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Calculated Ultraviolet Exposure Levels for a Healthy Vitamin D Status

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ABSTRACT

The dangers of overexposure to sunlight have been well publicized, but less attention has been given to an acknowledged benefit of exposure to UV radiation; that being the cutaneous synthesis of vitamin D_3. Here we define a standard vitamin D dose on the basis of recently recommended requirements for vitamin D that take account of its risk reduction role in a variety of diseases, and present a web-based tool that enables the reader to calculate associated exposure times for any time and place using either default values or user-selected conditions. Either it is not possible to synthesize vitamin D_3 at high latitudes in winter, or the exposure time required to reach a standard dose is sometimes impractical. Where solar UV is sufficient, a risk-benefit analysis of sunburn *vs.* vitamin D_3 synthesis shows that the best time for brief sun exposure is in the middle of the day. For low solar elevation angles common at high latitudes, a fine line exists between adequate UV exposure for vitamin D_3 synthesis and a risk of sun burn.

INTRODUCTION

Progress in the last century has removed much of our reliance on the cycles of the sun through artificial lighting, heating, air conditioning and transportation. This progress, and its influence on environment and lifestyle alike, has resulted in changing attitudes toward sun exposure and medical advice regarding sun exposure. By the end of the last century the overriding medical concern was of too much sun exposure leading to skin cancer, a message underlined by ozone depletion (1) and prognoses and observations of increased UV radiation at the surface (2). Skin cancer cases are still increasing as a result of exposure to both UVB (280–315 nm) and UVA (315–400 nm) radiation both by acute overdosing (causing sunburn) and lifelong cumulative exposure, although the mechanistic links to sun exposure are still under investigation (3–5). Now, however, the potential health risks due to inadequate UV exposure are being re-evaluated.

Vitamin D is synthesized in the skin after exposure to solar UVB radiation, and because only a few foods contain vitamin D most people gain the majority of their vitamin D intake from sunlight exposure (6). Regular replenishment of vitamin D is essential, although it can be stored in fatty tissue during times of plenty (*e.g.* summer), thus providing some stores for periods when availability is reduced (*e.g.* winter). At latitudes with long winters this storage is insufficient and vitamin D status declines. Submariners, with no UV exposure, had vitamin D levels that declined by half in a period of 2 months (7). Vitamin D levels are low in many populations and age groups. For example, in winter, one-third to one-half of preschool children in the United Kingdom have insufficient vitamin D status (8) and rickets, the childhood disease caused by lack of vitamin D, is being observed again, primarily but not exclusively, among children with pigmented skin (9,10). Adolescents in France exhibit vitamin D status that barely reaches sufficiency even in summertime (11), whereas vitamin D insufficiency has been shown among free-living healthy adults in the United States (12). Other examples can be found in the Nordic countries (13,14), other European countries (15), Canada (16) and India (17).

In addition to its long-established role in calcium metabolism, vitamin D appears to have a number of other beneficial effects. A protective effect of vitamin D against cancer of the colon, prostate, and breast has been suggested on the basis of epidemiological studies (18–21) and experimentally for prostate and breast cancers (22). The mechanisms for a reduction in risk of cancer incidence and death (23) are well documented, and sunlight exposure (suggesting vitamin D) has also been associated with improved cancer survival rates (24–26).

