that age-standardised incidence rates of prostate cancer will continue to increase by about 3 cases per 100,000 males per year to 200 new cases per 100,000 males. Based on expected population changes, this equates to about 31,000 new cases expected to be diagnosed in 2020 (Table 3.17a). This rise is expected to be most apparent in males aged 45–64 and 65–84; however, the rate in males aged 85 and over is expected to decrease slightly (Table 3.17b, Figure 3.17a).

Logarithmic trend

An alternative approach of logarithmic extrapolation of data from 1982 to 2007, supplemented by PSA testing data from 2008 to 2010, also suggests that age-standardised incidence rates of prostate cancer will continue to increase, but to a lower rate of about 164 new cases per 100,000 males (Table 3.17c). Based on expected population changes, this equates to around 25,310 new cases expected to be diagnosed in 2020. Similar to the linear model, the greatest increase is expected to be most apparent in males aged 45–64 and 65–84 (Table 3.17d, Figure 3.17b).

It is important to note that the prediction intervals for both the estimated number of new cases and the age-standardised rates of both models overlap.

Table 3.17a: Projected number of new cases and age-standardised rates with 95% prediction intervals, linear trend, 2011–2020: prostate cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	20,920	18,090	23,760	173.1	148.0	198.1
2012	22,020	19,080	24,970	176.0	151.0	201.1
2013	23,130	20,080	26,190	179.0	153.9	204.1
2014	24,230	21,050	27,410	181.9	156.8	207.1
2015	25,340	22,030	28,650	184.9	159.7	210.1
2016	26,480	23,020	29,930	187.8	162.6	213.1
2017	27,560	23,920	31,200	190.8	165.5	216.1
2018	28,680	24,870	32,490	193.7	168.4	219.1
2019	29,840	25,890	33,790	196.7	171.3	222.1
2020	31,000	26,920	35,090	199.7	174.2	225.1

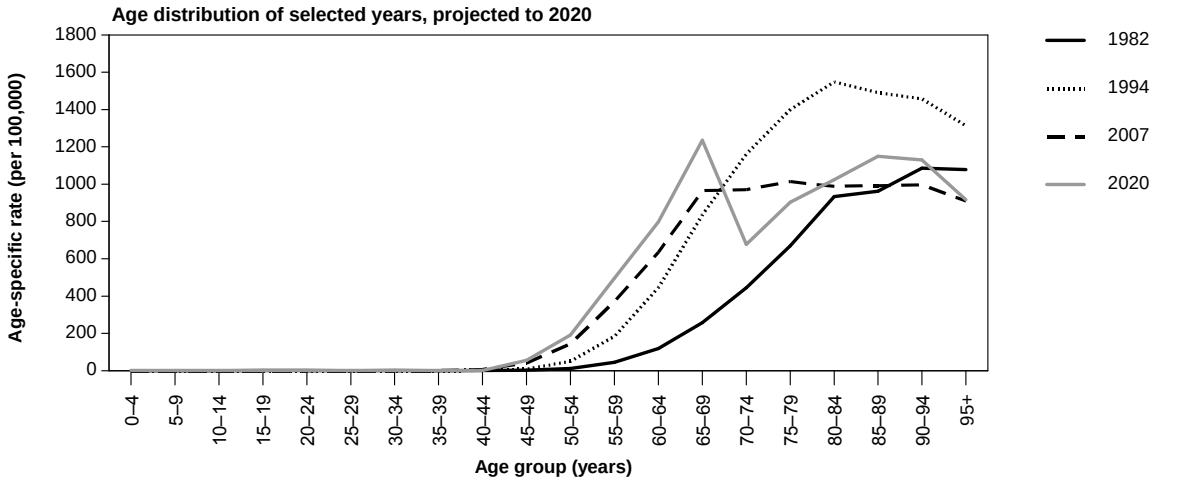
Table 3.17b: Projected number of new cases and age-specific rates, linear trend, 2011–2020: prostate cancer

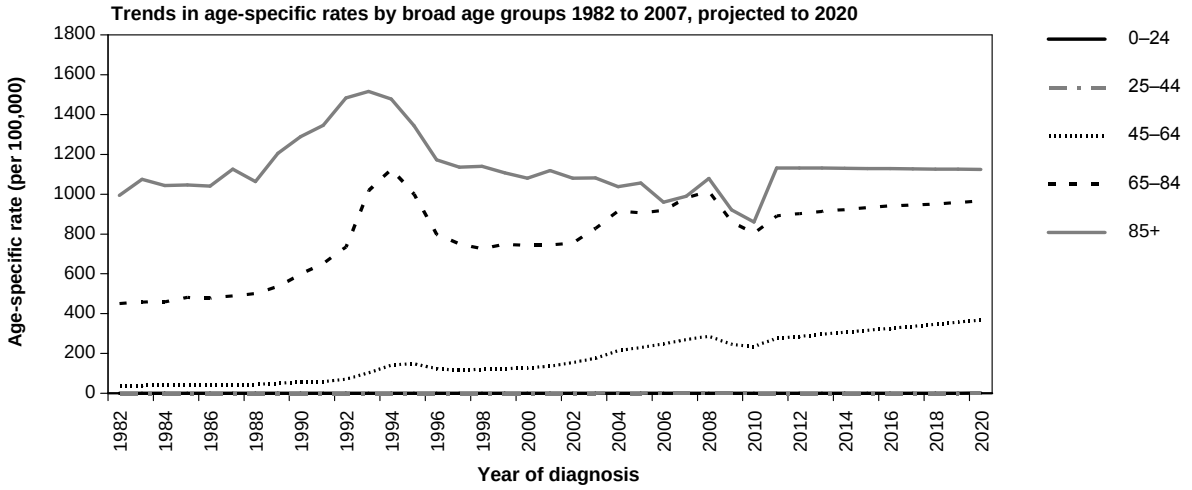
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	35	0.9	30	0.9	7,750	276.8	11,460	891.6	1,660	1131.1
2012	35	0.9	30	0.9	8,060	286.1	12,150	902.6	1,750	1130.8
2013	35	1.0	30	1.0	8,420	296.3	12,790	913.8	1,850	1130.7
2014	40	1.0	35	1.0	8,820	306.6	13,400	923.2	1,930	1130.3
2015	40	1.0	35	1.0	9,210	316.5	14,030	932.3	2,020	1129.2
2016	40	1.1	35	1.1	9,640	326.3	14,660	940.9	2,100	1128.2
2017	45	1.1	40	1.1	10,070	336.3	15,250	945.2	2,160	1127.1
2018	45	1.2	40	1.2	10,490	346.6	15,890	951.2	2,220	1126.2
2019	45	1.2	40	1.2	10,930	357.7	16,550	958.9	2,280	1125.3
2020	50	1.2	45	1.2	11,350	368.6	17,220	966.5	2,340	1124.4

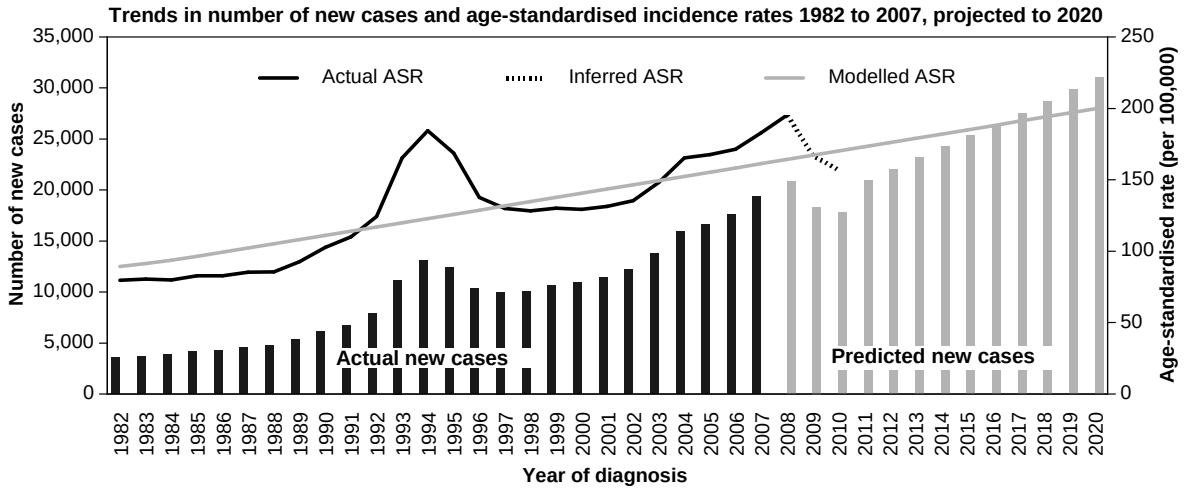
Notes

1. Prostate cancer includes ICD-10 code C61.
2. Projected estimates are based on a linear extrapolation of actual incidence data for 1982 to 2007, and estimated incidence for 2008 to 2010.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.17a: Prostate cancer (C61), linear trend, Australia: actual 1982–2007, estimated 2008–2010 and projected to 2020

Table 3.17c: Projected number of new cases and age-standardised rates with 95% prediction intervals, logarithmic trend, 2011–2020: prostate cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	18,580	15,360	21,810	156.0	128.6	183.4
2012	19,340	15,990	22,680	156.9	129.5	184.4
2013	20,080	16,620	23,540	157.8	130.4	185.3
2014	20,810	17,230	24,400	158.7	131.3	186.2
2015	21,550	17,840	25,270	159.6	132.1	187.1
2016	22,310	18,450	26,160	160.4	132.9	187.9
2017	23,030	19,010	27,060	161.2	133.7	188.8
2018	23,780	19,590	27,960	162.0	134.5	189.6
2019	24,540	20,230	28,850	162.8	135.2	190.4
2020	25,310	20,870	29,750	163.5	136.0	191.1

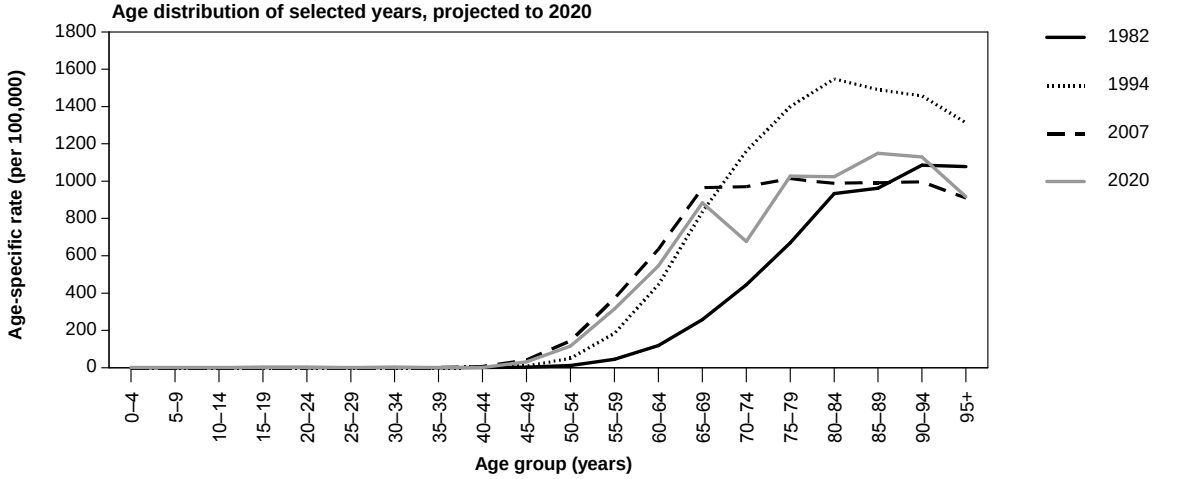
Table 3.17d: Projected number of new cases and age-specific rates, logarithmic trend, 2011–2020: prostate cancer

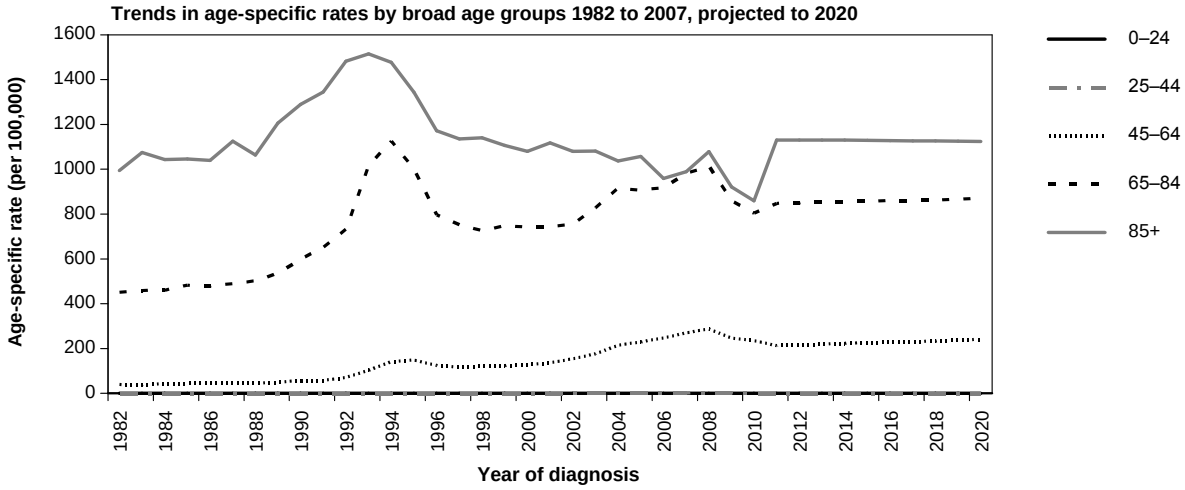
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	25	0.6	20	0.6	5,990	214.0	10,890	847.6	1,660	1131.1
2012	25	0.6	20	0.6	6,100	216.6	11,440	850.0	1,750	1130.8
2013	25	0.7	20	0.7	6,250	219.9	11,940	852.8	1,850	1130.7
2014	25	0.7	20	0.7	6,420	223.1	12,420	855.3	1,930	1130.3
2015	25	0.7	20	0.7	6,580	225.9	12,910	857.8	2,020	1129.2
2016	25	0.7	25	0.7	6,750	228.6	13,400	860.2	2,100	1128.2
2017	25	0.7	25	0.7	6,930	231.4	13,890	861.4	2,160	1127.1
2018	25	0.7	25	0.7	7,090	234.3	14,410	863.1	2,220	1126.2
2019	30	0.7	25	0.7	7,260	237.7	14,950	866.5	2,280	1125.3
2020	30	0.7	25	0.7	7,420	240.8	15,500	869.9	2,340	1124.4

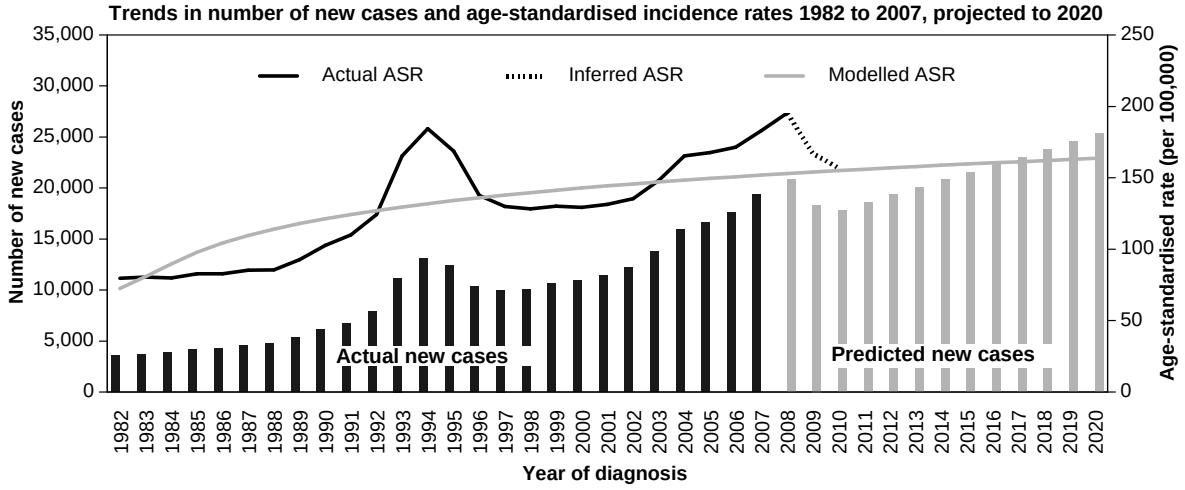
Notes

1. Prostate cancer includes ICD-10 code C61.
2. Projected estimates are based on a logarithmic extrapolation of actual incidence data for 1982 to 2007, and estimated incidence for 2008 to 2010.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.17b: Prostate cancer (C61), logarithmic trend, Australia: actual 1982–2007, estimated 2008–2010 and projected to 2020

Stomach cancer (C16)

There were 1,897 new cases of stomach cancer diagnosed in Australia in 2007 which represents 1.8% of all new cancer cases. Stomach cancer occurs mainly in older people and is twice as common in males as females, with age-standardised rates of 11.8 per 100,000 males, and 5.6 per 100,000 females in 2007.

A common stomach bacteria, *Helicobacter pylori,* is a strong risk factor for stomach cancer and is a necessary, although not sufficient, cause of almost all cases (Forman & Burley 2006; WCRF/AICR 2007). Diets low in fruit and vegetables and with a high intake of salted, smoked, cured and/or pickled foods, and heavily grilled or barbecued meat and fish are associated with increased risk of stomach cancer (Hu et al. 2011). Some researchers have suggested that since *Helicobacter pylori* and high salt intake correlate, these factors may interact to promote the development of stomach cancer (Wang et al. 2009). However, the evidence for the effect of diet-related risk factors is inconsistent (Brenner et al. 2009).

The incidence of stomach cancer has been decreasing in Australia since national data were available in 1982. This decrease is commonly attributed to improved diagnosis and treatment of infection with *Helicobacter pylori*, changes in diet and nutrition and improvements in food preservation (Parkin 2001). Cigarette smoking is also an associated risk factor (Ladeiras-Lopes et al. 2008) and reductions in the smoking rate in Australia may also translate into reductions in the incidence of smoking-related stomach cancer.

Males

Joinpoint analysis of age-standardised rates shows stomach cancer decreasing significantly at about 0.4 cases per 100,000 males per year since 1982. Extrapolation of age-specific trends from 1982 to 2007 indicate that rates are expected to continue to decrease in a logarithmic fashion to 8.9 per 100,000 males in 2020 which, taking into account expected changes to the population structure, will translate to an estimated 1,340 new cases diagnosed. Decreases in rates will be most apparent for those aged 85 and over, closely followed by those aged 65–84 (tables 3.18a and 3.18b, Figure 3.18a).

Females

Joinpoint analysis of age-standardised rates shows stomach cancer in females decreasing at a similar rate to males (0.4 cases per 100,000 females per year) from 1982 to 1988, but then slowing to a decrease of about 0.1 case per 100,000 females per year from 1988 to 2007.

