

Measurements of the upper body ultraviolet exposure to golfers: non-melanoma skin cancer risk, and the potential benefits of exposure to sunlight¹

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Abstract.

Background. Geographically, Queensland presents an extreme ultraviolet exposure climate to members of the public engaged in outdoor recreational activity. The risk of developing a skin cancer or an eye disease as a result of incidental exposure to naturally occurring ultraviolet radiation in the outdoor environment is proportionately high in a Queensland population compared to fair skinned population groups residing in comparable Northern Hemisphere latitudes. In contrast to these risks, elderly members of this high growth population group have been reported to be vitamin D deficient. The risks and potential benefits of exposure to sunlight in southern Queensland are assessed in this study with respect to recreational golfing. This sport is a popular recreational activity for the Queensland population and must be played during daylight hours.

Methods. The erythemal and vitamin D effective ultraviolet exposure measured to the forearm, upper back and vertex are presented for individuals playing golf under various atmospheric conditions in a seven month period extending from summer to winter.

Results. Mean summertime exposures were measured in the 2008 study period to be 1.4, 2.2 and 3.2 SED at forearm, upper back and vertex sites respectively compared to respective wintertime forearm, upper back and vertex exposures of 0.2, 0.3, 0.5 SED, where summertime exposures were recorded in the mean solar zenith angle ranges of 56° to 59° and wintertime exposures were recorded in the mean solar zenith angle range 74° to 83°. Vitamin D₃ effective exposures were determined to vary from between 225 Jm⁻², 325 Jm⁻² and 475Jm⁻² during summer and 48 Jm⁻², 59 Jm⁻² and 88 Jm⁻² during winter for the respective forearm, upper back and vertex body sites measured in the above mean solar zenith angle ranges.

Conclusion. Exposures to ambient UV during winter on the golf course between 3:00 pm and 5:30 pm could be beneficial for office workers for the production of vitamin D. Optimising exposure periods to late afternoon in the winter months and taking adequate sun protection measures in the summer months are important strategies that golfers can utilise for long term preventative health.

Keywords: Golfers; Ultraviolet; skin cancer; Vitamin D

Introduction

Across Australia during each week thousands of amateur and professional golfers are exposed to potentially damaging amounts of solar ultraviolet radiation (UVR) during their time spent on the golf course. It is well known that excessive exposure to UVR can increase the risk of life threatening diseases which include melanoma and non-melanoma skin cancers alongside other debilitating eye conditions such as conjunctivitis, keratitis and cataracts. Additionally, long-term UVR exposure can also lead to cosmetic skin damage by increasing the loss of skin elasticity and moisture and in turn, accelerating the onset of wrinkling. To date there has been limited qualitative research presented linking the effect of short and long-term UVR exposure to persons involved in various sports and other outdoor recreational activities. A number of studies have however previously detailed measurements of personal UVR exposure received while playing sport. *Bacillus subtilis* spore film dosimeters have been used to measure exposure to cyclists and athletes (1,2). Digital UVR dosimeters have been recently employed to measure UVB (280 – 320 nm) and UVA (320 – 400 nm) incident upon alpine skiers (3), and polysulphone dosimeters have been used to measure the UVR received by school children while playing soccer and basketball (4).

Exposures received while playing sport can make a significant contribution toward increasing the risk of developing melanoma and non-melanoma skin cancers. In Australia, the national occupational limit of exposure to solar UVR incident upon the skin or eye lies between approximately 105 Jm^{-2} and 135 Jm^{-2} for an 8 hour working day at mid latitudes (30°S) between 9:00 am and 5:00 pm (5,6,7). Measurements of UVR received by persons playing sport frequently exceed this level, where specifically the occupational exposure limit adopted by the NHMRC (6) and ARPANSA (7) standards is that specified by the International Radiation Protection Association (8) and represents an occupational weighted UV exposure of 30 Jm^{-2} (9).