The active form of vitamin D is 1,25-dihydroxyvitamin D ($1,25(OH)_2D$), which is produced after vitamin D_3 has formed in the skin, or after vitamin D_2 or vitamin D_3 taken orally has been hydroxylated first by the liver and then by the kidney. The first (liver) hydroxylation produces 25-hydroxyvitamin D (25(OH)D), and it is the plasma volume of 25(OH)D that is the usual measure of vitamin D status. 25(OH)D is observed to respond to exposure to UVB radiation, increasing in the summer months or with artificial radiation, and declining with lack of exposure; for example, in the wintertime. There is a well documented seasonal cycle of 25(OH)D in people living at mid to high latitudes (8). The active form, $1,25(OH)_2D$, is, however, very tightly regulated and has little response to sun exposure unless vitamin D status is low. Because the active form of the vitamin does not increase with sun exposure it was hard to explain how vitamin D could protect against cancer, even though $1,25(OH)_2D$ is known to be a potent inhibitor of abnormal cell growth (27,28). The explanation came with the discovery that colon cancer cells have receptors for 25(OH)D (29) and can internally metabolize this into the active form of the vitamin, $1,25(OH)_2D$ (21). $1,25(OH)_2D$ then exerts its antiproliferative action on the cell and so is a preventive measure against cancerous growth. Receptors for 25(OH)D have since been found in many other cells, including the breast and prostate (21,30–33), whose

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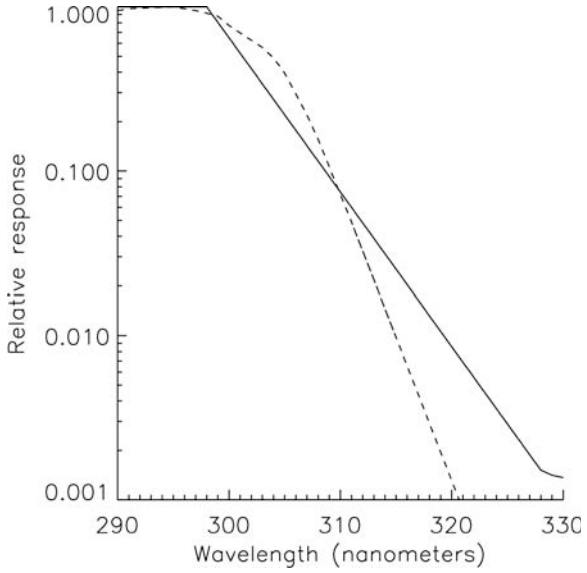

Figure 1. CIE (Commission Internationale de l'Eclairage) erythemal action spectrum (48) (solid) and the vitamin D weighted action spectrum (extrapolated from ref. 49; dashed).

cancers have been negatively correlated with sunlight exposure (34). Vitamin D has also been suggested as being effective against hypertension (35), with receptors found in blood vessels and hypertension decreasing with increasing UVB exposure (36,37); it may also help prevent the development of some autoimmune diseases such as multiple sclerosis and Type I diabetes (38,39). Further reviews of the benefits of vitamin D are provided by Heaney (40), Holick (41) and Peterlik and Cross (42).

There are clear benefits to UV exposure, and in the absence of careful dietary control UV exposure is necessary for skeletal health and has potential benefits for a range of other diseases. It is also clear that whereas a moderate amount of UV exposure is beneficial, too much is detrimental. In this paper we examine moderate UV exposure by optimally balancing the beneficial and harmful effects of UV radiation. To address the question, ''What is moderate exposure?'', we have modeled the erythemal and vitamin D effective solar radiation for all seasons and latitudes to enable estimates of the optimum exposures at different latitudes and for different skin types. We are not aware of any other study that provides guidelines on UV exposure duration taking all major variables into account, including variable atmospheric and surface conditions, time of day, percent body exposure and dietary vitamin D intake.