Extrapolation of age-specific trends from 1988 to 2007 indicate that rates are expected to continue to decrease in a logarithmic fashion to 4.5 per 100,000 females in 2020 which, taking into account expected changes to the population structure, will translate to an estimated 740 new cases diagnosed (Table 3.18c). As for males, decreases in rates will be most apparent for females aged 85 and over (Table 3.18c, Figure 3.18b).

Table 3.18a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: stomach cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,280	1,190	1,370	11.1	10.3	11.9
2012	1,290	1,200	1,380	10.8	10.1	11.6
2013	1,300	1,210	1,390	10.6	9.8	11.3
2014	1,300	1,210	1,400	10.3	9.6	11.0
2015	1,310	1,220	1,400	10.0	9.3	10.8
2016	1,320	1,220	1,410	9.8	9.1	10.5
2017	1,330	1,230	1,420	9.6	8.9	10.3
2018	1,330	1,230	1,430	9.3	8.6	10.0
2019	1,340	1,230	1,440	9.1	8.4	9.8
2020	1,340	1,240	1,450	8.9	8.2	9.6

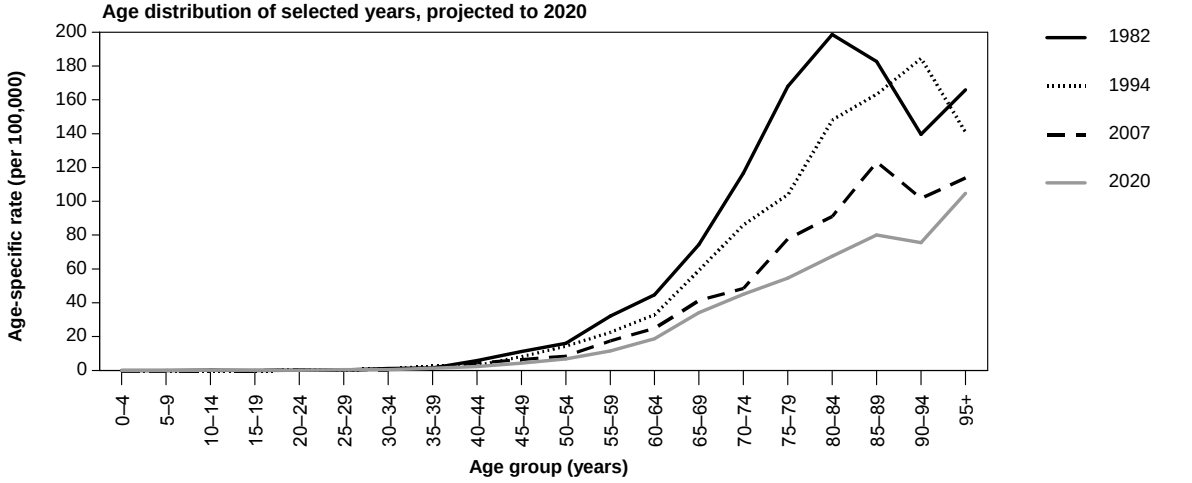
Table 3.18b: Projected number of new cases and age-specific rates, males, 2011–2020: stomach cancer

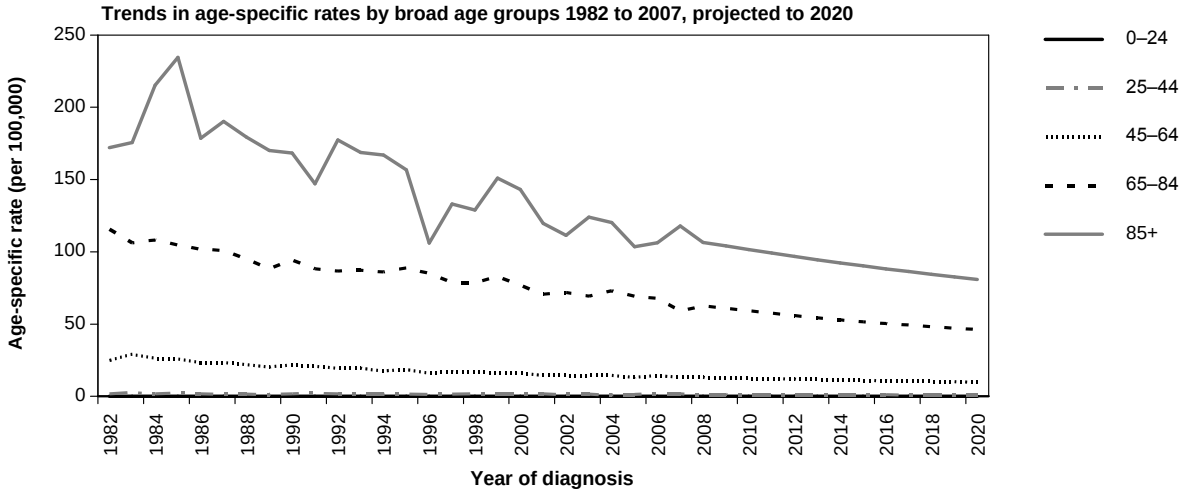
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	40	1.3	350	12.5	745	57.8	145	99.2
2012	0	0.0	40	1.3	340	12.1	755	56.1	150	96.8
2013	0	0.0	40	1.3	335	11.9	765	54.5	155	94.6
2014	0	0.0	40	1.2	335	11.6	770	53.1	160	92.3
2015	0	0.0	40	1.2	330	11.3	780	51.7	160	90.3
2016	0	0.0	40	1.2	325	11.0	785	50.5	165	88.2
2017	0	0.0	40	1.1	320	10.7	800	49.5	165	86.3
2018	0	0.0	40	1.1	315	10.5	810	48.4	165	84.4
2019	0	0.0	40	1.1	310	10.2	815	47.3	165	82.6
2020	0	0.0	40	1.1	310	10.0	825	46.3	170	80.8

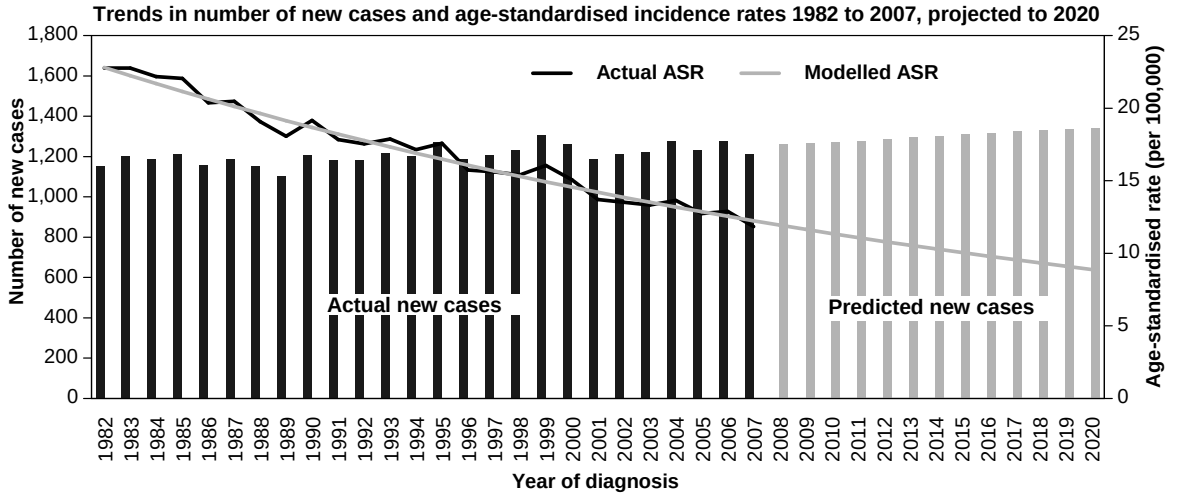
Notes

1. Stomach cancer includes ICD-10 code C16.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.18a: Stomach cancer (C16), males, Australia: actual 1982–2007 and projected to 2020

Table 3.18c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: stomach cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	705	640	765	5.2	4.8	5.7
2012	710	645	770	5.1	4.7	5.6
2013	710	645	775	5.1	4.6	5.5
2014	715	650	780	5.0	4.5	5.4
2015	720	655	790	4.9	4.4	5.3
2016	725	655	795	4.8	4.3	5.3
2017	730	660	800	4.7	4.3	5.2
2018	735	660	805	4.6	4.2	5.1
2019	740	665	810	4.6	4.1	5.0
2020	740	665	815	4.5	4.0	4.9

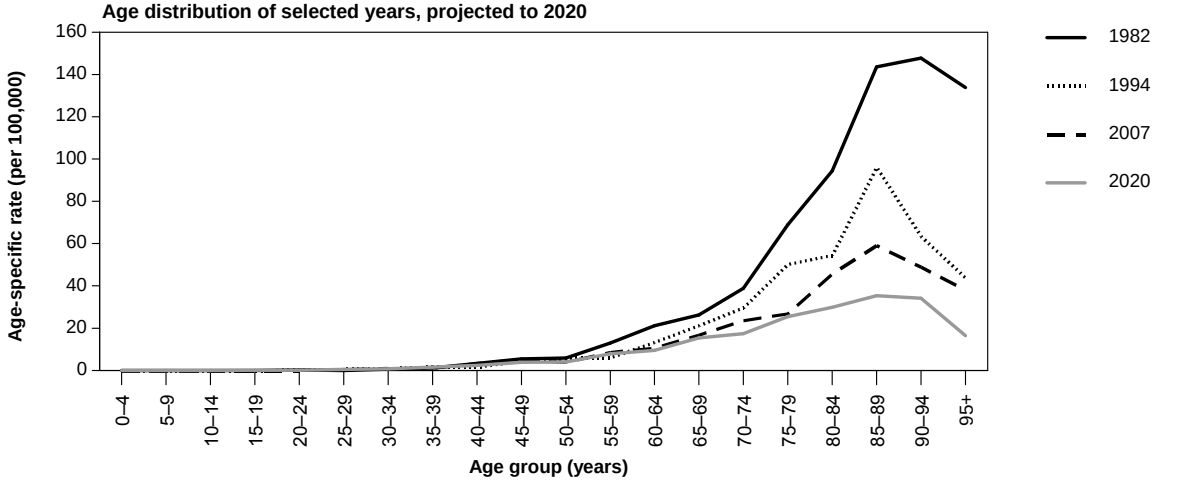
Table 3.18d: Projected number of new cases and age-specific rates, females, 2011–2020: stomach cancer

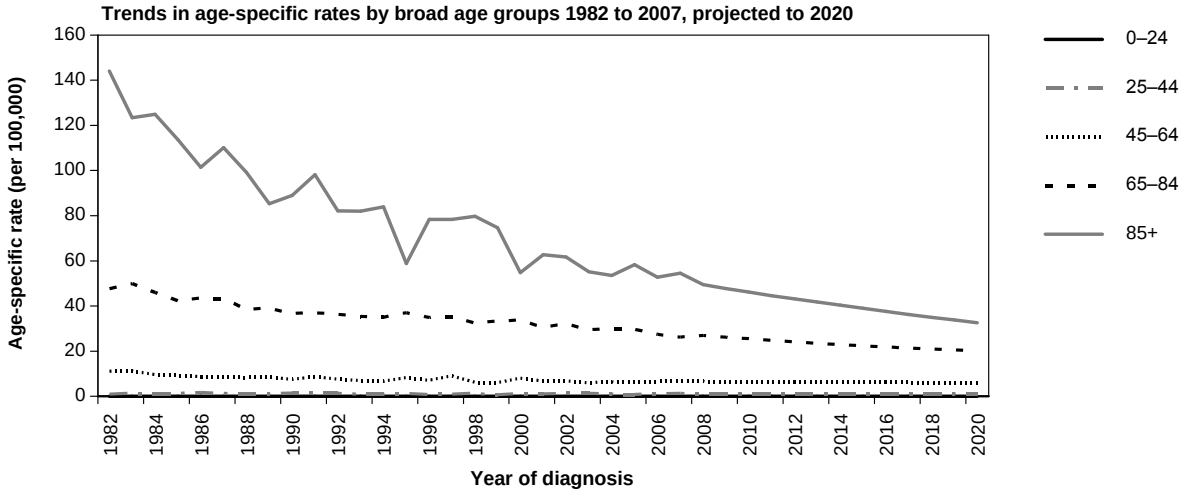
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	40	1.3	185	6.6	355	24.9	120	44.6
2012	0	0.0	40	1.3	185	6.5	355	24.2	120	43.1
2013	0	0.0	40	1.3	190	6.5	360	23.6	120	41.7
2014	0	0.0	40	1.3	190	6.4	365	23.0	120	40.4
2015	0	0.0	40	1.3	190	6.4	365	22.5	120	39.0
2016	0	0.0	40	1.2	190	6.3	370	22.0	120	37.6
2017	0	0.0	40	1.2	195	6.3	375	21.5	115	36.3
2018	0	0.0	40	1.2	195	6.2	380	21.1	115	35.0
2019	0	0.0	40	1.2	195	6.2	385	20.7	115	33.8
2020	0	0.0	45	1.2	195	6.2	390	20.3	110	32.6

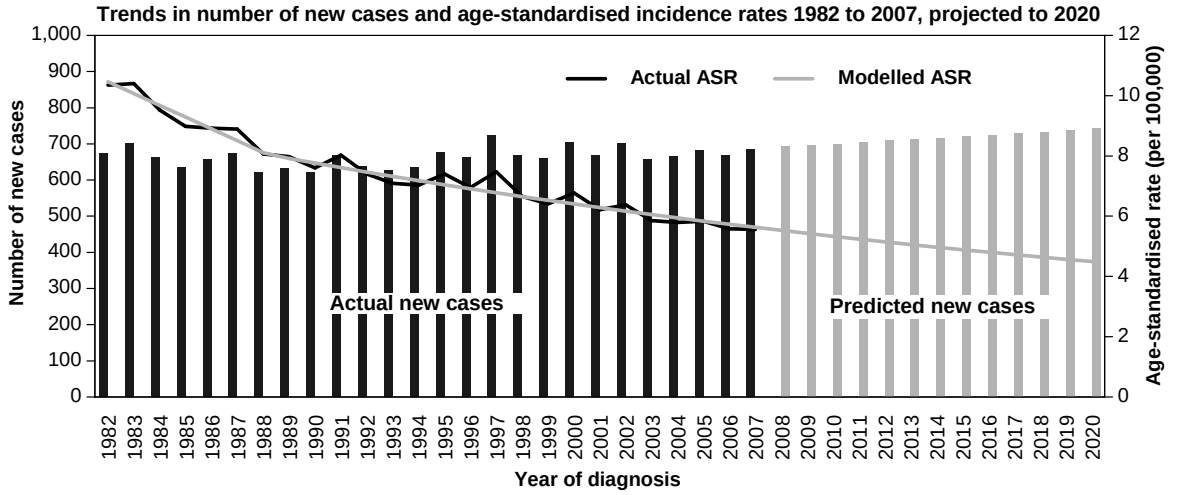
Notes

1. Stomach cancer includes ICD-10 code C16.
2. Projected estimates are based on incidence data for 1998 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.18b: Stomach cancer (C16), females, Australia: actual 1982–2007 and projected to 2020

Testicular cancer (C62)

Testicular cancer is uncommon, accounting for only 698 new cancer cases in Australia in 2007. Whereas most cancers are common among older age groups, testicular cancer occurs mostly in young and middle-aged men, specifically those between 15 and 50. In 2007, the mean age at diagnosis was 35, and it was the second most common cancer among young men between 20 and 39. Mortality rates declined between 1982 and 2007 and are very low due to improvements in treatment.

While the exact causes of testicular cancer are unknown, genetic factors are associated with the disease. Brothers, fathers and sons of men with testicular cancer have the highest associated risk, and dale infertility and undescended testis are also established risk factors (Akre et al. 2009; Richiardi et al. 2007). Some studies suggest that lifestyle and environmental factors, such as country of birth, may play a role in the development of testicular cancer (Beiki et al. 2010). Men who have had testicular cancer in one testicle are at an increased risk of developing it in the other testicle (Robinson et al. 2007).

In Australia, age-standardised incidence rates of testicular cancer have increased consistently between 1982 and 2007. Little is known about why rates are increasing (IARC 2008). The increasing trend may be at least partly due to changes over time in exposure to causal or protective factors around the time of birth (Baade et al. 2008).

Extrapolation of age-specific trends from 1982 to 2007 suggests that age-standardised rates are expected to continue to increase to about 8.5 new cases per 100,000 males in 2020. Based on expected population changes, this equates to about 1,020 new cases expected to be diagnosed in 2020 (Table 3.19a).

Consistent with the age distribution of testicular cancer, the main increase is expected to occur in males aged 25–44, with small increases also expected in males aged 0–24 and 45–64 (Table 3.19b, Figure 3.19a).

