Cancer Epidemiology 42 (2016) 199–205

Contents lists available at ScienceDirect

Cancer Epidemiology

The International Journal of Cancer Epidemiology, Detection, and Prevention

journal homepage: www.canceepidemiology.net

Has the incidence of brain cancer risen in Australia since the introduction of mobile phones 29 years ago?☆

Simon Chapman[a,*], Lamiae Azizi[a], Qingwei Luo[a,b], Freddy Sitas[a,c]

[a] School of Public Health, University of Sydney, Australia
[b] Cancer Council NSW, Sydney, Australia
[c] School of Public Health and Community Medicine, University of New South Wales, Australia

ARTICLE INFO

Article history:
Received 17 February 2016
Received in revised form 19 April 2016
Accepted 19 April 2016
Available online 5 May 2016

Keywords:
Mobile phone
Cell phone
EMF
Radiofrequency radiation
Brain cancer
Trends
Incidence

ABSTRACT

Background: Mobile phone use in Australia has increased rapidly since its introduction in 1987 with whole population usage being 94% by 2014. We explored the popularly hypothesised association between brain cancer incidence and mobile phone use.

Study methods: Using national cancer registration data, we examined age and gender specific incidence rates of 19,858 male and 14,222 females diagnosed with brain cancer in Australia between 1982 and 2012, and mobile phone usage data from 1987 to 2012. We modelled expected age specific rates (20–39, 40–59, 60–69, 70–84 years), based on published reports of relative risks (RR) of 1.5 in ever-users of mobile phones, and RR of 2.5 in a proportion of 'heavy users' (19% of all users), assuming a 10-year lag period between use and incidence.

Summary answers: Age adjusted brain cancer incidence rates (20–84 years, per 100,000) have risen slightly in males ($p < 0.05$) but were stable over 30 years in females ($p > 0.05$) and are higher in males 8.7 (CI = 8.1–9.3) than in females, 5.8 (CI = 5.3–6.3). Assuming a causal RR of 1.5 and 10-year lag period, the expected incidence rate in males in 2012 would be 11.7 (11–12.4) and in females 7.7 (CI = 7.2–8.3), both $p < 0.01$; 1434 cases observed in 2012, vs. 1867 expected. Significant increases in brain cancer incidence were observed (in keeping with modelled rates) only in those aged ≥70 years (both sexes), but the increase in incidence in this age group began from 1982, before the introduction of mobile phones. Modelled expected incidence rates were higher in all age groups in comparison to what was observed. Assuming a causal RR of 2.5 among 'heavy users' gave 2038 expected cases in all age groups.

Limitations: This is an ecological trends analysis, with no data on individual mobile phone use and outcome.

What this study adds: The observed stability of brain cancer incidence in Australia between 1982 and 2012 in all age groups except in those over 70 years compared to increasing modelled expected estimates, suggests that the observed increases in brain cancer incidence in the older age group are unlikely to be related to mobile phone use. Rather, we hypothesize that the observed increases in brain cancer incidence in Australia are related to the advent of improved diagnostic procedures when computed tomography and related imaging technologies were introduced in the early 1980s.

1. Introduction

The first call made on a mobile phone in Australia occurred on February 23, 1987. In the 29 years since, usage has grown rapidly, with over 90% of all Australians using the devices today. In 2011 the International Agency for Research on Cancer Working Group classified radiofrequency (RF) electromagnetic waves as 'possibly carcinogenic to humans'. Radiofrequency waves are emitted by electronic devices including radar, TV, radio, WiFi, Bluetooth, microwave and cordless devices and mobile phones. IARC issued a classification score of 2B for radio frequency electromagnetic radiation, which is defined as "*A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence*". The Working Group identified several methodological issues regarding measurement of RF from mobile phones and

☆ Dr. Freddy Sitas, a co-author of this paper, is an Associate Editor of Cancer Epidemiology. The Editor-in-Chief of Cancer Epidemiology managed the editorial process for this manuscript independently from Dr. Sitas and the manuscript was subject to the Journal's usual peer-review process.

