Comparison of human facial UV exposure at high and low latitudes and the potential impact on dermal vitamin D production

Kimlin, M.G.,*[+] Downs, N.J.[+] and Parisi, A.V.[+]

*National Ultraviolet Monitoring Center
Department of Physics and Astronomy
University of Georgia
Athens, Ga, USA. 30606
mkimlin@physast.uga.edu

[+]Centre for Astronomy, Solar Radiation and Climate
Faculty of Sciences
University of Southern Queensland
Toowoomba, Queensland, AUSTRALIA. 4350
parisi@usq.edu.au

*To whom correspondence should be sent

Abstract

The results presented in this paper allow for the estimation of the monthly UV exposure of the human facial region at various locations across the earth. The technique allows the graphical representation of the UV exposures over the face. The erythemal UV exposures as well as the vitamin D exposures to the human facial region have been investigated. The results gained in this paper, for clear sky and constant ozone indicate that the sun's capability to promote the development of vitamin D in the human body does not follow the erythemal UV irradiances, in particular at high latitudes. For Amsterdam (52^{o}N) in late winter, approximately 20% more UV is required to produce 215 $J.m^{-2}$ of vitamin D weighted UV than erythemal UV.

Introduction

Ultraviolet (UV) radiation's interaction on human skin has both positive and negative effects. The positive effect includes an important component for the formation of vitamin D in the skin, whilst the negative includes the formation of skin cancers and premature skin photo-aging. The active form of vitamin D is technically referred to as 1,25 hydroxyvitamin D (sometimes as calcitriol, or cholecalciferol). Sunlight interacts with the vitamin's pre-cursor, 7-dehydrocholesterol to produce pre-vitamin D_3[1]. (Vitamin D will be used in this paper to denote this compound.)

Vitamin D is also obtained from some foods, typically fortified milk. However, exposure to sunlight is considered the primary source of vitamin D, since the quantities of milk (the primary source other than sunlight) consumed in the U.S. is quite small[2]. In addition to the bone disease rickets, vitamin D has a suggested association with the development of various diseases, such as colon-rectal cancer, breast cancer, and type I diabetes. Recent research suggests that underexposure to UV radiation (hence Vitamin D production) is associated with premature cancer mortality in the American population[3].

The formation of non-melanoma skin cancers has been linked to exposure to solar UV with a higher incidence for people living in tropical and semi-tropical areas compared to those living further from the equator[4]. Research suggests that the sites of the lesions are coherent with the sites that receive high exposures to sunlight, for example, the head and neck in particular are likely to develop such lesions[5]. Previous research has calculated the ambient solar erythemal UV and UVA radiation on a horizontal plane at high and low latitudes in both the southern and northern hemispheres[6]. The research presented in this paper extends the previous research, as it allows the determination of erythemal UV exposures for the different latitudes to one of the highest UV exposure sites of the human body, namely, the face, for fairskinned populations. Additionally, this research compares the amount of UV radiation that is effective for the production of vitamin D at each of the low and high latitudes.

Materials & Methods

Locations

This study utilizes a previously developed UV radiation model to predict the biologically effective exposure on a horizontal plane[7] and a technique to calculate the subsequent exposure received by the human face[8]. The relevant UV exposures to the facial region at four separate geographic sites were selected for comparison. The sites selected were Brisbane (27^o S 153^o E), Cairo (30^o N 31.3^o E), Amsterdam (52^o N 5^o E) and Ushuaia (54^o S 68^o W). These sites were selected as they represent urban regions and therefore have a high population density, are located at or close to sea level, and are approximately evenly distributed on either side of the equator.

This study neglects the possible presence of cloud cover, altitude, and albedo contribution at these sites and the influence that these factors may have on the biologically effective horizontal plane UV and respective biologically effective UV exposure to the human face. Exposure levels calculated at each of these sites uses a nominal level of air, aerosol optical depth (AOD) and column ozone (320 DU) thickness, with the focus of this study being placed on geographical location, demonstrating the potential of the technique for assessing global scenarios. The standard ozone level of 320 DU was chosen as a first order approximation of the average ozone over these locations. Aerosol and air concentrations are based on the extinction coefficients and extinction amounts formulated primarily from the studies of Green et al[9]. Details of the model can be found in Appendix 1.

At each of the geographical sites selected, both the horizontal plane erythemal and vitamin D exposure is estimated for a single day in each month. Exposures are determined at sea level, which is a reasonable assumption given the locations of the selected sites. This analysis is performed so as to investigate the health outcomes associated with both excessive exposure to erythemally effective UV, and the possible insufficient production of vitamin D following under exposure to terrestrial levels of UV.

Furthermore, this study makes comparisons of the erythemally effective facial exposure at each of the geographical sites for a selected day in each season. In this way a "snapshot" of the likely facial exposures received in each of these regions will be provided for comparison.

UV model

The UV model employed to investigate the horizontal plane exposure is a developed hybrid of a number of semi-empirical UV transmittance equations and includes the variations in the earth-Sun distance. The model has been described previously[7], although a number of new modifications have been made to account for variation in albedo and altitude above sea level (Appendix 1). For this research, altitude is normalised to sea level and albedo contribution is neglected.

The UV model developed specifically for this research provides exposures that are on average within 10% of the horizontal plane daily erythemally effective exposures measured by the UV-Biometer (Solar Light Co. model 501) located at the University of Southern Queensland. A comparison of the modelled to measured daily erythemal UV irradiances, expressed in units of MED (minimum erythemal dose) for cloud free days, is shown in Figure 1. This accuracy may change depending on location and local atmospheric conditions.

The model calculates the spectral UV irradiance (units of $W/m^2/nm$) at 15 minute intervals across the entire biologically significant UV region, namely the 280 nm to 400 nm region. The modelled output irradiance is weighted according to the erythema[10] and vitamin D[11] biological action spectra (shown in Figure 2). The vitamin D spectra is based on the conversion of 7-dehydrocholesterol to pre-vitamin D_3 as measured at the four wavelengths of 296, 300, 306 and 316 nm. The biologically effective irradiance is then integrated with respect to time and the position of the sun (15 minute intervals) to determine the effective daily erythemal and vitamin D exposure.

