Sun beds and sunscreens - friends or foes?

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Abstract

When absorbed, a photon of a given energy has the same chemical and biological effect whether it comes from the sun or from an artificial, man-made source like a sun bed. If the spectrum and intensity of a sun bed are the same as those of the sun, the biological effects will be exactly the same. The main difference between radiation from sun and sun beds is that sun beds emit less visible light. This may affect the relative efficiency of photoreactivation, a process through which ultraviolet (UV) -induced DNA damage is repaired. Sun creams do not only reduce the intensity of the radiation reaching living skin tissue, but also changes the spectrum of the radiation, since they are mainly designed to reduce erythemogenesis, which is induced mainly by UVB radiation (280-320 nm). However, other biological effects, such as generation of melanomas, skin ageing and photosensitization associated with endogenous and exogenous dyes, may be caused also by UVA radiation (320-400nm) and even by visible light.

Solar UV radiation is the main source of vitamin D in humans, and most sun beds are equally efficient vitamin D producers. However, both sun and sun beds can cause skin cancer. Notably, intense, intermittent exposures, yielding sunburns, are carcinogenic, while more regular, moderate exposures are less so. The positive effect of such exposures, mainly related to vitamin D photosynthesis, may be much more important than the negative effects. Considerations of skin colours of populations living at different latitudes, as well as modern experimental and epidemiological work, clearly urge us to focus more on the positive effects of UV radiation from sun and sun beds.

The spectral region related to a given biological effect is given by the action spectrum of that particular effect. The action spectra of erythema, melanogenesis
and non-melanoma skin carcinogenesis are very similar and are all centred in the UVB region. Thus, it is the total exposure and the exposure pattern that determine which of the three effects is the dominating one. Sun creams are made to reduce or eliminate erythema and carcinogenesis risks. Then it is evident that they also reduce or eliminate vitamin D production, which has been experimentally proven. Reports in the literature on the effect of sun beds and sun creams on the risk of skin cancer are conflicting: Positive as well as negative effects have been demonstrated. Thus, so far no firm conclusions can be drawn with respect to the overall and dominating impact.

Health authorities frequently recommend that people should avoid midday sun and postpone their sun exposure to the afternoon. This is probably not the best recommendation to make, since moderate midday sun exposure gives maximal amounts of vitamin D at minimal melanoma risks.

**Introduction**

Sun beds are lamps constructed with the aim to emit radiation giving similar effects in human skin as solar radiation does. Sunscreens are made to play an opposite role: to reduce or eliminate the effect of ultraviolet radiation (UV) from the sun on human skin. Since the use of both sun beds and sunscreens has increased over many decades, their health effects should be continuously and critically investigated. Before attempting to answer the questions in the title, we will briefly review what is known about positive and negative effects of solar radiation on humans.

Any discussion on this topic should be based on action spectra of the biological effects. Such spectra show the wavelength dependency of the effects, and are almost inevitably needed whenever different radiation sources are to be compared with respect to biological effects. This is so because practically all radiation sources have different or varying spectra: the spectrum of the sun changes with solar elevation, ozone layer thickness, latitude, altitude, cloud cover and ground albedo. The spectra of sun beds are also widely different and change with hours of use. UVB ($\lambda = 280-320$ nm) is 10-1000 times more biologically active than UVA ($\lambda = 320-400$ nm), depending on the wavelengths chosen for comparison. In fact, the division between UVB and UVA is usually set at 320 nm, because DNA absorbs practically no radiation with longer wavelengths than 320nm, and DNA is thought to be the main chromophore for adverse effects in human skin. Other important chromophores, such as urocanic acid and 7-dehydrocholesterol, have similar spectra.

Two basic and undeniable facts should be kept in mind: Humans have developed and lived in solar radiation throughout their entire history, most of the time
without sunscreens and even without clothes. Their first home place is believed to be under the Equator, in Africa, where the solar radiation was, and still is, very strong. Those living under high fluences of solar radiation (i.e. close to the Equator) have dark skin; skin containing the natural, brown sunscreen melanin. Whether some forms of melanin, pheomelanins, can also act as photo sensitizers has to be considered and further studied. As humans moved away from the Equatorial areas and towards less sunny places, their skin gradually turned white, in fact whiter the further away from the tropics they live and the longer they have stayed there.

