COMPARISON OF BIOLOGICALLY DAMAGING SPECTRAL SOLAR ULTRAVIOLET RADIATION AT A SOUTHERN HEMISPHERE SUB-TROPICAL SITE

A.V. Parisi[1*], J.Sabburg[1], M.G. Kimlin[1,2]

[1]Center for Astronomy, Solar Radiation and Climate, Faculty of Sciences, University of Southern Queensland, TOOWOOMBA. 4350. AUSTRALIA. Fax: 61 74 6312721.

[2]National Ultraviolet Monitoring Center, Department of Physics and Astronomy, University of Georgia, Athens, GA, USA 30606

[*]To whom correspondence should be addressed. Email: parisi@usq.edu.au

Short title: BIOLOGICALLY DAMAGING SOLAR SPECTRAL UV

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Abstract

The first data set of a complete year of biologically damaging spectral UV at a sub-tropical latitude in the SH has been presented. The new data provides a baseline data set against which comparisons can be made in the future to establish if there have been any long term trends in the biologically damaging UV. The general shape of the variation of the daily biologically damaging exposures through the year depends on the relative response of the various action spectra at the different wavelengths. The ratio of the daily erythemal to actinic exposures drops by approximately 20 to 25% from winter to summer. The ratio of the erythemal to DNA exposures drops by approximately 50% over the same period. In contrast, the ratio of the erythemal to plant damage exposures is higher in summer compared to winter. This is due to the changes in the relative proportion of UVA to UVB wavebands and relative responses of the different action spectra. The relative changes for the different action spectra show that the erythemal action spectrum cannot be used as a proxy for other biologically damaging responses.

1. INTRODUCTION

Biologically damaging ultraviolet radiation (UV) may be calculated by measuring a solar spectrum with a spectroradiometer and weighting the spectrum with the action spectrum for the biologically damaging process being considered. Alternatively, it may be measured with a broadband meter or dosimeter with a response that has been designed to match as closely as possible one appropriate action spectrum to be investigated. Although more expensive and technically demanding, one of the advantages of measuring the UV spectrum with a spectroradiometer is that the spectrum may be weighted with any number of action spectra. Also, as new action spectra are developed, they may be applied to historical spectral UV data. Additionally, minor variations in the spectrum of the solar UV may have a more pronounced effect on the biologically damaging UV due to the wavelength dependence of the biologically damaging process. Broadband UV measuring instruments do not provide any information on the specific wavelength variations. Consequently, in order to further understand the solar UV environment on the surface of the earth, spectral UV measurements are required in addition to broadband measurements.

Spectral UV and biologically damaging UV have been quantified on a horizontal plane and in a number of different environments over relatively short periods, for example: over snow (McKenzie and Kotkamp, 1996); in tree shade (Parisi and Kimlin, 1999a); in cars (Kimlin et al., 2002a); and at orientations other than on a horizontal plane (Webb et al., 1999; Parisi and Kimlin, 1999b). The long term monitoring of the spectral UV is undertaken in the northern and southern hemisphere with spectroradiometers based on monochromators or filters. Despite the lower atmospheric ozone in the southern hemisphere (SH) and the reported cases of higher solar UV compared to the northern hemisphere (NH) (McKenzie et al., 1993), there are less sites undertaking long term monitoring of the spectral solar UV in the SH compared to the northern hemisphere. Examples of sites where monitoring of long term spectral UV have been established in the SH are Lauder (45^{o} S), New Zealand (McKenzie et al., 1992), Ushuaia (55^{o} S), Argentina (Diaz et al., 1996) and Palmer Station (64.8^{o} S) and McMurdo Station (77.9^{o} S), Antarctica (Frederick et al., 1998).

In the SH, Australia has the highest incidence rates of skin cancer (NHMRC, 1996) in the world with Queensland having the highest incidence rates of the Australian states. In Queensland, the monitoring of the spectral UV has been limited to short measurement campaigns (Bernhard et al., 1997). The Australian Radiation Protection and Nuclear Safety Agency has been running a network of broadband UV instruments across Australia, with some stationed in Queensland (Roy et al., 1995). Additionally, the analysis of broadband UV, ozone and cloud has been presented for a sub-tropical site (Sabburg et al., 1997; 2001). However, there has been no long term monitoring of the spectral UV in Queensland.

