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Prostate cancer prevalence in New South Wales Australia: A population-based study

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ABSTRACT

Background: Information on the current and future numbers of Australian men living with prostate cancer is limited. We describe a method for estimating complete prevalence of prostate cancer to provide a measure of the burden of prostate cancer in Australia.
Methods: Prostate cancer data from the New South Wales (NSW) Central Cancer Registry were used with PIAMOD (Prevalence and Incidence Analysis MODel) software to estimate future prostate cancer prevalence in NSW. We first fitted parametric incidence and survival models then used the modelled incidence and survival estimates to calculate complete prevalence. The estimated and projected prevalence incorporate past observed trends and take into account different assumptions about future survival trends. These models were validated against observed prevalence from the counting method.
Results: Based on data for 1996–2007, the number of men living with prostate cancer in NSW was estimated to rise by 59% to 73%, from 38,322 in 2007 to 60,910–66,160 in 2017. The increasing incidence rates and the ageing population were the major contributors to this estimated increase. Validation suggested that these projections were reasonable, as the estimated prevalence in 1996–2007 was in good agreement with the corresponding prevalence calculated using the direct counting method, and the incidence models were supported by the recent data on prostate-specific antigen testing.
Conclusions: As the number of men living with prostate cancer is expected to increase dramatically in the next decade in Australia, representing a significant challenge to the health system, careful planning and development of a healthcare system able to respond to this increased demand is required. These projections are useful for addressing the challenge in meeting the cancer care needs of men with prostate cancer.

1. Introduction

Prostate cancer has become the most frequently diagnosed cancer among men in developed countries around the world [1], and will likely remain so in the foreseeable future [2]. It is a major burden on health services in most high income countries, although it can be difficult to accurately assess the full extent of this burden. While it is fortunate that most prostate cancer patients live with the disease for many years after diagnosis, this does mean that the traditional cancer surveillance measures of incidence and mortality, which cover only the two extreme ends of the disease spectrum (diagnosis and death), are insufficient measures of the true magnitude of the disease burden in a given population. In this regard, cancer prevalence – defined as the number or proportion of people alive in a population at a given date who have been diagnosed with the disease – provides information that is crucial to

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the planning and provision of health services required by patients in the period following primary treatment and before death from prostate cancer.

There are, however, some significant challenges in determining the true prevalence of prostate cancer. Population-wide data used to estimate prevalence usually lag 3 to 5 years behind the current year due to the time required for data collection, compilation, and dissemination [3]. Moreover, prevalence estimates using observed data from cancer registries can only provide limited duration prevalence (e.g. 1-year prevalence or 5-year prevalence), as the majority of population-based cancer registries in the world have not been established long enough to capture all prior cancer diagnoses [4]. Thus, an estimate of the complete prevalence is often derived from observed data using statistical models, which can also be extended to estimate future prevalence.

Estimating future prostate cancer prevalence is particularly complicated due to the changes in patterns of incidence and survival that have occurred since the introduction of prostate-specific antigen (PSA) testing in the late 1980s [5]. These marked changes mean that historical data are a relatively unreliable foundation for modelling prevalence. In a recent report comparing the four most widely used age–period–cohort (APC) models to project cancer incidence in Canada, none of the approaches was found to work well for prostate cancer [6]. As a result of this, some authors avoided including prostate cancer [7] when they predicted future cancer incidence for major cancer types due to the uncertainty in the projections.

In this study, we used a valid PIAMOD (Prevalence and Incidence Analysis MODel) method [8] and data from an Australian population-based cancer registry to estimate the future prevalence of prostate cancer in the state of New South Wales (NSW).

2. Methods

The software we used in this study, PIAMOD [8], estimates and projects cancer prevalence as a function of modelled incidence and survival estimates. A more detailed description of the methods for using PIAMOD software to estimate future cancer prevalence can be found in previous publications [9,10]. In brief, the process of using the software to estimate prevalence involves three principal steps: modelling incidence (by fitting APC models to obtain incidence projections), modelling survival (by fitting a mixture cure model), and then the estimation and projection of complete prevalence. These steps are illustrated in Fig. 1, and the data and methods involved in each of these steps will be described in detail below.