Different UVR exposure distributions measured across different sports suggest that specific UVR exposures may not be transferable. Thieden et al. (10) measured the UVR exposure to the wrist of 24 Danish golfers and determined the daily erythemally effective UVR to range from 70 Jm^{-2} to 370 Jm^{-2} . This group was shown to have the highest median percentage of the ambient UV in the study population which included sun worshippers, school children, indoor workers and outdoor gardeners. The results of this study suggest that playing golf is a potentially high risk behaviour. The risk of developing a skin cancer is therefore likely to be significant for golfers as this population group is frequently exposed to high levels of ambient UVR. Sung and Slocum (11) determined using polysulphone dosimeters that the upper back

region of golfers received higher exposures than the front of the body. For golfers, these findings indicate that the back of the neck is likely to be a region of greater risk of over exposure to UVR than perhaps the face which can be protected to some degree by the brim of a hat.

Exposure to sunlight is however necessary for the healthy production of Vitamin D, with diet accounting for limited intake (12). Vitamin D deficiency has been linked to the development of diseases including rickets (13), type I diabetes (14), multiple sclerosis (15) and the possible development of some cancers (16,17,18). In Queensland, due to low geographical latitude, the biological response of vitamin D₃ production in human skin exceeds the erythemally effective UV. This is due to the vitamin D₃ response having a greater weighting at the shorter UVB wavelengths that are more abundant at sub-tropical and tropical latitudes. Effectively, healthy vitamin D₃ photolysis in fair skin is reached well before the erythema sunburning reaction is noticed. Nevertheless, vitamin D deficiencies are common in the elderly and are more likely to affect darker skin types even in locations that experience high levels of ambient UV (19,20). Unprotected exposures to UVR incurred while playing golf are therefore likely to be beneficial to certain population groups provided the exposure is received within set exposure intervals that do not elicit excessive and possibly carcinogenic exposures. In a position statement issued by the Australian and New Zealand Bone and Mineral Society, Endocrine Society, Osteoporosis Australia, Australian College of Dermatologists and the Cancer Council Australia, the recommended levels of exposure to sunlight for the adequate production of vitamin D were stated to be five minutes solar UV exposure either side of the peak UV period on most days of the week in summer and 2 to 3 hours solar UV exposure over a week in winter. In this research, the vitamin D₃ exposures recommended in this statement were calculated and compared to the exposures received by golfers frequenting golf courses for periods of play experienced between 3:00 pm and 5:00 pm during both summer and winter months.

Materials and Methods

Measurements of the erythemally effective (UVE) and vitamin D₃ (UVD) inducing ultraviolet were taken at the forearm, upper back and vertex sites of two golfers frequenting four golf courses across the Darling Downs, southern Queensland region between February and August 2008. These body sites represent those sites most frequently exposed to UV during a golf game as the legs are often covered by long trousers, and long sleeve shirts are not often worn as these inhibit the golf swing. The upper back site was chosen as a convenient site location

being a comfortable location for the golfer and for approximating exposures received at the back of the neck. Measurements of exposure to these sites were performed using calibrated polysulphone dosimeters. These dosimeters were miniaturised for this application and consisted of polysulphone film cast to an approximate thickness of 40 μm and adhered over a 6 mm diameter circular aperture punched into lightweight flexible cardboard frames measuring 10 mm by 15 mm. Dosimeters were attached to the body using medical tape. Figure 1 shows the position of three dosimeters attached to the forearm, upper back and vertex. The dosimeters used for this research application have been applied previously to measure personal exposures to school children and have a quoted uncertainty of $\pm 24\%$ (4). The dosimeters were calibrated to the horizontal plane ambient UV measured by a scanning spectroradiometer (Bentham Instruments, Reading, UK) located at the University of Southern Queensland Toowoomba campus (28°S, 153°E) over several days so that a wide range of solar zenith angles (SZA) and atmospheric conditions could be taken into account. Toowoomba is located at 690 m altitude in a semi-rural location and experiences limited atmospheric pollution, hence concentrations of atmospheric aerosol were not monitored during the measurement campaign. Calibrating measurements of the ambient horizontal plane UV exposure were taken over the 280 nm to 400 nm range and weighted to the respective erythemal (21) and vitamin D₃ human response (22).

