Projections of the future burden of cancer in Australia using Bayesian age-period-cohort models

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Abstract

Background

Accurate forecasts of cancer incidence, with appropriate estimates of uncertainty, are crucial for planners and policy makers to ensure resource availability and prioritize interventions. We used Bayesian age-period-cohort (APC) models to project the future incidence of cancer in Australia.

Methods

Bayesian APC models were fitted to counts of cancer diagnoses in Australia from 1982 to 2016 and projected to 2031 for seven key cancer types: breast, colorectal, liver, lung, non-Hodgkin lymphoma, melanoma and stomach. Aggregate cancer data from population-based cancer registries were sourced from the Australian Institute of Health and Welfare.

Results

Over the projection period, total counts for these cancer types increased on average by 2.7% annually to 100,385 diagnoses in 2031, which is a 50% increase over 2016 numbers, although there is considerable uncertainty in this estimate. Counts for each cancer type and sex increased over the projection period, whereas decreases in the age-standardized incidence rates (ASRs) were projected for stomach, colorectal and male lung cancers. Large increases in ASRs were projected for liver and female lung cancer. Increases in the percentage of colorectal cancer diagnoses among younger age groups were projected. Retrospective one-step-ahead projections indicated both the incidence and its uncertainty were successfully forecast.

Conclusions

Increases in the projected incidence counts of key cancer types are in part attributable to the increasing and ageing population. The projected increases in ASRs for some cancer types should increase motivation to reduce sedentary behavior, poor diet, overweight and undermanagement of
infections. The Bayesian paradigm provides useful measures of the uncertainty associated with these projections.

Key words

Neoplasms; Incidence; Forecasting; Models, Statistical; Australia

Abbreviations

ABS: Australian Bureau of Statistics

AE: Absolute error

AIHW: Australian Institute of Health and Welfare

APC: Age-period-cohort

ASR: Age-standardized incidence rate

CI: Credible interval

CRPS: Continuous ranked probability scores

HBV: Hepatitis B virus

HCV: Hepatitis C virus

*H. pylori*: *Helicobacter pylori*

INLA: Software for Bayesian modelling (Integrated Nested Laplace Approximation)

PAF: Population attributable fraction
1. Introduction

In Australia, 2% of the population are alive following a cancer diagnosis within the last 5 years and
over 145,000 cancer diagnoses are expected in 2020. (1) Quantitative estimates of future incidence
are needed to guide planning, policy development and resource management to meet demand 15
years in the future. It is also important to convey the uncertainty associated with future
projections. (2)

A range of statistical methods are available for projecting future cancer burden, including
generalized linear models, (3, 4) Joinpoint regression (5) and age-period-cohort (APC) models. (6, 7) In
APC models, age, period and cohort effects reflect latent processes, such as changes in risk factor
prevalence, interventions or diagnostic practices. Hence, estimates of the future burden of cancer
may be obtained without projections of the latent processes. There is flexibility in the way APC
models are fitted, with splines, power functions and Bayesian random effects models all finding
application. (4, 8-10)

When projecting cancer burden, uncertainty arises from several sources including uncertainty in the
fitted parameters, random year-to-year variation in the number of diagnoses and uncertainty in the
various assumptions inherent in the model. (2) Many published projections are limited in how
uncertainty is conveyed, with some lacking any confidence interval (7) and others missing at least
one component of uncertainty. (3, 6)

Bayesian APC models have been developed for projecting cancer incidence and mortality. (11)
Modelling uncertainty is intrinsic to the Bayesian paradigm and its credible intervals (CrIs) are
intuitive and readily understood by diverse audiences. Moreover, the Bayesian framework facilitates
hierarchical models, which have been used to address some identifiability problems associated with
APC models. (8) Bayesian APC models have not previously been applied to cancer incidence in
Australia, but examples from the literature have provided promising results. (8, 12, 13)
This study used Bayesian APC models to project the incidence of seven of the most common cancer types in Australia, 15 years beyond the most recent data.

2. Methods

2.1 Data

Data on primary cancers diagnosed for seven key cancer types were downloaded from the Australian Institute of Health and Welfare (AIHW) Australian Cancer Database. Counts were available for each year from 1982 to 2016, for both sexes and for 5-year age groups from 0-4 years to 85-89 years. The model assumes that the population is uniformly distributed within each age group, which was not true for the 90+ age group.

