

Erythema versus vitamin D production from sunlight and solaria

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Abstract. We compare production rates of vitamin D as a function of sun-burning radiation for two types of dermatological UV chambers, and for summer and winter sunlight. The main results presented here are based on experimental studies where the blood serum vitamin D of participants is measured before and after receiving multiple doses of UV radiation from two different UV booths. The relative increases in vitamin D between different lamps are inconsistent with the action spectrum for vitamin D published by the CIE, and suggest that the true action spectrum is confined to shorter wavelengths in the UV region. However, further measurements with low doses from each lamp are needed to verify that hypothesis. However, see **addendum** note added after presentation.

Introduction

A substantial contribution to our knowledge about the photochemical production of vitamin D has arisen from work carried out by Holick and his co-workers at Boston (42°N) since the 1980s. In addition to deriving the action spectrum for Vitamin D production that was recently adopted by the CIE (Bouillon et al. 2006), they have also shown that in summer at noon (UVI > 10), sufficient vitamin D is produced in fair skinned individuals after ~1 minute of full body exposure to sunlight, or ~10 minutes if just the hands and face are exposed, but that no vitamin D is produced during the winter months at mid-latitude sites such as Boston (when UVI < 2) (Webb et al. 1988). However, a recent study has challenged these findings (McKenzie, et al., 2009). Using spectral measurements taken at Lauder New Zealand (45°S), it was found that some vitamin D should be produced when UV intensities are similar to the Boston winter.

Spectral Distributions and Weighted Irradiances

Figure 1 shows the action spectra for erythema (McKinlay & Diffey 1987) and vitamin D production (Bouillon et al. 2006), as published by the CIE. The lower panel shows the ratio of these two weighting functions. For wavelengths less than 298 nm, or greater than 317 nm the ratio is less than one. But its peak value, near 308 nm, is nearly 3.5. Consequently, a monochromatic source centred at 308 nm would produce approximately twice as much vitamin D per unit of sun burning radiation as a monochromatic source centred near 301 nm or 313.5 nm; or four times as much as a monochromatic source centred at 317 nm. Here we compare the measured and calculated ratios of vitamin D production per unit of sun burning radiation for different sources: $R = UV_{vitD}/UV_{Ery}$, and find that the measured responses are inconsistent with those calculated.

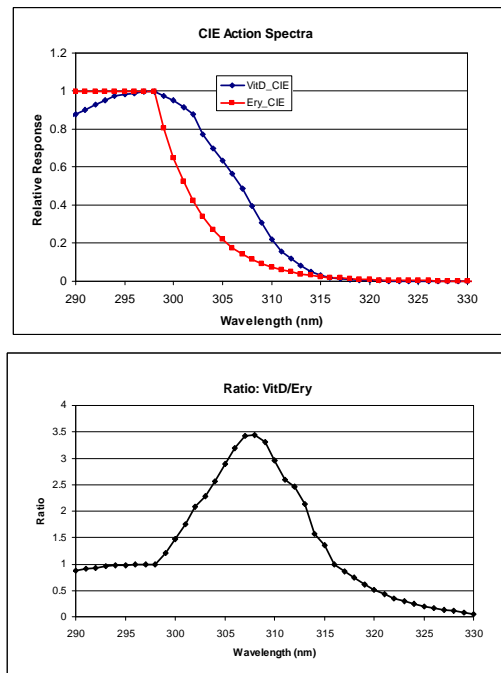


Figure 1. The upper panel shows the action spectra for erythema and vitamin D production as published by the CIE. The lower panel shows their ratio.

Parameter	Bed A	Bed B	Winter Sun	Summer Sun
UV_{Ery} (μWcm^{-2})	30.0	408.1	2.6	28.2
UV_{vitD} (μWcm^{-2})	34.8	997.4	3.2	56.7
$UVI = 0.4 \times UV_{Ery}$	12.0	163.3	1.0	11.3
Mins for 2SED	11.1	0.8	128.1	11.8
$R = UV_{vitD}/UV_{Ery}$	1.16	2.44	1.22	2.01

Table 1. Comparison of weighted irradiances and derived parameters from the two UV booths compared with sunlight at 45S in winter and summer.

Study Method

The study relates to UV exposures undertaken in phototherapy booths at a dermatological clinic in Auckland during the winters of 2006 and 2007 when ambient solar UV levels were low (McKenzie, et al. 2006). Participants were randomly assigned to receive regular twice-weekly sub-erythemal UV exposures from the phototherapy booths designated “Bed A” (62 participants), or “Bed B” (59 participants). The spectral distribution of Bed A is similar to that from a range of sun beds in common use in New Zealand. The spectrum of Bed B, which is especially designed for dermatological use, is dominated by a strong emission at 311 nm.

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Doses from Bed A were relatively low, as this was originally intended as a control. On the other hand, doses from bed B were as large as practicable without inducing erythema, and were generally increased from week to week (i.e., but always less than 1 MED³ per session). The cumulative dose over the 12-week period varied according to skin type, but was on average 11 SED from Bed A and 174 SED from Bed B.

