Association of Surface Ultraviolet B Radiation Levels with Melanoma and Nonmelanoma Skin Cancer in United States Blacks

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Abstract
Ultraviolet B (UVB) radiation exposure increases the risk of skin cancer in whites. Motivated by indications that United States geographic variation of relative skin cancer risk in blacks approaches that in whites, we used Poisson regression to estimate the risk of skin cancer in blacks as a function of average annual surface-levels of UVB radiation, measured by Robertson-Berger meters. United States data were used on deaths in 506 state economic areas, 1970–1994, and on incident cases in the nine areas of the Surveillance, Epidemiology, and End Results Program, 1973–1994. For black males, the age-adjusted relative risk of mortality for a 50% increase in UVB radiation was significantly above one for malignant melanoma, 1970–1994 (1.16; 95% confidence interval, 1.02–1.32) and nearly so for nonmelanoma skin cancer, 1970–1981 (1.18; 95% confidence interval, 1.00–1.39), for which the time period was chosen to avoid AIDS-related deaths from Kaposi’s sarcoma. However, for black females, the relative risk of mortality was not significantly elevated for either skin cancer, and, for both black males and females, the relative risk of incidence was not significantly elevated for melanoma in the period 1973–1994. Incidence data on nonmelanoma skin cancer were not available. Although the public health implication is uncertain because of the much lower absolute risk of skin cancer in blacks compared with whites, the findings suggest that sunlight exposure increases skin cancer risk in blacks.

Introduction
Sunlight exposure and low level of skin pigmentation are predominant risk factors for skin cancer in whites. Increased risk in whites of both malignant melanoma and nonmelanoma skin cancer (NMSC), including BCC and SCC, has been associated with UVB radiation exposure from sunlight, decreasing latitude, and decreasing level of skin pigmentation (1–5). Furthermore, blacks have much lower melanoma, BCC, and SCC incidence rates than whites (6, 7), and the dose of UV radiation required to produce a minimum perceptible erythema has been estimated to be 6–33 times greater in blacks than in whites (8, 9).

Some previous work suggests that sunlight exposure and low level of skin pigmentation are risk factors for skin cancer in blacks as well as whites. A Howard University Hospital study of 23 blacks with BCC and 291 blacks chosen randomly found that 60% of the former but only 10% of the latter had fair or olive skin (Ref. 10; P < 0.0001 for the difference). A survey of NMSC incidence at nine United States locations in 1977–1980 recorded 68 cases among blacks that suggest that NMSC rates increased with decreasing latitude (7). Telephone interview data from cases and controls at these locations indicate that the risk of NMSC was lower for dark-skinned whites than fair-skinned whites, but that NMSC rates increased with increasing UVB radiation level for both groups (3).

The present analysis was motivated by indications that United States geographical variation of relative skin cancer rates in blacks approaches that in whites (11, 12). After this discovery, we sought additional evidence linking skin cancer risk for blacks to potential UVB radiation exposure.

Materials and Methods
Data. We tabulated black and white incident cases of malignant melanoma in the nine areas of the SEER program (13). The nine areas are listed in Table 1. The area associated with a case was determined by place of residence at the time of diagnosis. We tabulated cases diagnosed during 1973–1994. Data were unavailable, however, for Seattle in 1973 and for Atlanta in 1973–1974. We stratified the cases by five anatomic site combinations: (a) the combined upper limb and shoulder sites; (b) the combined lower limb and hip sites; (c) the trunk site; and (d) site not specified. We could not obtain NMSC incidence data specifically for BCC and SCC because these data are not routinely reportable to SEER except for special surveys (for example, the survey reported in Ref. 2).

