Spatial variation in cancer incidence and survival over time across Queensland, Australia

Abstract

Interpreting changes over time in small-area variation in cancer survival, in light of changes in cancer incidence, aids understanding progress in cancer control, yet few space-time analyses have considered both measures. Bayesian space-time hierarchical models were applied to Queensland Cancer Registry data to examine geographical changes in cancer incidence and relative survival over time for the five most common cancers (colorectal, melanoma, lung, breast, prostate) diagnosed during 1997-2004 and 2005-2012 across 516 Queensland residential small-areas. Large variation in both cancer incidence and survival was observed. Survival improvements were fairly consistent across the state, although small for lung cancer. Incidence changes varied by location and cancer type, ranging from lung and colorectal cancers remaining relatively constant over time, to prostate cancer dramatically increasing across the entire state. Reducing disparities in cancer-related outcomes remains a health priority, and space-time modelling of different measures provides an important mechanism by which to monitor progress.

1. Introduction

With an estimated 14.1 million cancer cases diagnosed globally in 2012,[1] the impact of cancer is felt worldwide. With wide variation in cancer incidence and survival not only between countries,[1,2] but also within countries,[3,4] there are important disparities depending on where people live.

Quantifying and understanding the extent of small-area variation in cancer incidence and survival is becoming increasingly important, with government and other policy makers needing to make evidence-based decisions on resource allocation and planning interventions to address any known disparities. Consistent with this, an increasing number of small-area cancer atlases have been published, including those in Australia,[5-7] USA[8] and the UK.[9]

There is great variation in the statistical approaches used in these Atlases. These methods range from direct estimation of area-specific age-standardised incidence rates[5] through to modelling approaches incorporating smoothing such as Poisson kriging,[10] empirical Bayes[11] or fully Bayesian methods.[7] While each method has various benefits and disadvantages, some form of smoothing is often preferred to reduce spurious variation associated with very small area-specific counts.[12]

We have previously demonstrated the extent of small area variation in incidence and survival across the state of Queensland, Australia for around 20 of the most commonly diagnosed cancers.[6] This cancer atlas highlighted the extent of the geographical variability in incidence across Queensland, and how the survival outcomes were poorer in many of the more remote areas of the state.

However, it was unclear how these geographical patterns in cancer incidence and survival have changed over time. Since the ability to understand whether the spatial patterns are changing over time and in what direction is critical to guide efforts to reduce existing disparities, we have examined how the geographical variation in cancer incidence and survival in Queensland has changed over time for the five most commonly diagnosed cancers.

2. Methods

Ethical approval to conduct this study was obtained from the Darling Downs Hospital and Health Service Human Research Ethics Committee (HREC/15/QTDD/57).

2.1 Data and Analysis

De-identified data on all cases of colorectal (ICD-O-3[13] C18-C20, C218), lung (ICD-O-3 C33-C34), melanoma (ICD-O-3 C44 M872-M879), breast (ICD-O-3 C50) and prostate (ICD-O-3 C61) diagnosed in Queensland during 1997 to 2012 was obtained from the population-

based Queensland Cancer Registry (QCR). All non-keratinocytic cancers diagnosed are notifiable by law.

The patient's address at diagnosis was geocoded within the QCR, and assigned to one of 516 residential Statistical Area 2s (SA2s) based on the 2011 Australian Statistical Geography Standard (ASGS) boundaries.[14] SA2s with an average population below 5 during 1997-2012 were considered to be non-residential and were excluded (n=10). In 2011, the median population of a residential SA2 was 7996 (range:7 to 29,641). Cases with insufficient information to determine the SA2 at diagnosis were excluded.

The study cohort included those diagnosed with an invasive cancer and aged 15-89 years at diagnosis. Cases diagnosed through death or autopsy were excluded. Year of diagnosis was split into two diagnostic time periods: 1997-2004 and 2005-2012.

The QCR routinely conducts data linkage with the Australian National Death Index to determine the survival status of all cancer patients. Survival time (in days) was provided by the QCR, with follow-up of all patients to 2013. For the survival analyses, cases were censored at the earliest of five years from diagnosis or the specified censoring date, which was 31 December 2005 for the 1997-2004 cohort and 31 December 2013 for the 2005-2012 cohort.

As is the case for most population-based cancer survival studies, we used relative survival to estimate net survival. Since it compares the cohort mortality against the population mortality, relative survival has the advantage over cause-specific survival in not requiring cause of death information.[15]

To calculate the SA2-specific population mortality rates, unit record file death data were obtained from the Australian Bureau of Statistics (ABS) (for deaths from 1997 to 2005)[16] and the Australian Coordinating Registry (2006-2013).[17] Corresponding population data for each SA2, 5-year age group and sex was obtained from the Australian Bureau of Statistics (ABS) for 1997-2013. Concordance files provided by the ABS were used to adjust all the geographical information to the 2011 ASGS SA2 boundaries. To account for the low numbers of deaths in some SA2, single year age, sex and year categories, a smoothing process was used to increase the stability of the expected mortality. Briefly, population and mortality data for each SA2 were aggregated into strata comprising three time periods (1997-2002, 2003-2008, 2009-2013), by 5-year age group (to 90+ years) and sex. Neighbouring SA2s were identified based mainly on shared boundaries, although islands included nearby mainland areas. "Smoothed" population mortality rate estimates for specific SA2s by strata group were then calculated by combining the SA2-specific mortality and population with the corresponding data from all neighbouring areas. These smoothed estimates were then expanded so the same mortality rate was assigned to each single year age, single calendar

year, sex and SA2 within any given 5-year age group, 5-year or 6-year calendar time period, sex and SA2. These smoothed estimates were used in both the non-Bayesian and Bayesian relative survival models.