Table 3.19a: Projected number of new cases and age-standardised rates with 95% prediction intervals, 2011–2020: testicular cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	800	740	860	7.4	6.9	7.9
2012	825	765	885	7.5	7.0	8.1
2013	845	785	910	7.6	7.1	8.2
2014	870	810	935	7.7	7.2	8.3
2015	895	830	960	7.9	7.3	8.4
2016	920	855	985	8.0	7.4	8.6
2017	945	875	1,010	8.1	7.5	8.7
2018	970	900	1,040	8.2	7.6	8.8
2019	995	920	1,060	8.3	7.8	8.9
2020	1,020	945	1,090	8.5	7.9	9.0

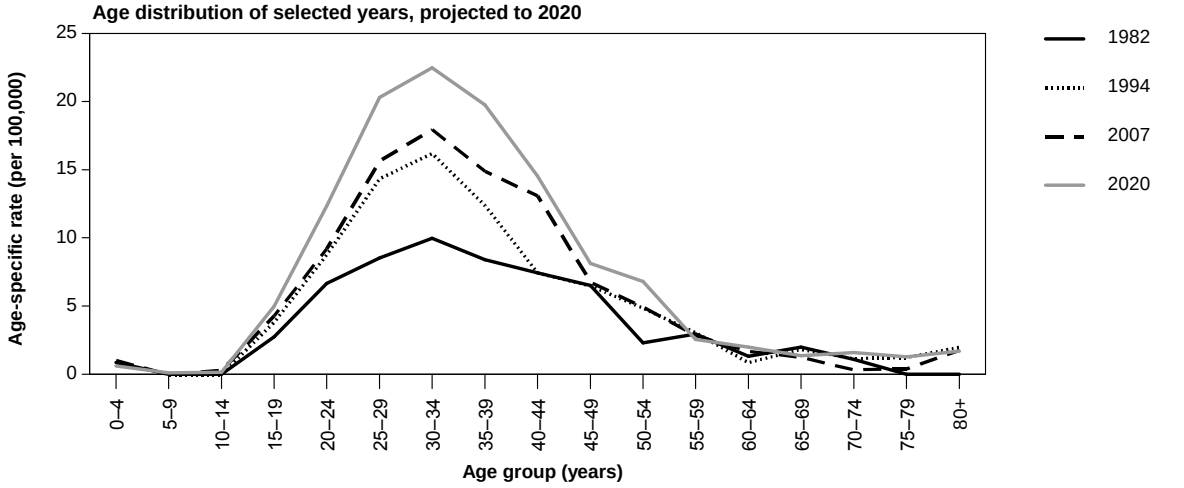
Table 3.19b: Projected number of new cases and age-specific rates, 2011–2020: testicular cancer

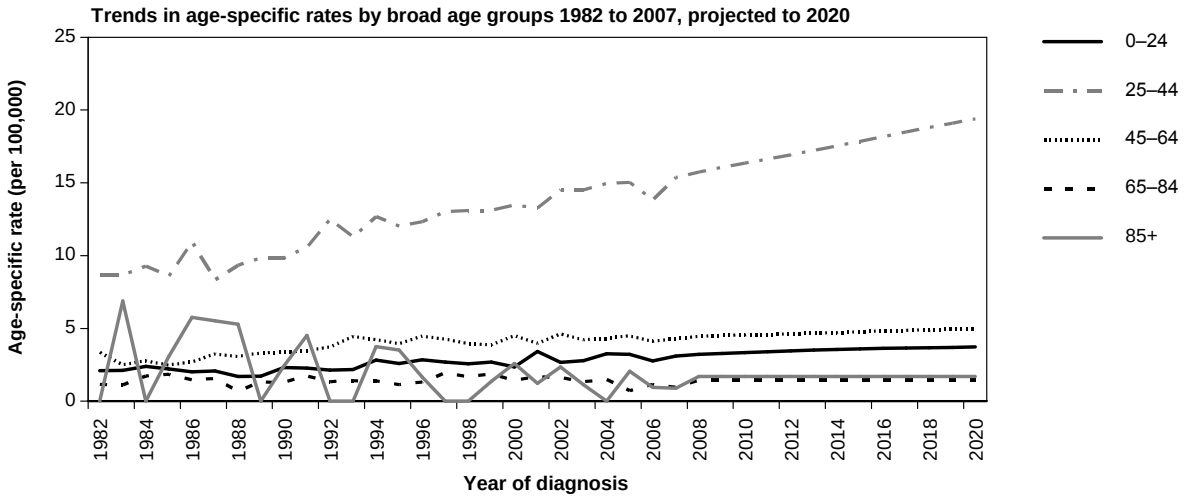
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	125	3.4	525	16.6	130	4.6	20	1.5	0	1.7
2012	130	3.5	540	16.9	130	4.6	20	1.4	5	1.7
2013	135	3.5	555	17.2	135	4.7	20	1.4	5	1.7
2014	135	3.6	575	17.5	135	4.7	20	1.4	5	1.7
2015	140	3.6	595	17.9	140	4.8	20	1.4	5	1.7
2016	140	3.6	610	18.2	145	4.8	25	1.4	5	1.7
2017	145	3.7	630	18.5	145	4.9	25	1.4	5	1.7
2018	145	3.7	645	18.8	150	4.9	25	1.5	5	1.7
2019	145	3.7	665	19.1	150	5.0	25	1.5	5	1.7
2020	150	3.7	685	19.4	155	5.0	25	1.5	5	1.7

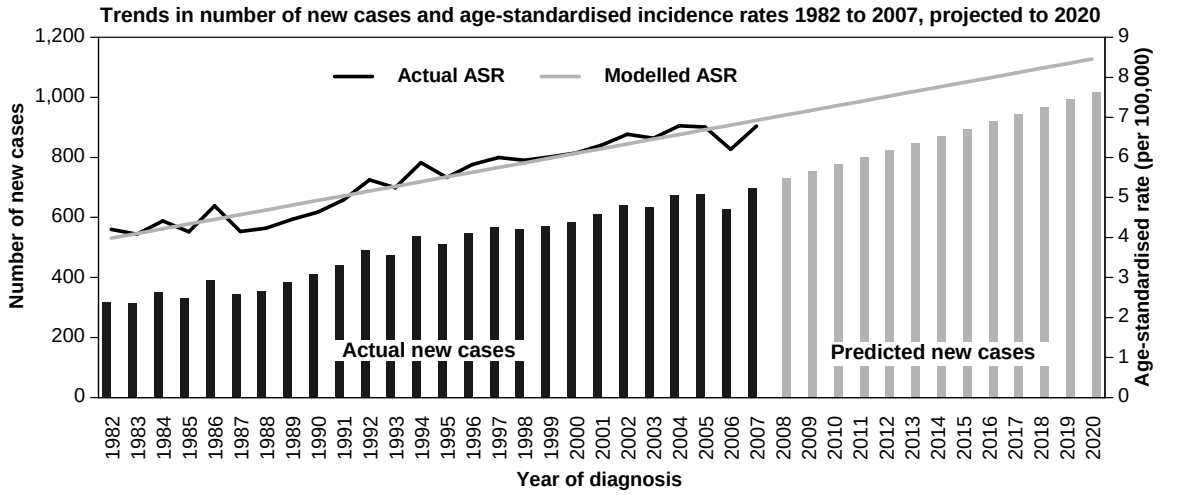
Notes

1. Testicular cancer includes ICD-10 code C62.
2. Projected estimates are based on a linear extrapolation of incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.19a: Testicular cancer (C62), Australia: actual 1982–2007 and projected to 2020

Thyroid cancer (C73)

Thyroid cancer accounted for 1,787 cases or 1.6% of all new cases of cancer in Australia in 2007. Age-standardised incidence rates have risen for both males and females from 1982 to 2007, while mortality rates have declined slightly. Numerous research studies attribute the rising incidence rates to increased medical surveillance and the use of improved diagnostics (IARC 2008; Enewold et al. 2009), specifically ultrasound and increased sampling of specimens by pathologists (Grodski et al. 2008).

The exact cause of thyroid cancer is unknown (Enewold et al. 2011). Ionising radiation exposure, particularly during childhood, is the principle risk factor. Potential sources of radiation exposure include radiation used in diagnostic and therapeutic medicine (Sprague et al. 2008). Thyroid cancer and thyroid diseases in first-degree relatives are also factors associated with an increased risk of developing the disease (Hoang 2010). Iodine deficiency and iodine excess are also associated with thyroid cancer (Dal Maso et al. 2009).

Females are three times more likely than males to develop thyroid cancer, making it the eighth leading cause of cancer in females in Australia. The exact reason for this is inconclusive, but experimental studies outline the strong role oestrogen and other sex hormones play in thyroid cancer progression (Yao et al. 2011). Other factors related to puberty and the effects of pregnancy also may influence the risk of females developing the disease (Horn-Ross et al. 2011).

Males

Joinpoint analysis of age-standardised rates shows thyroid cancer in males increased slightly, but significantly, by about 0.05 cases per 100,000 males per year between 1982 and 1993. After this time, the rate accelerated to increase by about 0.14 cases per 100,000 males per year. Extrapolation of age-specific trends from 1993 to 2007 indicate that rates are expected to continue to increase to 5.7 per 100,000 males in 2020 which, taking into account expected changes to the population structure, will translate to an estimated 775 new cases diagnosed. Increases in rates will be most apparent for those aged 45 to 84 (tables 3.20a and 3.20b, Figure 3.20a).

Females

Joinpoint analysis of age-standardised rates shows thyroid cancer in females increased at a non-significant rate from 1982 to 1989; however, from 1989 to 2000 rates increased significantly at about 0.4 cases per 100,000 females per year. The rate then accelerated to increase by 0.6 cases per 100,000 females per year to 2007.

Extrapolation of age-specific trends from 2000 to 2007 indicate that rates are expected to continue to increase to 19.8 new cases per 100,000 females in 2020 which, taking into account expected changes to the population structure, will translate to an estimated 2,660 new cases diagnosed (Table 3.20c). Unlike males, increases in rates will be most apparent for those aged 45–64, with smaller increases in those aged 25–44 and 65–84 (Table 3.20c, Figure 3.20b).

Table 3.20a: Projected number of new cases and age-standardised rates with 95% prediction intervals, males, 2011–2020: thyroid cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	520	470	565	4.5	4.1	4.9
2012	545	495	595	4.6	4.2	5.1
2013	570	520	625	4.8	4.3	5.2
2014	600	545	655	4.9	4.4	5.3
2015	625	570	685	5.0	4.6	5.5
2016	655	595	715	5.2	4.7	5.6
2017	685	620	745	5.3	4.8	5.8
2018	715	650	780	5.4	4.9	5.9
2019	745	675	810	5.5	5.0	6.0
2020	775	705	845	5.7	5.1	6.2

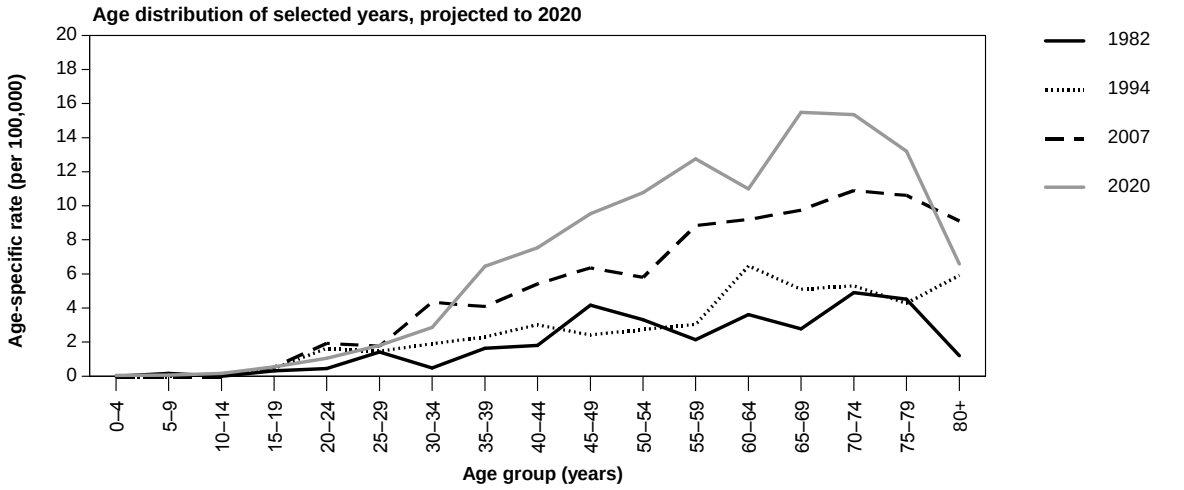
Table 3.20b: Projected number of new cases and age-specific rates, males, 2011–2020: thyroid cancer

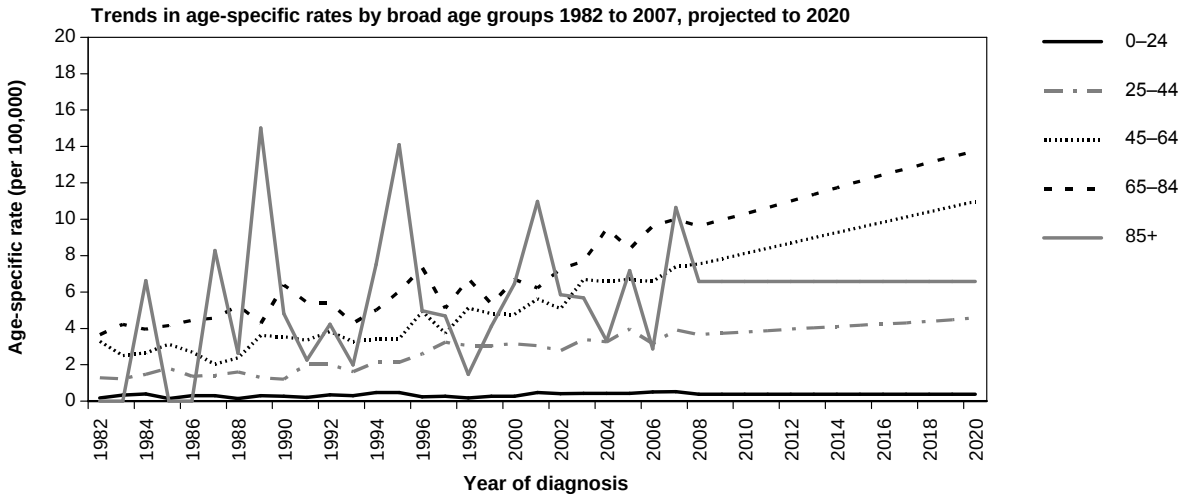
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	15	0.4	120	3.9	235	8.4	135	10.6	10	6.6
2012	15	0.4	125	4.0	245	8.7	150	11.0	10	6.6
2013	15	0.4	130	4.0	255	9.0	160	11.4	10	6.6
2014	15	0.4	135	4.1	265	9.3	170	11.7	10	6.6
2015	15	0.4	140	4.2	280	9.6	180	12.1	10	6.6
2016	15	0.4	140	4.2	290	9.8	195	12.4	10	6.6
2017	15	0.4	145	4.3	305	10.1	205	12.8	15	6.6
2018	15	0.4	150	4.4	315	10.4	220	13.1	15	6.6
2019	15	0.4	155	4.5	325	10.7	230	13.4	15	6.6
2020	15	0.4	160	4.6	340	11.0	245	13.8	15	6.6

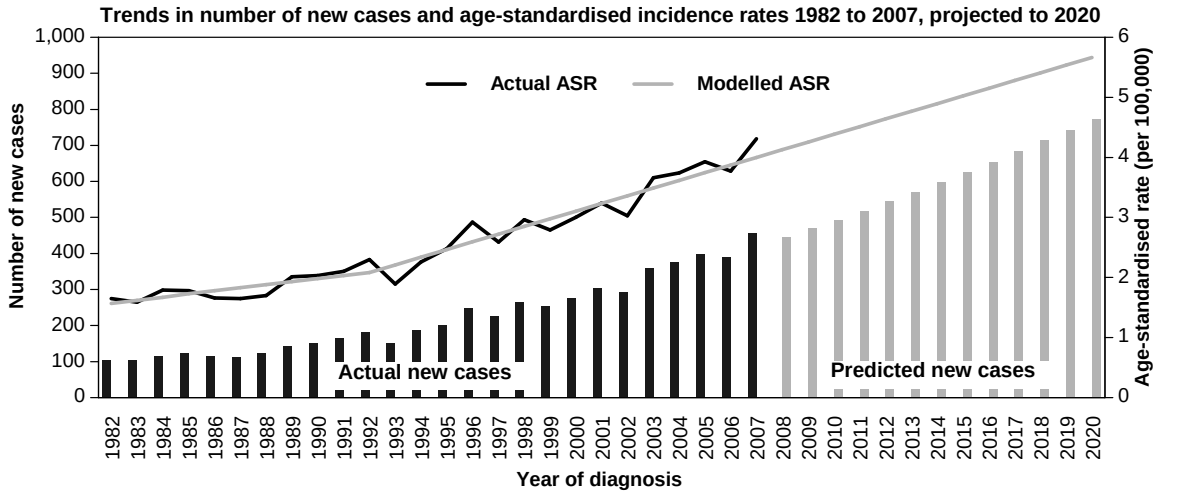
Notes

1. Thyroid cancer includes ICD-10 code C73.
2. Projected estimates are based on incidence data for 1993 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 males.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 males.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.20a: Thyroid cancer (C73), males, Australia: actual 1982–2007 and projected to 2020

Table 3.20c: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: thyroid cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	1,710	1,590	1,830	14.7	13.6	15.7
2012	1,800	1,670	1,940	15.2	14.1	16.3
2013	1,900	1,760	2,050	15.8	14.6	17.0
2014	2,010	1,850	2,160	16.4	15.1	17.6
2015	2,110	1,940	2,280	17.0	15.6	18.3
2016	2,220	2,030	2,400	17.5	16.1	19.0
2017	2,320	2,130	2,520	18.1	16.6	19.6
2018	2,430	2,220	2,640	18.7	17.1	20.3
2019	2,540	2,320	2,770	19.2	17.5	20.9
2020	2,660	2,420	2,900	19.8	18.0	21.6

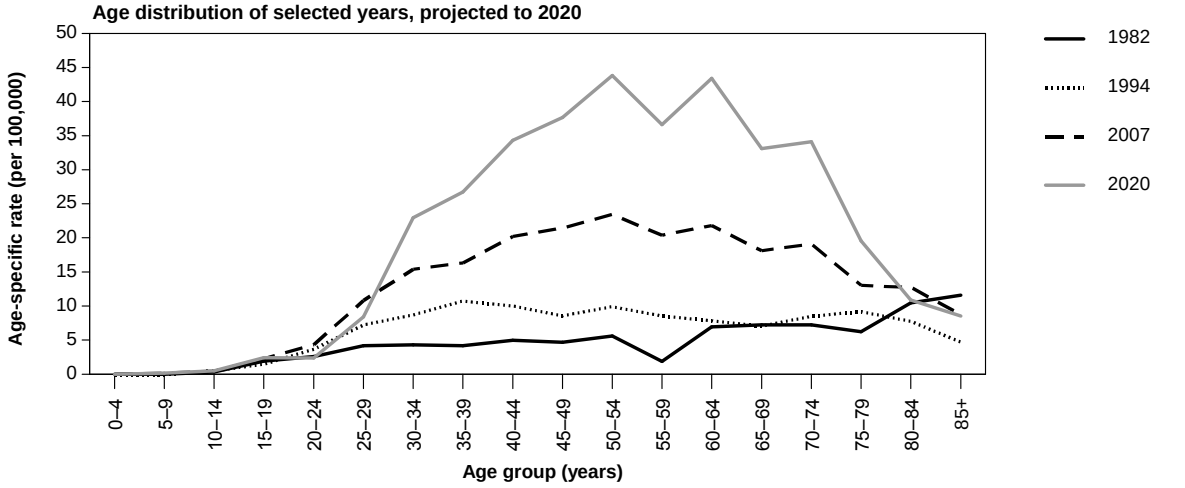
Table 3.20d: Projected number of new cases and age-specific rates, females, 2011–2020: thyroid cancer

