Published in final edited form as:
Int J Biometeorol. 2014 April ; 58(3): 361–370. doi:10.1007/s00484-013-0631-5.

The Relationship Between Long-Term Sunlight Radiation and Cognitive Decline in the REGARDS Cohort Study

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Abstract

BACKGROUND—Sunlight may be related to cognitive function through vitamin D metabolism or circadian rhythm regulation.

METHODS—Fifteen-year residential history merged with satellite and ground monitor data were used to determine sunlight (solar radiation) and air temperature exposure for a cohort of 19,896 cognitively intact black and white participants aged 45+ from the 48 contiguous United States. 15, 10, 5, 2, and 1-year exposures were used to predict cognitive status at the most recent assessment in logistic regression models.

RESULTS—1-year insolation and maximum temperatures were chosen as exposure measures. Solar radiation interacted with temperature, age, and gender in its relationships with incident cognitive impairment. After adjustment for covariates, the odds ratios of cognitive decline for solar radiation exposure below the median vs. above the median in the 3rd tertile of maximum temperatures was 1.88 (95% CI: 1.24, 2.85), the 2nd tertile was 1.33 (95% CI: 1.09, 1.62), and 1st tertile was 1.22 (95% CI: 0.92, 1.60). We also found that participants under 60 year old had an OR=1.63 (95% CI: 1.20, 2.22), those 60 to 80 years old had an OR=1.18 (95% CI: 1.02, 1.36), and those over 80 years old had an OR=1.05 (0.80, 1.37). Lastly, we found that males had an OR=1.43 (95% CI: 1.22, 1.69), and females had an OR=1.02 (0.87, 1.20).

CONCLUSIONS—We found that lower levels of solar radiation were associated with increased odds of incident cognitive impairment.

Keywords

Sunlight; Temperature; Weather; Climate; Remote Sensing Technology; Cognition

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BACKGROUND

Sunlight has a profound effect on vitamin D status as well on other aspects of human physiology, but there are little data on how sunlight directly affects human health (Reusch et al. 2009; Wong 2008; Turner and Mainster 2008). Vitamin D status can also be affected by diet, but for most people it is primarily determined by sunlight exposure (Reusch et al. 2009; Webb 2006). When exposed to UVB radiation, the skin produces vitamin D_3, which the liver then converts to 25(OH)D. Vitamin D supplements and fortifications often consist of vitamin D_2, which is also converted to 25(OH)D. The kidney is the primary site to convert 25(OH)D to its more active form, $1,25(OH)_2D$. While 25(OH)D serum levels vary widely with differing sun exposure, $1,25(OH)_2D$ is tightly regulated and rarely fluctuates. Blood serum levels of 25(OH)D are usually used to determine vitamin D status, thus can be affected by both cutaneous and ingested vitamin D sources (Holick 2007).

Vitamin D was previously thought to only affect bone metabolism, but may also be related to various chronic diseases (Rosen 2011). Sunlight is a widely accepted factor in depression, and cognitive function may be affected as well (Kent et al. 2009). Vitamin D status has been associated with cognitive function and seasonal affective disorder symptoms (Wilkins et al. 2009; Oudshoorn et al. 2008; Gloth et al. 1999). The brain has vitamin D receptors, and vitamin D may be neuroprotective (Eyles et al. 2005; Ibi et al. 2001; Brewer et al. 2001). Besides vitamin D status, environmental sunlight patterns may affect human physiology and cognitive function through the body's circadian rhythms. Amounts and patterns of light exposure affect mood through the body's internal clock, the suprachiasmatic nuclei (SCN) (Turner and Mainster 2008). Leonard and Myint, 2006 laid out a paradigm showing how environmental illumination and other stresses might lead to altered serotonin levels, neurodegeneration, depression, cognitive deficits, and ultimately dementia (Leonard and Myint 2006). Serotonin and melatonin regulation, mechanisms that are involved in the relationship between sunlight and light therapy on mood, are also involved in cognition, which suggests that cognitive function may also be influenced by light (Van Someren and Riemersma-Van Der Lek 2007; Turner and Mainster 2008; Srinivasan et al. 2006a; Winkler et al. 2006).