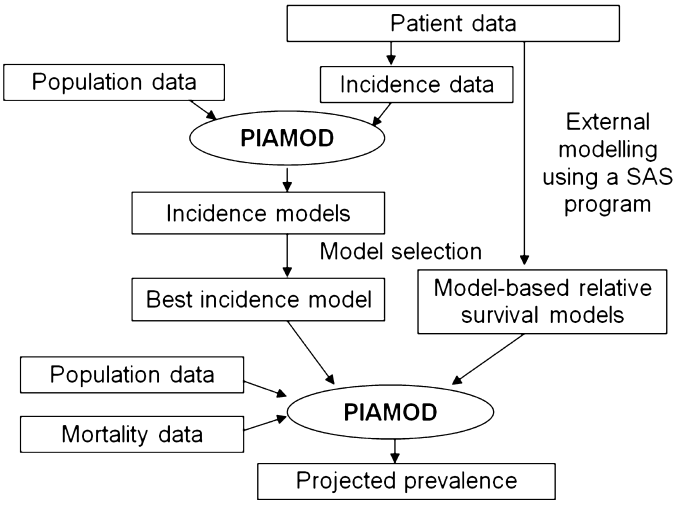

Fig. 1. Flow chart showing the use of PIAMOD for estimating future cancer prevalence.

2.1. Data

Incidence data for first primary prostate cancer (ICD-O3 C61) [11] diagnosed in 1972–2007 were extracted from the NSW Central Cancer Registry database. The Registry covers a population of 7.2 million people, approximately one-third of the national population of Australia, and maintains a record of all cases of cancer diagnosed in NSW residents since 1972 [12]. The Registry generally has high standards of data completeness and quality, and the data are accepted by the International Agency for Research on Cancer for publication in Cancer Incidence in Five Continents [13,14]. We included cases aged 18–84 years at diagnosis and excluded cases who were reported to the registry through death certificate only (DCO), or who were first identified post-mortem. The proportion of DCO cases in the Registry, an indicator of the quality of the cancer registry, is generally low (1.0% for 1993–1997 [15] and 0.9% for 2004–2008 [16]). Other input data required by the PIAMOD software were obtained from the Australian Bureau of Statistics (ABS): all-cause mortality data for NSW by single year of age and year (1972–2007), and the corresponding mid-year NSW residential male population data by single year of age and calendar year. Data on PSA tests performed (1996–2012) from Medicare Australia were used as a complementary validation for the fitted incidence models [17].

Cases were followed up for survival status to 31 December 2007 through record linkage of the cancer cases in the Cancer Registry with the death records from the NSW Register of Births, Deaths and Marriages and the National Death Index. 2007 was the most recent year for which follow up data were available. This significant lag was caused by the ABS reviewing its processes for release of its data including cause of death (http://www.cancer-institute.org.au/data-and-statistics/accessing-our-data/availability-of-nsw-central-cancer-registry-data#death-why-2008).

2.2. Ethics statement

This study involves analysis of routinely collected data and the records were de-identified (name, address, date of birth had been removed) before being provided to the research team. As a large proportion of the individuals would likely have moved or died since their diagnosis of cancer, which could have been up to 40 years ago, it would have been impracticable to seek consent, and thus the NSW Population and Health Service Research Ethics Committee waived the conditions for consent and approved the study (reference number: 2009/03/139).

2.3. Modelling incidence

Incidence rates are one of the main factors in predicting future cancer prevalence. Prostate cancer incidence changed dramatically in Australia with the introduction of PSA testing in the late 1980s. A sharp initial increase in the early 1990s (which peaked in 1994 at 187 cases per 100,000 men) was followed by a fall of 10% per year to a minimum of 126 per 100,000 men in 1998, and then another increase during 2001–2005 [18]. This incidence pattern poses a statistical challenge in terms of accurately projecting future incidence trends. While incidence data are available for 1972 onwards we chose to follow the approach used by previous researchers [19] and modelled incidence using data from 1996 to 2007. Selecting this period potentially helps reduce some of the impact of the introduction of PSA testing on our incidence models.