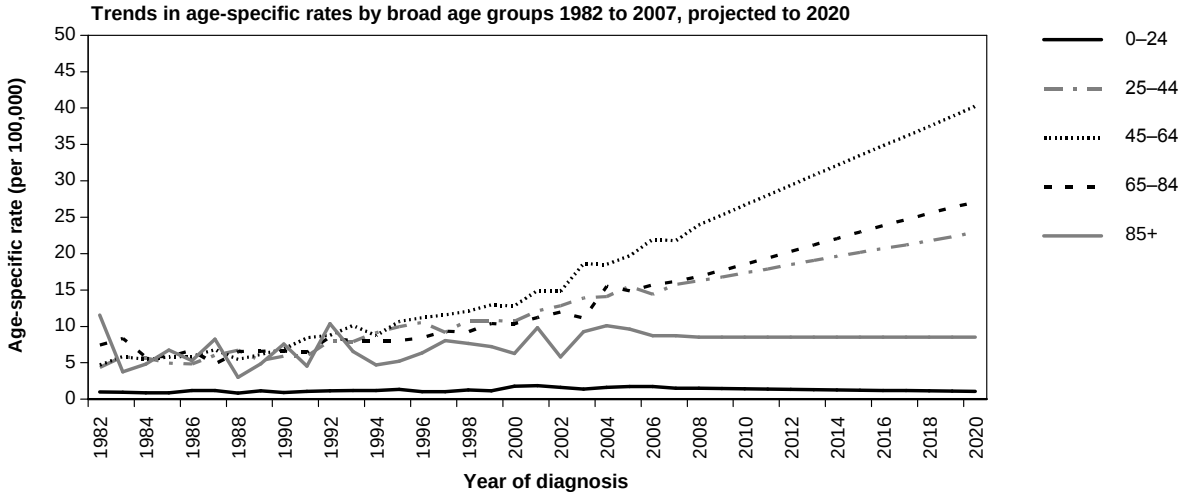
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	50	1.4	560	17.9	800	28.1	275	19.4	25	8.5
2012	50	1.3	590	18.5	845	29.4	300	20.3	25	8.5
2013	45	1.3	615	19.1	895	30.8	325	21.2	25	8.5
2014	45	1.3	640	19.6	945	32.1	350	22.1	25	8.5
2015	45	1.2	665	20.2	1,000	33.5	375	23.0	25	8.5
2016	45	1.2	685	20.7	1,050	34.8	405	23.9	25	8.5
2017	45	1.2	710	21.2	1,110	36.2	430	24.7	30	8.5
2018	45	1.1	735	21.8	1,160	37.5	460	25.5	30	8.5
2019	40	1.1	765	22.3	1,220	38.9	490	26.3	30	8.5
2020	40	1.1	795	22.9	1,270	40.3	525	27.1	30	8.5

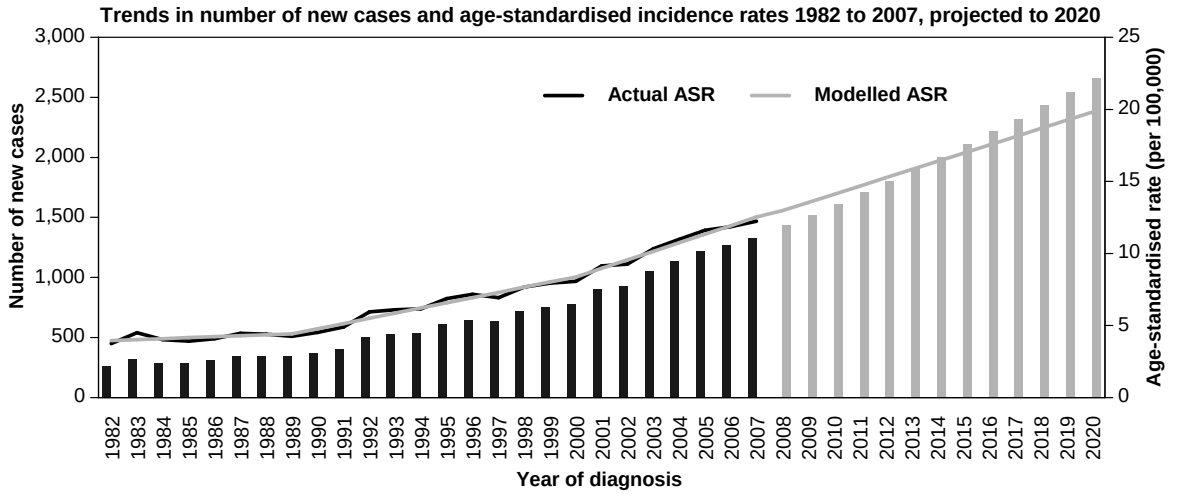
Notes

1. Thyroid cancer includes ICD-10 code C73.
2. Projected estimates are based on incidence data for 1998 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.20b: Thyroid cancer (C73), females, Australia: actual 1982–2007 and projected to 2020

Uterine cancer (C54–C55)

Uterine cancer (which includes endometrial cancer) is the fifth most common cancer diagnosed in Australian women, and the most common invasive gynaecological cancer, with 1,942 new cases diagnosed and 338 deaths in 2007. Uterine cancer is more common in women over the age of 50 than in younger women. There is no effective screening procedure for early detection, and the prognosis is poor for certain subtypes of the cancer, particularly those where the cancer is diagnosed at an advanced clinical stage.

Uterine cancer is an oestrogen-dependent disease. Chronic exposure to oestrogen, without the accompanying balancing effects of progesterone, is considered the major risk factor and may play a causal role in the development of the disease. Risk factors for development of uterine cancer include obesity, reproductive and menstrual history such as early onset of menstruation or late menopause, and oestrogen supplements (ANECS 2011; NCI 2011c).

Since 1982, age-standardised rates for uterine cancer have been increasing at a small but statistically significant rate of 0.1 cases per 100,000 females per year. This has been attributed in part to an increasing prevalence of obesity (ANECS 2011).

Extrapolation of this trend estimates that age-standardised incidence rates will continue to rise from 16.7 new cases diagnosed per 100,000 females in 2011 to 17.6 in 2020. Actual incidence is also expected to increase from an estimated 2,170 new cases in 2011 to 2,830 new cases in 2020 with the expected changes in the population (Table 3.21a).

Analysis of age-specific projections showed that incidence rates of uterine cancer in younger females (aged between 0–24 and 25–44) are expected to remain virtually non-existent, while rates for older females aged 45–64, 65–84 and 85 and over are expected to rise (Table 3.21b, Figure 3.21a).

Table 3.21a: Projected number of new cases and age-standardised rates with 95% prediction intervals, females, 2011–2020: uterine cancer

	Estimated number of new cases			Estimated age-standardised rate		
Year	Cases	Lower 95% PI	Upper 95% PI	Rate	Lower 95% PI	Upper 95% PI
2011	2,170	2,050	2,290	16.7	15.8	17.6
2012	2,240	2,110	2,360	16.8	15.9	17.7
2013	2,310	2,180	2,440	16.9	15.9	17.8
2014	2,380	2,240	2,510	17.0	16.0	17.9
2015	2,450	2,310	2,590	17.1	16.1	18.0
2016	2,520	2,380	2,670	17.2	16.2	18.1
2017	2,600	2,450	2,750	17.3	16.3	18.3
2018	2,670	2,520	2,830	17.4	16.4	18.4
2019	2,750	2,590	2,910	17.5	16.5	18.5
2020	2,830	2,660	2,990	17.6	16.6	18.6

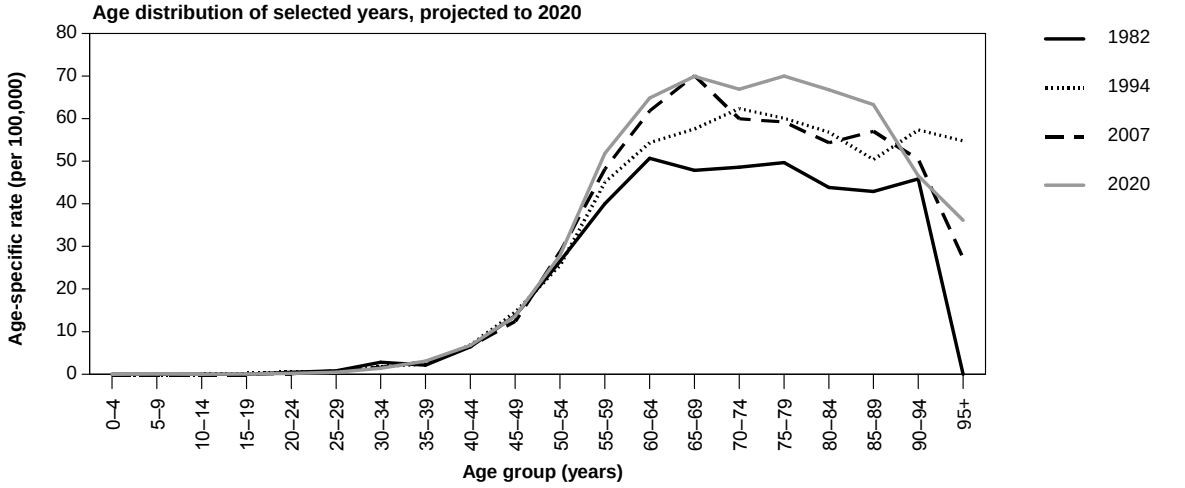
Table 3.21b: Projected number of new cases and age-specific rates, females, 2011–2020: uterine cancer

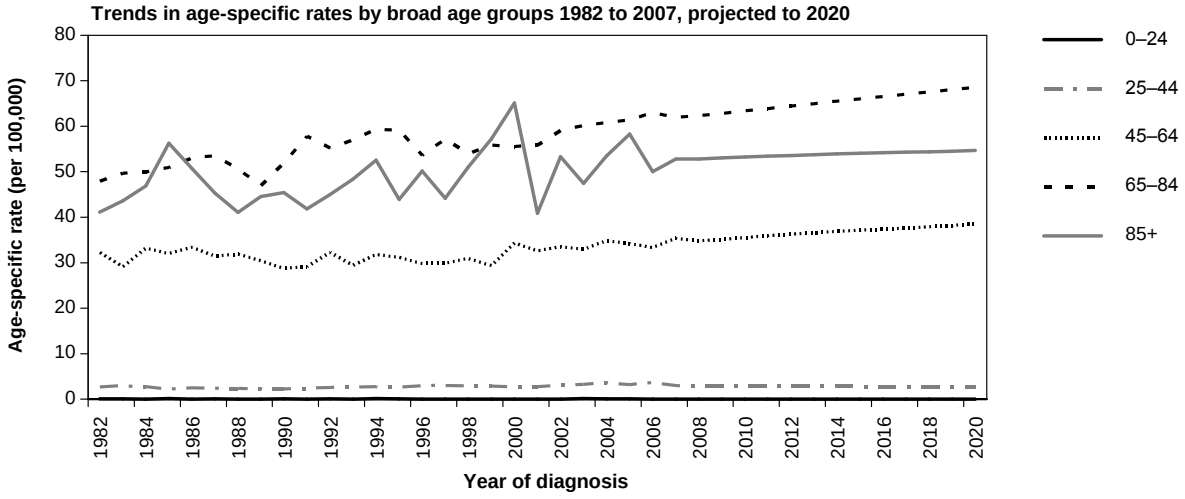
	0–24 years		25–44 years		45–64 years		65–84 years		85+ years	
Year	Count	Rate	Count	Rate	Count	Rate	Count	Rate	Count	Rate
2011	0	0.0	90	2.9	1,030	36.0	905	63.9	145	53.4
2012	0	0.0	95	2.9	1,040	36.3	950	64.5	150	53.6
2013	0	0.0	95	2.9	1,060	36.6	990	65.0	160	53.8
2014	0	0.0	95	2.9	1,090	36.9	1,030	65.5	165	53.9
2015	0	0.0	95	2.9	1,110	37.2	1,080	66.0	170	54.1
2016	0	0.0	95	2.9	1,130	37.4	1,120	66.6	170	54.2
2017	0	0.0	95	2.9	1,160	37.6	1,170	67.0	175	54.3
2018	0	0.0	95	2.8	1,180	37.9	1,220	67.5	180	54.4
2019	0	0.0	95	2.8	1,200	38.2	1,270	68.0	180	54.5
2020	0	0.0	100	2.8	1,220	38.6	1,320	68.5	185	54.7

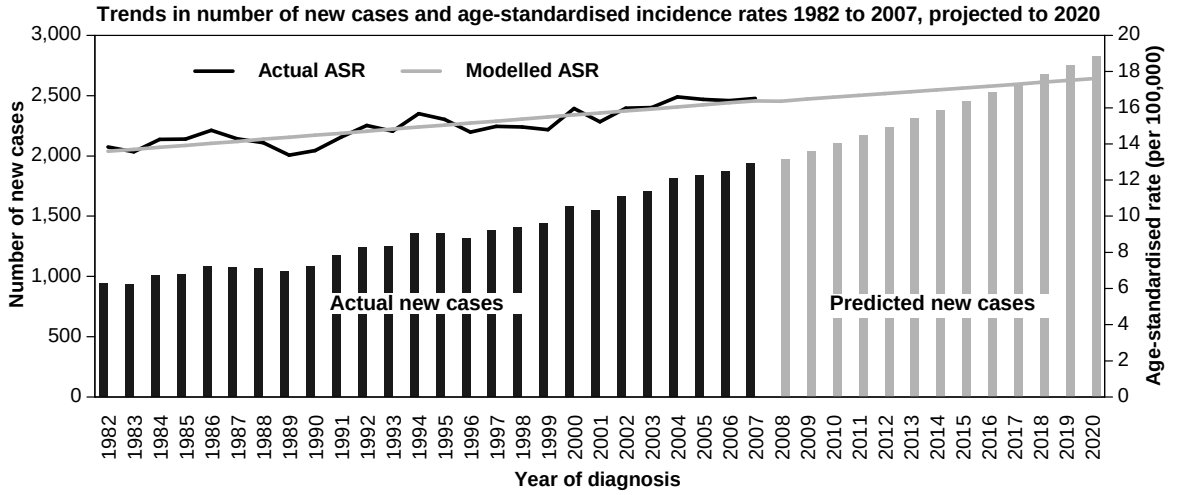
Notes

1. Uterine cancer includes ICD-10 codes C54–C55.
2. Projected estimates are based on incidence data for 1982 to 2007.
3. Counts are rounded to the nearest 10. For counts less than 1,000 estimates are rounded to the nearest 5.
4. Age-standardised rates are standardised to the Australian population as at 30 June 2001 and are expressed per 100,000 females.

Source: Australian Cancer Database (2007).







(a) Rates are expressed per 100,000 females.

(b) Age-standardised rates are age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007).

Figure 3.21a: Uterine cancer (C54–C55), females, Australia: actual 1982–2007 and projected to 2020

Appendix A: Cancer groups and base models

Table A.1: Cancer groups and base models

		Base model			
		Males		Females	
Cancer group	ICD codes	Observation window	Age groups	Observation window	Age groups
All cancers combined	C00–C96, D45, D46, D47.1, D47.3	1994–2007 (excluding prostate cancer) + 1982–2010 (prostate cancer)	Standard 5-year age groups to 95+	1995–2007	Standard 5-year age groups to 95+
Bladder	C67	1982–2007	Standard 5-year age groups to 95+	1982–2007	Standard 5-year age groups to 95+
Bowel	C18–C20	1996–2007	Standard 5-year age groups to 95+	1982–2007	Standard 5-year age groups to 95+
Brain	C71	1982–2007	Standard 5-year age groups to 90+	1982–2007	Standard 5-year age groups to 90+
Breast	C50	n.a.	n.a.	1995–2007	Standard 5-year age groups to 95+
Cervical	C53	n.a.	n.a.	2001–2007	Standard 5-year age groups to 90+
Hodgkin lymphoma	C81	1993–2007	Standard 5-year age groups to 75+.	1982–2007	Standard 5-year age groups to 85+.
Kidney	C64	1982–2007	Standard 5-year age groups to 90+	1992–2007	Standard 5-year age groups to 85+
Liver	C22	1982–2007	Standard 5-year age groups to 90+	1996–2007	Standard 5-year age groups to 90+
Lung	C33–C34	1982–2007	Standard 5-year age groups to 95+	1990–2007	Standard 5-year age groups to 95+
Melanoma of the skin	C43	1988–2007	Standard 5-year age groups to 95+	1987–2007	Standard 5-year age groups to 95+
Mesothelioma	C45	1994–2007	Standard 5-year age groups to 85+	1982–2007	Standard 5-year age groups to 85+
Non-Hodgkin lymphoma	C82–C85	1992–2007	Standard 5-year age groups to 85+	1997–2007	Standard 5-year age groups to 85+

(continued)

Table A.1 (continued): Cancer groups and base models

		Base model			
		Males		Females	
Cancer group	ICD codes	Observation window	Age groups	Observation window	Age groups
Oesophageal	C15	1994–2007	Standard 5-year age groups to 85+	1994–2007	Standard 5-year age groups to 90+
Ovarian	C56	n.a.	n.a.	1992–2007	Standard 5-year age groups to 95+
Pancreatic	C25	1982–2007	0–39, standard 5-year age groups to 90+	1982–2007	0–49, standard 5-year age groups to 90+
Prostate	C61	1982–2010[(a)]	Standard 5-year age groups to 95+	n.a.	n.a.
Stomach	C16	1982–2007	Standard 5-year age groups to 95+	1988–2007	Standard 5-year age groups to 95+
Testicular	C62	1982–2007	Standard 5-year age groups to 80+	n.a.	n.a.
Thyroid	C73	1993–2007	Standard 5-year age groups to 80+	2000–2007	Standard 5-year age groups to 85+
Uterine	C54–C55	n.a.	n.a.	1982–2007	Standard 5-year age groups to 95+

(a) See Appendix J of Cancer in Australia: An overview 2010 for a full explanation of imputed values for 2008–2010.

Appendix B: Statistical methods

Crude rates

A crude rate is defined as the number of events over a specified period of time (for example, a year) divided by the total population. For example, a crude cancer incidence rate is defined as the number of new cases of cancer diagnosed in a year divided by the population at risk. This is usually expressed as the number of new cases per 100,000 population.

Crude rates should not be used to compare cancer incidence in different populations or over time as the underlying populations may have different age structures and the risk of developing cancer varies with age.

As this report is based on analysis of historical trends over time, projected into the future, age-specific and age-standardised rates are used to describe the underlying changes in trend. Crude rates, however, are important to provide information about actual disease burden and are provided in the online supplementary tables for further information.

Age-specific rates

Age-specific rates provide information on the incidence of cancer in an age group relative to the total number of people at risk of cancer in the same age group. It is calculated by dividing the number of events occurring in each specified age group by the corresponding 'at risk' population in the same age group and then multiplying the result by a constant (usually 100,000) to derive the rate.

Age-standardised rates

Age-standardised rates adjust for age to facilitate comparisons between populations that have different age structures. This standardisation process effectively removes the influence of age structure on the summary rate.

There are two methods commonly used to adjust for age: direct and indirect standardisation. In this report, the direct standardisation approach presented by Jensen and colleagues (1991) is used. To age-standardise using the direct method, the first step is to obtain population numbers and numbers of cases in age ranges—typically 5-year age ranges. The next step is to multiply the age-specific population numbers for the standard population (in this case the Australian population as at 30 June 2001) by the age-specific incidence rates for the population of interest. The next step is to sum across the age groups and divide this sum by the total of the standard population to give an age-standardised rate for the population of interest. Finally, this can be converted to a rate per 100,000 population.

