Sunbeds as Vitamin D Sources

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ABSTRACT

The objectives of this work were: (1) To determine whether repeated exposures to small doses from a commercial sun bed (Wolff Solarium Super Plus 100 W) over 5 weeks gave less vitamin D than repeated exposures to twice as large, but still nonerythemogenic, doses. (2) To investigate whether the contribution to the vitamin D status from such sessions of exposures was dependent on the baseline status before the start of the sessions. (3) To determine the decay rate of the induced increment of vitamin D. The sun bed sessions raised the 25-hydroxyvitamin D levels from typical winter values to typical summer values the mean value after exposure being 80 nm (±14) and the increase being 15 nm on average. Persons with the lowest initial levels got the largest increase. The level in this group was back to the pre-exposure level after 2–4 weeks. To maintain a summer level through the winter, when no vitamin D is produced by the sun in northern countries, one should consider increasing the recommended intake of vitamin D intake significantly, or encouraging the population to get moderate, nonerythemal sun bed exposures.

INTRODUCTION

The health effects of vitamin D have been focused on for several years (1,2). A number of reports indicate that with a good vitamin D status prognosis is improved, adverse symptoms are alleviated and incidence rates are reduced for several forms of internal cancers (colon, prostate, lung, breast, lymphomas, etc.) (3,4), for multiple sclerosis (5), diabetes Type I and 2 (6,7), rheumatoid arthritis and several other autoimmune diseases (8), for influenza (9) and for cardiovascular diseases (10). Furthermore, rickets and osteomalacia, diseases related to a poor vitamin D status, are reappearing in certain population groups, notably immigrants with dark skin (11,12). Recently, a large IARC report (13) concluded that there was only limited evidence for any correlation between vitamin D levels and cancer risk or mortality. However, the report was criticized by a number of vitamin D experts for omissions, errors and lack of updating, and thus not describing the present state of knowledge correctly (14,15). Thus, the matter is not yet settled, and further investigations are certainly warranted.

In the United States and in a number of European countries a low vitamin D status has been reported for teenagers, elderly and even for apparently healthy adolescents (16–19). The vitamin D level is usually estimated by measuring the serum concentration of 25-hydroxyvitamin D (25[OH]D). Optimal levels are being discussed, and are certainly different for avoidance of different diseases (20–22). Levels below 50 nm are likely to be inadequate, and much higher levels are probably needed for optimal health.

Sun exposure in the summer months is regarded as a main human source of vitamin D. Consumption of fat fish (herring, mackerel, salmon), cod liver oil and supplements are also good sources (23). At latitudes above 40° solar radiation contains too little UVB (280–320 nm) during the winter months (October to March) to produce significant amounts of vitamin D (24). Thus, higher serum levels of 25(OH)D are found in the summer than in the winter in practically all published investigations (25). Our review of published data indicates that summer values are about 70–80 nm, while typical winter values are 40–50 nm in a number of countries (25). We have found that cancer prognosis is significantly better for summer diagnosis than for winter diagnosis, and, on the background of a number of experimental and epidemiological studies reported in the literature, we have suggested that our findings are likely to be related to 25(OH)D variations through the year (24,26–29). This indicates that significant health benefits might be obtained by raising the winter level of 25(OH)D in a population to a level similar to that in the summer. This could be achieved either by exposure to sun beds or by ingestion of sufficient amounts of food and supplements containing vitamin D. Before recommending general sun bed use, one has to evaluate the vitamin D yield and the accompanying skin cancer risk of such use more thoroughly than has yet been done.

The present work was carried out with the following aims: (1) To determine whether repeated sun bed exposures,
acquiring a total dose of 6.75 minimal erythema doses (MEDs), from a commercial sun bed (Wolff Solarium Super Plus 100 W, Basel, Switzerland) over five weeks gave a significantly smaller improvement of the vitamin D status than repeated exposures accumulating to a total dose of 13.5 MEDs. (2) To find out whether the sun bed-induced contribution to the vitamin D status was dependent on the baseline vitamin D status. (3) To estimate the decay rate of the increased serum 25(OH)D levels in groups with different baseline levels.

**MATERIALS AND METHODS**

**Volunteers.** Twenty-three healthy volunteers were included in the study, seven men and 16 women aged between 21 and 65 years. The average age was 34 years. All were living in Oslo (59°N). Most of the participants were Caucasians and had Fitzpatrick skin types II and III, except two men with skin type V.

