ORIGINAL ARTICLE

Sex differences in the association of cutaneous melanoma incidence rates and geographic ultraviolet light exposure

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Background: Cutaneous melanoma (CM) incidence rates continue to increase, and the reasons are unknown. Previously, we reported a unique age-specific sex difference in melanoma that suggested additional causes other than solar ultraviolet (UV) radiation.

Objective: This study attempted to understand whether and how UV radiation differentially impacts the CM incidence in men and women.

Methods: CM data and daily UV index (UVI) from 31 cancer registries were collected for association analysis. A second dataset from 42 US states was used for validation.

Results: There was no association between log-transformed female CM rates and levels of UVI, but there was a significant association between male rates and UVI and a significant association between overall rates and UVI. The 5-year age-specific rate−UVI association levels (represented by Pearson's coefficient ρ) increased with age in men, but age-specific ρ levels remained low and unchanged in women. The significant rate−UVI association in men and nonassociation in women was validated in a population of white residents of the United States.

Limitations: Confounders, including temperature and latitude, are difficult to separate from UVI.

Conclusions: Ambient UVI appears to be associated with melanoma incidence in males but not in females. (J Am Acad Dermatol http://dx.doi.org/10.1016/j.jaad.2016.08.027.)

Key words: age-standardized rates; gender difference; melanoma; UV index; UVI; sex.

INTRODUCTION

Incidence rates of cutaneous melanoma (CM) have been increasing in the past few decades in the United States and in European countries.[1] The causation of melanomagenesis remains under debate,[2,3] especially the role of ultraviolet radiation (UVR), which is the major known environmental risk factor for CM and nonmelanoma skin cancer (NMSC). CMs frequently occur on the trunk, where UVR does not usually reach, while NMSCs are mostly

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found on sun-exposed body sites, such as the head, neck, and limbs.[4] Unlike NMSCs, CMs are not associated with cumulative ultraviolet (UV) exposure.[5] In addition, there are fewer UV signature mutations in patients with CMs than in those with NMSCs.[6] Therefore, in contrast to NMSC, the involvement and effect of UVR in CM is much more complex.[7] It is now generally accepted that CM is associated with intermittent UV exposure.[5] Based on this concept, the primary melanoma preventive measure is the application of sunscreen. The use of sunscreen began as early as the 1930s and has boomed since the 1950s, but the incidence of CM has continued to increase during this time period.[8]

CAPSULE SUMMARY

- The influence of sex on the association of melanoma incidence with ultraviolet light index is uncertain.
- Our study shows that melanoma rates are associated with ultraviolet light index in males but not in females.
- The differential effect on ultraviolet light index on melanoma incidence suggests the possibility of sex-specific prevention strategies.

Our previous publication indicated that women from the United States and from Nordic countries had higher CM incidence rates than men until 45 years of age, with a peak difference at 20 to 24 years of age.[9] There was no evidence of such a pattern for NMSC.[9] Basically, men and women were at equal risk of developing NMSC at a young age, although elderly men were at a higher risk, as was true for melanoma. Based on this comparison, we speculated that the etiology of melanoma in older age groups, as for NMSC,[9] was largely attributable to cumulative UV exposure, but causative factors in younger females required additional investigation.

The purpose of our current study is to understand the heterogeneous etiologic factors that may contribute to sex and age differences in CM. In this study, we collected cancer registry data for melanoma and computed daily average UV index (UVI) for that registry area. The association between UVI and sex- and age-specific rates was analyzed.

METHODS

Data collection, inclusion, and exclusion criteria

Melanoma tumor classification was based on the standard of the *International Classification of Diseases for Oncology* (ie, code C43). Cancer registries were selected primarily based on availability of data and majority of white populations, which include select European countries, the United States, Australia, and New Zealand. The data from the Northern Territory in Australia (which contains considerable population of indigenous Australians) were extracted to contain only nonindigenous populations. The European registry selection is mainly based on light eye color, as reported earlier.[10] Countries with >50% of population with light eye color were selected; therefore, France, Italy, and many southern European countries were excluded.