**Negative health effects of solar radiation**

**Skin cancer**
The most important adverse effect of exposure to solar radiation is induction of skin cancer. There are three major forms of skin cancer: squamous cell carcinoma (SCC) arising mainly in keratinocytes in the epidermis, basal cell carcinoma (BCC) arising in dividing, basal cells on the border between the epidermis and the dermis, and cutaneous malignant melanoma (CMM) arising in melanocytes residing close to the basal layer. In Norway there are now about 9000 BCCs, 1300 SCCs and 1100 CMMs diagnosed each year (1). The survival rates of SCC and BCC are higher than 97%, while that of CMM is much lower. The five year survival rates of CMM have increased from about 50% and 60% in 1958 to about 78% and 90% in 2000 in men and women, respectively (1), supposedly due to earlier diagnosis and improved treatment. Nevertheless, about 260 Norwegians die from CMM every year, amounting to about 2.5% of the total number of total cancer deaths. About 4.7% of new cancer cases are CMMs (1).

Until about 1990 the incidence rates of all skin cancer forms increased with a doubling time of about 15 years (2). After 1990, however, the incidence rates of CMM have flattened out, and for younger people even decreased (see below). Solar radiation is the main cause of SCC and BCC. We have estimated that even in the Nordic countries more than 80% of the incidences are caused by the sun: Latitude variations and body localization patterns constitute the basis for this conclusion (2). The incidence rates are about three times higher in the south of Norway than in the north, where the annual fluence of UVB is 50% lower. The relative tumor density of SCC and BCC (here defined as the annual incidence rate divided by the skin area under investigation) is much higher on head and neck (heavily exposed areas) than on trunk and limbs. This pattern has not changed significantly over time. Although it has been claimed that intermittent exposure, leading to sunburn, is especially carcinogenic for all three skin cancer forms (3,4), most authors are of the opinion that the risk of SCC increases with accumulated dose (5,6). Mice get SCCs from UVB exposure, and experiments
with these animals generally support that the rates increase with accumulated
dose (7). However, mice get neither BCCs nor CMMs (8). The dose dependency
of BCC generation is less clear than that of SCC, but also in this case the
accumulated dose seems to be important. An intermittent exposure pattern may
be more BCC generating than a more constant one. Several reports indicate that
CMM generation is mainly related to intermittent exposure and sunburns (9,10).

The relationship between CMM and sun exposure is extremely complicated,
and, therefore, vigorously debated. We have earlier listed the main arguments
for and against a relationship (11,12). Our work indicates that intermittent sun
exposure has a high CMM-generating potential (2). In agreement with this, a
large review (13) of published data on CMM (29 studies on sun exposure and 21
on sunburn) indicates a significant, positive association (OR 1.71, 95 % CI:
1.54-1.9) between CMM and intermittent exposure and a reduced risk for heavy
occupational exposure (OR 0.86, 95% CI 0.77-0.96). Sunburn was found to be a
risk factor for all ages. This is in agreement with the early statement of Holman
et al. (14) that when the total sun exposure increases, the CMM risk first
increases, reaches a plateau and then decreases with further exposure. Thus,
farmers and fishermen, supposedly heavily exposed without sunscreens in all
seasons, have a surprisingly low chance to get CMM compared with white collar
workers. It is tempting to propose that a constant sun induced tan or skin
thickening acts protectively. That the exposure pattern is of great importance is
evident from our studies of relative tumor densities of CMM on different body
localizations: For younger women the relative tumor density on the lower limbs
is higher than that on the face and younger men get more CMMs per cm² on the
back than on the much more exposed face and neck (2). A rather strong
indication that sun exposure is a main CMM-causing factor is that practically no
Norwegian women got CMMs on the breasts before the top-less fashion came
(2,12).

Many scientists find it surprising that, as we have moved from agricultural
occupations to more office work, the CMM incidence rates have increased. This
may support the role of intermittent, vacational UV exposures as particularly
melanomagenic factors. Similarly, people working under fluorescent light tubes
have an increased CMM risk (15,16). This may simply be related to indoor
versus outdoor work, since such tubes emit very low fluence rates of UV.

