

Review

## Sun and Sun Beds: Inducers of Vitamin D and Skin Cancer

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**Abstract.** Solar radiation is both the main cause of all types of skin cancer, including cutaneous malignant melanoma (CMM), and the main source of vitamin D accompanied by its beneficial effects. The dilemma lies in that increased sun exposure could lead to an increase in skin cancers and yet is necessary for the better prognosis of internal cancers. Solar radiation varies in intensity and spectral composition with geographic location and time. Of central interest in the present context is that the UVA/UVB ratio can vary. Thus, the UVA/UVB ratio increases with decreasing solar elevation. The ratio is also larger for most sun beds than that in the midday sun, but similar to that in the afternoon sun. This may have large health implications, since vitamin D is exclusively generated by UVB, while UVA and UVB likely play a role in the onset of CMM. Sun and sun beds act similarly: one quantum of radiation at a given wavelength has the same biological effect, irrespective of the source from which it comes. The winter levels of vitamin D are 10 to 100% lower than the summer levels in most populations, but can be brought up to summer levels by moderate sun bed exposure. Doses of 200 IU of vitamin D per day are not sufficiently large to maintain a summer vitamin D status in winter. At high latitudes (>40 degrees) the sun provides no vitamin D in winter. A number of epidemiological studies, interventional studies, animal studies and cell experiments show that vitamin D reduces the risk and/or prognosis of internal cancers. Populations living at high latitudes would probably benefit from moderately increasing their exposure to UVB to provide a good vitamin D status.

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Key Words: Vitamin D, sun bed, skin cancer, cancer prognosis, review.

Ultraviolet (UV) radiation from the sun has been the main source of vitamin D throughout the evolution of humans. However, solar radiation can also have negative consequences, of which skin cancer induction and degradation of folic acid are the most important (1-3). The balance between beneficial and adverse effects of UV radiation is of great evolutionary and health significance, as accentuated by the development of different human skin colors: dark and protective colors where the solar radiation is strong and white colors where it is weak.

Sun beds are artificial UV sources, originally constructed for tanning purposes. However, the spectral region associated with tanning is essentially the same as that associated with non-melanoma skin cancer and with vitamin D photosynthesis. Thus, radiation from sun beds acts similarly to solar radiation with respect to biological effects: a quantum of radiation, when absorbed, has the same biological effect irrespective of its source. However, different types of sun beds emit radiation with different spectra and similarly, the sun's spectrum is not constant, changing with regards to geographical location and time of day. Many sun beds emit more UVA radiation (wavelengths between 320 and 400 nm) and less visible light and heat than the sun, notably at high solar elevations.

In the present work, the variation of the solar spectrum with regards to geographical location and time of day; sun bed spectra in comparison with solar spectra; yields of vitamin D from sun and sun beds in comparison with yields from food; risk of cutaneous malignant melanoma (CMM) associated with sun and sun beds; and the prevention of internal cancers and improvement of prognosis associated with sun and sun bed-induced vitamin D, are all discussed.

### Variation of the Solar Spectrum with Geographical Location and Time

Figure 1 shows the spectrum of the sun at noon, midsummer in Oslo, Norway (60 degrees North), at the equator and in Melbourne, Australia (37 degrees South). Only the UVB ( $\lambda=280-320$  nm) and UVA ( $\lambda=320-400$  nm) regions are

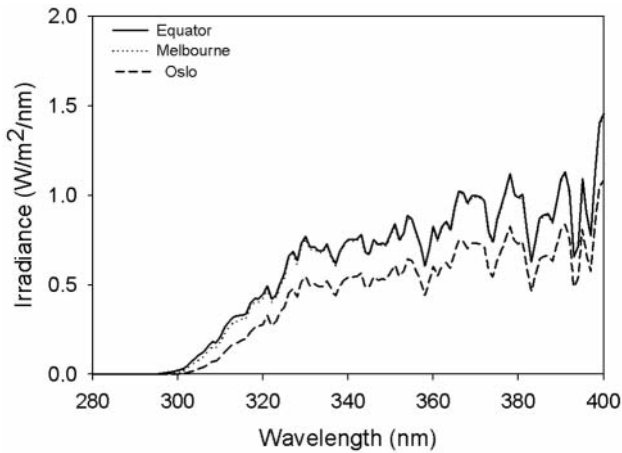


Figure 1. Solar spectra at noon, midsummer in Oslo, Melbourne and the equator.