For primary analysis, US data were retrieved from the Surveillance, Epidemiology, and End Results (SEER) 18 database using 2013 data (including data from 1973-2011), with all cutaneous melanomas (site group: 7.1 melanoma; *International Classification of Diseases for Oncology, 3rd revision* behavior recode: 3; primary site C000-C809, histology types 8720-8723, 8726, 8728, 8730, 8740-8746, 8761, 8770-8774, and 8780). Only white (race = 1) data were included for analysis. Registries 27, 37, and 47 (ie, Atlanta metropolitan, rural Georgia, and Georgia excluding Atlanta/rural Georgia) were pooled as "Georgia," and registries 1 and 31 (ie, San Francisco—Oakland and San Jose—Monterey) were pooled as "SFSJ" because the UVI is the same for these areas. Therefore, US SEER data generated a total of 13 areas. The age-standardized incidence rates (ASRs) are calculated according to the world standardized population for 2000-2025 (National Cancer Institute SEER website).

For the validation dataset, information was extracted from International Agency for Research of Cancer (IARC) CI5 volume X, which contains data for 2003 to 2007 only. To ensure homogeneity of the data, only US data of the white population was used. This US dataset contained some overlapping period and regions from the SEER data entries; even within the same SEER region, the data collected in this set were limited to 2003 to 2007, which was different from the SEER dataset where data were collected since the establishment of the registry. The source of data is listed in Supplemental Table I (available at http://www.jaad.org).

UVI calculation and estimation

Daily average UVI was calculated based on records from July 1, 2002 to June 30, 2014 from a satellite database (available at: http://www.temis.nl/uvradiation/SCIA/stations_uv.html). For country UVI estimation, either the data obtained from the station in the center of the country was used or, when that was not possible, average data from stations on

Abbreviations used:

ASR: age-standardized rate
CI: confidence interval
NMSC: nonmelanoma skin cancer
RR: relative risk
UV: ultraviolet
UVI: ultraviolet index

the borders of the country were calculated and used. For US and Australian registries, data from a satellite station within the registry area was used for that registry, or a location with similar latitude was used if no station was found within the registry region. For example, the UVI for Louisiana (29-33°N) was estimated to be 8.9, which was extrapolated from the monitoring data from a station in the Everglades National Park (25°N) in Florida. Denmark did not have a monitoring station; therefore, data from Manchester, United Kingdome was used because it has the closest latitude to Denmark. The average daily UVIs for selected countries are listed in Supplemental Table II (available at http://www.jaad.org).

Statistical analysis

All data were processed using SAS software (version 9.3; SAS Inc, Cary, NC) if not specified. The ASRs are calculated according to the world-standardized population for 2000 to 2025 (according to the NCI SEER website). The association between UVI and melanoma rates or the risk ratio was analyzed by a simple linear regression model and Pearson product-moment correlation method, as well as the Spearman correlation analysis. The normality test was carried out by all three defaulted test methods in SAS (ie, Kolmogorov–Smirnov, Cramer–von Mises, and Anderson–Darling), and the results are shown in Supplemental Table III. All P values were obtained for two-sided tests, with a significance level at 0.05.

RESULTS

The association of sex-specific melanoma incidence rates with UVI

To investigate the UVR impact on sex, we calculated ASRs of melanoma in men and women from 31 cancer registries in the United States, European, and Australian continents (Table I; Supplemental Table I). Daily average UVI was calculated using data collected by the GOME-2 satellite stations (Table I; Supplemental Table II). Ambient UVI was modeled with data collected at noon each day with consideration for the local cloud conditions and was well correlated with ground erythemal UV dose, without distinction between UVA and UVB wavelengths.[11] The UVI ranged from 1.8 (Finland) to 12.0 (Australia, Northern Territory) and followed a roughly normal distribution (Supplemental Table III; available at http://www.jaad.org).

Histogram ASRs from the 31 registries showed that these rates did not follow normal distributions (data not shown). Log transformation was then carried out for ASRs for men, women, and both; none of the log-transformed rates was significantly different from a normal distribution (Supplemental Table III), which enabled us to fit data into a linear regression model. Log-transformed rates were then used for regression analysis against UVI. Both Pearson and Spearman linear regression models were used to analyze the association of ASRs from each area and local UVIs. The Pearson method is based on the actual number, and the Spearman method is based on the rank of the data. As shown in Table II, male rates (log-transformed, same for all the following rates) showed moderate (Pearson ρ = 0.61) but significant (P = .0003) association with UVI, but females rates showed a nonsignificant low level of association (ρ = 0.31 and P = .09). When both sexes were considered, the association was moderate but significant (ρ = 0.49 and P = .005). The results from Spearman association analysis were similar (Table II).

