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Estimations of the human 'vitamin D' UV exposure in the USA

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Human exposure to sunlight promotes the formation of pre-vitamin D in the skin. Low or marginal levels of vitamin D has been linked to a wide range of human health outcomes, including the development of various types of cancer. However, few data exist on the actual exposure to human due to vitamin D producing ultraviolet radiation. Most studies of human disease and vitamin D have linked latitude and location of residence to expected exposure form the available ambient UV radiation. Human UV exposure for the development of vitamin D depends on a variety of factors such as time spent outdoors, percent available skin, skin type, UV protective devices used and distribution of UV over the human form. In this paper, we investigate how latitude impacts not only on the amount of UV available for vitamin D synthesis, but also the distribution of UV over the human form.

1.0 Introduction

Sunlight is the main source of vitamin D for humans, however, vitamin D may also be ingested through diet and dietary supplements. The relative effectiveness of sunlight to produce pre-vitamin D is governed by the vitamin D action spectrum.[1] This action spectrum indicates that the shorter UVB (280–320 nm) wavelengths are most responsible for pre vitamin D formation. Vitamin D has been linked to a wide range of human health outcomes. Grant[2] found that a latitude gradient exists for increased risks of colon, breast, rectal cancer in the USA and suggested that ambient levels of ultraviolet (UV) radiation are responsible for dermal vitamin D production. These findings have drawn us to consider the impact of our environmental exposure to the available UV, and how this impacts on our health and well-being. However, few data exist on the impact of location on human UV exposure. We directly address these issues through the results presented in this paper.

2.0 Methodology

The data presented in this paper are based on solar ultraviolet (UV) irradiance measurements are from the US Environmental Protection Agency (EPA) network of MK IV Brewer spectrophotometer instruments. These instruments have been modified to extend the spectral range of spectral solar UV measurements from 286.5 to 363 nm in 0.5 nm steps. Instruments undergo an annual UV irradiance calibration (to take into account fluctuations and degradation of the optical components of the instrument), using a standard UV lamp traceable to a US National Institute of Standards and Technology (NIST) 1000 W UV lamp. In addition to these annual calibrations by the National Ultraviolet Monitoring Center (NUVMC), independent quality assurance audits of the instruments take place by staff of the National Oceanic and Atmospheric Administration (NOAA) to ensure accurate measurements.

2.1 Corrections to Brewer UV data

Careful UV measurements require a full understanding of instrument performance in all conditions. Subsequently, UV data used in this paper were corrected for dark count, dead time and stray light using the algorithms of Sci-Tec.[3] In addition, the UV data use an estimated daily instrument response based on an annual UV irradiance calibration, using a 1000W secondary standard lamp traceable to the NIST 1000 W lamp. The instrument response function is calculated for each day based on a linear interpolation between the two temporally closest response functions. The data are then corrected for the instrument's angular (cosine) response and temperature dependence. The cosine correction leads to an increase in the UV irradiance relative to that of the uncorrected data since the full sky collector operates at a reduced throughput for rays at large angles from zenith, the angle for which the instrument is calibrated. The temperature response function of each instrument in the EPA/UGA network has its own wavelength dependent characteristic temperature dependence of about 1% per degree centigrade.[4]

2.2 Biologically effective UV–vitamin D and erythema

The biologically effective solar UV, UV_D, can be assessed using the following equation:

$$UV_D = T \int_{UV} S(\lambda)\, A(\lambda)\, d\lambda \quad (1)$$

where $S(\lambda)$ is the solar spectral irradiance, $A(\lambda)$ is the action spectrum for human vitamin D production[1] and T is the exposure time interval. The measured irradiance is weighted according to the vitamin D biological action spectra. The vitamin D spectra is based on the conversion of 7-dehydrocholesterol to pre-vitamin D3 as measured described previously by Galkin and Terenetskaya.[1] For this study, four sites were utilized: Albuquerque, New Mexico (latitude = 35.09°, longitude = 106.29°, altitude = 1615 m); Boulder, Colorado (48.14°, 123.40°, 1689 m), Chicago, Illinois (41.79°, 87.60°, 156 m); Denali National Park, Alaska (63.73°, 148.97°, 839 m); Hawaii National Park, Hawaii (19.42°, 155.29°, 1243 m) and the US Virgin Islands (18.33°, 64.79°, 0 m).