While geographic latitude corresponds to the number of daylight hours, atmospheric and geographic conditions can lead to different daily sunlight exposure times and intensities at similar latitudes (Chen et al. 2007). To better determine sunlight and air temperature exposure, this study used measures derived from ground and satellite data from the North American Land Data Assimilation System Phase 2 (NLDAS-2) dataset. These data were merged with the current and most recent 15-years of residential history for participants in the REasons for Geographic and Racial Differences in Stroke (REGARDS) study, which enrolled 30,239 African-American and white participants in the 48 contiguous United States (US). Using these data, this analysis sought to test whether ground and satellite measures of solar radiation are associated with cognitive decline.

METHODS

Study participants

To explore the relationships between risk factors and stroke incidence, the REGARDS study began enrollment in January, 2003 and completed enrollment in October, 2007 (Howard et al. 2005). REGARDS is a longitudinal study in which participants aged 45 years and older were sampled from the 48 contiguous United States, with oversampling in the "stroke belt", a high stroke mortality region consisting of the eight southeastern states of Arkansas, Louisiana, Tennessee, Mississippi, Alabama, Georgia, North Carolina, and South Carolina, and the "stroke buckle", a region with an even higher stroke mortality that lies within the

stroke belt along the coastal plains of Georgia, North Carolina, and South Carolina. At baseline, 35% of the participants were residing the non-buckle portion of the stroke belt, and 21% of the participants were sampled from the stroke buckle. The remaining 44% were from the rest of the nation. The cohort population at baseline was 42% African-American/58% white and 45% male/55% female. All participants provided written informed consent, and the study was approved by the Institutional Review Board for Human Subjects at the University of Alabama at Birmingham, as well as all other participating institutions. Further details on the study are available elsewhere (Howard et al. 2005).

Data collection

At baseline, a telephone interview was conducted which recorded the participant's self-reported demographic and behavioral factors, and medical history. In addition, at this time validated interview instruments were used to collect depression symptoms (the Center for Epidemiological Studies-Depression Scale-4 item version) (Melchior et al. 1993), cognitive status (Six-Item Screener derived from the Mini-Mental State Examination) (Callahan et al. 1995; Wadley et al. 2007), and physical function (Physical Component Summary score from the SF-12 Health Survey) (Ware et al. 1996). Shortly following the telephone call, participants were visited in their homes by a trained health professional from Examination Management Services, Inc., who collected blood pressure, height, weight, venipuncture, urine, and conducted electrocardiograms (ECG). Blood pressure was measured by a trained technician using a standard protocol and regularly tested aneroid sphygmomanometer and was calculated as an average of two measurements taken after the participant was seated for five minutes. The blood and urine were sent to the central repository at the University of Vermont and ECG data were sent to Wake Forest University to be read. During the in-home visit, the examiner also left several self-administered questionnaires; including a "Places You Have Lived" questionnaire used to determine residential history, and the Block98 food frequency questionnaire (NutritionQuest™, Berkeley, CA), which was used to determine dietary (but not supplemental) vitamin D intake.

Assessment of cognitive function—The Six-Item Screener (SIS), a screener based on the Mini-Mental State Examination, was used to evaluate global cognitive status by assessing short-term recall and temporal orientation (Callahan et al. 2002; Folstein et al. 1975; Wadley et al. 2007). The SIS was administered for each participant either during the baseline telephone interview or during the first follow-up after December 2003. Thereafter, a version of the SIS has been re-administered every 12 months. Following the practice of previous REGARDS studies, the score of this screener was dichotomized into an outcome of cognitively impaired or intact (Wadley et al. 2007). A score of four or fewer correct responses out of the six questions indicated cognitive impairment. Callahan et al. 2002 validated the screener in both a community-based population of 344 black adults aged 65 or older and a population of 651 subjects who were referred to an Alzheimer's Disease Center (84% white/16% black) (Callahan et al. 1995). Results from the community-based sample found that for a six-item screener score of 4 or fewer, using clinically confirmed cognitive impairment as the gold standard, the sensitivity was 74% and specificity was 80% (Callahan et al. 2002). It was also validated against other cognitive measures and diagnoses of both dementia and non-dementia cognitive impairment (Callahan et al. 2002).