Facial Exposure Contouring

This study includes a number of visual plots of the human facial region to highlight the potential of the developed model and how that model's output can be used to plot exposure estimates over the complex facial topography. The technique used to determine the biologically effective facial exposure has been discussed previously[7,8]. Polysulphone dosimeters were utilized to determine the exposure to various facial locations. The polysulphone film used was constructed by the authors at the University of Southern Queensland, and has a response approximating that of the erythemal action spectrum. The thickness of the produced film is monitored, as increases in thickness of the film causes a change in spectral response which deviates from the erythemal action spectrum and can cause errors of up to 35% in the measured exposures[12].

To summarize, polysulphone dosimeters were attached to a rotating human head manikin model and placed outdoors in an open, unshaded sports field in clear sky conditions. The radiometric uncertainty in the polysulphone measurements is of the order of ±10%. The nearest building was over 60 m away whilst the ground reflectivity of the grassy surface was estimated to be 5%. The method is detailed in Downs et al[7]. Briefly, the dosimeters were attached to the forehead, nose, left and right cheek, left and right ear, chin, front of the neck, left and right shoulders, and upper chest and measurements with these dosimeters were taken in solar zenith angle (SZA) ranges from 0° to 80° in 10° steps (no change in optical absorbency was recorded in the range 80^o to 90^o). The ratio of the UV exposure to a particular facial anatomical site compared to the exposure to a horizontal plane (known as the exposure ratio) was determined at each 10° interval. The modelled biologically effective horizontal plane UV (in 10^o intervals) for each location was weighted with the appropriate SZA facial exposure ratio (for SZA's above 80^o the 70^o to 80^o exposure ratio was used) for the appropriate anatomical site. These values were summed from 6:00 am to 6:00 pm to produce daily facial exposure estimates for the different facial sites. A series of contour plots over the facial region were drawn by bilinearly interpolating the exposures between each of the dosimeter sites over the entire facial region. The analysis of the exposure ratios and production of the contour plots was

handled using IDL: Interactive Data language *(Research Systems, Inc., IDL version 5.3.1).*

The model technique was used to determine the daily facial erythemal exposure for the first day of each season for Brisbane and Amsterdam using the assumption that no hat or sunscreen was employed and that the head was in an upright position. These two sites were selected for the comparison of facial exposure distributions as they exhibit the greatest seasonal contrast observable between the mid and high latitudes. Estimates of the erythemal facial exposure are made under clear sky conditions.

Results and Discussion

The estimated horizontal plane erythema and vitamin D response, for fair skinned people, at each of the four selected sites is shown in Table 1. Early represents the first day of each season, followed by the mid seasonal daily exposure of the season, and late refers to the last day of each season. Exposures listed are expressed relative to 215 J/m^2 of the effective biological exposure, which for erythema, approximates 1 MED. The table lists the effective daily exposures for the first day of each month within each respective season (both hemispheres). Exposure of an individual's whole body to one MED of sunlight is equivalent to ingesting about 250 µg (10,000 IU) of vitamin D[13]. Exposure to 1 MED of sunlight is 17 to 50 times the recommended adequate intake (AI) for vitamin D from dietary sources[14]. Therefore, for an older woman to obtain the equivalent of 15 µg (600 IU) of vitamin D per day (AI for women > 70 years of age), she would need to expose 6% of her body surface (hands, face, forearms) to sunlight for 15 to 30 minutes two or three times a week[13].

Figure 3 provides a comparison between the daily erythema and vitamin D response at each of the four sites for the first day of each month in a given year for a horizontal plane. This figure illustrates the difference between sites located at low and high latitudes, and how this affects the relative production of vitamin D. As expected there is little difference in the effective exposure between sites of approximately the same latitude and considerable contrast between sites of different latitudes. Of significant interest is the

relative production of vitamin D. Sites at low latitudes always receive sufficient sunlight to sustain consistently higher levels of vitamin D compared to effective erythemal exposures, while sites at high latitudes experience a "cross-over" in winter whereby the effective erythemal exposure exceeds the biologically effective vitamin D. The reduced effectiveness of the skin to produce vitamin D when the sun is lower towards the horizon has been noted before[11]. In the winter months at Amsterdam and Ushuaia, the amount of biologically effective UV for vitamin D production is less than that for erythema. This data shows that the health concerns associated with the insufficient production of vitamin D, such as the incidence of diseases such as rickets for example, are more likely to develop in locations where solar UV levels are low for much of the year and particularly in locations where populations tend to spend a lot of time indoors and out of direct sunlight. However, as is well known, there are also negatives associated with spending too much time in the sun, and Table 1 shows the possibility of the risk of over exposure to erythemal UV, particularly at low latitudes.

The lower relative production of vitamin D for the winter months at high latitudes is due to the weighting of the vitamin D action spectrum where the effective production of vitamin D is more dependent on the shorter wavelengths than the effective erythemal response. The low solar elevations and subsequently increased path through the atmosphere result in increased absorption and scattering of the shorter wavelengths. The relative proportions of absorption and scattering are both more pronounced at the shorter wavelengths due to increased absorption by ozone and increased Rayleigh scattering at the shorter wavelengths. Consequently, at these higher latitudes, the production of vitamin D due to UV falling on the skin tends to be lower than the effective erythemal response due to the reduced relative irradiances of the shorter wavelengths. In comparison, at lower latitudes, where the sun is able to reach high solar elevations, the relative production of vitamin D tends to be higher, due to reduced scattering and absorption of the more effective shorter wavelengths.

When dealing with health concerns due to over exposure caused by terrestrial UV, it is convenient to examine the anatomical distribution of the UV exposure. Figure 4 makes a

comparison between the erythemal facial exposure of a subject exposed for an entire day to the levels listed in Table 1 for the mid months of each season.

