CHAPTER

UV-RADIATION AND HEALTH
Optimal Time for Sun Exposure

Johan Moan,[1,2] Mantas Grigalavicius,[1] Arne Dahlback,[2]
Zivile Baturaite[1] and Asta Juzeniene*,[1]

[1]Institute for Cancer Research, the Norwegian Radium Hospital, Oslo University Hospital, Department of Radiation Biology, Oslo, Norway; [2]Institute of Physics, University of Oslo, Oslo, Norway
**Corresponding Author: Asta Juzeniene—Email: Asta.Juzeniene@rr-research.no*

Abstract: Positive as well as negative health effects of exposure of human skin to UV radiation depend on spectra and fluence rates, both of which being dependent on latitude, time of the day and several other factors. The major positive effects are related to vitamin D photosynthesis and the major negative effect is skin cancer development. The action spectra for these effects are different. This lead us to conclude that for optimal vitamin D synthesis at minimal risk of cutaneous malignant melanoma (CMM), the best time for sun exposure is between 10 a.m. and 1 p.m. Thus, the common health recommendation (that sun exposure should be avoided between the hours of 10 a.m. and 4 p.m. and postponed to the afternoon) may be wrong.

INTRODUCTION

Both UV radiation A (UVA, 315–400 nm) and UV radiation B (UVB, 280–315 nm) are involved in melanomagenesis, while only UVB gives vitamin D. Therefore, the UVA to UVB ratio of the radiation to which humans are exposed is important. This ratio changes with solar elevation, with cloud cover, with the ozone amount in the atmosphere and with several other less important parameters. The fluence rate of solar radiation reaching the earth is dependent on absorption and scattering. Scattering on clear days is caused by small elements, i.e., it is Rayleigh dominated and increases with decreasing wavelengths. A larger fraction of the incident UVB than of the UVA is absorbed by ozone and impurities in the atmosphere. Thus, a larger fraction of UVA than of UVB

Sunlight, Vitamin D and Skin Cancer, Second Edition, edited by Jörg Reichrath.

reaches the ground directly, unscattered from the sun, while more UVB is diffuse. Since, as discussed below, UVB and UVA have different impacts on health, and since radiation in these two wavelength regions are differently absorbed and scattered in the atmosphere, the choice of geometric representation of the human body is of fundamental importance. Equally important is the direction of the detector when UV from the sun is to be measured in a relevant way. At higher solar zenith angles (SZA) one cannot get maximal body exposures without lying horizontally, with the solar radiation falling perpendicularly on the skin. In theoretical, as well as in experimental approaches to evaluate positive and negative health effects of sun exposure and to balance these against each other, the human body is usually modeled as a horizontal, flat surface. This may lead to widely different predictions of health consequences of exposures than using a vertical cylinder model, which is more realistic. The choice of geometrical model is important for evaluations of latitudinal as well as of temporal effects, since the fluence per unit skin area is dependent both on the angle of incidence on the skin and on the zenith angle of the sun, the SZA. Larger SZAs result in relatively larger absorption and scattering of UVB (mainly vitamin D-inducing wavelengths) than of UVA (contributing together with UVB to immune- and carcinogenic effects) in the atmosphere. At the Equator there is about a 20% variation of the daily doses of vitamin D-inducing UVB radiation during a year, while at 50°N the corresponding variation is 250%.[1]

A large fraction of the solar UV radiation falling on skin is diffuse. This fact introduces problems in assessing risks and benefits. A horizontal plane model overestimates annual erythemally and vitamin D generating UVB doses by about a factor of two compared with a cylinder model.[2] The model dependency is even larger for UVA radiation. With the cylinder model, high UVA fluence rates last about twice as long after noon as high UVB fluence rates do. Thus, the human body can be more realistically represented by a vertical cylinder, as we do in this work, than by a horizontal, planar surface, as done in almost all calculations in the literature.