The log-transformed rates in men showed a moderate regression line with UVI (Fig 1, *A*) but the regression line in log-transformed female rates was much flatter, and the distribution was more scattered (Fig 1, *B*), indicating nonassociation between the two variables. In addition, coefficients of determination R^2 calculated from Pearson regression model for men, women and both sexes were 0.37, 0.09, and 0.24 (Table II), suggesting that the UVI only explained 9% of the melanoma incidence rates in women but 37% of the incidence rates in men.

Levels of ASR-UVI association increase with age in men

Melanoma incidence rates increase with age in both sexes, so we hypothesized that perhaps the CM rates in older females would show a better association with UVI. To test this hypothesis, the 5-year age specific rates in each registry were calculated. Age category (Agecat) 1 represents age 0 to 4, Agecat 2 represents age 5 to 9, and so on. The rates in each agecat were log-transformed and then fitted into a Pearson regression model with UVI as dependable variables, and the association levels (ρ and corresponding P values) for each agecat

Table I. Crude and age-adjusted melanoma incidence rates and rate ratios

		Crude Rate			ASR			
Registry/country	UVI	Male	Female	All	Male	Female	All	RR
Australia Capital Territory	7.2	44.2	35.9	39.8	39.8	29.8	34.2	0.75
Australia New South Wales	7.2	61.1	41.9	51.4	46.0	31.3	37.9	0.68
Australia NT nonindigenous	12.0	35.8	27.9	32.1	31.7	25.3	28.9	0.80
Australia Queensland	9.4	74.2	53.2	63.7	59.2	42.5	50.3	0.72
Australia Tasmania	5.6	54.5	46.5	50.4	40.5	34.5	37.0	0.85
Australia Victoria	6.3	45.4	36.3	40.8	34.8	27.1	30.6	0.78
Western Australia	7.7	60.0	41.6	50.8	48.7	32.8	40.4	0.67
South Australia	7.0	48.5	37.0	42.6	35.0	26.7	30.4	0.76
Austria	4.1	19.1	18.8	19.0	10.2	9.2	9.7	0.90
Belgium	3.4	25.5	35.3	30.5	18.7	26.6	22.7	1.43
Connecticut-US	4.9	22.2	17.3	19.7	17.6	13.3	15.4	0.75
Denmark	3.0	21.0	25.1	23.0	15.5	19.1	17.3	1.23
Detroit-US	4.9	16.8	13.1	14.9	14.4	10.8	12.6	0.75
Finland	1.8	15.3	14.1	14.7	11.3	9.5	10.4	0.84
Georgia-US	6.7	29.0	21.7	25.3	25.6	17.9	21.7	0.7
Germany	3.8	18.1	18.8	18.5	12.0	12.9	12.5	1.08
Hawaii-US	10.5	52.5	35.0	44.4	45.9	28.5	37.2	0.62
Iowa-US	4.9	15.5	13.4	14.4	12.7	11.0	11.8	0.86
Kentucky-US	5.7	28.6	21.0	24.7	22.0	16.3	19.1	0.74
Los Angeles-US	6.9	18.1	12.5	15.3	17.5	10.7	14.1	0.61
Louisiana-US	8.9	22.7	14.6	18.6	16.9	11.1	14.0	0.66
Netherlands	3.2	28.7	38.4	33.5	20.6	29.9	25.2	1.46
New Jersey-US	5.6	31.9	23.3	27.5	22.9	16.5	19.7	0.72
New Mexico-US	6.7	17.7	13.1	15.3	15.6	11.2	13.4	0.72
Norway	1.9	22.7	25.3	24.0	17.3	18.7	18.0	1.08
New Zealand non-Maori	5.4	64.7	56.4	60.5	46.5	40.2	42.9	0.87
Seattle-US	4.3	23.2	20.3	21.8	20.0	16.9	18.5	0.85
SFSJ-US	5.7	23.7	18.1	20.9	20.0	14.3	17.1	0.72
Sweden	2.2	21.4	21.5	21.5	14.6	15.0	14.8	1.03
United Kingdom	3.3	40.9	43.2	42.0	27.2	30.1	28.7	1.11
Utah-US	6.1	18.8	14.5	16.7	20.9	15.0	17.9	0.72