**Skin ageing** (17,18)

It is well known and proven that UVB exposure leads to accelerated elastosis
and skin ageing. UVA seems to act similarly, and, related to its larger
penetration depth, gives deeper effects and acts more on dermal structures.
Eye damage (19-21)
Photokeratitis (snow blindness) and cataract formation are accelerated by UVB exposure. The former is a transient damage to corneal cells, while the latter is mainly persistent cross linking, aggregation and “whitening” of lens proteins.

Immunological impairments (22,23)
This is a controversial topic: On one hand UVB reduces the number of melanocytes and Langerhans cells and makes mouse skin unable to reject transplanted skin tumors, but newer research shows that moderate UV exposures stimulates the immune system in skin in a positive way (24).

Positive health effects of solar radiation

Immune effects
The positive effects of moderate UV exposures on the immune system may largely be related to vitamin D formation. As long as this photoproduct is retained in the skin, it certainly acts immunomodulatory in a positive way (24). Recent research indicates that this is the biological basis for the positive effect of UV on skin tuberculosis (lupus vulgaris) (Finsen’s Nobel price in 1903) and the effect of sun-sanatory treatment of tuberculosis before the time of BCG vaccination (25-28). The balance of positive versus negative immune effects of UVB is probably related to dose- and exposure patterns. The reason why practically no influenza epidemies start in the summer season may, in addition to direct vitamin D effects, be caused by more general immune effects generated by solar radiation in skin (29). Before about 1930 positive health effects of solar radiation were strongly focused on. An editorial in The Lancet in 1932 expressed that the health effects of sun exposure were so large and important that governments should consider arranging areas for nude sunbathing! (30). Then, the medical societies and governments realized that solar radiation caused skin cancer, and the anti-sun campaigns slowly gained power and momentum. Peller’s observation that “skin cancer appeared to protect against other cancers”, published in The Lancet in 1936 (31), did not help much in creating a balanced view. In fact, Peller was wrong; it is not the skin cancer itself that acts protective, but the sun exposure through its vitamin D generation.

Positive effects related to vitamin D photosynthesis
Many of the following positive effects are related to interactions of vitamin D with the immune system. Most researchers in the field state that “solar radiation is the most important source of vitamin D for humans” (see Hollick M.F., Zittermann A., Reichrath J. in this book). Large population groups have an inadequate vitamin D status, primarily caused by lack of outdoor activities and sun-shielding by clothes or creams. What an “inadequate vitamin D status” really means is being debated by nutritionists, dermatologists and photo -
biologists. It seems clear that “recommended intake doses”, as well as values for “optimal calcidiol levels” in serum, should be substantially increased (see other chapters in this book). The fear of “vitamin D intoxication” may be extremely exaggerated.

Documented health effects of an optimal vitamin D status are numerous, and only an incomprehensive list will be given here: protection against a number of internal cancers (32-35), improved prognosis of a number of cancers (36-42), prevention of rickets, prevention of osteoporosis (the increase of incidence of this disorder may have two reasons: inadequate vitamin D intake, and increased protein intake which leads to increased calcium excretion (43)), prevention of osteomalacia (soft bone, chronic pain), reduction of risk of arthritis (44,45), reduction of development of muscle weakness and undefined chronic pain (46-51) control of psoriasis and eczema (52), reduction of hypertension, high blood cholesterol and cardiovascular diseases (53-55), reduction of diabetes type 2 incidence rates (56-58) and prevention of influenza epidemics (29). Sun exposure is beneficial also for autoimmune diseases: diabetes type 1, multiple sclerosis (see Kampmann M, chapter in this book) and rheumatoid arthritis (44,45). Finally, there are more or less well founded speculations concerning obesity, Parkinson’s disease, fibromyalgia, and Crohn’s disease (59-62).

**Effects on circadian rhythms**

This topic is treated in two other chapters of this book, and we will just mention that the “light governed”, or, more correctly stated, the “darkness-generated” hormone melatonin is interacting with many other hormonal systems, some of which are closely linked to cancer progression. Therefore, it has been proposed that solar radiation may play a role in cancer progression also through its interaction with circadian mechanisms (63). Night-working persons are more prone to get cancer than day-working people, an observation that may be related to effects of the visible part of solar radiation on hormonal systems, via receptors in the eye and signals to the pineal organ via the suprachiasmatic nuclei.