* Corresponding author.
E-mail address: simon.chapman@sydney.edu.au (S. Chapman).

http://dx.doi.org/10.1016/j.canep.2016.04.010
1877-7821/

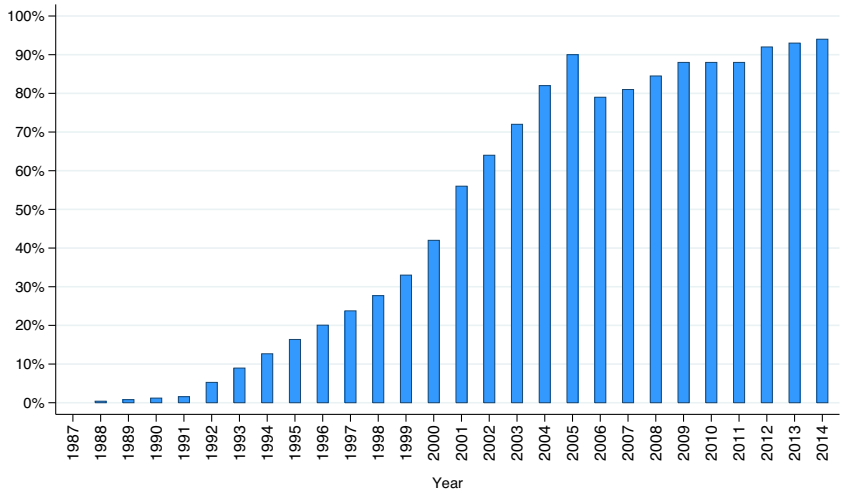

Fig. 1. Percentage of Australians with mobile phone accounts.

noted the inconsistency and poor replicability of most laboratory studies [1].

This view was strengthened by several independent national environmental health agencies. For example, a 2012 UK report of the Independent Advisory group on non-ionising radiation [2] and a 2015 review by the European Union's Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) [3] both found no evidence of health effects of mobile phones to humans at current EMF dosage levels. In addition, a number of groups in several countries have documented a stable or declining incidence of brain cancers [4–8]. In the USA [8] and the Nordic countries [7] several risk and latency scenarios about mobile phone use and brain cancers were investigated with the findings being consistent with a null effect or longer latency periods. However, Morgan et al. [9] recently argued that risks of mobile phone use are higher than previously thought, with relative risks in relation to a 'decade long mobile phone use' said to be between 1.8 and 7.8 (Ref. [9], Table 1). By contrast cohort studies in Denmark and the UK published after the IARC report [10,11] found a null effect.

Given these uncertainties, and continuing prominent media coverage of predictions of an eventual increasing incidence of mobile phone caused brain cancers, we investigated the association between mobile use coverage and brain cancer incidence in Australia using an ecological study design.

2. Methods

We obtained data on the percentage of all Australians with mobile phone accounts[1] from the Australian Mobile Telephone Association (AMTA) and the Australian Communications and Media Authority (ACMA) annual reports (see Fig. 1). Data on account holders for 1987–1990 and 1992–1997 were missing and were estimated by linear interpolation. Data by age and gender were unavailable.

These data refer to "accounts", not individuals. In 2014 there were 31.01 mobile phone million accounts in a population of some 23.86 million [12]. In 2009 (latest available data) nearly one in three children aged 5–14 and 76% of 12–14 years old had their own mobile phone [13]. The exact number of individuals using mobile phones in Australia is unknown but estimated to be approximately 90% of all people.

Reporting of incident invasive cancer is mandatory in all Australian states and territories, which send data to the Australian Institute of Health and Welfare (AIHW) for national reporting. We used AIHW national tabulated incidence data from 1982 to 2012 (the latest data available) for brain cancer [14] (80% of which are gliomas) to calculate (3-year smoothed) age-adjusted incidence rates (per 100,000) overall and for four age groups (20–39, 40–59, 60–69, 70–84 years). Data become unreliable after 84 years because they are combined into one category of 85 years and over.

To illustrate the purported effect of mobile phones on brain cancer incidence, we assumed a 10-year lag period between exposure to mobile phones, and estimated expected rates per age group over 20 years (R_E) assuming prevalence/use to be spread evenly across all age groups (due to lack of age specific usage data) (P_{mob}), by multiplying the pre-mobile phone baseline rate in 1982–1987 ($R_{1982-1987}$) by a (conservative) relative risk (RR_{mob}) of 1.5, the RR found for 'ever-users' of mobile phones, estimated by Hardell et al. [15], used by Little et al. [8] using the formula for each age group: $R_E = (R_{1982-1987} \times P_{mob} \times RR_{mob}) + (R_{1982-1987} \times (1 - P_{mob}))$, and then obtaining the all-age rate by summation of the age specific groups. Using a recent paper [9] we also modelled brain cancer incidence using a RR of 2.5, among heavy users (>896 h cumulative use), and assumed that 19% of the Australian population falls in this top category, based on data from the INTERPHONE study [16], an international pooled analysis of studies on the association between mobile phone use and brain cancer (which defines heavy users slightly differently, as being those with >735 h cumulative use). Confidence intervals and statistical significance of observed and expected incidence rates were calculated using formulas in Jensen et al. [17].