Assessment of sunlight exposure and temperature

Sunlight radiation and air temperature are freely available from the *National Aeronautics and Space Administration* (NASA) using the data product, the NLDAS-2. The NLDAS-2, which includes information from satellites and ground observations, is derived from a previous data product, the North American Regional Reanalysis (NARR), and consists of a grid surface with a ~14 km resolution over North America (NOAA/NCEP/EMC 2010). In

two previous published studies by the authors of this manuscript, NARR data has been matched to REGARDS participants' geocoded home residence during their in-home visit (Kent et al. 2009; Kent et al. 2011). In the NLDAS-2, solar radiation that was assessed at one-hour intervals was used to calculate a daily sum that is also referred to as "insolation". For time periods previous to the first SIS assessment, we compiled daily solar radiation and daily maximum temperature, which we then merged with the REGARDS self-administered residential history form. This form consists of locations where the participant had lived prior to enrollment into REGARDS, along with the ages s/he moved. Each location the participant recorded was matched with a feature in the US Geological Service's Geographic Names Information System using ArcGIS 9.2. To calculate monthly environmental exposures, it was assumed the participant moved in July of the indicated year for a residential move. For participants who had a period of missing residential data, due to having an unidentifiable location or residence outside of the contiguous 48 United States, we used only the existing residential history to compute environmental exposure averages.

We explored which models containing each of 15-year, 10-year, 5-year, 2-year, or 1-year exposures had better fits using Akaike Information Criterion (AIC) values. We initially categorized solar radiation and temperature exposures into quartiles. To avoid low cell numbers (<20 participants) when exploring interactions, we also categorized temperature as tertiles. Since our hypothesis was that lower levels of sunlight might have an adverse effect on incident cognitive impairment, the 4^{th} quartile of solar radiation was used as a reference. Since both high and low temperatures might have adverse effects on incident cognitive impairment, we did not a priori choose a reference.

Definitions

Main outcome: The main outcome was cognitive decline (coded yes or no), as defined by a shift in classification from intact cognitive status (SIS=5 or 6) at baseline to impaired (SIS≤4) at the most recent SIS assessment.

Main exposure variables: The main exposure variable was average solar radiation at residence during the past 15 years. We tested for additive interactions between solar radiation and air temperature, age, and gender. We also examined the relationships using the average daily maximum air temperature and average solar radiation for 10, 5, 2, and 1-year exposure periods.

Statistical Methods

Data management and statistical analyses were performed using SAS version 9.2 (SAS Corporation, Cary, North Carolina). We used likelihood ratio chi-square, *Wilcoxon* signed rank, and t-tests to univariately assess the relationships between covariates and cognitive decline. Of the 30,239 participants enrolled at baseline in REGARDS, 56 were excluded from the cohort due to data anomalies. Of the 30,183 participants available for analyses, 4,249 were excluded due to cognitive impairment at baseline (n=2,639), or self-reported stroke (n=3194), leaving 25,934 participants meeting the proper criteria for analysis. A further 4,910 were missing residential histories. Since many confounders had large numbers of participants missing data, we attempted to minimize selection bias by creating a separate "missing" category for any variable that had more than 500 participants missing data. After this, another 1,128 still had missing confounder data, leaving a final 19,896 participants for analyses.

