# Cancers in Australia attributable to exposure to solar ultraviolet radiation and prevented by regular sunscreen use

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n 1992, the International Agency for Research on Cancer (IARC) concluded that ultraviolet radiation (UVR) was carcinogenic to humans.<sup>1</sup> The main source of exposure to UVR is the sun (solar radiation). In 2009, IARC confirmed that there is sufficient evidence that solar radiation causes cutaneous malignant melanoma, and squamous cell carcinoma (SCC) and basal cell carcinoma (BCC) of the skin.<sup>2</sup>

Exposure to UVR can also occur through artificial sources, including UV-emitting tanning devices (solaria), medical and dental applications and industrial sources (e.g. electric arc welders, and some fluorescent and tungsten–halogen lamps). IARC has concluded that these artificial sources of UVR are also carcinogenic to humans.<sup>2</sup> In Australia, the overwhelming source of UVR is from the sun. Despite the importance of artificial UVR exposures, this analysis focuses on quantifying the contributions of natural UV exposure to the incidence of human cancer.

Epidemiological evidence for the causal role of solar UVR exposure in the development of melanoma and keratinocyte cancers (basal cell carcinomas [BCC] and squamous cell carcinomas [SCC] of the skin) includes observations of higher incidence rates in fair-skinned, sun-sensitive people than dark-skinned people, and higher incidence in locations closer to the equator.<sup>3,4</sup> Fairskinned migrants from high to low latitude countries have a lower melanoma incidence

#### Abstract

**Objectives**: To estimate the proportion and numbers of cancers occurring in Australia attributable to solar ultraviolet radiation (UVR) and the proportion and numbers prevented by regular sun protection factor (SPF) 15+ sunscreen use.

**Methods:** We estimated the population attributable fraction (PAF) and numbers of melanomas and keratinocyte cancers (i.e. basal cell carcinomas and squamous cell carcinomas) due to exposure to ambient UVR resulting from residing in Australia versus residing in the UK (for melanoma) or Scandinavia (for keratinocyte cancers). We also estimated the prevented fraction (PF): the proportion of cancers that would have occurred but were likely prevented by regular sunscreen use.

**Results**: An estimated 7,220 melanomas (PAF 63%) and essentially all keratinocyte cancers occurring in Australia were attributable to high ambient UVR levels in Australia. We estimated that regular sunscreen use prevented around 14,190 (PF 9.3%) and 1,730 (PF 14%) people from developing SCC and melanoma, respectively.

**Conclusions**: Although our approach was conservative, a high proportion of skin cancers in Australia are attributable to high ambient levels of UVR. Prevailing levels of sunscreen use probably reduced skin cancer incidence by 10–15%.

**Implications**: Most skin cancers are preventable. Sunscreen should be a component of a comprehensive sun protection strategy.

Key words: population attributable fraction, melanoma, skin cancer, solar radiation, sunscreen

rate than fair-skinned, native-born residents, and vice versa.<sup>5,6</sup> In addition, people with a past history of keratinocyte cancer<sup>7</sup> or solar keratoses<sup>8,9</sup> (widely considered as markers of accumulated sun exposure and phenotypic sensitivity) have markedly higher risks of melanoma than people with no history of keratinocyte cancers. Molecular studies have identified UV-specific mutations in the DNA of key regulatory genes in melanomas.<sup>10,11</sup> These epidemiologic observations are supported by a strong body of experimental evidence including animal, cellular and molecular studies.

Randomised trials have demonstrated that regular sunscreen use reduces the incidence of solar keratoses,<sup>12</sup> SCC<sup>13</sup> and possibly melanoma<sup>14</sup> in susceptible individuals. Interestingly, and despite the conclusive evidence that solar UVR causes BCC, the

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<sup>3.</sup> Cancer Research UK, Manchester Institute and Institute of Inflammation and Repair, University of Manchester, United Kingdom

only trial to examine this endpoint observed no effect of regular sunscreen on BCC incidence.<sup>13,15</sup> Various explanations for the null result are possible, such as the 'critical period' for sunlight on BCC was at younger ages than the lowest age for trial entry or that sunscreen really has no effect, but these remain to be explored. Based on current knowledge, we can conclude only that sunscreen prevents SCC, possibly prevents melanoma and its effect on BCC is uncertain.