FIGURE 1

Measurements of UVE and UVD exposure were recorded for a total of 26 person days in the February to August period where one person day represents the exposure measured for an individual playing a single round of golf. Exposure periods were limited to 9 hole rounds of golf for each of two players, extending from periods of 2 hours to 2 hours 30 minutes and were played at golf courses located near the city of Toowoomba, with the most distant course being approximately 40 km from the city in which the three other courses were located. Rounds of golf were played under varying levels of cloud cover which ranged from clear sky to completely overcast conditions. The SZA at this location in southern Queensland varied from between 41° to 95° during the golf playing periods which were limited to the mid to late afternoon and early morning.

Measurements of UVE and UVD exposure were recorded during the same time interval golf was being played to office workers located at the University of Southern Queensland. This served as the control exposure group and consisted of two office workers that were located

predominantly indoors during the golfing exposure periods. Measurements of forearm, upper back and vertex UVE and UVD exposure were recorded for the office workers.

The risk of developing a basal cell carcinoma (BCC) or a squamous cell carcinoma (SCC) was calculated for the golfers compared to the office workers. Both the golfers and office workers were assumed to have spent the equivalent amount of time in the sun during the early stages of life. Furthermore, the cancer risk calculated in this study assumes the golfers take up the sport on the commencement of their working life and follow a weekly playing pattern repeatable annually. For golfers that take up the sport later in life, the risk of developing a skin cancer will be reduced. Similarly, golfers taking up the sport earlier in life, or playing more frequently than once per week, increase their skin cancer risk above the estimates provided in this research. The annual contribution to the risk of developing a non-melanoma skin cancer (NMSC) may be expressed as (23):

$$Risk = kS^\beta A^\alpha \quad (1)$$

where the risk of developing NMSC is dependent upon the cumulative annual exposure, S and the age of the individual, A . In the equation, α and β are the respective age dependent and biological amplification factor constants which can be determined by epidemiological evidence for BCC and SCC respectively. The cumulative UVE exposure of a golfer's site exposure, S_0 at either the forearm, upper back or vertex body site was expressed as a ratio of the cumulative UVE exposure received by the office worker's exposure at the same site, S , in accordance with the method employed by Wong et al. (24). Using the method developed by Wong et al. (24), the dependence on age and the constant of proportionality, are removed in the ratio:

$$Risk_{RELATIVE} = \frac{k(S_0)^\beta A^\alpha}{k(S)^\beta A^\alpha} \quad (2)$$

leaving the NMSC risk of golfers expressed relative to the office workers as being dependent upon the cumulative yearly anatomical site erythematous exposure of the golfer compared to the office worker and the respective biological amplification factors for BCC and SCC. For this research, the biological amplification factors, β of 1.7 and 2.3 were employed for BCC and SCC risk respectively as cited by Vishvakarman and Wong (25).

Results

The relative body site distribution of erythemally effective ultraviolet

The calibrated erythemally effective body site UVE exposure measured to golfers in the February to August period is given in Figure 2. Measurements of the site distribution of UVE exposure indicate that the greatest exposures were recorded at vertex sites for most cases. This is most evident during the early part of the year during periods of high solar elevation (low SZA) which show a larger variation between body site exposures. UVE exposures received by the forearm were shown to be lower than exposures measured to the upper back site of both golfers during the early part of the year. This trend was observed to continue during the winter months of the February to August trial period. A clear seasonal variation in body site UVE exposure is evident in the figure.