APC models can be fitted using different approaches, including the generalised linear model (GLM), the generalised additive model (GAM) and the Bayesian model. We used the GLM polynomial method in this study, implemented using the PIAMOD software [8]. This approach considers age, year of diagnosis and birth cohort as continuous variables, and uses a smoothing method, a polynomial function, to fit the data. In the polynomial method, age, period and cohort trends are modelled by log-linear regression for the mean parameter of the Poisson distribution. For our analysis the linear term for period was excluded when estimating the parameters in order to avoid the collinearity due to the linear relationship between age, period and cohort [8]. The regression coefficients were obtained using the maximum likelihood method.

The parameters of the APC models were estimated using observed incidence for 1996–2007 and then this model was used for forward (after 2007) and backward (before 1996) projections. The resulting fitted incidence estimates were used as inputs for estimating future prevalence (for 2008–2017). The most appropriate model was selected based on the likelihood ratio statistic (LRS) combined with knowledge of the epidemiology of prostate cancer in Australia because '. . .forecasting is not possible without sufficient knowledge of the epidemiology of a given cancer. . .' [20].

2.4. Modelling survival

A two-step procedure was used to model survival. First, relative survival was estimated and tabulated, and then a mixture cure model was fitted to the tabulated relative survival estimates. Survival relative to the general population was calculated in this study because we used all-cause mortality data from a population-based cancer registry. We derived tabulated relative survival for prostate cancer from incidence and follow-up data using the Pohar–Perme method [21]. In line with the modelling of incidence we also used data for the period 1996–2007 for modelling survival. This period was chosen because survival patterns from the earlier period of 1972–1995 were markedly different from the survival patterns that occurred after the introduction of PSA testing, and the pre-PSA testing patterns would be unlikely to occur again in the future.

We modelled these tabulated relative survival estimates with a mixture cure model [22], which assumes that patients can be divided into two distinct groups based on their prognosis: those who are cured and those who are not. The definition of 'cure', as used in this context, is that 'cured' patients have a mortality rate equal to that of people of the same age and sex in the general population [22]. The model parameters are then the proportion cured and the scale and shape parameters of the Weibull distribution, which was assumed to describe the survival distribution for the uncured patients. The model parameters were estimated by means of a non-linear regression procedure using PROC NLIN (SAS code provided by Roberta De Angelis) and taking the inverse of the variances of the observed survival as weights [22]. In this study, the Weibull mixture model was stratified by age group (18–64, 65–74 and 75–84 years) so parameters were estimated for each age stratum at the reference year of diagnosis (central point of the period 1996–2007), and the period effect varied by age.

The modelled survival estimates were extrapolated backward to 1972 and forward to 2017, using the assumption that the survival trends would be dynamic and would have the same 'slope' as that for the 1996–2007 observed data. The model-based estimates of survival from this mixture cure model were used as inputs into PIAMOD for the next step of the analysis.

2.5. Prevalence projections

Prostate cancer prevalence was then estimated using PIAMOD software with the modelled prostate cancer incidence, model-based relative survival, and all-cause mortality as inputs. Because PIAMOD can only provide results for closed age groups and populations, our prevalence estimates include cases up to age 84 years only. Further details on the PIAMOD method have been previously described by Mariotto et al. [23]. The PIAMOD prevalence estimates for 1996–2007 were validated by comparing the estimated prevalence with the limited duration prevalence estimates derived by counting the number of cases diagnosed between 1972 and 2007 [24], which is considered to be the most reliable estimate for populations covered by a registry for a sufficient length of time [25].

2.6. Sensitivity analyses

Further analyses were also undertaken to investigate the effects of different assumptions regarding future incidence rates and survival on the estimated future prevalence. First, we estimated the future prevalence by choosing two different APC incidence models. Second, we estimated prostate cancer prevalence based on the assumption that the future survival rate will remain constant at the level observed in 2007 rather than continuing to follow the trends in the observed data. Third, we repeated the primary analyses using data with longer survival follow-up (1990–2007) to assess the sensitivity of the cure model to the length of follow-up and its impact on the projected prevalence. Finally, we assumed that there would be no changes in incidence rates in future years, so the 2007 age-specific prevalence rates were applied to the projected 2017 age-specific male population in NSW. This gave an estimate of the prevalence in 2017 due to population growth and ageing only.