2.2 Incidence models

To examine changes in cancer incidence over time, a Bayesian space-time model based on that introduced by Bernardinelli *et al.*[18] was used. A Poisson distribution:

$$O_{ij} \sim \text{Poisson}(\theta_{ij} E_{ij})$$

forms the foundation of this model, where O_{ij} are the observed new cancer cases in *i*=1,2…516 areas and *j*=1,2 time periods (representing 1997-2004 and 2005-2012), θ_{ij} is the corresponding modelled standardised incidence ratio (SIR) and E_{ij} represents the age- and sex-standardised expected counts. The log of the modelled SIR can then be written as:

$$\log(\theta_{ij}) = \alpha + \lambda\delta_j + s_i\delta_j + u_i + v_i$$

and each of these parameters were given prior distributions. The intercept term α and coefficients λ for the *j*th time period indicator δ have vague normal priors, u_i (structured spatial variation) and s_i (the differential trend) are assumed to follow an intrinsic conditional autogressive (CAR) prior with neighbours assigned based largely on geographically adjacent boundaries (since islands included the closest mainland areas as neighbours), and v_i represents unstructured spatial variation, with a vague normal distribution for each of i areas. Additional details on the prior distributions are provided in Supplementary Table S1.

Since the expected counts were standardised by age and sex, these variables were not included in this model.

2.3 Relative survival models

Recently, we introduced a Bayesian space-time flexible parametric relative survival model.[19] This approach had many advantages over Poisson-piecewise based models, including the feasibility of including individual-level data, time-varying components and complex interactions.[20]

Consider that the *d*th individual (*d*=1,…D) with covariate x_d lives in area *i* (represented as *i*[*d*], similar to Gelman and Hill[21]), then the space-time relative survival model can be written as:

$$\ln(-\ln R_{ij}(t)) = \ln(\Lambda(t)) = \ln(\Lambda_0(t)) + \mathrm{x}_d\beta + \lambda\delta_j + s_{i[d]}\delta_j + u_{i[d]} + v_{i[d]}$$

where $R_{ij}(t)$ is the relative survival function for the *i*th area (*i*=1,…516), and *j*th time period (1,2 representing cases diagnosed in 1997-2004 and 2005-2012, respectively), $\Lambda(t)$ is the cumulative excess hazard, $\Lambda_0(t)$ is the cumulative baseline excess hazard (the cumulative excess hazard when all covariates are 0) and $\beta = [\beta_1, \ldots, \beta_K]$ and represents the vector of coefficients relating to covariates $x_d = [x_{d1}, \ldots, x_{dK}]$. The indicator variable for the *j*th time period is δ_j, the overall temporal change is represented by λ, and $s_{i[d]}$ is the difference in excess mortality between the overall time change and the *i*th area.

Note that the cumulative excess hazard is composed of two terms,

$$\Lambda(t) = H(t) - H^*(t)$$

where $H(t)$ is the overall cumulative hazard, based on all deaths within the cohort, and $H^*(t)$ is the cumulative expected hazard, obtained from the population mortality estimates.

This model is based on the Weibull distribution, but the cumulative excess baseline hazard $\Lambda_0(t)$ is flexibly modelled using restricted cubic splines as a function of log time. The spline component enables flexible parametric models to fit the data better than parametric models.[22] Provided at least one interior knot is specified, the spline includes a constant term (γ_0), a parameter with a linear function of log time (γ_1), and, for each subsequent interior knot *m*=1,…,*M*, a basis function $(z_m(t))$ with associated parameter(γ_{m+1}). The model can thus be written as:

$$\ln\big(-\ln R_{ij}(t)\big) = \gamma_0 + \gamma_1 \ln(t) \ldots, + \gamma_{M+1} z_M(t) + x_d\beta + \lambda\delta_j + s_{i[d]}\delta_j + u_{i[d]} + v_{i[d]}$$

The covariates included in the model were age (centred continuous spline terms) and sex (males, females) for lung, colorectal, and melanoma.

Each model parameter was given a prior distribution, with vague normal distributions assumed for γ_m, β, λ and $v_{i[d]}$, while $u_{i[d]}$ and $s_{i[d]}$ were each given an intrinsic CAR prior with neighbours again based largely on shared boundaries (see Supplementary Table S1 for details on prior distributions).

2.4 Model assessment

All incidence and survival models were run as single chain Markov chain Monte Carlo (MCMC) in WinBUGS v1.4.3 interfaced with Stata v14.2. Running a single chain for an extended time adequately enables convergence assessment, and has theoretical justification.[23] Incidence and survival models discarded the first 70,000 iterations and monitored a further 50,000 iterations, keeping every 10th iteration to reduce the autocorrelation.

Convergence of the MCMC estimates was assessed graphically via trace plots and segment histograms for a subsample of areas with low populations (s_i, u_i, v_i), as well as all α, β, λ and γ_m terms. Autocorrelation was also graphically assessed. The Geweke diagnostic[24] was used to monitor convergence on all parameters, and is calculated as the difference between the means for the first 10% of iterations and the final 50% of iterations, divided by the asymptotic standard error of the difference. An estimate with a Geweke p-value below 0.01 was considered unlikely to have converged. Convergence diagnostics for colorectal cancer are available in Supplementary Figures S1 to S2.

Relative survival models were first run in Stata using the stpm2 command with just the fixed effects (γ_m, β, λ) to enable determination of model components, before running the full Bayesian model in WinBUGS. Since these initial models required population mortality data, the 'smoothing' was carried out prior to running the full Bayesian model. Likelihood ratio-tests and plots of predicted hazard components were used to determine which variables to include as time-varying components. The preferred number of knots for the restricted cubic splines (for centred continuous age, the baseline hazard, and any time-varying components) was selected based on Bayesian information criterion (BIC) values, as well as plotting the predicted hazard function. Details on the final model specifications are shown in Supplementary Table S2. Estimates for the γ_m, β, and λ terms from WinBUGS were checked against the estimates using the corresponding survival model in Stata and found to be similar for all five types of cancer.

Sensitivity analyses considered two vague distributions on the hyperprior variance component σ^2 for $v_{i[d]}$, $u_{i[d]}$ and $s_{i[d]}$ by comparing their impact on the final estimates. Models were run for the following options:

1. Gamma distribution (shape, scale) on the precision, $\frac{1}{\sigma^2} \sim \Gamma(0.1,100)$
    i. CAR distribution on s_i and $s_{i[d]}$
    ii. Vague normal distribution on s_i and $s_{i[d]}$
2. Uniform distribution (minimum, maximum) on the standard deviation, $\sigma \sim \mathrm{U}(0,20)$
    i. CAR distribution on s_i and $s_{i[d]}$
    ii. Vague normal distribution on s_i and $s_{i[d]}$

Although similar estimates were obtained under both distributions, option 1.i with the CAR distribution on the differential trend component was selected since it improved convergence of estimates (see Supplementary Figures S1-S2).