shown. Note the small differences in the UVA region between the equator and Melbourne (less than 0.01 W/m<sup>2</sup>/nm). The explanation is the relatively small difference in solar elevation between Melbourne and the equator, 76 and 90 degrees, respectively. In the UVB region the differences are larger than in the UVA region. However, the ozone level also plays some role in this difference. It has been previously shown that variations of the cloud cover play a much larger role than typical ozone variations for the annual doses of carcinogenic UV radiation in Southern Norway (4). Ozone depletion from anthropogenic sources is no longer a threat, as it was in the 1990s. Daily and monthly variations of UVA and UVB (chosen wavelengths for comparison being  $\lambda=360$  nm and 300 nm for UVA and UVB, respectively) throughout a year can be roughly estimated by considering the dependency of fluence rates on solar elevations, similar to those shown for a clear day in Figure 2. It can be seen that the UVA/UVB ratio increases strongly with decreasing elevation. This is due to the fact that UVB, but not UVA, is absorbed by the ozone layer. This means that at high latitudes the ratio is minimal in the summer and maximal in the winter. Furthermore, there is relatively more UVA in solar radiation in the afternoon than at midday. This may have large consequences for recommendations of sun exposure, since vitamin D is exclusively produced by UVB, while CMM may probably also be caused by UVA (5).

Snow increases the fluence rate of UV radiation. The increase is strongly dependent on cloud cover, being much larger for cloudy than for clear days. Furthermore, it is larger for vertical than for horizontal surfaces. Thus, the April dose of UV in Scandinavia on a vertical surface, such as the face, is 50-100% larger if the ground is snow-covered than if it is bare (6).

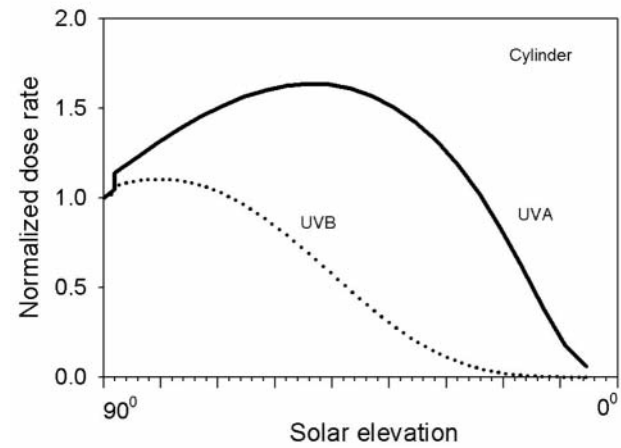
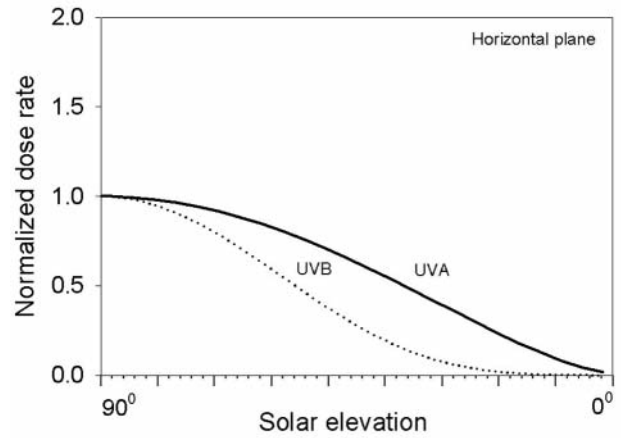


Figure 2. The variation of UVB (300 nm) and UVA (360 nm) with solar elevation, on a horizontal plane and on a vertical cylinder.

### Sun Bed Spectra in Comparison with Solar Spectra

Figure 3 shows two typical sun bed spectra in comparison with the sun spectrum at noon at the equator. Typically, the UVA/UVB ratio in a sun bed is about 50, which is similar to the ratio in solar radiation at a solar elevation of 45 degrees (Figure 2). Thus, around midsummer in Oslo, the UVA/UVB ratio of solar radiation four hours after noon is similar to that of a typical sun bed (7).

