AN ESTIMATION OF BIOLOGICAL HAZARDS

DUE TO SOLAR RADIATION

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ABSTRACT

A spectrum evaluator based on four different dosimeter materials was employed to estimate the spectral irradiances of exposure to solar radiation. The result was used to calculate the biologically effective irradiance using the erythemal action spectrum and a fish melanoma action spectrum. Measurements were made in winter at a sub-tropical site on the chest and shoulder of subjects during normal daily activities.

Up to 95% of the total UV exposure received was in the UVA waveband (320-400 nm). The UVA waveband was found to contribute approximately 14% of the erythemal UV and 93% of the biologically effective UV for fish melanoma. Extrapolation to humans suggests that the exposure to the UVA band will contribute to photodamage in human skin during an exposure to solar radiation.

1. Introduction

The assessment of the hazard of solar ultraviolet (UV) radiation on humans has been made with the erythemal action spectrum for human skin [1]. This action spectrum is normalised to unity at 297 nm with the value of the sensitivity dropping by about 3 decades in the UVB waveband (280-320 nm). In the UVA (320-400 nm) waveband, the value of the action spectrum varies from 10^{-3} to 10^{-4} . The incidence of melanoma may be linked to the exposure of solar UV [2]. There have been studies [3,4] on the relation of UV exposure and the induction of melanoma and the results suggest that the melanin in the melanocytes absorbs UV at all wavelengths. No action spectrum for induction of melanoma using a hybrid fish. The fish melanoma action spectrum has a relative effectiveness up to 800 times higher than that for erythema in the UVA waveband.

For sunlight, the spectral irradiance in the UVA is approximately one hundred times higher compared to the UVB in locations such as Australia. Furthermore, UVA wavelengths are employed in sunlamps for tanning sunbeds. Diffey [5] has estimated the population exposure to UVA in England to be 1,500 J cm⁻² based on the measurement of ambient radiation. No measurements on the contribution of the UVA waveband to personal biologically effective exposures have been previously undertaken. In addition, there have been very little studies on the estimation of the biological hazard of solar radiation using action spectra other than the erythemal action spectra. The result would yield information

on the contribution of the two waveband components (UVA and UVB) to the biological effects under selected conditions during the exposure.

To apply an action spectrum for estimating the biological hazard of personal exposure to solar radiation, a measurement of the solar spectrum is necessary. Measurement surveys of the personal exposures have been performed with polysulphone dosimeters [6-9] and CR-39 dosimeters [10]. These dosimeters were calibrated to measure personal erythemal exposures resulting predominantly from the UVB (280-320 nm) waveband. They are predominantly sensitive to the UVB wavelengths [11,12] and are impractical, if not impossible for the assessment of personal exposures to the UVA wavelengths. Furthermore, when applying the dosimeter to assess the effect of solar radiation using action spectra other than the erythemal action spectrum, the dosimeters must be recalibrated for that purpose. A detector [13] based on four different dosimeter materials was previously developed for estimating the solar UV spectrum and will be employed in this paper for evaluation of the hazard of the UVA waveband.

2. Materials and Methods

2.1 Dosimetric Method

The damaging effect of UV radiation on human skin can be expressed employing the concept of the biologically effective UV irradiance (UVBE) and the action spectrum, $A(\lambda)$, for the process under consideration as follows:

$$UVBE = \int_{280}^{400} S(\lambda) A(\lambda) d\lambda$$
 (1)

where $S(\lambda)$ is the source spectrum. A recently developed UV spectrum evaluator, [14] with four types of UV sensitive dosimeter films has been employed for evaluation of the filtered solar ultraviolet spectrum [15]. Its sensitivity at 340 nm is about 1000 times that of polysulphone [14]. The development and testing of the method has been previously described [16,13,14]. The dosimeter materials employed in the spectrum evaluator are polysulphone, nalidixic acid (NDA), 8-methoxypsoralen (8MOP) and phenothiazine [13]. The spectrum evaluator has an overall size of 3 cm x 3 cm in the form of a film badge with a piece of the material of approximately 1 cm² over a 0.6 cm diameter hole in a holder. Previous research has measured a difference of approximately up to 20% between spectra evaluated with the system and a calibrated spectroradiometer [13].

Briefly, for each type of dosimeter film exposed to a source spectrum $S(\lambda)$ over a time interval, T, the change in optical absorbance, ΔA_i at a set wavelength is given by [13]:

$$\Delta A_i = T \int_{uv} S(\lambda) R_i(\lambda) d\lambda \qquad i = 1,...4$$
⁽²⁾

where $R_i(\lambda)$ is the spectral response of each material. The subscript denotes one of the four different materials used.