3. Results

Fig. 1 shows mobile phone use in Australia from 1987 to 2014 increasing from 0% in 1987 to 94% in 2014.

A total of 19,858 males and 14,222 females aged 20–84 were diagnosed with brain cancer between 1982 and 2012. Brain cancer ranks as the 12th most common cancer in Australia, representing 1.4% of all newly diagnosed cancers [14].

Fig. 2 and Supplementary Tables S1 and S2 show the (3-year smoothed) observed and modelled expected rates per 100,000 population for brain cancer incidence for this period.

[1] Mobile phone plans are only available in Australia for people aged 18 or older, but many children have them supplied by parents.

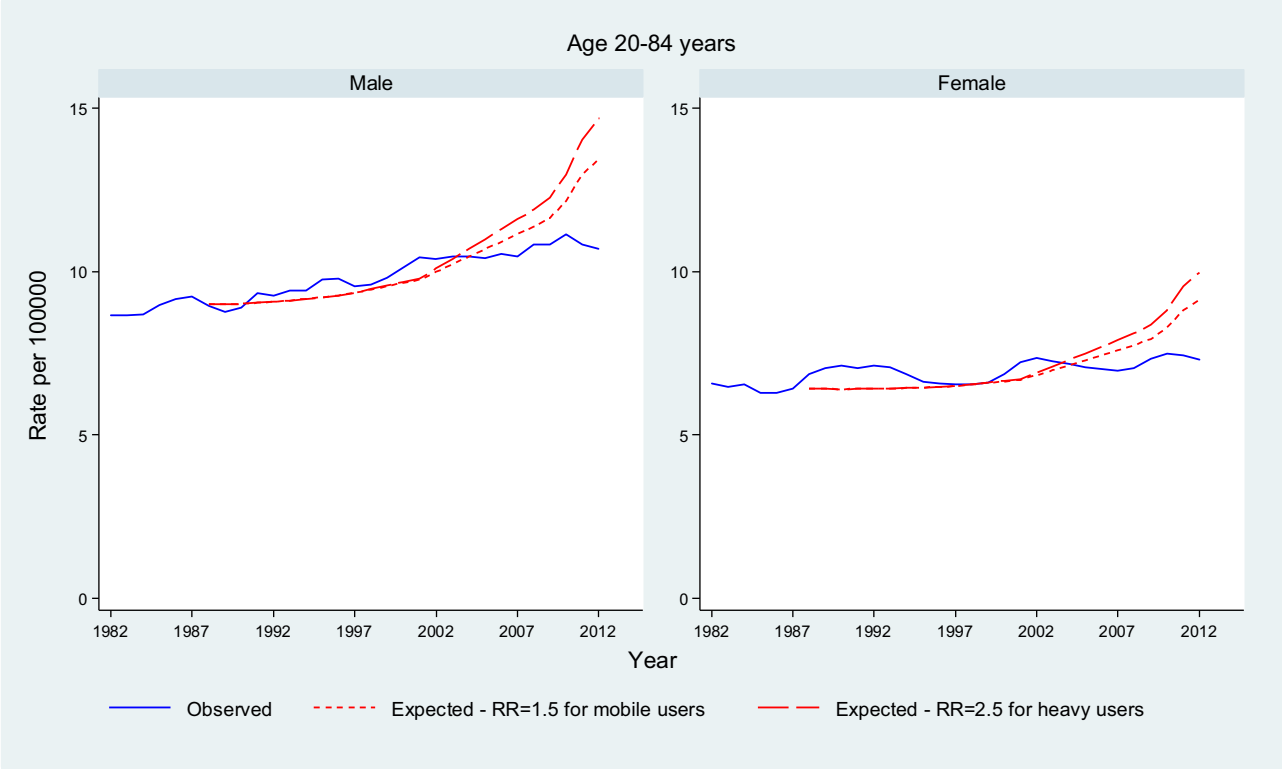

Fig. 2. Observed and expected brain cancer incidence rate in Australia (age standardised, World) assuming a RR of 1.5 for mobile users and RR of 2.5 for heavy users compared to non-users with a 10-year lag time.