Sun beds were originally made for tanning. The action spectrum (the wavelength dependence of the efficiency of UV radiation in producing a given effect) for permanent tanning of human skin is similar to the erythema action spectrum (8), and to the vitamin D action spectrum, *i.e.* to the absorption spectrum of 7-dehydrocholesterol (9), which is localized in the wavelength region 290-315 nm. Earlier it was assumed that the action spectra of all skin cancer forms, including CMM, were similar to the erythema spectrum. This

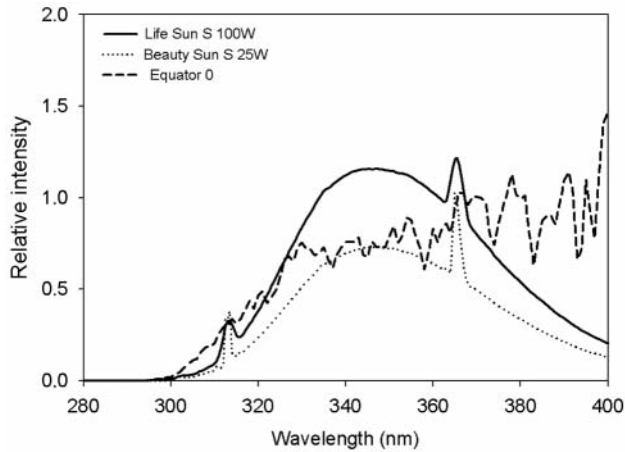


Figure 3. Spectra of two typical sun beds compared with the spectrum of zenith sun at the equator.

led to regulations demanding a low UVB emission of sun beds and thus, most sun beds emit more UVA than UVB radiation. However, it is the small UVB fraction that gives the main biological effect (vitamin D formation). The main effect of UVA in sun beds is to generate immediate pigment darkening, which has a UVA-weighted action spectrum (10). However, epidemiological and experimental studies have associated CMM risk with UVA exposure (5, 11-12), so the regulations should be reconsidered.

### Yields of Vitamin D from Sun, Sun Beds and Food

A minimum erythema dose (1 MED) amounts to about 30 minutes midsummer midday sun exposure in Oslo ( $=20 \text{ mJ/cm}^2$ ). Such an exposure given to the whole skin surface is equivalent to taking between 10,000 and 20,000 IU vitamin D orally, *i.e.* as much as 250-500  $\mu\text{g}$  vitamin D, a similar amount as that obtained when consuming 200-375 mL of cod liver oil (13). Clearly, the sun is an efficient vitamin D producer. Exposure to 1 MED from a sun bed (approximately 20 minutes) would give a similar amount of vitamin D. Several studies have reported that sun bed users get significant amounts of vitamin D from their exposures (14, 15). One study (Figure 4) showed that an exposure scheme with 0.5 MED given twice a week over five weeks in winter gave an increase in serum 25(OH)D of about 40%, *i.e.* a similar increase as seen from late winter to late summer (15). This “summer” level decayed back to “winter” levels within 6 to 8 weeks after termination of the sun bed sessions. Surprisingly, the recommended intake of 200 IU of vitamin D played no role in maintaining vitamin D levels (Figure 4). This is in agreement with other reports showing that 1,500 IU per day are needed to improve the vitamin D status (16).

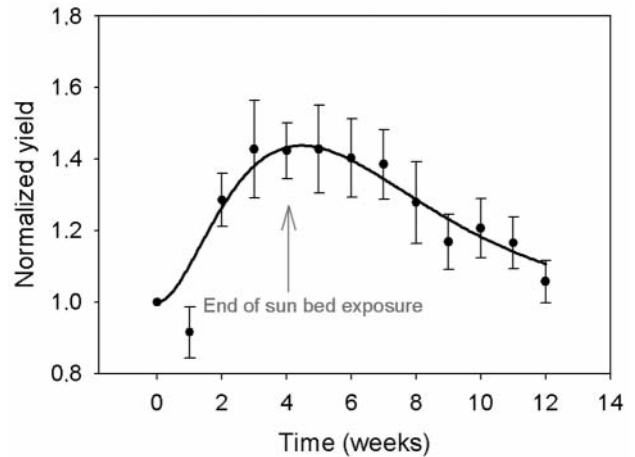


Figure 4. Increase in serum 25(OH)D levels after 10 sun bed exposure at sub-erythemal doses.