**Action spectra**

Since UVB is 10-1000 times more biologically efficient than UVA per incident photon, and since there is much more UVA than UVB in solar radiation, action spectra need to be known with great accuracy in the UVA range. For instance, so called UVA sun beds emit very little UVB, but it is still this small fraction that causes erythema, melanogenesis and vitamin D synthesis (64). For humans only three relevant action spectra are known with accuracy: those for erythema generation (65), melanogenesis (66) and vitamin D photosynthesis (67) (Figure 1). Those for elastosis, (68), immediate pigment darkening (IPD) (69) and
photoisomerization of urocanic acid are known with some accuracy (70). Concerning photo carcinogenesis, the action spectrum of SCC in mice (7) and that of CMM in a fish called Xiphophorus (71) are known. As shown in figure 1, the spectra of erythema, melanogenesis and SCC are very similar. The chromophore in the UVB range is believed to be DNA, since the spectra are almost identical with the absorption spectrum of DNA when the latter is corrected for scattering and absorption in the epidermis. DNA does not absorb UVA, so another chromophore must be acting in this wavelength region. One possibility might be porphyrins, although they are present only in trace amounts, so the question is unsolved. The urocanic acid isomerization spectrum is similar to the two above mentioned spectra in the UVB range, making it hard to decide whether DNA or urocanic acid is the main chromophore for immune effects. The vitamin D action spectrum is also similar to the mentioned spectra but lacks the UVA peak. It has been measured in human skin up to about 310 nm, but can also be calculated from the absorption spectrum of 7-dehydrocholesterol, the chromophore for photosynthesis of the vitamin D precursor previtamin D. Using known absorption and scattering characteristics of epidermis and assuming that the most important site of formation of vitamin D is the lower epidermis, one gets a calculated action spectrum for vitamin D formation which is blue-shifted compared with the measured spectrum. This may be due to competing photoreactions of previtamin D and vitamin D. In any case, the long-wavelength part (>300 nm) of the spectrum is not known with sufficient accuracy to allow exact comparisons of the vitamin D generating potential of UVA sources.

Figure 1. Action spectra for erythema (MED), melanogenesis, CMM in Xiphophorus, SCC in mice, immediate pigment darkening (IPD). That for vitamin D generation is similar to the SCC spectrum, but lacks the UVA peak.
although one can be almost sure that UVA alone produces negligible amounts of vitamin D. The elastosis spectrum is also similar to the erythema spectrum, while the IPD spectrum has a much larger UVA contribution than the other discussed spectra. The CMM spectrum resembles the IPD spectrum (Figure 1) and has also a surprisingly large UVA and even a visible light contribution, much larger than that of the SCC spectrum. This has been assumed to be caused by melanin absorption, since it is known that pheomelanin is photoactive and can generate free radicals upon light or UV exposure (72). We have proposed that melanin inside melanosomes acts as a photo carcinogen, while melanin in the upper skin layers acts as a sunscreen; the radicals formed there being unable to reach living and dividing cells during their lifetime (72).

Conclusions from spectral analysis

If the fish CMM spectrum is relevant for humans, large consequences will follow: UVA sun beds are more, rather than less, dangerous than UVB-weighted sun beds, and sunscreens, most of which have a much stronger absorption in UVB than in UVA, can efficiently prevent sunburn and vitamin D formation but not CMM induction. The inefficiency of sunscreens in CMM prevention (see below) may indicate that UVA plays a significant role in CMM generation. The latitude dose gradient is steeper for UVB than for UVA due to the fact that the ozone layer absorbs UVB, but not UVA (72). For instance, the annual UVB dose in South Australia is almost a factor of three larger than the corresponding dose in South Norway, while the annual UVA dose is only about 30% larger in Australia (72). This gives us an opportunity to evaluate the role of UVA and UVB for SCC, BCC and CMM on the basis of epidemiological data for Norway and Australia. The incidence rates of SCC and BCC have almost identical latitude gradients, and are 10-20 times more frequent in Australia than in Norway, while CMM is only a factor 1.7 more frequent in Australia than in Norway (72). This strongly indicates that UVA plays a larger role for CMM than for the non-melanoma skin cancers in humans. Furthermore, it suggests that the action spectra for BCC and SCC are similar, i.e. mainly UVB weighted, just as the spectra for erythema and vitamin D photosynthesis are.

