BJD
British Journal of Dermatology

Interdependence between body surface area and ultraviolet B dose in vitamin D production: a randomized controlled trial

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Accepted for publication
21 September 2010

Funding sources
Supported by the Danish Medical Research Council (271-08-0461), the Danish Medical Association Research Fund, the Danish Dermatological Society and Bispebjerg Hospital Research Fund.

Conflicts of interest
None declared.

DOI 10.1111/j.1365-2133.2010.10082.x

Summary

Background Ultraviolet (UV) B radiation increases serum vitamin D level expressed as 25-hydroxyvitamin-D_3 [25(OH)D], but the relationship to body surface area and UVB dose needs investigation.

Objective To investigate the importance of body surface area and UVB dose on vitamin D production after UVB exposure.

Methods We randomized 92 participants to have 6%, 12% or 24% of their skin exposed to 0·75 (7·5 mJ cm^{-2} at 298 nm using the CIE erythema action spectrum), 1·5 (15 mJ cm^{-2}) or 3·0 (30 mJ cm^{-2}) standard erythema doses (SED) of UVB. Each participant underwent four UVB exposures at intervals of 2–3 days. Skin pigmentation and 25(OH)D levels were measured before and 48 h after the final exposure.

Results The increase in 25(OH)D after irradiation [Δ25(OH)D] was positively correlated with body surface area ($P = 0·006$; $R^2 = 0·08$) and UVB dose ($P < 0·0001$; $R^2 = 0·28$), and negatively correlated with baseline 25(OH)D ($P < 0·0001$; $R^2 = 0·18$), for the entire data sample. However, when analysing each body surface area separately, we found a significant UVB response correlation for 6% ($P < 0·0001$; $R^2 = 0·48$) and 12% ($P = 0·0004$; $R^2 = 0·35$), but not for 24%. We also found a significant skin area response correlation for 0·75 SED ($P < 0·0001$; $R^2 = 0·56$), but not for 1·5 and 3·0 SED when analysing each UVB dose separately. The relationships did not change significantly after adjustment of Δ25(OH)D for baseline 25(OH)D.

Conclusion The increase in 25(OH)D depends mainly on the UVB dose; however, for small UVB doses the area of irradiated body surface is important.

Vitamin D is essential in calcium metabolism and may play a role in diseases such as diabetes, cancer and multiple sclerosis.[1–8] For most people, exposure to solar ultraviolet (UV) B (280–320 nm) radiation is thought to provide more than 90% of their vitamin D requirement.[9,10] Nevertheless, solar UVB radiation of the skin is also a recognized carcinogen.[11–13] Public health campaigns recommend limited sun exposure a few times each week during the summer to ensure sufficient production of vitamin D while minimizing the risk of skin cancer. However, there is uncertainty regarding the UVB dose and the size of body surface area that needs to be exposed. Only a few human studies have dealt with the importance of body surface area.[14,15] Barth *et al.*[14] irradiated different body surface areas and reported increasing vitamin D levels with increasing body area, whereas Matsuoka *et al.*[15] suggested that the vitamin D response after UVB irradiation reached a plateau when more than 33% of the body surface area was irradiated. However, both studies used minimal erythema dose (MED), an individual biological dose, assuming that skin pigmentation reduces vitamin D production after UVB exposure. A newly published study[16] calls into question whether winter skin pigmentation is of importance for the vitamin D response after UVB. Accordingly, it is difficult to interpret the results mentioned above. Therefore, in a randomized trial, we investigated the interdependence between body surface area and UVB dose in vitamin D production using a fixed physical UVB dose regardless of skin pigmentation.

Materials and methods

A randomized, controlled trial was conducted at Copenhagen University Hospital, Bispebjerg, Denmark (56°N), from February to March in 2008 and 2009, when the ambient UVB radiation is negligible and the low outdoor temperature prohibits

exposure, apart from the face and hands. This study is registered at ClinicalTrials.gov (http://clinicaltrials.gov/) and its unique identifier/registration number is NCT01042197. The objective was to investigate the importance of body surface area and UVB dose on the increase in serum vitamin D level after irradiation expressed as Δ25-hydroxyvitamin D_3 [Δ25(OH)D].

The Danish Medical Ethics Committee approved the study (H-B-2007-100), which was conducted according to Declaration of Helsinki principles. All participants gave written informed consent.