2.5 Visualisation

Maps were generated using MapInfo Pro v15.0. As the majority of SA2s are located in the urbanised south-east corner, this region is magnified on maps using an oval inset. The estimates used in the maps were the median of the posterior distributions calculated as follows:

- SIR for 1997-2004: $\exp(\alpha + u_i + v_i)$
- SIR for 2005-2012: $\exp(\alpha + \lambda\delta_j + s_i\delta_j + u_i + v_i)$
- Change in SIR between 1997-2004 and 2005-2012: $\exp(\lambda\delta_j + s_i\delta_j)$.

Likewise, the adjusted relative survival estimates were calculated as excess hazard ratios (EHRs) as follows:

- EHR for 1997-2004: $\exp(u_{i[d]} + v_{i[d]})$
- EHR for 2005-2012: $\exp(\lambda\delta_j + s_{i[d]}\delta_j + u_{i[d]} + v_{i[d]})$
- Change in EHR between 1997-2004 and 2005-2012: $\exp(\lambda\delta_j + s_{i[d]}\delta_j)$

For the second time period of 2005-2012, the baseline reference for the SIR and EHR was the 1997-2004 Queensland average. This meant that any of the maps with an average value (set to 1) had a similar incidence/excess risk of death within five years of diagnosis as the Queensland average during 1997-2004.

The same five categories were used across all maps to enable meaningful comparisons between the maps. These were deliberately selected to be wide, to reduce the likelihood of detecting spurious changes. Cut-points of 10% and 30% above the average (=1) estimate were selected (1.1 and 1.3 respectively), while the corresponding lower cut-offs were the inverse of these values (0.91 and 0.77).

The 80% credible intervals (CrIs) typically provide sufficient coverage of the posterior distributions generated from a well-fitting Bayesian model,[25] so are generally considered to correspond to the standard 95% confidence intervals reported in non-Bayesian analyses. Graphs of the 80% CrIs with the interval shading demonstrating the mapped colour of the median SIR/EHR estimate for each SA2 are provided in Supplementary Figures S3-S7. The graphs for 2005-2012 estimates also show the median SIR/EHR estimate for each SA2 during 1997-2004 as a grey dot, showing changes in the median estimate for each SA2.

Maps showing 'convincing' changes over time were also generated, and these were based on the 80% credible intervals for $\exp(\lambda\delta_j + s_i\delta_j)$ or $\exp(\lambda\delta_j + s_{i[d]}\delta_j)$ being above one (increase), below one (decrease), or, if it included one it was considered equivocal. These maps in Figure 2 show which areas are likely to have experienced change between the time periods, while the maps of median estimates (Figures 1 and 3) focus on the magnitude of changes.

3. Results

3.1 Incidence

During 2005-2012, there were almost 112,000 new diagnoses of the five most common cancers among our study cohort (Table 1). This was an increase of almost 31,000 new cases diagnosed compared to 1997-2004. Across total Queensland, incidence rates were lower for colorectal cancer in the later time period but higher for prostate cancer (Table 1). The age-standardised incidence rates for breast, lung and melanoma remained reasonably similar over the two time periods, with overlapping 95% confidence intervals. These findings were also reflected in the overall modelled time trends, although the small increase in breast cancer incidence was considered convincing in the modelled results with the 80% credible interval not including one (Table 1). Details on all modelled parameter estimates are available in Supplementary Table S3.

There was marked variation in cancer incidence across residential areas in both time periods for the five cancer types (Figure 1). The incidence of most cancer types was lower in the more rural and remote areas further away from the east coastline, particularly between 1997 and 2004. The exception was lung cancer, in which the incidence was generally higher in more remote areas, although some urban areas in the south-east corner of the state also had higher incidence in both time periods.

The patterns of change over time differed by cancer type and geographic location (Figures 1 and 2). In the majority of areas, colorectal cancer was relatively stable. Only 5 areas showed a convincing increase between the two time periods in colorectal cancer incidence, while 67 areas decreased (Figure 2). While more areas changed over time for melanoma incidence (67 areas increased, 62 areas decreased), these patterns depended on broad location with melanoma incidence rates tending to decrease in more remote areas, but increase in south-eastern and coastal northern Queensland areas. There was more stability in lung cancer incidence rates between the two time periods, with only 15 areas showing a decrease and 13 areas an increase. In contrast, the predominant pattern for breast (100 areas increased, 4 decreased) and prostate cancer (477 increased, 0 decreased) was for the incidence rates to increase between the two time periods. Again, this pattern varied by geographic location, with the increase in breast cancer incidence being predominately around northern and western Queensland, while the increases for prostate cancer incidence were larger and more widespread.

3.2 Survival

The five-year relative survival for cancers diagnosed during 2005-2012 ranged from 16.5% (lung) to 94.6% (prostate) (Table 1). For all cancers except melanoma, this represented an

increase in survival from those diagnosed during 1997-2004. After adjustment for age and sex (where applicable), the increase in survival across total Queensland for all five cancers was statistically convincing (Table 1). Prostate cancer showed the greatest change, with the risk of death within 5 years almost halving, while the reduction for lung cancer (8% decrease) was smallest. Details on all modelled parameter estimates are available in Supplementary Table S3.

For each individual cancer type diagnosed in 1997-2004, there was wide geographical variation in the median risk of dying from that cancer within 5 years of diagnosis (Figure 3). However, the pattern of geographical variation was consistent across the cancer types, with the excess risk of death within 5 years of diagnosis being lower in the south-east corner of the state, and higher in the other western and northern areas.

Between 1997-2004 and 2005-2012, the predominant pattern was for the median survival to increase across most areas (Figures 2 and 3). This meant that many areas with cancers diagnosed during 2005-2012 had a reduced excess risk of death within 5 years of diagnosis (Figure 3).