3.0 Results

The collected solar noon data for the various sites in this study are shown in Fig. 1. The data presented are the solar noon

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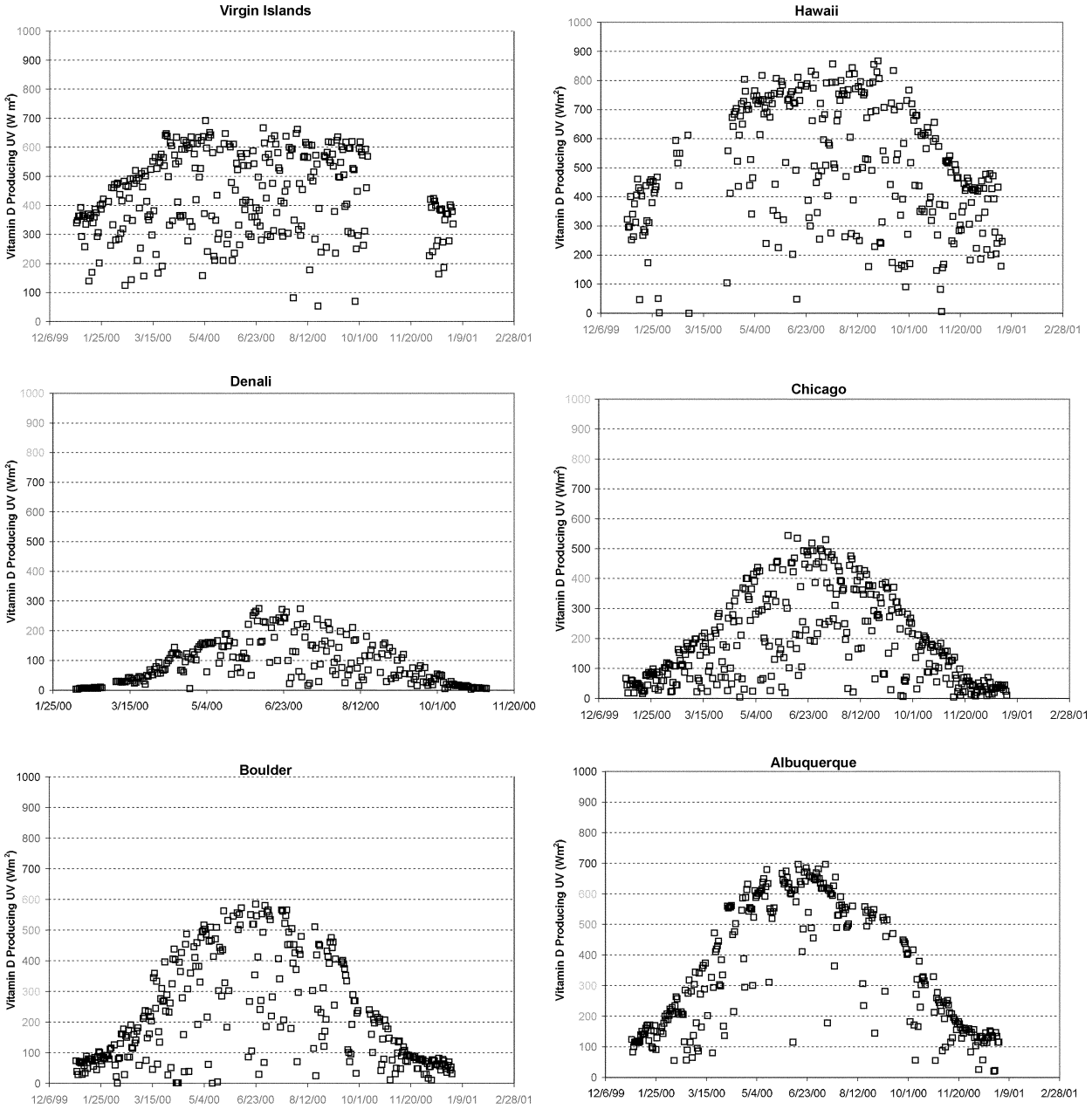

Fig. 1 'Solar noon vitamin D producing' UV at selected sites in the USA for the years 2000.

vitamin D producing UV irradiance. The solar noon time data were selected for presentation in this paper as it provides insights into the maximum potential for an individual to utilize the sun to produce dermal vitamin D. The limitations of such data presented include day to day variability of clouds and aerosols at scan time, however, they still provide a valuable insight into the trends in UV. The low latitude sites of the Virgin Islands and Hawaii have significantly higher vitamin D producing UV than the northern sites of Denali and Chicago.