Age adjusted brain cancer incidence rates rose slightly over time in males ($p < 0.05$) but not in females ($p > 0.05$). In 2012, rates were about 50% higher in males (8.7 per 100,000, 95%CI = 8.1–9.3) than in females (5.8 per 100,000, 95%CI = 5.3–6.3), $p < 0.001$ (Table S2). Using modelled assumptions of a 'causal' RR of 1.5 and a lag period of 10 years, expected incidences would have been significantly greater in both males and females (11.7; CI = 11.0–12.4, and 7.7; CI = 7.2–8.3, respectively), $p < 0.01$. Using our modelled assumptions, in 2012, 1867 cases (M&F combined) of brain cancer were expected vs. 1434 observed. Assuming a causal RR of 2.5 in 'heavy users' the expected incidences are increased further, to 2038.

Fig. 3 shows the observed and modelled expected age specific incidences of brain cancer across four age groupings, separately for males and females. In the oldest age group, 70–84 years, we observed an increase in brain cancer incidence in keeping with modelled expected incidence rates, however, this increase began from 1982 before the introduction of mobile phones. In all the remaining age groups, modelled expected incidence rates are significantly higher in comparison to what was observed.

4. Discussion

We used all the national incident brain cancer registrations available through Australia's high quality state and territory population-based cancer registration system. Registration is mandatory and histological verification rates exceed 85%. All registries conform to the International Agency for Research on Cancer's criteria for population based cancer registration, are "A" rated and have their data published in the "Cancer Incidence in Five Continents" series [18].

Publicly available Australian individual mobile phone usage data are unavailable in Australia, so of necessity, our analysis is an ecological trends analysis. Notwithstanding limitations of using subscription data to derive individual use patterns, we assumed phone use to be equal across all ages and between males and females. In Denmark in 1982–1995, for example when the prevalence of self reported use was just 19%, the predictive value of subscription data in ascertaining regular use was 56% [19]. Early mobile phones and accounts were very expensive by today's standards. Early subscribers were dominated by middle-aged working men on company mobile phone subscriptions, in Denmark and also in Australia. As costs fell dramatically, subscriptions rapidly extended throughout the population. This means that the per capita subscription rate we used would overestimate prevalence of use in males and underestimate it in females. While this may have pertained to the early years of mobile phone use in Australia, the picture changed quickly to almost full coverage of mobile phone use (Fig. 1). We had no data on the proportion of heavy users in Australia, and so assumed 19% of heavy mobile phone usage in Australia based on the INTERPHONE data [16].

Large proportions of Australians have been exposed to mobile phone and other EMR since the early 1990s and in 2012 (the latest available year for cancer incidence data), approximate adult per capita cell phone use was over 90% (Fig. 1). In the 25 years since the rapid and widespread adoption of mobile phones in Australia, the incidence of brain cancer has not risen in any age group other than those aged 70–84 years of age. However, in this age group, the increase in the incidence of brain cancer started before 1987, the advent of mobile telephony in Australia. While we have no Australian data on this, we suspect the population aged >70 would be those with the lowest prevalence of mobile phone use. Such an increase in cancer restricted to one age group is more consistent with improvements in access to diagnostic methods in older age groups over time.

The same observation was noted in other similar studies of brain tumours and cancers from New Zealand [4], Australia [5], England [6] and the Nordic countries [7]. In Australia, Dobes et al.