Of 102 participants screened for inclusion, nine were excluded under the protocol exclusion criteria. A total of 92 participants completed the study, one dropped out because of personal reasons. All participants responded to a questionnaire that ascertained information on chronic diseases, use of medications and dietary supplements, sun holidays and sunbed use. Weight and height were recorded from which the body mass index (BMI) and the total body area in square metres were calculated.[17]

The inclusion criteria were: age 18–65 years, avoidance of sunbed exposure, not holidaying south of 45° latitude during the 3 months before the study, and avoiding vitamin D supplementation for 3 months before the study and during the study period. Participants were ineligible for the study if they had any skin disease, psychiatric disease or drug addiction, if they took medication that could cause photosensitive skin, or took cholesterol-lowering medicine, or were pregnant.

Interventions

The participants were randomized to receive four UVB exposures of 0·75 [7·5 mJ cm^{-2} at 298 nm using the Commission Internationale de l'Eclairage (CIE) erythema action spectrum], 1·5 (15 mJ cm^{-2}) or 3·0 (30 mJ cm^{-2}) standard erythema doses (SED) at intervals of 2–3 days. Each participant wore specially designed sleeves, trousers, gloves and helmet with only the chest and back exposed (24%), only the back exposed (12%) or only the half of the back exposed (6%).[18] Exposure of half the back was achieved by placing an operation blanket on one side of the back. The group (n = 10) who received 3·0 SED for 10 min to the chest and back was derived from our recently published study.[16] All the UVB exposures were given for 10 min except in 10 subjects (10/92, 11%) who received exposure for 5 min (five subjects in each group who received 0·75 SED and 1·5 SED to 24% of the body surface area). There was no significant relationship between Δ25(OH)D and the exposure time (P = 0·8).

Randomization

A computer-generated randomization list was used to number sealed envelopes containing notes with the three different UVB doses and with the three different body surface areas. The researcher responsible for seeing and treating the participants used the envelopes consecutively.

Objective skin type/pigmentation and redness

Skin type may be determined objectively as the UV dose (erythema weighted) that can elicit just perceptible erythema of the skin (MED). The unit for erythema-weighted UV dose is SED. The number of SEDs to elicit MED is thus synonymous with objectively measured skin type and may be determined by a MED test or by remittance spectroscopy.

To follow the skin response to UVB radiation, percentage redness (measuring range 0–100%) and pigment protection factor (PPF, measuring range 1·0–24·0) were measured on the back (facultative skin pigmentation) and buttocks (constitutive skin pigmentation) at baseline and 2 days after the last UVB session using a skin reflectance meter (UV-Optimize Scientific 558; Chromo-Light, Espergaerde, Denmark).[19] PPF is a measure of melanin in the skin and is objectively measured as number of SED to MED by skin reflectance measurements.[20,21] The correlation between clinically measured MED and PPF is highly significant.[22] The individual dose expressed in MED is the exposure dose in SED divided by the same individual's PPF.[23] A typical person with skin type I–IV has a PPF value of 4–5 on the buttocks and up to 8–12 on other body locations, depending on previous sun exposure.[24]

The percentage redness (0–100%) is an objective skin reflectance measure of haemoglobin in the skin. Zero percentage skin redness is the reflectance from a blood-drained skin area, and 100% skin redness is the reflectance from a highly vascular skin lesion such as that found in a facial dark-blue naevus flammeus. There is linearity between the clinical scale of skin redness generated by UV lamp irradiation and the objective measure of skin redness by reflectance.[23] Fitzpatrick[25] skin types were registered at baseline (Table 1).

Irradiation

A broadband UVB light source consisting of a flat bank of Philips TL12 tubes (280–360 nm; Philips, Eindhoven, the Netherlands) was used. The UV source irradiated all body surface areas with equal intensity. To obtain the different UVB doses during the two different exposure times (5 and 10 min) the distance between the UV source and the subjects was varied. The UV source was chosen so that the emission spectrum fitted the action spectrum for optimal vitamin D production.[26] The UV intensity was checked weekly using a Sola-Hazard spectroradiometer (Solatell, Cornwall, U.K.) to monitor the stability of the light source during the trial period. The radiation dose was measured in the erythema-weighted international standard SED; one SED is defined as 10 mJ cm^{-2} at 298 nm using the CIE erythema action spectrum,[27,28] and it is the UV dose that elicits just perceptible erythema in the most sensitive person in a group of very sun-sensitive healthy individuals.[29]

