Photochemistry and Photobiology, 20**, **: *–*

UV Radiation: Balancing Risks and Benefits†

Richard L. McKenzie*[1], J. Ben Liley[1] and Lars Olof Björn[2]

[1]National Institute of Water & Atmospheric Research (NIWA), Lauder, Central Otago, New Zealand
[2]University of Lund, Lund, Sweden

Received 12 December 2007, accepted 13 May 2008, DOI: 10.1111/j.1751-1097.2008.00400.x

ABSTRACT

We use action spectra published by the International Commission on Illumination to examine diurnal, seasonal and latitudinal variations in erythemally weighted (sunburning) UV—a health risk, and vitamin D-weighted UV—a health benefit. Vitamin D-weighted UV is more strongly dependent on ozone and solar zenith angle. Consequently, its diurnal, seasonal and geographic variability is more pronounced than for erythemally weighted UV. We then investigate relationships between the two quantities. An algorithm is developed and used to relate vitamin D production to the widely used UV index, to help the public to optimize their exposure to UV radiation. In the summer at noon, there should at mid-latitudes be sufficient UV to photosynthesize optimal vitamin D in ~1 min for full body exposure, whereas skin damage occurs after ~15 min. Further, while it should be possible to photosynthesize vitamin D in the winter at mid-latitudes, the amount of skin that must be exposed is larger than from the hands and face alone. This raises the question of whether the action spectrum for vitamin D production is correct, since studies have reported that production of vitamin D is not possible in the winter at mid-latitudes.

INTRODUCTION

Ozone depletion and erythemally weighted UV

Since the realization that the world's protective ozone layer was at risk from a build-up of anthropogenic trace gases in the atmosphere, there has been increased interest in understanding the variability and trends in UV radiation. Good progress has been made through improvements in instrumentation, calibration procedures and data quality assurance. The widespread adoption of a standardized metric for reporting UV radiation risk—namely erythemally weighted UV (UV_{Ery}, in W m^{-2}) or the UV index (1) ($UVI = 40 \times UV_{Ery}$)—has also facilitated meaningful comparisons.

Assessments of our understanding of UV radiation and its effects on the environment are updated regularly. The most recent of these assessments predicts that although the ozone layer will gradually recover over the next few decades, the outlook for future UV is less certain (2–4). Despite the progress in instrumentation, any changes in UV_{Ery} attributable to ozone depletion have been difficult to detect, because of (i) uncertainties in UV measurement, (ii) a relatively low sensitivity of UV_{Ery} to changes in ozone and (iii) the effects of other changes in atmospheric composition (*e.g.* changes in aerosols and clouds).

The effects of ozone depletion are largest in the Antarctic region (5). In populated areas at lower latitudes, the effects have been smaller. At mid-southern latitudes, summertime ozone, and therefore UV_{Ery}, is influenced by the export of ozone-poor air from the Antarctic ozone hole. Long-term measurements at Lauder, New Zealand (45°S, 170°E, altitude 370 m) provide some of the strongest evidence for increases in UV_{Ery} attributable to ozone depletion outside the Antarctic region. The increases in peak UV_{Ery} due to ozone depletion were relatively modest, ~10–15%, with a peak in late 1990s, and a decrease since that time. Other measurement sites, which are generally more polluted, show larger variabilities from sources other than stratospheric ozone. These findings demonstrate that, outside the region affected by the Antarctic ozone hole, changes in UVI due to changes in ozone are rather small and are within the range of variability from other causes. That the changes in ozone and UV are relatively small is attributable to the success of the Montreal Protocol, and its subsequent amendments and adjustments.

In contrast to these ozone-induced changes in UV_{Ery}, the geographical and seasonal changes (as well as diurnal changes) are large. Compared with noon intensities, the corresponding daily total doses of available UV radiation show much larger seasonal variabilities because of the longer hours of daylight in summer. Generally, the daily doses of UV are a maximum at the subsolar locations (where the minimum solar zenith angle approaches zero), and tend to decrease rapidly in moving to locations where the noon solar zenith angle is larger. Thus, the highest daily doses and annual doses of UV tend to occur in the tropics, and the lowest doses occur in Polar regions where they fall to zero in mid-winter for all latitudes within the Arctic or Antarctic circles (latitudes $>68°$). There are two notable departures from the overall pattern. First, during the springtime Antarctic ozone hole period, UV doses can reach values comparable with mid to low latitudes, such as San Diego, CA (5). Secondly, UV doses can be exceptionally high in the Altiplano region of South America. There the daily dose of UV_{Ery} can exceed 12 kJ m^{-2} (or 120 standard erythemal dose [SED]) (6), which corresponds to ~50 minimum erythemal dose (MED) for fair skinned individuals. In terms of the peak irradiances, the UVI can reach values of 25 during the month

---

†Some of the material in this manuscript was presented at the WRC/PMOD "One Century of UV Radiation Research" meeting in Davos, Switzerland, September 18–20, 2007.

*Corresponding author email: r.mckenzie@niwa.co.nz (Richard L. McKenzie) of February in the high-altitude Altiplano region, when the noon Sun is approximately overhead (7). At other high altitude locations, such as the Tibetan Plateau and the Antarctic Continent, UV intensities are also elevated significantly.

At mid-latitudes (~45°), the daily UV_{Ery} dose in summer is comparable with that in the tropics, as longer day length compensates for a lower peak. But in the winter, it is less than 10% of the summer dose. As the latitude increases, the seasonal swing becomes more and more marked. It should be noted that estimates of UV_{Ery} from satellite sensors that use backscattered solar ultraviolet radiation are sometimes too large in polluted locations because extinctions within the atmospheric boundary layer are not well probed by these sensors (8,9).

In the southern hemisphere, the summer–winter contrast in UV_{Ery} is more marked because of (1) the phasing of the Earth's orbit about the Sun (closest in January and furthest in July), (2) differences in the seasonal patterns of ozone, with lower ozone in the south and (3) generally lower pollution. These huge seasonal changes in UV radiation have important implications for human health (10–12). There is a strong association between sunburn and melanoma (13), yet there is also increasing evidence that insufficient UV leads to vitamin D deficiency (14–17), and hence contributes to health problems (18–24). Thus, high UV irradiance in summer contributes to skin cancer, while low irradiance in winter contributes to ailments associated with vitamin D deficiency. The outcome can be fatal. In New Zealand, for example, the skin cancer rates are among the highest in the world, yet a significant fraction of the population has insufficient vitamin D in the winter (14), and the incidence of colon cancer is relatively high (17). Tanning of the skin, induced by the high summertime UV irradiance, and paling of the skin over winter may further exacerbate these problems.

Here, we use climatologies based on satellite data to show that seasonal differences in UV_{VitD} and UV_{Ery} are large, particularly at mid to high latitudes. We verify the large seasonal variability using spectral measurements of UV irradiance from a clean mid-latitude site, and then use those measurements to derive a relationship between UV_{VitD} and UV_{Ery}. We then make use of published physiological relationships involving these quantities to calculate the optimum exposure times as a function of UVI (or solar zenith angle [SZA]) to get sufficient vitamin D without erythema. Finally, we discuss the health implications of these findings and highlight an inconsistency which points to gaps in our knowledge of vitamin D production by sunlight.

METHODS

Weighting functions for erythema and vitamin D. The weighting functions for erythema (25) and for vitamin D production (26), as published by the International Commission on Illumination (CIE) are shown in Fig. 1a. Each of these is arbitrarily normalized to unity at its maximum value. The figure also includes typical global solar irradiance spectra measured at a mid-latitude site at local noon on cloudless days close to the summer and winter solstices. The resolution of the spectra is ~0.9 nm at full-width half-maximum, and the detailed structure is due mainly to absorption in the Sun's atmosphere. The sharp decrease at wavelengths shorter than 315 nm is due to absorption by atmospheric ozone. All of these curves are plotted on a logarithmic vertical scale. Although the erythemal weighting function extends to longer wavelengths, it does so at three orders of magnitude less than its peak. The vitamin D-weighted irradiance for the summer spectrum is about twice as large as for erythema because of its increased contribution between 300 and 315 nm. The weighted integrals for erythema (UV_{Ery}) and vitamin D (UV_{VitD}) are compared in Fig. 1b, with a linear vertical scale, but show only the wavelength region from 290 to 330 nm because the contribution from UV-A is small, especially in the case of vitamin D-weighted UV (UV_{VitD}).