Prediction intervals

No projections can be 100% accurate. Prediction intervals provide a quantification of the level of uncertainly around the projected figure. The prediction intervals in this report account for both the variance and covariance of the model parameters (reflected by conventional least squares confidence intervals) and the variance of the model residuals. The latter is an adjustment for the serial correlation experienced in the cancer data, due to repeated observation of a population that is static (apart from rates of birth, death and

migration that are negligible in this context). Even though the population can be regarded as fixed for projection purposes, the event of cancer diagnosis (by site) has low probability. Consequently, this adjustment for serial correlation is a conservative one. That is, our 95% prediction intervals will, over all sites considered, contain the true rate with a frequency greater than 95%.

These prediction intervals can be used in conjunction with the confidence intervals associated with other published rates to determine whether observed differences are statistically significant or simply due to chance variation. Intervals that do not overlap indicate a rate difference greater than that which could be explained by chance and should be regarded as statistically significant. Note, however, that overlapping confidence intervals do not necessarily mean that the difference between two rates is definitely due to chance. Instead, an overlapping confidence interval represents a difference in rates that is too small to allow differentiation between a real difference and one that is due to chance variation. It can, therefore, only be stated that no statistically significant differences were found, and not that no differences exist. The approximate comparisons presented might understate the statistical significance of some differences, but they are sufficiently accurate for the purposes of this report.

A projected age-standardised rate is the weighted average of mutually independent predicted age-specific rates. Hence, its variance is readily obtained as a linear combination of the age specific variances. In addition, it has the same interpretation with respect to the true value as the age specific rates.

Joinpoint analysis

Joinpoint statistical methodology (Kim et al. 2000) was used to examine temporal trends in cancer incidence in this report. A joinpoint regression model describes changing trends over successive segments of time and the amount of change within each segment. Trends are characterised by joined linear segments; a joinpoint is created where two segments meet, thus representing a statistically significant change in the trend. It is important to note that while the joinpoint analysis identifies a particular point in time that a trend changes, in reality changes in trend are not usually abrupt and may often be more gradual, depending on the underlying cause of the change in trend.

The software used to perform joinpoint analysis was Joinpoint Version 3.4.3, developed by the Statistical Research and Applications Branch of the National Cancer Institute in the United States of America (NCI 2011b). This software has been used frequently to examine cancer trends both internationally (Cancer Care Ontario 2006; Yang et al. 2009), and within Australia (Baade & Coory 2005; Tracey et al. 2009; AIHW 2011a).

The joinpoint software takes trend data in the form of age-standardised cancer rates and fits the simplest joinpoint model possible, where there is a minimum number of segments necessary to characterise a trend. The software begins with a model with zero joinpoints (that is, no changes in trend) and incrementally tests whether more joinpoints are statistically significant. The number of significant joinpoints is identified through performing several permutation tests and each *p*-value is found using Monte Carlo methods. The tests can be extended to datasets with non-constant variance to handle rates with Poisson variation and auto-correlated errors.

For this report, a number of parameters were also specified based on preliminary examination of the data:

- **Model specification**: to assess the most appropriate trends, both linear and logarithmic transformations were applied to joinpoint models.
- **Heteroscedastic errors**: preliminary testing showed little variation in results between assuming constant errors and inputting standard errors of the age-standardised rates. As a result, the constant error assumption was chosen for all models.
- **Number of joinpoints and observations**: the default values of 0 and 4 were selected for the minimum and maximum number of joinpoints. To ensure a minimum of 6 years of data in the most recent trend, values for the minimum number of observations from a joinpoint to either end of the data was set to 6 and the minimum number of observations between two joinpoints was set to 5.
- **Method**: the permutation method was used to determine the optimal number of joinpoints, which permuted the residuals from the null model and added them back on to fitted values. This method produced similar results to the alternative Bayesian Information Criterion, but was selected as it tended to be more conservative. The overall significance level for the permutation tests was 0.05.
- **Model selection method**: the grid search method (Lerman 1980) was used to fit the joinpoint model and compute point estimates of the coefficients. It created a 'grid' of all possible locations for joinpoints and tested the sum of squares at each one to find the best fit.

Projections model

This section summarises the mathematical details associated with the projection methodology such as the functional form of the predictive model and the derivation of variance formulae for the predicted incidence rates.

Model formulation

For each of the age groups 0–4, 5–9, . . ., 90–94, 95+ incidence prediction was carried out independently according to the procedure outlined below.

Base model

For future year t_0, the predicted incidence count of each age group, $\hat{I}(t_0)$, is given by

$$\hat{I}(t_0) = N(t_0)\hat{R}(t_0)$$

where

$$\hat{R}(t_0) = \text{projected incidence rate for year } t_0$$
$$N(t_0) = \text{projected population count for year } t_0$$

The projected population counts, $N(t_0)$, provided by the Australian Bureau of Statistics, are derived using a deterministic model. Consequently, they do not have an associated variance estimate; for this reason they are regarded as known 'constants'. This section describes the modelling technique used to calculate both $\hat{R}(t_0)$ and an appropriate measure of its accuracy.

Using the observed incidence rates $\{R(t): t = 1, 2, \cdots, n\}$ the ordinary least squares model is estimated as

$$R(t) = \alpha_0 + \alpha_1 t + \varepsilon_t \qquad (1)$$

where ε_t are assumed to be independent and identically distributed normal random variables with mean zero and unknown (but constant) variance σ^2.

The variance and covariance of the regression coefficients and the observed residual variance, $Var(\varepsilon_t)$, were all obtained in SAS using the least squares regression procedure PROC REG; these terms are denoted $Var(\hat{\alpha}_0), Var(\hat{\alpha}_1), Cov(\hat{\alpha}_0, \hat{\alpha}_1)$ and $\hat{\sigma}^2$, respectively.

Since incidence rates pertaining to distinct time points are repeated observations of essentially the same population (apart from births, deaths and migration) then the residuals of our model, ε_t, are correlated (and so not independent as assumed in (1)). However, according to the theory of Zeger and Liang (1992), these predictions are asymptotically unbiased for large sample size, n, but their variance-covariance matrix cannot be obtained using the conventional (least squares) formula. Instead, these authors recommend a variance estimator that is a (quadratic) function of the observed residuals; the methodology used in this report has taken an analogous, but computationally simpler approach by presenting prediction (rather than confidence) intervals. That is, the intervals displayed are a function of the observed residual variance and the inaccuracy associated with trend estimation; the latter is the only dispersion measure inherent in confidence interval calculation. The relevant formulae used in deriving these prediction intervals will be given throughout this section.

Model projection

Using model (1) the significance of the trend, $\hat{\alpha}_1$, is determined according to a two-sided hypothesis test with the significance level set at 5%. When the trend was significantly greater than zero we forecast using model (1), that is,

$$\hat{R}(t_0) = \hat{\alpha}_0 + \hat{\alpha}_1 t$$

and

$$Var\left(\hat{R}(t_0)\right) = Var(\hat{\alpha}_0) + 2t_0 Cov(\hat{\alpha}_0, \hat{\alpha}_1) + t^2 Var(\hat{\alpha}_1) + \hat{\sigma}^2 \qquad (2)$$

When the trend was not significantly different from zero we predict, at all future time points, the mean of the observed incidence rates. In this case we have

$$\hat{R}(t_0) = \hat{\alpha}_0$$

and

$$Var\left(\hat{R}(t_0)\right) = Var\left(\overline{R}\right) + \hat{\sigma}^2$$
$$= \left(\frac{1}{n} + 1\right)\hat{\sigma}^2$$
$$= \frac{n+1}{n} \times \text{sample variance of } \{R(t) : t = 1, 2, \cdots, n\}$$

When the trend is significantly less than zero, to guarantee projecting strictly positive rates, we apply the logarithmic link function. That is, we estimate the model

$$\log\left(R(t)\right) = \alpha_0 + \alpha_1 t + \varepsilon_t$$

To avoid undefined calculations where age-sex-cancer incidence rates are zero, an adjustment factor of 10^{-9} x the population is added to each incidence count in an age-sex group where there is at least one observed age-sex-cancer incidence count of zero.

In this case the predicted rate is

$$\hat{R}(t_0) = \exp\left(\hat{\alpha}_0 + \hat{\alpha}_1 t_0\right) \qquad (3)$$

Model (3) can be formulated as follows

$$\hat{R}(t_0) = \exp\left(\hat{\alpha}_0\right) \times \exp\left(\hat{\alpha}_1 \times t_0\right)$$
$$= A\beta^{t_0}$$

Projections are made according to a power law with common ratio $\hat{\beta}$ constrained (strictly) between zero and one.

For this case the variance of the predicted value is related to that for the increasing trend model, given in equation (2), using the so-called delta method. That is, model (3) can be written

$$R(t_0) = \exp\left(\theta(t_0)\right)$$

where $\theta(t_0) = \alpha_0 + \alpha_1 t_0$.

Using a first order Taylor approximation to $R(t_0)$ in variable $\theta(t_0)$ this gives

$$\begin{aligned}\hat{R}(t_0) - R(t_0) &= \frac{\partial R}{\partial \vartheta}\left(\hat{\theta}(t_0) - \theta(t_0)\right) \\ &= R(t_0)\left(\hat{\theta}(t_0) - \theta(t_0)\right) \\ &\cong \hat{R}(t_0)\left(\hat{\theta}(t_0) - \theta(t_0)\right)\end{aligned}$$

Hence, the variance of the predicted rate is given by

$$\begin{aligned}Var\left(\hat{R}(t_0)\right) &= \hat{R}^2(t_0)Var\left(\hat{\theta}(t_0)\right) \\ &= \hat{R}^2(t_0)\left\{Var(\hat{\alpha}_0) + 2t_0 Cov(\hat{\alpha}_0, \hat{\alpha}_1) + t_0^2 Var(\hat{\alpha}_0) + \hat{\sigma}^2\right\}\end{aligned}$$

Prostate cancer projections

To assess the length of the steep upward trend in prostate cancer incidence observed from 2002 to 2007, we imputed incidence counts, in age groups 0-44, 45-49, 50-54, …, 90-94 and 95+, for the years 2008, 2009 and 2010. These imputed values were obtained by exploiting the strong positive correlation between the annual count of PSA tests derived from MBS data, $A(t)$, and prostate incidence count, $I(t)$, for years t=2002, 2003,…,2007. That is, we have imputed according to the ordinary least squares model

$$I_t = \beta_0 + \beta_1 A_t + \eta_t \qquad (4)$$

Since the finest level of age aggregation at which PSA test counts are observable is the 10-year age group (45–54, 55–64, 65–74, 75–84 and 85+) the imputed values given by (4) were subsequently distributed over the two constituent 5-year age groups. For example, a count imputed for age group 45-54 was distributed between the *younger* and *older* finer categories 45–49 and 50–54. This allocation was also carried out according to an ordinary least squares regression model, estimated using observed cancer incidence counts from the 6 years from 2002 to 2007. In this case the model was

$$\frac{I_{\text{younger}}(t)}{I_{\text{broad}}(t)} = \gamma_0 + \gamma_1 t + \tau_t$$

Model diagnostics

This subsection is intended to provide an indication of how well the models formulated above fit the observed incidence rates. A diagnostic that examines both goodness-of-fit and the appropriateness of standard error estimation is the proportion of data points in the observation window used for model estimation contained within the 95% prediction interval.

By way of example, graphs are displayed for all cancers combined by gender, excluding the highly volatile prostate cancer for males (figures B.1 and B.2), and lung cancer (figures B.3 and B.4) showing:

- the observed incidence rates (per 100,000 individuals) for the years 1982 to 2007,
- fitted values from 1982 to 2020 and
- the 95% prediction intervals.

Due to space constraints, for each of these cancer type by gender cross-classifications, only one example of each of the following is displayed: a 5-year age category with significant increasing trend, a 5-year age category with significant decreasing trend and a 5-year age category where the trend is not significantly different from zero.

Females: all cancers combined

Figure B.1 shows examples of increasing, decreasing and constant trends for age categories 40–44, 20–24 and 70–74, respectively for all cancers in females combined. In this case, the predictive models were estimated using data observed from 1995 to 2007, and in all three graphs the prediction intervals have perfect coverage rates.

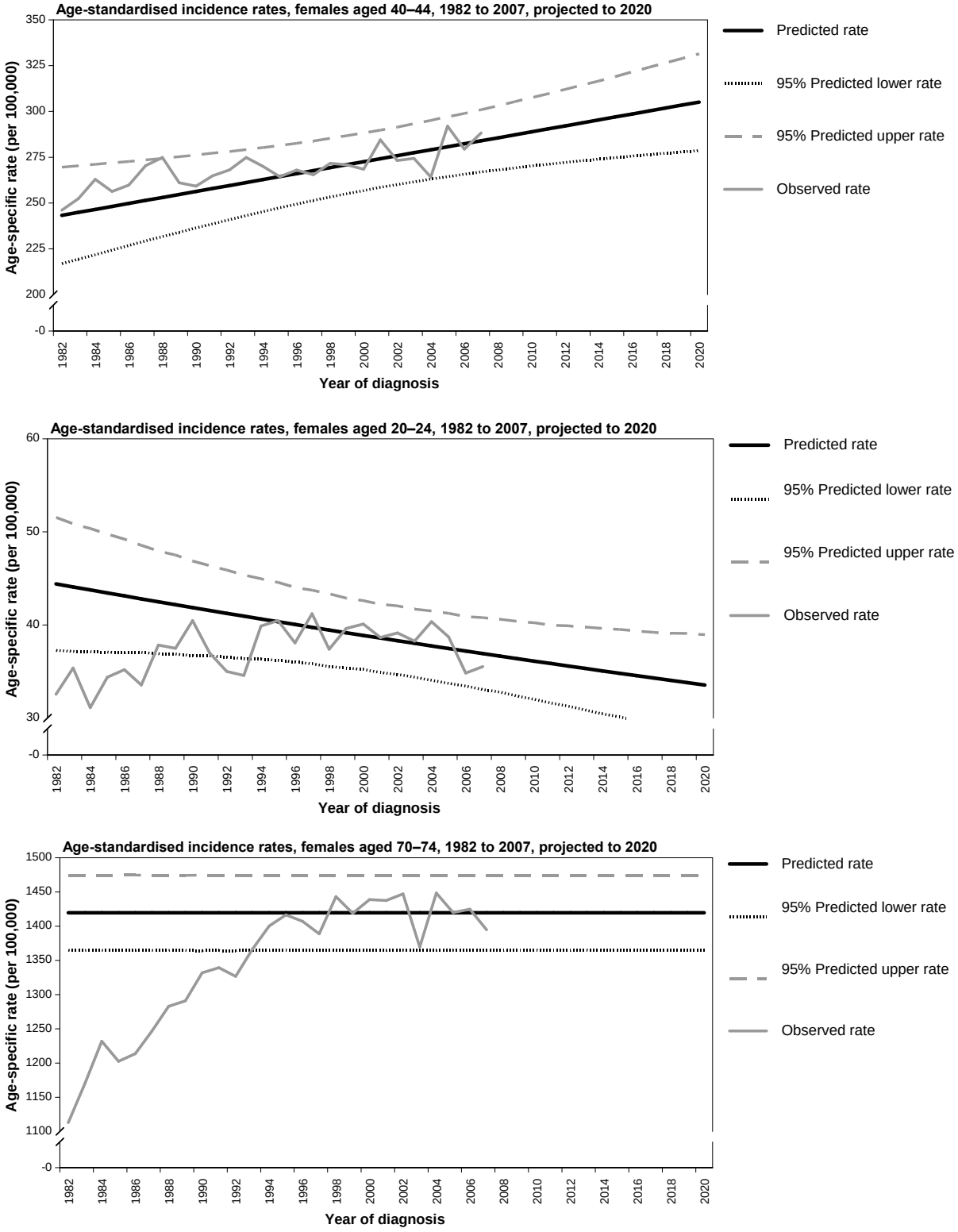
Males: all cancers combined (excluding prostate cancer),

For all cancers (excluding prostate cancer) in males combined, Figure B.2 displays typical models with increasing, decreasing and constant trend in respective age categories 80–84, 70–74 and 75–79. These models were estimated from the 14 observations from 1994 to 2007; in all three plots the prediction intervals contain 100% of data points in the observation window.

Lung cancer

Figure B.3 displays models for lung cancer males in age categories 95 and over (significant increasing trend), 75–79 (significant decreasing trend) and 80–84 (non-significant trend), respectively. In this case the predictive model was estimated using all 26 data points from 1982 to 2007; the number of points contained by the prediction intervals are 25 (96.2%), 26 (100%) and 24 (92.3%), respectively.

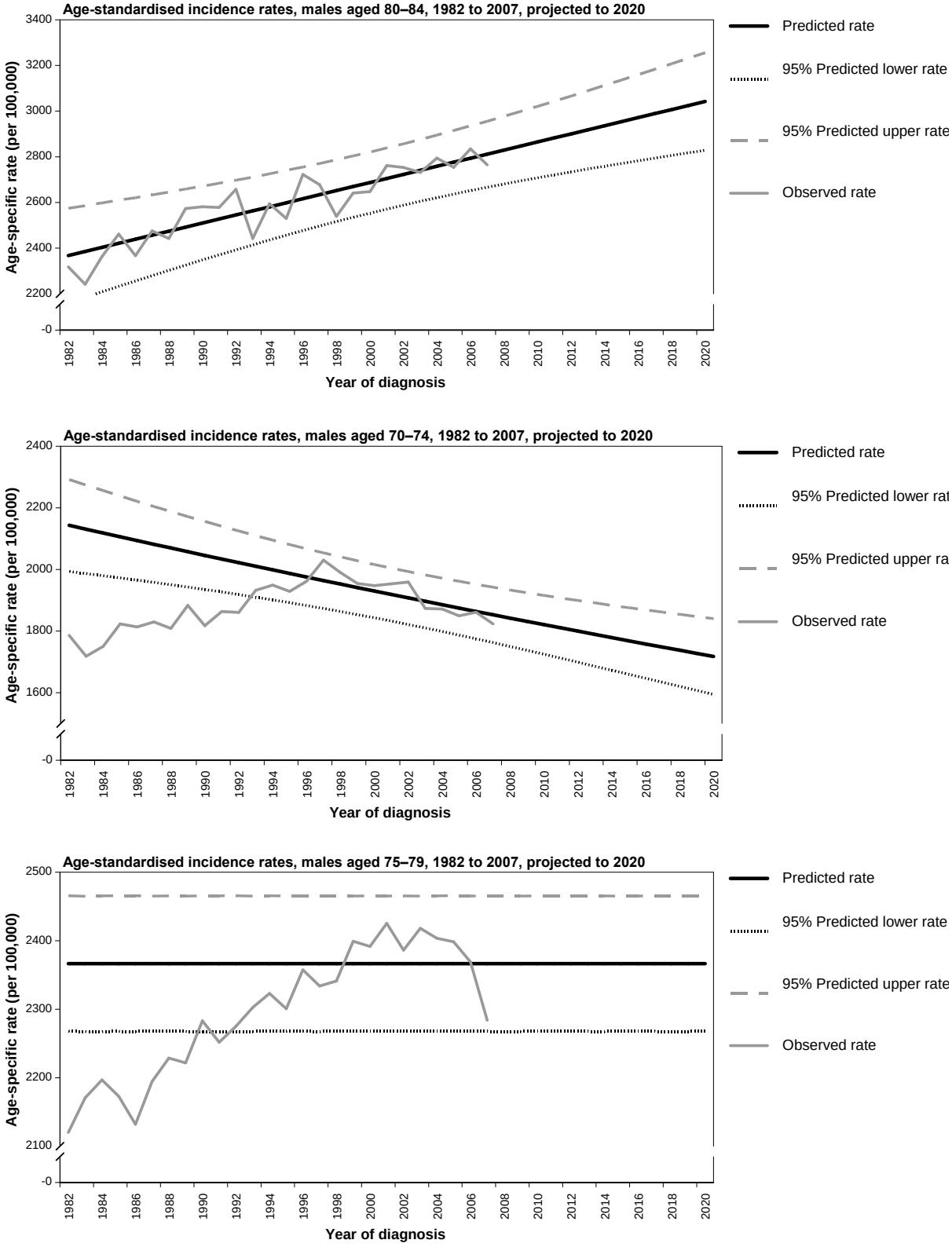
For females the projection window was 1990 to 2007 (18 data points). In all 5-year age categories, the trend over this window is either increasing or constant. Figure B.4 displays model diagnostics for females aged 55–59 and 60–64, respectively; in both graphs we see that the 95% prediction intervals have a 100% coverage rate.