ICD-10 codes were used to identify cases of colorectal (C18-C20), liver (C22), lung (C33-C34), melanoma (C43), non-Hodgkin lymphoma (C82-C86), stomach (C16), prostate cancer (C61) and female breast cancer (C50). These cancer types are among those with the highest incidence in Australia. Liver cancer was included because it is projected to have incidence rates similar to non-Hodgkin lymphoma by 2031. Moreover, 5-year relative survival rates for liver cancer are among the poorest of all cancers. Stomach cancer is unique since its rates have steadily decreased for decades.

Historical and projected population data were obtained from the Australian Bureau of Statistics (ABS) with projected populations based on assumptions of medium fertility, life expectancy and migration, as defined by the ABS.

2.2 Statistical modelling

Bayesian APC models were fitted using the INLA package (Version 20.3.17) in R (Version 4.0.0), using a sex-specific APC model (Equation 1). The number of cases, \( y_{ij} \), for each age group \( i \), period \( j \) and sex \( s \) was modelled as a Poisson process with mean the product of the population at risk, \( N_{ij} \), and the estimated rate. The log of the rate, \( \eta_{ij} \), was estimated as a linear
combination of a sex-specific intercept, \( \mu_s \), an overdispersion effect, \( z_{ijs} \), and the age, period and cohort effects, respectively \( \alpha_{is} \), \( \beta_{js} \) and \( \gamma_{ks} \), where \( k = M(I - i) + j \) is the birth cohort, \( M \) is the number of periods per age group and \( I \) is the number of age groups.

\[
\begin{align*}
    y_{ijs} & \sim \text{Poisson}(N_{ijs}\exp(\eta_{ijs})) \\
    \eta_{ijs} & = \mu_s + \alpha_{is} + \beta_{js} + \gamma_{ks} + z_{ijs}
\end{align*}
\]

Equation 1

Second-order random walk priors were applied to the age, period and cohort effects, with sum-to-zero constraints. Log-gamma priors were applied to the precision parameters with scale and shape parameters of 1 and 0.00005 for each of the age, period and cohort effects and 1 and 0.005 for the overdispersion effect. Overdispersion parameters were given Gaussian priors with mean zero. The posterior distributions of the age-standardized incidence rates (ASRs) were calculated using the 2001 Australian standard population as per Riebler and Held (13). Projected counts were calculated using the projected Australian population.

2.3 Additional measures

In the Bayesian paradigm, it is possible to use a normal approximation of the posterior distribution of the modelled count or rate, \( \tilde{\theta} \), to calculate \( P(\tilde{\theta}_{j+t} \geq m\tilde{\theta}_j) \), the probability that a projected estimate in year \( j + t \) is greater than the modelled value in the final year of observed data \( j \), by some margin \( m \).

The age distribution for each cancer was described by calculating the annual percentage of the projected counts that would fall in each of three age groups.

2.4 Model checking

Model fit and predictive accuracy were assessed by fitting one-step-ahead projections \( t = 1 \) for each year from 2002 to 2016.(13, 20) The absolute error (AE) between the observed and projected counts were calculated as were the continuous ranked probability scores (CRPS) using the
ScoringRules package. The CRPS is essentially the absolute error after accounting for normal variability (noise). The accuracy of the modelled uncertainty in the projections was assessed using calibration tests (based on the mean CRPS) and the coverage probabilities, which in the Bayesian paradigm are the proportion of observations that fall within the prediction intervals. The z-statistic arising from the calibration test has standard normal distribution. Negative values of the z-statistic combined with small p-values indicating over-dispersion of the projected CrIs compared with the data, while positive values indicate under-dispersion. Other model checking methods and results are provided in Supplementary material A.

3. Results

Age- and sex-specific trends in the observed incidence rates for each cancer type (Figure 1) demonstrate that rates for age groups less than 15 years old were comparatively negligible. Observed rates were lower for females than males across all cancers, age groups and birth years. Generally, incidence rates increased with increasing age group, except among the oldest age groups for certain cancer types. Differences by sex, notably for lung cancer, support the use of sex-specific models for these analyses.

Figure 1: Observed incidence rates for each cancer, plotted versus birth year by sex and age group (Note different scales on the vertical axis for each cancer type).