3. Results

A total of 49,866 cases of first primary prostate cancer were diagnosed in 1996–2007 in NSW. After excluding 2685 cases aged 85 years or over, 47,181 cases (median age: 69 years) were included in the incidence and prevalence analyses.

3.1. Incidence models

Six relatively simple APC models (APC101, 102, 201, 202, 103, and 301) were plotted against the observed incidence and are shown in Fig. 2. To select which models were most appropriate for predicting future incidence trends beyond the available data for NSW (to 2007), the projected incidence trends from these models were compared to more recent observed incidence data for prostate cancer from similar jurisdictions: data from another Australian state up to 2012 [26], and from the United States [27] and New Zealand [28]. All these data suggest that from around 2008–2010 prostate cancer incidence tended to stabilise or even fall. In addition, NSW data on PSA testing have also shown a trend towards a reduction in the number of PSA tests being performed (Fig. 3), which in turn is likely to result in a slower increase in prostate cancer incidence [29]. Thus the APC models 102 and 103, which showed slower increasing incidence trends, were considered to be the most appropriate models with which to project incidence for 2008–2017 (Fig. 2). The model-fit-statistics (Appendix) also suggest that these two models are a reasonably good fit to the observed incidence data.

3.2. Survival models

Five-year prostate cancer relative survival trends (assuming dynamic survival beyond the observed data window) by year of diagnosis are shown in Fig. 4. It can be seen that the fitted values for five-year relative survival were in close agreement with the observed data for the period 1996–2007.

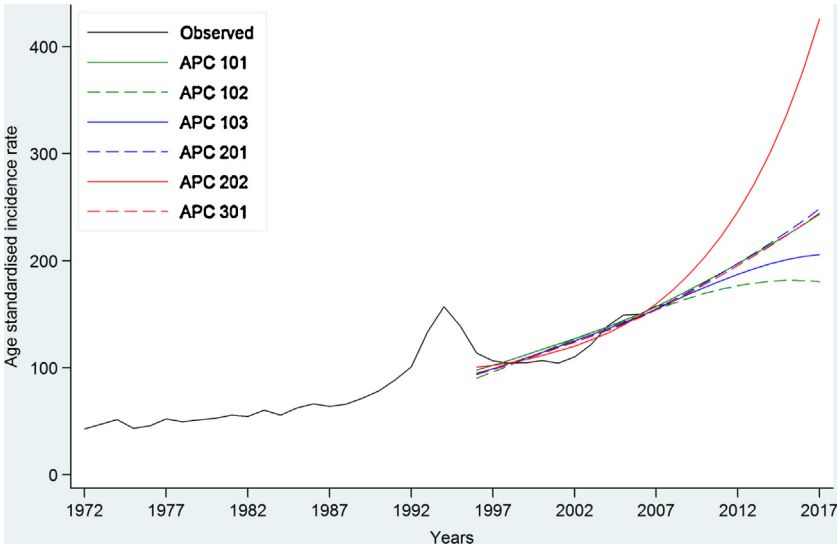

Fig. 2. Comparison of age–period–cohort incidence models and observed incidence rates for prostate cancer in NSW, Australia.

3.3. Projected prevalence

Based on these data, the number of men with prostate cancer in NSW is projected to rise by approximately 59% to 73% in 10 years: from 38,322 in 2007 to 60,910–66,160 patients in 2017 (Table 1). Fig. 5 shows the PIAMOD prevalence estimates based on data from 1996 to 2007 (with APC 102 and 103 incidence models) as well as the observed prevalence (to 2007). We observed (Fig. 5) that the PIAMOD estimates (1996–2007) were in good agreement with the prevalence (1996–2007) calculated from the direct counting method using observed data from 1972 to 2007.