We used logistic regression to determine relationships of solar radiation and daily maximum air temperature with cognitive decline, defined as yes or no. Because follow-up periods differed between participants, we included the total number of days in the follow-up interval

as a covariate in all regression models. We ran models to test for the relationship of solar radiation and temperature with cognitive decline, using two univariate models. We then considered models examining three separate additive interactions: (1) a 6-category variable indicating both the participant's temperature tertile and whether s/he was above or below the median solar radiation exposure; (2) a 6-category variable indicating both the participant's age range (under 60, 60 to 80, and over 80 years old) and whether s/he was above or below the median solar radiation exposure; (3) and a 4-category variable indicating both the participant's gender and whether s/he was above or below the median solar radiation exposure. We considered the potential demographic confounders of age in years, race (black or white), region (stroke belt/non-buckle, stroke buckle or non-belt at in-home visit), gender, rurality (based on the United States Department of Agriculture's rural-urban commuting area codes from 2000), education (years of schooling completed), individual income, and neighborhood poverty (percentage of census tract below poverty). We also considered the potential behavioral confounders of daily vitamin D dietary intake, number of times exercised per week, hours of TV/computer watched, statin use at baseline, alcohol use based on number of drinks per week (none, moderate: 0 to 7 for women and 0 to 14 for men, or heavy: over 7 for women and over 14 for men) and smoking status (never, past, current). Total vitamin D intake would be a variable of importance, considering our proposed mediating pathway may involve vitamin D metabolism; however our vitamin D intake did not include supplemental vitamin D, so we used this variable only as a confounder and did not directly analyze the relationship between Vitamin D dietary intake and cognitive decline. Lastly, we included medical risk factors: presence of depressive symptoms (≥4 points on a 12 point, 4-item screener derived from the Center for Epidemiological Studies Depression Scale), PSS (Perceived Stress Scale), the Physical Component Summary (PCS) score from the SF-12, body mass index (BMI), dyslipidemia (total cholesterol at least 200 or low-density lipids at least 160 or high-density lipids not greater than 40 or on lipid-lowering medication), diabetes (fasting glucose ≥126 mg/dL, non-fasting glucose ≥140 mg/dL, or use of insulin or other diabetes medications), history of reported TIA, or reported stroke symptoms at baseline (among those without self-reported stroke, using the 8-item *Questionnaire for Verifying Stroke-Free Status*).

Logistic regression models results are reported using odds ratios (ORs) accompanied by 95% profile likelihood confidence intervals and type III chi-square tests to determine p-values. Statistical significance was set at α=0.05. These analyses were not adjusted for the multiple comparisons that were made.

RESULTS

Solar radiation was found to have a threshold effect at the median and there were low numbers of participants in some cells (<20) when interaction variables were created, so solar radiation exposure was collapsed from quartiles to a 2 category variable that characterized whether the participant's residence received above or below the median exposure. Although 15-year meteorological exposure period were initially investigated as the main exposure variable, since sensitivity analyses showed 1-year exposures to have the best fit in the analyses below, we primarily displayed results using 1-year exposures.

Table 1 shows all exposure variables by cognitive decline status. Those with incident cognitive impairment showed shorter median follow-up intervals (1,475 vs. 1,833 days; p<0.0001; Table 1). 1-yr solar radiation exposure (p=0.028), but not temperature (p=0.20) showed a significant relationship with incident cognitive impairment (Table 1). Most covariates showed significant differences by cognitive decline status (Table 1). Of the demographics: males, blacks, less educated, lower income, higher neighborhood poverty, and older participants all showed higher proportions of cognitive decline (all p<0.0001;

Table 1). However, rurality (p=0.91) did not differ by incident impairment (Table 1). All behavioral confounders were significantly related to cognitive decline, as well as all medical factors, with the exception of dyslipidemia (p=0.066; Table 1).

In univariate regression analyses, participants below the median solar radiation exposure for one year before baseline showed 1.23 (95% CI: 1.10, 1.37) times the odds of cognitive decline. Participants in the middle tertile of previous year's temperature exposure showed the lowest odds of cognitive decline, with both the lower tertile (OR=1.11; 95% CI: 0.98, 1.27) and the upper tertile (OR=1.08; 95% CI: 0.95, 1.23) showing weak, non-statistically significant increases in odds of cognitive decline (data not shown).