Measures of model fit and predictive accuracy are presented in Table 1. Both the mean CRPS and AE were very low compared to the observed annual incidence counts (Table 2). According to the CRPS test, there was no evidence of miscalibration in the projections for liver cancer, lung cancer, melanoma or non-Hodgkin lymphoma. There was evidence of under-dispersion in the modelled estimates for breast and colorectal cancers and over-dispersion for stomach cancer. This means that the credible intervals may underestimate the uncertainty for breast and colorectal cancers and overestimate uncertainty for stomach cancer. However, the coverage probabilities indicate the miscalibration is small in magnitude at the 95% level.
3.1 Age-standardized rates

Modelled and projected ASRs for each cancer by sex are plotted in Figure 2 with the observed ASRs. CrIs calculated for the prostate cancer projections were un informatively wide. Hence, the results for prostate cancer are reported only in Supplementary material B (Figures B13-B15).

Figure 2; Age-standardized incidence rates versus period by cancer type and sex. Observed rates are shown as circles. Credible intervals, reflecting the total variance in the observed rates, are displayed as colored bands, where the outer edges of the lightest colored bands reflect the 95% credible intervals, followed by the 90% credible intervals and the width of the remaining credible intervals decreasing incrementally by 10%. (Note different scales on the vertical axis for each cancer type)

Temporal trends in observed ASRs for female breast cancer, non-Hodgkin lymphoma and melanoma were similar: all increased rapidly initially and slowly more recently. Over the 15 years to 2031, it is uncertain whether the ASRs for these cancers will change or remain stable. The probability that ASRs in 2031 will be greater than in 2016 was 51% for female breast cancer, 59% and 61% for melanoma and 51% and 42% for non-Hodgkin lymphoma among females and males, respectively.

ASRs for stomach and colorectal cancers have seen recent decreases, which was projected to continue until the mid-2020s, when slight increases may occur. The probabilities that the ASRs in 2031 will be greater than in 2016 were 32% and 40% for colorectal cancer and 30% and 23% for stomach cancers among males and females, respectively.

Liver cancer ASRs have been increasing and were projected to continue increasing, with probabilities of 92% among males and 89% among females that the ASRs in 2031 will be greater than in 2016.

Lung cancer ASRs decreased for males and increased among women, with projections indicating that these trends were likely to continue in the same directions. The probability that the ASRs in 2031 will be greater than in 2016 was 67% for females but only 20% for males.

3.2 Counts

Modelled and projected counts in 2016 and 2031 for males, females and persons under 90 years of age are provided in Table 2. The total count for these cancer types combined was 67 055 cases in
2016 and was projected to be 100,385 diagnoses in 2031 - a 50% increase. Projected counts in 2031 were greater than 2016 for each cancer type and sex.

The probabilities that the 2031 counts will be the same as or greater than the 2016 modelled counts by a margin of at least 15, 50 or 100% are shown in Table 2. It is considered likely that incidence counts in 2031 for most cancer types modelled will be at least 15% greater than 2016 counts. Some cancers, such as female lung cancer and melanoma, are likely to be more than 50% greater and liver cancer is likely to at least double.

The percentage of counts occurring in each of three age groups are presented versus time in Figure 3 and age-specific incidence rates by sex are in Supplementary material B. The percentage of diagnoses occurring in the 70 – 89 age group range was lowest for breast cancer (36% in 2031) and highest for lung cancer (62% in 2031). Liver cancer was expected to have the greatest increase in the percent of cases in the 70 – 89 age group, from 40% in 2016 to 62% in 2031. By 2031, the percentage of persons aged 70 to 89 at diagnosis was projected to be over 40% for all but breast cancer.

The percentage of colorectal cancer cases among people aged under 50 is projected to increase from 10% in 2016 to 18% in 2031, with incidence rates projected to increase among both men and women aged under 50 years. Melanoma had the greatest decrease in the percentage of projected cases among persons aged under 50, from 20% in 2016 to 10% in 2031.

4. Discussion

The total number of diagnoses for the cancer types included in this study was projected to increase on average by 2.7% or 2000 cases annually. Some of these increases may be attributable to increasing and ageing populations since ASRs are projected to decrease over time for some cancer types, including stomach and male lung cancer. For breast cancer, historical increases in ASRs coincided with the broadscale availability of screening and improvements in diagnosis. However,
changes in exposures to risk factors may also impact observed and projected trends in incidence rates. The projected counts were consistent with AIHW forecasts (1) (Supplementary material A, Table A1).

Bayesian APC models have several advantages over other projection methods. Firstly, the CrIs more accurately represent the uncertainty. (13) The CrIs are intuitive to interpret and facilitate calculation of the probability of increased estimates. Implementing Bayesian APC models with the INLA package is less computationally expensive and avoids the pitfalls of Markov Chain Monte Carlo convergence.