While the developed model can be used as an effective tool to examine the UV variation in the horizontal plane, it is useful for examining the facial distribution of exposure with respect to the epidemiology of cancerous lesions and familiar erythemal reactions. Figure 4 shows a marked contrast between a subject located in Amsterdam and one located in Brisbane. A mid summer exposure in Amsterdam, looks similar to that of a Brisbane spring time exposure, while the mid summer exposure in Brisbane indicates clearly the heightened risk of a much more severe sunburn over most of the face. It is interesting to note in all eight plots of Figure 4, that for an unprotected facial region, after the vertex of the head, the nose is the most likely location of the face to receive the highest facial exposure. In a similar fashion, the biologically effective exposures for vitamin D production may also be shown in the same graphical manner.

These predicted results are consistent with measured facial exposure data of other researchers[15,16]. Included with each plot of the figure is the horizontal plane exposure received in each 10^{o} range recorded over the course of a day. Facial exposures are influenced the most by exposures received during the middle of the day, with high solar elevations having the greatest effect on horizontal facial topography such as the vertex of the head and the nose. The effective distribution of exposure is thus more likely to affect these areas of the face when the sun reaches high solar elevations at low latitudes, however at high latitudes, low solar elevations reduce the vertical component of the direct UV that reaches the face and so broader facial exposure distributions are received. Such results may indicate an increased incidence of cancerous lesions across a broader facial range at progressively high latitudes, although at a much lower incidence than at low latitudes.

Conclusions

The results presented in this paper allow for the estimation of the monthly UV exposure of the human facial region at various locations across the earth. The authors investigated

the erythemal UV exposures as well as the vitamin D exposures. The vitamin D exposures are an important tool for the estimation of the sun's ability to produce vitamin D in the human body. For some of the cold, northern locations, the facial region may be the only area of the body with skin exposed to sunlight, and hence vitamin D production capability. The results presented in Figure 3 highlight the need for understanding of the physical environment at high latitudes and its effect on vitamin D formation.

For both the Ushuaia and Amsterdam locations, the capability of the sun to produce vitamin D is diminished in the winter months. For Amsterdam in late winter, approximately 20% more UV is required to produce 215 $J.m^{-2}$ of vitamin D weighted UV than erythemal UV. The higher relative proportion of absorption and scattering at the shorter UV wavelengths at these high latitudes alters the amount of UV required to produce vitamin D. These results neglect the influence of facial skin type and individual variations in the skin. The model also neglects to consider the position of the subject with respect to the environment, variation in the atmospheric conditions, and variations in the physical environment that inevitably change throughout the year.

This paper has extended the previous research to allow the determination of the UV exposures to the human facial region for the different latitudes. In this case, the results were shown graphically for the erythemal UV exposures, however the technique allows the graphical representation of the UV exposures over the face for the vitamin D production or for any other biological process and highlights the need for further research in this area.

REFERENCES

1. J. J. McGrath, Does ‘imprinting’ with low prenatal vitamin D contribute to the risk of various adult disorders?, *Med. Hyp.*, 2001, **56**, 367-371.

2. M. Holick, 1988; reported in J. J. McGrath, Does ‘imprinting’ with low prenatal vitamin D contribute to the risk of various adult disorders? *Med. Hyp.*, 2001, **56**, 367-371.

3. W.B. Grant, An estimate of premature cancer mortality in the U.S. due to inadequate doses of solar UV-B radiation, *Canc.*, 2002, **94**, 1867-1875.

4. F. Urbach, Ultraviolet radiation and skin cancer of humans, *J. Photochem. Photobiol. B: Biol.*, 1997, **40**, 3-7.

5. B.L. Diffey, Solar ultraviolet effects on biological systems, *Phys. Med. Biol.*, 1991, **36,** 299-328.

6. J.M. Elwood and B.L. Diffey, A consideration of ambient solar ultraviolet radiation in the interpretation of studies of the aetiology of melanoma, *Mel. Res.*, 1993, **3**, 113-122.

7. N.J. Downs, M.G. Kimlin, A.V. Parisi and J.J. McGrath, Modelling human facial UV exposure, *Rad. Prot. Australas.*, 2001, 17, 103-109.

8. M.G. Kimlin, A.V. Parisi and J.C.F. Wong, The facial distribution of erythemal ultraviolet exposure in south east Queensland, *Phys. Med. Biol.*, 1998, 43, 231-240.

9. A.E.S. Green, T. Sawada and E.P. Shettle, The middle ultraviolet reaching the ground, *Photochem. Photobiol.*, 1974, **19**, 251-259.

10. CIE (International Commission on Illumination), A reference action spectrum for ultraviolet induced erythema in human skin, *CIE J.* 1987, **6**, 17-22.

11. A.R. Webb, Vitamin D synthesis under changing UV spectra, in *Environmental UV Photobiology*, ed. A.R. Young, L.O. Bjorn, J. Moan and W. Nultsch, Plenum Press, New York, 1993, pp.185-202.

12. A.V. Parisi, L.R. Meldrum and M.G. Kimlin, Polysulphone film thickness and its effects in ultraviolet radiation dosimetry, *Protection Against the Hazards of UVR, Internet Conference*, 18 Jan – 5 Feb 1999.

13. M.F. Holick, *Vitamin D*, in Modern Nutrition in Health and Disease, ed. M.E. Shills, J.A. Olson, M. Shike and C.A. Ross, . 9th edition, Williams & Williams, Baltimore 1999, pp.329-345.

14. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine (IOM). *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride*. Washington, DC: National Academy Press, 1997.

15. D.K. Airey, J.C.F. Wong, R.A. Fleming and L.R. Meldrum, An estimate of the total UV-B exposure for outdoor workers during a south-east Queensland summer, *Health Phys.*, 1997, **72**, 544-549.

16. M.G. Kimlin and A.V. Parisi, Usage of real-time ultraviolet radiation data to modify the daily erythemal exposure of primary schoolchildren, *Photodermatol. Photoimmunol. Photomed.*, 2001, **17**, 130-135.