It would be perfectly possible to provide sun beds with much better properties with respect to vitamin D photosynthesis than those available today. Solar radiation, and presumably also current sun beds, can convert only 10-15% of the 7-dehydrocholesterol in skin to pre-vitamin D (17). This is due to the facts that back reactions occur, and that different intermediates in vitamin D photosynthesis have different action spectra and different sensitivities of photodegradation (18). An optimal sun bed for vitamin D photosynthesis in human skin should have a peak emission at about 295 nm (18). Such a sun bed would convert about 65% of the 7-dehydrocholesterol in skin to pre-vitamin D, and additionally, it would give good permanent tanning. The small exposures needed for this would probably not increase the photocarcinogenic risk significantly.

### CMM Risk Associated with Sun and Sun Beds

Based on epidemiological investigations, it can be concluded that solar radiation is the main cause of CMM in caucasians, even in a country at a high latitude (4, 19). The distribution pattern of CMM on different body localizations provides the main argument. However, this pattern changes with time, and for younger cohorts the relative CMM density (RTD=relative incidence rate on a given body localization divided by the skin area of that localization) is larger on the back than on the head/neck (4, 20). For older generations, RTD is always largest for the head and neck. This led to the conclusion that intermittent exposures (such as those most people get on the trunk) is more CMM-generating than constant or regular exposures (such those on the head/neck). Most investigations support this (21).

Table I. Relative risks of CMM of sun bed users and non-users.

| Reference             | Country (period)                     | Study design (population size) | Results (users vs. non-users of sun beds or sunlamps) <sup>a,b</sup> |
|-----------------------|--------------------------------------|--------------------------------|--|
| Adam 1981 (38)        | UK (1971-1976)                       | Case-control (169-207)         | OR=2.93 (1.16-7.40)  |
| Holman 1986 (39)      | Australia                            | Case-control (511-511)         | OR=1.1 (0.6-1.8)   |
| Osterlind 1988 (40)   | Denmark (1982-1985)                  | Case-control (474-926)         | OR <sub>artificial UV</sub> =0.7 (0.5-1.0)                           |
| Swerdlow 1988 (41)    | UK (1979-1984)                       | Case-control (180-197)         | OR <sub>sunbeds+sunlamps</sub> =2.9 (1.3-6.4)                        |
| MacKie 1989 (42)      | UK (1987)                            | Case-control (280-180)         | RR=1.3 (0.2-7.9) M   |
| Garbe 1993 (43)       | Germany (1984-1987)                  | Case control (856-705)         | RR=1.2 (0.5-3.0) F (artificial UV sources)                           |
| Dune-Lane 1993 (44)   | UK                                   | Case-control (100-100)         | RR <sub>sunbeds</sub> =1.5 (0.9-2.4)                                 |
| Autier 1994 (45)      | Germany, Belgium, France (1991-1993) | Case-control (420-447)         | OR=1.16 (0.54-2.47)  |
| Westerdahl 1994 (46)  | Sweden (1988-1990)                   | Case-control (400-640)         | OR <sub>sunbeds+sunlamps</sub> =1.16 (0.83-1.61)                     |
| Holly 1995 (47)       | USA – San Francisco (1981-1986)      | Case-control (452-930)         | OR <sub>sunbeds or sunlamps</sub> =1.3 (0.9-1.8)                     |
| Chen 1998 (48)        | USA – Connecticut (1987-1989)        | Case-control (624-512)         | OR <sub>&lt;30 yr</sub> =2.7 (0.7-9.8)                               |
| Walter 1999 (49)      | Canada (1984-1986)                   | Case-control (583-608)         | OR <sub>sunlamps</sub> =0.94 (0.74-1.2)                              |
| Westerdahl 2000 (50)  | Sweden (1995-1997)                   | Case-control (571-913)         | OR <sub>sunbeds+sunlamps</sub> =1.13 (0.82-1.54)                     |
| Naldi 2000 (51)       | Italy (1992-1995)                    | Case-control (542-538)         | OR <sub>sunbeds</sub> =1.54 (1.16-2.05)                              |
| Kaskel 2001(52)       | Germany (1996-1997)                  | Case-control (271-271)         | OR <sub>sunbeds</sub> =1.2 (0.9-1.6)                                 |
| Veierod 2003 (53)     | Sweden, Norway                       | Female cohort (106,379)        | OR <sub>sunlamps</sub> =1.10 (0.50-2.39)                             |
| Bataille 2004 (54)    | UK (1989-1993)                       | Case-control (413-416)         | OR <sub>sunbeds + sunlamps</sub> =0.78 (0.45-1.37)                   |
| Bataille 2005 (55)    | 5 European countries (1998-2001)     | Case-control (597-622)         | OR <sub>artificial UV</sub> =1.00 (0.6-1.8)                          |
| Ting 2007 (56)        | USA - Iowa                           | Cohort (1518)                  | >5 per year vs. 5 per year   |
| Faurshou 2007 (57)    | Denmark (1977-1989, 1990-2002)       | Ecological                     | OR <sub>sunbeds</sub> =1.55 (1.04-2.32)                              |
| Clough-Gorr 2008 (58) | USA (1995-1998)                      | Case-control (423-678)         | OR <sub>sunbeds</sub> =1.19 (0.84-1.68), p=0.33                      |
|                       |                                      |                                | OR <sub>sunbeds</sub> =0.90 (0.71-1.14)                              |
|                       |                                      |                                | OR <sub>first used&lt;15 yr</sub> =1.82 (0.92-3.62)                  |
|                       |                                      |                                | OR <sub>sunbeds</sub> =1.65 (1.01-2.67)                              |
|                       |                                      |                                | OR <sub>first used&lt;45 yr</sub> =3.22 (1.01-11.46)                 |
|                       |                                      |                                | No correlation found   |
|                       |                                      |                                | OR <sub>sunlamps</sub> =1.39 (1.00-1.96)                             |
|                       |                                      |                                | OR <sub>sunbeds</sub> =1.14 (0.80-1.61)                              |
|                       |                                      |                                | OR <sub>sunlamps +sunbeds</sub> =1.96 (1.06-3.61)                    |