While increases in breast cancer ASRs observed in the early 1990s may be largely attributable to the free breast screening program, (23) smaller increases before that may be from increases in sedentary behavior and the prevalence of being overweight. (24, 25) Other causative factors for breast cancer either have low population attributable fractions (PAFs) or the prevalence of the exposure has been decreasing. (26-29) Hence, the slow increases in ASRs projected for breast cancer is consistent with the continuing increase in population rates of physical inactivity and being overweight.

Projected shifts in the age distribution for colorectal cancer are consistent with the increasing prevalence of inadequate dietary fiber, increases in consumption of red and processed meats, being overweight and physical inactivity, (30, 31) each of which are known causative agents for colorectal cancer, (24, 25, 32, 33) and are increasing particularly among younger age groups. (30, 31, 34) The number of participants in the National Bowel Screening Program, which began in 2006, increased 2.5-fold between 2014-15 and 2018-19, before becoming available nationwide in 2020. (35) Hence, it is likely screening had minimal impact on the observed trends. Changes to participation rates may affect future incidence counts, although the screening program is designed to detect precancerous polyps as well as cancer.

Consistent with previous reports, (36) liver cancer incidence rates were projected to increase for the entire projection interval. A large majority of cases of liver cancer are attributable to modifiable factors, including Hepatitis B (HBV) or C (HCV) infection (37), tobacco smoke (38), alcohol...
consumption (39) and obesity (40). The observed increase in liver cancer rates has occurred despite recent decreases in rates of tobacco smoking (41) and excessive alcohol consumption (42).

Promisingly, HCV and HBV rates have declined in recent years and HCV treatment rates have increased 4-fold between 2015 and 2016 (43), which may result in lower liver cancer ASRs in the future, possibly within the projection window.

Population lung cancer incidence rates are strongly associated with the population’s tobacco smoking history up to 20 or 30 years earlier (3, 38, 44, 45) with 80% of cases attributable to tobacco smoking and a further 6% of cases occurring among people who have never smoked but have cohabited with a smoker (38). The changes in projected incidence over the next 15 years are consistent with observed smoking rates, with the prevalence of smoking, the annual number of cigarettes per capita and the per capita tar exposure decreasing steadily among men for many decades, particularly among younger cohorts (3). Among women, prevalence and per capita cigarette consumption have been decreasing since 1990, although very slowly among older age groups (3), which is consistent with the projected plateau in the lung cancer incidence among women in coming years.

The ASRs for melanoma were expected to continue increasing and there is a 50% probability that the number of melanomas diagnosed will more than double between 2016 and 2031. The burden of melanomas is primarily due to the effect of previous UV exposure, with nearly two-thirds of melanomas attributable to exposure to solar UV radiation (46). While recent decreases in incidence rates among younger cohorts have been observed (47), the overall trends are dominated by the higher and still-increasing incidence rates among older people. As the proportion of the population born after 1980 increases, the ASRs should start to decrease.

Non-Hodgkin lymphoma is caused by infectious diseases such as *Helicobacter pylori* (*H. pylori*), HCV and Human immunodeficiency virus (PAF from all infectious diseases was 3.6%), (37) as well as being overweight (PAF 4.3%) (37) and occupational exposures (48). The population of people employed in
industries with strong evidence of an association with non-Hodgkin lymphoma, such as agriculture and manufacturing, has steadily decreased in recent decades (49), which is consistent with decreases in observed age-specific incidence rates in younger age groups. Although incidence is projected to increase very slowly over the next 15 years, improvements in the incidence and treatment of infectious diseases (50) and reduced occupational exposures may result in decreases further in the future.

Our projections suggest the incidence rates of stomach cancer will continue to decrease, however with a gradual plateauing in later years. The continual decrease in stomach cancer ASRs observed globally for decades is a consequence of improved diets including more fresh fruit and vegetables and less preserved food and, more recently, reductions in H. pylori infection,(32, 37, 42) as well as reductions in tobacco smoking.(38, 40) Data from the ABS indicates that recently diets in Australia are including less fresh fruit and non-starchy vegetables, by both period and birth cohort.(30, 31)

The measures of error, the mean AE and CRPS, were both small compared to the total annual counts for each cancer, suggesting that the models fitted the data well for all cancer types. Although there is evidence of miscalibration in the projections for some cancer types, the 95% CrIs appear reliable. However, the calculated probabilities of an increase in estimates may be affected. The over-dispersion in the model for stomach cancer will result in an underestimate of the probabilities that ASRs or counts will be greater in 2031 than 2016, particularly where these probabilities are closer to 0.5. The under-dispersion in the models for colorectal and breast cancer will result in small overestimates of the probabilities.