Winter solar noon irradiance minimums at these locations are at times higher than the values recorded at the higher latitude sites in summer. However, the mid-latitude sites of Boulder and Albuquerque have considerable variation in the measured solar noon vitamin D irradiances between summer and winter. The average monthly vitamin D solar noon irradiances for the Boulder site for January and July were 70 and 414 W m^{-2}, respectively. Meanwhile, the Hawaii site recorded average monthly vitamin D solar noon irradiance of 343 and 605 W m^{-2} for January and July, respectively. The Boulder site had 83% lower vitamin D UV between July and January while the Hawaii site had only a 43% for the same time period. This is due to the sensitivity of the vitamin D action spectrum to the shorter UVB (280–320 nm) wavelengths. During winter at the more northern locales the larger solar angle during winter allows for more absorption and scattering of the shorter wavelengths, hence decreasing the UVB component of the solar UV spectrum, which is key for the development of pre-vitamin D.

These data, whilst useful to assess the environmental (ambient) levels of solar vitamin D UV and how it is distributed geographically, do not provide insight into what sections of the body are exposed to sunlight. Using a technique described in ref. 5, we estimated the vitamin D UV at various anatomical locations for the different geographical sites in this study. Fig. 2 shows the average monthly solar noon vitamin D producing UV for selected anatomical locations for each of the sites in this study. The exposure ratios were determined through a technique described in ref. 6, and the exposure ratios used for this research were those contained in that work. Briefly, the exposure ratio was calculated through the use of polysulfone dosimeters attached to selected anatomical locations over a manikin in an upright position. The manikin was exposed to clear sky conditions for a 10° SZA range increment from 0 to 90°, giving exposure

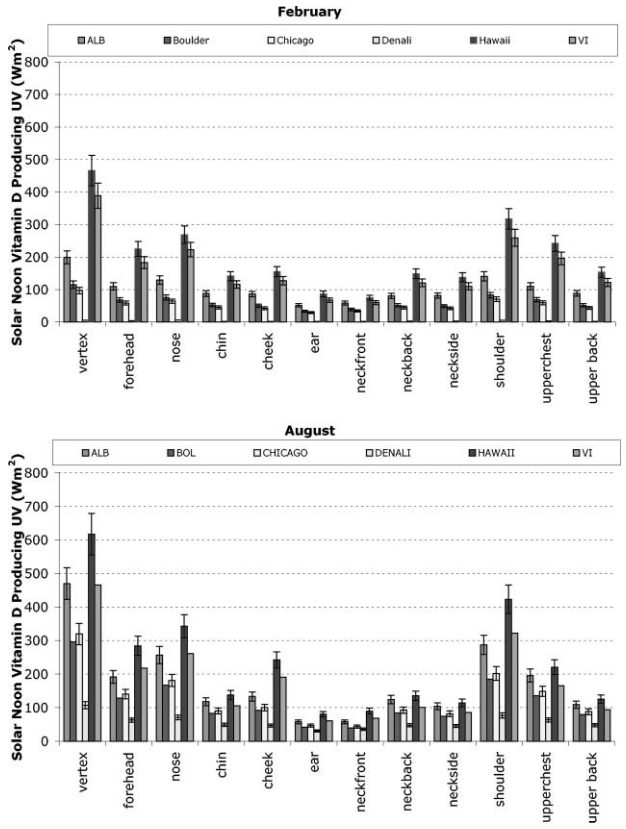

Fig. 2 Anatomical distribution of 'vitamin D producing' UV at selected US sites for the years 2000 for February and August.

ratios in 10° intervals. Ambient measurements of solar UV were also recorded at the same time the manikin was exposed, again with the use of polysulfone dosimeters. Data from the selected anatomical location were normalized to the ambient UV exposure to obtain an exposure ratio, presented as a fraction, of the ambient radiation for that particular 10° solar zenith angle range. We calculated the anatomical exposure of vitamin D through:

$$UV_{VitD} = AE_{VitD}{*}ER \quad (2)$$

where UV_{VitD} is the vitamin D UV exposure to a selected anatomical location, AE_{VitD} is the ambient vitamin D UV radiation and ER is the UV exposure ratio.