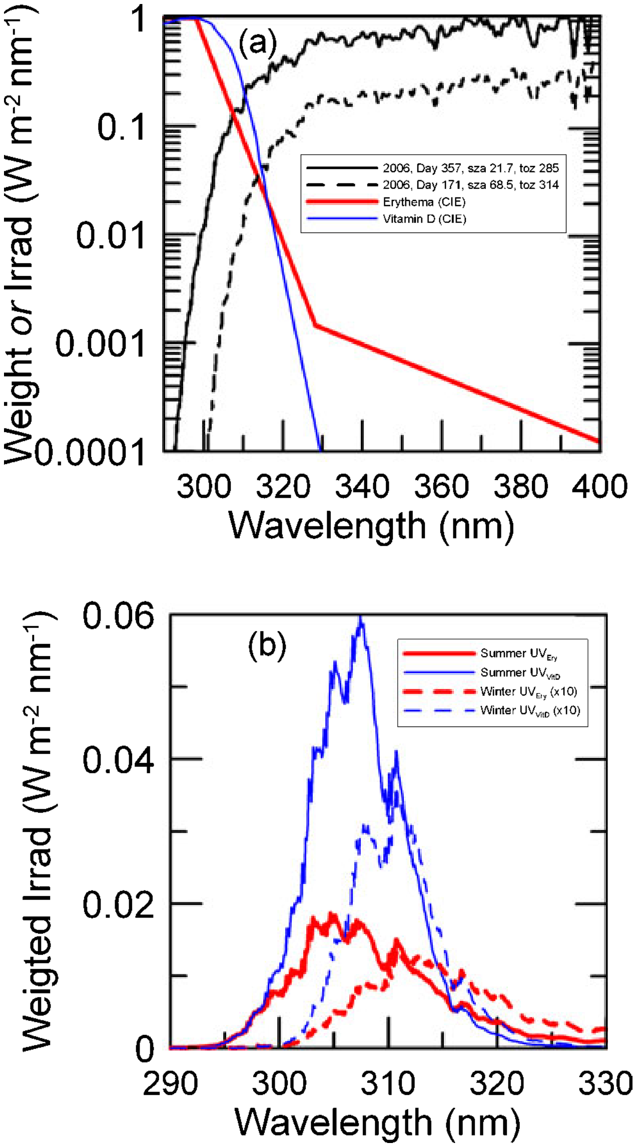

Figure 1. (a) Weighting functions for erythema and for vitamin D production, along with sample spectra measured at Lauder New Zealand at noon in the summer and winter. (b) Weighted spectral irradiances for the spectra shown in the upper panel between 290 and 330 nm, with winter values (dashed curves) scaled up by a factor of 10.

Note again that action spectra are normalized, so the use of units (*e.g.* W m^{-2} nm^{-1}) can be misleading in suggesting that UV_{Ery} and UV_{VitD} are directly comparable. Absolute, rather than relative, measures of either would have to be in terms of a defined physiological effect, such as MED for a given skin type. For vitamin D, skin type and area exposed must both be specified. In the following, we discuss the ratio UV_{VitD}/UV_{Ery} with regard to its spectral, seasonal, and diurnal variation, and subsequently introduce relevant units and dimensions.

For the summer spectrum, the weighted irradiances are UV_{Ery} = 0.28 Wm^{-2} and UV_{VitD} = 0.54 Wm^{-2} (UV_{VitD} is reduced by ~5% if the weighting function is cut at 315 nm). For the winter spectrum, the weighted irradiances are approximately 10% and 5%, respectively, of their summertime peaks UV_{Ery} = 0.026 Wm^{-2} and UV_{VitD} = 0.027 Wm^{-2} (UV_{VitD} is reduced by a further ~10% if the weighting function is cut at 315 nm). There are substantial differences in the spectral shape of these weighted irradiances. Further, in the winter, the contribution from longer wavelength components has a greater relative importance for both weightings.

Seasonal and diurnal variation. Spectral measurements of global solar UV irradiances, which have been undertaken by NIWA in New Zealand over several years, have been used to demonstrate the seasonal and diurnal variability of both UV_{VitD} and UV_{Ery} irradiances at this mid-latitude site. There are large diurnal, day-to-day and seasonal changes due to the combined effects of solar zenith angle, clouds, ozone and Earth–Sun separation. While the means of daily values including cloud effects are about 70% of corresponding clear sky values, on some days clouds attenuate the irradiances to less than 30% of the clear sky values The seasonal variation in UV_{VitD} is very large at this site, with noon values at mid-winter being only 5% of those in summer. This seasonal swing is significantly larger than for UV_{Ery}. The large swings are in contrast to the situation found for noon time UV_{VitD} at several sites in the USA over several months (27). However, those were based on measurements at lower latitudes where seasonal changes are smaller. Furthermore, in the northern hemisphere, the seasonal changes in Sun–Earth separation tend to cancel some of the effects due to seasonal changes in SZA.

Figure 2 shows the diurnal variability of UV_{Ery} and UV_{VitD} and their ratios on cloudless days near the summer and winter solstices at Lauder. The total column amount of ozone was stable through both of these days and was quite similar: 300 DU for the summer day and 310 DU for the winter day. Most of the difference is therefore attributable to differences in SZA and Earth–Sun separation. The peak UV_{Ery} in winter is a factor of 10 less than for the summer day, and the peak UV_{VitD} is nearly a factor of 20 less than the summer day. At larger SZAs, the UV_{VitD}/UV_{Ery} ratio is greatly reduced compared with that for high sun, resulting in lower ratios in the winter compared with the summer and lower ratios at twilight compared with midday.

Global climatologies. Recently, global climatologies of vitamin D-weighted UV have become available (6), permitting a direct comparison between UV_{VitD} and UV_{Ery}. In both cases, the weighting functions are as adopted by the CIE, extending to 400 nm in the case of UV_{Ery} (25) and to 330 nm in the case of UV_{VitD} (26). If the truncated version of the vitamin D action spectrum had been used, the peak value would be reduced by ~5%, with larger percentage reductions for smaller doses. The climatologies are based on over 20 years of satellite-derived data from the NASA Total Ozone Monitoring Spectrometer instruments. As changes in ozone have been relatively small over most of the globe, these climatologies still apply for the present day ozone fields, and for those expected in decades to come. In the Antarctic region, ozone amounts have been lower in spring, but the summer and winter conditions illustrated have been less affected by the springtime ozone hole. Furthermore, as ozone is expected to recover only slowly in the future, the means over the period of satellite data used are likely to be similar to the means over the next decades.

Zonal means of these UV climatologies are shown for solstice months in Fig. 3a. The UV doses are largest at the subsolar latitudes (20°S in December and 20°N in June) and decrease rapidly as the latitude diverges from there. Doses in southern hemisphere summer are significantly larger than in the northern hemisphere summer. The winter values in the southern hemisphere are much more comparable with those in the northern hemisphere. The UV_{VitD} doses show a stronger latitudinal gradient than the UV_{Ery} doses. When cloud effects are included these tend to reduce the southern hemisphere values more than in the northern hemisphere, especially at latitudes pole-ward of about 60°S (not shown).

Figure 3b shows the ratio UV_{VitD}/UV_{Ery} for these daily doses. Near the latitudes with peak UV, the daily doses of UV_{VitD} are approximately twice those of UV_{Ery}. This shows that the daily doses are dominated by the contributions from near noon when the SZA is smallest. The decrease is more rapid in the case of UV_{VitD}, and the ratios reduce to below unity at latitudes where the UV is low (*e.g.* approaching the poles). These calculated climatological mean values were compared with the measured values shown for the clear summer and winter days at Lauder discussed previously, by integrating the daily irradiances shown in Fig. 2 and plotting them as symbols at latitude 45°S. There is excellent agreement between the measurements and the climatology for both the weighted irradiances and their ratios. This gives confidence in both the measurements and the radiative transfer calculations.

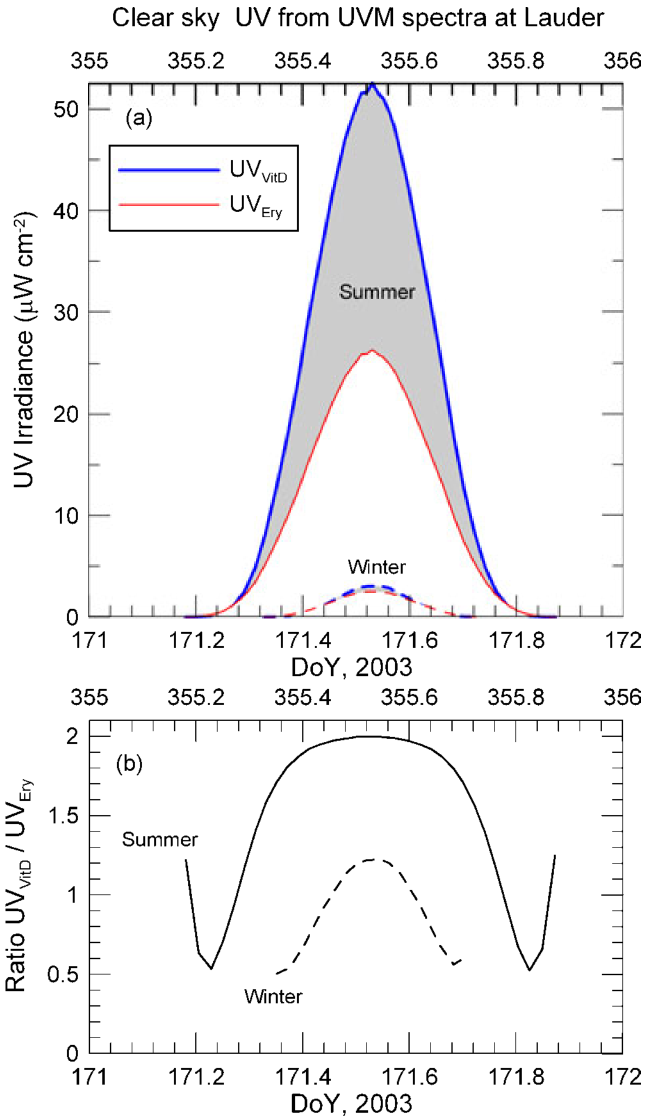

Figure 2. (a) Diurnal variations in weighted irradiances on a clear day near the solstice periods at Lauder, New Zealand 45°S in 2003 are shown in the upper panels, and (b) corresponding ratios of UV_{VitD}/UV_{Ery}: Solid curves are for the summer solstice, day 355 (upper axes), when the total ozone was 300 DU, and the minimum SZA was 21.6°. Dashed curves are for day 171 (lower axes), which is close to the winter solstice, when the total ozone was 310 DU, and the minimum SZA was 68.5°.

