Vitamin D and its association with personal UV exposure.

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Abstract. In this study we relate levels of Vitamin D with personal UV exposures in two centres from measurements taken over an 8 week period for each participant. The results presented here are an assessment of analytical methods and a demonstration of these methods using preliminary partial data. Depending on assumptions the dose response measure differs so we must be clear what relationship we are interest in and which analytical model gives this estimate.

Introduction

Low levels of vitamin D have been implicated in a wide variety of health issues including rickets, osteomalacia, osteoarthritis, many forms of cancer (eg. colon, breast), multiple sclerosis, diabetes, hypertension, coronary heart disease and reduced lung function. New Zealanders have low vitamin D levels compared to other developed countries, with the mean level in adult New Zealanders being 50 nmol/L [*Rockell et al, 2006*]. A significant proportion have sub-optimal levels of vitamin D, particularly Pacific, Maori and South Asian people.

The primary source of vitamin D is sun exposure; and the amount of vitamin D produced from sun exposure varies with age, skin pigmentation, time of year and day, and latitude. However, there is limited information on how much sun exposure is needed to maintain vitamin D at the optimum levels required for good health. The time required outdoor in the sun to receive 1/6 to 1/3 of one MED, in order to produce between 1500-3750 IU of vitamin D, has been estimated for people with white (Fitzpatrick type II) skin living in the main Australian cities [*Samenek, et al, 2006*]. There is no indication from this study of the serum $25OHD_3$ levels that would be achieved by the above sun exposures in people with varying skin colours.

The aim of the current study is to determine the association between sun exposure and changes in serum $25OHD_3$, and how this varies with age, skin pigmentation, latitude and season. At the time of the workshop, preliminary data were available from only the about half of the total number of people surveyed, for presentation in this report.

Study Design

The study related UV exposure to vitamin D level measured twice, once after 4 weeks and again after another 4 weeks of UV exposure measurement. The potential UV exposure was measured by a dosimeter worn like a watch on the wrist, above any clothes. The measure of exposure used in these analyses is this recorded measure which is then multiplied by the proportion of skin exposed. Each participant, for the 8 weeks of the study, recorded the clothes they were wearing for each of three periods each day (up to 11am, 11am to 4pm and after 4pm). The cumulative UV exposure in each of these periods was multiplied by the proportion of the skin exposed and averaged over the 4 weeks to give a measure with the units of SED/day. To obtain a wide range of UV exposures the study was conducted over a wide seasonal range (February to November) and in two centres with quite different latitudes.

In 2008, 250 participants recruited from work places and the community, aged 18 to 81 years, from a wide-range of ethnicities (Maori, Pacific, Asian and European) were enrolled into the study, (164 in Auckland (36° 52´S) and 86 in Dunedin (45° 50´S)) and 239 have complete data which has been used for demonstration analyses in this presentation. The observation time for these participants is evenly spread over the year except for summer where there were very few observations.

Simple linear regression and linear mixed models have been considered as possible analysis techniques. The simple association of UV exposure over 4 weeks with the level of vitamin D at the end of that period is a simple possibility. This model needs all confounding variables to be included to get the appropriate assessment of UV exposure. To reduce the effect of subject variation the change in vitamin D level from the 4 week to 8 week measure can be associated with UV exposure. To use all the collected data a more complex model, a linear mixed model can be used. This model has two UV exposure and 2 vitamin D measures for each individual. The UV exposure data is modelled as fixed effects in the model and the subject variation is modelled by including subject as a random effect. The UV exposure can be modelled as the observed exposure during each of the 4 week intervals or it could be modelled with the two variables, the UV exposure mean for each subject and the deviation of the observed exposure from this mean [*Neuhaus and Kalbfleisch*, 1998]. This enables examination of the within and between subject effects.

Results

As expected, both UV exposure and vitamin D levels vary by time of year (Figure 1).

In the winter months there is very little UV exposure as recorded by the participants of this study and so very little variation. The vitamin D level also drops during the winter months and for 2 months none of the participants had a vitamin D level above 75 nmol/L.

