

Vitamin D Level in Summer and Winter Related to Measured UVR Exposure and Behavior

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Received 1 April 2009, accepted 22 June 2009, DOI: 10.1111/j.1751-1097.2009.00612.x

ABSTRACT

The influence of the summer UVR exposure on serum-25-hydroxyvitamin D (25(OH)D) in late summer and winter was investigated in an open study on 25 healthy, adult volunteers. The UVR exposure dose in standard erythema dose (SED) was monitored continuously during a summer season with personal, electronic wristwatch UVR dosimeters and sun exposure diaries. Constitutive and facultative skin pigmentation was measured in September. 25(OH)D was measured in September and February and was in mean $82 \text{ nmol/L} \pm 25$ (mean \pm SD) in September and $56 \text{ nmol/L} \pm 19$ (mean \pm SD) in February. The received cumulative UVR dose measured during a mean of 121 days was $156 \text{ SED} \pm 159$ (mean \pm SD). The following UVR exposure parameters correlated with 25(OH)D in September and February, respectively: (1) The cumulative UVR dose ($r = 0.53$; $P < 0.01$) and ($r = 0.43$; $P = 0.03$); (2) Mean daily hours with UVR measurements monitored by the dosimeter ($r = 0.64$, $P = 0.001$) and ($r = 0.53$; $P = 0.007$); (3) Days “with sun-exposed upper body” ($r = 0.58$, $P = 0.003$) and ($r = 0.50$; $P = 0.01$); (4) Facultative pigmentation ($r = 0.47$; $P < 0.02$) and ($r = 0.7$; $P < 0.001$); (5) Constitutive pigmentation ($r = 0.06$, n.s.) and ($r = 0.43$, $P = 0.03$). Neither days “sunbathing” nor days with “sunscreen applied” correlated with 25(OH)D. The fall in 25(OH)D during winter was dependent on the entry value.

INTRODUCTION

In addition to its well-known influence on the calcium metabolism, vitamin D insufficiency has in recent years been connected to other diseases such as cancer, diabetes and multiple sclerosis (1–3). Solar radiation is the natural source for vitamin D synthesis in the skin. In the summer half year in Denmark, 56°N, 10–20 min exposure of the arms, hands and face two to three times a week is said to be sufficient to maintain a sufficient vitamin D blood level in summer (4). However, from October to April the solar UV radiation (UVR) is insufficient to maintain the D vitamin level and the climate too cold to expose the naked skin (4–6). Disregarding a possible increased skin cancer risk it has been proposed to recommend unprotected solar exposure in the middle of the day to increase the vitamin D levels (4). To be able to better advise the public there has therefore been a growing interest in

finding the correlation between UVR exposure dose, time and skin area exposed and the resulting vitamin D level.

Most information about cutaneous vitamin D production after UVR exposure has been based on retrospective interviews about UVR exposure or theoretical models (7,8). However, these studies do not actually monitor the individual sun exposure dose, time and behavior while outdoors (7,9). The serum concentration of 25-hydroxyvitamin D (25(OH)D) is considered the best measure of the total vitamin D status of an individual. We have therefore chosen to assess 25(OH)D in September on volunteers that have worn a personal, electronic UVR dosimeter in a wristwatch (10) for the summer season from May to September and reported corresponding sun behavior information in a diary. Our aim was to investigate if 25(OH)D in September and the following February were influenced by (1) UVR dose received in the spring and summer months, (2) number of days sunbathing with the intention to tan and with sun-exposed upper body, (3) skin pigmentation, constitutive and facultative (tanning) in a relatively homogenous white population (Fitzpatrick skin type II–IV) (11).