Relationship between UV_{VitD} *and* UV_{Ery}. The relationship between UV_{Ery} and UV_{VitD} is demonstrated in Fig. 4. A scatterplot of the relationship from several years of spectral UV data from Lauder, New Zealand is shown in Fig. 4a. In this plot, we used the full CIE action spectrum (26), which extends to 330 nm. At first impression, the plot seems to indicate a close proportionality between the two quantities, with UV_{VitD} being approximately 2 × UV_{Ery} (for standard normalization). However, at lower values, which are characteristic of the situation throughout winter months, this proportionality breaks down. For the truncated version of the action spectrum, the plot is very similar (not shown), but the slope is reduced by ~3%.

The relationship is examined in more detail in Fig. 4b where the ratio UV_{VitD}/UV_{Ery} is plotted as a function of SZA. Because there is uncertainty about the validity of extrapolating the action spectrum for vitamin D beyond the last measured point at 315 nm, this figure is also plotted for results using a truncated version of the CIE action spectrum, limited to wavelengths less than 315 nm (Fig. 4c). For the first case, the

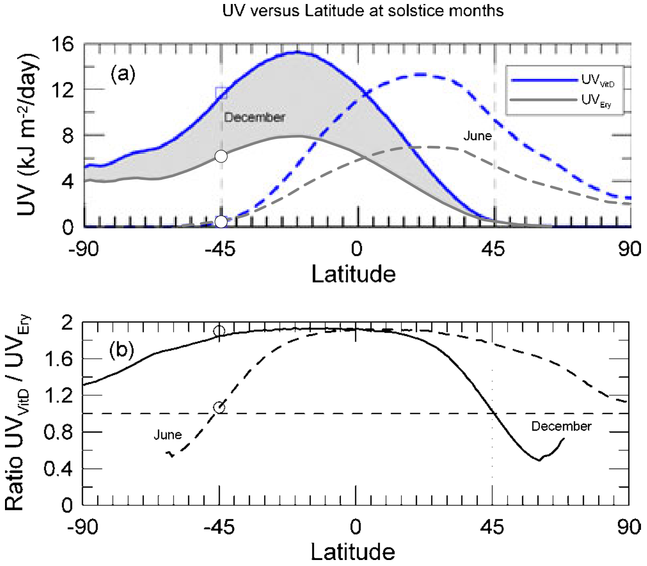

Figure 3. (a) Latitudinal distribution of the UV_{Ery} and UV_{VitD} daily dose, calculated for the two solstice months for clear skies, and (b) corresponding ratios of UV_{VitD}/UV_{Ery}. Solid curves are for December (southern hemisphere summer) and dashed curves are for June (northern hemisphere summer) clear skies. The larger symbols at 45°S are integrated values at Lauder from the summer and winter days shown in Fig. 2.

ratio is ~2 when the SZA is small and reduces to a value close to unity at SZA ~70°. The ratio reaches a minimum for SZA ~85° and increases thereafter. As expected, the departures from proportionality with UV_{Ery} are more marked, and the ratios are slightly lower, with the truncated version of the action spectrum. In both cases, there are horizontal groupings of the data at 5° steps in SZA, which are an artifact of the sampling intervals in the spectral measurements, and comprise the large majority of the spectra. Several spectra are also taken at 15 min intervals over a 2 h period around the solar noon. The largest vertical separation in points occurs at intermediate SZAs, where two distinct clusterings of data points are apparent. These are a consequence of the seasonal variations in ozone. In the spring, when ozone amounts are larger, the ratio is smaller than in autumn when ozone amounts are smaller.

Although UV_{VitD} is not directly proportional to UV_{Ery}, it is possible to estimate UV_{VitD} from UV_{Ery} using a radiative transfer model if ozone and SZA are known. Ratios of UV_{VitD}/UV_{Ery} have been calculated for a wide range of ozone and SZA using the TUV radiative transfer code (28) and are shown in the Appendix. The measured ratios in Fig. 4b and c are consistent with those calculated with the TUV radiative transfer model. Note that the ratios here are slightly different from those reported previously (29,30), which used a different digitization of the previously published action spectrum for vitamin D production (31).

Calculation of exposure time to induce erythema. The time taken (t_E in minutes) to induce skin damage (1 MED) is given by

$$t_E = \frac{4000}{60}\frac{\mathrm{MEDF}\cdot\mathrm{SPF}}{\mathrm{UVI}}, \qquad (1)$$

where the factor 4000/60 accounts for the conversions from UV_{Ery} to UVI, and seconds to minutes; UVI is the UV index ($= 40 \times UV_{Ery}$, where UV_{Ery} has units of Wm^{-2}); MEDF is a factor to account for differences in skin type. It is expressed here as the number of SED (1 SED = 100 J m^{-2} of UV_{Ery}) required to induce erythema, according to the Fitzpatrick skin classification (32) (see Table 1); and SPF is the sun protection factor of any sun block applied.

For example, for unprotected skin (SPF = 1) of type II (1 MED = 2.5 SED for erythema according to the Aust/NZ standard), the time taken to receive an erythemal dose at UVI = 12 would be 13.9 min. Under the same conditions, with a sun block of SPF = 20, it would take ~278 min (>4.5 h) for damage to occur.

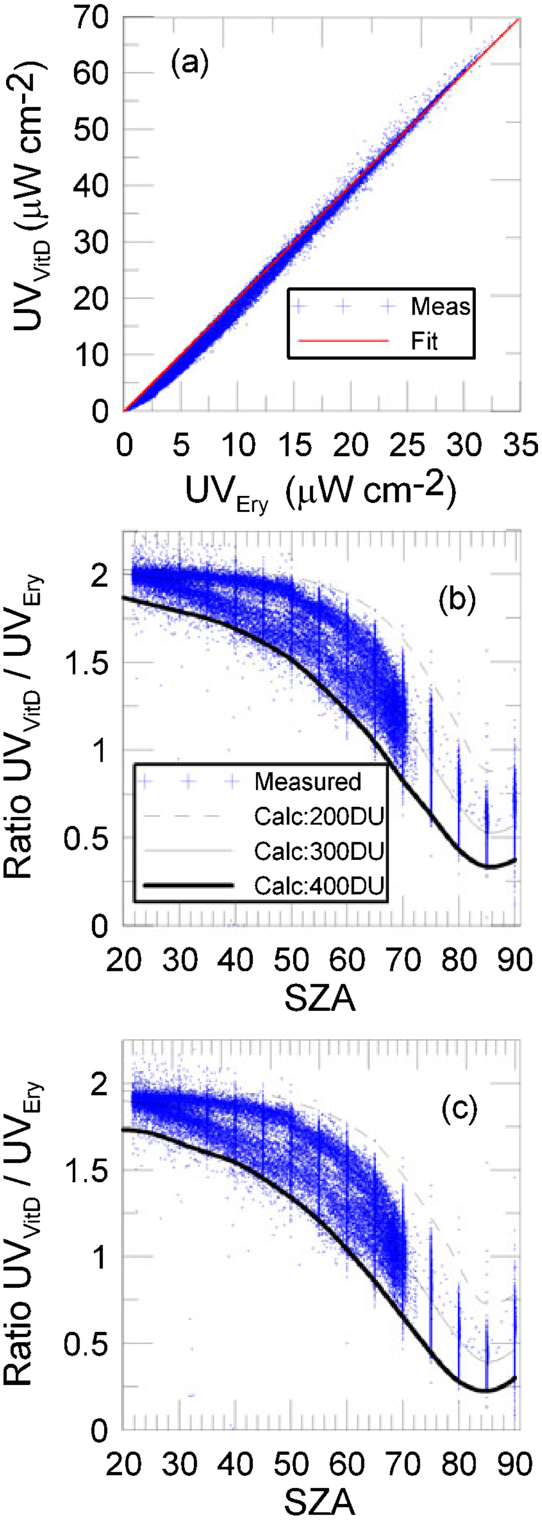

Figure 4. Relationship between UV_{VitD} and UV_{Ery} based on 100 000 spectra measured at Lauder, New Zealand over the period 1998–2007. (a) Scatterplot showing the near linear relationship, which breaks down for low values of UV_{Ery}, (b) ratios of UV_{VitD}/UV_{Ery} as a function of solar zenith angle. The curves show model calculated ratios for three ozone amounts: 200, 300 and 400 DU. (c), as for (b) except that instead of using the full CIE action spectrum for vitamin D production that extends to 330 nm, the weighting is limited to wavelengths less than 315 nm.

Table 1. Skin type classifications according to the Fitzpatrick scale (32) and the Australian/New Zealand Standard (58).