<sup>a</sup>95% CIs are given in parenthesis after the main OR; <sup>b</sup>M, males and F females.

Table I, which is a summary of literature data on odds ratio of CMM incidence rates of sun bed users and non-users, indicates that no firm conclusion can be drawn. However, overall there seems to be a slight increase in the risk associated with sun bed use.

Similarly, sunscreen use has, thus far, not shown any major impact on CMM risk, as far as literature data are concerned. Some studies showed no increase in risk of CMM among sunscreen users (22, 23), some more recent studies argue for a protective effect of modern sunscreens (24), while other studies found an increased risk (25), especially for sunscreens that absorb only UVB and in populations at high latitudes (26).

An important finding to keep in mind is that the progression of CMM seems to be slower for lesions localized on skin that has large UV exposure (sun exposure is associated with increased survival from CMM (27)). Furthermore, survival from melanoma depends on the season of diagnosis: the mortality rates of melanomas diagnosed in summer are lower than those diagnosed in the winter (28).

This apparent protective effect of sun exposure may be due to vitamin D formation.

### Sun Exposure and Internal Cancers

The scientific interest in this topic was ignited by observations of higher incidence rates of a number of internal cancer forms in geographic locations of low annual UV fluences (29-31). Later, a number of studies, including cell studies, animal studies and human interventional studies, supported and indicated that incidence is likely to be related to the production of vitamin D by solar radiation (32, 33).

Whilst investigating north-south gradients of cancer incidence and mortality in Norway, which spans over more than 10 degrees, with annual UVB fluencies 50% larger in the south than in the north, no differences were found. This is probably due to the fact that people in the north consume more vitamin D than people in the south, and that this higher intake compensates for the low UV exposure in the north.



Knowing that there is a seasonal variation of vitamin D status in Norway as in practically all countries at latitudes above 30 degrees (34), seasonal variations of internal cancer incidence and progression were investigated (35-37). No seasonal variation of incidence rates was found, indicating no seasonal variation of cancer awareness. However, with a few exceptions, a seasonal variation of prognosis was observed. Cancers diagnosed in the summer and autumn had a significantly better prognosis than cancers diagnosis in the winter and spring. This was tentatively attributed to sun-induced vitamin D. It seems that vitamin D mainly affects tumor progression, so that it acts synergistically with standard cancer treatments. The fact that the best time point for therapy commencement appears to be after the time point of maximal 25(OH)D in serum (35) may be related to the time it takes for 25(OH)D to be converted to active metabolites and to be transported to the tumors.

### Acknowledgements

We acknowledge the Norwegian Cancer Association and The Research Foundation of The Norwegian Radium Hospital for financially supporting this work. We thank Kristian P. Nielsen for providing us with the sun bed spectra.

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Received January 29, 2009

Revised May 26, 2009

Accepted June 9, 2009