ASR, Age-adjusted melanoma incidence rate; *NT*, Northern Territory; *RR*, rate ratio; *SFSJ*, San Francisco—San Jose; *UVI*, ultraviolet light index.

Table II. Association between log-transformed rates with ultraviolet light index

Statistics	Male	Female	All
Pearson's ρ	0.61	0.31	0.49
P value	0.0003*	0.09	0.005*
R^2	0.37	0.09	0.24
Spearman's ρ	0.63	0.28	0.48
P value	0.0002*	0.13	0.006*
R^2	0.39	0.076	0.23

*Statistically significant.

were computed and listed in Table III. As shown in Fig 2, there was a significant linear increase of ρ (association levels) with age in men (eg, the rate—UVI association became stronger in older age groups; ρ_{m1} = 0.62; P = .006). Again, no association of ρ with age was observed in women (Fig 2, *B*; ρ_{f1} = -0.11; P = .67).

Validation of the observation with a second dataset

To validate our observation of the different association of CM rates with UVI, a separate dataset was obtained from IARC CI5 Volume X, where 42 states from US provided cancer data from 2003 to 2007 with race information (California counted for 2 registries therefore a total of 43 registries; Table IV). Only white race data was used for analysis, and therefore this dataset contained more homogeneous data on race (white race) and data collection period (2003-2007). For this dataset, 12 registries (28%) overlapped with the primary dataset used in the main analysis, but the data collection period was shorter compared to the one used in the main analysis. Pearson regression analysis indicated that in this new dataset, the log-transformed rates in men again showed a moderate but significant association with UVI (ρ = 0.34 and P = .02) while that in women

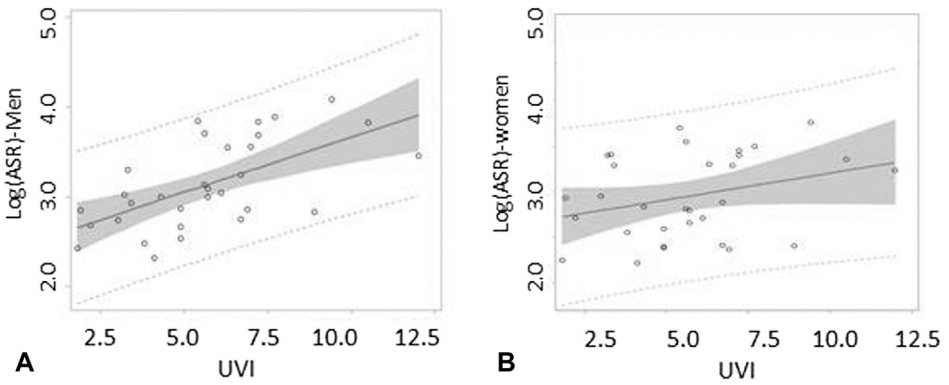

Fig 1. Melanoma incidence rates. Linear regression of log-transformed age-standardized melanoma incidence rates (ASRs) from 31 registries with average local daily ultraviolet light index (UVI). **A**, Men. **B**, Women. Solid lines represent the fitted line; shaded areas represent the 95% confidence interval.