		SED to burn	
Skin type	Description	Fitzpatrick (1988)	Aust/NZS (2002)
I	Celtic (always burns)	2–3	< 2.5
II	Pale (burns easily)	2.5–3	2.5
III	Caucasian (may burn)	3–5	3.5
IV	Mediterranean (burns rarely)	4.5–6	4.5
V	S. American (rarely burns)	6–20	–
VI	Negroid (rarely burns)	6–20	–

Calculation of exposure time for sufficient vitamin D. Generally, our dietary intake of vitamin D is far below the level required to maintain optimal levels of blood serum vitamin 25(OH)D, so some UV exposure is desirable to maintain healthy vitamin D levels. Unlike the risk of erythema, the beneficial effects of UV radiation scale with the area of skin exposed. If you expose twice the area, then you get twice the benefit. We can use the relationships derived above to estimate the range of optimal exposures for various skin types as a function of UVI. We could also develop a similar relationship as a function of SZA, but that would ignore the effect of ozone, which can be appreciable.

The dose of vitamin D obtained can be described by:

$$\mathrm{VitD} = k\frac{\mathrm{UVI}\cdot R\cdot t_{\mathrm{D}}\cdot A}{\mathrm{MEDF}\cdot \mathrm{SPF}}, \qquad (2)$$

where k is a proportionality constant, which includes geometrical, biochemical and physiological considerations; UVI is the UV index as defined above; R (SZA, TOZ) is the ratio of $\mathrm{UV_{Ery}}/\mathrm{UV_{VitD}}$ for those conditions; t_{D} is the exposure time (in minutes); A is the fractional area of skin surface exposed; and MEDF and SPF are as defined above.

The MED factor (MEDF) can be understood by considering that for a given exposure to skin type IV, the radiation transmitted would be just under half of that for the same exposure to skin type II (Table 1). It has been shown previously that larger UV doses are needed for darker skinned people (33,34). The SPF can be understood by considering that if a sunscreen of SPF 20 were applied, the dose would be reduced by the same factor of 20.

We eliminate the unknown proportionality factor k in equation 2 by considering the vitamin D produced compared with that produced for a known reference condition, as follows. The recommended daily dose of vitamin D is thought to be in the range 400–1000 IU (18,22,35–37). Here, we assume that optimal vitamin D levels are easily maintained by a daily intake of 1000 IU of vitamin D. If an intake of 400 IU is sufficient, as some have suggested, then the times to achieve the desired UV dose would be decreased by a factor of 2.5 from those that we calculate. It has been previously estimated (18,38) that a full body exposure of pale skin under high sun conditions (UVI = 10) produces 1000 IU in less than 1 min. We therefore adopt a set of reference conditions as follows:

UVI_0 = 10 (peak UV for mid-latitudes in the northern hemisphere);
R_0 = 2 (see Fig. 4);
T_{D0} = 1 min (from previous paragraph);
A_0 = 1 (full body exposure);
MEDF_0 = 2.5 (skin type II); and
SPF_0 = 1 (no sunscreen applied).

If the time taken to produce a specific increase in vitamin D resulting from one given set of reference conditions (0) is known, then the time for any other set of conditions can be deduced by

$$t_{\mathrm{D}} = t_{\mathrm{D0}}\frac{\mathrm{UVI}_0}{\mathrm{UVI}}\frac{R_0}{R}\frac{A_0}{A}\frac{\mathrm{MEDF}}{\mathrm{MEDF}_0}\frac{\mathrm{SPF}}{\mathrm{SPF}_0} \qquad (3)$$

If we assume the same skin type, body area exposed and no sunscreens, the exposure time is just

$$t_{\mathrm{D}} = t_{\mathrm{D0}}\frac{\mathrm{UVI}_0}{\mathrm{UVI}}\frac{R_0}{R} \qquad (4)$$

The only troublesome part of the expression is the value for R, which varies from approximately 2 to 0.5. However, on the basis that very little vitamin D is produced for high SZA, the effective range of R that needs to be considered is between 1 and 2. Figure 5a shows how R varies as a function of UVI, as calculated from the spectral measurements at Lauder. It can be seen that for each UVI value there is a range of possible values of R. The range becomes larger at smaller values of UVI. The lowest values for each UVI correspond to times when the SZA is smaller, or when ozone amounts are greater. Here, we consider the lower limit of the envelope of these values to estimate the maximum time needed to produce a given dose of vitamin D in the worst case. In reality, the required exposure times for smaller UVI values could be as little as half of the calculated values using this procedure.

The required exposure times for other conditions can then be derived. As shown by Fig. 5b, the corresponding relationship between SZA and UVI is variable, as it depends also on ozone amount, Sun–Earth separation and air clarity. However, taking the extreme cases

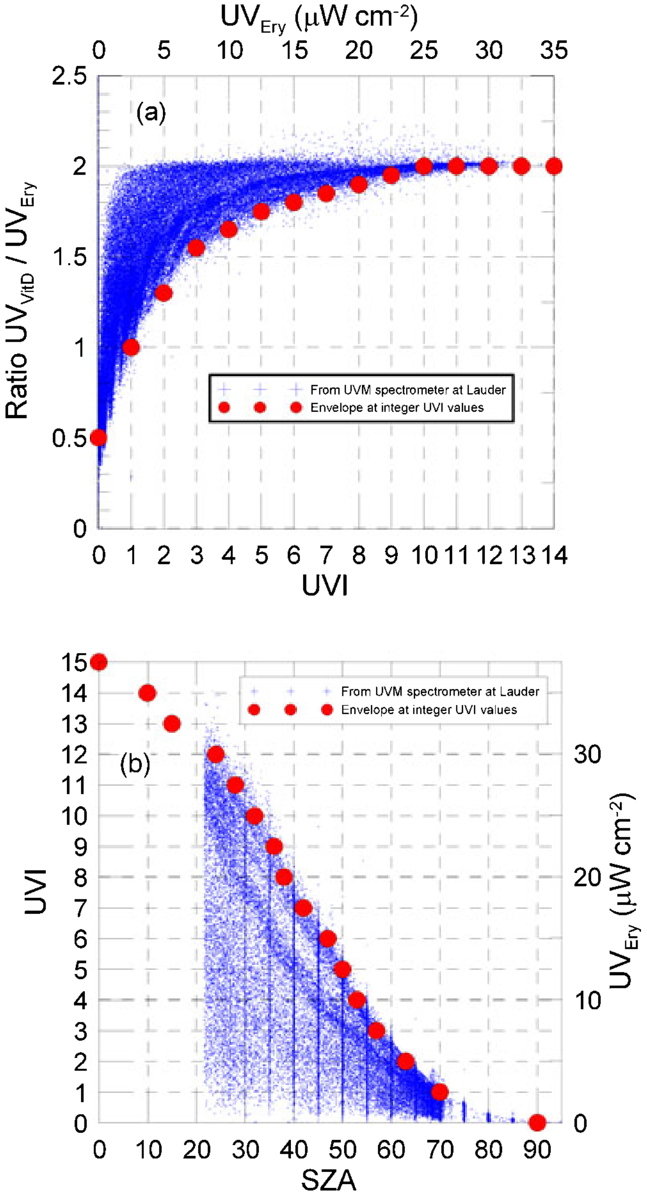

Figure 5. Scatterplots showing relationships between (a) the ratio $\mathrm{UV_{VitD}}/\mathrm{UV_{Ery}}$ and UVI and (b) the relationship between UVI and SZA. The small points are from UVM spectrometer measurements at Lauder, and the larger circles are empirical fits to the outer envelopes of those data.

from our UV climatology (Fig. 5b) still gives a useful guide for the maximum UVI expected in each case. Model calculations were used to extrapolate for SZA < 22°, which is the minimum SZA at Lauder. For sites in the northern hemisphere, these peak UVI values decrease by 1–2 UVI units.

A threshold condition for producing sufficient vitamin D without inducing erythema can then be estimated by equating the time to induce erythema (t_E) with the time required to synthesize sufficient vitamin D (t_D). Then, assuming that the UV blocking capacity of skins and sun block creams is similar over the ranges of erythema and vitamin D production, we can calculate the minimum skin area exposure that is necessary to produce enough vitamin D without inducing erythema as

$$A = 0.12\frac{t_{D0}}{R}, \tag{5}$$

where R is the ratio as before, and t_{D0} is the time to synthesize sufficient vitamin D with whole-body exposure of skin type II to UVI of 10.

Note that although this relationship depends on a calibration at a given skin type, it applies to all skin types on the above assumptions.

RESULTS

Comparing exposure times for sufficient vitamin D *versus* erythema

Results of the calculations described in the previous section are summarized in Table 2. Because UVI information is not always available, we also provide these times as more approximate functions of tan(SZA), which corresponds to the ratio of one's shadow length to one's height. For small SZA (tan[SZA] < 0.5), your shadow length is less than half your height and the UVI exceeds 10 (*i.e.* UVI "extreme") (1). For SZA = 45°, your shadow length is as long as your body (tan[SZA] = 1), and the UVI is 6–7 (*i.e.* UVI "high"). For SZA = 63°, your shadow is twice as long as your body (tan[SZA] = 2): a vertical 1 m ruler casts a shadow of length 2 m. For that particular SZA, which corresponds to noon on a mid-winter day at latitude ~41°, the UVI is typically between 2 and 3 (*i.e.* UVI "low"), and the ratio of UV_{VitD}/UV_{Ery} is about 1.6. The time to produce a MED for skin type II is between 1 and 2 h, and the time to achieve 1000 IU is about 6 min for full body exposure (A = 1.0), and about 1 h if only the hands and face are exposed (A = 0.1).