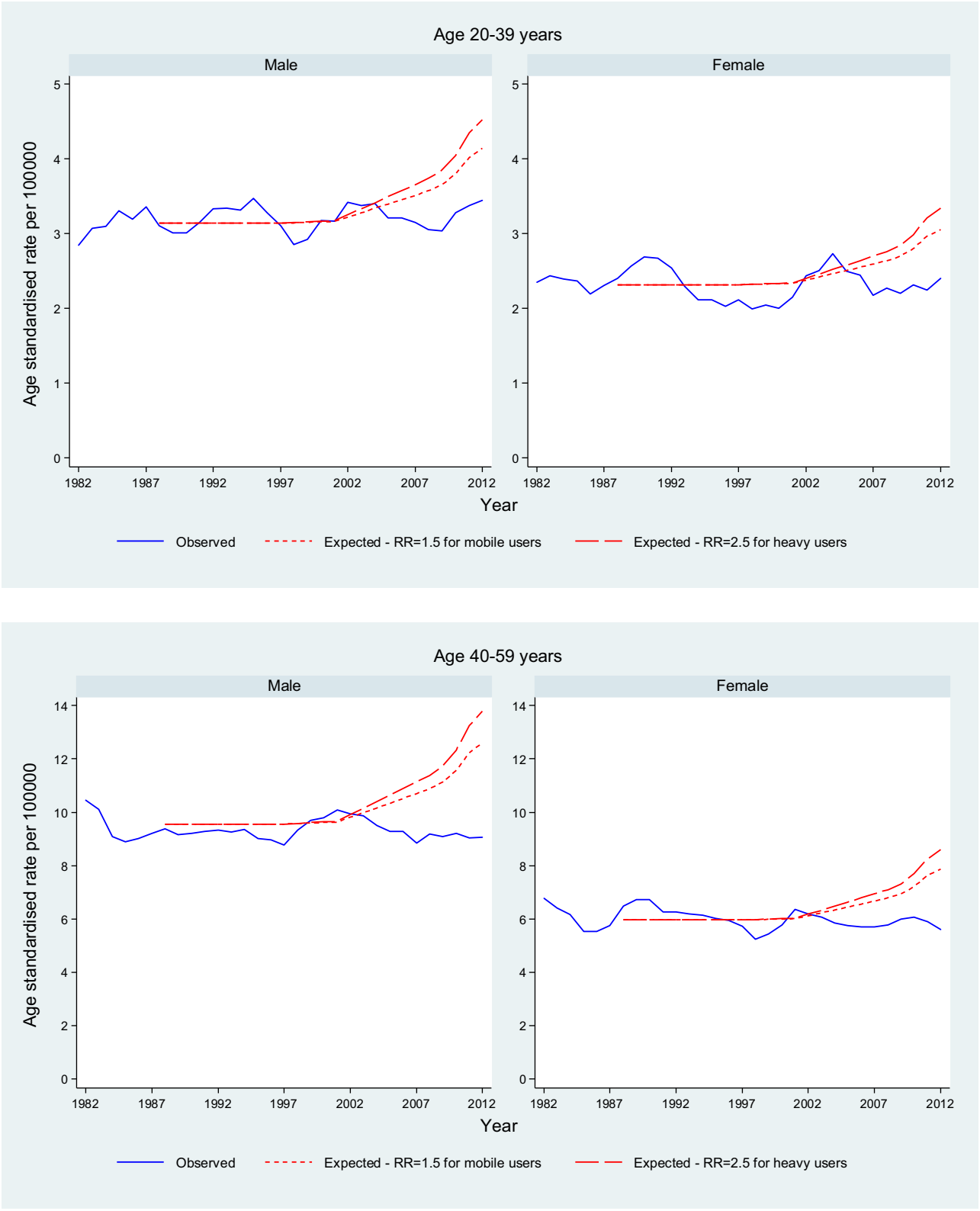

Fig. 3. Observed and modelled expected estimates of brain cancer incidence in Australia, in four age groups, assuming a RR = 1.5 for mobile users and RR = 2.5 for heavy users compared to non-users with a 10-year lag time.

[5] analysed 7251 histologically confirmed cases of brain cancer from all pathology and neurosurgical centres and major teaching hospitals in NSW and ACT between 2000 and 2008 and, in keeping with our data, found an increase in incidence in those aged 65 and over and a decline in Schwannomas (acoustic neuromas, not malignant and not reported by cancer registries).

In the USA, mobile phones have been available for the longest period of time (33 years, since 1983). In the 29 years of mobile