MATERIALS AND METHODS

Participants and procedure. The study took place in Copenhagen vicinity, Denmark, 56°N in May 2006 to March 2007. Twenty-five healthy, Caucasian volunteers of Danish ancestry with a mean age of 52 years (range 32–71 years) (male 12 and female 13) were recruited. The group was selected to comprise indoor workers with known low, middle or high UVR exposure and gardeners representing outdoor workers. The subjects wore a personal electronic UVR dosimeter in the form of a wristwatch and completed sun exposure diaries in a mean of 121 days (10,12,13). To be included in the data analysis, the volunteers should have (1) more than 30 days with both UVR dosimeter readings and corresponding diary data of which 21 days or more had to be in June, July or August and (2) vitamin D intake and number of fish meals were registered and no supplementary vitamin D ingestion above the level in a multivitamin tablet ($10 \mu\text{g}$ per day) was allowed. The Ethical Committees for Copenhagen and Frederiksberg approved the study KF 11 320779, which was conducted according to the Declaration of Helsinki.

Skin type and pigmentation (PPF). Self-reported skin type according to Fitzpatrick (11) was registered as follows: Skin Type II, 4; Skin Type III, 18; and Skin Type IV, 3. The pigmentation expressed as pigment protection factor (PPF) was measured by a skin reflectance meter (UV-Optimize, Skin Type, Chromo-light, Denmark) (14,15) in September on the same day as a blood sample for 25(OH)D assessment was drawn. PPF on the buttock was used as a measure of the constitutive skin pigmentation and PPF on the shoulder as a measure of the achieved facultative pigmentation on a body position sun exposed only when a major part of the body is uncovered, i.e. during sunbathing.

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Efficacy measures. Blood samples for assessment of biomarkers were obtained twice from each subject at baseline after the summer season in September and in February the following year. Serum concentrations were assessed of (1) 25(OH)D, (2) parathyroid hormone (PTH), (3) ionized calcium, Ca²⁺ and (4) alkaline phosphatase (AP). The primary outcome was to identify possible differences in serum levels of 25(OH)D in relation to UVR exposure parameters in the preceding summer.

Biochemical analyses. 25(OH)D was measured by a commercially available radioimmunoassay kit (IDS Immunodiagnosics, Boldon, UK). PTH was measured by a chemiluminescence immunometric assay (Immulate 2500 biochemistry analyzer; Diagnostic Products Corporation, Los Angeles, CA). Ca²⁺ was measured in whole-blood on an ABL700 (Radiometer a/s, Denmark) using an E733 ion selective electrode. AP was measured on a VITROS Vitros 5.1 FS (Ortho Clinical Diagnostics Inc.) using VITROS ALKP slides and VITROS calibrator kit 3. All analyses have earlier been described in detail (16).

Statistics. The distribution of 25(OH)D, PTH, Ca²⁺ and AP did not deviate significantly from normal distributions at baseline, thus permitting the use of parametric statistics on untransformed values. The paired *t*-test was used to test differences in biomarker levels in September compared with the end of study in February while the Pearson correlation test was used to test a possible correlation between the September and February level. The Pearson correlation test was also used to compare the UVR exposure parameters and the biomarkers in September and February including the seasonal change in biomarkers (Δ serum concentration) defined as September values minus February values. In each case, $P < 0.05$ was considered significant. Multiple regression models were used to determine the effect of the different significant UV exposure parameters on 25(OH)D. We used SPSS for Windows 13 (SPSS Inc, Chicago, IL) for data analysis.

RESULTS

The serum concentrations of 25(OH)D, PTH, Ca²⁺ and AP measured in September and February the following year are shown in Table 1. Significant differences and correlations were found between the serum concentrations measured in September compared with February for each of the biomarkers. However, there was no inter correlation between 25(OH)D and PTH or the other biomarkers nor between the Δ -biomarkers (defined as the difference in serum concentration of a biomarker from September to February): Δ 25(OH)D, Δ PTH, Δ Ca²⁺ and Δ AP. Only Δ 25(OH)D did correlate with the entry value in September ($r = 0.68$, $P < 0.001$). None of the Δ -biomarkers was related to the exit values in February or to each other.