Every SA2 showed a convincingly decreased risk of death within 5 years for breast cancer, and almost every area for prostate and colorectal cancers (Figure 2). Even lung cancer had convincing decreases (albeit generally small) in 342 areas. Of these, the change ranged from 5% to 15% lower risk of death than during 1997-2004, so many were not noticeable when reporting the median risk estimates in Figure 3. Melanoma likewise had widespread decreases across the state (Figure 3), but only 266 areas were considered to have convincing decreases (Figure 2). In comparison, the large changes in prostate cancer over time, ranging from 28% to 60% lower excess risk of death within 5 years compared to those diagnosed during 1997-2004, meant that there was no inconsistency between the different maps.

4. Discussion

In Queensland, the risk of a cancer diagnosis or cancer-related death varies by residential location. This is true for all five of the most commonly diagnosed cancers, whether diagnosed during 1997-2004, or during 2005-2012. The general improvement in survival over most areas between the two time periods means that geographical disparities have remained. This suggests that it is not sufficient to just ensure that diagnostic and management strategies are equivalent across the state, rather reasons for the disparities need to be understood to develop geographically focused strategies to ensure that the cancer-related outcomes are equivalent across the state.

Of the five cancer types examined, prostate cancer showed the greatest and most consistent improvement in survival across all areas of the state between the two time periods, coinciding with similar consistent increases in incidence. This pattern supports the hypothesis of an

impact of prostate specific antigen (PSA) testing, in that the subsequent overdiagnosis of localised prostate cancers leads to increased incidence and artificially increased survival due to lead-time bias. While it has been shown that the use of PSA testing is higher in capital cities of Australia compared to the rest of the country,[26] the testing rate increased in both areas. However, the recent release of guidelines recommending a reduction in the use of PSA[27] may impact future spatial and temporal patterns.

Breast cancer also showed large survival improvements, with incidence generally increasing, particularly in more remote areas. All women aged between 50-69 years were eligible for publicly-funded mammography screening every 2 years throughout the time period examined, and nationally participation remained around 55% consistently, with variation by remoteness (lowest participation in very remote areas, highest participation in outer regional areas).[28] Breast cancer treatment has also improved[29] and the rural mobile mammography services went digital in 2009.[30]

Melanoma likewise showed large, albeit rather uncertain, improvements in survival, with incidence increasing only among certain regions in northeastern and southeastern Queensland, and decreasing elsewhere. As regular skin checks can assist in early detection, the lower diagnosis rate of melanoma in more remote areas suggests the availability of skin checking services might be influencing incidence patterns. It is possible the survival improvements are driven more by improved therapies. For advanced melanoma, there have been recent advances in targeted therapies and immunotherapies.[31]

In contrast, the decrease in colorectal cancer incidence was observed over most areas of Queensland, yet survival improvements were consistent over the state. In contrast to prostate cancer, screening for colorectal cancer through FOBT testing is designed to detect pre-cancerous polyps, which, if found and removed, would result in lower incidence. However, the participation in the national colorectal cancer screening program since it was introduced in 2006 has been low, particularly among remote areas,[32] so it is unlikely to have impacted on the observed results. Targeted therapies have also become available for advanced colorectal cancer,[33] and it is possible they may be influencing the improved survival.

The almost negligible change in lung cancer incidence over the two time periods is likely a result of combing both males and females with their contrasting incidence trends.[34] Survival improvements, although often convincing, were small in magnitude, reflecting that lung cancer is often diagnosed at a later stage and there have been limited improvements in treatment during the time periods examined.[35]

Other Bayesian space-time models have been developed and applied to cancer incidence data, including age-period-cohort,[36,37] dynamic[38,39] and mixture models.[40] Age-period-cohort models can be useful when strong cohort effects are expected, such as for lung cancer, but

assume minimal migration between areas. In contrast, mixture models are focused on detecting disparate areas which will not be smoothed over, while dynamic models are more useful when there are many time periods involved. In this analysis, we expected neighbouring regions to have similar outcomes, so did not use a model that allowed for disparate changes. Others have examined different forms of interactions between spatial and temporal components,[41,42] but as we only had two time components, this was not investigated. However, when several time periods are available, some form of correlation across time should be considered.

The choice of intervals for maps requires careful consideration, since different intervals can lead to different interpretation. For this reason we presented maps showing the median estimates for each SA2 (categorised into five groups), as well as those areas with a convincing difference to the Queensland average (based on 80% credible intervals). Both maps have different but complementary purposes. The median captures the magnitude of the difference, although ignores the level of uncertainty, while the statistically convincing changes captures areas we are confident do differ to some extent from the average, without quantifying the magnitude of that difference. We acknowledge that there are many other ways of defining and visualising the magnitude, significance and uncertainty of spatial estimates, and the optimal methods of presenting static and dynamic disease maps continues to be an active field of research.[43]

Within the Bayesian framework, convergence, sensitivity and identifiability of model parameters need to be considered. We divided the residual component into three terms: s_i, u_i and v_i. It is recognised that spatial models are often unable to identify even two separate terms (u_i and v_i),[44] and given our specific interest in the differential trend component s_i, we chose to place a stronger hyperprior distribution on the variance for these three terms to aid in identifiability.

Interpreting the driving factors behind geographical variation and changes in incidence and survival is difficult without information about tumour stage at diagnosis. Currently the Queensland Cancer Registry does not record stage at diagnosis for all cancers, although a broad measure of spread of disease is available for breast cancer[45] and stage for colorectal cancer estimated using pathology forms.[46] For consistency across cancers, the impact of stage was not examined in this study. However, our previous work has found that differences in stage at diagnosis was the key driver of poorer breast cancer survival among more remote areas, while for colorectal cancer, factors related to the time after diagnosis played a greater role in the survival disparities.[20]

Other limitations included the lack of treatment information, and the substantial computation time involved in running the models through WinBUGS, especially for the more complex relative survival models which ranged from 11.5 hours for breast cancer to 20.5 hours for

prostate cancer on a dual CPU Quad Core Xeon E5520 computer. The use of emerging approximation methods, such as integrated nested Laplace approximation (INLA), is increasing within the spatial field, and offers much potential to reduce computation times although it does not have the flexibility of likelihood specification available in WinBUGS.