Table 2 shows that the different time intervals of solar radiation and temperature exposure had different relationships, but there was no clear trend for shorter or longer-term intervals to carry stronger relationships. We chose the 1-year time interval, since it had the best fit (AIC=10400.14; Table 2), but the second best fit was the 15-year interval (AIC=10410.67; Table 2), and the worst fit was in the 5-year interval (AIC=10423.79; Table 2). Associations between sunlight and incident impairment generally had monotonic increases in strength with increasing temperature tertiles (Table 2). For the 1-year time period, the 3rd tertile of temperature had the strongest relationship (OR=2.19; 95% CI: **1.**46, 3.28), the 2nd tertile had a weaker, but still significant relationship (OR=**1.**47; 95% CI: **1.**21**, 1.**80), and the 1st tertile did not have a significant relationship (OR=**1.**27; 95% CI: **0.**96**, 1.**67) (Table 2). After adjustment of all covariates in Table 2, the 6-category solar radiation/temperature variable was still significant (p=0.0015). The relationships between solar radiation exposure and cognitive decline in the 3rd tertile (OR=1.83; 95% CI: 1.19, 2.81), the 2nd tertile (OR=1.37; 95% CI: 1.11, 1.69), and 1st tertile of temperature (OR=1.30; 95% CI: 0.97, 1.73) were only slightly attenuated (data not shown).

AICs for different exposure time intervals for models including either the 6-category solar radiation/age or 4-category solar radiation/gender interaction variables showed similar patterns to models including the 6-category solar radiation/temperature variable, with the 1-year time interval again marginally showing the best fit (data not shown). The addition of potential confounders did not change the relationships for either of these interaction models. After the addition of potential confounders, those under 60 year old had an OR=1.63 (95% CI: 1.20, 2.22), those 60 to 80 years old had an OR=1.18 (95% CI: 1.02, 1.36), and those over 80 years old had an OR=1.05 (0.80, 1.37). In the last covariate-adjusted model, males had an OR=1.43 (95% CI: 1.22, 1.69), and females had an OR=1.02 (0.87, 1.20).

DISCUSSION

The results of this analysis find that lower levels of solar radiation are related to increased odds of incident cognitive impairment. This relationship was not significantly altered by the addition of various confounders. Possible pathways for a relationship between sunlight and cognitive decline could be either through vitamin D metabolism or circadian rhythm regulation via the SCN. Since sunlight increases vitamin D blood levels, it is of interest that recent studies have found a relationship between higher vitamin D blood levels and improved cognitive function (Wilkins et al. 2009; Oudshoorn et al. 2008; Rosen 2011). However, this relationship is not yet established and a recent study even found a significant inverse relationship between vitamin D blood levels and academic performance in children (Tolppanen et al. 2012; Barnard and Colon-Emeric 2010). An alternative pathway may be through sunlight's effect on the hypothalamic SCN. The SCN are already known to be involved in the relationship between light and depression, and through these same mechanisms may affect cognitive function (Van Someren and Riemersma-Van Der Lek 2007; Turner and Mainster 2008). One of the SCN's regulatory functions is to inhibit the

pineal gland from turning serotonin into melatonin in the presence of daytime light (Miller 2005). Melatonin and serotonin regulation abnormalities have been found to vary according to sunlight and light therapy in both patients with and without mood disorders (Khait et al. 2002; Leppamaki et al. 2003; Srinivasan et al. 2006a; Jakovljevic et al. 1997; Golden et al. 2005). Serotonin and melatonin have also been implicated in many mental and cognitive disorders, such as Alzheimer's disease, Parkinson's disease, and sleep disorders (Khait et al. 2002; Srinivasan et al. 2006b).