A serum concentration of 25(OH)D above 50 nmol/L is considered sufficient, while a level between 25 and 50 nmol/L is considered insufficient and a level below 25 nmol/L as deficient. Except for one person in September (32 nmol/L) and eight in February (range, 26–48 nmol/L) the subjects had 25(OH)D levels above 50 nmol/L. None of the participants thus

experienced vitamin D deficiency during the study. The PTH levels were within the normal range for all in September and for all but one in February (9 pmol/L). All subjects had normal Ca²⁺ and AP levels.

Table 2 shows the sun exposure parameters measured in the spring and summer preceding blood sampling. Table 3 shows the correlations between these UVR exposure parameters and the 25(OH)D in September and February.

Dosimeter-monitored UVR exposure dose and time and diary-reported behavior

UVR exposure dose and hours monitored by the dosimeter correlated significantly with 25(OH)D in September as well as

Table 2. Distribution of sun exposure parameters in a summer season of mean 121 days ($n = 25$)

	Mean	SD	Min	Max
(1) UVR doses and hours				
Cumulated measured UVR dose (SED)	156	159	18	790
Estimated annual UVR dose (SED)	232	260	25	1337
Mean UVR dose per day (SED)	1.4	1.5	0.15	8.6
Mean UVR dose 1200–1500 h (SED)	0.6	0.8	0.05	4.2
Mean hours outdoor per day*	2.9	1.6	0.3	6.5
(2) UVR exposure behavior				
Number of days with sun-exposed shoulders	24	20	0	75
Number of days sunbathing "to get a tan"	11	13	0	44
Number of days with sunburn	2.6	4	0	14
Number of days with sunscreen use	9.8	13.7	0	50
(3) PPF (September)				
Facultative pigmentation (tanned skin), shoulder	9.0	2.4	4.5	13.1
Constitutive pigmentation, buttock	4.5	1.4	2.8	8.6

SED = standard erythema dose; PPF = pigment protection factor.

*Hours with UVR measurements monitored by the dosimeter.

(1) Electronic, dosimeter monitored UVR dose and hours; (2) Diary-reported behavior; and (3) Constitutive and facultative pigmentation (tanned skin) expressed as PPF on the buttock and shoulder in September.

Table 1. Serum concentrations, Mean (SD), of 25(OH)D, PTH, Ca²⁺ and AP in September and the following February ($n = 25$).

	September	February	Paired <i>t</i> -test	Pearson's correlation September vs February	
	Mean (SD)	Mean (SD)	<i>P</i>	<i>r</i>	<i>P</i>
25(OH)D (nmol/L)	82.2 (25)	56.4 (19)	<0.001	0.52	0.007
PTH (pmol/L)	3.7 (1.4)	4.4 (1.9)	0.012	0.71	<0.001
Ca ²⁺ (mM)	1.25 (0.03)	1.24 (0.03)	0.018	0.77	<0.001
AP (U/L)	64 (14)	72 (18)	0.021	0.53	0.006

AP = alkaline phosphatase; PTH = parathyroid hormone.

Paired *t*-test showed the differences in September and February levels. The Pearson's correlation test showed that the February levels were dependent on the entry values in September.

Table 3. Pearson correlations (*r*) and level of significance (*P*) between 25(OH)D in September and February and UVR exposure parameters in a summer season (*n* = 25).

UVR exposure parameters	25(OH)D (nmol/L)			
	September		February	
	<i>r</i>	<i>P</i>	<i>r</i>	<i>P</i>
(1) UVR doses and hours				
Cumulated measured UVR dose (SED)	0.53	0.006	0.43	0.03
Estimated annual UVR dose (SED)	0.50	0.011	0.46	0.02
Mean UVR dose per day (SED)	0.49	0.014	0.43	0.03
Mean UVR dose 1200–1500 h (SED)	0.47	0.02	0.44	0.03
Mean hours outdoor per day*	0.64	0.001	0.53	0.007
(2) UVR exposure behavior				
Days with sun-exposed shoulders	0.58	0.003	0.50	0.011
Days sunbathing	0.35	0.09 n.s.	0.30	0.14 n.s.
Days with sunburn	–0.003	0.99 n.s.	–0.21	0.31 n.s.
Days with sunscreen applied	0.34	0.1 n.s.	–0.031	0.9 n.s.
(3) PPF (September)				
Facultative pigmentation (tanned skin), shoulder	0.47	0.02	0.70	<0.001
Constitutive pigmentation, buttock	0.06	0.77 n.s.	0.43	0.03