We tabulated black and white deaths due to melanoma and NMSC by 506 SEAs in the coterminous United States, i.e., excluding Alaska and Hawaii, using data from the National Center for Health Statistics (14). SEAs are groups of counties defined by the United States Bureau of the Census that do not cross state boundaries and are similar in demography, economy,
For the SEER areas, the average was obtained for 1977–1980 from meter readings (4). For Hawaii, meter readings were available only from 1974–1987 and explains up to 97% of the variation in the meter readings (4). For California, meter readings were available only for southern California (Table 1). The SEAs assigned to northern California were San Francisco, San Jose, Sacramento, Eureka, Santa Rosa, Santa Cruz, Chico, Modesto, and Woodland.

To estimate the effect of UVB radiation exposure on the rates, we used available SEER area and state estimates (Ref. 4, Tables 17-3a and 17-2 therein) of average annual UVB radiation reaching the earth’s surface (Table 1). The estimates are based on surface-level readings from meters developed by Robertson (17) and Berger et al. (18) that were placed at ground level at various National Weather Service stations. The meters were calibrated to an action spectrum that parallels that for human skin erythema and provide a single reading that weights the UVB wavelengths by their relative erythema response. The estimates are given in Robertson-Berger (R-B) counts $\times 10^{-4}$.

The UVB levels used for the SEER areas were averages of UVB readings taken from 1977–1980 as part of a special survey on NMSC (4), except for Connecticut, Iowa, and Hawaii, which were not included in the survey. For Connecticut and Iowa, we appropriated the UVB levels used for the states. The state UVB levels were based on predictions from a regression model that linearly related the log of UVB to latitude, altitude, and sky cover; the regression model is based on UVB meter readings for Mauna Loa, a volcano on which no one lives, and a state estimate was not readily available. We, therefore, predicted the value for Honolulu from the regression model log(UVB) $= 15.545 - 0.039(L) - 0.0001038(A)$ [given in Ref. 4], where, for Honolulu, $L =$ latitude $= 21.32$ degrees and $A =$ altitude in meters $= 4.0$ meters; this model explains up to 91% of the variation in meter readings (4).

For SEAs, we used the corresponding state levels of UVB. The states include DC and separate areas for northern and southern California (Table 1). The SEAs assigned to northern California were San Francisco, San Jose, Sacramento, Eureka, Santa Rosa, Santa Cruz, Chico, Modesto, and Woodland.

### Geographic Variation

Before undertaking this paper, we estimated geographical variation in relative melanoma and NMSC mortality rates for blacks and whites as part of a United States cancer mortality atlas (11). We used the methods derived in Ref. 12 to estimate the RRSD among geographical areas and its SE. These are computed under a Poisson model on age-specific counts with means multiplicative in fixed-age effects and random-area effects. The random-area effects represent relative risks and are assumed to be independently gamma distributed with mean one and SD RRSD. We applied the same methods to the time periods considered here.

<table>
<thead>
<tr>
<th>States</th>
<th>SEER areas</th>
<th>Estimated annual surface level of UVB radiation in Robertson-Berger counts $\times 10^{-4}$ by SEER area and state$^a$</th>
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<tbody>
<tr>
<td>Alabama</td>
<td>Atlanta</td>
<td>153</td>
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<tr>
<td>Arkansas</td>
<td>Connecticut</td>
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<tr>
<td>California (north)</td>
<td>California (south)</td>
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<tr>
<td>Colorado</td>
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<td></td>
<td>Wyoming</td>
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$^a$ For the SEER areas, the average was obtained for 1977–1980 from meter readings except for Connecticut, Hawaii, and Iowa. For these areas and the states, the average was predicted from latitude, altitude, and cloud cover based on models of 1974–1987 meter readings (4).
uted with the log of the Poisson mean additive in the log of the person-years at risk, an age effect, and \( \beta (\log x) \), where \( \beta \) is the coefficient of the log of UVB level \( x \). In this model, the relative risk of skin cancer for a proportional increase in UVB level from \( x \) to \( cx \) is \( e^{\beta (\log cx)} / e^{\beta (\log x)} = e^\beta \). We estimated \( \hat{\beta} \) with \( \hat{\beta}^c \), where \( \hat{\beta} \) is the MLE of \( \beta \), and we obtained an approximate 95% CI as \( \hat{\beta} \pm 1.96 \text{se}(\hat{\beta}) \), where \( \text{se}(\hat{\beta}) \) is the asymptotic SE of \( \beta \). We chose \( c = 1.5 \) for a 50% increase in UVB level, and we call \( 1.5^\beta \) the RR50. For example, a 50% increase corresponds to living in Texas versus Indiana or in San Francisco versus Michigan (Table 1).