An important factor to keep in mind is that UV exposure of skin leads to nonlinear biological effects.[3] This should be paid more attention because different anatomical sites are exposed to widely different fluence rates and fluences. Nonlinearity and geometrical model are even more important to take into account when circadian rhythms of cellular damage and repair are brought into consideration, as we will show. UV induction of DNA damage, as well as DNA repair, follow a circadian rhythm, at least in mice. For humans, this points toward optimal repair and minimal damage of UV exposure before noon. With the above mentioned facts in mind, we hope to be able to give improved recommendations with respect to an optimal exposure pattern to solar radiation.

METHODS

Both direct and diffuse UV radiation on a vertical cylinder and on a horizontal surface[4] were calculated with a radiative transfer model.[5,6] Fluence rates of solar radiation were calculated for the lowest SZA summer day in Oslo (59.95°N), in Sydney (33.87°S) and in the Christmas Islands (1.86°N) where average total ozone columns are 350 Dobson units (DU), 300 DU and 250 DU, respectively. When different wavelength regions are to be compared, the cylinder model will give more relevant results than the horizontal plane model. Even for persons lying on the ground a large fraction of the skin surface will be vertically rather than horizontally oriented.

We have used the fish melanoma action spectrum found by Setlow et al.[7] as an approximation to the human CMM spectrum, despite the fact that there is a discrepancy between Setlows work and a recent publication from Mitchells group,[8] in which it is claimed that the Setlow spectrum is wrong and that UVA does not give melanoma in the Xiphophorus fish.[8] A reason for the discrepancy may be that the fish used in the two investigations were genetically different. Furthermore, the experiments of Noonan et al.,[9] in which hepatocyte growth factor/scater transgenic mice C57BL/6-HGF (black) were used to study melanoma induction, demonstrated that UVA may be a melanoma inducer in the presence of melanin.[9] Finally, our own epidemiological work indicates that UVA is likely to play a significant role also in human CMM etiology,[10] and a recent review on the topic by Mitchell et al.[11] concludes similarly. Thus, even if UVA does not have an initiating effect under all conditions, it is likely to have a promotive effect and act melanomagenic together with UVB. For vitamin D generation we have used the action spectrum of Galkin and Terentskaya,[12] which is supposedly more accurate in the wavelength region above 300 nm, (which counts most heavily), than the spectrum measured in human skin.[13] However, for the present evaluations the two spectra lead to qualitatively similar results and conclusions.

RESULTS AND DISCUSSION

As representative examples we performed calculations for midsummer days at three latitudes with different maximal solar elevations at noon, using cylinder geometry, the CMM and the vitamin D action spectra as argued for in Materials and Methods. Since differences between vertical cylinder and horizontal plane models show no significant dependence on ozone levels between 230 and 400 DU,[14] typical midsummer ozone values for all three geographic locations were used. A high cloud cover would reduce the difference between these two models.[2] The SZA, and consequently the time of the day, is the most interesting variable, i.e., the dominant determinant for UVA and UVB fluence rates.

Figure 1 shows that vitamin D synthesis and CMM risk varies widely during a day at all three latitudes, CMM risk lasting much longer in the afternoon than vitamin D synthesis does. This is due to the fact that UVA plays a role for CMM generation. At least five observations indicate that UVA plays a major role in CMM induction by the sun and that melanin, which absorbs UVA and even visible light, may be a chromophore for this: (1) African albinos who lack dark melanin, have very low incidence rates of CMM in spite of the fact that they have high incidence rates of non melanoma skin cancers;[15] (2) The latitude gradient of CMM is much smaller than those of non melanoma skin cancers, just as the latitude gradient of annual doses of UVA is smaller than that of annual doses of UVB;[10] (3) Some of the mutations found in CMMs are not due to UVB induced pyrimidine dimers, but rather to UVA induced oxidative DNA damages;[16] (4) The action spectrum for light activation of melanin in Xiphophorus resembles that of CMM induction in the same fish;[17] (5) Melanin pigmentation plays a significant role for the induction of CMM in a hepatocyte growth factor transgenic mouse model.[9]

Sunbeds emit relatively more UVA than what is found in solar radiation[18] except for large SZAs. In view of this, it is surprising that some investigations report that sunbed use increases the risk of basal cell carcinoma (BCC) but not that of CMM.[19] Other investigations indicate that UVA plays a similar role for CMM and for BCC.[9,20] Measurements of the