Parameter	Period 1. Six Weeks		Period 2. 12 Weeks	
	Bed A	Bed B	Bed A	Bed B
R (Calculated, from UV spectra)	1.16	2.44	1.22	2.01
R (Measured, from ΔVitD)	4.5 ± 0.3	1.0 ± 0.1	3.1 ± 0.3	0.4 ± 0.1

Table 2. Calculated and measured ratios of vitamin D production per SED of UV exposure.

Blood serum vitamin D was measured at the start of the study, then at the end of a 6 week period in which 12 UV exposures were administered, and again at the end of a second 6-week period after a further 12 UV exposures.

Spectral irradiances from the beds are compared with those for noon sunlight in summer and winter in Figure 2, and the weighted irradiances are compared in Table 1. The calculated sensitivity of vitamin D production per SED for Bed B (R_B) is approximately twice R_A . Coincidentally, the sensitivity value for Bed B is similar to summer sunlight whereas the value for Bed A is similar to winter sunlight.

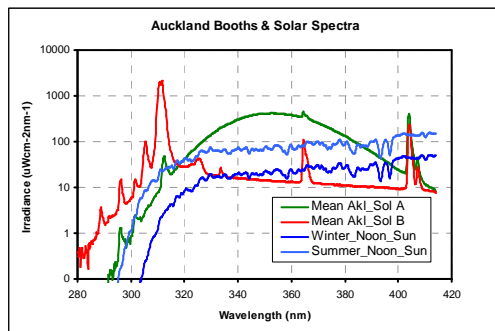


Figure 2. Spectral irradiances of two UV booths in Auckland, compared with mid-latitude irradiances measured under clear skies at noon in summer and winter.

Changes in vitamin D status due to the lamp exposures were calculated, including a subtraction of the population-average seasonal pattern in blood serum vitamin D. Inclusion of this term represents the assumption that apart from sunbed use, the study group was typical in its UV and dietary behaviour. These results were expressed in terms of the change in vitamin D per SED, as shown in Table 2. Note that whereas the calculated sensitivities are greater for Bed B, the measured sensitivities are greater for Bed A.

³ 1 SED = 1 Standard Erythral Dose = 100 Jm⁻² of erythemally-weighted irradiance. For sensitive skins, one Minimum Erythral Dose (MED) is approximately 2 SED. For less sensitive skins, 1 MED can exceed 10 SED.

Contrary to expectations based on the measured changes in vitamin D status, the efficiency of vitamin D production from Bed A appears to be much more than Bed B per SED. Some of the discrepancy is attributable to non-linearities in production, as evidenced by the fact that the sensitivity in the 2nd 6-week period, where UV intensities were increased, was smaller than in the first period, especially for Bed B where the erythral doses administered were much larger.

Parameter	6 Wk	12 wk
R_B/R_A (Calc from UV spectra)	2.10 ± 0.05	1.65 ± 0.03
R_B/R_A (Meas from ΔVitD)	0.22 ± 0.03	0.13 ± 0.02
Calc/Meas	9.5 ± 1.2	12.7 ± 1.6

Table 3. Comparing calculated and measured efficiencies of vitamin D production per SED for the two beds.

Table 3 shows there is more than a factor of 8 difference between observation and theory for period 1, and an even greater difference for period 2. Huge non-linearities in vitamin D production for dose ~1 SED, or as yet unresolved temperature effects would be required to resolve the discrepancy. Analysis of data from a follow-up study where similar UV doses were administered from each bed should help resolve these issues. These discrepancies would be reduced by a factor of two if the new action spectrum for vitamin D production (Olds et al. 2010) were used instead of the CIE action spectrum.

Addendum. After presentation of this material at the UV Workshop, subsequent analysis of data from the sun beds discussed here (which were also used in our HRC UV-Vitamin D study) showed that the production of vitamin D from these sun beds, was much less than as stated during the presentation. The error was due to an incorrect calibration factor which has now been corrected in this version. With these corrected values, the discrepancy between theory and measurement is reduced slightly.

References

- Bouillon, R., Eisman, J., Garabedian, M., Holick, M., Kleinschmidt, J., Suda, T., Terenetskaya, I., Webb, A. 2006. Action spectrum for the production of previtamin D3 in human skin. No. 174:2006. 12 p.
- McKenzie, R.L., Bodeker, G.E., Scott, G., Slusser, J. 2006. Geographical differences in erythemally-weighted UV measured at mid-latitude USDA sites. *Photochemical & Photobiological Sciences* 5(3): 343-352.
- McKenzie, R.L., Liley, J.B., Björn, L.O. 2009. UV Radiation: Balancing Risks and Benefits. *Photochemistry and Photobiology* 85: 88-98;doi:10.1111/j.1751-1097.2008.00400.x.
- McKinlay, A.F., Diffey, B.L. 1987. A reference action spectrum for ultra-violet induced erythema in human skin. In: Passchier, W.F., Bosnjakovic, B.F.M. (eds). *Human Exposure to Ultraviolet Radiation: Risks and Regulations*, pp. 83-87. Elsevier, Amsterdam.
- Olds, W.J., Lucas, R.M., Kimlin, M.G. 2010. Action spectrum for vitamin D synthesis. Poster 53 at NIWA UV Workshop, Queenstown, 7-9 April.
- Webb, A.R., Kline, L., Holick, M.F. 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. *Journal of Clinical Endocrinology and Metabolism* 67(2): 373-378.