The fractional decrease in relative risk for a proportional decrease in UVB level from \( cx \) to \( x \) is \( e^\beta - 1 \) or \( 1 - e^{-\beta} \). We estimated this quantity with \( 1 - e^{-\hat{\beta}} \) and obtained an approximate 95% CI as \( 1 - e^{-\hat{\beta}} \pm 1.96 \text{se}(\hat{\beta}) \). We chose \( c = 1.5 \) to indicate a 50% decrease in UVB level, and we call the percentage decrease in relative risk, \( 100 \times (1 - 1.5^\beta) \), the PDRR50.

**Correction for Overdispersion.** To account for possible overdispersion in the counts relative to Poisson variation, we assumed the commonly used quasilikelihood model that the variance of the counts equals the Poisson variance times a scale factor (19). We estimated the scale factor by the Pearson statistic divided by the degrees of freedom of the model. When the scale factor estimate was >1, indicating overdispersion, we adjusted \( \hat{\beta} \) by multiplying by the square root of this estimate. When the scale factor estimate was <1, indicating underdispersion, to be conservative, we made no adjustment. Underdispersion occurred only in the analyses of black counts (see “Results” section), which were sparse, and we were concerned that the scale factor estimates might be unreliable in these cases.

To check the reliability of the scale factor estimate, we computed the score statistic \( P_s \) derived in (20) to test for overdispersion due to unmodeled random effects that are additive in the log of the Poisson mean. When there are no unmodeled random effects, \( P_s \) converges quickly to the standard normal distribution. If \( P_s \) is positive and large compared with the standard normal distribution, then it indicates overdispersion. If \( P_s \) is negative and large in magnitude compared with the standard normal distribution, then it indicates underdispersion.

**Results**

**Geographic Variation.** Table 2 lists MLEs of the geographical variation parameter, RRSD, for melanoma mortality, 1970–1994, and NMSC mortality, 1970–1981, by race and gender. Within each type of skin cancer, the RRSD MLE for black males approaches that for white males. Because of sparse data, each RRSD MLE for black females was unreliably estimated as zero with an infinite SE.

We were surprised that the RRSD MLEs for black males were as large as they were, given that skin cancer mortality rates are much lower in blacks than whites. These results are not influenced by the low random variation of low black rates because the RRSD method separates the random variation of observed rates from the variance of area-specific relative risks, which is the component of interest (12). The results motivated the forthcoming Poisson regressions of skin cancer counts on UVB level to see whether some of the geographical variation in blacks could be explained by UVB radiation exposure.

**Rates.** Age-adjusted rates for groups of geographical areas defined by tertiles of UVB level give preliminary indications of UVB gradients in skin cancer incidence (Table 3) and mortality

![Table 2](Table 2)

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![Table 2](Table 2)
score statistic for overdispersion ($P_9^A$) was much greater than 1.645, the 95th percentile point of the standard normal distribution. For black males and females, RR50 and PDRR50 CIs were adjusted only for black females, trunk, because, in all of the other cases, the scale factor estimates were $<1$; in all cases, $P_9^A$ was between $-1.645$ and 1.645, which indicated no adjustment was needed.

**Relative Risks of Melanoma and NMSC Mortality.** For melanoma mortality at the 506 SEAs in the coterminous United States, 1970–1994, relative risks increased significantly with UVB radiation level for white males, white females, and black males (Table 4). The RR50 MLEs were 1.19 for white males, 1.12 for white females, and 1.16 for black males. Corresponding PDRR50 MLEs were 15.9, 10.5, and 13.6%, respectively. For both whites and blacks, the RR50 MLE was significantly greater in males than in females ($P = 0.0001$ for whites and 0.0439 for blacks), which indicated greater increased risk in males than in females for the same increase in UVB radiation level.