4.1 Limitations

APC models account for trends in risk factors, without requiring measurements of exposures or even identifying the carcinogen. However, if large unforeseeable changes occur, for example broadscale decreases in exposures or new screening programs, then the projected rates may over- or underestimate the actual future burden. As with all projections, the key assumption with these models is
that the historical trends will continue in the future, and it is impossible to know to what extent this assumption is valid. Other assumptions built into the models are designed to reflect the historical patterns with greater accuracy, including the use of the log link function,(51) but it is likely these model-based assumptions would play a smaller role in the overall uncertainty. Populations with heterogeneous subpopulations and therefore heterogeneous changes in the underlying risk factors may be better described by a model that includes geographic or sociodemographic effects.(12)

Despite the wide uncertainty in projections beyond 5 or 10 years, governments require longer term projections for effective planning and policy development. The increased uncertainty provides a more realistic picture of the accuracy of projections, particularly when unforeseen interventions and changing circumstances are considered. The CrIs for melanoma were wide and those of prostate cancer were so wide that the projections were deemed uninformative. Rates of diagnoses for these cancer types are prone to fluctuation, particularly for prostate cancer, which fluctuated following the introduction of the Prostate Serum Antigen test (Figure 1). The large degree of uncertainty for these cancer types may result from these artefacts, as the model accommodates the possibility of further sudden changes.

The uncertainty in the projected counts does not include uncertainty in the population size, however there was less than 7% difference in the populations projected for 2031 by the ABS using lower and higher modelling assumptions.(16)

Because cohort is a linear combination of age and period, the effects are not identifiable and cannot provide statistical evidence of change in the incidence rates.(8) Nevertheless, the models provide information on the incidence and distribution of diagnoses across the population. We have addressed the lack of a time coefficient by calculating the probability that the number of diagnoses at time \( J + t \) will be greater than the number of modelled diagnoses at time \( J \).

There is evidence that the COVID-19 pandemic has impacted certain behaviours like alcohol and drug consumption(52) and screening for breast cancer(53). Moreover, the population projections...
are likely to overestimate immigration in 2020-2021. Diagnoses of breast cancer may be lower because of decreased screening rates between March and July 2020, however, this is likely to be a short-term anomaly. Increases in risk behaviours may result in increased cancer incidence beyond 2031. Decreases in migration may result in decreased numbers of diagnoses over the projection period. The potential impact of the unforeseen pandemic on future cancer incidence highlight the caution required when interpreting estimates of cancer projections.

5. Conclusions

These projections highlight the increasing burden of cancer in Australia over the next decade, with the incidence of some cancer types projected to more than double between 2016 and 2031. The ability to quantify these projections, combined with an appropriate measure of uncertainty, provides important information to guide planning and resourcing of cancer management and support services. Since our projections assume that previously observed age, period and cohort trends will continue, these results should motivate increased efforts to intervene and change these trends by reducing the prevalence of risk factors, such as poor diet, overweight or sedentary behavior. In addition, efforts to increase the proportion of cancers diagnosed early, by screening or greater awareness of symptoms, would serve to reduce the mortality burden associated with the increasing numbers of cases.

Acknowledgements

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### Tables

**Table 1:** Model predictive quality calculated from one-step-ahead predictions for males and females combined, including the mean continuous ranked probability score (CRPS), mean absolute error (AE), z-score and P-value for the calibration test\(^a\) and coverage probabilities\(^b\) for the 50, 80 and 95% credible intervals (CrI).

<table>
<thead>
<tr>
<th></th>
<th>Calibration test(^a)</th>
<th>Coverage probabilities(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CRPS</td>
<td>AE</td>
</tr>
<tr>
<td>Breast</td>
<td>29.5</td>
<td>41.4</td>
</tr>
<tr>
<td>Colorectal</td>
<td>14.4</td>
<td>20.6</td>
</tr>
<tr>
<td>Liver</td>
<td>3.3</td>
<td>4.8</td>
</tr>
<tr>
<td>Lung</td>
<td>9.4</td>
<td>13.1</td>
</tr>
<tr>
<td>Melanoma</td>
<td>13.9</td>
<td>19.5</td>
</tr>
<tr>
<td>Non-Hodgkin lymphoma</td>
<td>5.9</td>
<td>8.4</td>
</tr>
<tr>
<td>Stomach</td>
<td>3.2</td>
<td>4.5</td>
</tr>
</tbody>
</table>

**Notes:**

\(^a\) The calibration test determines whether the data-generating distribution is the same as the forecast distribution. The resulting z-statistic has standard normal distribution.\(^{(22)}\)

\(^b\) The proportions of observed values that fall within the prediction intervals calculated using the one-step-ahead projections between 2002 and 2016.
Table 2: Modelled and projected counts per year (95% credible interval) for each cancer and sex in 2016 and 2031 and the probability of an increase of at least 0, 15, 50 or 100% on 2016 numbers by 2031.