Here, we estimated the population attributable fractions (PAF) and numbers of cutaneous melanomas and keratinocyte cancers arising in the Australian population that were attributable to exposure to solar radiation. We also estimated the proportion of melanomas and cutaneous SCCs that were likely to have been prevented by regular sunscreen use in the Australian population (the 'prevented fraction', PF).

#### **Methods**

## Solar UVR – population attributable fractions

In calculating the fractions of melanomas and keratinocyte cancers attributable to UVR, the traditional formula using population prevalence of exposure and relative risk of cancer is difficult to apply. This is because exposure to sunlight is ubiquitous and quantification of accumulated personal dose is prone to error. Instead, we adopted an approach similar to previous reports<sup>16,17</sup> in which the fraction of melanoma cases attributable to solar UVR was calculated as the proportional difference between the melanoma incidence in 'exposed' and 'unexposed' populations of similar ethnic composition. While this method cannot provide a precise measure of the excess burden of melanoma due to any UVR exposure, we believe it provides a clearer sense of the burden due to high levels of ambient UVR experienced by the Australian population when compared to ethnically similar populations residing in environments with far lower levels of ambient UVR.<sup>18,19</sup> The annual total UVR in Australian cities is about 3-5 times higher than reported for the UK (e.g. 9,760 standard erythermal dose [SED] for Sydney and 2,950 SED for Leeds).<sup>20</sup>

We used the following formula to calculate the PAF:

$$PAF = (I_p - I_u)/I_p$$

where  $I_p$  is the incidence of melanoma in the Australian population, and  $I_u$  is the incidence in the reference population.

For our primary melanoma analysis, we estimated the difference between the observed numbers of melanoma cases in Australian residents<sup>21</sup> (i.e. 'exposed' to high ambient UVR in Australia) and the expected number of cases assuming the population was exposed to levels of ambient UVR experienced by an 'ancestral' population for many Australians. As our reference, we used the UK population (2009–11).<sup>22</sup> This reference population was chosen on the basis that the majority of susceptible Australian residents trace their ancestry to northern Europe, and particularly the British Isles,<sup>23</sup> and because the difference in average ambient UV levels in each location is substantial.<sup>24,25</sup> As a sensitivity analysis we used the same reference population as Parkin and colleagues<sup>17</sup>(i.e. the 1903 birth cohort from the South Thames, UK).

The total number of cancers attributable to UVR was also expressed as a percentage of the total number of all incident cancers (excluding basal cell and squamous cell carcinoma of the skin) recorded in the Australian population (children and adults) in 2010.

## Potential impact of changing UVR exposure in the Australian population

In our primary analyses, we used the contemporary UK population as the comparator, which generated a PAF representing the maximum but unattainable target for solar protection strategies in the Australian population. To estimate the fraction of melanomas that might feasibly be prevented by population-wide behaviour change, we performed additional analyses:

- 'Time shift' analysis: the melanoma incidence rates experienced by the Australian population in 1982 were applied to the 2010 Australian Estimated Resident Population.
- 2. 'Geographic shift' analysis: we estimated the number of melanomas that would have occurred if the population residing in each state or territory experienced melanoma at the rate of the population in the nearest state with lower melanoma incidence. For Queensland, we used New South Wales incidence rates;<sup>26</sup> for New South Wales, Australian Capital Territory, and Western Australia, we used Victorian incidence rates:<sup>27</sup> for Victoria and the Northern Territory we used South Australian incidence rates,<sup>28</sup> since rates in South Australia were the lowest in mainland Australia (see supplementary file: Table S1, available with the online version of this

article). South Australia and Tasmania were unchanged.