FIGURE 2

Table 1 lists the body site UVE exposure of the golfers expressed relative to the ambient UVE measured by the University of Southern Queensland's spectroradiometer. The mean vertex UVE exposure expressed relative to the ambient UVE was determined to be 0.69 ± 0.30 (1σ). The upper back site was the next highest relative exposure measured to the golfers and was expressed relative to the ambient UVE exposure as 0.49 ± 0.29 (1σ), followed by exposures measured to the forearm site 0.31 ± 0.19 (1σ). Both the data presented in Table 1 and Figure 2 indicate the relative distribution of UVE exposure received by golfers. In all measurements of golfer body site UVE exposure, the relative trend of the greatest exposure being received at the vertex, followed by the upper back and the forearm was preserved for most trials. Golfer 2 experienced a higher upper back exposure in early May. A possible explanation for this measurement may be the presence of dust on the dosimeter film during post exposure measurement.

Uncertainty of the calibrated dosimeters accounts largely for the variation in the exposures represented relative to the measured ambient exposure. Local golf course variations in cloud cover and atmospheric conditions relative to the ambient measurement site also influence the golfers exposure presented in Table 1. Furthermore, the proportion of time each golfer spends in a shaded environment during their individual round affects the site exposure relative to the available ambient UV.

TABLE 1

The likelihood of receiving a high UVE exposure increases with decreasing SZA. Figure 3, plots the vertex, upper back and forearm UVE exposure of each trial relative to mean SZA. Clearly evident in the figure is the dependence of each body site exposure on SZA.

FIGURE 3

Skin cancer risk

The risk of developing a non-melanoma skin cancer, determined as the ratio of the cumulative annual exposure of a golfer playing weekly for two hours compared to an office worker was determined by application of the cumulative yearly exposure where this exposure is determined by:

$$E_A(b) = \sum_{d=1}^{d=246} E_n(d) + \sum_{h=1}^{h=119} E_w(d) + \sum_{d=1}^{d=52} E_g(d) \quad (3)$$

where E_A is the estimated total annual UVE exposure of the golfer, received at a specific body site b , $E_n(d)$ is the UVE exposure received during the number of office days in the year, $E_w(d)$ is the UVE exposure received on the number of weekend and leave days, and $E_g(d)$ is the UVE exposure received on the number of office days in the year during which the golfer plays a 9 hole round, assuming a round is played weekly. UVE exposures and the relative BCC and SCC risk are presented in Table 2 whereby the relative BCC and SCC risk was determined by the $E_A(b)$ ratios of the golfers to the office workers who have 0 $E_g(d)$ days. The exposure for the 52 days when golf was played, $E_g(d)$ was determined in this instance from an interpolation of each measured exposure of forearm and upper back data presented in Figure 2 using both cloud free and cloud affected data and assumes this UVE exposure was received in addition to exposures estimated for the indoor office workers on those particular days. The interpolation of the measured forearm and upper back data was determined using a linear estimate of annual UVE exposure to both body sites where exposure decreased from summer to winter and increased from winter to summer. Here, UVE exposures in the later part of the year were mirrored from the interpolated estimate measured from February to August according to a linear fit of the measured exposure data provided in Figure 2. Non melanoma skin cancer risk was not calculated for a vertex site as this site is often protected by either a hat or hair cover.

Using this method, the total $E_g(d)$ exposure for every seventh day of the year (52 days) estimated at the latitude of 28°S was 47 SED for a forearm site and 69 SED for a golfer's upper back site. The total annual exposure for the number of office days, $E_n(d)$ was substituted from the estimate of Vishvakarmen and Wong (25) which assumes an indoor worker spends approximately 1.5 hours outdoors travelling to, from and at work daily. This estimate is given for indoor workers for hand and neck sites which for this research is assumed to approximate UVE exposures received at forearm and upper back sites and is stated to be 32 kJm⁻² or 320 SED. The estimated annual exposure received on weekends and during three weeks annual leave was also substituted from the estimates of Vishvakarmen and Wong (25), where this exposure was approximated to be 45 kJm⁻² or 450 SED and is taken to represent $E_w(d)$ based on ambient UVE exposure measured to the neck and hand sites of teachers and postal workers in central Queensland (26) and assumes an outdoor exposure of 8 hours per weekend and 6 hours per day during three weeks annual leave.