Biochemical analyses

Blood samples were analysed for 25(OH)D, parathyroid hormone (PTH), ionized calcium, alkaline phosphatase and total

Table 1 Baseline characteristics among the 92 participants and the relationship between the baseline factors and the increase in 25-hydroxyvitamin-D_3 [Δ25(OH)D] after ultraviolet (UV) B exposure [not adjusted for UVB dose (0·75–3·0 standard erythema dose, SED) or exposed body surface area (6–24%)]

Baseline factors	Baseline characteristics	P-value
Sex, men/women (%)	31 (33·7)/61 (66·3)	0·93
Skin type,[25] I/II/III/IV	10/43/21/18	0·78
Age (years)	32·8 ± 10·5 (18–62)	0·15
Weight (kg)	72·6 ± 15·2 (50–116)	0·46
Height (m)	1·75 ± 0·09 (1·55–1·96)	0·74
Body mass index (kg m^{-2})	23·7 ± 3·9 (18·2–37·2)	0·51
Total body area (m^2)[a]	1·91 ± 0·24 (1·49–2·55)	0·50
Serum 25(OH)D (≥ 50 nmol L^{-1})[b]	35·8 ± 16·8 (9–104)	< 0·0001*
Serum PTH (1·1–7·1 ρmol L^{-1})[b]	4·4 ± 2·0 (0·4–10·3)	0·21
Serum ionized Ca^{2+} (1·15–1·30 mmol L^{-1})[b]	1·22 ± 0·04 (1·13–1·34)	0·10
Serum alkaline phosphatase (35–105 U L^{-1})[b]	66 ± 15·3 (21–125)	0·34
Serum total cholesterol (2·9–7·1 mmol L^{-1})[b]	4·8 ± 1·0 (2·5–8·0)	0·76
PPF buttock[c]	4·8 ± 1·4 (1·1–9·4)	0·33
PPF back[d]	5·8 ± 1·7 (2·8–10·0)	0·06**
Redness, back (%)	23·1 ± 6·3 (9·2–38·8)	0·36

Values are mean ± SD (range) unless otherwise indicated. PPF, pigment protection factor; PTH, parathyroid hormone. [a]Total body area[17] (m^2) = 0·024 × height$^{0·40}$ × weight$^{0·54}$; [b]reference intervals for biochemical parameters; [c]constitutive skin pigmentation; [d]facultative skin pigmentation; *P = 0·002 when adjusted for UVB dose (0·75–3·0 SED) and body surface area (6–24%); **P = 0·11 when adjusted for UVB dose (0·75–3·0 SED) and body surface area (6–24%).

cholesterol at baseline and 2 days after the fourth and last UVB session. The blood samples were centrifuged (5000 *g* for 10 min) within 2 h of sampling. All the analyses have been described previously.[16]

Definition of vitamin D levels

In this study, the definition of vitamin D sufficiency is defined as 25(OH)D level > 50 nmol L^{-1} (20 ng mL^{-1}), vitamin D insufficiency as 25(OH)D level < 50 nmol L^{-1} (20 ng mL^{-1}) and vitamin D deficiency as 25(OH)D level < 25 nmol L^{-1} (10 ng mL^{-1}).

Statistical analysis

Before the study, the following assumptions were made: given a significance level of 5% and an assumed standard deviation of 9 nmol L^{-1} for the 25(OH)D analysis at the 50 nmol L^{-1} level, the study was designed to show a difference of at least 12 nmol L^{-1} between the groups with a power of 80% if at least nine subjects per group completed the study.

The data were statistically handled using SPSS 17.0.2 for Windows (SPSS Inc., Chicago, IL, U.S.A.). The relationship between Δ25(OH)D, and sex, age, height, weight, BMI, body surface area, UVB dose, baseline 25(OH)D, biochemical parameters, PPF and percentage redness was examined by analysis of variance (ANOVA) both individually and with the body surface area and the UVB dose included for all variables in every analysis. For all significant analyses, multiple linear regression was performed with body surface, UVB dose and baseline 25(OH)D as independent variables. The significance limit was $P < 0·05$.