MATERIALS AND METHODS

Throughout this study we applied the fast yet accurate FastRT UV simulation tool (43). We assumed a representative cloudless ''base case'' atmosphere over a nonreflecting surface with an ozone layer thickness fixed at a typical level (350 Dobson Units [DU]) and a rural aerosol (44) optical depth given by $\tau = \beta * \lambda^{-\alpha}$ where the ångström coefficient (α) was set to 1.3, and the wavelengths (λ) are in micrometers. The ångström coefficient (β) was related to 25 km visibility R_m[km] using the parameterization provided by Iqbal (45) (*i.e.* $\beta = 0.55^{1.3}\,(3.912/R_m[\text{km}] - 0.01162)[0.02472 * (R_m[\text{km}] - 5) + 1.132]$. Otherwise, for all simulations we assumed a U.S. standard atmosphere (46). Clearly, this is an idealized situation, and changes in ozone, cloud, aerosol, albedo and altitude will all change the calculation. The Web site sponsored by the Norwegian Institute for Air Research (47) allows the user to select local conditions rather than taking the default values that we use for illustration. Deviations from the ''base case'' scenario are stated, when applicable. For results associated with UV indices, the matching cloud liquid water column was applied. FastRT was used to compute erythema (48) and vitamin D_3 effective (49) UV doses.

Defining the standard vitamin D dose

Vitamin D_3 effective doses were computed using the action spectrum for conversion of 7-DHC to previtamin D_3 in human skin (49) (Fig. 1) with an exponential decay extrapolation. We then defined a standard vitamin D dose (SDD) corresponding to the UV equivalent of an oral dose of about 1000 IU vitamin D (50) (*i.e.* one dose recommended to provide all the possible health benefits of vitamin D [25,51]). Because radiation is incident on the skin, and the response to either irradiation or oral dosing is measured in the blood, the SDD must be qualified by the conditions of skin exposure. Following the formula provided by Holick (50,51), which recommends exposure to ¼ of personal minimal erythemal dose (MED) on ¼ skin area (hands, face and arms; *i.e.* corresponding to the UV equivalent of an oral dose of 1000 IU vitamin D), we calculated the equivalent D_3 effective UV.

We calculated UV doses under a reference condition (*i.e.* midlatitude, midday in spring; Boston, 21 March, 42.2°N, ozone = 350 DU). The reasons and uncertainties associated with this choice of reference condition are revisited in the Discussion. First, for this reference condition, we calculated the time to acquire a ¼ MED around solar noon using FastRT model (43) simulations at 0.3 min time steps. Next, using the same simulated solar spectra at the ground over the same time interval, but weighting with the action spectrum for previtamin D_3 synthesis (49) instead of the erythema action spectrum (48), we then calculated the vitamin D_3 effective dose acquired over the same time interval. This is then the SDD based on exposure of ¼ body surface area, and like the MED, the absolute amount of radiation required to obtain an SDD changes with skin type (see results, Table 1).

A person exposing hands, face and arms would now make sufficient vitamin D_3 with 1 SDD, and will suffer minimal erythema after 1 MED, which by definition is four times the SDD exposure under the reference conditions (*i.e.* Boston, 21 March, 42.2°N, ozone = 350 DU), but not necessarily for other conditions with a different shape of the solar spectrum at the ground. Once more, following the recommendations offered by Holick (51) and others (52), this UV exposure of 1 SDD should be achieved approximately every other day.

RESULTS

The time required to obtain the recommended UV dose for adequate vitamin D_3 synthesis in human skin (*i.e.* 1 SDD) depends on the solar elevation angle, as well as the surface and atmospheric conditions. For fixed typical atmospheric conditions, a significant variation with respect to season and latitude is evident (Fig. 2). The period and locations at which the recommended UV exposure is not available (red field, Fig. 2) is not as extensive as the vitamin D_3 winter found by Webb *et al.* (53) who used realistic exposure times of up to 3 h and not the extensive periods accumulated in the model scenario. In reality, the variability of UVB radiation, and consequently the vitamin D_3 effective dose, is immense even at one location, primarily due to variations in cloudiness, ozone, surface reflection and aerosols. The recommended exposure times must account for skin type (Table 1 [54]) and changes in the radiative regime. Roughly speaking, variations in skin type and parameters influencing UV radiation shift the extent of the red and black fields in Fig. 2 up and down while the essential shape is preserved. Examples of the effects of some of these influences are given in Table 2 for Skin Types I, III and V. For each skin type, Table 2 shows for a variety of atmospheric conditions the maximum latitude at which 1 SDD could theoretically be reached in midwinter (in the daylight hours available), and the period for which 1 SDD cannot be reached at 70°N.