Recently, Kimlin et al., (2002b) compared a year of erythemally weighted UV data, calculated from spectral UV, from a site in Queensland to data from similar latitude sites in the NH. This current paper extends this previous research by reporting a complete year of biologically damaging UV for a range of other action spectra for a site in Queensland. This

is the first data set of this biologically damaging UV over a complete year in Queensland, Australia and in any southern sub-tropical latitudes.

2. MATERIALS AND METHODS

2.1 Spectral Measurements

The spectral UV was measured with a Brewer spectroradiometer MKIII model (Kipp and Zonen Inc. Saskatoon, Canada) that scanned from 286.5 to 363 nm in 0.5 nm steps. This instrument employs a double monochromator for the wavelength dispersion and a quartz dome and Teflon diffuser as the input optics. The instrument was in a weather proof box and located at the University of Southern Queensland, (27.5°S, 151.9°E, 693 m above sea level) Toowoomba, Australia on the top of a building with an unobstructed field of view except for an office block, approximately 50 meters to the south west, and 20 meters higher than the instrument. However, this did not shade the instrument at any time of the year. Data was collected over a year between 18 July 2000 and 17 July 2001. The Brewer also measured the ozone column amounts. The control and data logging software was located at a distance of approximately 80 m on a computer in a laboratory in the building. A user defined scanning schedule designed to maximize the number of solar UV spectral measurements throughout the course of the day was employed.

The spectral UV data was corrected for dark count, dead time and stray light using the algorithms of Sci-Tec (1999). A correction for the cosine response was applied as described by Sabburg et al. (2002). The Brewer employs the spectral lines of an internal low pressure mercury lamp for wavelength calibration. A daily response function, based on a calibration performed by Kipp & Zonen using a 1000 W FEL lamp calibrated for spectral irradiance traceable to NIST, was applied to calculate the UV irradiances. This function was based on the single response function that was determined at the beginning of the data collection period. A stability check was performed at least each month using three sets of 50 W tungsten lamps at each time. This was performed on-site with the lamps in a light tight housing that mounted directly over the input optics of the instrument. It was found that the overall stability of the Brewer (due to both temporal and temperature dependence), was approximately ± 2% during the data collection period. As a result no temporal or temperature corrections were applied to the data (Kipp & Zonen, personal communication).

2.2 Biologically Damaging UV

In order to calculate the biologically damaging UV, action spectra specific to biologically damaging processes for human skin and eyes and for plants were employed (Figure 1), namely, the action spectra for erythema, which will be abbreviated as DUV (CIE, 1987), actinic hazard (IRPA, 1989), photoconjunctivitis (CIE, 1986a), photokeratitis (CIE, 1986b), DNA damage (Setlow, 1974), generalized plant damage (Caldwell, 1971) and plant damage (Coohill, 1989). The generalized plant damage action spectrum is the average of eight different action spectra and the plant damage action spectrum is for a combination of results for a variety of photoresponses in intact cucumbers. These action spectra were selected as they provide examples with a relative effectiveness over a range of wavelengths.

Knowledge of the spectral UV measured at a wavelength increment $\Delta\lambda$ allows the biologically damaging UV, UVBE to be calculated as follows:

$$UVBE = \sum_{UV} S(\lambda) A(\lambda) \Delta\lambda \quad (1)$$

where $S(\lambda)$ is the spectral irradiance, $A(\lambda)$ is the action spectrum for the particular biological process and the wavelength range is over the solar terrestrial UV waveband. For erythema, DNA and generalized plant damage, the numerical parameterization available in the literature has been employed. For the remainder, each of the action spectra have been linearly interpolated between the data points to 0.5 nm steps to coincide with the spectral UV wavelength increment.

The biologically damaging UV irradiances for each action spectrum were calculated employing Equation 1 at each of the scan times. Simpson's rule was employed to calculate the daily biologically damaging UV from these irradiances for each respective action spectrum. Over the day, if the ratio of the number of scans divided by the maximum possible number of scans was less than 0.95, the daily exposure was rejected to minimize the uncertainty in the daily exposure. The overall uncertainty in the UV irradiances measured by the Brewer was estimated to be ± 6% (Sabburg et al., 2002).