Strengths of this study include a high quality population-based cancer registry with full coverage of the cancers diagnosed among Queensland residents, the use of an innovative relative survival methodology that appropriately captures both spatial variation and temporal changes in that variation, and a population dispersed over areas of varying remoteness.

In conclusion, our space-time analyses have provided unique insights into how the geographical patterns in cancer incidence and survival have changed over time in Queensland. While the improvements in survival are encouraging, the lack of reduction in the spatial variation between the two time periods means that cancer patients who live in specific geographical areas continue to experience poorer survival outcomes than those living in other areas. To reduce these disparities in outcomes, greater focus needs to be placed on understanding why these disparities occur, and then implementing appropriate interventions to address the barriers to equal outcomes.

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Table 1: Cohort incidence, survival and time changes by cancer type, persons, aged 15-89 years, Queensland

	Incidence							5-year relative survival					
	1997-2004			2005-2012			Modelled change in	1997-2004		2005-2012		Modelled change in	
	N cases	ASR	[95% CI]	N cases	ASR	[95% CI]	SIR [80% CrI]	RS	[95% CI]	RS	[95% CI]	EHR	[80% CrI]
Colorectal	17,628	63.9	[63.0,64.9]	21,953	61.2	[60.4,62.1]	0.96 [0.95, 0.97]	64.6	[63.2, 66.0]	70.2	[68.9, 71.4]	0.80	[0.77, 0.82]
Melanoma	18,025	71.9	[70.8,72.9]	23,062	71.5	[70.5,72.4]	1.00 [0.99, 1.02]	94.7	[93.8, 95.6]	94.2	[93.4, 95.0]	0.86	[0.80, 0.93]
Lung	12,129	43.9	[43.1,44.7]	15,624	43.2	[42.5,43.9]	0.99 [0.97, 1.00]	14.2	[13.1, 15.3]	16.5	[15.5, 17.6]	0.92	[0.90, 0.93]
Breast (females)	16,621	127.7	[125.8,129.7]	21,583	130.1	[128.4,131.9]	1.04 [1.02, 1.05]	88.5	[87.4, 89.5]	91.0	[90.2, 91.8]	0.75	[0.71, 0.79]
Prostate	16,711	126.5	[124.6,128.5]	29,691	171.8	[169.9,173.8]	1.34 [1.33, 1.36]	84.7	[83.1, 86.2]	94.6	[93.8, 95.4]	0.53	[0.49, 0.56]

N=number, ASR=age-standardised rate, CI=confidence interval, CrI=credible interval, RS=relative survival, SIR=standardised incidence ratio, EHR=excess hazard ratio.

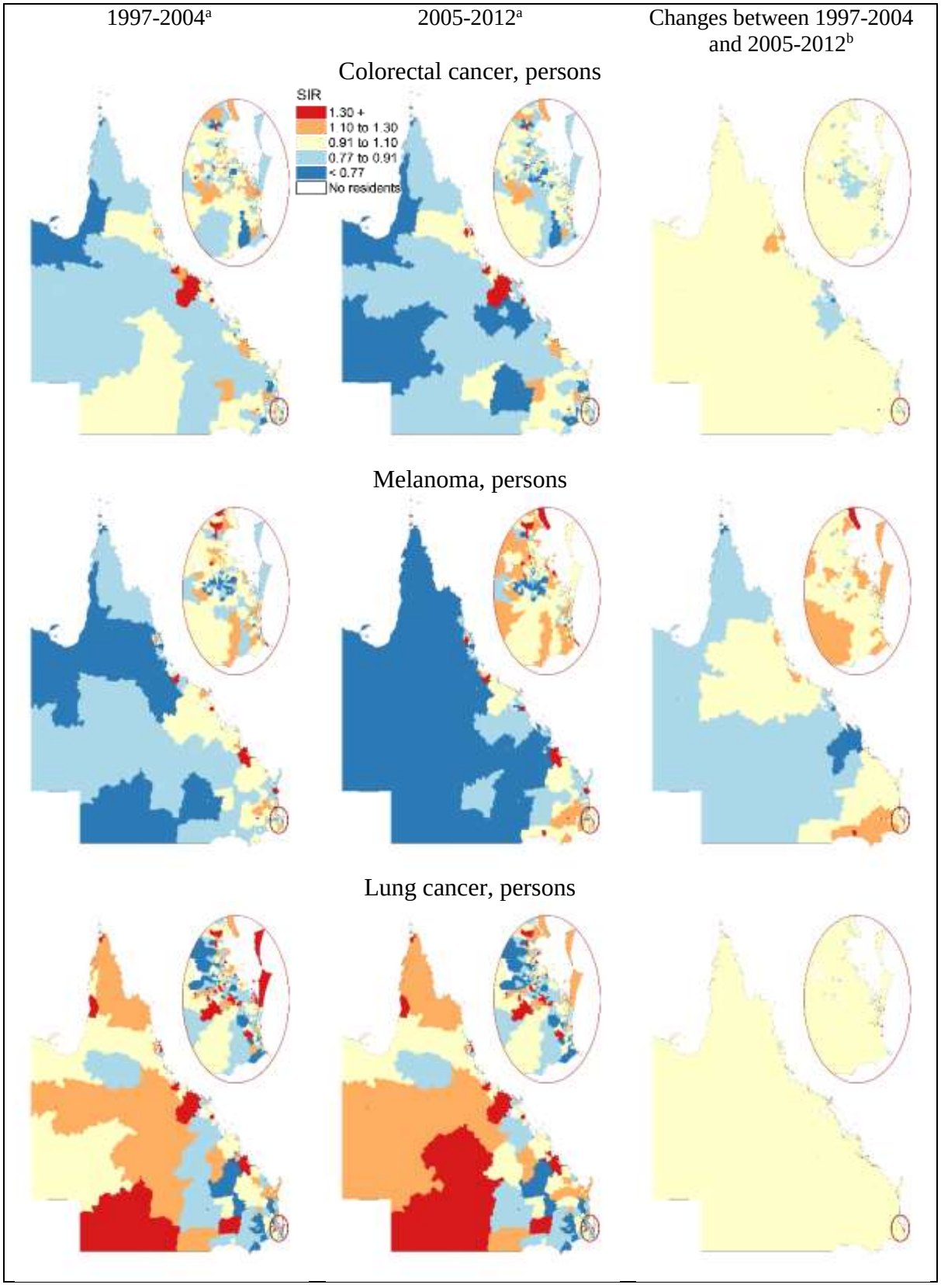
Note than an 80% CrI is considered equivalent coverage to a 95% CI.