For each location in this study, the site with the highest vitamin D UV exposure is the vertex of the head. Other locations on the face received an exposure from 0.5 to 0.2 that of the vertex of the head, making their potential to produce dermal vitamin D less. Fig. 2 also compares the UV exposure to selected anatomical locations with respect to month of the years for February and August. Due to the changes in solar zenith angle during the course of the years, the anatomical distribution of UV changes, which can impact on the capability of the sun to produce dermal vitamin D.

Fig. 3 shows the ratio of the monthly average vitamin D UV exposure to the nose compared to the upper chest for each of the sites in this study. Depending on location and time of years, the ratio can change, but at all times, the exposure to the nose is higher than that of the upper chest. For the lower latitude sites of Hawaii and Virgin Islands, during the summer months (June, July, August), the nose received a higher exposure than for the same time period at the high latitude site of Denali. Fig. 4 shows the ratio of nose to upper chest plotted as a function of latitude of location for the months of January and July. We found that for July, the ratio of nose to upper chest decreased with an increase in latitude, indicating that during July, the distribution of vitamin D UV is associated with location, caused by the maximum solar

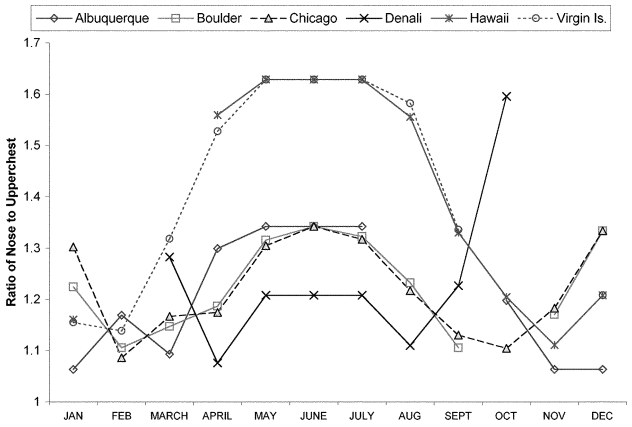

Fig. 3 Ratio of the 'vitamin D producing' UV to the nose to the upper chest for the years 2000.

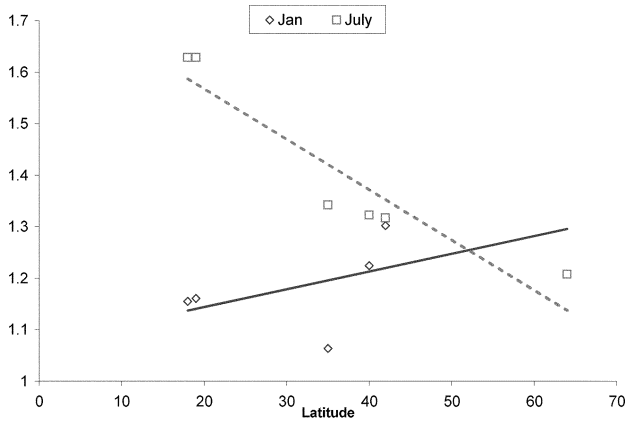

Fig. 4 Ratio of the 'vitamin D producing' UV to the nose to the upper chest as a function of location.

zenith angle reached during the day. At the lower latitudes, with the small solar zenith angle, the total length of the air-mass column that the radiation passes through is less, causing less scattering of the radiation compared with the high latitude site. This variation of the scattering of incoming radiation causes the distribution of vitamin D over the human form to vary.

A similar result is found in Fig. 5 and 6 where the ratio of the vitamin D UV exposure to the chin compared with the shoulder is plotted as a function of time of years. For all locations, a trend was noted for the ratio reaching a nadir during the summer months (June, July, Aug) with the apogee in the winter months (December, January).