For the sake of brevity, we only present sensitivity analysis results from incidence model APC 102 here (Table 2). It suggested that the impact of variations in survival (either assuming a constant trend or using a longer time series) on the projected prevalence was limited (2%). This is due to the high survival for prostate cancer, which means there is very little room for further improvement. If the effect of the growth in the ageing population is solely considered, prostate cancer prevalence is estimated to increase by 26% from 2007 to 2017.

4. Discussion

This study used population-based cancer registry data and the PIAMOD software to model current and future prostate cancer prevalence in NSW. Our results indicated that in the 10 years from 2007 to 2017 the number of men living with a diagnosis of prostate cancer in NSW is likely to rise by between 59% (low bound) and 73% (high bound). We found that for the period 1996–2007 there was close agreement between the fitted and observed prevalence, so we believe that our predictions are reasonable and that the true future prevalence is likely to lie between these two predictions. We would however, like to emphasise that there is considerable uncertainty in these projections, and that our models are likely to become less accurate as time passes, especially considering how

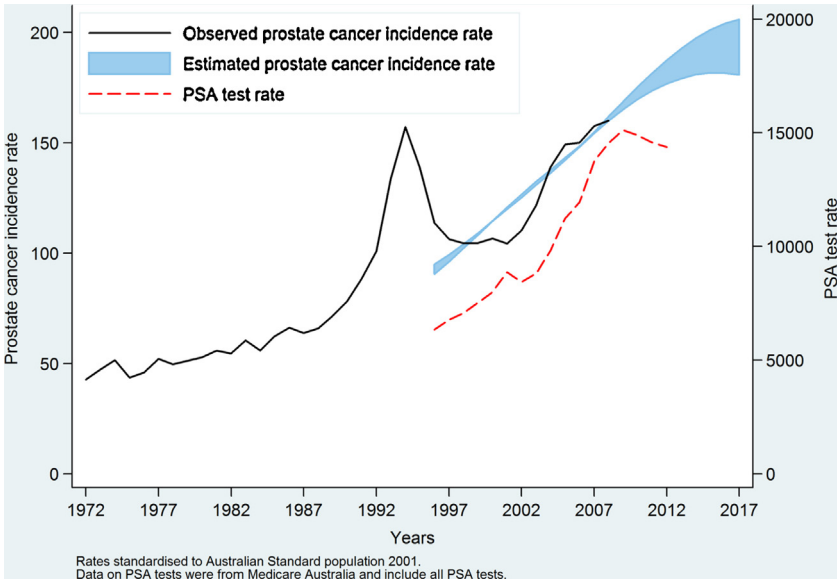

Fig. 3. Estimated age-standardised prostate cancer incidence rates (1972–2007), and projected incidence rates (1996–2017) and age-standardised rates of PSA testing (1996–2012) in NSW, Australia.

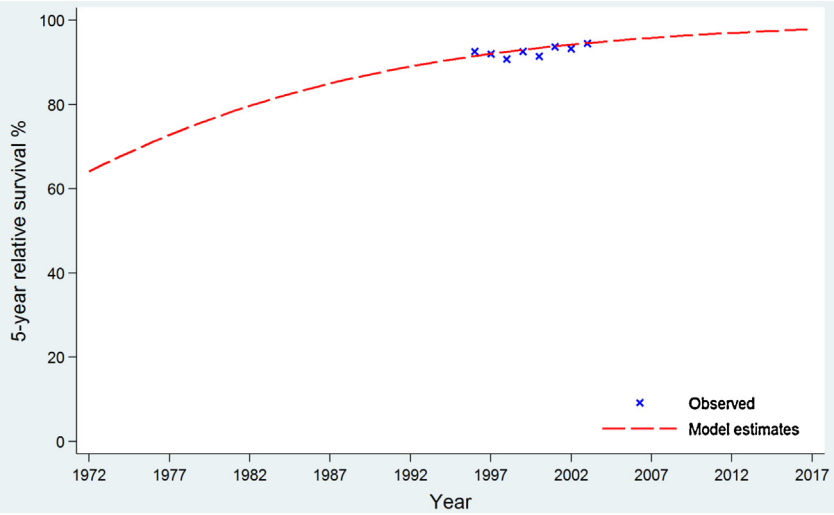

Fig. 4. Comparison of fitted five-year relative survival with observed survival data for prostate cancer in NSW, Australia.