There has not been much previous research directly examining a relationship between sunlight and cognitive function or decline. We have previously published a cross-sectional analysis also using REGARDS and NASA data, which showed a relationship between SIS-determined impairment at baseline and average solar radiation exposure for the previous two weeks (Kent et al. 2009). This relationship was found only among participants exhibiting depressive symptoms, and only for the lowest of 5 solar radiation categories that approximated quintiles. For the long-term solar radiation exposures used in the current analysis, we found a threshold effect at the median rather than the lowest solar radiation category. We found that the 1-year exposure exhibited the relationship with the best fit, although there was not a general trend that shorter exposure periods had better-fitting relationships and the 15-year exposure had the next best fit. The only other study we are aware of that examined a relationship between sunlight and cognitive function found that higher barometric pressure (a proxy for sunny weather) was associated with improved cognitive performance (Keller et al. 2005).

The relationship between sunlight and cognitive decline was stronger among those residing in warmer temperatures, among males, and among younger participants. It is possible we found these results because the relationship between solar radiation and cognitive function is stronger in participants that spend more time outdoors (Graham and McCurdy 2004). Those spending more time outdoors would be more able to take advantage of the available sunlight, whereas those spending little time outdoors would be less exposed to variations in solar radiation. The only previous study finding a relationship between sunny weather and cognitive function found a stronger relationship among those who spent more time outdoors (Keller et al. 2005). These findings may seem to contradict previous literature examining the relationship between temperatures and mortality, which have found stronger relationships among females and older individuals. Some hypothesized mechanisms in these previously found interactions may also apply to this analysis: females might be more vulnerable to the effects of weather due to clothing that generally exposes more skin or through physiological differences, and older individuals might be more vulnerable due to cardiovascular aging processes (Basu and Samet 2002; Barnett et al. 2007; Alperovitch et al. 2009). However, physiological responses to differing temperature and sunlight exposures are bound to differ, so comparing the health effects of temperature and sunlight exposures may be of limited use.

Potential limitations

As noted in our previous study using the similar datasets and methods, exposure misclassification exists as a possible source of bias for the study. This could happen if during the time period of an exposure measurement a participant spent a large amount of time in a climate different from that indicated by the outdoor exposures linked to his or her residence. However, this analysis represents on improvement on previous studies involving meteorological variables, where differences have been shown simply by comparison of latitudes or by proxy variables such as air pressure (Keller et al. 2005; Rostand 1997). Another potential limitation is that there may be confounders for which we have not accounted. In particular, there may be other environmental variables that correlate with those in this study that may account for the results. Our study had no measures of air pollution, which is related to temperature and sunlight and has been found to be related to

neuroinflammation and cognitive deficits (Block and Calderon-Garciduenas 2009; Calderon-Garciduenas et al. 2008). In the future, studies should account for confounding or effect modification by pollutants such as ozone and particulate matter. In addition, repeated measures of cardiovascular factors, vitamin D blood serum levels, sleep patterns, and other factors may help explain mediating pathways in future analyses.

CONCLUSIONS

This longitudinal analysis used a large cohort to give evidence of a significant and novel relationship between reduced sunlight exposure and cognitive decline. This study is innovative in its use of REGARDS' residential histories and NASA-obtained NLDAS-2 data to estimate long-term climate exposure. This adds to our previous research which used the same cohort and NASA data to show evidence of a cross-sectional relationship between 2 weeks sunlight exposure and cognitive function. Mechanisms are yet undetermined, but are hypothesized to be via vitamin D metabolism or circadian rhythm regulation.

Acknowledgments

The authors thank the other investigators, the staff, and the participants of the REGARDS study for their valuable contributions. A full list of participating REGARDS investigators and institutions can be found at http://www.regardsstudy.org. This research project is supported by a cooperative agreement U01 NS041588 from the National Institute of Neurological Disorders and Stroke. Additional funding, data, data processing, and consultation were provided by an investigator-initiated grant from National Aeronautics and Space Administration (grant# NNX09AV81G). The NLDAS hourly data used in this study were acquired as part of the mission of National Aeronautics and Space Administration's Earth Science Division and archived and distributed by the Goddard Earth Sciences Data and Information Services Center.