We used a similar approach to calculate the population fraction of keratinocyte cancers attributable to solar UVR (i.e. calculating the proportional difference between incidence in 'highly sun exposed' Australian population and 'minimally sun exposed' Scandinavian populations). We sourced incidence rates from the 2002 National Non-melanoma Skin Cancer Survey.<sup>29</sup> The choice of a reference population was limited by the availability of reliable incidence data; we used incidence rates from Nordcan for all participating countries (Denmark, Sweden, Norway, Finland, Iceland and the Faroe Islands).<sup>30</sup> Incidence rates of BCC and SCC were not provided separately in the Nordcan database, so analyses were conducted for all keratinocyte cancers combined. We performed a sensitivity analysis using the 2002 time period. While more reliable than most countries, incidence of keratinocyte cancers in Nordcan may be under-reported by up to 30%,<sup>31</sup> so we performed a sensitivity analysis assuming this extent of underreporting.

#### Sunscreen-prevented fractions

Relative Risk estimates

The relative risk estimate for the protective effect of regular sunscreen use on SCCs of the skin was sourced from long-term follow-up of participants of the Nambour Skin Cancer Prevention Trial.<sup>15</sup> The Nambour trial commenced in 1992 and randomised 1,621 participants to receive either regular application of broad-spectrum sunscreen (SPF 16) to the head, neck, arms and hands (intervention arm) or discretionary sunscreen (control arm).<sup>14,15</sup> The age range for trial participants was 25-75 (median 48 years). We used the relative risk for SCC incidence (persons affected) over 11 years from commencement of the intervention<sup>15</sup> (RR=0.65, 95%CI 0.45-0.94).

The evidence that regular sunscreen use prevents melanoma is weaker than for SCC. Again, we sourced effect estimates from the only randomised trial to assess the efficacy of sunscreen on melanoma, the Nambour Skin Cancer Prevention Trial, which reported a marginally significant reduced incidence among regular sunscreen users (RR=0.50, 95%CI 0.24–1.02).<sup>14</sup>

*Exposure prevalence estimates* We sought population-based prevalence estimates for sunscreen use that best aligned with the intervention delivered in the randomised trial. Of several possible sources of data, we selected the 2010 NSW Population Health Survey as best meeting this criterion.<sup>32</sup> As the NSW sunscreen prevalence data may not have reflected national patterns of sunscreen use (or use during earlier time periods arguably more relevant to melanoma development), we used prevalence data from other population-based surveys to conduct sensitivity analyses: the NSW Population Health Survey 2004,<sup>33</sup> Victorian Sun Survey 2006-07,<sup>34</sup> Queensland Self-reported Health Status for 2009,<sup>35</sup> and the National Sun Protection Survey 2010-11.<sup>36</sup> The questions relating to sunscreen use in each of these surveys are summarised in supplementary file: Table S2, available online.

#### Cancer incidence data

Using age-specific rates obtained from the 2002 National Non-melanoma Skin Cancer Survey<sup>29</sup> and the estimated resident population for 2008, it was estimated that there were 83,901 new cases of SCC of the skin in men and 53,699 new cases of SCC of the skin in women in 2008.<sup>29</sup> For consistency with the SCC analyses, we used incidence data for 2008 for melanoma of the skin (11,029 cases).<sup>29</sup>

#### Statistical analysis

As sunscreen has a protective effect, and as the natural exposure level is zero, the Prevented Fraction (PF) is the most appropriate measure to quantify population impact:

$$PF_x = P_x \left(1 - RR\right)$$

where  $P_x$  is the prevalence of regular sunscreen use by sex category.

We estimated the number of SCCs prevented through regular sunscreen use using the following formula:

Number of prevented cancers =  $\sum \left(\frac{N_{\chi}}{1 - PF_{\chi}}\right) - N_{\chi}$ 

where  $N_x$  is the number of observed cancers in 2008 in each sex category and  $PF_x$  is the prevented fraction in each sex category.

The overall prevented fraction was then calculated by summing the total number of prevented SCCs or melanomas across all categories of age and sex, and expressing this sum as a percentage of the total numbers of observed plus prevented cancers.