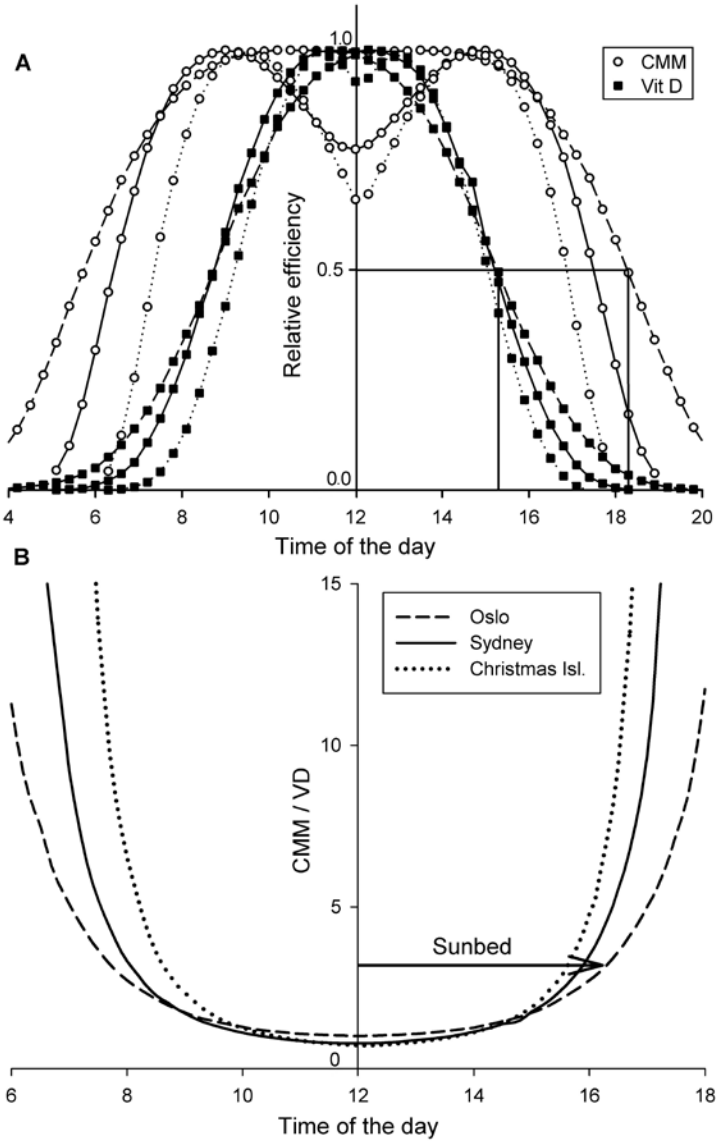

Figure 1. The time dependence of vitamin D–inducing and melanoma–generating fluence rates of solar radiation on a midsummer day in Oslo (59.95°N), in Sydney (33.87°S) and on the Christmas Islands (1.86°N). All curves are normalized to unity at the maximal value. Open circles represent CMM inducing fluence rates, squares represent vitamin D generating fluence rates. The lower part of the figure shows the UVA to UVB ratios.

UVB and UVA fluence rates of a commercial sunbed in our laboratory, (Solarium Super Plus 100 W tubes (Wolff system, Basel, Switzerland) showed that its fluence rate of UVB was similar to that in solar radiation at noon in the summer in Oslo and that the UVA to UVB ratio was similar to that of solar radiation at 4:30 p.m. (Fig. 1).

It is important to take into account that the day length is different at different latitudes. Even when the same time- and intensity scales are used for all locations, the curves showing the UVA to UVB ratio (Fig. 1, lower part) are similar around noon. However, the absolute UVA and UVB doses depend on latitude. After noon fluence rates of vitamin D generating radiation falls off much faster than those of CMM generating radiation do (Fig. 1). For example, the rate of synthesis of vitamin D is halved at about 3:20 p.m., while the CMM induction rate is not halved before 6:30 p.m. in Oslo.