For NMSC mortality at the 506 SEAs, 1970–1981, relative risks increased significantly with UVB radiation level for white males and white females and nearly so for black males (Table 4), the same groups for which significant increases in relative risk were found for melanoma mortality. The RR50
MLEs were 1.37 for white males, 1.19 for white females, and 1.18 for black males. The corresponding PDRR50 MLEs were 27.2, 15.9, and 15.1%. For whites, the RR50 MLE was significantly greater in males than in females (\(P = 0.0001\)), indicating greater increased risk in males than in females for the same increase in UVB radiation level. For blacks, the RR50 MLE was greater in males than in females, but not significantly (\(P = 0.3251\)).

For white males (\(P = 0.0001\)) and white females (\(P = 0.0241\)), the RR50 MLE was significantly greater for NMSC than for melanoma, which indicated greater increased risk of NMSC than melanoma for the same increase in UVB radiation level. For black males (\(P = 0.8682\)) and black females (\(P = 0.5220\)), the RR50 MLE was also greater for NMSC than melanoma, but not significantly.

For each of the mortality analyses for white males and white females, the RR50 and PDRR50 CIs were adjusted for overdispersion by scale-factor estimates that were >1, and the corresponding score statistics \(P_s\) were much greater than 1,645, which indicated the need for adjustment (Table 4). For black males and black females, the CIs were not adjusted because the scale factor estimates were <1, and \(P_s\)'s were between 1,645 and 1,645.

For NMSC mortality for the time periods 1970–1981 and 1987–1994 combined (the latter period excluding AIDS-related deaths) RR50 MLEs for white males and white females were slightly smaller than they were for 1970–1981 considered alone but still significant, which indicated increased relative risk (results not shown). However, the RR50 MLE for black males was only 1.00, indicating no increase in relative risk, in stark contrast to its nearly significant value of 1.18 for the 1970–1981 data considered alone (Table 4).

For comparison with the skin cancer mortality analyses, we obtained RR50s for mortality from all cancers combined for each of the time periods 1970–1981 and 1970–1994. For each time period and each of the four race-gender groups, the RR50 was significantly less than one, which indicated decreased risk with increasing UVB level (results not shown).

### Discussion

We found that, for black males, age-adjusted mortality rates of melanoma 1970–1994 increased significantly, and those of NMSC 1970–1981 increased nearly significantly with increasing levels of surface UVB radiation. We did not find corresponding significant increases in black females, nor did we find significant increases in incidence rates of melanoma, 1973–1994, in black males or black females. A previous United States study indicated that NMSC incidence rates for blacks increased with decreasing latitude (7), but we are unaware of previous United States studies that indicated that melanoma incidence or mortality rates for blacks increased with increasing UVB radiation level or with decreasing latitude.

The nearly significant increase in NMSC mortality in black males disappeared, however, when the data for 1970–1981 were combined with the data for 1987–1994 that supposedly excluded AIDS-related deaths. In 1982–1986, the incidence of Kaposi’s sarcoma rapidly rose in association with increased cases of AIDS (Ref. 13, p. 215), which made NMSC incidence and mortality data difficult to interpret. Beginning in 1987, AIDS-related Kaposi’s sarcoma deaths were supposed to be coded to the human immunodeficiency virus infection if AIDS was mentioned on the death certificate, and to NMSC otherwise (14). Perhaps some of the AIDS-related NMSC deaths after 1986 were reported as skin cancer without specifying AIDS on the death certificate, diluting the potential observable association with UVB radiation. The relative impact of misclassifying AIDS-related NMSC is much greater for blacks than for whites because non-AIDS-related NMSC is much more frequent among whites than blacks.