#### Results

#### Solar UVR

## Number and proportion of melanomas attributable to ambient UVR exposure

In our primary analysis applying contemporary UK melanoma rates to the Australian population, 7,220 melanomas in 2010 (4,668 in men and 2,552 in women) were attributable to the ambient UVR exposure experienced by Australian residents. This represents 63% of the observed melanoma cases in Australia in that year (Table 1). Our sensitivity analysis using the historical UK comparison (i.e. the 1903 birth cohort from the South Thames, UK) resulted in a PAF of 95% (97% for men and 92% for women).

### Potential impact of changing UVR exposure in the Australian population

The potential impact on melanoma incidence of reducing solar UVR exposure in the Australian population is summarised in Table 2.

#### Time shift analysis

Briefly, assuming an intervention was able to successfully decrease sun exposure such that the Australian adult population of 2010 had developed melanoma at the rates prevailing in 1982, we estimate that 5,148 fewer cases of melanoma would have occurred (a reduction in incidence of 45%).

#### Geographic shift analysis

Assuming a more modest intervention, whereby the incidence of melanoma in each state or territory was reduced to the incidence observed in the nearest state with lower incidence, we estimate that 2,020 fewer cases of melanoma would have occurred (incidence reduction 18%).

Number and proportion of keratinocyte cancers attributable to ambient UVR exposure

When the age- and sex-specific incidence rates for keratinocyte cancer from the Nordic countries for 2002 were applied to the Australian population (2002), virtually 100% of keratinocyte skin cancers were attributable to sun exposure. Assuming that keratinocyte cancers were under-reported by 30% in the Nordcan database made no material difference to the PAF estimates (99.4% using 2002 incidence rates).

#### Sunscreen

Prevalence of regular sunscreen use The New South Wales Population Health Survey (2010) reported that 28% of participants always applied a broad-spectrum sunscreen (SPF 15+) to exposed skin when they were out in the sun for longer than 15 minutes.<sup>32</sup> The proportion of women (35%) who applied sunscreen regularly was higher than men (21%).<sup>32</sup>

## Proportion of keratinocyte cancers and melanomas prevented due to regular sunscreen use

Assuming the prevalence of regular sunscreen use above and the protective effects reported in the long-term follow-up of the Nambour trial, we estimated that 9.3% of Australians who would otherwise have developed cutaneous SCC in 2008 had their cancers prevented through regular sunscreen use, equating to 14,192 people (Table 3). Similarly, about 14% of people who would otherwise have developed melanoma in 2008 had their cancers prevented through regular sunscreen use; that is 1,729 prevented cases. Table 3 summarises the prevented fraction, and estimated number of prevented cases of cutaneous SCC of the skin, when other sources of sunscreen prevalence were used. In all instances, the estimated prevented fractions of SCC were higher than those derived using 2010 NSW Population Health Survey data and ranged from 11% to 17%.

#### Discussion

UVR is the major environmental cause of melanoma and keratinocyte cancers. We estimate that 63% of all melanomas and virtually all keratinocyte cancers could be attributed to the high background levels of UVR experienced by the Australian resident population. We also estimated that the number of people diagnosed with SCC in the Australian adult population in 2008 was reduced by at least 9.3% (or about 14,200 cases) because of prevailing levels of sunscreen use. Similarly, our analyses suggest that melanoma incidence was about 14% lower than would otherwise have been observed because of sunscreen use, assuming that regular sunscreen use prevents this cancer.