3. RESULTS

3.1 Daily UV

The diurnal variations of the actinic, DNA and plant damage irradiances are shown for a typical relatively cloud free day and a partially cloudy day for each of the seasons in Figure 2. Cloud is a major factor, both in the unpredictability and the size of the variations of the biologically damaging UV throughout the day. The average ozone for each of the days varies from 260 to 333 DU (Dobson Units) with the highest ozone value occurring in spring (September to November). The maximum reduction in the daily exposures due to clouds is of the order of a factor of 10.

For the relatively cloud free days the increase in the biologically damaging UV from the morning to noon depends on the relative shape of the action spectrum. This is illustrated in Table 1 where the irradiances at noon are compared to those at 9 am for a relatively cloud free day in summer (23 Dec 2000) and in winter (11 August 2000). The irradiances representing 9 am and noon were measured by a scan within 10 minutes either side of each of these times. In all cases, the largest change from 9 am to noon occurs in winter. In both summer and winter, the difference is highest for the action spectrum with more relative sensitivity towards the shorter wavelengths and lowest for action spectra with sensitivity towards the longer wavelengths. The ratio of UVA (320-400 nm) to UVB (280-320 nm) is higher in the morning and afternoon time periods, providing a greater weighting to the UVA part of the action spectrum (Kimlin et al., 2002c).

3.2 Annual UV

The daily actinic, DNA and plant damage exposures calculated from the spectral UV scans for the year are shown in Figure 3 as examples of the variation throughout the year. The daily exposures were calculated from a total of 8,139 spectral scans. The clear sky envelope of the maximum exposures can be seen. The general shape of the variation through the year is similar for the actinic and DNA exposures. The reason for this is that both these action spectra have a high relative response for UVB wavelengths. In comparison, the plant damage action spectrum has a higher response for UVA wavelengths compared to the other two. As a result the envelope of the maximum exposures has a different shape relative to the other two. This is due to the ratio of the UVA to UVB irradiances changing with the seasons. Specifically, the relative proportion of UVA compared to UVB is higher in winter than in summer due to the longer atmospheric path length and increased attenuation of the shorter wavelengths in winter (Kimlin et al., 2002c).

In order to illustrate the relative change of the biologically damaging exposures throughout the year, the ratios of the DUV exposures to the other biologically damaging exposures are plotted in Figure 4. The data points that lie above or below the general envelopes of data points may be due to variations in the UVA to UVB ratio. For the actinic, DNA, photoconjunctivitis, photokeratitis and generalized plant damage, their relative response in the UVA is lower than for the erythema action spectrum. The drop in the ratio from winter to summer is due to lower relative proportion of UVA to UVB in summer compared to winter. The ratio DUV/Actinic drops by approximately 20 to 25% from winter to summer. As a comparison for DUV/DNA, the drop over the same period is of the order of 50%. The difference in the amount of drop is due to the relative shape of each action spectrum and the relative responses in the UVB and UVA wavebands. In contrast the ratio DUV/Plant is higher in the summer compared to the winter months. This is due to the higher relative response of the plant damage action spectrum compared to the erythema action spectrum.

The daily UV exposures for each action spectrum are averaged over each of the seasons and the maximum daily exposures over the year for the different action spectra (last row) are shown in Table 2. The ratio of the average summer to winter exposures is different for the various action spectra. It is a maximum (5.5) for the photoconjunctivitis action spectrum that has a response in the UVB only and a minimum (2.2) for the plant damage that has a higher response in the UVA compared to the other action spectra.

4. DISCUSSION

The first data set of a complete year of biologically damaging spectral UV at a sub-tropical latitude in the SH has been presented. Knowledge of detailed spectral UV at the earth's surface increases the understanding of the biologically damaging UV for plants and humans. The new data provides a baseline data set against which comparisons can be made in the future to establish if there have been any long term trends in the biologically damaging UV. Baseline data at the southern latitudes of this research is important due to the scarcity of spectral UV measuring stations in the SH.

The maximum daily DUV (7,794 J m^{-2}) reported by Bernhard et al. (1997) for Townsville (19.33^{o} S), was comparable to that found in this current research. The short term measurement campaign in Townsville was from 24 December, 1995 to 20 January, 1996. The average daily DUV over that period in Townsville was 6,060 J m^{-2}. The average for the same days of the year for this research was 5,535 J m^{-2}. The main factor causing the lower average would be the difference in latitude; however, it is worth noting that the maximum values were comparable. This may be due to the relatively low tropospheric pollution and higher altitude of the sub-tropical site in this research. This illustrates the high biologically damaging UV exposures for both DUV and the other action spectra at the sub-tropical latitudes of this current research.