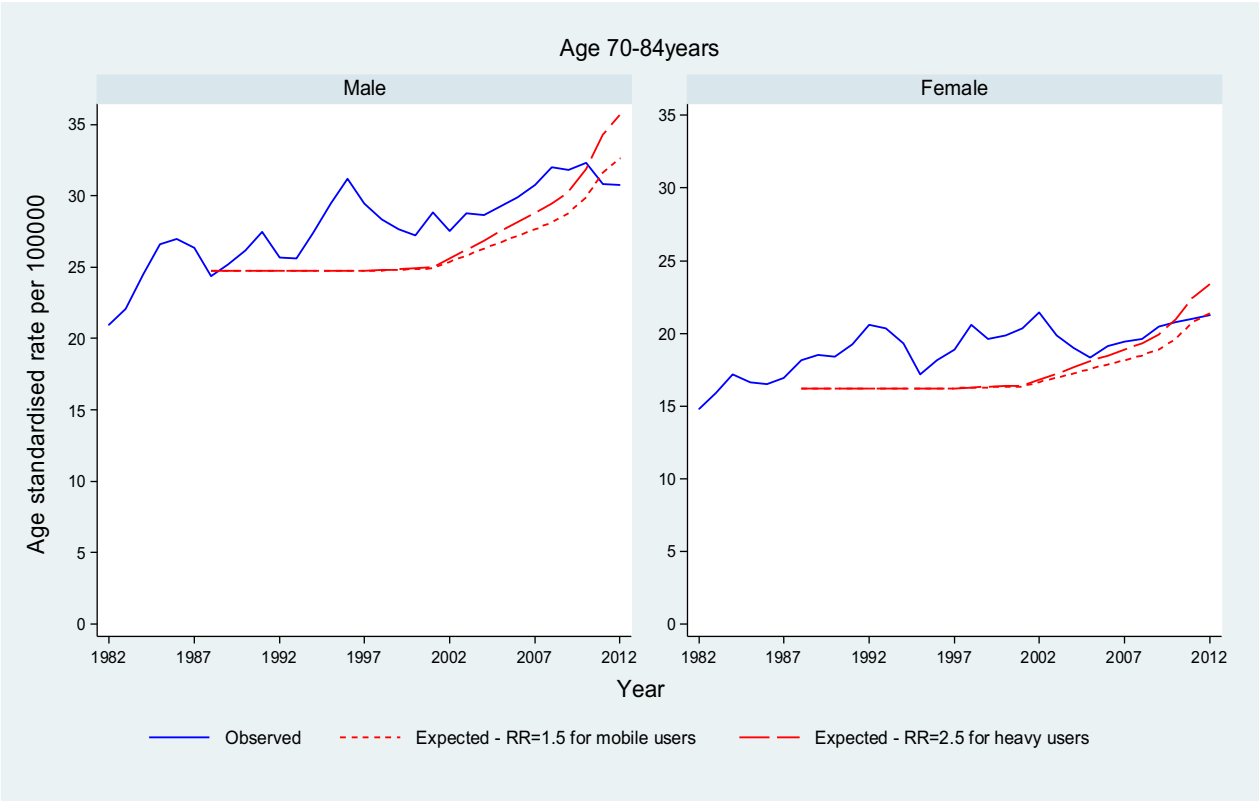

Fig. 3. (Continued)

telephony in Australia, assuming a purported RR of 1.5 and a latency of 10 years we predicted a 30% increase in brain cancer incidence. Likewise, a similar modelling study of USA brain cancer incidence which assumed brain cancer risks of those who had ever used a mobile phone would be 1.5 fold higher than those who never used them [8], predicted a 40% increase in brain cancer incidence based on a 10-year latency period, but no such increase has yet been observed in either country. We chose conservative

estimates of RR 1.5 and 2.5 to model our assumptions. Higher RRs would have yielded even higher expected numbers.

The incidence of brain cancer in Australia from 1982 to 2012 in females has been consistently some 50% lower than that in males. Data on mobile phone use by gender are sparse in Australia, but in New South Wales, the most populous state (7.6 m people out of 23.5 m) 20.9% of adult males and 19.5% females are exclusive mobile users and 71% reported dual mobile and landline use [20]. If one assumes those with dual use are also approximately evenly distributed between the sexes and these results generalizable across the rest of Australia, it is difficult to reconcile this prevalence of exposure with the observed sex differences in brain cancer patterns in Australia. These have been roughly constant (in mortality) since 1969 (the earliest records available) with females having 57% lower mortality compared to males [14]. We know of no data that show that women speak for about half as long on mobile phones as men. This is a further counterfactual to the hypothesis that mobile phone EMR causes brain cancer.

As concluded by post-IARC [1] international reviews [2,3], other similar descriptive studies [4–8], and cohort studies [10,11] we hypothesize that the observed increase in brain cancer in Australia in older age groups is due to improved diagnostic acuity. Computed tomography, magnetic resonance imaging and related techniques, introduced in Australia in the late 1970s, are able to discern brain tumours which could have otherwise remained undiagnosed [21]. It has long been recognised that brain tumours mimic several seemingly unrelated symptoms in the elderly including stroke and dementia (e.g. [22]), and so we postulate that their diagnosis had been previously overlooked.