SED = standard erythema dose; PPF = pigment protection factor.

*Hours with UVR measurements monitored by the dosimeter.

(1) Dosimeter-monitored UVR dose and hours; (2) Diary-reported behavior; (3) Constitutive and facultative pigmentation (tanned skin) expressed as PPF on the buttock and shoulder in September.

in February. The Pearson correlations were of the same order ($r = 0.49$ – 0.63 , $P = 0.001$ – 0.02 in September) and ($r = 0.43$ – 0.53 , $P = 0.01$ – 0.03 in February). The correlations were strongest between 25(OH)D and “the mean daily hours outdoors per day” (defined as hours with UVR measurements monitored by the dosimeter). This indicates that 25(OH)D depends more on mean daily exposure time in the preceding summer than the size of the daily UVR dose received, in spite of the exposed area. “Days with exposed upper body/shoulders outdoors” correlated also significantly with the 25(OH)D in September and February. Excessive UVR exposure measured as numbers of days: (1) “sunbathing to get a tan,” (2) “with sunburn” or (3) “with sunscreen applied” did not correlate to 25(OH)D. None of the UVR exposure parameters correlated with PTH, Ca^{2+} , AP nor with Δ 25(OH)D or the other Δ -biomarkers.

Pigment protection factor

The constitutive skin pigmentation (PPF, on the buttock) did not correlate with 25(OH)D in September but was positively correlated in February ($r = 0.43$, $P = 0.03$). The facultative skin pigmentation (PPF on the shoulder) correlated positively with 25(OH)D in September ($r = 0.47$, $P = 0.02$) and even better in February ($r = 0.70$, $P < 0.001$). When adjusted for constitutive pigmentation (PPF on shoulder minus PPF on buttock) the correlation with 25(OH)D was still significant in September ($r = 0.45$, $P = 0.02$) as well as in February

($r = 0.46$, $P = 0.02$). In addition, the facultative pigmentation correlated significantly with the mean hours outdoor per day ($r = 0.65$, $P < 0.001$).

In addition, no significant correlations were found between 25(OH)D and sex, age, number of fish meals or oral vitamin D supplements, which according to the inclusion criteria should be less than 10 μ g per day.

Multiple regression models were used to determine the effect of all the significant UV parameters measured during the summer on 25(OH)D in September and the following February, and only “the mean daily hours outdoors per day” (defined as hours with UVR measurements monitored by the dosimeter) was a significant predictor in September and February. If the constitutive and facultative pigmentation assessed in September are included in the multiple regression models as well, “the mean daily hours outdoors per day” was a significant predictor in September while the facultative pigmentation was the only significant predictor of 25(OH)D in February.

DISCUSSION

Most information about the influence of UVR exposure dose on vitamin D production has been based on models where UVR exposure has been calculated from ambient UVR measurements and self-reported information about time spent outdoors. However, these questionnaires and models do not assess objectively the UVR dose and time outdoors (7–9,17). We have earlier documented that different behavior resulted in huge differences in UVR doses among individuals (12,13,18). To be able to quantify cutaneous vitamin D production it is thus important to relate the individual vitamin D level to measured individual UVR exposure. In the actual study, the subjects have worn a personal, electronic wristwatch UVR dosimeter and reported sun exposure behavior in a diary from May to September (10). 25(OH)D was assessed in September just after the summer season, when the vitamin D level is expected to have stabilized close to the yearly maximum and 25(OH)D was assessed again in February, close to the yearly minimum. This allow us to investigate how different sun exposure parameters influence vitamin D level just after the summer and the following winter in a climate as the Danish, 56°N, where little or no solar-induced cutaneous vitamin D production takes place from October to April. In addition, none of the participants were on holiday to sunny places from September to February, which might have had implications for the winter 25(OH)D.