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Table 1 The ratio of the biologically damaging irradiance at noon compared to 9 am for each of the action spectra for a relatively clear day in winter and in summer.

	Ratio						
	DUV	Actinic	Photo-keratitis	DNA	Gen. Plant	Photo-conjunctivitis	Plant damage
Winter	2.3	2.9	2.6	3.7	3.1	6.0	1.8
Summer	1.7	1.7	1.7	2.1	1.8	2.2	1.4

Table 2 Seasonal averages, summer/winter ratio and the maximum of the daily exposures for the action spectra.

	Biologically damaging UV (J m^{-2})						
	DUV	Actinic	Photo-keratitis	DNA	Gen. Plant	Photo-conjunctivitis	Plant damage
Winter	1657	405	989	24	448	5	26,805
Spring	3949	1051	2407	72	1203	16	50,349
Summer	5718	1594	3463	119	1832	27	60,740
Autumn	2899	775	1772	53	890	12	36,271
Summer/Winter	3.5	3.9	3.5	5.0	4.1	5.5	2.3
Maximum	7795	2317	4925	180	2769	42	83,487

Figure Captions

Figure 1 The action spectrum (response versus wavelength), for (1) erythema (CIE, 1987), (2) actinic hazard (IRPA, 1989), (3) DNA damage (Setlow, 1974), (4) photoconjunctivitis (CIE, 1986a), (5) photokeratitis (CIE, 1986b), (6) generalized plant damage (Caldwell, 1971) and (7) plant damage (Coohill, 1989).

Figure 2 Daily variation of the biologically damaging UV for the actinic, DNA damage and plant damage action spectra for relatively cloud free days (left column) and cloudy days (right column).

Figure 3 Daily actinic, DNA and plant damage exposures calculated from the spectral UV scans during July 2000 to July 2001 where day 1 corresponds to 18 July 2000.

Figure 4 The ratio of the daily DUV exposures to the other biologically damaging UV exposures where day 1 corresponds to 18 July 2000.

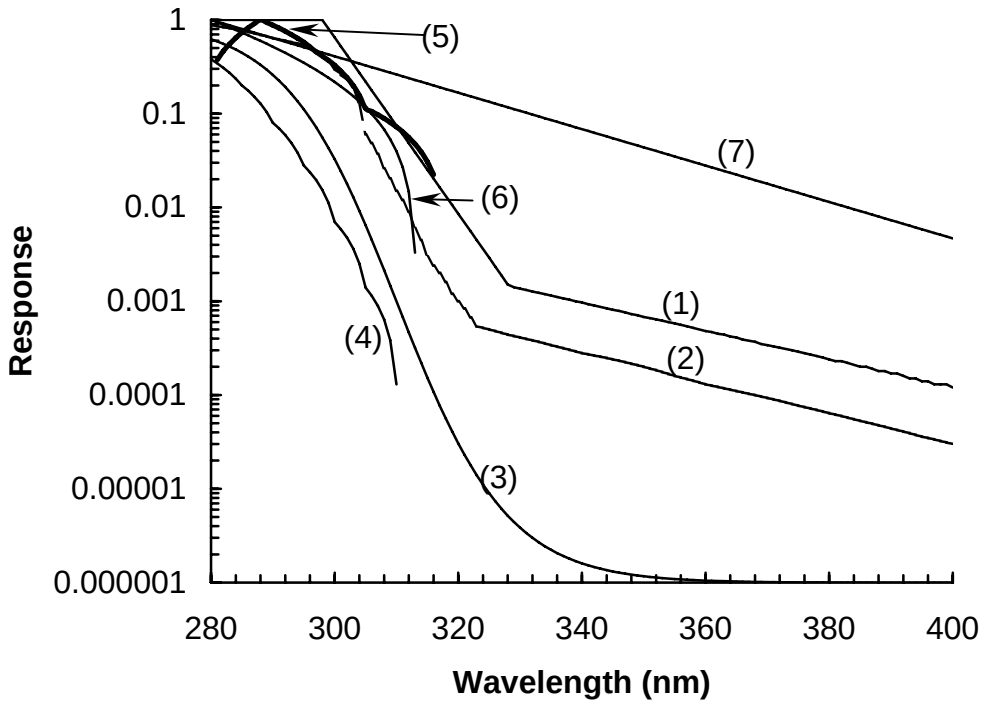
1
0.1
0.01
0.001
0.0001
0.00001
0.000001
Response
280
300
320
340
360
380
400
Wavelength (nm)
(1)
(2)
(3)
(4)
(5)
(6)
(7)