The dietary vitamin D intake was estimated by using a specially designed food frequency questionnaire that was offered to all the participants at the beginning of the study. Questions about weight and height for calculation of body mass index (BMI) were included. The individual vitamin D intakes were estimated on the basis of the questionnaire. All participants were asked to stick to their normal diet during the study, to avoid any changes in vitamin D intake that might be caused by increased awareness of the problem of vitamin D deficiency.

To avoid any contribution from solar radiation, the study was conducted during the winter months (November to March), a time of the year when no vitamin D is synthesized in skin by sun exposure at our latitudes (24). None of the participants had used sun beds for at least 1 month prior to the start of the study.

The Regional Ethical Committee approved the study, and each participant gave informed consent. All completed the entire study. A pilot study had indicated that reliable trends would be obtainable even with the present low number of participants.

**Protocol.** The participants were split in two groups, similarly composed with regard to distribution of age, gender, BMI and vitamin D intake. The study extended over 12 weeks, including two phases: 1: intervention and observation and 2: only observation. For both groups a Norwegian summer was simulated by moderate exposures to a commercial sun bed two times per week. Individual MEDs were determined before starting the exposures (see below), to avoid any sun burns. Group A started with an exposure of 0.5 MED, and then the exposures were escalated by approximately 0.1 MED per session to reach 1 MED. Group B started with an exposure of 0.25 MED, escalating by 0.05 MED per exposure to reach 0.5 MED. In both groups the maximum exposure per session was reached at the fifth session. Exposures at this level continued up to 15 sessions, totally. After this part of the study (intervention), serum samples were collected for a 5 week observation period to determine the decay rate of serum 25(OH)D.

**Ultraviolet exposure.** The source of UV radiation was a commercially available and approved sun bed, equipped with Solarium Super Plus 100 W tubes (Wolff System). The spectrum given by the producer is shown in Fig. 1. The fluence rates were measured using a UV-meter (Solar Light Company Inc.) and the CIE adjusted exposures were 16.4 mW cm$^{-2}$ in UVA and 0.2 mW cm$^{-2}$ in UBV. Figure 1 shows the spectral characteristics of the sun bed radiation compared with midsummer sun at noon in Oslo and at the Equator. Spectra relevant for vitamin D photosynthesis are also shown: that for 7-DHC absorption in an organic solution (32), that for vitamin D formation in human skin (33) and that for the yield of vitamin D and 1,25(OH)$_2$ vitamin D (they are similar) in human skin (34). To be comparable with the exposure set-up in a sun bed, the solar fluence rates are given for a plane surface perpendicular to the direction from the sun.

**RESULTS**

The spectrum of two sun beds, the one used in the present work and that used in our earlier work (30), compared with the solar spectrum at noon, midsummer in Oslo and under the Equator, are shown in Fig. 1. General characteristics of the study population are given in Table 1.

Moderate, nonerythromogenic exposures to the sun bed twice a week up to a total of 15 exposures were surprisingly efficient in increasing the serum 25(OH)D level (Fig. 2). The increase was almost the same for the two exposure patterns. The average MED was about 23 min (SD ± 3 min). The total exposure was 13.5 MEDs (5.4 h [SD ± 0.5 h]), with 1 MED in each of the last nine exposures for group A and 6.75 MEDs (2.5 h [SD ± 0.5 h]), with 0.5 MED in each of the last nine exposures for group B. Both exposure patterns gave a 25–30% increase in the 25(OH)D levels.

We studied this further and divided the data in two (Fig. 3) and three (Fig. 4) groups based on initial 25(OH)D level as given in the figure legends and in the discussion. BMI, within the range in the present population (19–29 kg m$^{-2}$), had a small influence on the initial level of 25(OH)D, but not on the increase (Fig. 5).
DISCUSSION

The sun bed UVB fluence rates were larger in our earlier study (30) than in the present one, but none of them are widely different from that in Equatorial sun. However, the UVA fluence rates are larger for the sun beds, notably for the one used in the present work. Several spectra relevant for photosynthesis of vitamin D are also shown in Fig. 1. The action spectra for generation of vitamin D in vivo are redshifted compared with the absorption spectrum of 7-dehydrocholesterol (7-DHC) in organic solutions (31). This is clearly shown

Table 1. General characteristics of the study population.