Table 7.2 in Holick (50) provided recommendations for ''safe and effective'' sun exposure for vitamin D production for different skin types, latitude ranges, season and times of day. Our Table 3 shows exposure times needed to gain 1 SDD as a function of

Table 1. General characteristics of skin types

Skin type	Color	Reaction to UV radiation	Reaction to sun	1 MED (J m^{-2} erythemal)*	1 SDD (J m^{-2} D effective)	Minimum exposure (minutes)†
Type I	Caucasian; blonde or red hair, freckles, fair skin, blue eyes	Very sensitive	Always burns easily, never tans; very fair skin tone	200	37.2	16
Type II	Caucasian; blonde or red hair, freckles, fair skin, blue eyes or green eyes	Very sensitive	Usually burns easily, tans with difficulty; fair skin tone	250	46.5	20
Type III	Darker Caucasian, light Asian	Sensitive	Burns moderately, tans gradually; fair to medium skin tone	300	55.8	25
Type IV	Mediterranean, Asian, Hispanic	Moderately sensitive	Rarely burns, always tans well; medium skin tone	450	83.6	37
Type V	Middle Eastern, Latin, light-skinned black, Indian	Minimally sensitive	Very rarely burns, tans very easily; olive or dark skin tone	600	111.4	49
Type VI	Dark-skinned black	Least sensitive	Never burns, deeply pigmented; very dark skin tone	1000	185.1	83

*1 MED varies for different skin types (54). †Indicates the minimum recommended exposure time to achieve SDD for Boston (42.2°N) on 21 March. Note that exposure times are calculated with solar noon at the midpoint of the period. If exposure is taken at times other than the midday period then the required exposure to achieve 1 SDD will be increased.

latitude, season, time and skin type under our standard atmospheric and skin area conditions. Note, there is little change in exposure times in the hours around solar noon (illustrated by noon and 1030 h) but at 0900 h (or 1500 h) exposure times are approximately double those at noon.

The public generally has access only to UV indices (55) as a guide to the UV environment, either measured or forecast. However, the UV index is an instantaneous value, whereas the SDD is a time-accumulated dose. We can therefore show only a general relationship between the UV indices and minimum recommended exposure times (Fig. 3). Exact relations will depend on time, location and the radiative regime. The forecast UV index is often given in integer numbers only, shown in parentheses in the examples below. As a rough guide, if the UV index is less than 1.7 (2) (*e.g.* when exceeding a latitude of 59° on 21 March for cloudless conditions at solar noon), we calculate that it will be difficult for a fair-skinned person to achieve an SDD in less than 1 h. Smaller amounts of vitamin D_3 may be made in a brief exposure as long as the UV index is greater than ~0.5. If the UV index is less than 0.5 (1) then casual exposure will not result in any appreciable vitamin D_3 synthesis. This so-called vitamin D winter (53) is observed for