Figure 1

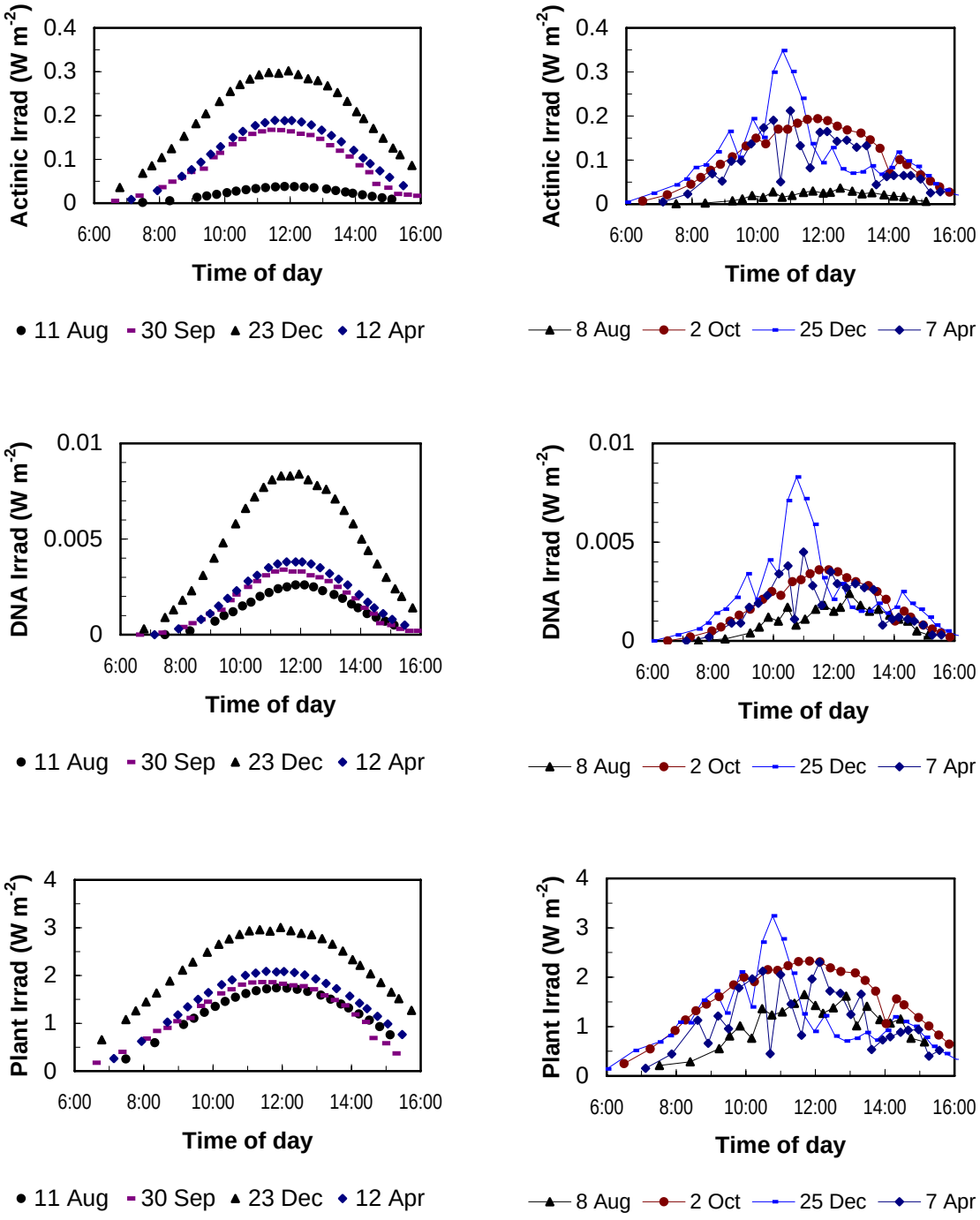
Actinic Irrad (W m-2)
0.4
0.3
0.2
0.1
0
6:00
8:00
10:00
12:00
14:00
16:00
Time of day
11 Aug
30 Sep
23 Dec
12 Apr
8 Aug
2 Oct
25 Dec
7 Apr
DNA Irrad (W m-2)
0.01
0.005
Plant Irrad (W m-2)
4
3
2
1