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<td>Number of participants</td>
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<td>Initial 25(OH)D levels, nmol L⁻¹ (SD)</td>
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<td>63.9 (± 6.09)</td>
<td>86.5 (± 8.6)</td>
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<td>25(OH)D after 15 exposures, nmol L⁻¹ (SD)</td>
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<td>77.6 (± 13.7)</td>
<td>95.6 (± 14.2)</td>
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<td>Absolute 25(OH)D increase, nmol L⁻¹ (SD)</td>
<td>22 (± 13)**</td>
<td>13.7 (± 14)*</td>
<td>9 (± 13.3)</td>
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<td>BMI, kg m⁻² (SD)</td>
<td>23.8 (± 3.5)</td>
<td>23.07 (± 3.4)</td>
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<td>Gender, % female</td>
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<td>75</td>
<td>83.3</td>
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25(OH)D = 25-hydroxyvitamin D; BMI = body mass index. *P = 0.022; **P = 0.0004.

Figure 2. Level of 25(OH)D (nmol) at baseline and as function of time in the intervention and observation phase for the two exposure patterns. The total exposure was 13.5 MEDs in group A and 6.75 MEDs in group B.

Figure 3. Level of 25(OH)D (nmol) at baseline and as function of time in the intervention and observation phase for the groups with high (larger than 62 nmol L⁻¹) and low (smaller than 62 nmol L⁻¹) initial levels of 25(OH)D.

Figure 4. 25(OH)D increase in three groups: persons with initial levels in the regions 40–50 nmol L⁻¹ (nine persons), 50–75 nmol L⁻¹ (eight persons) and 75–100 nmol L⁻¹ (six persons).

Figure 5. Initial and final 25(OH)D levels for persons of different body mass indexes (BMIs).
by the spectrum of Galkin and Terenetskaya (32) for solutions.

compared with action spectrum for human skin (33), and the
spectra showing the yields of vitamin D and 1,25(OH)₂ vitamin
D in skin (34). The latter spectra have peaks around 295–
305 nm, a region where both the sun and the sun beds emit
significant fluence rates. There are two reasons for the redshift.

First, the penetration depth of UV radiation into human tissue
decreases with decreasing wavelengths, due to absorption by
chromophores like proteins, nucleic acids, uracil and
melanin, and to scattering, which is Rayleigh-dominated, as
the majority of scattering elements are smaller than the
wavelength. The cross section of pure Rayleigh scattering
increases inversely with the fourth power of the wavelength,
thus being 40% larger at 295 nm than at 320 nm. Secondly,
the binding of 7-DHC in a lipid bilayer leads to a significant
redshift (31). Without this redshift only small amounts of
vitamin D would have been formed in skin by solar radiation,
which contains very little radiation with wavelengths covering
the 7-DHC spectrum in solution.

Our sun bed, being marketed as a “UVA sun bed”, gave
significant contributions to the serum 25(OH)D level. It seems
that even exposure pattern B (6.75 MEDs) gave saturation so
that no more could be gained by increasing the total dose to
13.5 MEDs. However, as Fig. 2 shows, there is almost a linear
increase up to the last exposure, so complete saturation did
probably not occur. This is in contrast to our earlier work (30)
which indicated that saturation might be approached already
after 4 weeks. However, another type of sun bed—one
emitting more UVB—was used in the first study, which
included only 10 persons. Several investigations have indicated
that larger exposures than those applied in the present work
are needed before saturation occurs (35–39).

The reason for the similarity in increase for the two groups
is probably that by chance the initial average level of 25(OH)D
was lower in group B than in group A (Fig. 2). Thus, when all
data were brought into one group and then divided in two
similarly large groups on the basis of initial 25(OH)D level,
one with initial levels below 62 nm (average 46 nm) and one
with initial levels above 62 nm (average 72 nm), it is evident
that those with low levels got more vitamin D from the
exposures than those with high initial levels (Fig. 3). We
studied this phenomenon in more detail and looked at the
25(OH)D increase in three groups: persons with initial levels in
the regions 40–50 nm (nine persons), 50–75 nm (eight persons)
and 75–100 nm (six persons) (Fig. 4). The increase in 25(OH)D
was largest for the group with the lowest initial 25(OH)D level
and decreased with increasing initial level. This cannot be
explained by differences between the groups in BMI, in
vitamin D intake or in gender composition (Table 1). Similar
trends have been observed in other recent studies (40,41). In
the study conducted by Carbone et al. a serial exposure to a
broadband UV radiation source resulted in 170% increase in
25(OH)D levels in persons with initial 25(OH)D concentra-
tions ≤75 nm and just 24% increase in the group with baseline
levels >75 nm (41). Other works (38,40) also showed that
persons with serum 25(OH)D ≤ 30 nm respond earlier to
seasonal changes in UV fluence rates. Moreover, the work
by Steingrimsdottir et al. (42) showed that the amplitude of
seasonal variation of 25(OH)D is highest for individuals not
taking vitamin D supplements, and, therefore, having low
circulating levels of 25(OH)D. Furthermore, Davie et al. (43)
found surprisingly large contributions to the vitamin D status
by exposing only 600–900 cm² of skin to UV radiation at doses
of only 3 min per day, three times per week. They even found
that the 25(OH)D level reached steady state after 5–6 weeks.