17. R.D. Rundel, Computation of spectral distribution and intensity of solar UVB radiation, in *Stratospheric Ozone Reduction, Solar Ultraviolet Radiation and Plant Life*, ed. R.C. Worrest and M.M. Caldwell, Springer-Verlag, Berlin, 1986.

18. P.F. Schippnick and A.E.S. Green, Analytical Characterization of Spectral Actinic Flux and Spectral Irradiance in the Middle Ultraviolet, *Photochem. Photobiol*., 1982, **35** 89-101.

Appendix 1

Specifically the model determines both the direct and diffuse components of the UV irradiance. In this latest version of the developed hybrid, the equations are arranged as follows whereby (1) represents the direct irradiance[9] and (2) the diffuse[17]

$$B_{dir}(\lambda,\theta,y) = \mu H(\lambda) e^{-A_t} \quad \ldots(1)$$

In equation (1), $B_{dir}(\lambda,\theta,y)$ is the local ground level direct spectral irradiance at a given wavelength, solar zenith angle, and altitude, A_t is the attenuating thickness of the atmosphere along the path of the direct solar beam, $H(\lambda)$ is the extra-terrestrial spectral irradiance, and μ is the cosine response, $\cos(\theta)$ that describes the direct solar irradiance falling vertically on a horizontal plane.

$$\text{And,} \quad A_t = \sum_j \frac{\tau_j}{\mu_j}$$

Where τ_1, τ_2, τ_3 are the resultant product of the wavelength dependent species optical depth and altitude dependent concentrations of air, ozone and aerosols respectively, and μ_1, μ_2, μ_3 are geometric cosine functions that describe each of the aforementioned species relative to a spherical earth.

$$\text{And, } \mu_j = \left(\frac{\mu^2 + t_j}{1 + t_j} \right)^{1/2}$$

t_1, t_2, t_3 are constants that depend on the altitude distributions of air, ozone, and aerosol species.

$$B_{diff} = H(\lambda)e^{-D_t(\theta,\lambda)} + A_d \quad \ldots(2)$$

In equation (2) $D_t(\theta,\lambda) = K_{oz}(e^{\kappa k_0 w_{oz} - (\lambda-\lambda_0)/(\delta speq\theta)})seq(\theta, q_1) + K_{ap} seq(\theta, q_2)$

$$speq\theta = \frac{1}{\left(1 - \frac{\sin p_\theta}{q}\right)^{1/p}}$$

$$seq_i(\theta, q_i) = \left(1 - \left(\frac{\sin^2 \theta}{q_i}\right)\right)^{-1/2}$$

$K_{ap} = 1.255$ at sea level, $q_2 = 1.32$ at sea level, $K_{oz} = 1.62$, $\kappa = 2.40$, $\delta = 7.48$, $q_1 = 1.10$, $q = 1.148$, and $p = 4$

and at altitude, $K_{ap} = 0.872(1 + 0.179y + 0.0487(\tau_p - 0.538)^2)$

$$q_2 = 1.06(1 + 0.106y)$$

Here *y* is the altitude above sea level expressed in kilometres, and τ_p is a wavelength and altitude dependent atmospheric parameter (product of the aerosol optical depth and aerosol species concentration).

$$\text{And, } A_d = r(\lambda)S_u$$

S_u is the upward irradiance resulting from the local surface albedo, and $r(\lambda)$ is the air reflectivity function describing the spectral reflectivity of the above atmosphere such that A_d is the total downward albedo contribution of the diffuse irradiance resulting from downward atmospheric backscatter. The formulation of $r(\lambda)$ is that used by Ruundel[17] and given by Schippnick and Green[18] p. 96 equations (28) and (29).

$$\text{Furthermore, } S_u = \frac{A_g G_s E_d}{1 - r(\lambda)A_g}$$

Where A_g is the surface albedo measured as a percentage, G_s is the global ultraviolet irradiance measured at sea level without an albedo contribution and, E_d is the normalised altitude dependence defined as the ratio of the albedo contribution at altitude to the albedo contribution at sea level.

Table 1 – Daily erythemal and vitamin D effective exposures on a horizontal plane expressed relative to 215 J m^{-2}.

	Brisbane		Cairo		Ushuaia		Amsterdam	
	Erythema	Vitamin D	Erythema	Vitamin D	Erythema	Vitamin D	Erythema	Vitamin D
Season	/ 215Jm^{-2}	/ 215Jm^{-2}	/ 215Jm^{-2}	/ 215Jm^{-2}	/ 215Jm^{-2}	/ 215Jm^{-2}	/ 215Jm^{-2}	/ 215Jm^{-2}
Early	30.7	58.0	29.0	54.6	21.3	38.4	22.0	39.8
Summer	31.2	59.0	29.2	55.2	22.4	40.6	22.8	41.6
Late	29.3	55.1	27.5	51.8	17.2	30.3	18.8	33.6
Early	24.6	45.8	23.1	42.8	10.0	16.2	11.7	19.4
Autumn	17.2	30.9	16.5	29.6	3.6	4.6	5.2	7.2
Late	10.7	18.0	9.9	16.4	1.0	0.8	1.5	1.5
Early	6.9	10.7	6.2	9.3	0.3	0.2	0.5	0.3
Winter	6.3	9.6	5.6	8.2	0.3	0.1	0.4	0.2
Late	8.7	14.1	8.3	13.3	0.6	0.4	1.0	0.8
Early	14.2	25.0	13.7	23.8	2.3	2.5	3.3	4.0
Spring	21.3	39.2	21.0	38.5	7.0	10.6	9.1	14.4
Late	27.6	51.8	26.4	49.4	14.9	25.6	16.4	28.7

FIGURE CAPTIONS

Figure 1: Comparison of the modelled to measured erythemal exposures.

Figure 2: Erythemal Action Spectrum and Vitamin D spectrum

Figure 3: Comparison of the daily erythemal and vitamin D exposure plotted for the first day of each month.