Melanoma incidence and mortality rates have increased significantly over time for whites but not for blacks. Estimated annual percent changes in rates for 1973–1996 indicate, for melanoma mortality, a 1.0% significant decrease for black males and a 0.0% increase for black females; for melanoma incidence, they indicate an increase for black males and a decrease for black females, neither of which is significant (21).
At the same time, melanoma mortality and incidence rates for white males and white females have increased significantly from 0.6 to 4.4% (21). Melanoma survival rates are poorer for blacks than whites (13), and the significant decrease in black-male melanoma mortality could indicate an improvement in timely diagnosis and treatment. Detection of a consistent time trend for melanoma incidence among blacks is hampered by lack of power due to very low rates for blacks.

The UVB measurements that were used were not representative of individual cumulative exposure to UVB but rather were average annual surface levels in geographical areas corresponding to residence at the date of diagnosis. Furthermore, our use of average annual surface levels for 1977–80 and 1974–1987 does not reflect UVB levels from 1970 to 1994, during which skin cancer data were collected, nor do they reflect yearly changes in UVB levels due to changes in industrialization, motorizing, the ozone hole, sunspot activity, and other factors. Therefore, the effects of UVB exposure on skin cancer rates are probably diluted by our use of this surrogate measure. One possible explanation for our failure to link melanoma and NMSC mortality with UVB level for black females is that the UVB measurements are less representative of actual exposure in females than in males, who have been estimated to spend 1.5–2 times more time outdoors (3). Other factors that could explain individual variation in UVB exposure within geographical areas include outdoor recreational habits and the mobility of the United States population. In particular, the migration of blacks from the South to other parts of the United States during this century could lead to the underestimation of the lifetime cumulative exposure of blacks outside the South.

Nonetheless, the same UVB measurements that we used for SEER areas have been used to associate UVB exposure with NMSC incidence (3, 4). As a reviewer has pointed out, we could have used theoretical UVB levels based on applying radiative transfer models to satellite-based measurements of ozone. Such theoretical measurements have indicated increases in UVB levels over time because of ozone depletion (22), although Robertson-Berger meter readings have not indicated increases over a similar time period (23). A possible explanation for the discrepancy is that ozone-based measurements do not account for possible increases in air pollution. Although the time trends are different, the area-specific UVB levels for each method could be proportional, in which case, the use of either of the two sets of levels would lead to essentially the same results.

Our study can be compared with other melanoma incidence studies that report the biological amplification factor (BAF), which is the limit of the ratio of the percent increase in skin cancer to the percent increase in UVB as the latter approaches zero. In symbols, for a model relating rate \( R \) to UVB level \( x \), the BAF is

\[
\frac{dR}{dx} = \beta \frac{R}{x}
\]

In our model, \( BAF = \beta \), as defined in the “Materials and Methods” section, and is constant over \( x \). For melanoma incidence, the estimated BAFs in our study were 0.5 (95% CI, 0.4–0.5) for white males and 0.4 (95% CI, 0.3–0.4) for white females. Kricker et al. (24) estimated this BAF to be between 0.3 and 2.5, and Scotto and Fears (25) estimated it between 0.6 and 0.8 for males and 0.5 and 1.0 for females, depending on body site. All of these estimates depend on the time period, geographical locations, and action spectrum used to compute the UVB dose. Our BAFs are slightly lower, probably because they encompass more recent time periods, and the strength of the association of melanoma risk with UVB has decreased over time, as implied by associations with latitude (26).