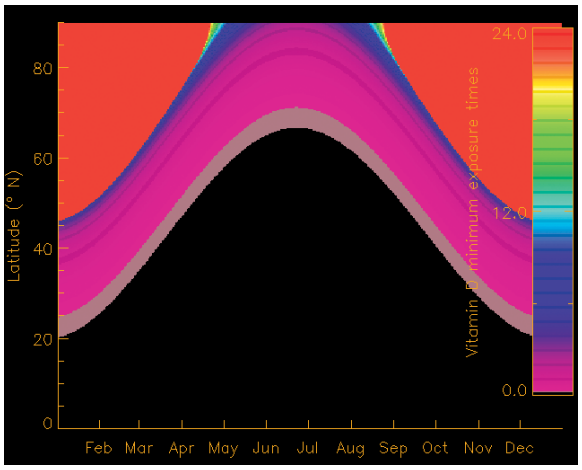

Figure 2. Recommended UV exposure times around noon for a cloudless sky and base conditions (see Materials and Methods) with respect to latitude and day of year to obtain 1 SDD for Skin Type I (MED 200 Jm^{-2}; *i.e.* 37.2 Jm^{-2} vitamin D weighted UV dose). The red areas illustrate when and where unity SDD is not achievable. The recommended SDD dose can be obtained in minutes in the black area. Reprinted with permission from Elsevier (A. R. Webb [2006] Who, what, where and when—Influences on cutaneous vitamin D synthesis. *Prog. Biophys. Mol. Biol.* **92**, 17–25).

Table 2. Limits of sufficient UV exposure as a function of atmospheric and surface conditions for Skin Types I, III and V*

Sufficient solar UV levels for a healthy Vitamin D status	Midwinter maximum latitude [°N] (solar zenith angle [°]) of incidence	Period at 70°N
Skin Type I		
Reference atmosphere	47 (70)	18 March–28 September
Low ozone (200 DU)	54 (77)	1 March–15 October
High ozone (500 DU)	40 (63)	31 March–14 September
Low visibility (5 km)	44 (67)	23 March–22 September
High visibility (210 km)	47 (70)	16 March–29 September
High altitude (3 km)	48 (71)	15 March–30 September
Snow-covered ground	49 (72)	12 March–3 October
Skin Type III		
Reference atmosphere	44 (67)	23 March–22 September
Low ozone (200 DU)	52 (75)	6 March–10 October
High ozone (500 DU)	37 (60)	7 April–7 September
Low visibility (5 km)	41 (64)	29 March–16 September
High visibility (210 km)	45 (68)	22 March–23 September
High altitude (3 km)	45 (68)	21 March–24 September
Snow-covered ground	47 (70)	17 March–28 September
Skin Type V		
Reference atmosphere	39 (62)	4 April–10 September
Low ozone (200 DU)	48 (71)	15 March–1 October
High ozone (500 DU)	31 (54)	22 April–23 August
Low visibility (5 km)	36 (59)	11 April–3 September
High visibility (210 km)	40 (63)	1 April–12 September
High altitude (3 km)	40 (63)	31 March–14 September
Snow-covered ground	42 (65)	27 March–18 September

*The table shows the maximum latitude at which 1 SDD could theoretically be reached in midwinter, and the period for which 1 SDD is achievable at 70°N.

Table 3. Sun exposure times (in minutes) for vitamin D production as a function of latitude, date, time and skin type*

Skin type by latitude	0900 h			1030 h			1200 h		
	21 December	21 March	21 June	21 December	21 March	21 June	21 December	21 March	21 June
11.5°N									
I	18	10	9	7	4	4	6	3	4
II	22	13	11	9	5	5	7	4	4
III	25	15	13	11	7	6	8	5	5
IV	35	22	19	16	10	9	13	7	8
V	44	28	25	21	13	12	17	10	10
VI	63	42	38	33	20	20	28	16	16
29°N									
I	50	15	8	20	6	4	16	5	3
II	58	19	9	25	8	5	20	6	4
III	65	22	11	30	9	6	23	7	5
IV	84	30	16	43	14	9	35	10	7
V	100	38	21	55	18	11	48	14	10
VI	136	56	33	86	29	19	85	23	16
42.5 °N									
I	131	26	8	75	11	4	70	8	4
II	148	31	10	91	14	6	94	10	5
III	165	36	12	107	16	7	127	12	6
IV	213	49	17	161	24	10	999	19	8
V	274	60	22	999	31	13	999	25	11
VI	999	86	35	999	49	21	999	41	19
62.5°N									
I	−999†	89	13	−999	52	8	999	44	7
II	−999	103	16	−999	63	10	999	55	9
III	−999	116	19	−999	74	12	999	67	11
IV	−999	152	27	−999	106	18	999	106	16
V	−999	184	35	−999	139	24	999	163	21
VI	−999	276	55	−999	264	39	999	999	36