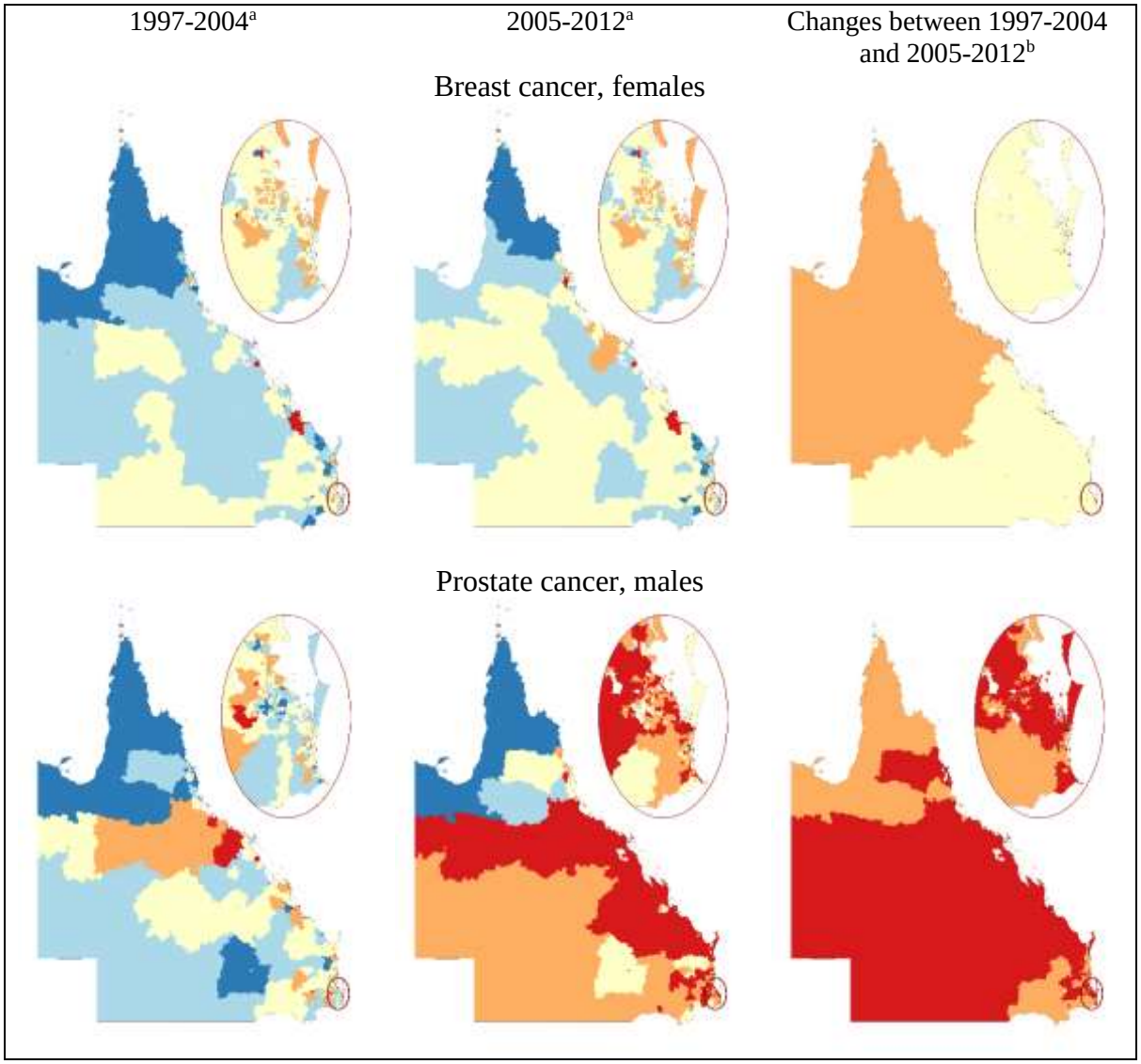
Rates age-standardised to the WHO world 2000 standard population as modified by SEER.

Relative survival is calculated using an unadjusted cohort analysis of the lifetable method.

Change in EHR estimates have been adjusted for age and sex (where applicable).

Figure 1: Spatial variation in risk of diagnosis by cancer type, Queensland





SIR=Standardised incidence ratio.

The median posterior estimate is mapped.

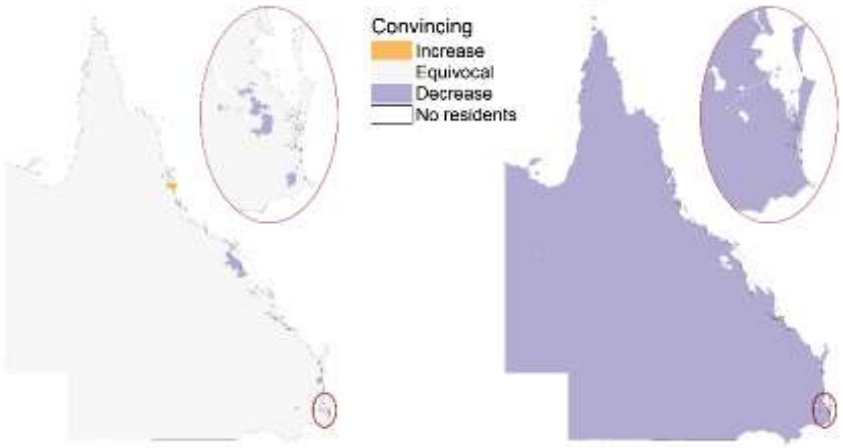
a. Results are in comparison to the Queensland average incidence in 1997-2004 (set to 1).

b. Results for changes between time periods are in comparison to each individual SA2, so 1=the same incidence in that SA2 during 1997-2004.