The estimated relative risk of mortality was greater for NMSC than for melanoma for the same increase in UVB level for white males, white females, black males, and black females. Although the differences were significant only for white males and white females, the overall pattern suggests that the etiological role that sunlight plays may be more direct for NMSC than for melanoma for both whites and blacks. The anatomical site distribution of NMSC in whites is weighted towards the frequently sun-exposed face, head, and neck sites (4, 7, 27, 28), which suggests that it is associated primarily with chronic sun exposure (29). In contrast, the distribution of melanoma in whites is weighted towards the face, head, and neck sites (6, 27, 28), and the trunk and leg in females in younger age groups (30–32), which suggests that part of it is associated with chronic sun exposure, and part by recreational sun exposure (29). In contrast, the distributions of NMSC (7) and melanoma (6) in blacks are weighted more towards infrequently sun-exposed sites than frequently sun-exposed sites, probably because of the protection from sunlight afforded by melanin. A special review of medical documents revealed that on the sole of the foot, where sun exposure has presumably little effect, United States whites and blacks have similar melanoma incidence rates (33). Routinely coded data on melanoma incidence are available at five general site combinations, which we studied individually for UVB effects. For black males and black females, melanoma incidence rates increased (insignificantly) with increasing UVB level at the face, head, and neck sites combined and at the lower limb and hip sites combined, suggesting that both recreational and chronic sun exposure may contribute to the risk of melanoma in blacks. However, the site-specific rates for blacks were not consistent with those for whites in that at the trunk site, rates were much higher for white males than for white females, and, at the lower limb and hip sites, rates were much lower for white males than for white females, in accordance with other studies; yet, at both of these site combinations, the rates for black males and black females were about equal (Table 3). Possible explanations include melanin protection from sunlight and the lack of power to detect differences between very low black male and black female rates.

The major limitation of a study of skin cancer mortality rates is that early treatment may prevent death from melanoma and from NMSC, especially the common BCC and SCC forms of NMSC. Thus, factors such as low socioeconomic status that inhibit access to timely diagnosis and treatment could influence skin cancer mortality rates. In order for such factors to confound the association between UVB radiation exposure and skin cancer mortality, poor access to treatment would also need to be associated with latitude. Treatment of prostate (34) and breast cancer (35) has been found to vary geographically in the United States, although not in a consistent north-to-south fashion. It is possible that access to treatment of skin cancer is poorer in more southern latitudes, resulting in higher mortality rates in such locations, but it seems more likely that the latitude effects on mortality reflect biological effects of UVB radiation exposure. If poorer access to treatment were associated with latitude, then one might expect mortality rates for all cancers combined to increase with UVB radiation level, but these rates decreased significantly for all of the four race–gender groups. One might also expect the risk increase in males and females to
be about the same for the same increase in UVB level, but it was significantly smaller for black females than black males for melanoma.

Another factor that could confound associations of UVB radiation exposure with skin cancer is geographical variation of skin color. Light skin color has been associated with increased risk of NMSC among whites (1–5) and with BCC among blacks (10). Therefore, our geographical associations of UVB radiation exposure with skin cancer rates among black males could be explained by a higher prevalence of lighter-skinned blacks in more southern latitudes. We are unaware, however, of empirical data demonstrating that the proportion of admixture of blacks and whites varies by latitude or that the migration of blacks from the South to the rest of the United States during this century varied by skin color.

A reviewer pointed out that since the end, centuries ago, of the trans-Atlantic importation of slaves, a gradual depigmentation of North American blacks may have occurred because of the lack of an environmental basis for skin pigmentation, leading to increased risk of skin cancer caused by UV radiation exposure. We are unaware of studies that compare the skin color of African-Americans to that of native-African blacks. An extensive review of evolutionary theories of skin color is given in Robins (36).

Future case-control studies of incident skin cancer could help to clarify the role of UVB radiation exposure on the risk of melanoma and NMSC in blacks. However, even if increases in risk are confirmed for blacks and other nonwhites, the absolute increases in risk for these population groups will be much smaller than for whites, because these groups have incidence rates much lower than whites (Table 3; Refs. 6, 7). For example, based on the incidence rates in Ref. 7, an increase in UVB radiation exposure that doubles the incident risk of BCC increases the incidence rate in blacks from 2 to only 4 cases per 100,000 person-years at risk, whereas it increases the incidence rate in whites from 360 to 720. Thus, care would be required to fashion recommendations for prevention, such as sun-blocking agents, that are warranted and acceptable for blacks and other nonwhite populations.

References


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