*All calculations represent clear sky conditions. The latitudes are the midpoints of the ranges selected by Holick (51). Times are related to local solar time, so 1200 h is local solar noon, and do not take account of daylight saving (summer) time. In an idealized model the radiation regime would be symmetrical with time so the autumn equinox (in September) is the same as that in March, and 1500 h is the same 0900 h. –999 and 999 signify that an SDD was not available. †–999 Means that the solar zenith angle was never below 87 degrees, which is the practical limit for the FastRT UV simulation tool.

latitudes above 51°N on 21 December for a clear atmosphere (56). Using the UV index as a rough guide to vitamin D_3 synthesis has the advantage that the UV index forecasts account for the predicted ozone and usually the predicted cloud cover for a location.

To investigate the balance between a harmful (erythema) and beneficial (vitamin D_3 synthesis) effect of UV exposure we plotted the UV exposure in MEDs after obtaining 1 SDD (Fig. 4). Because the action spectra for erythema and cutaneous vitamin D_3 synthesis differ considerably (Fig. 1) and the solar spectrum at Earth's surface also changes with solar zenith angle, a required UV dose of ¼ MED (where 1 SDD is achieved at ¼ MED) is only valid for the same radiative regime used for defining SDD. For other solar elevation angles (illustrated as functions of latitude and season), as well as other altitudes, aerosol, cloud and ozone amounts, the solar spectrum and the erythemal UV dose will change. Note the fine balance between recommended UV exposure and harmful exposure when the solar UV is just about strong enough to provide recommended UV exposure (*i.e*. the rim around the black boundary in Fig. 4).

DISCUSSION

This work is based on a number of assumptions that must be recognized. The real atmosphere varies widely, as do real people. Examples of the effects of such variations are shown in Table 2. In addition, we have taken a single recommendation for vitamin D requirements, yet the optimum requirement for vitamin D is still a matter of debate (57–59). In addition, we have taken no account of particular sections of the population such as elderly people or pregnant women who may have different requirements (60,61), nor have we considered confounding factors such as body fat (62).

For the illustrations in this manuscript we use a recommendation that takes account of the proposed benefits of vitamin D beyond the long-established calcium metabolism. The web site (47) allows the user to vary the dietary equivalent dose of vitamin D they require from UV exposure, either to take account of known dietary intake, or alternative recommendations for vitamin D.

The action spectrum we have used is the only one measured for human skin, and in our opinion is the most appropriate choice. However, to estimate the uncertainty in the results due to this choice the results in Table 3 have been recomputed using two alternative action spectra: the 7-DHC absorption spectrum (Terenetskaya, personal communication), and the D-dosimeter action spectrum (63). None of the entries in Table 3 are more than 3 min longer than those calculated using the D-dosimeter action spectrum, or 5 min shorter than when using the 7-DHC absorption spectrum. The D-dosimeter action spectrum overestimates the exposure times required with respect to the action spectrum offered by MacLaughlin *et al*. (49) at low solar elevations, and underestimates them at high solar elevations. The converse is true for the 7-DHC absorption spectrum.