Table 2. Range of UVI and corresponding exposure times for erythema and for photosynthesizing optimal vitamin D for skin type II, where 1 MED = 2.5 SED (58).

UVI	Approx. SZA	Shadow multiplier (tan[SZA])	Multiplier (ratio VitD/Ery)	Time for erythema (min)	Time for 1000 IU VitD (min)	
					Full body (100%)	Face and hands (10%)
1	70	2.7	1	180	20	200
2	63	2.0	1.3	120	7.7	77
3	57	1.5	1.55	60	4.3	43
4	53	1.3	1.65	45	3.0	30
5	50	1.2	1.75	36	2.3	23
6	47	1.1	1.8	30	1.9	19
7	42	0.9	1.85	26	1.5	15
8	38	0.8	1.9	22	1.3	13
9	36	0.7	1.95	20	1.1	11
10	32	0.6	2.0	18	1.0	10
11	28	0.5	2.0	16	0.9	9
12	24	0.4	2.0	15	0.83	8.3
13	15	0.3	2.0	14	0.8	8
14	10	0.2	2.0	13	0.71	7.1
15	0	0.0	2.0	12	0.67	6.7

For lighter skins, the times should be decreased to ~0.7 of these values. For skin type IV, the time should be increased by a factor of 2, while for skin type V or VI (black) the times should be increased by up to a factor of ~5–10 (32). MED = minimum erythemal dose.

These times are shown in Fig. 6. The area shaded at the top right gives times when erythema occurs on exposed skin for each UVI value. The area shaded at the bottom left gives the times when there is insufficient UV to maintain optimal levels of vitamin D, even for full body exposures. The other three curves give the approximate exposure times needed to maintain vitamin D for different areas of the body exposed. These fractional body areas were estimated using the so-called "rule of nines" guidelines recommended by St John Ambulance for assessing burns (39). Exposing only the head corresponds to 9% of the body area (A = 0.09, but we will use face and hands as 10%); face, hands and arms corresponds to 27% (A = 0.27); face, hands, arms and legs corresponds to 63% (A = 0.63). Figure 6 shows that for full body exposures, there is a wide window between the time for sufficient UV and the time for too much UV. As the fraction of body that is exposed decreases, the window of optimum UV exposure times also decreases. If only hands and face are exposed, there is generally only a small window between receiving insufficient UV for vitamin D production, and too much UV for skin damage (erythema).

DISCUSSION

Uncertainties and caveats

These optimal exposure times calculated above should be considered as only very approximate. In particular, there is some uncertainty regarding the applicability of the action spectrum for vitamin D, and the possible role of temperature in converting pre-vitamin D to vitamin D (40). If the action spectrum were confined to shorter wavelengths, then the R values would be reduced commensurately. For example, as shown in Fig. 4, if the upper limit were reduced from 330 to 315 nm, the R values are typically 5–10% lower, so the required exposure time would be increased by a similar proportion. There is also the question of how well the radiation received by the skin relates to that incident on a horizontal surface. A recent study (17) has addressed this issue by considering the radiation incident on a cylindrical surface, which for human exposure is usually a better representation than for the radiation on a horizontal surface. As the SZA increases, the differences become larger. When their latitudinal variations are compared with those used here (6), we find (not shown) that by latitude 70°, the dose on a cylindrical surface is ~60% more than on a horizontal surface. It is difficult to account for this uncertainty because the actual dose will depend on other factors, including body shape, clothing and posture, as well as the surface albedo and cloud cover. A better approximation may be to use actinic fluxes, which are independent of direction, rather than irradiances, which are a vector quantity. Unfortunately, there are few measurements of actinic fluxes available. They can be derived from irradiances, but there is an additional penalty in the uncertainty of

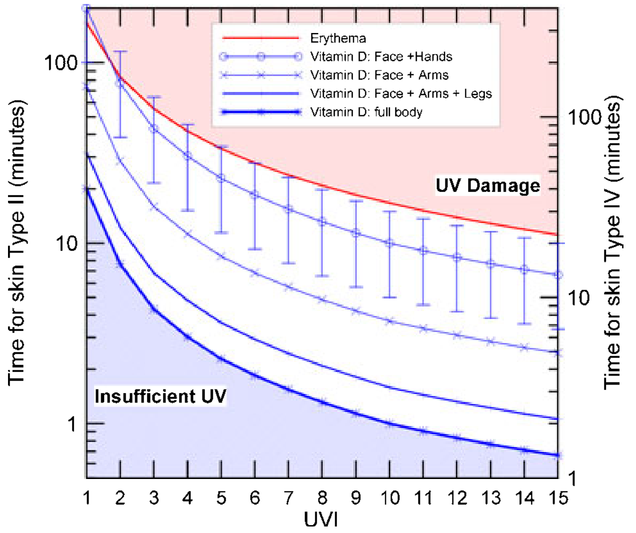

Figure 6. Range of exposure times required for optimal UV (white region), plotted as a function of UVI. The upper right shaded area represents times when you have received too much UV, leading to erythema (skin reddening). The lower left shaded region represents the time when you do not receive enough UV to maintain an intake of 1000 IU for full body exposure.

10–20% (41). Fortunately, for moderate SZA the effects of these geometric differences are relatively small, and their importance to this study is less important because they affect erythemally weighted and vitamin D-weighted radiation in similar proportions. We also note that the relationships derived here are from high quality spectral irradiance measurements; they are specific to a single location with its own particular conditions. Although we consider the results should be generally applicable elsewhere, the minimum *R* values do depend on the range of ozone variability. At locations where ozone amounts are higher (such as at high northern latitudes), the minimum *R* values would be smaller, and at locations with lower ozone amounts (such as within the tropics), the minimum *R* values would be larger.

Here, we have assumed that the blocking factors for skin (MEDF) and sun screens (SPF) are similar for UV_{Ery} and for UV_{VitD}. This is reasonable, since both are dominated by radiation within the UV-B region (see Fig. 1). There is some evidence that melanin in the skin blocks shorter wavelengths more efficiently than longer wavelengths (42–46). Similarly, many sunscreens block more efficiently at shorter wavelengths (47,48). In both of these cases, there would be a larger effect on vitamin D production than on protection from sunburn. A similar effect has been noted previously in the case of a sunscreen with SPF 15 for which the peak absorption occurs near 315 nm (49). However, the changes in absorption are relatively small over the UV-B region, and in the same sense for both effects. Consequently, their effect should be small.

As the UVI becomes smaller, it becomes more difficult to produce sufficient vitamin D without inducing erythema. For low sun conditions typical of midday in the mid-latitude winter (SZA ~65°), where *R* ~1, the skin area exposed must be more than 12% (see Eq. 5) This means that for these low sun conditions it is improbable that sufficient vitamin D can be produced from exposing the hands and face alone, since that area is less than 12% of the full body. In practice, the exposure could be somewhat larger for nonhorizontal surfaces, as discussed above. For still larger SZA, the minimum *R* value is ~0.5, so about 25% of the body would have to be exposed to produce sufficient vitamin D without erythema. However, that is of academic interest only, because at those low UVI values there is insufficient time in a day to produce either of those outcomes.

Two further caveats should be mentioned here. First, over periods longer than ~1 h, there will be substantial changes in the UVI, so the mean UVI value over the period should be applied. Secondly, it is implicitly assumed that reciprocity between time and UVI applies: meaning that a constant UVI of 10 for 1 h, say, would give the same erythemal effect as a constant UVI value of 1 for 10 h. This reciprocity has not been demonstrated for sunlight, although it does seem to apply for a range of UVI values greater than ~5 using artificial lamps that emit much higher proportions of UV-B and UV-C radiation (50).

Finally, we note that this margin between UV sufficiency (for vitamin D) and UV excess (for sunburn) depends critically on the assumed vitamin D cutaneous productivity. In Fig. 6, we assumed that 1 min of exposure to UVI = 10 provided sufficient UV to maintain vitamin D levels. In that case, whenever the UVI is less than 2, one cannot manufacture sufficient vitamin D from exposure to hands and face alone without inducing erythema. The results also depend on the accuracy of the assumed time for vitamin D sufficiency for high sun, which was taken here as 1 min. If, for example, the time was 1.5 min, then the vitamin D curves in Fig. 6 all move up, and the curve for exposure to hands and face only intersects the UV damage curve for when UVI = 6. Conversely, if lower doses of vitamin D are sufficient, then the curves move downward. The curve for exposures to hands and face includes estimated error bars to encompass these uncertainties. Percentage errors are similar for the other curves, but have been omitted for clarity.