Each type of film is responsive to different UV wavebands [14]. The spectral responses give the effectiveness of each wavelength for the production of a change in the optical absorbance of each film. For each material, it is defined as the change in optical absorbance at a set wavelength for each film due to unit irradiance in the wavelength interval λ to $\lambda + d\lambda$ [13]. Polysulphone exhibits a high response at UVB wavelengths with a rapid drop for wavelengths longer than 320 nm. NDA has a peak in response at

approximately 325 nm and is sensitive up to 370 nm. 8-MOP responds to wavelengths up to 360 nm with a peak at 300 nm. Phenothiazine responds to all UV wavelengths with a peak at 330 nm. The composite of all four materials covers the entire UV waveband.

The materials change their optical absorbance after exposure to UV radiation and this was determined for each material by measuring the absorbance at 330 nm for both polysulphone and NDA, 305 nm for 8MOP and 280 nm for phenothiazine before and after exposure in a spectrophotometer (Shimadzu Co., Kyoto, Japan). These wavelengths are used as it is at approximately these wavelengths that the greatest change in absorbance occurs. The post-exposure absorbance was measured as soon as practical following the exposure, thus eliminating errors due to any possible changes in absorbance in these films caused by the exposure to solar radiation was used to evaluate the time averaged UV spectrum over the period [13]. Knowledge of the spectrum incident to each site allowed calculation of both the biologically effective irradiance employing Equation (1) and the broadband irradiances.

An estimate of the source spectrum was extracted by using an assumed function, $S(\lambda)$, for the source spectrum based on a prior estimate of the spectrum as follows [13]:

$$S(\lambda) = (\lambda - \lambda_0) \left(\sum_{i=1}^n a_i \lambda^{i-1} \right)$$
(3)

where λ_0 is the wavelength where the irradiance is approximately zero (300 nm in this case), n is the order of the polynomial and a_i are coefficients determined by an iterative technique by minimising the $\chi 2$ value defined as:

$$\chi^{2} = \sum_{i=1}^{4} \frac{1}{\sigma^{2}} (\Delta A_{i} - \Delta A_{i})^{2}$$
(4)

where ΔA_i are the measured change in optical absorbance, σ is the error in ΔA_i and $\Delta A_i^{'}$ was calculated using the assumed function (Equation 3) in Equation (2).

2.2 UV Exposures

A spectrum evaluator was employed at each of the chest and shoulder of five human subjects undertaking outdoor activities in winter in Toowoomba $(27.5^{\circ} \text{ S} \text{ latitude})$ at approximately noon. Each spectrum evaluator was attached on top of the clothing with a clip. Subject 1 was a jogger on 2 August between 13:10 and 13:40 EST and subjects 4 to 5 were undertaking recreational activities in the park on 4 August between 11:55 and 12:25 EST. For each case, the spectrum evaluator was exposed for a period of 30 minutes. This time period was long enough to produce a measurable change in the optical absorbance of the dosimeter materials. From the measured changes in absorbance, the UV spectrum incident on each of the sites for each of the subjects was evaluated and the biologically effective irradiance was calculated employing the erythema action spectrum and the action spectrum is normalized to unity at 297 nm and the fish melanoma is normalized to unity at 302 nm.

3. Results

3.1 Spectral Irradiances

The action spectra for human erythema and fish melanoma in Figure 1 show the relative differences, particularly in the UVA waveband. In this Figure, the fish melanoma has been linearly interpolated between the measurement wavelength points of 302, 313, 365 and 405 nm employed by Setlow et al [4]. The fish melanoma action spectrum is approximately up to 5 times greater in the UVB waveband compared to the UVA waveband. In comparison the erythema action spectrum is approximately 1,000 to 10,000 greater in the UVB compared to the UVA.

The evaluated UV spectra time averaged over the exposure period for the shoulder and chest for subject 2 are provided in Figure 2(a) as an example of the different spectra. This illustrates the differences in UV spectra to the different body sites. The spectral biologically effective irradiances calculated from these spectra are provided in Figure 2(b) both for the erythemal and fish melanoma action spectra for both the shoulder and chest sites. The consequences of the higher effectiveness of the fish melanoma in the UVA waveband are evident here. Due to the higher UVA spectral irradiances, the spectral UVBE for fish melanoma is higher in the UVA compared to the UVB. On the other hand the spectral UVBE for erythema is of the order of 10 to 100 times less in the UVA waveband compared to the UVB waveband.

3.2 Broadband Irradiances

The irradiances integrated over the UVB waveband, the UVA waveband and total UV irradiance to the chest and shoulder of each subject and averaged over the five subjects are provided in Table 1. The error is represented as the standard error in the mean. The irradiances to the shoulder ranged from 62 to 80 μ W cm⁻² for the UVB and from 1006 to 1316 μ W cm⁻² for the UVA. Similarly, for the chest, the irradiances ranged from 23 to 49 μ W cm⁻² for the UVB and 391 to 991 μ W cm⁻² for the UVA. For both the chest and shoulder, the broadband UVA irradiances contribute 94 to 95% of the total irradiances. The differences in the irradiances to the chest and shoulder are due to the differences in the time averaged UV spectra to each of the sites as a result of the different locations and orientations as illustrated in Figure 2(a). These differences are also dependent on the season, time of day and any influences that affect the ratio of diffuse to direct UV radiation. It should be noted that from previous research, [13], the differences for the UV exposures between those calculated with the evaluated spectra and spectra measured with a calibrated spectroradiometer were found to be less than 20%.