By using univariate analysis of variance with the present data, a combined model was made to imitate the production of vitamin D after different UVB doses and for different body surface areas. In order to simulate the nonlinear increase in 25(OH)D as found in the results (Tables 2 and 3), we transformed the UVB dose and body surface area using the natural logarithm function. This transformation was only used in the combined model as the other statistical analysis was stronger with a linear regression. In Figure 3 of our previous study,[16] we showed the relationship: Δ25(OH)D = 31·4–0·223 × [baseline 25(OH)D] ($P = 0·000024$; $R^2 = 0·313$). Therefore the Δ25(OH)D was adjusted for baseline 25(OH)D level using: adjusted Δ25(OH)D = Δ25(OH)D + 0·223 × [baseline 25(OH)D].[16]

Results

Baseline data

Relevant personal and biochemical characteristics at baseline for the 92 participants are shown in Table 1. Of the 92 subjects, 76 (83%) were vitamin D insufficient [25(OH)D < 50 nmol L^{-1}] including 28 (30%) who were vitamin D deficient [25(OH)D < 25 nmol L^{-1}].

Analyses of the entire data sample

We found that Δ25(OH)D was positively correlated with the exposed body surface area ($P = 0·006$; $R^2 = 0·08$) and the UVB dose ($P < 0·0001$; $R^2 = 0·28$) and negatively correlated with baseline 25(OH)D level ($P < 0·0001$; $R^2 = 0·18$) when the entire data sample was analysed. No significant

Table 2 The actual increase (mean) in 25-hydroxyvitamin-D_3 [Δ25(OH)D, nmol L^{-1}] after ultraviolet (UV) B exposure (0·75–3·0 standard erythema dose, SED); n = 10 or 11 in each UV group

	Body surface area (%)			
UVB dose (SED)	6	12	24	P (R^2), relation to body surface area
0·75	1·9	9·0	19·9	< 0·0001 (0·56)
1·5	13·5	13·4	19·7	0·15 (0·073)
3·0	22·7	30·7	25·0	0·90 (0·001)
P (R^2), relation to UVB dose	< 0·0001 (0·48)	0·0004 (0·35)	0·08 (0·108)	

Table 3 The increase (mean) in 25-hydroxyvitamin-D_3 [Δ25(OH)D, nmol L^{-1}] after ultraviolet (UV) B exposure (0·75–3·0 standard erythema dose, SED), adjusted for baseline 25(OH)D[a]; n = 10 or 11 in each UV group

	Body surface area (%)			
UVB dose (SED)	6	12	24	P (R^2), relation to body surface area
0·75	10·2	16·5	26·5	< 0·0001 (0·54)
1·5	22·8	25·1	27·6	0·22 (0·05)
3·0	29·4	38·5	30·7	0·91 (0·00004)
P (R^2), relation to UVB dose	< 0·0001 (0·45)	< 0·0001 (0·41)	0·14 (0·08)	

[a]Adjusted Δ25(OH)D = Δ25(OH)D + 0·223 × [baseline 25(OH)D]. The adjusted Δ25(OH)D was calculated using the relationship: Δ25(OH)D = 31·4–0·223 × [baseline 25(OH)D] from Figure 3 in reference 16.

relationships were found between Δ25(OH)D and the following baseline factors: sex, Fitzpatrick skin type, age, weight, height, BMI, total body area, PTH, ionized calcium, alkaline phosphatase, total cholesterol, percentage redness and constitutive or facultative pigmentation (Table 1). Multiple linear regressions were performed on all significant single parameters (body surface area, UVB dose and baseline vitamin D), showing that they remained significant (P < 0·0001; R^2 = 0·45).

As expected, the facultative skin pigmentation increased [mean (SD)] significantly (P = 0·046) from 5·3 (1·7) to 5·8 (1·4) as did the percentage redness (P = 0·03) [mean (SD)] from 22·1 (6·5) to 24·0 (4·7) during the course of UVB treatments for the participants who received a UVB dose of 3·0 SED per session, whereas the other UVB doses did not result in a significant increase in pigmentation or skin redness. The PTH level [mean (SD)] decreased significantly from 4·4 (2·0) to 4·0 (1·9) pmol L^{-1} (P = 0·04).