It may seem that the decay of the levels of 25(OH)D after
the sun bed sessions is fastest for the group with the lowest
initial level (Fig. 3). This deserves further investigations, as it
indicates that low 25(OH)D levels may be related to fast
metabolism and degradation of vitamin D derivatives.

Body mass index seems to influence the initial level of
25(OH)D but not the increase (Fig. 5). The observation that
the initial level decreases with increasing BMI is in agreement
with other published investigations (44–47) and with a large
yet unpublished study in our group. This can be explained by
the fact that vitamin D, and its precursors, are lipophilic, and,
therefore, partly stored in adipose tissue (47,48).

A main conclusion of our work is that small exposures to
UV radiation, amounting to only 6.75 MEDs, from a commercial
sun bed give large improvements to the vitamin D status.
This finding is in agreement with our earlier investigation
(30) as well as with the results of others (43–49), and may
have significant health consequences. Regular use of
sun beds with more than one exposure per week for
6 months or more resulted in 90% higher 25(OH)D serum
values and in higher bone mineral densities (0.97 ± 0.03 vs
0.92 ± 0.01 g cm⁻²) (39,50).

Sun beds are manufactured so as to follow the legislation
in different countries. This means that the biologically
weighted fluence rates have to be below a certain limit, often
comparable with Mediterranean, midsummer and midday
fluence rates (51). Furthermore, based on the belief that UVB
is more carcinogenic than UVA (320–400 nm), according to
the action spectrum of squamous cell carcinoma in mice (52),
sun beds are made to emit orders of magnitude more UVA
than UVB. A change in the legislation is the reason why we
have used a different type of sun bed in the present than in
the earlier work. Notably, the UVB/UVA ratio is lower in
the present work than in earlier investigations (0.02 vs 0.039).
However, even in the radiation of the present sun bed, the
small UVB fraction is large enough to give summer levels of
vitamin D.

As the action spectrum of vitamin D photosynthesis lies
mainly in the UVB region (Fig. 1) and is similar to the DNA
absorption spectrum (53), to the melanogenesis action spe-
crum and to the erythema action spectrum (54), or possibly
slightly redshifted compared with these spectra (as argued for
above), we can assume that whenever a sun bed can give
erythema and DNA damage, it can also generate vitamin D.
This would not be true for a sun bed emitting only UVA
radiation, but even so-called “UVA-sunbeds,” such as our sun
bed, emit small amounts of UVB.

The risk of cutaneous malignant melanoma (CMM) asso-
ciated with sun bed use has been the topic of a number of
investigations, as summarized in several reviews (55–58). Some
investigations show a CMM-generating effect of sun bed use
(59,60), while other investigations show no such effect, or even
protective effects (61,62). It has been stated that the discrep-
cy may be related to methodological shortcomings (63,64),
and that one has to wait for further results before firm
conclusions can be drawn. A Norwegian investigation indi-
cated that a relatively large melanoma risk might result from
According to the estimations of Giovannucci et al. (24), by 1500 IU (67), corresponding to 20 mL cod liver oil. However, before making any firm conclusion one should await more interventional investigations about the relationships between vitamin D levels and cancer. Furthermore, making any firm conclusion one should await more interventional investigations about the relationships between vitamin D levels and cancer. Such an increase might have a significant impact on death rates of internal cancers (4,66).

Raising winter levels of 25(OH)D up to summer levels (50 nm up to 80 nm in the Nordic countries [24]), would lead to a 22 nm higher average annual 25(OH)D level than the present one. This might be achieved by moderate sun bed exposures during the winter or by increasing the daily intake of vitamin D by 1500 IU (67), corresponding to 20 mL cod liver oil. According to the estimations of Giovannucci et al. (66) this would reduce the total number of cancer deaths by 29%, corresponding to a reduction in the annual number of cancer deaths in Norway by 3000 from the present level of 11 000. This is 10 times more than the number of melanoma deaths in Norway per year (about 250 at present) (68). However, before making any firm conclusion one should await more interventional investigations about the relationships between vitamin D levels and internal cancer.

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