Table III. Association between age-specific rates and ultraviolet light index based on 31 registries

		Men		Women	
Age category	**Age range, y**	ρ	***P* value**	ρ	***P* value**
1	0-4	0.41	.071	0.26	.286
2	5-9	0.41	.053	0.59	.004
3	10-14	0.35	.074	0.20	.332
4	15-19	0.64	<.0001	0.27	.141
5	20-24	0.48	.0051	0.43	.013
6	25-29	0.58	.0005	0.32	.075
7	30-34	0.49	.0045	0.24	.184
8	35-39	0.52	.0023	0.31	.081
9	40-44	0.51	.0031	0.33	.064
10	45-49	0.60	.0003	0.39	.027
11	50-54	0.66	<.0001	0.37	.039
12	55-59	0.66	<.0001	0.31	.089
13	60-64	0.58	.0006	0.24	.190
14	65-69	0.51	.0031	0.32	.072
15	70-74	0.54	.0013	0.34	.055
16	75-79	0.67	<.0001	0.32	.072
17	80-84	0.53	.0018	0.29	.110
18	85+	0.66	<.0001	0.34	.064

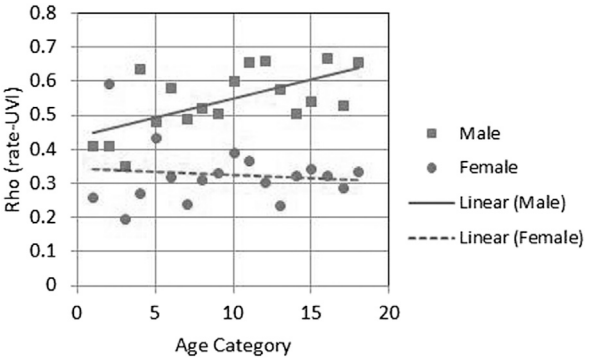

Fig 2. Melanoma rates and age. Levels of rate—ultraviolet light index association increase with age in men but not in women. Five-year age-specific rates were calculated for each registry for each sex, and association of these rates (log-transformed) with ultraviolet light index was analyzed to obtain Pearson's ρ for each age category. These ρ values were plotted against age categories. Squares and solid line: ρ for males in each age categories and the regression line. Filled circles and dotted line: ρ for females in each age categories and the regression line.

did not (ρ = 0.08 and P = .68; Table IV). When both sexes were combined, the rate—UVI association was no longer significant (Table IV).

DISCUSSION

This ecologic study provides evidence of the following: (1) there is a significant difference in the association of geographic UVI with male and female CM incidence rates, and CM rates in men are associated with UVI but not that in women; and (2) the levels of association between male rates and UVI increase with age. It was previously reported that the mean daily UVR accounted for 82% of BCC and 85% of SCC incidence rates.[12] In our study, UVI only accounted for approximately 1% to 2% of melanoma rates in women, approximately 33% in men, and approximately 13% to 15% for the entire population (Table II). While it was known that UVR played a complex role in melanoma etiology, our observation on sex difference is quite interesting.

Potential major confounders include temperature and latitude. Higher UVIs are usually associated with higher temperatures and lower latitudes, both of which may be associated with different sun behaviors (eg, how much clothing individuals wear and how long they stay outdoors).[13] Limited information is available on how external ambient temperatures impact CM incidence,[14] and additional investigations are needed to address this question.[3]

Table IV. Melanoma age-adjusted incidence rates in US white population from 2003 to 2007 and their association with ultraviolet light index