Figure 2

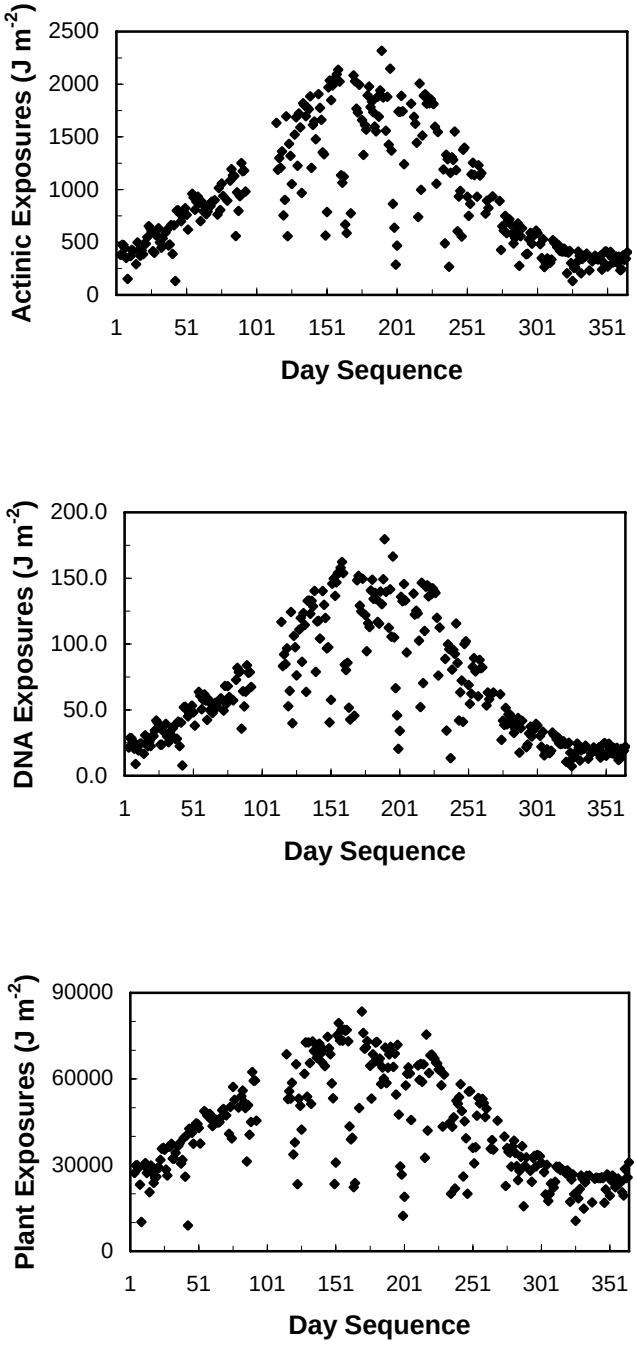
Actinic Exposures (J m-2)
2500
2000
1500
1000
500
0
1
51
101
151
201
251
301
351
Day Sequence
DNA Exposures (J m-2)
200.0
150.0
100.0
50.0
0.0
1
51
101
151
201
251
301
351
Day Sequence
Plant Exposures (J m-2)
90000
60000
30000
0
1
51
101
151
201
251
301
351
Day Sequence