We employed a similar approach to previous evaluations of the fraction of melanoma cases related to UVR exposure,<sup>16,17</sup> although our choice of an 'unexposed' or 'reference' population differed from those earlier reports. Armstrong and Kricker<sup>16</sup> modelled three alternative exposed/unexposed populations: the first compared incidence in white Americans with incidence in black Americans

Table 1: Number and fraction of cutaneous melanoma cases diagnosed in Australia in 2010 attributable to the										
difference in ambient UVR exposure between Australia and the United Kingdom. Melanoma (C43) All cancer										
Age(years)	Expected	Observed	Excess att	ributable cases	Observed	All cancer®				
	Cases	cases	No.	%	cases	No.	%			
Males										
0-4	0	1	1	100.0	159	1	0.8			
5–9	0	0	0	0.0	85	0	0.1			
10-14	1	3	2	48.5	85	2	1.6			
15-19	8	1/	9	54.9	1/6	9	5.3			
20-24	21	43 105	22	50.6	310	22 62	/.U			
25-29	43 50	105	02 100	58.9	440 505	02 100	15.0			
30-34	38 79	220	100	63.9	889	100	15.8			
40-44	114	220	172	60.4	1 324	172	13.0			
45-49	137	402	265	65.8	2,448	265	10.8			
50-54	169	566	397	70.1	4,184	397	9.5			
55-59	195	688	493	71.7	6,691	493	7.4			
60-64	257	845	588	69.5	9,588	588	6.1			
65–69	228	817	589	72.1	10,143	589	5.8			
70–74	232	800	568	70.9	9,370	568	6.1			
75–79	201	683	482	70.6	7,960	482	6.1			
80-84	157	619	462	74.7	6,560	462	7.1			
85+	132	447	315	70.5	4,968	315	6.3			
TOTAL	2,032	6,700	4,668	69.6%	65,983	4,668	7.1%			
Females										
0-4	0	0	0	0.0	147	0	0.1			
5–9	0	1	1	100.0	50	1	1.8			
10–14	1	4	3	84.7	77	3	4.9			
15–19	12	18	6	29.4	138	6	3.8			
20–24	45	61	16	26.5	236	16	6.9			
25–29	81	157	76	48.3	568	76	13.3			
30-34	108	180	72	40.1	833	72	8.7			
35-39	142	248	106	43.0	1,494	106	7.2			
40-44	1/3	31/	144	45.3	2,142	144	6.7			
45-49	196	402	206	51.2	3,416	206	6.0			
50-54	199	443	244	55.1	4,396	244	5.6			
55-59	202	4/1	269	57.2	5,038	269	5.3			
00-04	220	523	297	56.9	6,004	297	5.0			
00-09	187	4/1	284	60.Z	5,859	284	4.8			
70-74 75 70	104	303	201	55.I	5,Z14	201	5.8			
13-19	142	20/	100	01.3 E9.7	4,958	100	4.5			
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Persons	2,133	U, UJ	2,332	J7.370	70,370	2,332	J.070			
				100.0	244					
U-4	0	1	1	100.0	306	1	0.5			
5-9	U			0.0	135	1	0./			
10-14 15 10	2	/	5	/0.8	102	5	3.Z			
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20-24	124	202	130	50.8	1,015	130	13.3			
25_20	100 221	00CC 168	1/2	57.8	1,420	1/Z 2/7	12.0			
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50-54 55_50	202	1,009	041 762	65.8	11 700	767	7.J 65			
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65_60	د <del>ەت</del> 115	1 אַפּג	872	0 <del>4</del> ./ 67.7	15,372	872	5.7 5.5			
70-74	396	1,200	769	66.0	10,005	769	5.3			
75_79	370	1,103	709	67.4	14,004	709	J.J 5 5			
80-84	290	940	650	69.7	11 477	650	5.5			
85+	270	20TC 803	529	65.9	10 134	529	5.7			
TOTAL	4.185	11.405	7.220	63.3%	116.580	7,220	6.2%			

(assumed to be the incidence in unexposed whites) - PAFs 96% for males and 92% for females; the second compared incidence rates in Australians to those in migrants from areas of lower ambient UVR (overall PAF 68%); and the third compared the incidence of melanoma on all body sites to the incidence on unexposed body sites (buttocks and, in women only, the scalp) - PAFs 97% in males and 96% in females. Thus, our estimate of 63% of melanomas arising in Australia were attributable to the higher UVR levels to which the population are exposed is similar in magnitude to the estimate of Armstrong and Kricker using similar methods.<sup>37</sup> Parkin and colleagues<sup>17</sup> used historical data from the South Thames region of England, defining the 1903 birth cohort as the "minimum risk" population and estimated PAFs of 90% in males and 82% in females.