Population location	Men	Women	All	UVI
Alabama	18.9	13.4	16.2	6.7
Arizona	13.9	9.0	11.5	6.7
Arkansas	13.0	9.0	11.0	6.7
California (Los Angeles)	15.9	9.8	12.9	5.7
California (San Francisco)	20.3	14.4	17.4	5.6
Colorado	17.7	14.5	16.1	5.9
Connecticut	20.6	16.1	18.4	4.9
Delaware	22.8	16.2	19.5	5.6
Florida	16.5	11.6	14.1	8.7
Georgia	23.3	17.1	20.2	6.7
Hawaii	55.3	36.9	46.1	10.5
Idaho	20.1	14.8	17.5	4.7
Illinois	13.9	10.8	12.4	4.9
Indiana	13.9	11.1	12.5	4.9
Iowa	15.0	12.8	13.9	4.9
Kentucky	17.8	14.2	16.0	5.7
Louisiana	14.9	9.7	12.3	8.9
Maine	18.2	14.7	16.5	4.2
Massachusetts	18.5	14.3	16.4	4.9
Michigan (Detroit)	16.8	14.2	15.5	4.9
Mississippi	12.3	8.6	10.5	6.7
Missouri	15.3	11.1	13.2	5.7
Montana	13.5	12.2	12.9	4.3
Nebraska	13.0	10.0	11.5	4.9
New Jersey	19.5	15.1	17.3	5.0
New Mexico	15.4	11.0	13.2	6.7
New York	14.9	10.7	12.8	4.9
North Carolina	19.3	15.0	17.2	6.0
North Dakota	12.0	12.7	12.4	4.3
Ohio	15.4	12.9	14.2	5.6
Oklahoma	17.4	13.0	15.2	5.7
Oregon	19.9	18.1	19.0	4.7
Pennsylvania	15.2	12.4	13.8	5.0
Rhode Island	18.7	14.1	16.4	4.9
South Carolina	23.4	18.1	20.8	6.8
Tennessee	17.9	12.9	15.4	6.3
Texas	13.2	8.4	10.8	6.8
Utah	22.4	16.2	19.3	6.1
Vermont	23.3	21.3	22.3	4.2
Virginia	20.2	14.6	17.4	5.5
Washington (Seattle)	20.7	18.3	19.5	4.3
West Virginia	15.1	11.5	13.3	5.3
Wisconsin	14.0	11.6	12.8	4.9
log(rates) vs UVI ρ (*P* value)	0.34 (.02)	0.075 (.63)	0.23 (.14)	—

UVI, Ultraviolet light index.

Estimated solar UVR (as well as latitude) was shown to be strongly associated with NMSC, but data from 10 US metropolitan populations from 1977 to 1978 showed limited association with melanoma rates.[15] Armstrong and Kricker[15] concluded in 2001 that "inconsistencies have been observed in these patterns (association of incidence rates with geographical UV) depending on the populations studied, particularly for melanoma." In addition, not much analysis was based on separated sex and age categories. Our analysis included more registries with relatively homogenous population backgrounds, and used more updated incidence rates.

Nevertheless, a limitation of this study is still the heterogeneous population from different countries. One would argue that because nonwhites usually are a minority population and have much lower rates of melanoma, the overall error may not affect our conclusion. Validation from US white population from the 2003 to 2007 dataset therefore provides important support to our conclusion.

The weakness of this study, because of its ecologic nature, is that the conclusion is not applicable to individuals (ie, an individual's risk for CM may not have such a linear correlation with individual UV exposure because we cannot assume all people are exposed to the same level of UVR in the same area). Also, intermittent sun exposure, a known risk factor for melanoma,[16] cannot be assessed from UVI and is therefore not a variable in this study.

The underlying mechanisms as to why women did not show an association with UV index is likely complex, reflecting either differences in sun behavior, physiologic differences, or both. One immediate argument is that use of cosmetics or sunscreen in women provides protection against UVR, and therefore cosmetic use masks the UV effect. A recent survey showed that compared to men, women were more likely to use sunscreen and therefore were protected.[17] However, our previous study showed that younger women exhibited significantly higher (not lower) CM rates than younger men.[9] Using more cosmetics or sunscreen by women does not completely explain the nonassociation between UVI and rates, at least for younger age.

Several lines of evidence suggest that physiologic differences between the sexes may play a crucial role in melanoma development. First, male and female skin differs substantially in both structure and biology.[18] Male skin is thicker, richer in collagen and elastin, secretes more sebum, has less subcutaneous fat, more hair, and different hair patterns because of androgen stimulation and estrogen suppression.[18,19] These differences lead to differences in skin responses to environmental stress, including UVR.[19] Potentially, female skin

may naturally be more protected from UVR or may be equally prone to UV-induced damage but more capable of repair. Evidence from the literature supports both hypotheses.[20,21] A Netherlands study revealed that male subjects had a lower minimal erythema dose than female volunteers,[20] suggesting that male skin was more sensitive to UVR. A similar study using simulated solar UVR revealed that the doses required to elicit a similar immunosuppression response in men were 3 times lower than those in women.[21]

In addition, studies in animal models have suggested that hormones play a role in UV-induced skin responses. The female hormone 17-β-estradiol inhibits UVR-induced immunosuppression in female mice,[22] and the male hormone androgen impedes acute skin wound healing in male rats.[23] Finally, melanomas have been shown to clinically respond to treatment with tamoxifen, follicle-stimulating, melatonin, and nerve growth factor hormones.[24,25] The outcome of melanoma treatment is different in men and women,[26] which also implies a role for hormones in response to treatment.