Figure 4a: Seasonal erythemal exposure in Brisbane.

Figure 4b: Seasonal erythemal exposure in Amsterdam.

Figure 1: Comparison of the modelled to measured erythemal exposures.

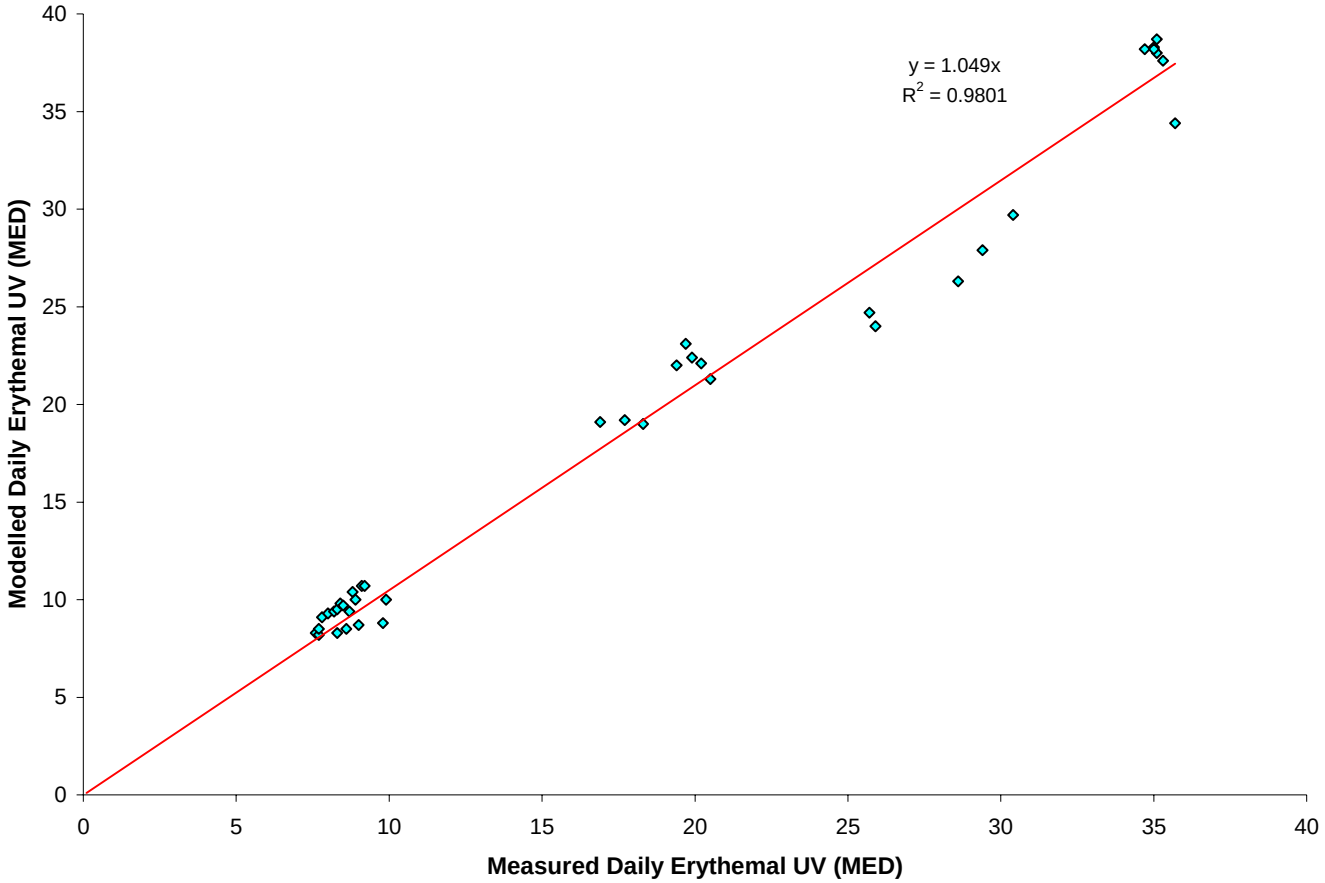


Figure 2 – Erythemal Action Spectrum and Vitamin D Spectrum