Production of vitamin D from sunlight

While there is considerable uncertainty regarding the wavelength dependence for vitamin D production, evidence suggests that there is insufficient vitamin D produced in the winter at latitudes pole-ward of about 40° (51). Based on the statement that no vitamin D is produced in the winter at Boston, MA, we may take an upper limit threshold for insufficient vitamin D production as the daily available dose at the latitude of Boston, 42°N. This threshold is about 0.7 kJ m^{-2} per day (see Fig. 3). Under the same conditions, UV_{Ery} dose is ~0.55 kJ m^{-2} per day, with a corresponding peak UVI = 1.3 at noon. Yet at the same location in summer, the UV_{Ery} dose can exceed 5 kJ m^{-2}, with corresponding peak UVI values reaching ~9. The only places where the minimum required vitamin D-weighted is exceeded throughout the winter months are at latitudes between 40°S and 40°N. On the other hand, the peak UVI at all of these latitudes can often exceed 10 (*i.e.* "extreme" UV) during the summer months, so at some times of the year there is a risk of skin damage in times as short as 15–20 min. Surprisingly therefore, it appears that there is no "Goldilocks" region on the planet which is "just right": where there is no risk of sunburn in summer, yet ample UV for vitamin D production in the winter. Public advice regarding personal behavior in response to UV variability needs further development, and should recognize both the benefits and the risks of UV exposure.

An inconsistency

There seems to be an inconsistency with the CIE spectrum for vitamin D (26) and the statements regarding our inability to photosynthesize vitamin D in winter, namely that

- a few minutes' daily exposure to sunlight in summer is sufficient at mid-latitudes D (18,38) and
- no vitamin D is produced in Boston in winter (51)

The first criterion above is based on the statement that 1 MED full body exposure corresponds to an oral dose of 10 000–25 000 IU of vitamin D. For a fair skinned person, 1 MED (*i.e.* 2.5 SED) is accumulated in ~14 min when UVI = 12 (UV_{Ery} = 0.3 W m^{-2}). The vitamin D produced from this is more than 10 times the recommended daily dose of up to ~1000 IU (18,22,35,36). As we have noted above, this implies that a full body exposure of ~1 min should suffice to meet daily requirements in the summer. In the winter, the UV_{VitD} incident on a horizontal surface is approximately 1/20th of the summer value at mid-latitudes. And at these larger SZAs, the radiation on more realistic body-surface geometries could be significantly higher as discussed above, so sufficient vitamin D should easily be produced in less than 20 min of full body exposure. Even if there were an error of 300% in this calculation, sufficient vitamin D should be produced in less than 1 h. For darker skinned individuals, the exposure time required would be longer, but the amount of vitamin D produced should not be zero. This inconsistency remains, regardless of whether the long wavelength limit of the action spectrum for vitamin D extends to 315 or 330 nm.

There is, of course, the question of whether individuals would be prepared to expose a large enough area of their bodies at the low temperatures in winter. They probably would not. However, even for more limited exposures, the vitamin D produced would be non-zero. The experiments to determine the action spectrum of pre-vitamin D did not use live subjects. Instead, they used samples of skin tissue exposed in a Petri dish (51). It is surprising that these did not yield any vitamin D, since these exposure periods were for 3 h over the midday period. This raises the question of whether the action spectrum has been specified sufficiently. For example, it is known that the conversion from pre-vitamin D to vitamin D is temperature dependent (18,52), so it is reasonable to assume some temperature dependence in the overall conversion from sunlight to blood serum vitamin 25(OH)D. Although the temperature-dependent reaction takes place in the skin, the temperature can vary significantly from normal body temperature of 37°C. Some have suggested that there is a threshold below which vitamin D is not produced (53). But the evidence for this is not strong. More probably, any perceived threshold is actually caused by an inability to detect the smaller amounts produced.

Notwithstanding the above arguments, there is ample evidence that individuals do not receive sufficient UV to maintain optimal vitamin D (18–23). This may be in part because of our modern lifestyles, where outdoor exposure is rare, even in the summer months. A recent study in New Zealand found that schoolchildren typically received less than 5% of the available UV dose, even in the summer (54). Another study in Germany found that even outdoor workers receive only 5–10% of the available dose, which is a factor of five more than indoor workers (55). More work is clearly needed to characterize the relationship between sunlight and vitamin D status.

Behavioral strategy

Optimally, individuals should attempt to receive the minimum dose that provides the necessary vitamin D. It is not necessary to receive this dose every day, because vitamin D has a residence time in the body of a few weeks (24,35,56,57). But the average daily dose over a period of a week or two should match the values calculated here. During the winter, when cold temperatures may preclude exposures of large areas of skin, it may not be possible to receive adequate UV. When exposures are limited to the hands and face, there is a relatively small margin of error between getting sufficient UV for vitamin D production, and not getting too much for sunburn. The calculations show that the best advice would be to expose as much area as possible for the minimum time necessary. For high sun conditions, the time for skin damage is about twice the time for sufficient vitamin D production if only the hands and face are exposed. And when the UVI is 1 or less, it is not possible to get sufficient UV for vitamin D without acquiring a mild erythemal dose.

In order to make sensible choices on sun exposure, the public needs knowledge of the UV environment as it relates to sunburn and to vitamin D production. Currently, the necessary information is not generally available to the public. If UV information is available at all, it is usually confined only to the summer months, and is provided for clear sky conditions. Further, often only the peak daily value is provided. This is because the UVI was originally designed only to give the risk of skin damage. However, since there is a close relationship between UV_{Ery} and UV_{VitD}, the UVI scale can also be used as a proxy for optimal guidance as far as vitamin D is concerned. Although, the relationship is not a simple direct proportionality, some simple "rules of thumb" can be used as a guide, as discussed above. This approach is probably preferable to introducing yet another index for the public to understand.

CONCLUSION

There are huge geographical and seasonal differences in UV, which have more important implications for health than any trends due to ozone depletion. Highest UV intensities occur in the tropics, but latitude for latitude, the peak UV intensities are relatively much higher in the southern hemisphere.

There are even larger geographical and seasonal variabilities for beneficial UV. The ratio between the doses of UV_{VitD} and UV_{Ery} depends on arbitrary normalizations of the weighting functions as well as on ozone and SZA. With the normalizations adopted by the CIE, UV_{VitD} is approximately twice UV_{Ery} in summer, but the two are approximately equal for mid-latitude winter. At high latitudes in the winter hemisphere, UV_{Ery} becomes larger than UV_{VitD}. There is no place on the planet where UVI is "optimum" all year round.

Despite these departures from proportionality at larger SZA, UV_{VitD} can still be estimated from knowledge of UV_{Ery} (or UVI). We have used measured spectral irradiances to develop a simple algorithm to estimate vitamin D production from UVI. The algorithm has been applied to determine optimum conditions of UV exposure.

The production of vitamin D from sunlight is dominated by the midday period when UV intensities are at a maximum. When the sun is high in the sky, such as near noon at mid to low latitudes in the summer, sufficient vitamin D can be produced from a few minutes of sun exposure to the face and hands. But the exposure time should be limited to less than about 15 min to avoid erythema. A better strategy would be to expose a larger fraction of the body for a shorter time period, preferably when the sun is lower in the sky to allow a greater margin of error. When the UVI is 3, skin damage occurs after approximately 1 h, but sufficient vitamin D can still be produced in a few minutes.

The results of this study imply that there is sufficient UV radiation available in the mid-latitude winter to produce sufficient vitamin D. However, under those conditions it is necessary to expose larger areas than hands and face alone. Because of the low temperatures, this proviso sets a practical limit on our ability to maintain adequate levels of vitamin D in the winter: a situation which is exacerbated by our modern lifestyle in which periods spent outdoors are greatly diminished. The situation could be improved by promoting physically strenuous outdoor activities, such as jogging, during the midday period in winter, but it is likely that there would remain a problem from overexposure during the shoulder seasons either side of winter, when UVI values increase rapidly. During the winter at mid-latitudes, most people will probably require supplementation of vitamin D from other sources. These could be dietary (*e.g.* increased consumption of oily fish or from vitamin D supplements), or from exposure to higher UV intensities from holidays abroad or from artificial sources. The latter options carry risks of overexposure.

For the public to be able to make informed decisions about appropriate "sun-smart" behavior to maintain vitamin D status without inducing erythema, it is essential that they have access to UV information throughout the year, throughout the day, and for all sky conditions Until better information is available, our table relating shadow length to optimal exposure times may be a useful practical guide.

Finally, the results point to an inconsistency between the action spectrum of vitamin D and statements that have been made about the production of vitamin D. In particular, if the rate of production stated for the summer is correct, then it should be possible to produce vitamin D at mid-latitudes in the winter, contrary to the current advice. The calculations here show that current advice to the public has also been overly simplistic and inadequate.

Acknowledgements—The authors are grateful to Michael Kotkamp of NIWA Lauder for maintaining the spectral UV observations and Dan Smale of NIWA Lauder for carrying out the RT calculations with the TUV model. We also thank Dr. Sasha Madronich of NCAR for providing the UV climatologies and Dr. Robert Scragg of the University of Auckland for helpful suggestions.

APPENDIX

Calculated factors, as functions of ozone and SZA, to convert from UV_{Ery} to UV_{VitD}, assuming, respectively (a) the CIE action spectrum (to 330 nm) (26) and (b) a modified version of the CIE action spectrum, truncated to 315 nm.

(a) Using the CIE action spectrum (to 330 nm).