The causes of brain cancer are unknown but given current evidence it is unlikely that the modest increases in brain cancer observed in the older age groups can be attributed to the widespread use of mobile phones. Large cohort studies are underway [23], and they may shed further light on the health effects of mobile phones. Ionising radiation causes DNA damage in brain cells and it is thought that the latency between exposure and cancer is about 5 or more years [8,24]. EMF radiation is non-ionising, unlikely to be causing DNA damage directly and more likely to affect cells at a later stage of carcinogenesis. For these reasons, Little et al. argue that exposure to EMF radiation is more likely to have a shorter latency [8,24], in which case the putative effects on brain cancer incidence would have happened sooner (and the number of expected cases would have been greater). Until better laboratory information is available regarding the type of damage EMF radiation actually causes in human brain cells, assumptions around latency between EMF exposure and increased risk of brain cancer remain speculative.

Morgan et al. [9] claim that "the latency reported between known causes of brain cancer and development of the disease appears to range from 10 to 50 years". However they report results of increased risks between 1 and 10 years post-use, which if true, would imply latencies between mobile phone use and brain cancer of below 10 years. Claims that insufficient years of exposure to mobile phone radiation have yet occurred for the hypothesised increases in cancer incidence to become manifest fail to account for why there has been no observed rise in brain cancer in any age group in Australia (this study and Ref. [5], nor in England [6], New Zealand [4], and the Nordic countries [7]) across 25 years other than in the most aged group, which we have discussed. Such a hypothesis would require an induction time incidence profile where there was no rise for 30 or more years, followed by a sudden rise after that time. There are no precedents in cancer epidemiology for such a profile. Induction times always will have a distribution, and a risk would be expected to increase from the minimum (earliest) induction time.

5. Conclusion

After nearly 30 years of mobile phone use in Australia among millions of people, there is no evidence of any rise in any age group that could be plausibly attributed to mobile phones.

Conflict of interest

We have no conflicts of interest to declare and confirm that we did not receive any funding for the study reported in this paper.

Authors' contribution

SC conceived the study, FS devised the study design, QL and LA performed the analyses. All the authors contributed to the manuscript and approved the final copy.

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.2016.04.010.

References

[1] IARC Working Group, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Non Ionising Radiation. Part 2: Radio Frequency Electromagnetic Fields, vol. 102, IARC, Lyon, 2013.
[2] Health Protection Agency, Health effects from radiofrequency electromagnetic fields. Report of an independent advisory group on non-ionizing radiation. Documents of the Health Protection Agency. Radiation, Chemical and Environmental Hazards, https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/333080/RCE-20_Health_Effects_RF_Electromagnetic_fields.pdf.
[3] European Union's Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), Potential health effects of exposure to electromagnetic fields (EMF), 27 January 2015. http://ec.europa.eu/health/scientific_committees/consultations/public_consultations/scenihr_consultation_19_en.htm (accessed 17.04.16).
[4] J.-H.S. Kim, S.J. Ioannides, J.M. Elwood, Trends in incidence of primary brain cancer in New Zealand, 1995–2010, Aust. N. Z. J. Public Health (2015) 148–152, doi:http://dx.doi.org/10.1111/1753-6405.12338 Epub 25 February 2015. PubMed PMID: 25715883.
[5] M. Dobes, V.G. Khurana, B. Shadbolt, S. Jain, S.F. Smith, R. Smee, M. Dexter, R. Cook, Increasing incidence of glioblastoma multiforme and meningioma, and decreasing incidence of Schwannoma (2000–2008): findings of a multicenter Australian study, Surg. Neurol. Int. 2 (2011) 176, doi:http://dx.doi.org/10.4103/2152-7806.90696 Epub 13 December 2011.
[6] F. de Vocht, I. Burstyn, J.W. Cherrie, Time trends (1998–2007) in brain cancer incidence rates in relation to mobile phone use in England, Bioelectromagnetics 32 (May (5)) (2011) 1998–2007, doi:http://dx.doi.org/10.1002/bem.20648 Epub 28 January 2011. PubMed PMID: 21280060.
[7] I. Deltour, A. Auvinen, M. Feychting, C. Johansen, L. Klaeboe, R. Sankila, J. Schüz, Mobile phone use and incidence of glioma in the Nordic countries 1979–2008: consistency check, Epidemiology 23 (2) (2012) 301–307, doi:http://dx.doi.org/10.1097/EDE.0b013e3182448295.
[8] M.P. Little, P. Rajaraman, R.E. Curtis, S.S. Devesa, P.D. Inskip, P.D. Check, M.S. Linet, Mobile phone use and glioma risk: comparison of epidemiological study results with incidence trends in the United States, BMJ 344 (2012) e1147, doi:http://dx.doi.org/10.1136/bmj.e1147.
[9] L.L. Morgan, A.B. Miller, A. Sasco, D.L. Davis, Mobile phone radiation causes brain tumours and should be classified as a probable human carcinogen, Int. J. Oncol. 46 (5) (2015) 1865–1871.
[10] P. Frei, A.H. Poulsen, C. Johansen, J.H. Olsen, M. Steding-Jessen, J. Schüz, Use of mobile phones and risk of brain tumours: update of Danish cohort study, BMJ 343 (2011) d6387, doi:http://dx.doi.org/10.1136/bmj.d6387.
[11] V.S. Benson, K. Pirie, J. Schüz, G.K. Reeves, V. Beral, J. Green, Million Women Study Collaborators, Mobile phone use and risk of brain neoplasms and other cancers: prospective study, Int. J. Epidemiol. 42 (3) (2013) 792–802, doi:http://dx.doi.org/10.1093/ije/dyt072 Epub 8 May 2013.
[12] J. Thomas, Australia has more phones than people, SBS January 2, 2015 http://www.sbs.com.au/news/article/2015/01/02/australia-has-more-phones-people.
[13] Australian Bureau of Statistics. 1370.0 Measures of Australia's progress. Children and mobile phones. September 9, 2010 http://www.abs.gov.au/