Table 1
Estimated (2007) and projected (2017) prevalence of prostate cancer in NSW, Australia.

2007 (Baseline) estimate	Projected prevalence estimates in 2017 and percent increase from baseline			
	Lower bound[a]	% Increase	Upper bound[b]	% Increase
38,322	60,910	59%	66,160	73%

[a] Based on incidence model APC 102.
[b] Based on incidence model APC 103.

difficult it is to predict the future trends in prostate cancer testing behaviours.

Despite the difficulties involved, several previously published international studies have estimated future prostate cancer prevalence. Gatta et al. [30] used the completeness index method with data from 76 European cancer registries, and reported a 17.6% increase in prostate cancer prevalence from 2003 to 2010 due to demographic changes (which is a very similar result to our estimates of increase in prevalence due to population changes only: 2.5% vs 2.6% annual increase). An American study by Mariotto et al. [31] used the PIAMOD method [8] and data from 9 SEER registries, and estimated a 41.3% increase in prostate cancer prevalence from 2010 to 2020. Given the different methods, time periods and data used in these studies it is very difficult to make any direct comparisons between these studies and ours, but it does seem at least that the prediction of an increasing trend in prostate cancer prevalence is common amongst studies in developed countries.

Several Australian studies of cancer prevalence [32–34] provided estimates of limited duration prevalence. This means that they reported the number of people alive at the date of interest with a diagnosis of cancer within a past specific number of years (e.g. 2, 10, or 25 years). Our 36 year prevalence estimate at 2007 was comparable to those reported by the Cancer Institute NSW [33] and the Australian Institute of Health and Welfare (AIHW) [34], although different time periods were used in these studies. This close agreement is not surprising, as the results were based on the same (Cancer Institute NSW report) or similar (AIHW report) data sources. However, our study extended these results by providing estimates of the future prevalence that are more useful for health service planning for prostate cancer patients. Extrapolating these projections to the national population would equate to about 185,700 to 201,700 men living with prostate cancer in Australia in 2017, representing a significant challenge to the

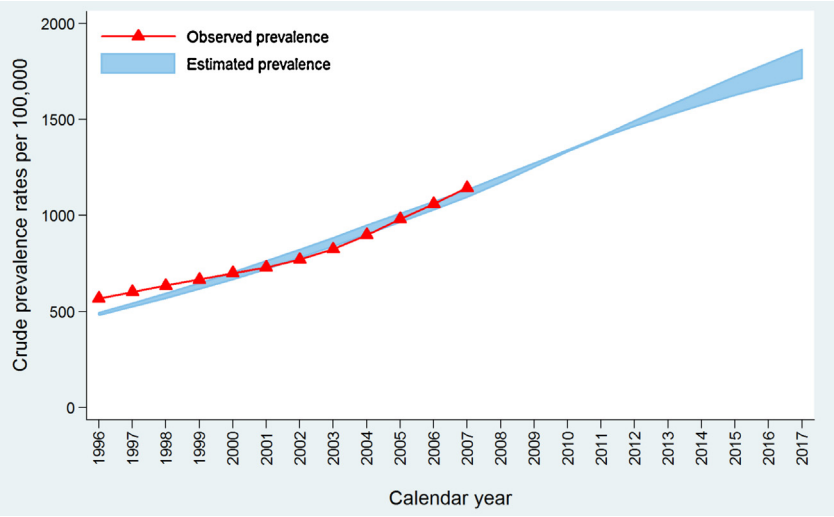

Fig. 5. Comparison of PIAMOD estimated complete prevalence and observed data for prostate cancer in NSW, Australia.

Table 2
Impact of various factors on the projected prevalence of prostate cancer in 2017, NSW Australia.

2007 (Baseline) estimate	Projected number of prevalent cases (2017) using different assumptions[a]					
	Assuming constant survival trend	% Change	Based on data for 1990–2007	% Change	Based on population change[b] only	% Change
38,322	60,313	57%	60,163	57%	48,269	26%

[a] Incidence model APC 102 was used in these analyses.
[b] Population projections from the Australian Bureau of Statistics (2007).