Since UVB is almost absent from late September to early March in North Europe, including UK, there is no sunburn risk and no vitamin D formation in this period. This is supported by experiments (73). Similarly, during a summer day the UVA/UVB ratio is smallest around noon. Thus, if one wants to get maximally of vitamin D at a minimal CMM risk, midday midsummer is the best time for sun exposure. The general recommendation to avoid midday sun and rather sunbathe in the afternoon may be totally wrong! Of course, one should always limit the exposure time to avoid erythema.
The action spectra tell that as skin gets thicker and more pigmented during the summer season and the melanin moves upwards in the skin, the erythema risk, as well as the efficiency of vitamin D photosynthesis, decreases. The CMM risk, however, will not decrease to the same extent, and neither does immediate pigment darkening.

Of importance for our topic is that all sunscreens that reduce erythema and non-melanoma skin cancer risk, also reduce vitamin D formation and melanogenesis. One might construct a sun bed with a UVA spectrum giving pigmentation, at least IPD, but the erythema risk would exist and the CMM risk would probably be very large. However, people with naturally dark skin (skin types IV-VI) might benefit slightly from such UVA sun beds, as long as the CMM risk is not taken into account (74).

**Sunscreens - epidemiological and clinical investigations**

Practically all of the following results can be predicted by the spectral evaluations above. Since these two approaches, epidemiological investigations and spectral evaluations, are so different in nature, agreements between them should be given strong emphasis.

Sunscreens, properly applied, practically eliminate vitamin D photosynthesis in skin (75,76). It may be true, however, that they are rarely applied in recommended amounts (2 mg/cm²), and that they, therefore, allow some vitamin D photosynthesis. However, most sunscreen campaigns strongly stress that “correct amounts of high SPF (≥15) should be applied, and frequently reapplied, during sun exposure”.

Such campaigns have led to an enormous increase of sunscreen sale: In USA the sale increased from 18 million dollars in 1972 to 640 million dollars in 2005 (77). In spite of this, the CMM incidence rate among Caucasians in USA has increased from 6.3/100 000 in 1973 to 16.5/100 000 in 1997 (78). In Norway the CMM rate increased from about 6/100 000 in 1970 to about 16/100 000 in 1990 (1).

Several reviews and meta-analyses of epidemiological investigations on the relationship between sunscreen use and CMM have been published (79-84). Most of these analyses conclude that no protective role of sunscreens can be demonstrated. This is true for the first analysis (from 1970ies) as well as for those carried out after 2000, so improvements of the sunscreens have not been documented so far. Many of the individual studies show that those applying sunscreens regularly had significantly higher risks of CMM than others (84,85). Several hypotheses for this have been discussed (85). The most frequently
mentioned one is that sunscreen use may tempt people to stay longer in the sun and thus get large UVA doses to the deeper part of their skin. Autier et al (80) showed that use of sunscreens increased the duration of recreational sun exposure of young Europeans. Another hypothesis is that the phenomenon is due to the fact that sunscreens reduce vitamin D photosynthesis in skin (75,76), and that sunscreen users have lower calcidiol levels than others and also lower bone mass density.