<table>
<thead>
<tr>
<th>Cancer</th>
<th>2016 Count (95% CI)</th>
<th>2031 Count (95% CI)</th>
<th>Probability of stated increase^a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>≥0%</td>
</tr>
<tr>
<td><strong>Breast</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>17 056 (16 702 – 17 411)</td>
<td>22 855 (5266 – 40 444)</td>
<td>0.74</td>
</tr>
<tr>
<td><strong>Colorectal</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>6607 (6397 – 6818)</td>
<td>8275 (4517 – 12 033)</td>
<td>0.81</td>
</tr>
<tr>
<td>Males</td>
<td>8168 (7932 – 8404)</td>
<td>9899 (5414 – 14 384)</td>
<td>0.78</td>
</tr>
<tr>
<td>Persons</td>
<td>14 775 (14 459 – 15 091)</td>
<td>18 174 (12 322 – 24 025)</td>
<td>0.87</td>
</tr>
<tr>
<td><strong>Liver</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>558 (502 – 614)</td>
<td>1229 (574 – 1884)</td>
<td>0.98</td>
</tr>
<tr>
<td>Males</td>
<td>1608 (1508 – 1708)</td>
<td>3515 (1748 – 5283)</td>
<td>0.98</td>
</tr>
<tr>
<td>Persons</td>
<td>2166 (2051 – 2280)</td>
<td>4744 (2859 – 6629)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td><strong>Lung</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>5087 (4906 – 5269)</td>
<td>8122 (4691 – 11 553)</td>
<td>0.96</td>
</tr>
<tr>
<td>Males</td>
<td>6688 (6478 – 6899)</td>
<td>8083 (4705 – 11 460)</td>
<td>0.79</td>
</tr>
<tr>
<td>Persons</td>
<td>11 776 (11 498 – 12 053)</td>
<td>16 205 (11 390 – 21 019)</td>
<td>0.96</td>
</tr>
<tr>
<td><strong>Melanoma</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>5824 (5617 – 6032)</td>
<td>11 513 (0 – 37 978)</td>
<td>0.66</td>
</tr>
<tr>
<td>Males</td>
<td>8240 (7992 – 8488)</td>
<td>17 232 (0 – 56 744)</td>
<td>0.67</td>
</tr>
<tr>
<td>Persons</td>
<td>14 064 (13 741 – 14 388)</td>
<td>28 745 (0 – 76 302)</td>
<td>0.73</td>
</tr>
<tr>
<td><strong>Non-Hodgkin lymphoma</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>2190 (2073 – 2306)</td>
<td>3104 (1508 – 4700)</td>
<td>0.87</td>
</tr>
<tr>
<td>Males</td>
<td>2956 (2819 – 3093)</td>
<td>3973 (1952 – 5993)</td>
<td>0.84</td>
</tr>
<tr>
<td>Persons</td>
<td>5145 (4966 – 5325)</td>
<td>7077 (4502 – 9652)</td>
<td>0.93</td>
</tr>
<tr>
<td><strong>Stomach</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>671 (611 – 732)</td>
<td>808 (450 – 1166)</td>
<td>0.77</td>
</tr>
<tr>
<td>Males</td>
<td>1401 (1311 – 1491)</td>
<td>1778 (1034 – 2523)</td>
<td>0.84</td>
</tr>
<tr>
<td>Persons</td>
<td>2072 (1964 – 2181)</td>
<td>2586 (1760 – 3412)</td>
<td>0.89</td>
</tr>
</tbody>
</table>

^a The probability of increase was calculated using \( P(\bar{y}_{f+t} \geq m\bar{y}_f) \), where the probability distribution for \( \bar{y}_{f+t} \) is its posterior distribution and \( \bar{y}_f \) was the modelled count in 2016 and \( m \) was 1, 1.15, 1.5 or 2.
References