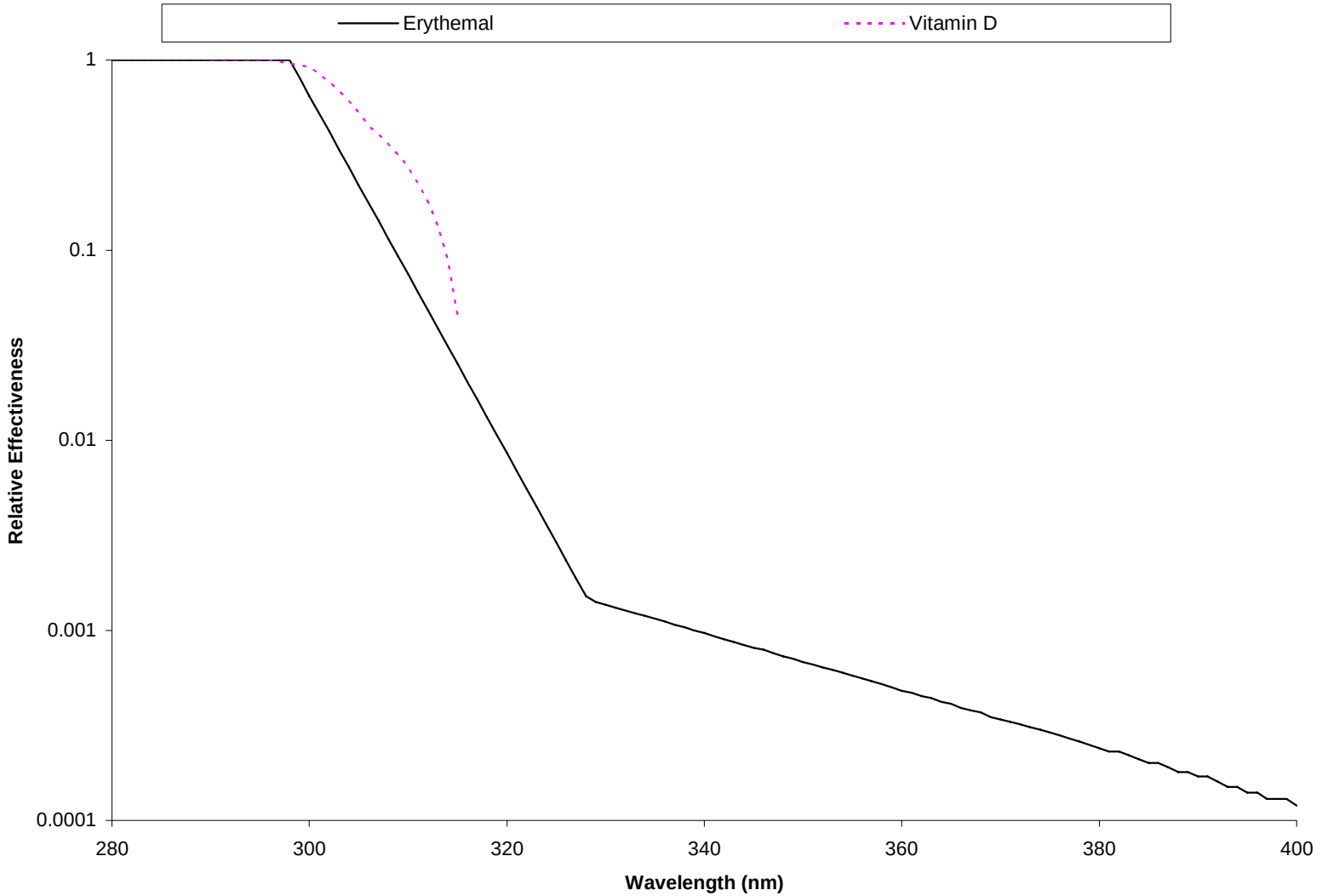
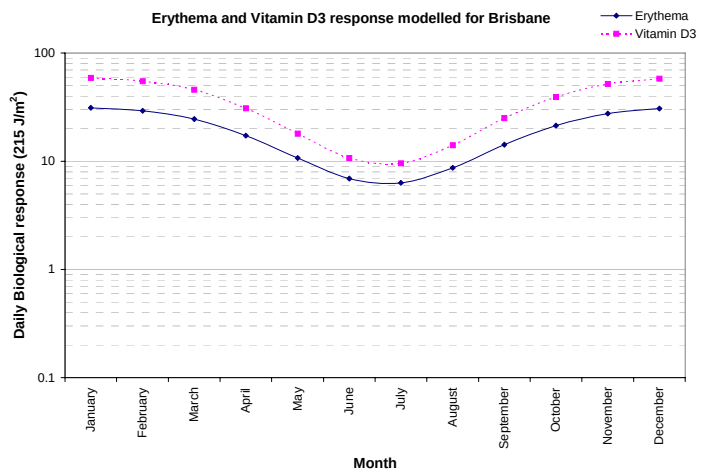
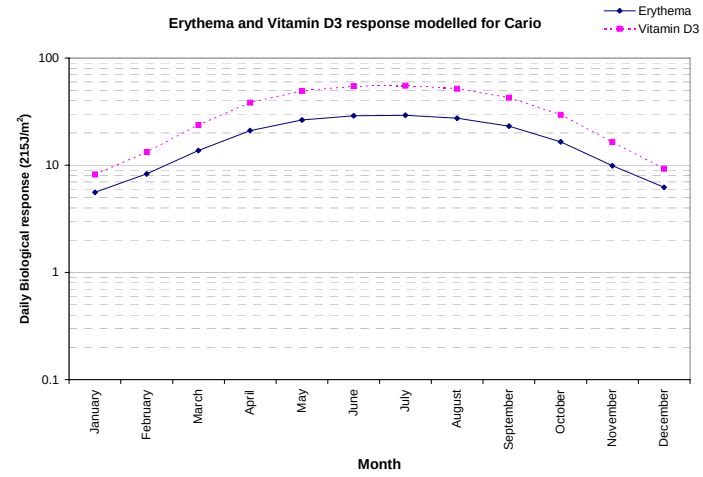


Figure 3: Comparison of the daily erythemal and vitamin D exposure plotted for the first day of each month.