Z/Toz	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95
100	1.79	1.79	1.79	1.79	1.81	1.83	1.83	1.85	1.89	1.91	1.93	1.97	1.99	2.01	2.01	1.93	1.79	1.56	1.44	1.58
150	1.93	1.93	1.93	1.95	1.95	1.97	1.97	1.99	2.01	2.01	2.03	2.03	2.01	1.95	1.85	1.69	1.44	1.18	1.14	1.34
200	2.01	2.01	2.01	2.01	2.03	2.03	2.03	2.03	2.03	2.01	1.99	1.95	1.89	1.79	1.64	1.40	1.12	0.89	0.91	1.10
250	2.03	2.03	2.03	2.03	2.03	2.03	2.03	2.01	1.99	1.95	1.91	1.83	1.73	1.60	1.40	1.14	0.89	0.69	0.71	0.91
300	2.01	2.01	2.01	2.01	2.01	1.99	1.97	1.95	1.91	1.85	1.79	1.69	1.56	1.38	1.18	0.93	0.69	0.53	0.57	0.73
350	1.97	1.97	1.97	1.95	1.95	1.93	1.89	1.85	1.81	1.73	1.65	1.54	1.38	1.20	0.99	0.77	0.55	0.41	0.45	0.61
400	1.91	1.91	1.89	1.89	1.87	1.83	1.79	1.75	1.69	1.62	1.52	1.38	1.22	1.04	0.83	0.63	0.43	0.33	0.37	0.51
450	1.83	1.83	1.81	1.79	1.77	1.73	1.69	1.65	1.58	1.48	1.38	1.24	1.08	0.91	0.71	0.51	0.35	0.28	0.32	0.41
500	1.73	1.73	1.73	1.71	1.67	1.65	1.60	1.54	1.46	1.36	1.24	1.12	0.97	0.79	0.61	0.43	0.30	0.22	0.26	0.35
550	1.65	1.65	1.64	1.62	1.60	1.56	1.50	1.44	1.34	1.26	1.14	1.00	0.85	0.69	0.51	0.35	0.24	0.20	0.22	0.30
600	1.56	1.56	1.56	1.52	1.50	1.46	1.40	1.32	1.24	1.14	1.02	0.89	0.75	0.59	0.43	0.30	0.20	0.16	0.18	0.26

(b) Using a modified version of the CIE action spectrum, truncated to 315 nm.

Z/Toz	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95
100	1.75	1.75	1.75	1.77	1.77	1.79	1.80	1.82	1.84	1.86	1.88	1.92	1.93	1.93	1.92	1.84	1.67	1.41	1.30	1.47
150	1.88	1.88	1.88	1.90	1.90	1.92	1.92	1.93	1.93	1.95	1.95	1.93	1.90	1.84	1.73	1.54	1.28	1.02	1.00	1.21
200	1.95	1.95	1.95	1.95	1.95	1.95	1.95	1.95	1.93	1.93	1.90	1.84	1.77	1.66	1.47	1.23	0.95	0.73	0.78	0.97
250	1.95	1.95	1.95	1.95	1.95	1.93	1.93	1.92	1.88	1.84	1.79	1.71	1.58	1.43	1.23	0.97	0.71	0.52	0.60	0.78
300	1.92	1.92	1.92	1.92	1.90	1.88	1.86	1.82	1.79	1.73	1.64	1.54	1.40	1.21	0.99	0.74	0.52	0.39	0.47	0.61
350	1.86	1.86	1.86	1.84	1.82	1.80	1.77	1.73	1.67	1.60	1.49	1.38	1.21	1.02	0.80	0.58	0.37	0.30	0.37	0.50
400	1.79	1.79	1.77	1.75	1.73	1.71	1.66	1.60	1.54	1.45	1.34	1.21	1.04	0.86	0.65	0.45	0.28	0.22	0.30	0.39
450	1.69	1.69	1.67	1.66	1.64	1.60	1.54	1.49	1.41	1.32	1.19	1.06	0.89	0.71	0.52	0.33	0.20	0.17	0.24	0.32
500	1.60	1.60	1.58	1.56	1.53	1.49	1.43	1.38	1.28	1.19	1.06	0.93	0.76	0.60	0.41	0.26	0.17	0.13	0.19	0.26
550	1.49	1.49	1.49	1.45	1.43	1.38	1.32	1.25	1.17	1.06	0.95	0.80	0.65	0.48	0.33	0.20	0.13	0.11	0.15	0.22
600	1.40	1.40	1.38	1.36	1.32	1.28	1.21	1.15	1.06	0.95	0.84	0.71	0.56	0.41	0.28	0.17	0.09	0.09	0.13	0.19

REFERENCES

1. WHO (2002) *Global Solar UV Index: A Practical Guide*. World Health Organisation (WHO), World Meteorological Organisation (WMO), United Nations Environment Program (UNEP), and International Commission on Non-Ionising Radiation Protection (ICNRP), Geneva.
2. WMO (2007) *Scientific Assessment of Ozone Depletion: 2006*. World Meteorological Organisation Global Research and Monitoring Project, Report No. 50, Geneva.
3. UNEP (2007) Environmental effects of ozone depletion and its interactions with climate change: 2006 assessment. *Photochem. Photobiol. Sci.* **6**, 201–332.
4. UNEP (2006) Environmental effects of ozone depletion and its interactions with climate change: Progress report, 2005. *Photochem. Photobiol. Sci.* **5**, 13–24 (DOI: 10.1039/b515670 j).
5. Kerr, J. B., G. Seckmeyer, A. F. Bais, G. Bernhard, M. Blumthaler, S. B. Diaz, N. A. Krotkov, D. Lubin, R. L. McKenzie, A. A. Sabziparvar and J. Verdebout (2003) Surface ultraviolet radiation: Past and future. In *Chapter 5 of WMO Scientific Assessment of Ozone Depletion: 2002* (Edited by A.-L. Ajavon, N. D. L. Albritton, G. Megie and R. T. Watson). World Meteorological Organization, Global Ozone Research and Monitoring Project, Report No. 47, Geneva.
6. Lee-Taylor, J. and S. Madronich (2007) *Climatology of UV-A, UV-B, and Erythemal Radiation at the Earth's Surface, 1979-2000. NCAR Technical Note* TN-474 + STR, pp. 52. NCAR, Boulder, CO.
7. Liley, J. B. and R. L. McKenzie (2006) Where on Earth has the highest UV? In *UV Radiation and its Effects: An Update* (Edited by R. L. McKenzie and E. Davis for NIWA and RSNZ), pp. 36–37. Royal Society of New Zealand, Wellington, New Zealand. Available at: http://www.niwascience.co.nz/rc/atmos/uvconference. Accessed on 10 June 2008.
8. McKenzie, R., D. Smale, G. Bodeker and H. Claude (2003) Ozone profile differences between Europe and New Zealand: Effects on surface UV irradiance and its estimation from satellite sensors. *J. Geophys. Res.* **108**, 4179 (DOI: 4110.1029/2002JD002770).
9. McKenzie, R. L., G. Seckmeyer, A. F. Bais, J. B. Kerr and S. Madronich (2001) Satellite retrievals of erythemal UV dose compared with ground-based measurements at northern and southern midlatitudes. *J. Geophys. Res.* **106**, 24051–24062.
10. Grant, W. B., C. F. Garland and M. F. Holick (2005) Comparisons of estimated economic burdens due to insufficient solar ultraviolet irradiance and vitamin D and excess solar UV irradiance for the United States. *Photochem. Photobiol.* **81**, 1276–1286.
11. Berwick, M. and D. Kesler (2005) Ultraviolet radiation exposure, vitamin D, and cancer. *Photochem. Photobiol.* **81**, 1261–1266.
12. Godar, D. E. (2005) UV doses worldwide. *Photochem. Photobiol.* **81**, 736–749.
13. Gandini, S., F. Sera, M. S. Cattaruzza, P. Pasquini, O. Picconi, P. Boyle and C. F. Melchi (2005) Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. *Eur. J. Cancer* **41**, 45–60.
14. Livesey, J., P. Elder, J. Ellis, R. McKenzie, B. Liley and C. Florkowski (2007) Seasonal variation in vitamin D levels in the Canterbury population in relation to available UV radiation. *NZ Med. J.* **120**(1262): http://www.nzma.org.nz/journal/120-1262/2733/.
15. Brustad, M., K. Edvardsen, T. Wilsgaard, O. Engelsen, L. Aksnesc and E. Lunda (2007) Seasonality of UV-radiation and vitamin D status at 69 degrees north. *Photochem. Photobiol. Sci.* **6**, 903–908.
16. Davies, P., C. Bates, T. Cole, A. Prentice and P. Clarke (1999) Vitamin D: Seasonal and regional differences in preschool children in Great Britain. *Eur. J. Clin. Nutr.* **53**, 195–198.
17. Moan, J., A. C. Porojnicu, A. Dahlback and R. B. Setlow (2008) Addressing the health benefits and risks, involving vitamin D or skin cancer, of increased sun exposure. *Proc. Natl Acad. Sci. USA* **105**, 668–673.
18. Holick, M. F. (2007) Vitamin D deficiency. *N. Engl. J. Med.* **357**, 266–281.
19. Gorham, E. D., C. F. Garland, F. C. Garland, W. B. Grant, S. B. Mohr, M. Lipkin, H. L. Newmark, E. Giovannucci, M. Wei and M. F. Holick (2007) Optimal vitamin D status for colorectal cancer prevention. A quantitative meta analysis. *Am. J. Prev. Med.* **32**, 210–216.
20. Vieth, R. (2006) Critique of the considerations for establishing the tolerable upper intake level for vitamin D: Critical need for revision upwards. *J. Nutr.* **136**, 1117–1122.
21. Gillie, O. (2006) A new government policy is needed for sunlight and vitamin D. *Br. J. Dermatol.* **154**, 1052–1061.
22. Bischoff-Ferrari, H. A., E. Giovannucci, W. C. Willett, T. Dietrich and B. Dawson-Hughes (2006) Estimation of optimal serum concentrations of 25-hydroxyvitamin D for multiple health outcomes. *Am. J. Clin. Nutr.* **84**, 18–28.
23. Young, A. R. and S. L. Walker (2005) UV radiation, vitamin D and human health: An unfolding controversy introduction. *Photochem. Photobiol.* **81**, 1243–1245.
24. Cannell, J., B. Hollis, M. Zasloff and R. Heaney (2008) Diagnosis and treatment of vitamin D deficiency. *Expert Opin. Pharmacother.* **9**, 1–12.
25. McKinlay, A. F. and B. L. Diffey (1987) A reference action spectrum for ultra-violet induced erythema in human skin. In *Human Exposure to Ultraviolet Radiation: Risks and Regulations* (Edited by W. F. Passchier and B. F. M. Bosnajakovic), pp. 83–87. Elsevier, Amsterdam.
26. Bouillon, R., J. Eisman, M. Garabedian, M. Holick, J. Kleinschmidt, T. Suda, I. Terenetskaya and A. Webb (2006) *Action Spectrum for the Production of Previtamin D3 in Human Skin*. UDC: 612.014.481-06, CIE, Vienna.
27. Kimlin, M. G., W. J. Olds and M. R. Moore (2007) Location and vitamin D synthesis: Is the hypothesis validated by geophysical data? *J. Photochem. Photobiol. B* **86**, 234–239.
28. Madronich, S. and S. Flocke (1995) Theoretical estimation of biologically effective UV radiation at the earth's surface In *Solar Ultraviolet Radiation* (Edited by C. S. Zerefos and A. F. Bais), pp. 23–48. NATO Series I: Advanced Study Institute. Springer, Berlin.
29. McKenzie, R., D. Smale and M. Kotkamp (2004) Relationship between UVB and erythemally weighted radiation. *Photochem. Photobiol. Sci.* **3**, 252–256.
30. McKenzie, R. L. (2007) UV radiation climatology and trends (Invited presentation). In *One Century of UV Radiation Research* (Edited by J. Grobner), pp. 13–14. PMOD/WRC, Davos, Switzerland.
31. MacLaughlin, J. A., R. R. Anderson and M. F. Holick (1982) Spectral character of sunlight modulates photosynthesis of previtamin D3 and its photoisomers in human skin. *Science* **216**, 1001–1003.
32. Fitzpatrick, T. B. (1988) The validity and practicality of Sun-reactive skin types I through VI. *Arch. Dermatol.* **124**, 869–871.
33. Nielsen, K. P., L. Zhao, J. J. Stamnes, K. Stamnes and J. Moan (2006) The importance of the depth distribution of melanin in skin for DNA protection and other photobiological processes. *J. Photochem. Photobiol. B* **82**, 194–198.
34. Armas, L. A. G., S. Dowell, M. A. RN, S. Duthuluru, C. Huerter, B. W. Hollis, R. Lund and R. P. Heaney (2007) Ultraviolet-B radiation increases serum 25-hydroxyvitamin D levels: The effect of UVB dose and skin color. *J. Am. Acad. Dermatol.* **57**, 588–593.
35. Chel, V. G. M., M. E. Ooms, C. Popp-Snijders, S. Pavel, A. A. Schothorst, C. C. E. Meulemans and P. Lips (1998) Ultraviolet irradiation corrects vitamin D deficiency and suppresses secondary hyperparathyroidism in the elderly. *J. Bone Miner. Res.* **13**, 1238–1242.
36. Vieth, R., H. Bischoff-Ferrari, B. J. Boucher, B. Dawson-Hughes, C. F. Garland, R. P. Heaney, M. F. Holick, B. W. Hollis, C. Lamberg-Allardt, J. J. McGrath, A. W. Norman, Robert. Scragg, S. J. Whiting, W. C. Willett and A. Zittermann (2007) The urgent need to recommend an intake of vitamin D that is effective. *Am. J. Clin. Nutr.* **85**, 649–650.
37. Wolpowitz, D. and B. A. Gilchrest (2006) Clarifying the vitamin D controversy: The health benefits of supplementation by diet versus sunshine. In *Skin Aging*, Edited by B. A. Gilchrest and J. Krutmann), pp. 81–102. Springer, Berlin Heidelberg.
38. Holick, M. F. (2002) Vitamin D: The underappreciated D-lightful hormone that is important for skeletal and cellular health. *Curr. Opin. Endocrinol. Diab.* **8**, 87–98.