In summary, we found no association between UVI and female CM incidence rates. In contrast, there was a significant association between male CM incidence rates and UVI, and this association showed a moderate increase with age. Our results reveal a previously underappreciated area of sex-specific causative factors in melanoma development, which may potentially lead to novel sex-specific research on etiology and possibly preventive strategies in the future.

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Supplemental Table I. Total case numbers and data sources

Country/region	Male	Female	Years	Data source
Austria	10,305	10,902	1990-2009	IARC European Cancer Observatory
Australia	29,380	21,624	2003-2007	IARC Volume X
Belgium	1876	2811	2004-2006	IARC European Cancer Observatory
Canada	39,020	35,250	1992-2010	Chronic Disease Infobase Canada
Denmark	12,753	15,601	1990-2012	The NORDCAN project
Finland	8959	8632	1990-2012	The NORDCAN project
Germany	86,705	80,330	2000-2010	The German Centre for Cancer Registry Data
New Zealand	5152	4664	2003-2007	IARC Volume X, non-Maori only
Nordic Countries	56,349	60,490	1990-2012	The NORDCAN project
Norway	12,349	13,319	1990-2012	The NORDCAN project
Sweden	21,925	22,369	1990-2012	The NORDCAN project
The Netherlands	33,067	43,303	1995-2012	The Dutch Cancer Registry
United Kingdom	37,275	40,741	2005-2012	Office for National Statistics
United States (primary)	125,442	97,740	1973-2011	SEER 18 registries (only white race)
United States (validation)	120,066	90,668	2003-2004	IARC CI5 Volume X (only white race)

Supplemental Table II. Selected daily average ultraviolet light index from monitoring stations in different countries

Country	Station location	UVI
Austria	Vienna	3.8
	Sonnblick	4.3
Australia		
Northern Territory	Darwin	12.0
South	Sydney	7.0
Tasmania	Paraparaumu, New Zealand	5.6
Victoria	Melbourne	6.3
Capital Territory	Kingston	5.2
Western	Perth	7.7
New South Wales	Newcastle	7.3
Belgium	Uccle	3.4
Denmark	Goosebay	2.9
Finland		
	Jokioinen	2.2
	Sodankyla	1.5
New Zealand	Paraparaumu	5.6
Norway	Trondheim	1.9
Sweden	Stockholm	2.2
Netherlands	Bilthoven	3.2
US		
Georgia, New Mexico	Atlanta	6.7
Seattle	Acadia Park (ME)	4.3
New Jersey	Shenandoah Park (VA)	5.6
Louisiana	Everglades Park (FL)	8.9
Hawaii	Mauna Loa	10.5
Utah	Canyonlands	6.1
SFSJ	San Francisco	5.7
Iowa, Detroit, Connecticut	New York*	4.9

SFSJ, San Francisco—San Jose; *UVI*, ultraviolet light index.

Supplemental Table III. Test of normal distribution

	Test	Statistic	*P* value	
log(ASRM)	Kolmogorov–Smirnov	0.126	Pr > D	>.150
	Cramer–von Mises	0.097	Pr > W-Sq	.121
	Anderson–Darling	0.561	Pr > A-Sq	.14
log(ASRF)	Kolmogorov–Smirnov	0.151	Pr > D	.07
	Cramer–von Mises	0.119	Pr > W-Sq	.062
	Anderson–Darling	0.708	Pr > A-Sq	.061
log(ALL)	Kolmogorov–Smirnov	0.108	Pr > D	>.150
	Cramer–von Mises	0.075	Pr > W-Sq	.235
	Anderson–Darling	0.444	Pr > A-Sq	>.250
UVI	Kolmogorov–Smirnov	0.106	Pr > D	>.150
	Cramer–von Mises	0.038	Pr > W-Sq	>.250
	Anderson–Darling	0.289	Pr > A-Sq	>.250

ASRF, Age-standardized rate for females; *ASRM*, age-standardized rate for males; *UVI*, ultraviolet light index.