In the future circadian rhythms should be brought into this discussion. It seems that a given fluence acts differently before and after noon. Mouse experiments suggest that in humans DNA repair may be maximally efficient in the morning.[21] It should be noted that mice, in contrast to humans, are nocturnal animals. Experiments with human epidermal cells seem to indicate that DNA synthesis (the S-phase) has a peak around noon and that the number of mitotic cells is lowest before noon.[22] Experiments with hair follicle cells show that nucleotide excision repair rate is maximal around 7 a.m.[23] These factors suggest that the best and safest time for sun exposure might be a couple of hours before and around noon and that recommendation to avoid sun exposure between the hours of 10 a.m. and 4 p.m.[24,25] may be non-optimal with respect to health.

CONCLUSION

We conclude that postponing moderate nonerythemogenic sun exposures from noon to afternoon yields less vitamin D at a similar melanoma risk. The relative effects of sunbed exposure are similar to those of sun exposure 4–5 hours after noon with respect to the UVA/UVB ratio (Fig. 1B). However, at that time longer sun exposures than sunbed exposures are needed to give the same vitamin D yield. In view of the present work, recommendations concerning "healthy and unhealthy" sun exposures should be adjusted to agree with recent research. Early noon seems to be optimal.

ACKNOWLEDGMENTS

The present work was supported by the South-Eastern Norway Regional Health Authority and by Oslo University Hospital. The study has used data from the Cancer Registry of Norway. The interpretation and reporting of these data are the sole responsibility of the authors, and no endorsement by the Cancer Registry of Norway is intended nor should be inferred.

REFERENCES

1. Chaplin G, Jablonski NG. Vitamin D and the evolution of human depigmentation. Am J Phys Anthropol 2009; 139:451-61; PMID:19425101.
2. Pope SJ, Godar DE, Solar UV. Solar UV geometric conversion factors: horizontal plane to cylinder model. Photochem Photobiol 2010; 86:457-66; PMID:20059727; http://dx.doi.org/10.1111/j.1751-1097.2009.00679.x.
3. Streicher JJ, Culverhouse WC Jr., Dulberg MS, Fornaro RJ. Modeling the anatomical distribution of sunlights. Photochem Photobiol 2004; 79:40-7; PMID:14974714.