Figure 3

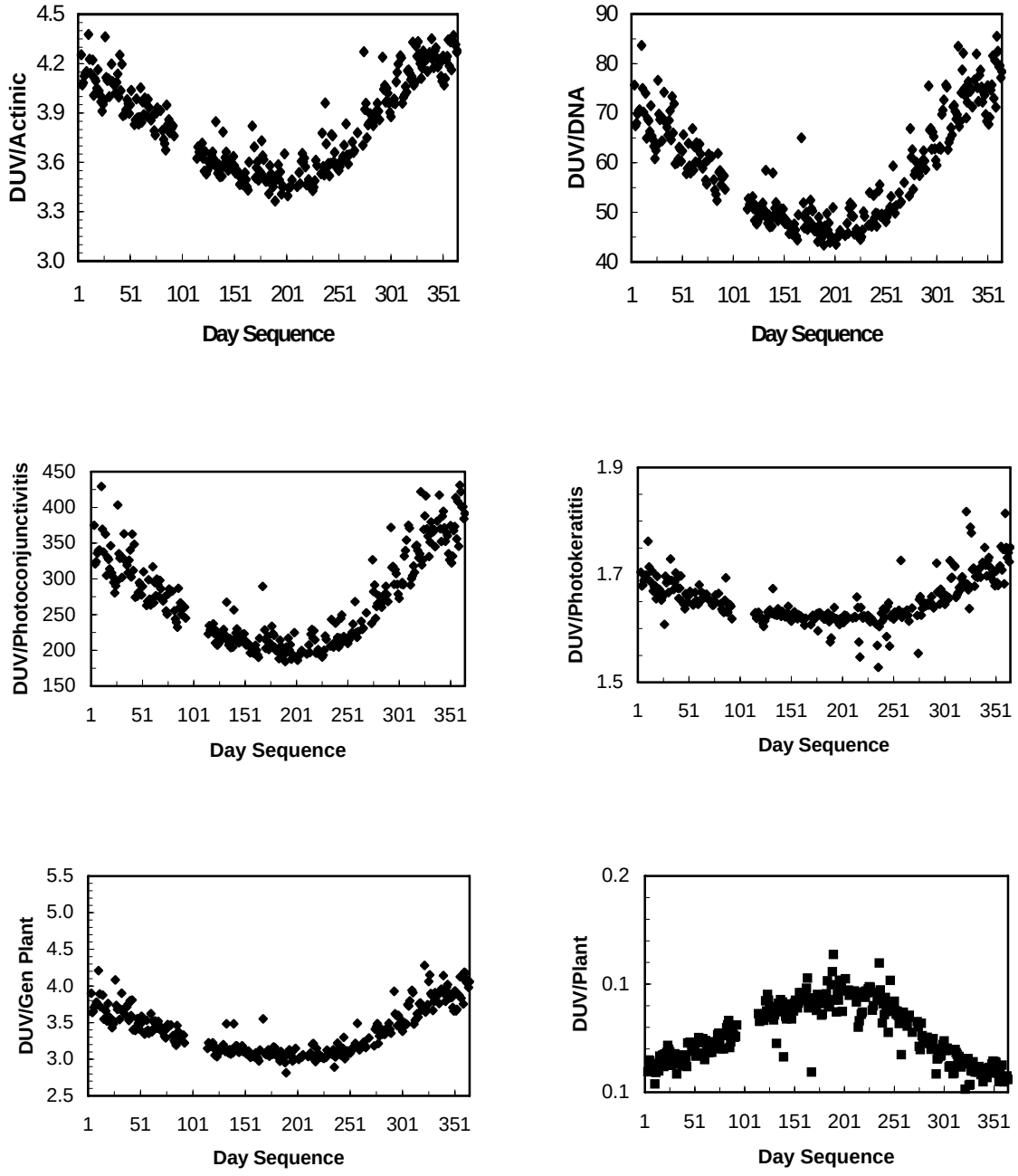
DUV/Actinic
4.5
4.2
3.9
3.6
3.3
3.0
1 51 101 151 201 251 301 351
Day Sequence
DUV/DNA
90
80
70
60
50
40
1 51 101 151 201 251 301 351
Day Sequence
DUV/Photoconjunctivitis
450
400
350
300
250
200
150
1 51 101 151 201 251 301 351
Day Sequence
DUV/Photokeratitis
1.9
1.7
1.5
1 51 101 151 201 251 301 351
Day Sequence
DUV/Gen Plant
5.5
5.0
4.5
4.0
3.5
3.0
2.5
1 51 101 151 201 251 301 351
Day Sequence
DUV/Plant
0.2
0.1
0.1
1 51 101 151 201 251 301 351
Day Sequence

Figure 4