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Table 1

Characteristic distributions by incident impairment (n=19,896)

	Incident impairment		
Characteristic	**No (n=19896)**	**Yes (n=1470)**	**p-value**
Median follow-up interval (Q1, Q3)	1833 (1110, 1879)	1475 (1099 (1848)	<0.0001
Below median one year solar radiation, n (%)	9167 (49.8%)	771 (52.4%)	0.047
One year maximum temperature, n (%)			
1st quartile	6158 (33.4%)	499 (33.9%)	
2nd quartile	6270 (34.0%)	468 (31.8%)	0.20
3rd quartile	5995 (32.5%)	503 (34.2%)	
Solar radiation/Temperature, n (%)			
< median solar radiation, 1st tertile temperature	5289 (28.7%)	436 (29.7%)	
< median solar radiation, 2nd tertile temperature	3703 (20.1%)	306 (20.8%)	
< median solar radiation, 3rd tertile temperature	175 (0.9 %)	29 (2.0 %)	0.0006
> median solar radiation, 1st tertile temperature	869 (4.7 %)	63 (4.3 %)	
> median solar radiation, 2nd tertile temperature	2567 (13.9%)	162 (11.0%)	
> median solar radiation, 3rd tertile temperature	5820 (31.6%)	474 (32.2%)	
Demographics			
Male, n (%)	7826 (42.5%)	758 (51.6%)	<0.0001
Black, n (%)	6363 (34.5%)	725 (49.3%)	<0.0001
Education, n (%)			
< High school	1513 (8.2 %)	282 (19.2%)	
High school	4565 (24.8%)	413 (28.1%)	<0.0001
Some college	5059 (27.5%)	366 (24.9%)	
College graduate	7286 (39.5%)	409 (27.8%)	
Income, n (%)			
< $20k	2586 (14.0%)	359 (24.4%)	
$20k–$34k	4265 (23.2%)	403 (27.4%)	<0.0001
$35k–$74k	6009 (32.6%)	370 (25.2%)	
>= $75k	3516 (19.1%)	156 (10.6%)	
Refused	2047 (11.1%)	182 (12.4%)	
Neighborhood poverty, n (%)			
<25% poverty	12838 (69.7%)	922 (62.7%)	<0.0001
>=25% poverty	3835 (20.8%)	416 (28.3%)	
missing	1750 (9.5 %)	132 (9.0 %)	
Rurality, n (%)			
Urban	13336 (72.4%)	1066 (72.5%)	
Large rural	1940 (10.5%)	158 (10.7%)	0.91
Small rural/isolated	1397 (7.6 %)	114 (7.8 %)	
missing	1750 (9.5 %)	132 (9.0 %)	
Age, mean yrs (SD)	63.9 (9.0)	69.7 (9.4)	<0.0001[c]
Behavioral			