39. British Red Cross (2002) *First Aid Manual: The Authorised Manual of St John Ambulance, St Andrew's Ambulance Association of the British Red Cross*. Dorling Kindersley, London.
40. Matsuoka, L. Y., J. Wortsman, J. G. Haddad and B. W. Hollis (1989) In vivo threshold for cutaneous synthesis of vitamin-D3. *J. Lab. Clin. Med.* **114**, 301–305.
41. Blumthaler, M., B. Schallhart, M. Schwarzmann, R. McKenzie, P. Johnston, M. Kotkamp and H. Shiona (2008) Spectral UV measurements of global irradiance, solar radiance, and actinic fluxes in New Zealand: Inter comparison between instruments and model calculations. *J. Oceanic Atmos. Sci. Technol.* **25**(6), 945–958.
42. Jacques, S. L. (1998) Skin Optics. Available at http://omlc.bme.ogi.edu/news/jan98/skinoptics.html. Accessed on 10 June 2008.
43. Meinhardt, M., R. Krebs, A. Anders, U. Heinrich and H. Tronnier (2008) Effect of UV adaptation on the UV absorption spectra of human skin in vivo. *Photodermatol. Photoimmunol. Photomed.* **24**, 76–82.
44. Meinhardt, M., R. Krebs, A. Anders and U. H. Tronnier (2008) Absorption spectra of human skin in vivo in the ultraviolet wavelength range. *Photochem. Photobiol.* doi: 10.1111/j.1751-1097.2008.00398.x (In press).
45. Kaxiras, E., A. Tsolakidis, G. Zonios and S. Meng (2006) Structural model of eumelanin. *Phys. Rev. Lett.* **97**, 1–4.
46. Tran, M. L., B. J. Powell and P. Meredith (2006) Chemical and structural disorder in eumelanins: A possible explanation for broadband absorption. *Biophys. J.* **90**, 743–752.
47. Damiani, E., L. Rosati, R. Castagna, P. Carloni and L. Greci (2006) Changes in ultraviolet absorbance and hence in protective efficacy against lipid peroxidation of organic sunscreens after UVA irradiation. *J. Photochem. Photobiol. B* **82**, 204–213.
48. Diffey, B. L., P. R. Tanner, P. J. Matts and J. F. Nash (2000) In vitro assessment of the broad-spectrum ultraviolet protection of sunscreen products. *J. Am. Acad. Dermatol.* **43**, 1024–1035.
49. Sayre, R. M. and J. C. Dowdy (2007) Darkness at noon: Sunscreens and vitamin D3. *Photochem. Photobiol.* **83**, 459–463.
50. Meanwell, E. F. and B. Diffey (1989) Reciprocity of ultraviolet erythema in human skin. *Photodermatology* **6**, 146–148.
51. Webb, A. R., L. Kline and M. F. Holick (1988) Influence of season and latitude on the cutaneous synthesis of vitamin D3: Exposure to winter sunlight in Boston and Edmonton will not promote vitamin D3 synthesis in human skin. *J. Clin. Endocrinol. Metab.* **67**, 373–378.
52. Holick, M. F., X. Q. Tian and M. Allen (1995) Evolutionary importance for the membrane enhancement of the production of vitamin D3 in the skin of poikilothermic animals. *Proc. Natl Acad. Sci. USA* **92**, 3124–3126.
53. Hollis, B. W. (2005) Circulating 25-hydroxyvitamin D levels indicative of vitamin D sufficiency: Implications for establishing a new effective dietary intake recommendation for vitamin D1. *J. Nutr.* **135**, 317–322.
54. Wright, C. Y., A. I. Reeder, G. E. Bodeker, A. Gray and B. Cox (2007) Solar UVR exposure, concurrent activities and sun-protective practices among primary schoolchildren. *Photochem. Photobiol.* **83**, 749–758.
55. Knuschke, P., I. Unverricht, G. Ott and M. Jansen (2007) *Personenbezogene Messung der UV-Exposition von Arbeitnehmern im Freien*. Forschung Projekt F 1777, pp. 195. Bundesanstalt fur Arbeitsschutz und Arbeitsmedian (BAUA), Dortmund/Berlin/Dresden.
56. Zittermann, A. (2003) Vitamin D in preventive medicine: Are we ignoring the evidence? *Br. J. Nutr.* **89**, 552–572 (DOI: 510.1079/BJN2003837).
57. Holick, M. F. and M. Jenkins (2003) *The UV Advantage*. I Books, New York.
58. ANZS Standards Association of Australia and New Zealand (2002) *Solaria for Cosmetic Purposes*, pp. 14. AS/NZS 2635:2002, Wellington.