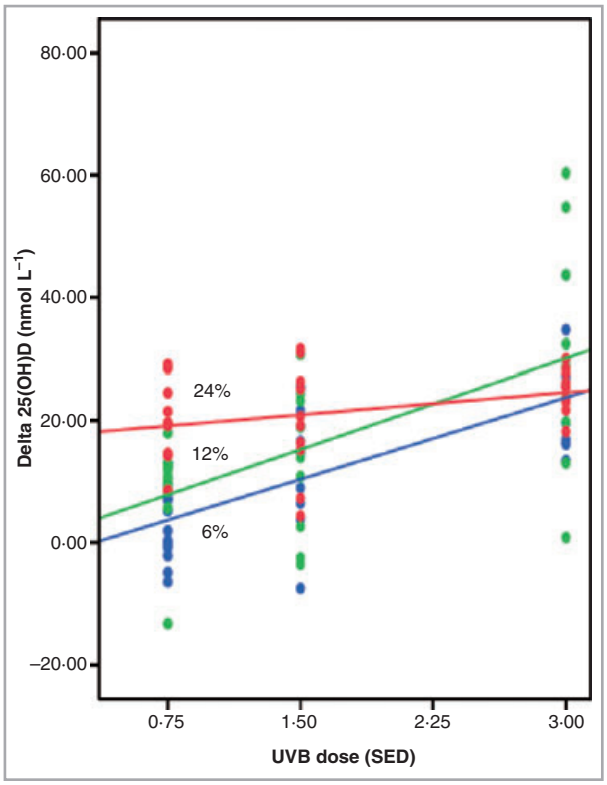

Fig 1. Relationship between the increase in vitamin D [25-hydroxyvitamin-D_3; Δ25(OH)D] and the ultraviolet (UV) B dose of 0·75 standard erythema dose (SED), 1·5 SED and 3·0 SED. Red line, 24% body surface area (P = 0·08; R^2 = 0·108); green line, 12% body surface area (P = 0·0004; R^2 = 0·35); blue line, 6% body surface area (P < 0·0001; R^2 = 0·48).

Separate ultraviolet B dose analysis

When analysing each body surface area separately (6%, 12% or 24%), we found a significant positive correlation between Δ25(OH)D and UVB dose for 6% (P < 0·0001; R^2 = 0·48) and 12% (P = 0·0004; R^2 = 0·35), while a state of saturation was reached when 24% of the body area was exposed (P = 0·08) (Table 2; Fig. 1). Table 3 shows that the relationships between Δ25(OH)D and UVB dose were almost identical when Δ25(OH)D was adjusted for baseline 25(OH)D with positive significant relationships for 6% (P < 0·0001; R^2 = 0·45) and 12% (P < 0·0001; R^2 = 0·41), but not for 24% (P = 0·14).

Separate body surface area analysis

We also found a significant positive correlation between Δ25(OH)D and body surface area for 0·75 SED (P < 0·0001; R^2 = 0·56) but with a state of saturation for 1·5 SED (P = 0·15) and 3·0 SED (P = 0·90) when analysing each UVB dose separately (Table 2; Fig. 2). When we adjusted Δ25(OH)D for baseline 25(OH)D, the relationship between Δ25(OH)D and body surface area remained almost unchanged