Australian health system. Moreover, these projections are likely to be underestimated because we did not include cases aged 85 years or over in the estimation and those with second primary prostate cancer. There would be approximately 6922–7518 men aged 85 years or older living with prostate cancer in NSW in 2017 if we apply the proportion of cases in this oldest group in 2007 (for which we have data) to the projected 2017 total prevalence in NSW. The corresponding national figure would be between 21,103 and 22,922 prevalent cases aged over 85 years; this highlights the importance of including the elderly population in studies of cancer survivors.

The advantage of the method we used is that the models for incidence and survival were fitted separately, similar to the method adopted by Crouch et al. [35], with the estimates of future prevalence being obtained as a product of both. This method allows us to construct and evaluate different scenarios regarding future incidence and survival, as well as population ageing, and to model their effects on future prevalence estimates.

Our models are based on both statistical and clinical foundations. First, most projection models for prostate cancer are likely to be poor due to the fluctuation in incidence caused by the introduction of PSA testing in the late 1980s [6] and this would inevitably lead to biased estimates of both incidence and prevalence. Hence, from a statistical point of view it is well justified to exclude the period where the data fluctuate considerably, as has also been done by other researchers [19]. As a general rule, it is more appropriate to separate the patient population into a set of homogeneous groups and exclude those groups that add little value to the modelling of future incidence and prevalence. Therefore, the more recent data have greater predictive power than much older data, and in the case of prostate cancer this is particularly important, as the introduction of PSA testing has significantly altered the way in which disease first presents and its subsequent management. Modelling the recent data allows for a more clinically relevant prediction of future prevalence.

Projections of cancer prevalence depend on the estimates and assumptions of future trends in incidence and survival. We found that the projected incidence trend was the most important factor in estimating future prostate cancer prevalence, as was shown in the results, with the estimated prevalence being very sensitive to the incidence models selected. For example, if we chose incidence model APC 202, which had the lowest LRS (Appendix), this would result in a 164% increase in prostate cancer prevalence by 2017 (rather than 59–73%), which we believe to be highly unlikely. This demonstrates that it may be risky to select the incidence model based on statistical assessment criteria only, and shows why an understanding of the epidemiology of the cancer under study is extremely important for the selection of the most appropriate models for predicting future trends.

Based on the models we selected, there would be much slower growth in the incidence rates of prostate cancer in the period 2008–2017 than was seen from 2001 onward, and it tended to flatten out near the end of the period. Based on our understanding of the driving forces behind changes in prostate cancer incidence and independent data from similar jurisdictions [26–28], we believe these assumptions are reasonable. Many studies show an association between the level of PSA testing and prostate cancer incidence [18,29,36], so the trend in PSA testing should provide some insight into the future trends in incidence. Recent data from the Medicare Benefits Scheme (Australia's universal health care scheme) suggested that the peak of PSA testing in NSW may have been reached, although it still remains at a very high level (Fig. 3). However, it is as yet unclear how patterns of future prostate cancer testing in Australia will be affected by recent recommendations such as those of the U.S. Preventive Services Task Force against routine PSA screening for men of any age [37], and the American Urological Association's more conservative recommendations to only test men aged 55 to 69 [38]. Evidence from the USA indicates that the impact is likely to be minimal [39,40].

Age is another important factor in predicting prostate cancer incidence patterns, as the risk of diagnosis increases exponentially after age 50. The number of men aged over 65 years in Australia is projected to increase from 1.23 million in 2006 (representing 12.1% of the total male population) to 1.78 million (15.7% of the total male population) in the year 2016 [41]. This ageing of the population will inevitably lead to a significant increase in the number of men diagnosed with prostate cancer. In regards to the extent of this increase, incidence projections from Canada and the USA may provide some insight into future Australian trends, as the population age structures of these countries are broadly similar to that of Australia [41]. The incidence in Canada was projected to increase by 39% from 2009 to 2021 due to population ageing only [2], while the corresponding increase in the USA is estimated to be 30% over the period 2010 to 2020 [42]. Our projected increase due to population changes only (26% from 2007 to 2017) is similar to the estimates from Canada and the USA.