As with CMM, BCC incidence rates, and even nevi counts, seem to be higher rather than lower among sunscreen users (75,76,86-91). However, sunscreen use seems to decrease the risk of actinic keratosis (a SCC precursor) and SCC (92,93). Some sunscreens contain ingredients that penetrate into the circulation, notably that of small children with thin skin (94-96). Aromatic compounds might, in principle, be carcinogenic, and should be thoroughly checked before launched for human use that might involve systemic effects. Sun-induced mutagenicity of a common sunscreen ingredient has been clearly demonstrated (97). The physical sunscreens (TiO$_2$, ZnO and FeO$_2$) were introduced because they were thought to be chemically inert and just scatter UV radiation. However, they are made in small particles (100-400nm) and may penetrate the stratum corneum. As long as they are outside living skin, their scattering properties are beneficial. However, inside skin they may act photo-catalytically and even lead to increased space irradiance of UV in certain skin layers. Some sunscreen products inhibit inducible nitric oxide synthesis (98). UV induced erythema is partly due to the synthesis of NO in the skin. NO is also an important regulator in the induction of cell-mediated immune responses in skin. NO synthesis in macrophages seems to delay progression of UV induced mouse tumours (99). In correspondence with this, some sunscreens are made to act even when applied after UV exposure (see references in (98)). It should also be kept in mind that sunscreens seem to reduce radical formation in skin much less efficiently than they reduce erythema (100). This might be important in the case of CMM, for which UVA seems to be important, since UVA is thought to act partly via radical formation. Psoralen-containing suncreams are quite efficient in reducing erythema without reducing pigmentation (79). UVB filters seemed to inhibit the carcinogenic properties of psoralens, and it was proposes that such creams might be a good alternative for people who wanted cancer protection and at the same time a good tan (101,102). However, according to the review of Autier et al (79) the assumed good properties of psoralen containing sunscreens have not been epidemiologically proven.

Sunscreens seem to protect against photoimmunosuppression (103), although the immune protection factor is less than the erythema protection factor (103,104). This may indicate that UVA is involved in the immunosupression. Growth of CMMs transplanted to UV exposed mice is believed to be caused by immunosuppression. Sunscreens fail to protect well against such growth (105).
Benign melanocytic lesions, so called acquired nevi, increase in number with age, and are to a large extent caused by UV exposures, notably intermittent, burning ones. Such lesions are believed to be strong risk factors for CMM (106). Oppositely, congenital nevi, which are rare, seem to be markers for persons with decreased CMM risk (107). Since acquired nevi are associated with CMM in 50% of the cases, one might suggest that such nevi are CMM precursors (108-110). Two important arguments for sunscreens are that they appear to reduce development of acquired nevi (111) and inhibit mutations (in p53) which are believed to be involved in skin carcinogenesis (112).

Sun beds

**UVA and UVB sun beds** (See references in (113))

UVA acts deeper than UVB. UVB-tanning is dispersed throughout the epidermis, while UVA-tanning is principally confined to the basal layer. Some investigators believe that due to this, UVB provides a better protection against later UV-induced erythema than UVA. UVB-exposure leads to epidermal thickening. Both UVA and UVB alter the morphology of Langerhans cells and induce antigen presenting cells and suppressor T cells. On the other hand, there is suppression of natural killer cell activity. UVA induces immediate erythema and tanning, while UVB acts in a more delayed manner. Vascular occlusion prevents UVA-tanning, indicating that oxygen is needed in the initial step. This is probably due to the fact that DNA does not absorb UVA, which, therefore, acts through a radical or photosensitization process. A practical consequence of this is that skin in contact with the tanning bed, and therefore oxygen depleted, does not pigment significantly.

**Conclusions based on spectra**

There is nothing magic about artificial UV radiation, and no rational reason exists why radiation from such sources should be more dangerous than radiation from the sun. The effects totally depend on spectra and fluence rates. The fluence rate of UVB from sun beds is similar to, or slightly larger than that of UVB in solar radiation (114). It was believed that UVA gave a safer tanning than UVB, which was considered more carcinogenic. This was based on action spectra for melanogenesis in persons with dark skin of type V (74). Sun beds with rather large UVA fluence rates were then constructed. Thus, the UVA/UVB ratio is often larger for sun beds than for solar radiation at noon at low and mid latitudes (114). For low solar elevation, however, this ratio in solar radiation is large, even larger than that in sun beds. The magnitude of fluence rates may play a role for carcinogenesis. It seems that a UV dose given at a low fluence rate is more carcinogenic than the same dose given at a high fluence rate. This would be in favour of sun beds. A high UVA/UVB ratio, as found in newer sun beds and in solar radiation at low solar elevation, is disadvantageous.
if the fish CMM action spectrum applies to humans. There is also another argument for decreasing this ratio, i.e. for producing sun beds with relatively more UVB, namely the physical efficiency of vitamin D photosynthesis. The action spectrum for this effect is exclusively in the UVB region (67). More than that: Due to complex back reactions and photoproduct degradation, only 15-20% of the 7-dehydrocholesterol in human skin can be converted to previtamin D by solar radiation, while as much as 65% can be converted by semi-monochromatic radiation around 290 nm (67). Small doses of such radiation would give plenty of vitamin D and good tanning at a moderate CMM risk. The visible light contribution of a source is another important issue, since visible light is able to repair DNA damage through photoreactivation. The cyclobutyl pyrimidine dimers removed in photoreactivation are believed to represent an important step in photocarcinogenesis (115). Again, the issue is complex, since pyrimidine dimers are also involved in melanogenesis. UVA acts mainly through radical generation, and the damage caused by this component is probably not photorepairable. In conclusion, at the present stage of knowledge one can propose that an optimal sun bed would be one emitting radiation around 290 nm and visible light. This would give an optimal yield of vitamin D at a minimal CMM risk.