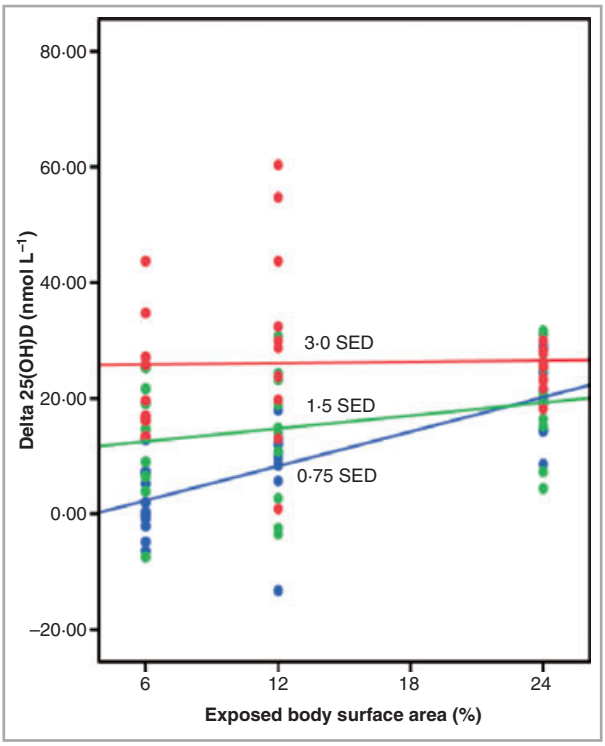

Fig 2. Relationship between the increase in vitamin D [25-hydroxyvitamin-D_3; Δ25(OH)D] and the exposed body surface areas of 6%, 12% and 24%. Red line, 3 standard erythema dose (SED) ($P = 0{\cdot}9$; $R^2 = 0{\cdot}001$); green line, 1·5 SED ($P = 0{\cdot}15$; $R^2 = 0{\cdot}073$); blue line, 0·75 SED ($P < 0{\cdot}0001$; $R^2 = 0{\cdot}56$).

(Table 3) with positive significant relationships for 0·75 SED ($P < 0{\cdot}0001$; $R^2 = 0{\cdot}54$), but not for 1·5 SED ($P = 0{\cdot}22$) and 3·0 SED ($P = 0{\cdot}91$).

Estimation of 25-hydroxyvitamin-D_3 increase after ultraviolet B in a combined model

When knowing that the 25(OH)D response after UVB exposure depends on baseline 25(OH)D, UVB dose and body surface area, the approximate Δ25(OH)D can be described by the following equation: $\Delta 25(OH)D = 7{\cdot}1 - 0{\cdot}223 \times [\text{baseline } 25(OH)D] + 11{\cdot}0 \times \ln(\text{UVB dose}) + 5{\cdot}5 \times \ln(\%\text{ body area})$, ($P < 0{\cdot}0001$; $R^2 = 0{\cdot}47$) where $\ln(x)$ is the natural logarithm of x. The equation is based on UVB doses from 0·75 to 3·0 SED and for a body surface area between 6% and 24%.

Discussion

When all 92 participants were included in the statistical analyses, we found a significant positive relation between Δ25(OH)D and body surface area. However, when analysing each body surface area separately, we found a significant correlation only when we irradiated with the smallest UVB dose of 0·75 SED. A state of saturation was reached for the higher UVB doses of 1·5 and 3·0 SED, where no significant relationship to body surface area was found. We also found a significant positive correlation between Δ25(OH)D and UVB dose for the entire data sample. However, when viewing each UVB dose separately, we found a significant dose–response relationship for 6% and 12% of body surface area, while a state of saturation was reached for the largest body surface area of 24%. This means that there is an interdependence between body surface area and UVB dose, where the increase in 25(OH)D depends mainly on the UVB dose; however, for small UVB doses the area of the irradiated body surface becomes important.

Notably, a very small UVB dose of 0·75 SED (~8 min of sun exposure on a clear day around the summer solstice in Denmark, 56°N) and a small body surface area of 12% resulted in significant 25(OH)D production. Exposure to higher UVB doses and to larger body areas gives a less favourable UV risk–benefit ratio (Tables 2 and 3). A body surface area of 6% equals that of the face and hands, 12% equals the face and arms and 24% equals the face, arms and legs.[18] We also found the limit at which almost no vitamin D is produced: no significant increase in 25(OH)D was achieved with exposure of 6% body surface area with 0·75 SED for the unadjusted actual vitamin D increase (Table 2).

Only two studies have dealt with the relationship between body surface area and vitamin D production.[14,15] Both studies found a nonlinear relationship between vitamin D production and body surface area, indicating that saturation occurs rapidly. Matsuoka *et al.*[15] suggested that vitamin D production reaches a plateau with irradiation of more than 33% of the body surface area. However, the authors concluded that the data were inadequate and that the phenomenon remains to be determined.[15] In addition, both studies assumed that skin pigmentation reduces vitamin D production in the skin after UVB exposure. Accordingly, the authors dosed in MED, an individual biological dose. However, a newly published study from our group calls into question whether winter skin pigmentation is of importance for vitamin D production.[16] It can be argued that 3·0 SED administered four times to 24% of the body surface area might have been sufficient to reach saturation in our previous study;[16] however, in the same study we found a remarkable increase in 25(OH)D after UVB exposure among the dark-skinned group, showing that skin pigmentation, saturation or not, might not be as important as earlier assumed in reducing the production of vitamin D after UVB exposure. In fact, there was a factor of 2·5 difference (10·1/4·1) in the UVB dose, if the UVB doses were adjusted for the pigmentation – the PPF buttock (mean) among the fair-skinned was 4·1, and the PPF buttock (mean) among the dark-skinned was 10·1. In addition, the relationship between Δ25(OH)D and the UVB dose in this study ($P = 0{\cdot}00000006$; $R^2 = 0{\cdot}28$) grew weaker when the UVB dose was adjusted for pigmentation (MED = UVB dose/PPF back) ($P = 0{\cdot}0000179$; $R^2 = 0{\cdot}20$). Therefore a fixed physical UVB dose was chosen. Consequently, it is difficult to draw firm conclusions from the previous studies, although we, too, found saturation after exposure of about 24% of the body surface area.