In contrast to the selection of the incidence models, different assumptions of trends in future survival had less of an impact on the estimated future prevalence than was seen with changes in incidence rates and population ageing (Table 2). This may be because survival rates for prostate cancer are already very high (five year relative survival being over 90%). Therefore, a less robust assumption (of cure) would not have too great an impact on the predicted prevalence. Sensitivity analysis indicates that the predicted prevalence estimates were not sensitive to the length of the survival follow-up.

Cancer prevalence is a function of incidence, survival and population ageing. It is challenging to completely separate the contribution of each of these factors, as it is not simply an additive relationship. For example, an increased incidence of PSA detected cases will likely lead to prolonged survival even if no improvements in treatment occur. Nevertheless, we conducted sensitivity analyses designed to investigate the sensitivity of the estimated future prevalence to different factors. Compared to 2007, our results indicate that population ageing and growth only would result in an increase of around 26% in the number of men living with prostate cancer in 2017 (assuming incidence and survival remain static for the period 2007–2017). This also implies that the remaining predicted increase in prevalence for 2017 would be caused by the increased incidence rates and improved survival. As

discussed previously, changes in survival patterns would only contribute about 2% to the change in the estimated prevalence in 2017, and thus population ageing and increased incidence rates should be the major contributors to the future increase in prevalence. Although these results are consistent with previous international studies [2,42], we would emphasise that it is extremely difficult to accurately isolate the effects of individual factors, and the results of this sensitivity analysis only provide a rough indication of the contribution of each of these variables.

The method used here has several limitations that must be considered if it is to be applied to other data. As these prevalence projections are based on assumptions about incidence and survival, any future changes in the methods used to undertake prostate biopsy or changes to PSA testing thresholds could have marked short-term effects, leading to unreliable projections. It is therefore important to emphasise that these projections are very sensitive to any future changes in prostate cancer diagnostic practices and the accuracy of the projections tends to decrease with time. Thus, we suggest that these projections should be updated as new data become available and assumption underpinning the projections be periodically reviewed. Also, integrating data on PSA testing trends and the method and number of biopsy cores taken with observed incidence data would have increased the accuracy of our model, but as registry data are not routinely linked with data on screening history, and the software we used does not allow for the integration of such factors, this was not possible. This may warrant further investigation in future modelling exercises, as the model accuracy could be significantly increased by incorporating data regarding important predictors of incidence [43]. The final limitation of our methods is that the assumption of cure is not always reasonable for prostate cancer because of its high survival rate [44]. When cure does not occur, the mixture cure model used here provides an approximation only [22]. However, as previously described, we found that a less robust assumption of cure would only have a small impact on the predicted prevalence.

Despite these limitations, this study does provide a more complete measure of the future burden of prostate cancer in Australia than has previously been available. With an increasing number of men being diagnosed with prostate cancer and living for many years after diagnosis, this is an area of research of critical importance for the planning of healthcare services. Despite its high prevalence (about 20% of the total cancer survivor population), research on prostate cancer survivors is relatively rare, with only 5% of all studies of cancer survivors having a specific focus on these men [45]. By comparison, breast cancer survivors have been the focus of 40% on studies of cancer survivors, despite representing a similar proportion of the survivor population as prostate cancer survivors (22%) [45]. As prostate cancer prevalence is expected to increase dramatically in the next decade, it is important that more is known about this population and that reliable projections of the future burden are available. It is hoped that this study will begin to address some of these issues and will help enable an evidence-based approach to health-care planning and service delivery.

Conflict of interest statement

The authors declare that they have no conflict of interests.

Authorship contribution

XQY and DOC conceived the project; DPS and MSC assisted with further refinement of the project. XQY obtained the cancer registry data, QL performed the data analysis, and XQY provided oversight of the data analysis with inputs from DOC, DPS and MSC. XQY drafted the manuscript with help from QL for Section 2 and 3. DOC, DPS, MSC and QL revised the manuscript. All authors read and approved the final version of the manuscript.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.canep.2014.11.009.

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