Note: Rates are expressed per 100,000 population and age-standardised rates age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007)

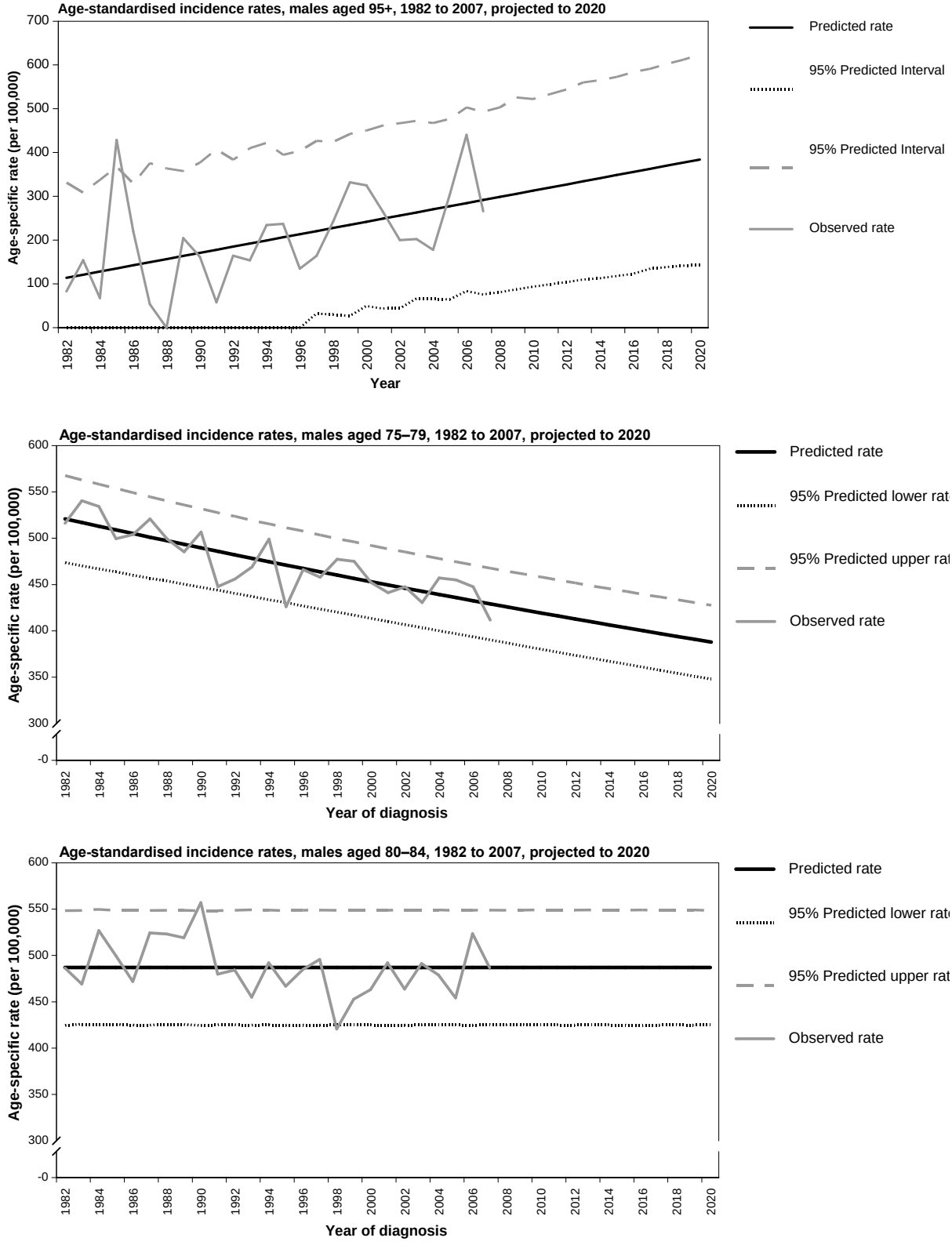
Figure B.1: Model diagnostics: all cancers combined, females, actual 1982–2007 and projected to 2020



Note: Rates are expressed per 100,000 population and age-standardised rates age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007)

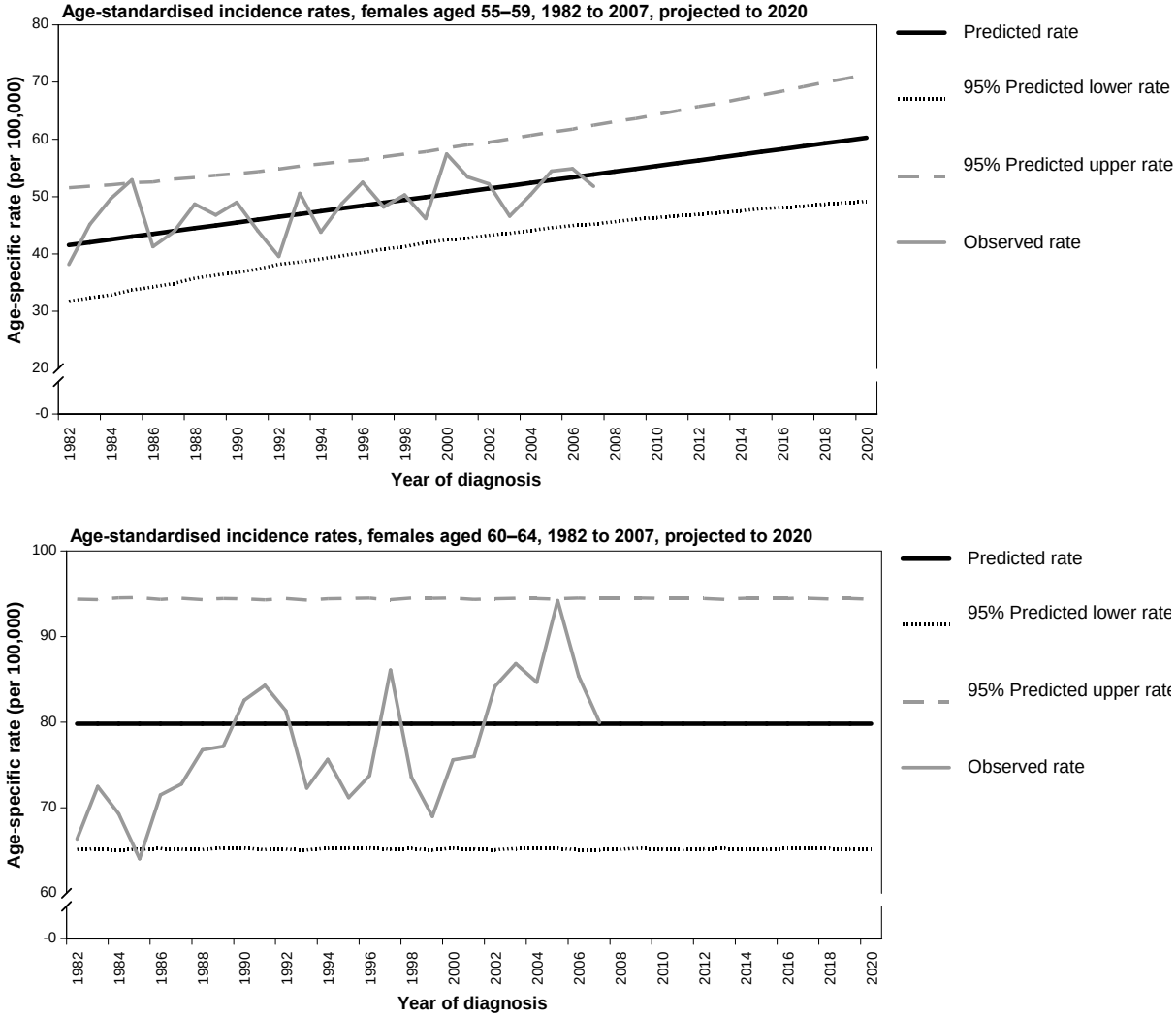
Figure B.2: Model diagnostics: all cancers combined (excluding prostate cancer), males, actual 1982–2007 and projected to 2020



Note: Rates are expressed per 100,000 population and age-standardised rates age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007)

Figure B.3: Model diagnostics: Lung cancer, males, actual 1982–2007 and projected to 2020



(a) Rates are expressed per 100,000 population

(b) Age-standardised rates age-standardised to the Australian population as at 30 June 2001.

Source: Australian Cancer Database (2007)

Figure B.4: Model diagnostics: lung cancer, females, actual 1982–2007 and projected to 2020

Impact of autocorrelation on modelling and projection

As mentioned in the section on model formulation, the data observed are serially correlated. Consequently, preliminary analyses included an assessment of time series methods as an alternative to the chosen regression methodology.

An empirical comparison of predicted rates obtained by the ordinary least squares (OLS) regression technique used in this report and those obtained from an autoregressive moving average (ARMA) model was undertaken using the five-year age-specific incidence of lung cancer (by gender), all female cancers combined and all male cancers (excluding prostate cancer) combined. Since there is (potentially) a linear trend in the observed rates, the ARMA model to their first order difference, $\{R_t - R_{t-1} : t = 2, 3, \cdots, n\}$ was fitted.

Significance was tested using the following Student's *t* statistic, *T*, that is approximately distributed with $n-2$ degrees of freedom.

$$T = \frac{\hat{R}_A - \hat{R}_O}{\sqrt{Var\left(\hat{R}_A\right) + Var\left(\hat{R}_O\right) - 2 * Cov\left(\hat{R}_A, \hat{R}_O\right)}}$$
$$= \frac{\hat{R}_A - \hat{R}_G}{\sqrt{Var\left(\hat{R}_A\right) + Var\left(\hat{R}_O\right) - 2 * SE\left(\hat{R}_A\right) * SE\left(\hat{R}_O\right) * Corr\left(\hat{R}_A, \hat{R}_O\right)}}$$

where $\hat{R}_A$ and $\hat{R}_O$ are the predicted values of the ARMA and OLS techniques, respectively,

These two predictors have a strong positive correlation, that is, $Corr\left(\hat{R}_A, \hat{R}_O\right)$ is close to one. Therefore,

$$T \cong \frac{\hat{R}_A - \hat{R}_O}{\sqrt{\left(SE\left(\hat{R}_A\right) - SE\left(\hat{R}_O\right)\right)^2}}$$
$$= \frac{\hat{R}_A - \hat{R}_O}{\left|SE\left(\hat{R}_A\right) - SE\left(\hat{R}_O\right)\right|} \qquad (5)$$

As expected, discrepancies between the two methods were largest in 2020 and, for this reason, comparison focuses on this particular year. For all female cancers combined (Table B.1), the absolute value of *T* is less than two in all age categories except in the age group 10–14. In this age group *T* takes value 2.502, which implies a *p*-value of 0.0294 (since there are 13 observations, taken from 1995 to 2007, then *T* is distributed with 11 degrees of freedom). In view of the (slight) positive bias of *T* (compared with a random variable with exact *t*-distribution) and the profound non-significance (of difference) in the other 19 age categories, this single apparent significant difference was not considered cause for concern. In the other three site-by-gender classifications considered, the results (shown in tables B.2, B.3 and B.4) provided even more compelling evidence in favour of inferential equivalence with the absolute value of the test statistic bounded by 1.5.

Table B.1: Difference in the ARMA and OLS predicted age specific rate, 2020, all female cancers; together with the associated Student's *T* and *p*-values.

Age category (years)	ARMA prediction	OLS prediction	Difference in predictions	*T*	*p*-value
0–4	23.4 (12.2)	19.5 (1.8)	3.9 (10.4)	0.38	0.71
5–9	7.1 (9.0)	9.2 (1.6)	-2.1 (7.5)	0.28	0.79
10–14	7.4 (2.9)	11.7 (1.2)	4.3 (1.7)	2.50	0.03
15–19	13.5 (10.5)	22.3 (2.1)	-8.8 (8.4)	1.05	0.32
20–24	32.5 (9.2)	33.6 (2.5)	-1.1 (6.7)	0.16	0.88
25–29	62.7 (20.2)	66.0 (3.5)	-3.4 (16.6)	0.20	0.84
30–34	104.9 (22.1)	111,1 (5.2)	-6.3 (16.8)	0.37	0.72
35–39	161.7 (18.7)	172.1 (4.4)	-10.5 (14.3)	0.73	0.48
40–44	312.2 (48.2)	305.1 (12.1)	7.1 (36.0)	0.20	0.85
45–49	446.2 (55.7)	415.9 (12.2)	30.2 (43.5)	0.69	0.50
50–54	519.9 (61.5)	519.0 (16.2)	1.0 (45.3)	0.02	0.98
55–59	745.7 (86.8)	760.8 (18.3)	15.1 (68.4)	0.22	0.83
60–64	973.8 (100.6)	1041.1 (40.7)	-67.3 (59.9)	1.12	0.28
65–69	1361.5 (87.6)	1341.6 (26.3)	19.9 (61.3)	0.32	0.75
70–74	1380.4 (167.8)	1419.6 (25.2)	-39.2 (142.6)	0.27	0.79
75–79	1683.7 (157.3)	1685.5 (35.4)	-1.8 (121.9)	0.01	0.99
80–84	1994.9 (118.7)	2063.5 (42.8)	-68.3 (75.9)	0.90	0.39
85–89	2025.0 (210.7)	2196.3 (71.8)	-171..3 (138.9)	1.23	0.24
90–94	2180.0 (386.7)	2319.9 (106.1)	-139.9 (280.6)	0.50	0.63
95+	2100.8 (707.6)	2219.0 (193.1)	-118.2 (514.3)	0.23	0.82

Table B.2: Difference in the ARMA and OLS predicted age specific rate, 2020, all male cancers excluding prostate; together with the associated Student's *T* and *p*-values.

Age category (years)	ARMA prediction	OLS prediction	Difference in predictions	*T*	*p*-value
0–4	24.6 (10.8)	22.0 (1.9)	2.6 (9.0)	0.28	0.78
5–9	8.8 (6.6)	11.7 (1.3)	2.9 (5.3)	0.55	0.59
10–14	14.6 (6.5)	12.5 (1.1)	2.1 (5.4)	0.39	0.70
15–19	18.8 (12.4)	26.3 (2.0)	-7.6 (10.3)	0.73	0.48
20–24	35.8 (11.6)	39.6 (2.2)	-3.8 (9.40)	0.40	0.69
25–29	42.7 (19.4)	49.6 (5.1)	-6.9 (14.3)	0.48	0.64
30–34	76.4 (15.4)	81.7 (2.3)	-3.3 (13.1)	0.25	0.81
35–39	102.5 (12.3)	110.1 (2.5)	-7.6 (9.8)	0.77	0.45
40–44	152.4 (33.3)	158.9 (5.0)	-6.6 (28.3)	0.23	0.82
45–49	267.2 (27.5)	248.5 (5.8)	18.7 (21.8)	0.86	0.41
50–54	388.7 (41.8)	377.3 (12.3)	11.3 (29.5)	0.38	0.71
55–59	594.6 (46.4)	576.1 (12.0)	18.5 (34.5)	0.54	0.60
60–64	790.9 (126.7)	822.6 (32.1)	-31.7 (94.6)	0.34	0.74
65–69	1303.5 (72.8)	1262.5 (29.3)	41.0 (43.5)	0.94	0.36
70–74	1709.2 (148.1)	1717.8 57.0	-8.6 (91.1)	0.09	0.93
75–79	2265.7 (157.1)	2366.6 (45.8)	-100.9 (111.3)	0.91	0.38
80–84	3019.1 (347.9)	3042.4 (99.1)	-23.3 (248.8)	0.09	0.93
85–89	3088.5 (238.3)	3179.6 (73.5)	-91.1 (164.8)	0.55	0.59
90–94	3434.1 (348.6)	3492.8 (159.9)	-58.7 (188.7)	0.31	0.76
95+	3401.9 (1189.4)	2174.2 (367.3)	1227.7 (822.1)	1.49	0.16

Table B.3: Difference in the ARMA and OLS predicted age specific rate, 2020, male lung cancer; together with the associated Student's *T* and *p*-values.

Age category (years)	ARMA prediction	OLS prediction	Difference in predictions	*T*	*p*-value
0–4	0.2 (0.3)	0.1 (0.1)	0.2 (0.2)	0.69	0.49
5–9	0.0 (0.1)	0.0 (0.0)	-0.0 (0.1)	0.07	0.95
10–14	0.0 (0.2)	0.0 (0.1)	-0.0 (0.2)	0.13	0.90
15–19	0.1 (0.4)	0.1 (0.1)	-0.0 (0.3)	0.01	0.99
20–24	0.4 (0.6)	0.3 (0.2)	0.1 (0.4)	0.31	0.76
25–29	1.0 (0.8)	0.7 (0.2)	0.3 (0.6)	0.53	0.60
30–34	0.5 (1.1)	1.0 (0.4)	-0.5 (0.7)	0.68	0.50
35–39	2.8 (2.0)	2.9 (0.6)	-0.1 (1.4)	0.06	0.95
40–44	5.1 (3.9)	4.2 (0.7)	0.9 (3.2)	0.28	0.78
45–49	10.5 (10.2)	9.7 (1.5)	0.8 (8.7)	0.09	0.93
50–54	6.6 (11.5)	20.1 (1.7)	-13.6 (9.8)	1.39	0.18
55–59	21.2 (22.4)	44.2 (2.7)	-23.1 (19.7)	1.17	0.25
60–64	66.3 (27.1)	93.9 (6.5)	-27.6 (20.6)	1.34	0.19
65–69	167.8 (41.7)	174.5 11.8	-6.7 (30.0)	0.22	0.82
70–74	250.7 (56.0)	294.2 (15.7)	-43.5 (40.3)	1.08	0.29
75–79	341.8 (72.2)	388.0 (19.4)	-46.2 (52.9)	0.87	0.39
80–84	496.6 (102.8)	487.1 (30.1)	9.5 (72.7)	0.13	0.90
85–89	452.1 (90.4)	457.3 (29.4)	-5.3 (61.0)	0.09	0.93
90–94	409.7 (173.6)	366.8 (46.8)	42.9 (126.8)	0.34	0.74
95+	326.0 (342.6)	384.1 (116.4)	-58.1 (226.2)	0.26	0.80

Table B.4: Difference in the ARMA and OLS predicted age specific rate, 2020, female lung cancer; together with the associated Student's *T* and *p*-values.