4. Dahlback A, Moan J. Annual exposures to carcinogenic radiation from the sun at different latitudes and amplification factors related to ozone depletion. The use of different geometrical representations of the skin surface receiving the ultraviolet radiation. Photochem Photobiol 1990; 52:1025-8; PMID:2287633; http://dx.doi.org/10.1111/j.1751-1097.1990.tb01820.x.
5. Dahlback A, Stamnes K. A new spherical model for computing the radiation field available for photolysis and heating rate at twilight. Planet Space Sci 1991; 671-83; http://dx.doi.org/10.1016/0032-0633(91)90061-E.
6. Stamnes K, Tsay SC, Wiscombe W, Jayaweera K. Numerically stable algorithm for discrete-ordinate-method radiative transfer in multiple scattering and emitting layered media. Appl Opt 1988; 27:2502-9; PMID:20531783; http://dx.doi.org/10.1364/AO.27.002502.
7. Setlow RB, Grist E, Thompson K, Woodhead AD. Wavelengths effective in induction of malignant melanoma. Proc Natl Acad Sci U S A 1993; 90:6666-70; PMID:8341684; http://dx.doi.org/10.1073/pnas.90.14.6666.
8. Mitchell DL, Fernandez AA, Nairn RS, Garcia R, Paniker L, Trono D, et al. Ultraviolet A does not induce melanomas in a Xiphophorus hybrid fish model. Proc Natl Acad Sci U S A 2010; 107:9329-34; PMID:20439744; http://dx.doi.org/10.1073/pnas.1000324107.
9. Noonan FP, Zaidi MR, Wolnicka-Glubisz A, Anver MR, Bahn J, Wielgus A et al. Melanoma induction by ultraviolet A but not ultraviolet B radiation requires melanin pigment. Nat Commun 201;3:884. doi: 10.1038/ncomms1893.
10. Moan J, Dahlback A, Setlow RB. Epidemiological support for an hypothesis for melanoma induction indicating a role for UVA radiation. Photochem Photobiol 1999; 70:243-7; PMID:10461463; http://dx.doi.org/10.1111/j.1751-1097.1999.tb07995.x.
11. Mitchell D, Fernandez A. The photobiology of melanocytes modulates the impact of UVA on sunlight-induced melanoma. Photochem Photobiol Sci 2012; 11:69-73; PMID:21887451; http://dx.doi.org/10.1039/c1pp05146f.
12. Galkin ON, Terenetskaya IP. 'Vitamin D' biodosimeter: basic characteristics and potential applications. J Photochem Photobiol B 1999; 53:12-9; PMID:10672524; http://dx.doi.org/10.1016/S1011-1344(99)00115-3.
13. MacLaughlin JA, Anderson RR, Holick MF. Spectral character of sunlight modulates photosynthesis of previtamin D3 and its photoisomers in human skin. Science 1982; 216:1001-3; PMID:6281884; http://dx.doi.org/10.1126/science.6281884.
14. Koepke P, Mech M. UV irradiance on arbitrarily oriented surfaces: Variation with atmospheric and ground properties. Theor Appl Climatol 2005; 81:25-32; http://dx.doi.org/10.1007/s00704-004-0106-z.
15. Diffey BL, Healy E, Thody AJ, Rees JL. Melanin, melanocytes, and melanoma. Lancet 1995; 346:1713; PMID:8551863; http://dx.doi.org/10.1016/S0140-6736(95)92882-0.
16. Hocker T, Tsao H. Ultraviolet radiation and melanoma: a systematic review and analysis of reported sequence variants. Hum Mutat 2007; 28:578-88; PMID:17295241; http://dx.doi.org/10.1002/humu.20481.
17. Wood SR, Berwick M, Ley RD, Walter RB, Setlow RB, Timmins GS. UV causation of melanoma in Xiphophorus is dominated by melanin photosensitized oxidant production. Proc Natl Acad Sci U S A 2006; 103:4111-5; PMID:16537493; http://dx.doi.org/10.1073/pnas.0511248103.
18. Nilsen LT, Aalerud TN, Hannevik M, Veierød MB. High UV-A exposure from sunbeds. Pigment Cell Melanoma Res 2012; 25:639-40; PMID:22776093; http://dx.doi.org/10.1111/j.1755-148X.2012.01035.x.
19. Faurschou A, Wulf HC. Ecological analysis of the relation between sunbeds and skin cancer. Photodermatol Photoimmunol Photomed 2007; 23:120-5; PMID:17598864; http://dx.doi.org/10.1111/j.1600-0781.2007.00289.x.
20. Pfeifer GP, Besaratinia A. UV wavelength-dependent DNA damage and human non-melanoma and melanoma skin cancer. Photochem Photobiol Sci 2012; 11:90-7; PMID:21804977; http://dx.doi.org/10.1039/c1pp05144j.
21. Gaddameedhi S, Selby CP, Kaufmann WK, Smart RC, Sancar A. Control of skin cancer by the circadian rhythm. Proc Natl Acad Sci U S A 2011; 108:18790-5; PMID:22025708; http://dx.doi.org/10.1073/pnas.1115249108.
22. Brown WR. A review and mathematical analysis of circadian rhythms in cell proliferation in mouse, rat, and human epidermis. J Invest Dermatol 1991; 97:273-80; PMID:1830075; http://dx.doi.org/10.1111/1523-1747.ep12480379.
23. Akashi M, Soma H, Yamamoto T, Tsugitomi A, Yamashita S, Yamamoto T, et al. Noninvasive method for assessing the human circadian clock using hair follicle cells. Proc Natl Acad Sci U S A 2010; 107:15643-8; PMID:20798039; http://dx.doi.org/10.1073/pnas.1003878107.
24. Berg AO. Counseling to prevent skin cancer: recommendations and rationale. Am J Nurs 2004; 104:87-91; PMID:15171121; http://dx.doi.org/10.1097/00000446-200404000-00027.
25. Lin JS, Eder M, Weinmann S. Behavioral counseling to prevent skin cancer: a systematic review for the U.S. Preventive Services Task Force. Ann Intern Med 2011; 154:190-201; PMID:21282699.