For our primary analysis, we used the contemporary UK population as our reference to reduce possible bias introduced by changes in modes of diagnosis and cancer registration that almost certainly apply to historic datasets. Nonetheless, our choice of reference population was conservative, and is likely to underestimate the 'true' fraction of melanomas attributable to ambient sunlight. This is because the reference population is not 'unexposed' to UVR but rather 'less exposed than the Australian population'. Hence, the fraction we estimated was the proportion of melanomas attributable to residing in Australia rather than England. As shown by Parkin,<sup>17</sup> however, around 90% of melanomas in the UK can also be attributed to sun exposure. Thus, to estimate the 'true' fraction of melanomas in Australia attributable to any sun exposure would require comparison with a cohort that had very limited sun exposure. We are not aware of any contemporaneous cohorts that would meet this criterion, so we conducted further sensitivity analyses using the same population as Parkin and colleagues<sup>17</sup> (i.e. the 1903 birth cohort from the South Thames, UK) which resulted in a PAF more comparable with that reported by Parkin.<sup>17</sup>

A limitation of the analyses for keratinocyte cancers is the lack of reliable national incidence data, both for Australia and international comparator populations. Most Australian states and territories do not capture notifications of keratinocyte cancers, and other health registers (e.g. Medicare) do not record details of skin cancer histology. We therefore used data from a 2002 National Survey to estimate Australian

a excluding basal cell carcinoma and squamous cell carcinoma of the skin

incidence rates,<sup>29</sup> and we used Scandinavian registry data for comparisons. Although the Scandinavian registries are considered some of the most complete, they are also subject to a degree of under-reporting.<sup>38,39</sup> This may have resulted in an inflated estimate of the proportion of keratinocyte cancers attributable to UVR.

Although the numbers of cancers attributable to UV generated by these analyses may appear precise, we remind readers that there is potential for error in these estimates due both to statistical uncertainty (precision) as well as variation in prevalence and risk estimates. We did not calculate confidence intervals for the PAF as these is no universally agreed approach. Instead, we performed sensitivity analyses under various scenarios, which convey a sense of the range of uncertainty of our estimates.

We did not calculate PAFs associated with solarium use. While solarium use in Australia grew in popularity in the 1990s and early 2000s,<sup>40</sup> publicity and active campaigning has recently seen the number of solaria decline. In addition, all Australian states and territories except Western Australia and Northern Territory had passed legislation to ban solaria (except for medical use in Tasmania) by the end of 2014.<sup>41</sup>

Very few data are available with which to estimate the effectiveness of sunscreen for preventing skin cancers. We used data from the only trial of sunscreen use and skin cancer ever conducted.<sup>13,15</sup> While the relative risk estimates from that trial were generated from a 5-year intervention with 11 years of followup, it is possible that the observed effect of sunscreen on skin cancer incidence may have been greater had there been longer follow-up and a greater age span. If so, our calculations of prevented fraction may be underestimates. Further, while we assumed that regular sunscreen use exerts the same magnitude of effect at all ages; it is possible that it may have different magnitudes of effect on younger or older people. For melanoma, UV exposure in early life is thought to be particularly important,<sup>5</sup> and thus sunscreen use among children and adolescents may lead to even greater benefits in cancer prevention and may include BCC prevention.

The estimates of prevalence of sunscreen use applied in our primary analysis (from the 2010 NSW Population Health Survey) were chosen because the question most closely reflected the administration of sunscreen in the Nambour trial. The NSW Population Health Survey was a survey of 10,245 adults aged 16 years and over with a participation rate of 57%.32 The survey was conducted by telephone, so respondents were all from households with private telephones; and adult males were under-represented (40% of the sample versus 50% of the NSW adult population).<sup>32</sup> We conducted sensitivity analyses using prevalence data from other Australian surveys and the resulting

prevented fractions were similar to those from our primary analysis.