ausstats/abs@.nsf/Lookup/by%20Subject/1370.0~2010~Chapter~Children%20and%20mobile%20phones%20(4.8.5.3.2).
[14] Australian Institute of Health and Welfare (AIHW), 2015. Australian Cancer Incidence and Mortality (ACIM) Books: Brain Cancer. AIHW, Canberra. http://www.aihw.gov.au/acim-books. Updated January 2015.
[15] L. Hardell, M. Carlberg, K. Hansson Mild, Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects, Int. J. Oncol. 38 (5) (2011) 1465–1474, doi:http://dx.doi.org/10.3892/ijo.2011.947 Epub 17 February 2011.
[16] The Interphone Study Group, Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study, Int. J. Epidemiol. 39 (3) (2010) 675–694, doi:http://dx.doi.org/10.1093/ije/dyq079.
[17] O.M. Jensen, D.M. Parkin, R. MacLennan, C.S. Muir, R.G. Skeet, Cancer Registration: Principles and Methods, IARC Scientific Publication No. 95, Lyon, 1991.
[18] IARC, Cancer Incidence in Five Continents, IARC, Lyon, 2016. http://ci5.iarc.fr/Default.aspx.
[19] J. Schüz, C. Johansen, A comparison of self reported cellular telephone use with subscriber data: agreement between the two methods and implications for risk estimation, Bioelectomagnetics 28 (2) (2007) 130–136, doi:http://dx.doi.org/10.1002/bem.20197.
[20] M.L. Barr, R.A. Ferguson, D.G. Steel, Inclusion of mobile telephone numbers into an ongoing population health survey in New South Wales Australia, using an overlapping dual-frame design: impact on the time series, BMC Res. Notes 7 (August (12)) (2014) 517, doi:http://dx.doi.org/10.1186/1756-0500-7-51.
[21] J.D. Mathews, A.V. Forsythe, Z. Brady, M.W. Butler, S.K. Goergen, G.B. Byrnes, G. G. Giles, A.B. Wallace, P.R. Anderson, T.A. Guiver, P. McGale, T.M. Cain, J.G. Dowty, A.C. Bickerstaffe, S.C. Darby, Cancer risk in 680,000 people exposed to computed tomography scans in childhood or adolescence: data linkage study of 11 million Australians, BMJ (2013), doi:http://dx.doi.org/10.1136/bmj.f2360.
[22] M. Salcman, Brain tumors in elderly patients, Am. Fam. Physician 27 (4) (1983) 137–143.
[23] J. Schüz, P. Elliott, A. Auvinen, H. Kromhout, A.H. Poulsen, C. Johansen, J.H. Olsen, L. Hillert, M. Feychting, K. Fremling, M. Toledano, S. Heinävaara, P. Slottje, R. Vermeulen, A. Ahlbom, An international prospective cohort study of mobile phone users and health (Cosmos): design considerations and enrolment, Cancer Epidemiol. 35 (1) (2011) 37–43, doi:http://dx.doi.org/10.1016/j.canep.2010.08.001.
[24] M.P. Little, R. Wakeford, E.J. Tawn, S.D. Bouffler, A. Berrington de Gonzalez, Risks associated with low doses and low dose rates of ionising radiation: why linearity may be the best we can do, Radiology 251 (2009) 6–12.