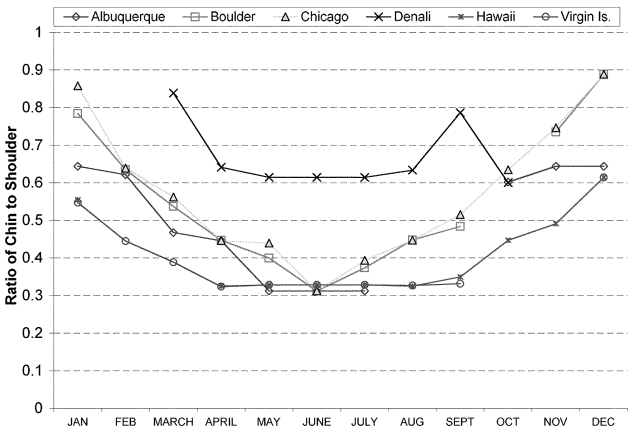

Fig. 5 Ratio of the 'vitamin D producing' UV to the chin to shoulder for the years 2000.

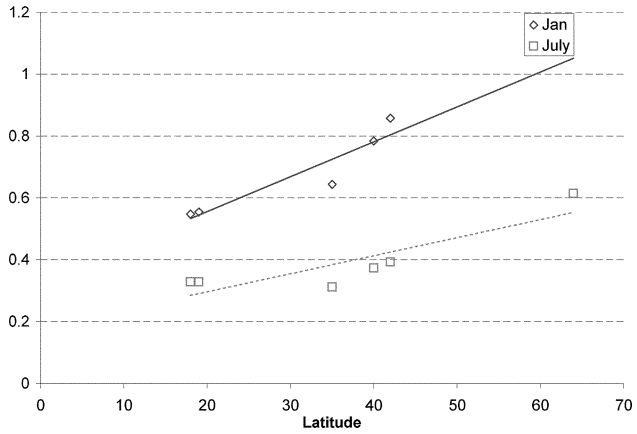

Fig. 6 Ratio of the 'vitamin D producing' UV to the chin to shoulder as a function of location.

Conclusions

The results presented in this paper indicate how human vitamin D UV exposure varies over the United States and support findings that the location of exposure influences human disease rates.[7] The data suggest that there is extreme sensitivity of the vitamin D action spectrum to the shorter UVB wavelengths. These wavelengths are highly sensitive to variability to ozone and solar zenith angle, which accounts for the variability with respect to location in the results presented in this paper. The results presented suggest that one needs to consider not only the ambient vitamin D UV irradiance (as measured on a horizontal plane), but consideration is needed on the anatomical distribution of exposure. From the results presented in this paper, we show that the anatomical distribution of UV radiation that synthesizes pre-vitamin D changes throughout the years and also with latitude. This is important due to clothing considerations (for amount of skin available for vitamin D synthesis) and the use of hats and sun-screens. The data presented in this paper suggest that careful personal vitamin D UV exposure measurements to assess the impact of latitude and time of years would help us better understand the exposure required for vitamin D synthesis.

Acknowledgements

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References

1 O. N. Galkin and I. P. Terenetskaya, Vitamin D biodosimeter: basic characteristics and potential applications, *J. Photochem. Photobiol. B: Biol.*, 1999, **53**, 12–19.
2 W. B. Grant, An estimate of premature cancer mortality in the U.S. due to inadequate doses of solar UV-B radiation, *Cancer*, 2002, **94**, 1867–1875.
3 *Brewer MKIV Spectrophotometer Operator's Manual*, OM-BA-C231 REV B, SCI-TEC Instruments Inc., Saskatoon, Canada, August 15, 1999.
4 E. Weatherhead, A. Theisen, J. Stevermer, B. Enagonio, P. Rabinovitch, P. Disterhoft, K. Lantz, R. Meltzer, J. Sabburg, J. DeLuisi, J. Rives and J. Shreffler, Temperature Dependence of the Brewer Ultraviolet Data, *J. Geophy. Res.*, 2001, **106**, 34121–34129.
5 M. G. Kimlin, A. V. Parisi and J. C. F. Wong, The facial distribution of erythemal ultraviolet exposure in south east Queensland, *Phys. Med. Biol.*, 1998, **43**, 231–240.
6 N. Downs, MSc Thesis, University of Southern Queensland, Toowoomba, Australia, 2002.
7 C. L. Hanchette and G. C. Schwartz, Geographic patterns of prostate cancer mortality, *Cancer*, 1992, **70**, 2861–2869.