	Incident impairment		
Characteristic	**No (n=19896)**	**Yes (n=1470)**	**p-value**
Vitamin D intake, n (%)			
1st quartile (1–66)	3765 (20.4%)	243 (16.5%)	
2nd quartile (66–112)	3806 (20.7%)	272 (18.5%)	<0.0001
3rd quartile (112–184)	3754 (20.4%)	265 (18.0%)	
4th quartile (184–1870)	3680 (20.0%)	277 (18.8%)	
Missing	3418 (18.6%)	413 (28.1%)	
Taking statins, n (%)	5589 (30.3%)	522 (35.5%)	<0.0001
Smoking status, n (%)			
Current	2484 (13.5%)	184 (12.5%)	0.0033
Past	7278 (39.5%)	647 (44.0%)	
Never	8661 (47.0%)	639 (43.5%)	
Alcohol Status, n (%)			
None	10935 (59.4%)	993 (67.6%)	<0.0001
Moderate	6698 (36.4%)	427 (29.0%)	
Heavy	790 (4.3 %)	50 (3.4 %)	
Exercise status, n (%)			
None	5895 (32.0%)	523 (35.6%)	0.0012
1 to 3 times/week	7022 (38.1%)	493 (33.5%)	
>=4 times/week	5506 (29.9%)	454 (30.9%)	
TV/Video watched, n (%)			
1 hr/day or less	12668 (68.8%)	1011 (68.8%)	<0.0001
2 hrs/day or more	3367 (18.3%)	219 (14.9%)	
Missing	2388 (13.0%)	240 (16.3%)	
Medical factors			
Depressive symptoms present, n (%)	1656 (9.0 %)	168 (11.4%)	0.0025
Perceived stress scale, n (%)			
1st quartile	4794 (26.0%)	377 (25.6%)	
2nd quartile	4604 (25.0%)	330 (22.4%)	0.018
3rd quartile	5528 (30.0%)	439 (29.9%)	
4th quartile	3497 (19.0%)	324 (22.0%)	
PCS, n (%)			
1st quartile (11–40)	3738 (20.3%)	391 (26.6%)	
2nd quartile (40–50)	4293 (23.3%)	390 (26.5%)	<0.0001
3rd quartile (50–55)	4701 (25.5%)	337 (22.9%)	
4th quartile (55–69)	5060 (27.5%)	282 (19.2%)	
Missing	631 (3.4 %)	70 (4.8 %)	
BMI, n (%)			
<18.5	177 (1.0 %)	12 (0.8 %)	
18.5–24.9	4384 (23.8%)	391 (26.6%)	0.028
25–29.9	6895 (37.4%)	561 (38.2%)	
>=30	6967 (37.8%)	506 (34.4%)	

	Incident impairment		
Characteristic	**No (n=19896)**	**Yes (n=1470)**	**p-value**
SBP, mean mmHg (SD)	126 (16)	130 (18)	<0.0001[c]
Dyslipidemia			
Yes	10320 (56.0%)	869 (59.1%)	0.066
No	7481 (40.6%)	557 (37.9%)	
Missing	622 (3.4 %)	44 (3.0 %)	
Diabetic, n (%)			
Yes	3226 (17.5%)	376 (25.6%)	<0.0001
No	14587 (79.2%)	1048 (71.3%)	
Missing	610 (3.3 %)	46 (3.1 %)	
Has Stroke Symptoms, n (%)	2566 (13.9%)	286 (19.5%)	<0.0001
Self-reported TIA, n (%)	648 (3.5 %)	89 (6.1 %)	<0.0001

SD=standard deviation; n=Number; IQR=InterQuartile Range;; PCS=Physical Component Score; BMI=Body Mass Index; SBP=Systolic Blood Pressure; CRP=C-reactive protein; TIA=Transient Ischemic Attack

P-values for categorical variables determined by likelihood-ratio chi-square tests and for continuous variables determined by Satterthwaite t-tests

[a] p-value obtained from Wilcoxon

[b] p-values obtained from pooled t-test

[c] p-values obtained from Satterthwaite t-test

Table 2

ORs for the unadjusted relationships between below the median solar radiation exposure and incident impairment, by maximum temperature tertile (n=19,896)

Time Period	Among 1st temperature tertile	Among 2nd temperature tertile	Among 3rd temperature tertile	AIC
1 year	1.27 (0.96, 1.67)	1.47 (1.21, 1.80)	2.19 (1.46, 3.28)	10400.14
2 year	1.30 (0.99, 1.71)	1.29 (1.06, 1.57)	2.25 (1.33, 3.79)	10414.12
5 year	1.20 (0.92, 1.56)	1.26 (1.03, 1.53)	1.26 (0.65, 24.5)	10423.79
10 year	1.21 (0.92, 1.61)	1.25 (1.03, 1.52)	2.05 (1.20, 3.51)	10418.61
15 year	1.33 (0.97, 1.81)	1.42 (1.17, 1.72)	1.97 (1.11, 3.49)	10410.67