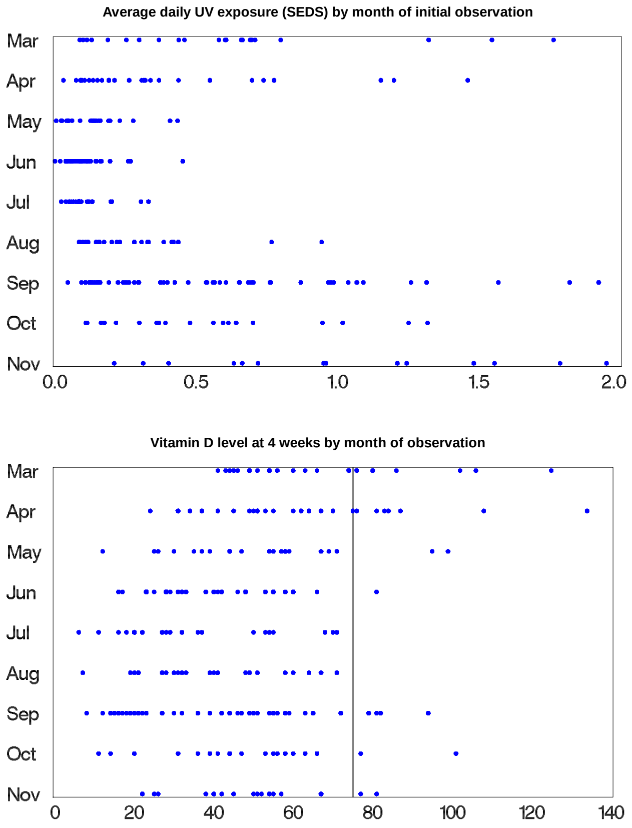

Figure 1. Upper panel: The average daily exposure (SED/day) by month of the year of initial observation. Lower panel: The vitamin D (nmol/L) reading after 4 weeks in the study by month of the year of initial observation.

The simple regression assessing the 8 week vitamin D level shows a strong relationship with UV exposure but this is strongly influenced by individual characteristics. To obtain a satisfactory estimate of effect all the confounding variables need to be included in the model. The assessment with change in vitamin D showed a positive but small relationship, for an exposure increase of 1 SED/day the vitamin D change between measurements would by 6.9 (3.5). Because we are assessing a within subject measure the effect of between subject differences is removed. The mixed model has the two terms, mean UV exposure and the deviation of the observed UV exposure from the mean. In this case, their being only two observations, one deviation is the negative of the other. As the mixed model includes the random subject effect and the mean exposure level is included as a fixed effect this leaves the deviation to describe the effect of a within person UV exposure. In the demonstration data this effect is 5.9 (4.4) (Figure 2). The coefficient of the mean effect is 37 (7) which reduces to 24 (6) when a number of subject specific covariates (ethnicity, skin colour, BMI and time of year) are included in the model.

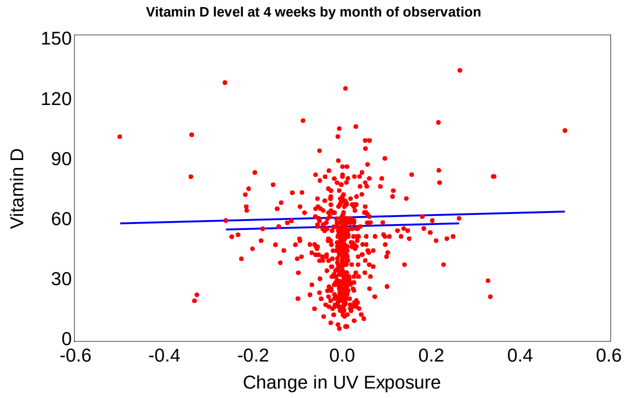

Figure 3. The predicted regression line (Auckland – upper and Dunedin – lower) associated with the deviation from the mean vitamin D level. The slope of this line is 5.9 (4.4) SED/day. The points are the observed deviations. These points are similar to but not quite a reflection about 0 as they come from the measured vitamin D values at 4 and 8 weeks of observation.

Discussion

Models that remove subject effects by use of confounder variables show a much greater association between UV exposure and vitamin D levels. This is probably because we have recorded only some of the confounder variables and those we have are not accurately measured. The models that remove the subject effects by use of the vitamin D levels themselves show a lower level of association and are reasonable consistent. However, it could be that there is a different effect of UV exposure between individuals compared with that between individuals. We need to ensure that we use an appropriate model, one that removes the subject effect, in our reporting of the results from this study.

References

Neuhaus, J.M., and J.D. Kalbfleisch, Between- and within-cluster covariate effects in the analysis of clustered data, Biometrics, 58 (2), 638-645, 1998.

Rockell J.E., C.M. Skeaff, S.M. Williams, T.J. Green, Serum 25-hydroxyvitamin D concentrations of New Zealanders aged 15 years and older, Osteoporos Int 17 (9), 1382-9, 2006.

Samanek A.J., E.J. Croager, P. Gies, E. Milne, R. Prince, A.J. McMichael, R.M. Lucas and T. Slevin, Estimates of beneficial and harmful sun exposure times during the year for major Australian population centres. Med J Aust 184 (7), 338-41, 2006.

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