Vitamin D, UVR dose and exposure time

The major findings from this study were that the maximal 25(OH)D in late summer as well as the minimal 25(OH)D in late winter was dependent on the UVR exposure dose received the preceding summer. The Pearson’s correlation coefficients were at the same level whether we compared 25(OH)D with the cumulative UVR dose, the estimated annual dose, the mean UVR dose per day, the mean UVR dose between 1200–1500 h or the mean daily hours with UVR measurements on the dosimeter. As 25(OH)D is a little better correlated to the daily UVR exposure time compared with UVR dose further studies are needed to find the lowest UVR dose for optimal vitamin D

production. The $\Delta 25(\text{OH})\text{D}$, representing the decrease in vitamin D level during autumn and winter was not correlated to any of the UVR behavioral parameters, however, positively related to the maximum $25(\text{OH})\text{D}$ level measured in September. This indicates that subjects who had a high vitamin D level in September lose more vitamin D during the winter compared with people with a low vitamin D level in September.

Examining a possible linearity between $25(\text{OH})\text{D}$ and UVR dose and exposed skin area

The assumption that the vitamin D synthesis is linear with respect to UVR exposure dose and body area exposed has been generally believed but has not been validated (4,8,19,20). In an earlier study using sunbeds as UVR source we have not been able to verify the linearity in dose–response, as a five-fold increase in UVR dose only led to a two-fold increase in vitamin D level (16). Recent but not yet published data from our group point at a linear dose–response when small skin areas are exposed while the response is more complex when larger skin areas are exposed as during sunbathing or full body sunbed exposure where a maximal effect is obtained after few minutes' exposure. It is very difficult to evaluate the influence of "exposed body area" in long-term real life situations, as continuous monitoring of exposed skin area is almost impossible. To get an impression of the time spent with a large area of the body UVR exposed, the subjects in the actual study reported the number of days with the upper body sun exposed with the purpose to get a tan or during other outdoor activities such as gardening. Days with "upper body exposed" did correlate significantly with $25(\text{OH})\text{D}$, while "days sunbathing to get a tan" did not. This indicated a positive relation between $25(\text{OH})\text{D}$ and the skin area exposed, but on the other hand showed that excessive sun exposure, such as during sunbathing, did not give rise to further vitamin D production. This is in line with earlier findings that cutaneous vitamin D production reaches a plateau after only 15–30 min of UVB exposure, thereafter only lumisterol and tachysterol and other vitamin D-inert substances are produced (21). To try to verify that, we excluded days with high UVR exposure above 5 standard erythema dose from the analyses, which led to a minor increase in the correlation coefficients, but the results are too uncertain to make conclusions.

Use of sunscreen did not affect the vitamin D level

Concern has been expressed about inhibition by sunscreen of vitamin D synthesis in humans (22). In this highly cited study of 20 long-term regular sunscreen users and 20 controls a lower $25(\text{OH})\text{D}$ level was found among the sunscreen users. However, regular use of sunscreen was determined retrospectively and in addition only a single blood sample was taken not allowing for comparison of changes in $25(\text{OH})\text{D}$ level between sunscreen users and controls. In contrast hereto an Australian group conducted a randomized, double-blind controlled study of the daily use of a broad spectrum sunscreen (sun protection factor 17) vs placebo cream over a summer period in Australia in 113 people. The mean change in the level of $25(\text{OH})\text{D}$ was significant but similar among sunscreen users and controls showing that regular sunscreen use did not influence vitamin D level (23). As the Australians, we did not find any relation

