# Association between sun exposure and serum 25-hydroxyvitamin D<sub>3</sub> levels

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Abstract. Low vitamin D levels are associated with increased risk of many diseases. However, there is uncertainty about the amount of sun exposure required to increase vitamin D. Personal UV dosimeters were used to quantify the association between sun exposure over 8-weeks, corrected for the proportion of skin not covered by clothing, and change in serum 25-hydroxyvitamin D<sub>3</sub> (25OHD<sub>3</sub>) concentration. There was a non-linear association between UV exposure and 25OHD<sub>3</sub>. This finding suggests that vitamin D status can be increased by regular small sun exposures and that greater exposures result in only small additional increases in 25OHD<sub>3</sub>.

## **Background**

There is increasing evidence from prospective epidemiological studies that low vitamin D status, as measured by blood 25-hydroxyvitamin D (25OHD) concentration, predicts a wide range of diseases (Scragg 2011). About 90% of vitamin D in humans is synthesised through sun exposure. A better understanding of the relationship between sun exposure and vitamin D status is required so that sun exposure policies can be developed that result in increased vitamin D levels without also increasing risk of skin cancer.

Two approaches have been used to quantify the association between sun exposure and vitamin D status. These have been either: a) solaria studies of change in 25OHD level from measured doses of artificial UV-B; or b) observational studies comparing 25OHD levels with recent UV exposure. Most of the latter have measured UV exposure with questionnaires, complemented by measures of ambient UV exposure. Few have used objective measures of UV exposure (such as polysulphone badges or electronic dosimeters) and only two have quantified the association between UV exposure and 25OHD status (Datta et al, 2012; Kimlin et al, 2014). The aim of the current study is to examine, in a large multi-ethnic community sample, the association between natural outdoor UV exposure and change in serum 25OHD levels over 4-weeks using electronic UV dosimeters.

### Methods

The participants in this report comprise 503 volunteers recruited from the community in Auckland (n=332) and Dunedin (n=171), equally distributed across the adult ages of 18-85 years and the four main ethnic groups (European 126, Maori 126, Pacific 123 and Asian 128). Baseline interviews were carried out during February-October in 2008 and 2009, in which the following information was collected: self-defined ethnicity, self-reported reaction to sun exposure (Fitzpatrick scale), weight and height to estimate body mass index and body surface area, and skin colour measured by a portable spectrophotometer. At this

visit, participants were given a personal electronic dosimeter to wear on the outer wrist when outdoors and a daily diary to record their main clothing during following three day-time periods: before 11am, 11am-4pm, and after 4pm. The diary was used to estimate the proportion of their skin exposed to UV radiation in each time period. Participants returned weekly for 8-weeks for the dosimeter data to be downloaded and clothing diary to be checked and replaced. Blood samples were collected at the end of week 4 and week 8, to measure serum 25OHD<sub>3</sub> concentrations using Liquid Chromatography-Tandem Mass Spectrometry.

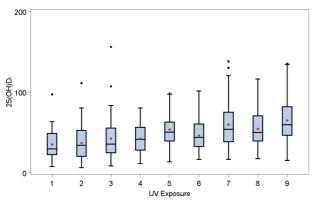
The dosimeters sampled erythemally-weighted UV irradiance at 8-second intervals (Allen & McKenzie 2005). These data were aggregated for each of the three daily time periods (above) and converted to standard erythemal doses (SED), which were corrected for clothing to generate equivalent full-body exposures, SED<sub>EFB</sub>. Data were analysed using a multivariate linear mixed model with an autoregressive structure for repeated measures to estimate simultaneously both between-person and within-person effects (Neuhaus & Kalbfleisch 1998). Full details of methods and results have been reported (Scragg et al, 2014).

#### Results

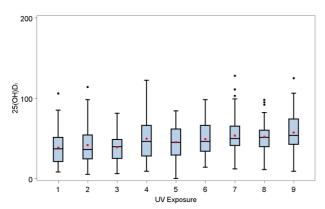
Median SED of UV radiation received was 7.3 in weeks 1-4 and 6.5 in weeks 5-8, less than 2% of ambient UV radiation. When converted to SED<sub>EFB</sub> per week, this was 0.33 in weeks 1-4 and 0.34 in weeks 5-8. Mean (SD) 25OHD3 concentration was 48.2 (24.2) nmol/L at the end of week 4and 47.4 (22.4) nmol/L at the end of week 8.

Participants were ranked into nine groups by increasing cumulative  $SED_{EFB}$  per week during each 4-week period, and mean  $25(OH)D_3$  concentrations at the end of each 4-week period were calculated for each group (Figures 1 & 2). The variation in mean  $25(OH)D_3$  from the lowest to the highest SED group for each 4-week period was substantial, being from 34.9 up to 64.7 nmol/L for week-4 and 38.4 to 57.3 nmol/L for week-8. This represents the between-person variation in  $25(OH)D_3$  concentration.