Age category (years)	ARMA prediction	OLS prediction	Difference in predictions	*T*	*p*-value
0–4	0.6 (0.5)	0.1 (0.1)	0.5 (0.3)	1.5	0.16
5–9	0.0 (0.2)	0.0 (0.0)	-0.0 (0.2)	-0.1	0.95
10–14	-0.1 (0.3)	0.0 (0.1)	-0.2 (0.2)	-0.8	0.41
15–19	0.4 (0.8)	0.2 (0.1)	0.2 (0.7)	0.3	0.73
20–24	0.3 (0.6)	0.2 (0.2)	0.1 (0.4)	0.2	0.86
25–29	0.8 (1.1)	1.1 (0.3)	-0.3 (0.8)	-0.4	0.72
30–34	1.1 (2.1)	1.0 (0.4)	0.2 (1.7)	0.1	0.92
35–39	5.0 (3.0)	2.5 (0.6)	2.5 (2.4)	1.0	0.32
40–44	6.6 (3.1)	6.8 (1.0)	-0.1 (2.1)	-0.1	0.95
45–49	23.7 (7.7)	15.5 (2.1)	8.2 (5.6)	1.5	0.16
50–54	28.8 (10.0)	28.7 (2.4)	0.1 (7.6)	0.0	0.99
55–59	58.1 (18.4)	60.3 (5.3)	-2.2 (13.1)	-0.2	0.87
60–64	76.4 (23.4)	79.8 (6.9)	-3.4 (16.5)	-0.2	0.84
65–69	144.5 (28.1)	138.0 (9.3)	6.5 (18.8)	0.3	0.73
70–74	191.6 (32.3)	191.0 (9.4)	0.6 (22.9)	0.0	0.98
75–79	249.7 (41.2)	259.0 (13.6)	-9.3 (27.6)	-0.3	0.74
80–84	291.3 (39.7)	276.5 (13.8)	14.8 (25.9)	0.6	0.58
85–89	249.4 (32.5)	236.7 (11.6)	12.7 (20.9)	0.6	0.55
90–94	230.7 (86.0)	198.6 (24.9)	32.1 (61.1)	0.5	0.61
95+	174.8 (106.1)	151.4 (32.9)	23.4 (73.2)	0.3	0.75

Appendix C: Data sources

Australian Cancer Database

The ACD holds information about 1.9 million cases of Australians who were diagnosed with cancer (other than basal cell and squamous cell carcinomas of the skin) between 1982 and 2007.

The AIHW compiles and maintains the ACD, in partnership with the AACR, whose member registries provide data to the AIHW on an annual basis. Each Australian state and territory has legislation that makes the reporting of all cancers (excluding basal cell and squamous cell carcinomas of the skin) mandatory. Pathology laboratories and Registrars of Births, Deaths and Marriages across Australia must report on cancer cases, as do hospitals, radiation oncology units and nursing homes in some (but not all) jurisdictions.

The data provided to the AIHW by the state and territory cancer registries include, at a minimum, an agreed set of items that provide information about the individual with the cancer, the characteristics of the cancer and, where relevant, deaths from malignant tumours. In addition to the agreed set of items, registries often provide other data which are also included in the ACD. For example, data on ductal carcinoma in situ (DCIS) are not part of the agreed ACD data set but are regularly provided by the state and territory registries.

Once the data are received from the state and territory cancer registries, the AIHW assembles the data into the ACD. Internal linking checks are undertaken to identify those who had tumours diagnosed in more than one state or territory; this process reduces the degree of duplication within the ACD to a negligible rate. The ACD is also linked with information on deaths (from the National Death Index) to add information on which people with cancer have died (from any cause). Any conflicting information and other issues with the cancer data are resolved through consultation with the relevant state or territory cancer registry.

The registration of cases of cancer is a dynamic process and records in the state and territory cancer registries may be modified if new information is received. Thus, records in the cancer registries are always open and updated as required. For these changes to be incorporated into the ACD, a new complete file for all years of cancer data is provided by each of the jurisdictions annually. As a result, the number of cancer cases reported by the AIHW for any particular year may change slightly over time, and data published by a cancer registry at a certain point in time may differ from what is published by the AIHW (AIHW 2009).

The data in the ACD are protected both physically with built-in computer security systems and by legislation under the Australian Institute of Health and Welfare Act 1987 and agreements with the state and territory cancer registries. More information about physical security and legislative protection of the ACD is in the National Cancer Statistics Clearing House protocol (AIHW 2009).

Data Quality Statement

Data Quality Statement: Australian Cancer Database

Important note

To avoid excessive repetition in what follows, the word 'cancer' is used to mean 'cancer, excluding basal cell carcinomas of the skin and squamous cell carcinomas of the skin'. In most states and territories these two very common skin cancers are not notifiable diseases and as such are not in the scope of the ACD.

Summary of key issues

- All states and territories maintain a population-based cancer registry to which all cancer cases and deaths must be reported.
- The AIHW compiles the Australian Cancer Database using information from state and territory registers.
- Some duplication may occur where the same person and cancer have been registered in two or more jurisdictions. The AIHW temporarily resolves these instances, but full resolution usually occurs with the following year's release.
- The level of duplication is small, about 0.17% of all records.
- Cancer registry databases change every day, adding new records and improving the quality of existing records as new information becomes available. Information on ACD records may therefore change from year to year.

Description	All states and territories have legislation that makes cancer a notifiable disease. All hospitals, pathology laboratories, radiotherapy centres and registries of births, deaths and marriages must report cancer cases and deaths to the state/territory population-based cancer registry. Each registry supplies incidence data annually to the AIHW under an agreement between the registries and the AIHW. These data are compiled into the only repository of national cancer incidence data—the Australian Cancer Database (ACD).
Institutional environment	The Australian Institute of Health and Welfare (AIHW) is a major national agency set up by the Australian Government under the *Australian Institute of Health and Welfare Act* 1987 to provide reliable, regular and relevant information and statistics on Australia's health and welfare. It is an independent statutory authority established in 1987, governed by a management Board, and accountable to the Australian Parliament through the Health and Ageing portfolio. The AIHW aims to improve the health and wellbeing of Australians through better health and welfare information and statistics. It collects and reports information on a wide range of topics and issues, ranging from health and welfare expenditure, hospitals, disease and injury, and mental health, to ageing, homelessness, disability and child protection. The Institute also plays a role in developing and maintaining national

	metadata standards. This work contributes to improving the quality and consistency of national health and welfare statistics. The Institute works closely with governments and non-government organisations to achieve greater adherence to these standards in administrative data collections to promote national consistency and comparability of data and reporting. One of the main functions of the AIHW is to work with the states and territories to improve the quality of administrative data and, where possible, to compile national datasets based on data from each jurisdiction, to analyse these datasets and disseminate information and statistics. The *Australian Institute of Health and Welfare Act* 1987, in conjunction with compliance to the *Privacy Act* 1988 (Cth), ensures that the data collections managed by the AIHW are kept securely and under the strictest conditions with respect to privacy and confidentiality. For further information see the AIHW website www.aihw.gov.au. The AIHW has been maintaining the ACD since 1986.
Timeliness	The version of the ACD used in this analysis contained data on all cancer cases diagnosed between 1982 and 2007. Each jurisdictional cancer registry supplies data annually to the AIHW. Because each jurisdiction operates on its own data compilation and reporting cycle, the ACD cannot be fully compiled until the final jurisdiction supplies its data.
Accessibility	The AIHW website provides cancer incidence and mortality data, which can be downloaded free of charge. Numerous reports, including the biennial *Cancer In Australia*, are published and are available on the AIHW website where they can be downloaded without charge. Users can request data not available online or in reports via the Cancer and Screening Unit of the Australian Institute of Health and Welfare on (02) 6244 1000 or via email to cancer@aihw.gov.au Requests that take longer than half an hour to compile are charged for on a cost-recovery basis. General enquiries about AIHW publications can be made to the Communications, Media and Marketing Unit on (02) 6244 1032 or via email to info@aihw.gov.au. Researchers who are following a cohort of people enrolled in a longitudinal study of health outcomes can request the AIHW to undertake data linkage of their cohort to the ACD. Such requests must be approved by the AIHW Ethics Committee as well as the ethics committees governing access to the state/territory cancer registries.
Interpretability	Information on the ACD is available on the AIHW website. While numbers of new cancers are easy to interpret, other statistical calculations (for example, calculations of age-standardised rates and confidence intervals) are more complex and their concepts may be confusing to some users. In most publications there is an appendix on statistical methods as well as technical notes.
Relevance	The ACD is highly relevant for monitoring trends in cancer incidence. The data are used for many purposes: by policy makers to evaluate health

	intervention programs and as background data for health labour force planning, health expenditure, and so forth; by pharmaceutical companies to assess the size of the market for new drugs; by researchers to explore the epidemiology of cancer; by insurance companies to evaluate the risk of people being diagnosed with cancer. The ACD contains information on all reported cancer cases and deaths in Australia. Data can be provided at state and territory level and at Remoteness Area level. The 3rd edition of the International Classification of Diseases for Oncology (ICD-O-3) is used to classify cancer cases. Data can also be provided classified according to the 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10). While all state and territory cancer registries collect information on Indigenous status, in some jurisdictions the level of identification of Indigenous Australians is not considered to be sufficient to enable analysis. The ACD also contains the name and date of birth of each person who has been diagnosed with cancer. This allows researchers who have enrolled people in a study to link their database to the ACD to find out which of their study subjects have been diagnosed with cancer, what kind of cancer, and when. (Such data linkage can only be undertaken after receiving approvals from various ethics committees.) This kind of research gives insight into cancer risk factors. Data linkage is also undertaken when a researcher has been contracted to investigate a potential cancer cluster in a workplace or small area.
Accuracy	The publication *Cancer incidence in five continents* is issued about every 5 years as a collaborative effort by the International Agency for Research on Cancer (IARC) and the worldwide network of cancer registries. Australia's cancer registries continue to pass IARC's numerous tests for data quality. Details of the tests and Australia's cancer registries' results in them can be found in the above-mentioned book and appendices of the registries' annual incidence reports. Each year when all the registries' new data have been compiled into the new ACD a data linkage process called the national deduplication is undertaken. This process detects instances where the same person and cancer have been registered in two or more jurisdictions. This could happen, for example, when a person attends hospitals in different jurisdictions. All such instances that are found are temporarily resolved at the AIHW by removing one record while the relevant jurisdictions are notified of the situation so that they can determine in which jurisdiction the person was a usual resident at the time of diagnosis. Their resolution will flow through to the ACD in the next year's data supply. In recent years the national deduplication has resulted in the removal of about 3,500 records from the ACD, which is about 0.17% of all records supplied by the jurisdictions. While all state and territory cancer registries collect information on

	Indigenous status, in some jurisdictions the level of identification of Indigenous Australians is not considered to be sufficient to enable analysis. Data for four states and territories—New South Wales, Queensland, Western Australia and the Northern Territory—are considered suitable for analysis. Cancer registry databases change every day, and not just because new records are added. Existing records are changed if new, more precise, information about the diagnosis becomes available. Also, any typographical errors that are discovered by routine data checking procedures are corrected by referring to the source documentation. Finally, existing records can be deleted if it is discovered that the initial diagnosis of cancer was incorrect, for example, the tumour was in fact benign, or the person is found to be not a resident of that state/territory. As a result of all these issues, the number of cancer cases reported by AIHW for any particular year may change slightly over time, and data published by a cancer registry at a certain point in time may differ slightly from what is published by the AIHW at a different time.
Coherence	Cancer data are reported and published annually by the AIHW. While there are sometimes changes to coding for particular cancers, it is possible to map coding changes to make meaningful comparisons over time.

Estimated resident populations

To derive estimates of the resident populations, the ABS uses the 5-yearly Census of Population and Housing data and adjusts it as follows:

- all respondents in the Census are placed in their state or territory, statistical local area and postcode of usual residence; overseas visitors are excluded
- an adjustment is made for persons missed in the Census (about 2%)
- Australians temporarily overseas on Census night are added to the usual residence Census count.

Estimated resident populations are then updated each year from the census data using indicators of population change, such as births, deaths and net migration. More information is available from the ABS website <www.abs.gov.au>.

Population projections

*Population projections, Australia, 2006 to 2101 (*series B*)* are based on preliminary 2007 Census-based estimated resident populations and projected to 2101 using the cohort-component method (ABS 2008). In this method the base population is projected forward annually by calculating the effect of births, deaths and migration within each age-sex group according to the specified fertility, mortality and migration assumptions. Assumptions of birth rates, death rates and migration are based on historical patterns and trends over the previous decade.

Medicare data

Medicare Benefits Schedule (MBS) data were used for analysis of the relationship between PSA testing and prostate cancer incidence. MBS data are available by year, age and jurisdiction (Australian Government Department of Human Services 2011).

Data extracted include only those services that are performed by a registered provider for services that qualify for Medicare Benefit and for which a claim has been processed by Medicare Australia. They do not include services provided by hospital doctors to public patients in public hospitals or services that qualify for a benefit under the Department of Veterans' Affairs National Treatment Account.

Month is determined by the date the service was processed by Medicare Australia, not the date the service was provided, and may vary due to the varying number of processing days in a month, which depends on the number of days and public holidays in the month.

Glossary

Age-specific rate: A rate for a specific age group. The numerator and denominator relate to the same age group.

Age-standardisation: A method of removing the influence of age when comparing populations with different age structures. This is usually necessary because the rates of many diseases vary strongly (usually increasing) with age. The age structures of the different populations are converted to the same 'standard' structure; then the disease rates that would have occurred with that structure are calculated and compared.

Australian Cancer Database (2007): A collection of all primary cancers diagnosed in Australia from 1982 to 2007 maintained by the Australian Institute of Health and Welfare on behalf of the Australasian Association of Cancer Registries.

Body Mass Index: The most commonly used method of assessing whether a person is normal weight, underweight, overweight or obese. It is calculated by dividing the person's weight (in kilograms) by their height (in metres) squared; that is, kg ÷ m^2. For both men and women, underweight is a BMI below 18.5, acceptable weight is from 18.5 to less than 25, overweight is 25 and above (includes obese), and obese is 30 and over.

Cancer (malignant neoplasm): A large range of diseases in which some of the body's cells become defective, begin to multiply out of control. These can invade and damage the area around them and can also spread to other parts of the body to cause further damage.

Crude rate: The number of events in a given period divided by the size of the population at risk in a specified time period.

Incidence: The number of new cases (of an illness or event) occurring during a given period.

International Statistical Classification of Diseases and Related Health Problems: The World Health Organization's internationally accepted classification of death and disease. The tenth revision (ICD-10) is currently in use and is used to define cancer classifications in the Australian Cancer Database.

Obesity: Marked degree of overweight, defined for population studies as a *body mass index* of 30 or over. See also *overweight.*

Overweight: Defined for the purpose of population studies as a *body mass index* of 25 or over. See also *obesity.*

Population estimates: Official population numbers compiled by the Australian Bureau of Statistics at both state and territory and statistical local area levels by age and sex, as at 30 June each year.

Prediction interval: There are 19 chances in 20 that a 95% prediction interval for a forecasted incidence rate (or count) will contain the (true) attained value. This interval is derived from measures of both the inaccuracy of trend estimation and its effect on the associated predictive model; the corresponding narrower confidence interval reflects only the inaccuracy of the trend estimate.

Primary cancer: A tumour that is at the site where it first formed.

Projection: Longer-term extrapolation of recent trend data using unknown parameters such as expected future populations.

Secondary cancer: A tumour that originated from a cancer elsewhere in the body. Also referred to as a metastatic cancer.

Statistical significance: An indication from a statistical test that an observed difference or association may be 'real' because it is unlikely to be due to chance. A statistical result is usually said to be 'significant' if it would occur by chance only once in 20 times or less often.

Year-to-date estimate: Simple extrapolation of recent trend data to the current year. This uses known parameters such as current populations.

References

ABS (Australian Bureau of Statistics) 2008. 3222.0 Population projections, Australia, 2006-2101. Canberra: ABS.

ABS 2009. Experimental estimates and projections, Aboriginal and Torres Strait Islander Australians, 1991 to 2021. ABS Cat. No. 3238.0. Canberra: ABS.

AIHW (Australian Institute of Health and Welfare) 2007. Cancer in Australia: an overview, 2006. Cancer series 37. Cat. no. CAN 32. Canberra: AIHW.

AIHW 2008. 2007 National Drug Strategy Household Survey: detailed finding. Cat. no. PHE 107. Canberra: AIHW.

AIHW 2009. National Cancer Statistics Clearing House protocol 2009. Canberra: AIHW.

AIHW 2010. Australia's health 2010. Cat. no. AUS 122. Canberra: AIHW.

AIHW 2011a. Cancer in adolescents and young adults: an analysis of incidence, mortality and survival in Australia. Can. Canberra: AIHW.

AIHW 2011b. BreastScreen Australia monitoring report 2008-2009. Canberra: AIHW.

AIHW & AACR (Australasian Association of Cancer Registries) 2010. Cancer in Australia: an overview, 2010. Cancer series 60. Cat. no. CAN 56. Canberra: AIHW.

AIHW & CA (Cancer Australia) 2008. Non-melanoma skin cancer: general practice consultations, hospitalisations and mortality. Cancer series 43. Cat. no. CAN 39. Canberra: AIHW.

AIHW & CA 2011. Lung cancer in Australia: an overview. Cat. no. CAN 58. Canberra: AIHW.

AIHW & DoHA (Australian Government Department of Health and Ageing) 2008. National Bowel Cancer Screening Program : annual monitoring report 2008. Cancer series no. 49. Cat. no. CAN 40. Canberra: AIHW.

AIHW & DoHA 2011. Cervical screening in Australia 2008-2009. Cancer series 54. Cat. no. CAN 61. Canberra: AIHW.