between $25(\text{OH})\text{D}$ and number of "days with sunscreen applied." The reason is probably that vitamin D can be synthesized even through sunscreen and the fact that sunscreen is not applied systematically on all the exposed body area or in the recommended dose all the time. Another explanation could be that people who often apply sunscreen also have a UVR exposure above average, which may compensate for a probable decrease in cutaneous skin synthesis of vitamin D caused by sunscreen (24). In the actual study we do not find any correlation between UV exposure dose and "days with sunscreen applied." An explanation could be that the participants having the highest UV exposure were partly female sun worshippers who were keen sunscreen users and partly male golfers and gardeners who did not use sunscreen at all.

The influence of skin pigmentation on vitamin D level

In general vitamin D status is considered negatively related to the skin pigmentation (8). In contrast hereto, we found that the constitutive skin pigmentation did not affect the achieved $25(\text{OH})\text{D}$ in September, while the facultative pigmentation did. In addition the facultative pigmentation was UV dose related as it correlated significantly with the mean hours outdoor per day with UVR measurements. This indicated that a probable negative effect of a dark constitutive skin pigmentation as well as a high facultative pigmentation because of tanning in a summer season are of less importance for $25(\text{OH})\text{D}$ synthesis than the positive effect of the UVR exposure dose and the skin area exposed. The fact that fair skinned subjects with the highest constitutive pigmentation in September were found to have the highest $25(\text{OH})\text{D}$ in the next winter could be explained by our earlier findings that people with darker skin who actually tolerate UVR better as skin types IV are more UVR exposed than the more fair-skinned and UVR-sensitive skin types I and II (18). The subjects in the actual study were all with Danish ancestry and thereby a relatively homogeneous pigmented group with the same sun cultural background. The results could be different if the study had included persons of another ethnicity or inclination for sun exposure.

Our findings are in harmony with recent results from New Zealand where tanning measured on the forearm in late summer but not constitutive skin color was considered an important positive determinant of $25(\text{OH})\text{D}$ even though the volunteers comprised 255 fair skinned subjects with European ancestry as well as 87 with Pacific ancestry and a darker skin color (25). Opposite hereto a recent American study among 69 volunteers who were given 12 UVB sessions of 20–80 mJ cm^{-2} reported an association between unexposed skin color and $25(\text{OH})\text{D}$ with lighter skin being associated with higher $25(\text{OH})\text{D}$ (26), but the result is difficult to interpret as the UVB dose was increased with skin pigmentation. A saturation especially at the highest UVR doses that blocks the vitamin D synthesis can have taken place (16). Skin pigmentation and ambient UVR seems to be of minor importance for vitamin D levels than earlier anticipated, as a recent meta-analysis of cross-sectional studies of $25(\text{OH})\text{D}$ globally among healthy subjects in 394 studies indicates that vitamin D is more or less of the same level independent of skin pigmentation and latitude as a surrogate parameter for available ambient UVR (27).

Further studies are needed

In a just published report on Vitamin D and cancer the International Agency for Research on Cancer, WHO argues that more controlled studies measuring objectively the correlation between UVR exposure and 25(OH)D are needed before enhanced UVR exposure in order to increase vitamin D level is recommended (28). We recommend that these studies should be performed both in standardized laboratory settings taking baseline values of vitamin D, skin pigmentation, UVR dose, exposure time and area into consideration. However, as this study has elucidated that the situation often is different in real life settings, we highly recommend thorough field studies measuring how behavioral, cultural but also climatic differences influence vitamin D.

Acknowledgements—The authors are grateful to Henrik Jørgensen, MD PhD, for good advice regarding the biomarkers, to the laboratory technicians at the biochemical departments at Bispebjerg and Hvidovre Hospital, Copenhagen University Hospitals, Copenhagen, Denmark for blood test sampling and analysis, and to the volunteers for their high compliance with a tight study program. No external funding was used and the authors declare no conflicts of interest.

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