We acknowledge counter-arguments that relate to potential harm associated with sunscreen use, including the potential to extend intentional sun exposure<sup>42</sup> and a potential effect on vitamin D production, although evidence for the latter is lacking.43 For the Australian population, where knowledge about skin cancer and sun protection is high,<sup>44</sup> it is not known whether sunscreen is used to extend intentional sun exposure. The Nambour trial found no increase in sun exposure for the group randomised to sunscreen and, regardless, there was a net benefit for SCC of the skin associated with regular sunscreen use, and possibly also for melanoma.

The high proportion of melanomas and keratinocyte cancers attributable to solar UVR exposure underscores the potential for preventing these cancers. High sun exposure is modifiable through various practices including minimising outdoor activity during periods of peak ambient UVR (such as in summer and in the middle hours of the day), wearing sun-protective clothing and applying sunscreen. The analyses here indicate that prevailing levels of sunscreen are likely to have substantially reduced the incidence of SCC and perhaps also melanoma. More widespread regular use would be expected to reduce the incidence further. At a population level, it has recently been shown that treatment rates for keratinocyte skin cancers declined over the period 2000-11 among Australians aged under 45 years.45 One interpretation of those data is that skin cancer prevention programs that have been prominent in Australia for more than 30 years have led to changes in sun protection among more recent birth cohorts.46 Continued monitoring of these trends will be important to determine whether they are sustained into the future.

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attributable fractions (PAF).	

Sex	Observed Cases 2010		Time Shift <sup>a</sup>			Geographic Shift <sup>b</sup>			
		Expected Cases	Excess cases	PAF%	Expected Cases	Excess cases	PAF%		
Males	6,700	3,185	3,515	52.5	5,434	1,266	18.9		
Females	4,705	3,072	1,633	34.7	3,951	754	16.0		
Persons	11,405	6,257	5,148	45.1	9,385	2,020	17.7		

a: Australian 1982 incidence rates as reference

b: Incidence rates of nearest lower level jurisdiction as reference for each State and Territory (South Australia and Tasmania unchanged)

#### Table 3: Summary of results: prevented fraction and number of cutaneous SCCs prevented (2010) through regular sunscreen use, primary and sensitivity analyses.

	Estimated		Sensitivity analyses													
	Cancer	analysis														
	incidence	NSW Population Health Survey 2010		NSW Population Health Survey 2004		Victorian Sun Survey 2006-07			Queensland Self-reported Health Status 2009			National Sun Protection Survey 2010-11				
	(2008) <sup>a</sup>															
		%	PF <sup>b</sup>	Cancers	%	PF <sup>b</sup>	Cancers	%	PF <sup>b</sup>	Cancers	%	PF <sup>b</sup>	Cancers	%	PF <sup>b</sup>	Cancers
		Sun-screen		prevented	Sun-screen		prevented	Sun-screen		prevented	Sun-screen		prevented	Sun-screen		prevented
		Use			Use			Use			Use			Use		
Males	83,901	21.4	7.5	6,793	40.9	14.3	14,017	27.0	9.5	8,379	39.1	11.2	10,602	36.0	12.6	12,096
Females	53,699	34.6	12.1	7,399	59.5	20.8	14,124	44.0	15.4	9,385	51.8	15.0	9,462	36.0	12.6	7,742
Persons	137,600		9.3	14,192		17.0	28,141		11.4	17,764		12.7	20,064		12.6	19,837
a: Source: Australian Institute of Health and Welfare <sup>29</sup>																

b: PF: Prevented Fraction (expressed as a percentage)

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#### **Supporting Information**

Additional supporting information may be found in the online version of this article:

**Supplementary Table 1**: Theoretical "geographic shift" in State and Territory incidence (/100,000) of melanoma.

**Supplementary Table 2**: Summary of questions about sunscreen use in State and National Health Surveys.