The fact that our recently published study[16] was carried out in the laboratory with broadband UVB lamps (TL12) and not with lamps emitting natural sunlight represents a limitation. One might speculate that the short-wave components in the broadband UVB lamps (TL12) would induce vitamin D production above the main melanin layer in the skin. However, we agree with a newly published review[30] about skin pigmentation and vitamin D status, which states that the role of melanin on vitamin D status is unclear and requires further investigation. We also showed a significant negative correlation between Δ25(OH)D and baseline 25(OH)D, as was also the case in our recent study.[16] The Δ25(OH)D was therefore adjusted according to baseline 25(OH)D level in the statistical analyses of the importance of UVB dose and body surface area (Table 3).

To strengthen our findings, we excluded participants who took vitamin D supplements or travelled south of the 45° latitude, included two serum samples from each subject before and after UVB exposure, measured pigmentation and conducted the study in February to March when the ambient UV radiation is negligible and the blood concentration of vitamin D can be considered at the year's lowest in Denmark at 56°N. The light intensity was measured frequently to monitor the stability of the light sources during the trial. UVB radiation was used instead of artificial sunlight because the spectrum of the sun varies according to the time of day and the time of year; artificial sun delivers only one of these spectra and it is difficult to obtain uniform irradiation of large body surface areas with artificial sunlight. A limitation of this study was the variance in the liquid chromatography–mass spectrometry analysis of 25(OH)D of approximately 4%, despite performing each 25(OH)D analysis twice.

We did not divide the participants into subgroups according to their previous sun exposure, as we did in our recent study[16] where we needed a homogeneous population according to baseline vitamin D level when analysing the correlation between total cholesterol (precursor to vitamin D) and Δ25(OH)D. This may explain the lack of a relationship between Δ25(OH)D and total cholesterol in this study.

Our study supports the theoretical calculations made by McKenzie *et al.*[31] and the time stated by Holick[32] that significant vitamin D can be produced by only a few minutes of sun exposure [assuming the UV index (UVI) is three in the middle of a clear sky day] on ~24% of the body surface area. In addition, our results support the guidelines given by public health campaigns, which recommend a few short sun exposures in a week during the summer. Accordingly, our study shows (Table 3) that given the same baseline 25(OH)D level (31·4 nmol L^{-1}) and a UVI of 3, a sufficient level of vitamin D (> 50 nmol L^{-1}) would be reached by four exposures of either 15 min sun exposure (0·75 SED) to ~24% of the body surface area or 30 min sun exposure (1·5 SED) to only ~6% of the body surface area. However, because of its skin carcinogenic effect, we would not recommend increasing UVB exposure as a vitamin D source, because sufficient vitamin D levels can easily be obtained with vitamin D supplements.[33–35]

In conclusion, the increase in 25(OH)D depends mainly on the UVB dose; however, for small UVB doses the area of the irradiated body surface is important.

What's already known about this topic?

- Few studies have dealt with the relationship between body surface area, ultraviolet (UV) B dose and vitamin D production. These studies used minimal erythema dose assuming that skin pigmentation reduces vitamin D production. However, the importance of skin pigmentation is debatable. Consequently, it is difficult to draw firm conclusions.

What does this study add?

- In this study, we investigated the relationship between body surface area, UVB dose and vitamin D production using a fixed UVB dose regardless of skin pigmentation.

Acknowledgments

We thank the participants, Henrik Jørgensen MD, PhD, and the laboratory technicians for their help in the project. This work was supported by grants from the Danish Medical Research Council (271-08-0461), the Danish Medical Association Research Fund and the Danish Dermatological Society and Bispebjerg Hospital Research Fund.

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