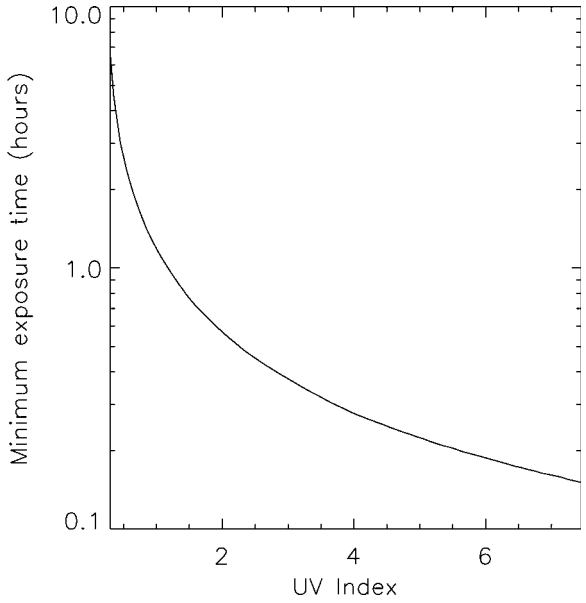

Figure 3. Recommended UV exposure times around noon for base conditions (see Materials and Methods) with respect to UV index on 21 March in Boston (42.2°N) for Skin Type I. The recommended UV exposure is not available for UV indices below 0.5 (65). It is not possible to calculate UV indices above 7.5 for this time and location.

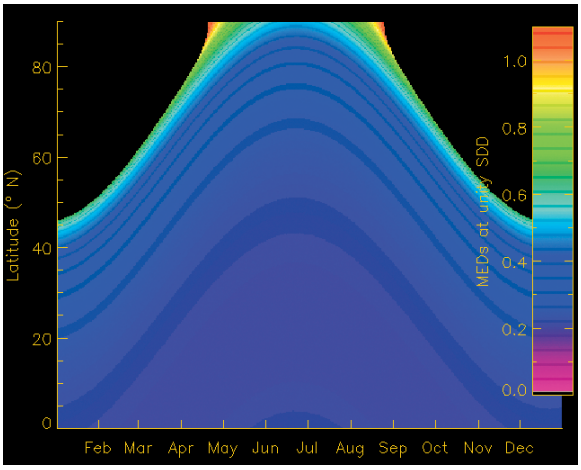

Figure 4. Number of MEDs acquired when unity SDD is obtained for Skin Type I, as a function of latitude and day of year for a cloudless sky. Black areas indicate where and when the recommended UV dose for vitamin D (SDD) is not achievable. Close to these areas, the margin between the recommended minimum UV for SDD and UV exposure liable to produce erythema is the smallest.

At low solar elevations the differences in the calculations increase, and exceed ±10 min in some cases, but under these conditions the hypothetical exposure times would be practically unrealistic. Our results therefore fall between those calculated using two other candidate action spectra, and for low latitudes and high solar elevations where exposure times are shortest the absolute differences in exposure times are ±1 min.

A further choice we made was the solar spectrum at the ground used for defining the SDD, because this was calculated via the erythemal action spectrum, and the relation between erythema and vitamin D synthesis changes with changing solar spectrum. Unfortunately, Holick and coworkers (50,51) did not specify the exact shape of the spectrum of the radiation source they used for their background experiments, except that ''healthy young and middle-aged adults were exposed to simulated sunlight equivalent to being on a sunny beach.'' We do not have adequate information to know or accurately reconstruct the spectrum from this artificial light source and to thus know the exact relevant vitamin D weighted doses during the experiments that Holick *et al.* conducted. Lacking this data we chose for reference a calculated (modeled) spectrum for noon at the spring equinox in Boston (solar zenith angle [SZA] = 42°), but selecting noon for the summer solstice in Boston (SZA = 19°), or at the equator at spring equinox (SZA = 2°) as the reference spectrum would lead to an increase in recommended exposure times of about 20%. The springtime Boston spectrum is more representative of summertime sunlight at higher latitudes than are the low SZA spectra and so was chosen to define the SDD. It is then known that vitamin D_3 can be synthesized in human skin. This latter assumption is based on the work of Webb *et al.* (53) who showed that from November to February there was insufficient solar UVB to synthesize vitamin D_3 in Boston, but by March, previtamin D_3 was formed from 7-DHC in both solution and the skin. The total ozone of 350 DU is approximately the total ozone defined in the U.S. standard atmosphere, and thus compatible with most other atmospheric parameters assumed here.