Clinical and epidemiological work
Sun beds are more frequently used in the Nordic countries than in Southern Europe (116). Experiments clearly show that moderate and nonerythemogenic sun bed exposures give large contributions of vitamin D to humans (117-119). We carried out such an experiment with 10 exposures below the individual MED (minimal erythema dose) from a commercial sun bed over four weeks, and found an increase of the calcidiol level from 65 nM to about 92 nM. The experiment was carried out in winter time with no vitamin D contribution from the sun. The vitamin D status at the end of the sun bed sessions was similar to that in summer and autumn. This level decayed to the pre-sun bed level in about 6-8 weeks. Surprisingly, intake of the recommended winter dose of vitamin D (200 IU/day) played no role: The decay of calcidiol was as fast in persons supplemented with vitamin D as in those taking no supplements. Our experiments seem to indicate that the recommended vitamin D intake is too small to keep a summer vitamin D level constant in the absence of vitamin D generating radiation.

Some epidemiological investigations indicate that sun beds are CMM-generating (120,121) and refs in (116,122-124)). There has been no variation in the odds ratio over time (116,123-125), so the introduction of UVA sun beds has played no major role. In Norway sun bed use has increased steeply since 1990 (data from the Norwegian sun bed Association) while the CMM incidence rates have decreased rather than increased since 1990 (11). Other investigations show no effect, or even a protective effect of sun beds (see references in (124,125)).
Several review papers and meta analysis of such data have been published (116,123-125). Overall, these analyses indicate a slightly elevated CMM risk for sun bed users, with an odds ratio of 1.1 – 1.5.

When other cancers are brought into consideration the conclusion would be opposite. Our work has shown that the relative survival of breast-, prostate-, colon-, lung cancer and Hodgkin lymphoma is larger for diagnosis and treatment start in the season of high calcidiol levels (summer and autumn) (37-39,41,42). If such high levels could be maintained throughout the year, a large increase in cancer survival would result (126).

Conclusions

Solar radiation, notably UVB, is likely to be the main cause of SCC. The same is probably true for BCC. For CMM, however, no firm conclusion can be drawn. Regular, moderate UV exposures, from sun or sun beds, may act protectively, at least with respect to survival. Intermittent exposures may be significantly CMM generating. The action spectra for BCC, SCC, erythema, elastosis and melanogenesis are similar, with a main peak coinciding with DNA absorption in UVB and a shoulder around 360 nm in UVA. The action spectrum for immediate pigment darkening is more UVA-weighted than the spectra mentioned above. This is also true for the action spectrum of CMM in the fish Xiphophorus. Epidemiological data indicate that this spectrum may apply also to humans, although this has not been proven.

Sun beds are likely to act similarly as the sun, although they have a higher UVA contribution than midday summer sun. Epidemiological investigations have given no conclusive answers as to the role of sun beds for humans, although recent meta-analyses indicate that they may increase the CMM risk slightly (odds ratio around 1.15).

Concerning sunscreens, the situation is even more confusing. In the worst case they may be photocarcinogenic and increase the CMM risk. Sun beds give good contributions of vitamin D, since practically all of them emit some UVB (>1%). Sunscreens, properly applied, eliminate vitamin D photosynthesis from any source, sun or sun beds.

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