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THE AUTHORS REPLY

We thank Dr. Grant (1) and Dr. Hu (2) for their comments regarding our prospective study of ambient ultraviolet radiation (UVR) and mortality risk in a geographically diverse cohort of Americans (3). Dr. Grant’s comments focus on vitamin D production, whereas our study examined exposure to ambient UVR, which may have effects, beneficial or detrimental, in addition to vitamin D production. He suggests that UVR doses estimated from satellite measurements from NASA’s Total Ozone Mapping Spectrometer are in the range for ultraviolet A but not ultraviolet B radiation. The ambient UVR estimate was the erythemal exposure data product (an integral from 280 nm to 400 nm), which is a standard measure of UVR exposure but is not specifically designed to estimate vitamin D production potential. This UVR dose is an estimate of the daily integrated ultraviolet irradiance from a model for the susceptibility of Caucasian skin to sunburn and can be interpreted as an index of the potential for biological damage due to solar irradiation after taking into account the ozone and cloud conditions (4).

The erythemal exposure data product was the appropriate measure for a study focused on the effects of ambient UVR exposure, which is not associated with any specific process but is likely correlated with many processes, including vitamin D production. Others have used the same data source to examine associations with health outcomes (e.g., Freedman et al. (5)). A conversion between erythemal and vitamin D action spectra for various conditions may be conducted for studies specific to vitamin D production (6).

We agree with Dr. Grant that the use of wintertime UVR doses could yield different results. We selected the July UVR dose rather than a wintertime dose for a number of reasons, including the fact that summer is when surface UVR is strongest, noise factors such as clouds and aerosols are not as influential (7), and the satellite UVR data are in better agreement with ground-based UVR data (8). Moreover, for the regions in our analysis, the dynamic range for the UVR dose is broad in the summer but narrow in the winter, and it is unlikely that an effect would be apparent in the narrow range of exposure in our cohort in the winter. Dr. Hu states that some states may have longer summers, but we chose to use a consistent method across states for assigning ambient UVR exposure. Dr. Hu also suggests that the exposure window should have covered enrollment to death or end of follow-up, but we chose to integrate the doses across all measured periods because NASA data were not available for the full follow-up period.

Dr. Grant discusses squamous cell carcinomas and basal cell carcinomas, both of which are types of nonmelanoma skin cancers. Because they are both linked to sun exposure (9), we previously reported that our measure of ambient UVR exposure was associated with self-reported nonmelanoma skin cancer (10) as a positive control for our analysis with the UVR data. Our data did not allow for separation of squamous from basal cell carcinoma, and given the low rate of nonmelanoma skin cancer mortality in the United States (0.69 deaths per 100,000 people) (11), it was not feasible to examine the associations between ambient UVR exposure and this cause of death in our study.

We agree with Dr. Hu’s comments that our lack of area-level health care access indicators may be a potential limitation, which we had noted in the text. Both Drs. Grant and Hu suggest that air pollution may be an important confounder. We accounted for potential residual correlations due to geography as a proxy for additional environmental factors, among other factors, by including a random effect for census tract in our Cox models and saw no difference in risk estimates. Given that our crude and multivariate adjusted models yielded similar risk estimates, additional adjustment for other area-level confounders may not lead to major changes in our risk estimates. However, in the future, some air pollution data will be linked to our cohort to allow for further adjustments. Dr. Hu suggests that several states in which our enrolled subjects reside are in the stroke belt. We agree that there are potential regional differences across these states, but as the cause of this phenomenon is yet unclear, we did not exclude these subjects from our analysis.

Dr. Grant suggests that changes in subject characteristics may have occurred in our population during follow-up. An ideal study would collect data on subject characteristics at regular intervals throughout follow-up. Given that our crude and multivariate-adjusted models yielded similar risk estimates, updated information would likely have had little impact on our estimates. Furthermore, we tested for and found no deviations from the proportionality assumption in our Cox models. Some of our mortality outcomes, such as cancer mortality, have a long latency period, so collecting characteristics at baseline may better reflect the relevant etiologic window.

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REFERENCES


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