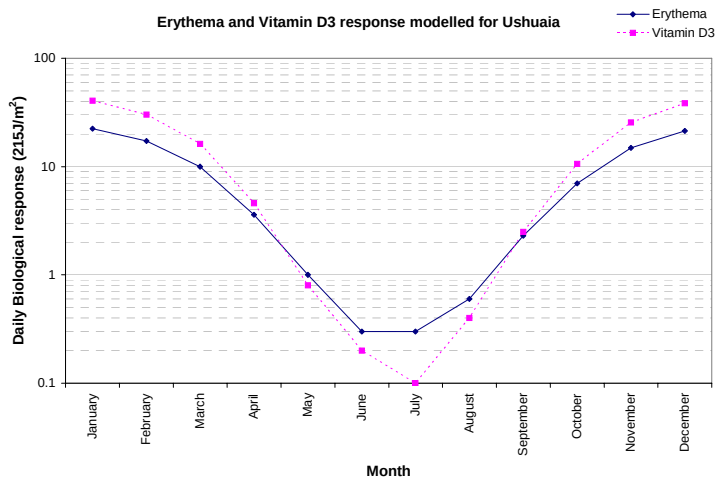
Brisbane



Cairo



Ushuaia



Amsterdam

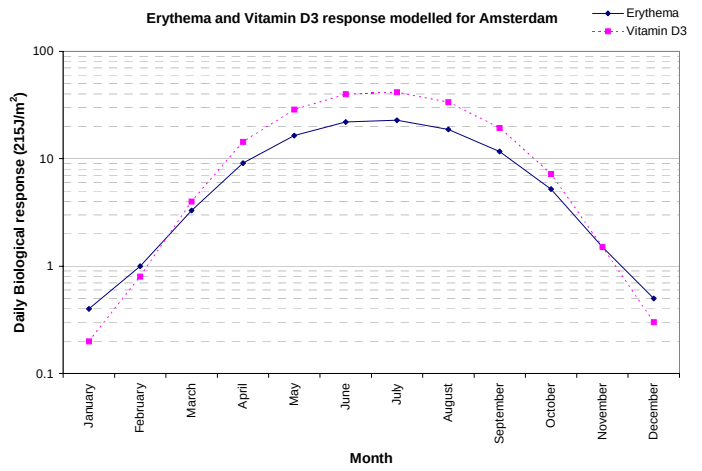
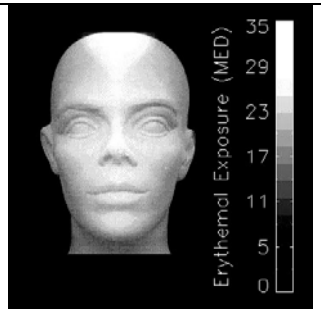
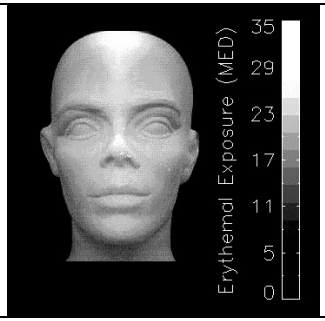
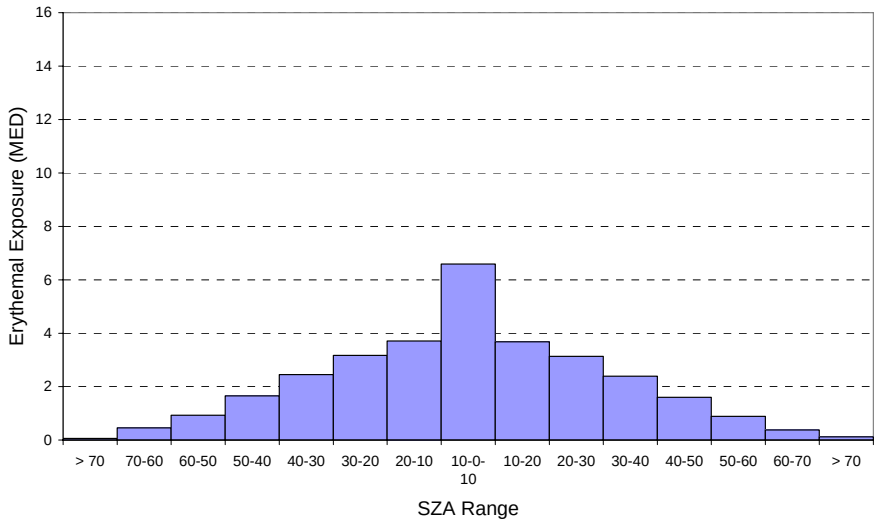
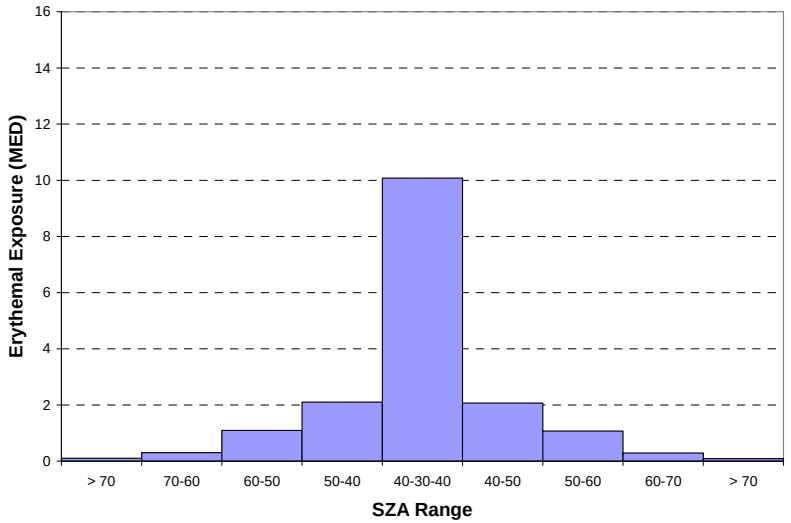


Figure 4a: Seasonal erythemal exposure in Brisbane.

Summer (1 January)	Autumn (1 April)
	
	