AIHW & NBOCC (National Breast and Ovarian Cancer Centre) 2010. Ovarian cancer in Australia: an overview, 2010. Cancer series 52. Cat. no. CAN 48. Canberra: AIHW.

AIHW & NBOCC 2009. Breast cancer in Australia: an overview, 2009. Cancer series no. 50. Cat. no. CAN 46. Canberra: AIHW.

AIHW: Ridolfo B & Stevenson C 2001. The quantification of drug-caused mortality and morbidity in Australia, 1998. Drug statistics series no. 7. Cat. no. PHE 29. Canberra: AIHW.

Akre O, Pettersson A & Richiardi L 2009. Risk of contralateral testicular cancer among men with unilaterally undescended testis: a meta analysis. International Journal of Cancer 124:687-9.

Alam N, Chen W, Baker D & Bishop J 2009. Liver cancer in New South Wales. Sydney: Cancer Institute NSW.

ANECS (Australian National Endometrial Cancer Study) 2011. Viewed 21 November 2011. <http://www.anecs.org.au/>.

Armstrong BK and Kricker A 2001. The epidemiology of UV induced skin cancer. Journal of Photochemistry and Photobiology B: Biology 63 (1–3): 8–18.

ARPANSA (Australian Radiation Protection and Nuclear Safety Agency) 2011. Fact sheet 13: Mobile telephones and health effects. Viewed 11 November 2011, <https://www.arpansa.gov.au/mobilephones>.

Australian Cancer Network Colorectal Cancer Guidelines Revision Committee 2005. Clinical practice guidelines for the prevention, early detection and management of colorectal cancer. Sydney: Cancer Council Australia and Australian Cancer Network.

Australian Government Department of Human Services (DoHS) 2011. Medicare Item Reports. Canberra: Australian Government Department of Human Services. Viewed 3 November 2011, <https://www.medicareaustralia.gov.au/statistics/mbs_item.shtml>.

Australian Mesothelioma Registry 2011. Australian Mesothelioma Registry. Sydney: Cancer Institute NSW. Viewed 21 October 2011, <http://www.mesothelioma-australia.com/home-page.aspx>.

Baade P, Carriere P & Fritschi L 2008. Trends in testicular germ cell cancer incidence in Australia. Cancer Causes and Control 19:1043-9.

Baade P & Coory M 2005. Trends in melanoma mortality in Australia: 1950–2002 and their implications for melanoma control. Australian and New Zealand journal of public health 29:383-6.

Beiki O, Granath F, Allebeck P, Akre O & Moradi T 2010. Subtype-specific risk of testicular tumors among immigrants and their descendants in Sweden, 1960 to 2007. Cancer Epidemiology Biomarkers & Prevention 19:1053-65.

Bergström A, Hsieh C, Lindblad P, Lu C, Cook N & Wolk A 2001. Obesity and renal cell cancer–a quantitative review. British Journal of Cancer 85:984.

Bianchi C & Bianchi T 2007. Malignant mesothelioma: global incidence and relationship with asbestos. Industrial Health 45:379–387.

Bouvard V, Baan R, Straif K, Grosse Y, Secretan B, El Ghissassi F, Benbrahim-Tallaa L, Guha N, Freeman C, Galichet L, Cogliano V 2009 on behalf of the WHO International Agency for Research on Cancer Monograph Working Group. A review of human carcinogens—Part B: biological agents. The Lancet Oncology 10(4):321-322.

Brenner H, Rothenbacher D & Arndt V 2009. Epidemiology of stomach cancer. Methods in Molecular Biology 2:467-78.

Calle E, Rodriguez C, Dal Maso L, Talamini R, Bain CJ, Green AC et al. 2008. Ovarian cancer and oral contraceptives: collaborative reanalysis of data from 45 epidemiological studies including 23,257 women with ovarian cancer and 87,303 controls. The Lancet 371:303-14.

Cancer Care Ontario 2006. Cancer in young adults in Canada. Toronto: Cancer Care Ontario.

Cancer Council NSW 2007. Liver cacer - what is liver cancer. Sydney: Cancer Council NSW. Viewed 2 November 2011, <http://www.cancercouncil.com.au/editorial.asp?pageid=2638>.

Chang ET, Smedby KE, Hjalgrim H, Porwit-MacDonald A, Roos G, Glimelius B et al. 2005. Family history of hematopoietic malignancy and risk of lymphoma. Journal of the National Cancer Institute 97:1466-74.

Chang ET, Zheng T, Weir EG, Borowitz M, Mann RB, Spiegelman D et al. 2004. Childhood social environment and Hodgkin's lymphoma: new findings from a population-based case-control study. Cancer Epidemiology, Biomarkers & Prevention 13:1361-70.

Chatenoud L, Gallus S, Altieri A, Negri E, Talamini R, Franceschi S et al. 2005. Number of siblings and risk of hodgkin's and other lymphoid neoplasms. Cancer Epidemiology, Biomarkers & Prevention 14:552.

Clements M, Berry G & Shi J 2007a. Actuarial projections for mesothelioma: an epidemiological perspective. Presented to Actuaries of Australia XIth Accident Compensation Seminar 2007a.

Clements M, Berry G, Shi J, Ware S, Yates D & Johnson A 2007b. Projected mesothelioma incidence in men in New South Wales. Occupational and Environmental Medicine 64:747-52.

Colt JS, Baris D, Stewart P, Schned AR, Heaney JA, Mott LA et al. 2004. Occupation and bladder cancer risk in a population-based case-control study in New Hampshire. Cancer Causes and Control 15:759-69.

Coory M & Armstrong B 1998. Cancer incidence projections for Area and Rural Health Services in New South Wales. Sydney: NSW Cancer Council.

COSMOS (Cohort study of mobile phone use and health) 2011. COSMOS. Imperial College: London. Viewed 9 November 2011, <http://www.ukcosmos.org/index.html>.

Creighton P, Lew JB, Clements M, Smith M, Howard K, Dyer S et al. 2010. Cervical cancer screening in Australia: modelled evaluation of the impact of changing the recommended interval from two to three years. BMC Public Health 10:734-48.

Cust A, Armstrong B, Goumas C, Jenkins M, Schmid H, Hopper J, Kefford R, Giles G, Aitken J and Mann G 2011. Sunbed use during adolescence and early adulthood is associated with increased risk of early-onset melanoma. International Journal of Cancer 128: 2425–2435.

Dal Maso L, Bosetti C, La Vecchia C & Franceschi S 2009. Risk factors for thyroid cancer: an epidemiological review focused on nutritional factors. Cancer Causes and Control 20:75-86.

De P, Neutel CI, Olivotto I & Morrison H 2010. Breast cancer incidence and hormone replacement therapy in Canada. Journal of the National Cancer Institute 102:1489-95.

De Sanjose S, Benavente Y, Vajdic CM, Engels EA, Morton LM, Bracci PM et al. 2008. Hepatitis C and non-Hodgkin lymphoma among 4784 cases and 6269 controls from the International Lymphoma Epidemiology Consortium. Clinical Gastroenterology and Hepatology 6:451-8.

Devesa SS & Fears T 1992. Non-Hodgkin's lymphoma time trends: United States and international data. Cancer Research 52:5432-40.

DoHA 2008. The Australian Immunisation Handbook. Canberra: DoHA.

DoHA 2011a. National Tobacco Campaign 2011. Canberra: DoHA. Viewed 2 November 2011, <http://www.quitnow.gov.au/internet/quitnow/publishing.nsf/Content/ntc-2009-2013-lp>.

DoHA 2011b. The National Skin Cancer Awareness Campaign. Canberra: DoHA. Viewed 2 November 2011, <http://www.skincancer.gov.au/internet/skincancer/publishing.nsf/Content/home>.

Enewold L, Zhou J, Devesa SS, de Gonzalez AB, Anderson WF, Zahm SH et al. 2011. Thyroid cancer incidence among active duty US military personnel, 1990-2004. Cancer Epidemiology Biomarkers & Prevention epub ahead of print.

Enewold L, Zhu K, Ron E, Marrogi AJ, Stojadinovic A, Peoples GE et al. 2009. Rising thyroid cancer incidence in the United States by demographic and tumor characteristics, 1980-2005. Cancer Epidemiology Biomarkers & Prevention 18:784.

Engel LS, Chow WH, Vaughan TL, Gammon MD, Risch HA, Stanford JL et al. 2003. Population attributable risks of esophageal and gastric cancers. Journal of the National Cancer Institute 95:1404-13.

Evens AM & Chiu BCH 2008. The challenges of epidemiologic research in non-Hodgkin lymphoma. The Journal of the American Medical Association 300:2059-61.

Forman D & Burley V 2006. Gastric cancer: global pattern of the disease and an overview of environmental risk factors. Best Practice & Research Clinical Gastroenterology 20:633-49.

Freedman ND, Abnet CC, Leitzmann MF, Mouw T, Subar AF, Hollenbeck AR et al. 2007. A prospective study of tobacco, alcohol, and the risk of esophageal and gastric cancer subtypes. American journal of epidemiology 165:1424-33.

Freedman ND, Silverman DT, Hollenbeck AR, Schatzkin A & Abnet CC 2011. Association Between Smoking and Risk of Bladder Cancer Among Men and Women. The Journal of the American Medical Association 306:737-45.

Ganem D & Prince AM 2004. Hepatitis B virus infection—natural history and clinical consequences. New England Journal of Medicine 350:1118-29.

Grodski S, Brown T, Sidhu S, Gill A, Robinson B, Learoyd D et al. 2008. Increasing incidence of thyroid cancer is due to increased pathologic detection. Surgery 144:1038-43.

Grulich AE, Vajdic CM & Cozen W 2007a. Altered immunity as a risk factor for non-Hodgkin lymphoma. Cancer Epidemiology Biomarkers & Prevention 16:405-8.

Grulich AE, van Leeuwen MT, Falster MO & Vajdic CM 2007b. Incidence of cancers in people with HIV/AIDS compared with immunosuppressed transplant recipients: a meta-analysis. The Lancet 370:59-67.

Hassan MM, Bondy ML, Wolff RA, Abbruzzese JL, Vauthey JN, Pisters PW et al. 2007. Risk factors for pancreatic cancer: case-control study. The American journal of gastroenterology 102:2696-707.

Hoang J 2010. Thyroid nodules and evaluation of thyroid cancer risk. Australasian Journal of Ultrasound in Medicine November 13:33-6.

Hollingsworth JM, Miller DC, Daignault S & Hollenbeck BK 2006. Rising incidence of small renal masses: a need to reassess treatment effect. Journal of the National Cancer Institute 98:1331-4.

Horn-Ross PL, Canchola AJ, Ma H, Reynolds P & Bernstein L 2011. Hormonal Factors and the Risk of Papillary Thyroid Cancer in the California Teachers Study Cohort. Cancer Epidemiology Biomarkers & Prevention 20:1751.

Hu J, La Vecchia C, Morrison H, Negri E & Mery L 2011. Salt, processed meat and the risk of cancer. European Journal of Cancer Prevention 20:132-9.

Hunt JD, Van der Hel OL, McMillan GP, Boffetta P & Brennan P 2005. Renal cell carcinoma in relation to cigarette smoking: Meta analysis of 24 studies. International Journal of Cancer 114:101-8.

Huxley R, Ansary-Moghaddam A, de Gonzalez AB, Barzi F & Woodward M 2005. Type-II diabetes and pancreatic cancer: a meta-analysis of 36 studies. British Journal of Cancer 92:2076-83.

International Agency for Research on Cancer (IARC) 2008. World cancer report 2008. Lyon: IARC.

Iodice S, Gandini S, Maisonneuve P & Lowenfels AB 2008. Tobacco and the risk of pancreatic cancer: a review and meta-analysis. Langenbeck's Archives of Surgery 393:535-45.

Jarrett RF 2002. Viruses and Hodgkin's lymphoma. Ann Oncol 13 Suppl 1:23-9.

Jensen O, Parkin D, MacLennan R, Muir C & Skeet R (eds) 1991. Cancer registration:principles and methods. Lyon: IARC.

Kim HJ, Fay MP, Feuer EJ & Midthune DN 2000. Permutation tests for joinpoint regression with applications to cancer rates. Statistics in Medicine 19:335-51.

La Vecchia C 2006. Oral contraceptives and ovarian cancer: an update, 1998-2004. European Journal of Cancer Prevention 15:117-124.

Ladeiras-Lopes R, Pereira AK, Nogueira A, Pinheiro-Torres T, Pinto I, Santos-Pereira R et al. 2008. Smoking and gastric cancer: systematic review and meta-analysis of cohort studies. Cancer Causes and Control 19:689-701.

Lahti TA, Partonen T, Kyyrönen P, Kauppinen T & Pukkala E 2008. Night time work predisposes to non Hodgkin lymphoma. International Journal of Cancer 123:2148-51.

Larsson S, Orsini N, Brismar K & Wolk A 2006. Diabetes mellitus and risk of bladder cancer: a meta-analysis. Diabetologia 49:2819-23.

Lerman P 1980. Fitting segmented regression models by grid search. Applied statistics:77-84.

Liang KY, Zeger SL & Qaqish B 1992. Multivariate regression analyses for categorical data. Journal of the Royal Statistical Society. Series B (Methodological):3-40.

Lochan R, Daly A, Reeves H & Charnley R 2008. Genetic susceptibility in pancreatic ductal adenocarcinoma. British Journal of Surgery 95:22-32.

Masood J, Lane T, Koye B, Vandal M, Barua J & Hill J 2001. Renal cell carcinoma: incidental detection during routine ultrasonography in men presenting with lower urinary tract symptoms. BJU international 88:671-4.

MakinJK & Dobbinson SJ 2009. Changes in solarium numbers in Australia following negative media and legislation. Australian and New Zealand Journal of Public Health 33(5): 491–494.

Muir C 1995. Cancer of unknown primary site. Cancer supplement: An interdisciplinary international journal of the American Cancer Society 75:353–356.

Müller AMS, Ihorst G, Mertelsmann R & Engelhardt M 2005. Epidemiology of non-Hodgkin's lymphoma (NHL): trends, geographic distribution, and etiology. Annals of hematology 84:1-12.

NCI (National Cancer Institute) 2011a. Cell phones and cancer risk. Bethesda, MD: National Institutes of Health, United States Department of Health and Human Services. Viewed 9 November 2011, <http://www.cancer.gov/cancertopics/factsheet/Risk/cellphones>.

NCI 2011b. Joinpoint regression program. Bethesda, MD: National Institutes of Health, United States Department of Health and Human Services. Viewed 3 November 2011, <http://surveillance.cancer.gov/joinpoint/ >.

NCI 2011c. What you need to know about cancer of the uterus. Bethesda, MD: National Institutes of Health, United States Department of Health and Human Services. Viewed 21 November 2011. <http://www.cancer.gov/cancertopics/wyntk/uterus/page4>.

Natoli C, Ramazzotti V, Nappi O, Giacomini P, Palmeri S, Salvatore M, Landriscina M, Zilli M, Natali PG, Tinari N & Iacobelli S 2011. Unknown primary tumors. Biochimica et Biophysica Acta (BBA) Reviews on Cancer. 1816:13–24.

OECD (Organisation for Economic Co-operation and Development) 2010. OECD Health Data 2010. Paris: OECD. Viewed 1 March 2011, <http:// www.ecosante.org/index2.php?base=OCDE&langh=ENG&langs=ENG&sessionid=>.

Parkin DM 2001. Global cancer statistics in the year 2000. The Lancet Oncology 2:533-43.

Pelucchi C, Bosetti C, Negri E, Malvezzi M & La Vecchia C 2006. Mechanisms of disease: the epidemiology of bladder cancer. Nature Reviews Urology 3:327-40.

Richards M 2008. Trends and inequalities in survival for 20 cancers in England and Wales 1986-2001: population-based analyses and clinical commentaries. British Journal of Cancer 99:S1.

Richiardi L, Pettersson A & Akre O 2007. Genetic and environmental risk factors for testicular cancer. International journal of andrology 30:230-41.

Robinson D, Møller H & Horwich A 2007. Mortality and incidence of second cancers following treatment for testicular cancer. British Journal of Cancer 96:529-33.

Safe Work Australia 2010. Mesothelioma in Australia: incidence 1982 to 2006, deaths 1997 to 2007. Canberra: Safe Work Australia.

Scélo G & Brennan P 2007. The epidemiology of bladder and kidney cancer. Nature Clinical Practice Urology 4:205-17.

Smith DP, Supramaniam R, Marshall VR & Armstrong BK 2008. Prostate cancer and prostate-specific antigen testing in New South Wales. Med J Aust 189:315-8.

Sprague BL, Warren Andersen S & Trentham-Dietz A 2008. Thyroid cancer incidence and socioeconomic indicators of health care access. Cancer Causes and Control 19:585-93.

Tracey E, Ling L, Baker D, Dobrovic A & Bishop J 2009. Cancer in New South Wales: incidence and mortality 2007. Sydney: Cancer Institute NSW.

Tucker MA and Goldstein AM 2003. Melanoma aetiology: where are we? Oncogene 22: 3042–3052.

Wang XQ, Terry PD & Yan H 2009. Review of salt consumption and stomach cancer risk: Epidemiological and biological evidence. World Journal of Gastroenterology: WJG 15:2204.

WHO (World Health Organization) 2010. Fact sheet no 287. Sunbeds, tanning and UV exposure. Viewed 16 February 2012. <http://www.who.int/mediacentre/factsheets/fs287/en/>.

WHO 2011. Fact sheet No. 193. Electromagnetic fields and public heatlh: mobile phones. Viewed 14 November 2011.
<http://www.who.int/mediacentre/factsheets/fs193/en/index.html>.

World Cancer Research Fund / American Institute for Cancer Research (WCRF/AICR) 2007. Food, nutritian, physical activity, and the prevention of cancer: a global perspective. Washington DC: American Institute of Cancer Research.

Wrensch M, Minn Y, Chew T, Bondy M & Berger MS 2002. Epidemiology of primary brain tumors: current concepts and review of the literature. Neuro-oncology 4:278-99.

Yang L, Fujimoto J, Qiu D & Sakamoto N 2009. Trends in cancer mortality in Japanese adolescents and young adults aged 15–29 years, 1970–2006. Annals of Oncology 20:758-66.

Yao R, Chiu CG, Strugnell SS, Gill S & Wiseman SM 2011. Gender differences in thyroid cancer: a critical review. Expert Review of Endocrinology and Metabolism 6:215-43.

Zeger, S & Liang, K 1992. An overview of methods for the analysis of longitudinal data. Statistics in Medicine 11:1825-1839.

List of tables

List of figures

Related publications

This report, *Cancer projections in Australia, 2011–2020*, is a companion report to *Cancer in Australia: an overview, 2010* which can be downloaded free on the AIHW website < http://www.aihw.gov.au/publication-detail/?id=6442472459>

Additional data tables providing projected counts and rates for 5-year age groups are also available on the AIHW website.