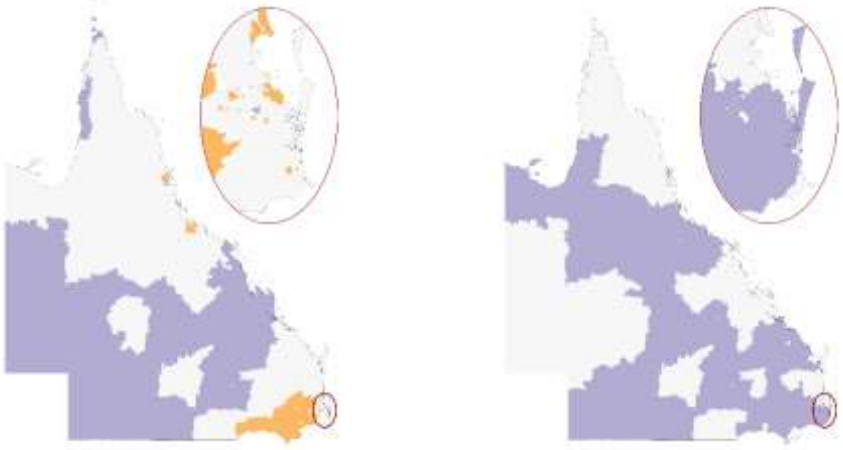
Figure 2: Convincing changes between 1997-2004 and 2005-2012 by cancer type, Queensland

Risk of diagnosis	Excess risk of death within five years of diagnosis

Colorectal cancer, persons



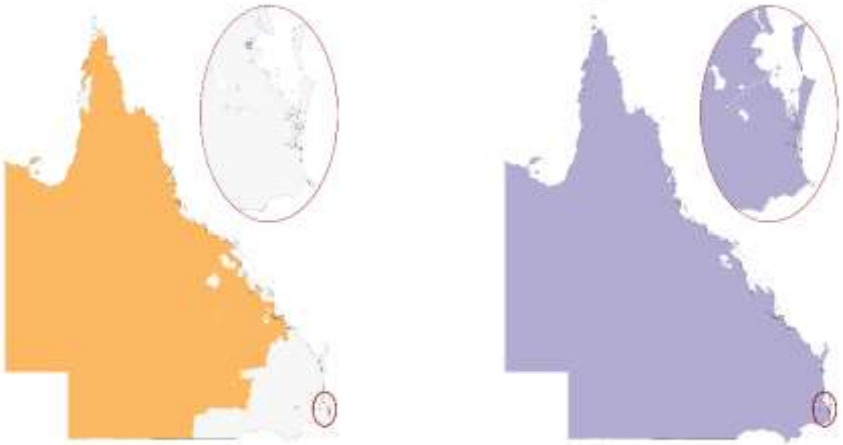
Melanoma, persons



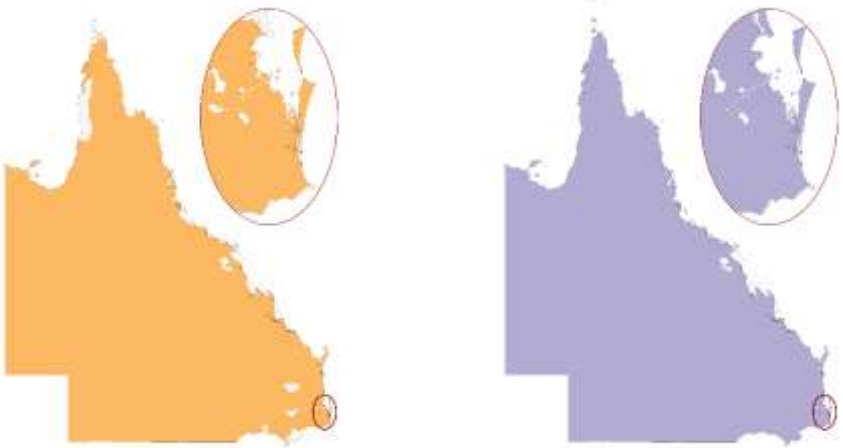
Risk of diagnosis	Excess risk of death within five years of diagnosis