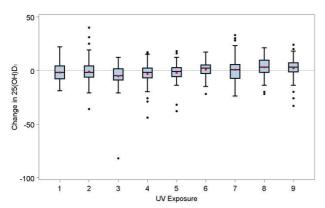
The change in 25(OH)D<sub>3</sub> from week-4 to week-8 for each participant was calculated, and then ranked by increasing SED<sub>EFB</sub> per week received during weeks 5-8 (Figure 3). This change, which represents the within-person variation in vitamin D status, was much smaller than the between-person variation, and varied from a mean change of -5.4 nmol/L up to 2.1 nmol/L across the range of grouped SED<sub>EFB</sub> exposures in weeks 5-8. UV exposures < 0.4 SED<sub>EFB</sub> per week (groups 1-5) were associated with decreasing 25OHD<sub>3</sub> concentrations in weeks 5-8, from weeks 1-4.



**Figure 1.** Boxplots showing the distribution of 25-hydroxyvitamin  $D_3$  (nmol/L) at end of week 4, by increasing rank of cumulative UV SED<sub>EFB</sub> exposure during preceding 4 weeks (weeks 1-4). Dot = mean, lines are 25<sup>th</sup>, 50% and 75% percentiles, "whiskers" are lowest and highest values within 1.5 \* inter-quartile range.



**Figure 2.** Boxplots showing the distribution of 25-hydroxyvitamin  $D_3$  (nmol/L) at end of week 8, by increasing rank of cumulative UV SED<sub>EFB</sub> exposure during preceding 4 weeks (weeks 5-8). See Figure 1 legend for explanation of box-plot.



**Figure 3.** Change in 25-hydroxyvitamin  $D_3$  (nmol/L) from week 4 to week 8, by increasing rank of cumulative UV SED<sub>EFB</sub> exposure during weeks 5-8. See Figure 1 legend for explanation of box-plot.

Statistical analyses identified a non-linear association between SED<sub>EFB</sub> exposure and 25(OH)D<sub>3</sub> concentration over the 8-week period. Using the multivariate mixed model described above, the coefficient for the betweenperson variation showed that average exposures of 0.1, 1.0 and 10 SED<sub>EFB</sub> per week were associated with 25(OH)D<sub>3</sub> levels that were greater by 10.9, 21.9 and 32.8 nmol/L, respectively, than an average exposure of 0.01 SED<sub>EFB</sub> per week (ie. almost zero), adjusting for age, sex, ethnicity, body mass index, skin colour, number of days of UV exposure and within-person variation in UV exposure. A graph of this coefficient showed that most of the increase in 25(OH)D<sub>3</sub> associated with between-person variation in UV exposure occurred at exposures of < 2 SED<sub>EFB</sub> per week, and that greater exposures were associated with only small additional increases in 25(OH)D<sub>3</sub> concentration.

#### Conclusions

Our results indicate a non-linear association between sun exposure and serum vitamin D concentration which suggest that vitamin D status can be increased by regular small sun exposures (< 2 SED<sub>EFB</sub> per week), and that greater exposures result in only small additional increases in  $25(OH)D_3$ .

### References

Allen, M., McKenzie, R. 2005. Enhanced UV exposure on a ski-field compared with exposures at sea level. Photochemical & Photobiological Sciences 4(5): 429-437.

Datta. P., Bogh, M.K., Olsen, P., et al. 2012. Increase in serum 25-hydroxyvitamin-D3 in humans after solar exposure under natural conditions compared to artificial UVB exposure of hands and face. Photochemical & Photobiological Sciences 11(12):1817-1824.

Kimlin, M.G., Lucas, R.M., Harrison, S.L., et al. 2014. The Contributions of Solar Ultraviolet Radiation Exposure and Other Determinants to Serum 25-Hydroxyvitamin D Concentrations in Australian Adults: The AusD Study. American Journal of Epidemiology 179(7):864-874.

Neuhaus, J.M., Kalbfleisch, J.D. 1998. Between- and within-cluster covariate effects in the analysis of clustered data. Biometrics 54(2):638-645.

Scragg, R. 2011. Vitamin D and public health: an overview of recent research on common diseases and mortality in adulthood. Public Health Nutrition 14(9):1515-1532.

Scragg, R.K.R., Stewart, A.W., McKenzie, R.L., Reeder, A.I., Liley, J.B. and Allen, M.W. Sun exposure and 25-hyrdoxyvitamin D3 levels in a community sample: quantifying the association with electronic dosimeters (submitted).