Other authors who have estimated vitamin D production in sunlight include Beadle (64) and Holick (51). Beadle (64) attempted to calculate the rate of UV absorption by 7-DHC in epidermis for clear sky conditions as a function of latitude, season and skin type to provide an upper limit estimate of epidermal vitamin D production. He estimated that nonpigmented skin produces about 10 times more than our findings for Skin Type I at midlatitudes in spring. For heavily pigmented skin (Skin Type 6) his estimates were about 40 times higher. The differences are somewhat dependent on solar elevations, but our estimates never exceed Beadle's estimates of dermal production. Our results are thus never in conflict with Beadle's aim to define an upper limit.

Our estimates for minimum recommended exposure times are in broad agreement but on the long side of Holick's range for the idealized cloudless conditions. Elsewhere, Holick (65) stated that 5–15 min of exposure between 1000 h and 1500 h during the spring, summer and autumn is usually enough for individuals with Skin Type II or III. We concur with this rough general recommendation for moderate latitudes for cloudless conditions, although for most conditions we would recommend the longer end of this range of exposures (*i.e.* 15 min). For high latitudes (>60°) or cloudy conditions (or both), a significantly longer UV exposure is recommended. Note that our web page (47) offers the opportunity to assess minimum exposure times for a variety of surface and atmospheric conditions, in addition to the cloudless conditions referred to by Holick (50,65).

Thus, absolute values of exposure times reported in this paper are only guidelines. In addition, cloudy skies or pigmented skin will increase the times, low ozone or high altitude, and highly reflective environments will decrease the times, and their effect can be computed more accurately using our web page (47). For example, we calculate that the radiation amplification factor (*i.e.* percentage change in the effect per percentage change in ozone) for vitamin D_3 synthesis is 2.5 for small changes in ozone on 21 March in Boston. For the same conditions, the modeled recommended exposure time decreases by about 7% per kilometer increase in

altitude. Adding a snow cover (albedo of 0.9) in the model instead of a nonreflecting surface decreases the recommended exposure time by 31%. In addition, the calculations are for UV radiation on a flat horizontal surface—not the geometry of the human body during casual exposure—this would require further modification of exposure times that would depend on the solar elevation angle and atmospheric conditions (66,67). The study indicates where casual sun exposure is able to provide sufficient cutaneous synthesis of vitamin D_3 to benefit from all the vitamin's proposed health impacts (34–42,50,51,56). It is clear that acquiring vitamin D_3 in this way is not possible at all times and all seasons. Where it is theoretically possible, further consideration must be given to skin type, real atmospheric conditions and the practicalities of time, skin orientation and unprotected skin area. In regions where cutaneous vitamin D_3 synthesis is obviously inadequate for significant periods of the year consideration should be given to other sources of the vitamin. Readers are encouraged to visit the web site (47) to explore situations not covered in the illustrations provided here.

In addition, we have shown the changing erythema risk: vitamin D_3 benefit analysis of sun exposure as a function of solar elevation angle (that is, latitude and season). A fine line exists between adequate UV exposure for vitamin D_3 synthesis and a risk of sunburn for the low solar elevation angles common at high latitudes (Fig. 4). At the high solar elevation angles found at low latitudes the brief time required for 1 SDD provides a much smaller fraction of an MED. Optimizing this risk-benefit of UV exposure therefore implies short exposures at maximum solar elevation angle (around noon) rather than longer exposures at other times of day, in contrast to common advice about behavior in the sun. It is stressed that efficient and pragmatic vitamin D_3 synthesis occurs at suberythemal doses. Where gaining 1 SDD requires more than ~0.5 MED UV exposure, the exposure times become unrealistically long (56).

In conclusion, public access to vitamin D through diet and UV exposure requires more attention from public health authorities, particularly at high latitudes and in countries with prevailing cloud cover, for naturally pigmented migrants and those with little ability to gain sun exposure. With indications that vitamin D may protect against more than bone disease it is no longer sufficient to assume that sunlight will provide for the needs of the global population.

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