3.3 Biologically Effective Irradiances

As explained in Equation 1, the biologically effective irradiances for both the human erythema and fish melanoma action spectrum may be obtained by integrating the product of the spectral irradiances and action spectra as given in Figure 2(b) over the entire UV waveband. The averages of the biologically effective irradiances for the chest and shoulder are provided in Table 2. The error is the standard error in the mean. The erythemal irradiances ranged from 1.7 to 3.6 μ W cm⁻² for the chest and from 4.6 to 5.9

 μ W cm⁻² for the shoulder. The relative contribution of the UVA waveband to the erythemal UV ranges from 13 to 16%. In comparison, the fish melanoma UV ranges from 91 to 223 μ W cm⁻² for the chest and 237 to 310 μ W cm⁻² for the shoulder with the UVA contributing 92 to 93% of the biologically effective irradiance. As seen from Figure 2(b), this is due to the higher relative effectiveness of this action spectrum in the UVA along with the higher UVA irradiances.

4. Conclusion and Discussion

The data presented in this paper indicate that the UVA waveband contributes about 95% of the personal exposure to solar radiation. In terms of erythemally effective exposure, the UVA waveband contributes about 14%. On the other hand, using the fish melanoma action spectrum, the contribution of the UVA waveband amounts to about 93%. The extrapolation of this action spectrum to humans is an open question. Nevertheless, the action spectrum suggests the photobiological importance to humans of the UVA waveband [17]. If the fish melanoma action spectrum is found to apply or at least resemble that for melanoma in humans, the UVA waveband could contribute to the risk of melanoma development during exposure to solar radiation. The usage of the fish melanoma action spectrum in this paper to calculate the biologically effective irradiances poses the possibility and opens the debate on the increased relative importance of the UVA waveband.

The choice of the action spectrum is critical in assessing the hazard of the UV exposure. The erythemal UVA contribution found in this research is still significant with regards to skin damage as previous research [18] has found that repeated exposures to suberythemal doses of UVA induce human skin damage. On the other hand, the high relative contribution of the UVA waveband to fish melanoma indicate the possibility that the UVA waveband may contribute to human melanoma.

This contribution of the UVA waveband to the biologically effective irradiance will vary with the atmospheric conditions, time of year and day and with the environment. The relative contribution of the UVA waveband may be even more significant on cloudy days when the ratio of UVA to UVB is higher compared to clear days [19] and on winter days when this ratio is higher compared to summer days [14]. Similarly, the UVA does not vary with time of day as much as the UVB and the relative contribution may be higher in the morning and afternoon compared to noon. For other body sites, geographic locations and seasons, the UVA irradiance will generally be higher than the UVB irradiance. Consequently, the contribution of the UVA waveband to the biologically effective exposure for the fish melanoma action spectrum will generally be high for other body sites, geographic locations and seasons.

Additionally, patients on photosensitising drugs need to be aware of the large UVA component with the requirement to reduce daylight exposure. If further data become available on an action spectrum for melanoma in humans, the method employed in this paper for evaluating personal biologically effective exposures can be utilised to determine the nature of the hazard for the induction of skin cancers.

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Site		Irradiances (μ W cm ⁻²)			
	UVB	UVA	Total UV		
Chest	37±5	698±98	735±102		
Shoulder	74±4	1180±68	1253±72		

Table 1 – The means of the broad band UV irradiances to the chest and shoulder of the subjects. The error is the standard error in the mean.

Table 2 – The means of the irradiances weighted by the human erythema and fish melanoma action spectra and the relative contribution of the UVA waveband to the total effective irradiances. The error is the standard error in the mean. The standard error for the relative contribution data is less than 0.005 in each case.

Site	Effective Irradiances (µW cm ⁻²)		Relative contribution of the UVA	
	Erythemal UV	Fish	Erythemal UV	Fish
		Melanoma UV		Melanoma UV
Chest	2.7±0.3	160±22	0.15	0.93
Shoulder	5.4±0.3	279±16	0.14	0.92

FIGURE CAPTIONS

- Figure 1 (1) The fish melanoma action spectrum [4] and (2) the human erythema action spectrum [1].
- Figure 2 (a) The evaluated spectra for the (1) shoulder and (2) chest of subject 2 and (b) the spectral UVBE for the (1) shoulder and (2) chest for the fish melanoma action spectrum and for the (3) shoulder and (4) chest for human erythema.



Figure 1



(a)

(b)



Figure 2

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