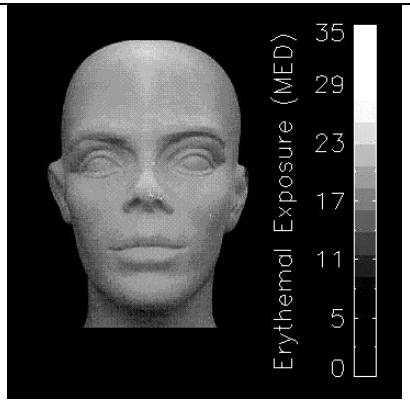
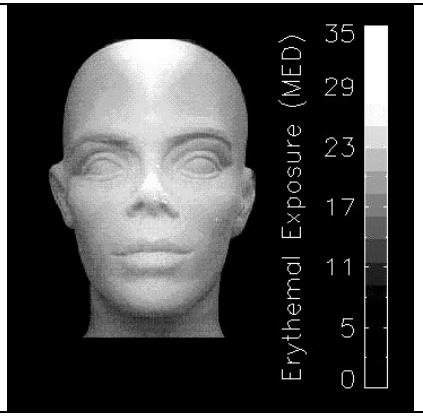
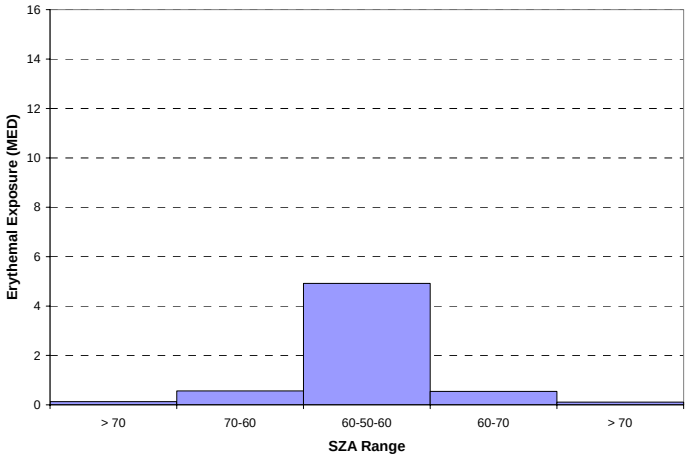
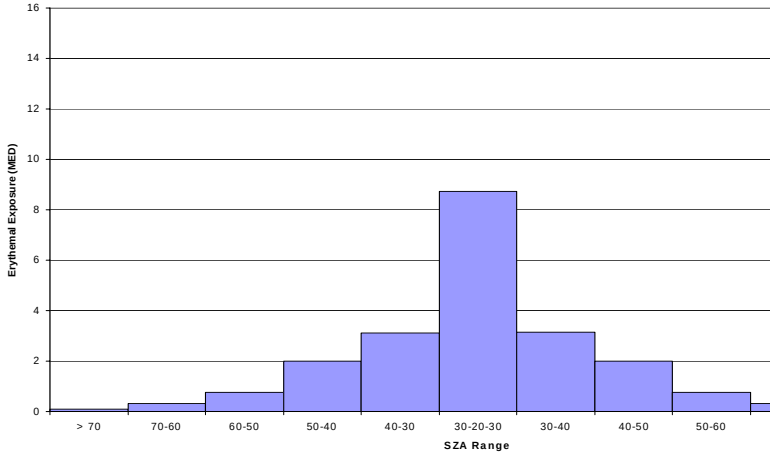
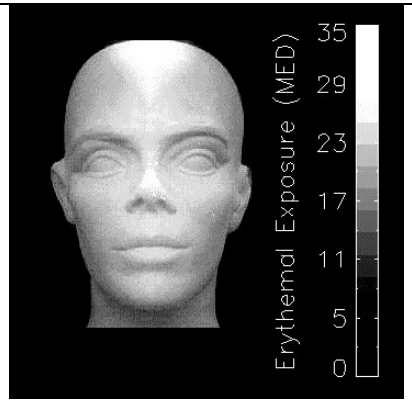
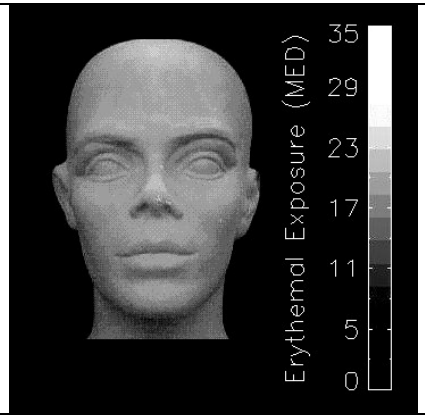
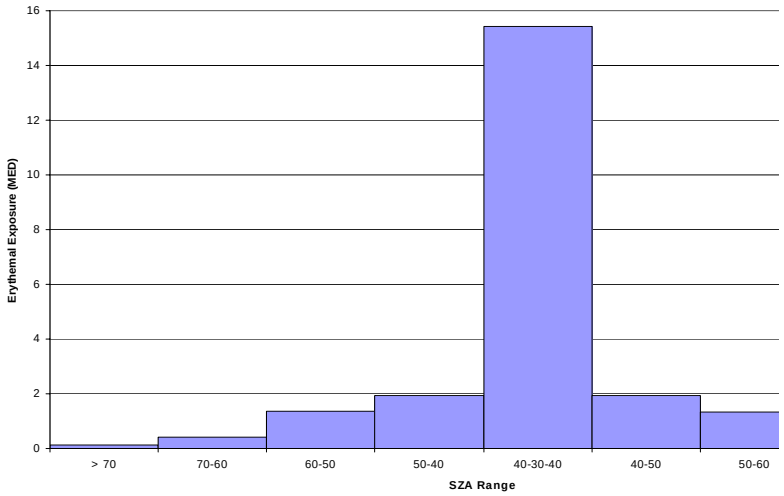
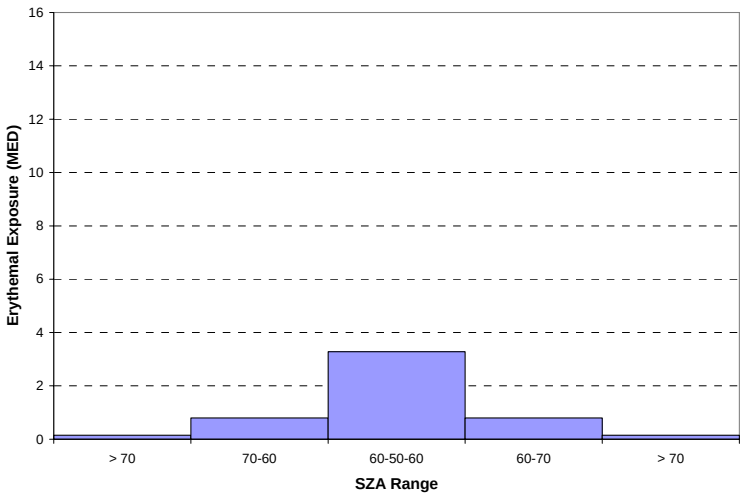
Winter (1 July)	Spring (1 October)
	
	

Figure 4b: Seasonal erythemal exposure in Amsterdam.

Summer (1 July)	Autumn (1 October)
	
	

Winter (1 January)	Spring (1 April)