Lung cancer, persons

Breast cancer, females

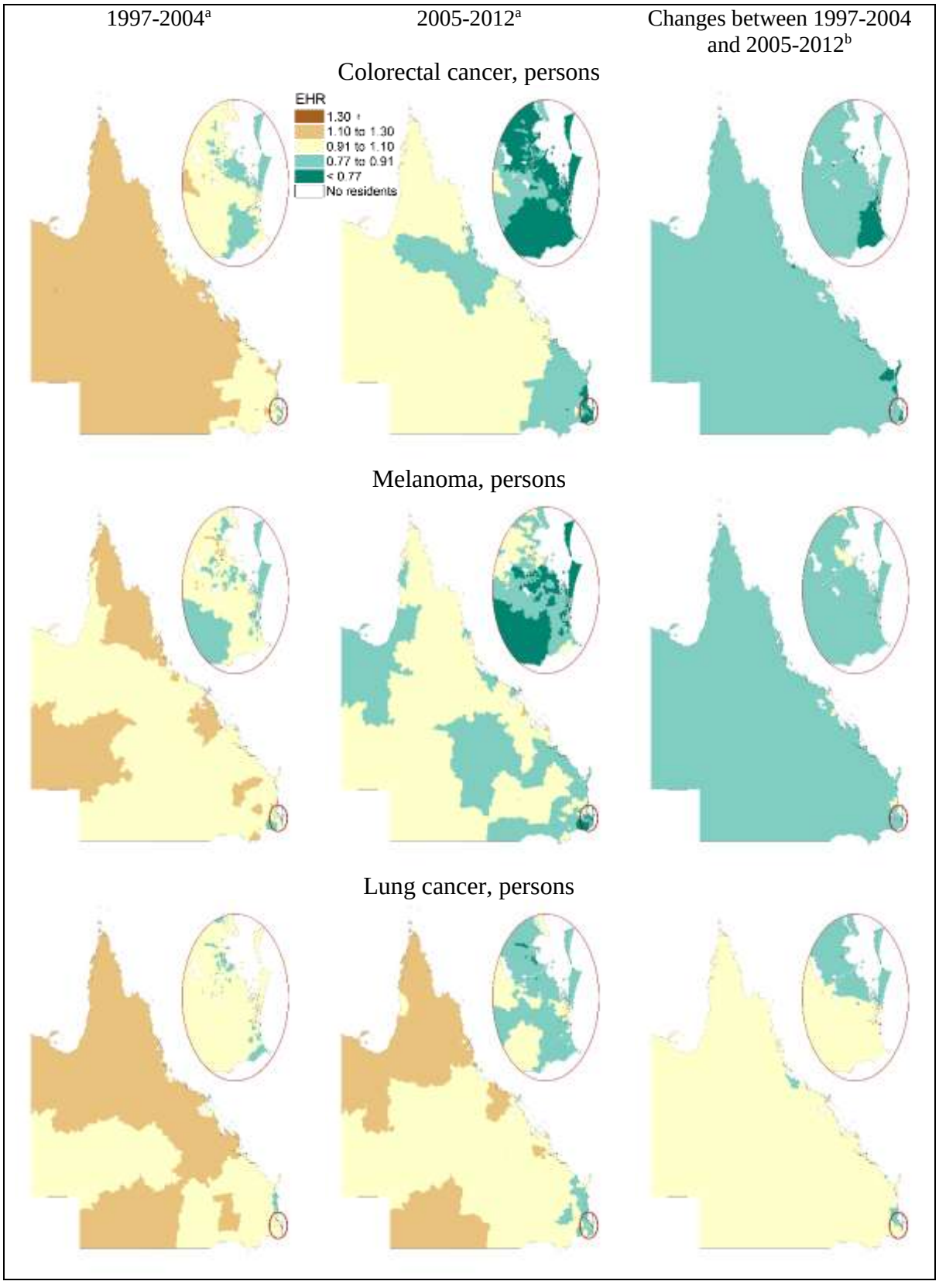


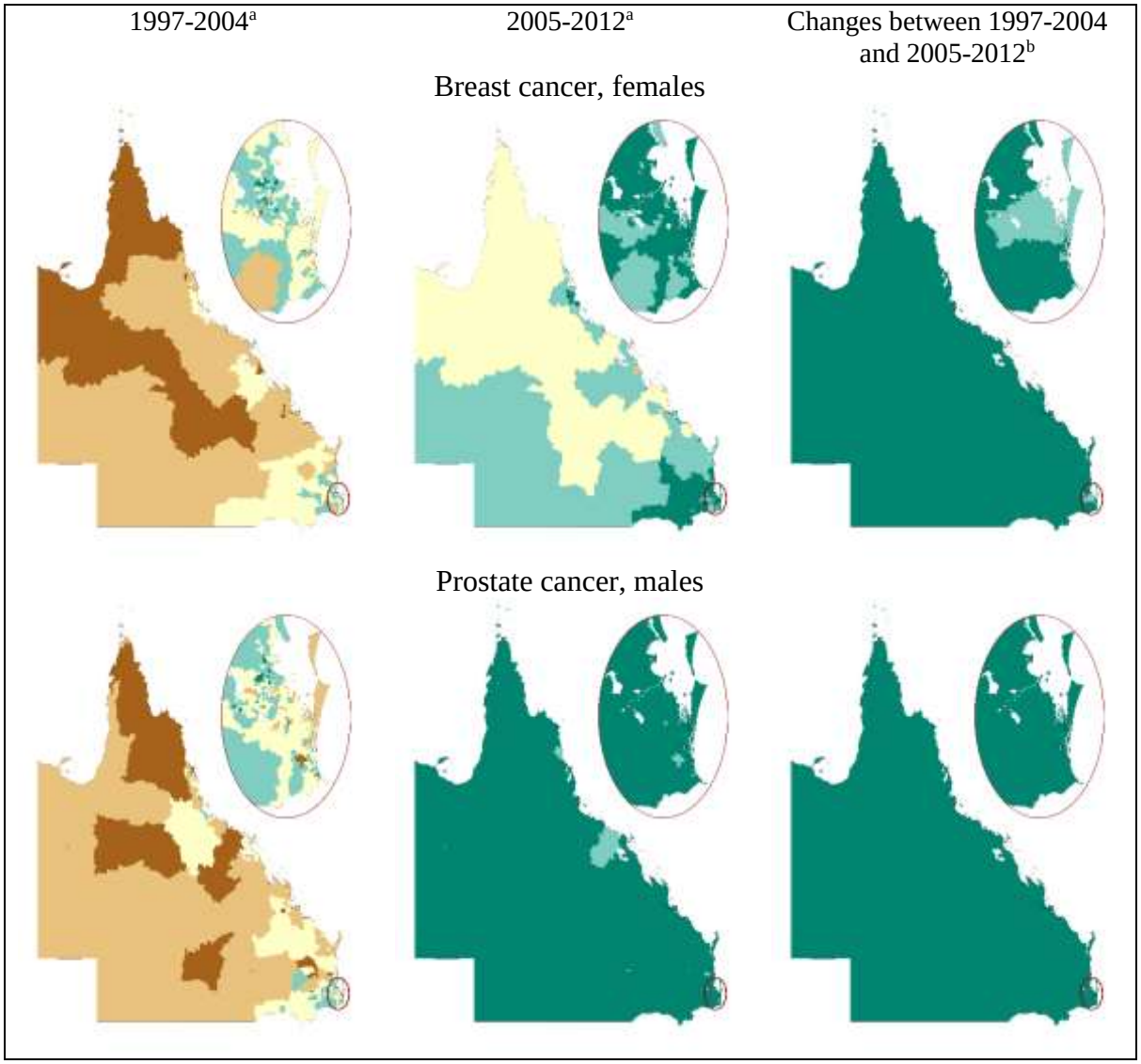
Prostate cancer, males



Notes: Convincing is defined as the 80% credible interval not including one. These estimates are for the risk of diagnosis/excess risk of death in 2005-2012 in comparison to each individual SA2 in 1997-2004.

Figure 3: Spatial variation in excess risk of death within five years of diagnosis by cancer type, Queensland, 1997-2012





EHR=Excess hazard ratio.

The median posterior estimate is mapped.

a. Results are in comparison to the Queensland average excess risk of death in 1997-2004 (set to 1).

b. Results for changes between time periods are in comparison to each individual SA2, so 1=the same excess risk of death in that SA2 during 1997-2004.