TABLE 2

The increased lifetime risk of developing either a BCC or SCC due to playing a weekly round of golf is clearly a result of increased outdoor behaviour compared to the office worker. Figure 4(a) distinctly shows that on average across all the trials, the golfers received far more UVE in comparison to the office workers.

Vitamin D effective exposure

The UVD response of human skin is higher in the UVB wavelengths. The result is that at the latitudes of this research, the ambient UV is more likely to illicit a positive UVD response than UVE under similar conditions. This was immediately noticeable in a comparison between UVE and UVD exposures measured to the office worker group (Figure 4(b)). As was the case with the UVE, the golfers received a much more substantial amount of UVD when compared to the office workers. However under low ambient UV conditions experienced in the office, the UVD effective exposure was generally in greater proportion for the office worker group compared to the golfers than the proportional UVE exposures. In direct comparison of the UVE on the vertex region, the golfers received average exposures 52 times greater than the office workers. Quantification of the different amounts of UVD exposure received by both golfers and office workers showed that on the vertex region, the golfers

accumulated average exposures 28 times more substantial in comparison to those delivered to the office workers. A reasonable conclusion to draw from this observation is that UVD exposure need not be substantial to produce a positive reaction.

FIGURE 4

Recommended exposures to sunlight are currently set at 5 minutes exposure either side of peak UV periods in summer and 2 to 3 hours exposure per week in winter. As a guide for this research, these UVD exposures were calculated about the periods of maximum and minimum solar UV irradiance occurring near to 21 January (one month from summer solstice and near to the earth's closest approach to the sun) and 21 June (winter solstice) for the year 2008. In summer on 21 January, 5 minutes exposure to sunlight modelled using a previously discussed algorithm (27) on a horizontal plane at 300 DU for the latitude of the study location under clear sky conditions between 3:00 pm to 3:05 pm represents approximately 80 Jm^{-2} UVD. In winter, using the same physical parameters to model exposure in summer, 20 minutes exposure to sunlight between 3:00 pm to 3:20 pm for 7 days of the week beginning on 17 June and ending 23 June represents 140 minutes of exposure time, which lies close to the lower recommended winter exposure of 2 hours per week, giving approximately 120 Jm^{-2} UVD. Although UVD exposure will vary for different periods of the day, atmospheric conditions and cloud cover, a tentative estimate of 100 Jm^{-2} UVD was taken in this research to represent a sufficiently healthy exposure to sunlight for the golfers in this study.

Figure 5 compares the calibrated UVD exposure measured at vertex, forearm and upper back sites of the golfers normalised to 100 Jm^{-2} UVD in the February to August study period showing clearly the limited value of a weekly game of golf in the summer months for the healthy synthesis of vitamin D_3 as the exposure is too high. However, it highlights also that winter time exposures received by the golfers between May and August were on par with our tentative estimate for recommended weekly winter exposures. The results suggest, that for the study latitude a weekly 9 hole round of golf played in winter after 3:00 pm is a suitable outdoor activity for healthy vitamin D_3 synthesis. The mean exposure, normalised to 100 Jm^{-2} UVD for exposures recorded between May and August was 0.8 ± 0.3 (1σ) at the vertex, 0.4 ± 0.3 (1σ) at the forearm and 0.6 ± 0.3 (1σ) on the upper back site.

FIGURE 5

Discussion

It appears that golfers can receive a healthy amount of vitamin D exposure during a round played in the winter months of the year. However, this is coupled with a large erythemally effective exposure experienced during the summer months for the same playing period between 3:00 pm and 5:30 pm. Playing golf in this time period is likely to be beneficial during the winter months in order to maximise potential UVD exposure while reducing the risk of excessive erythema exposure. Similarly, limiting playing times to the late afternoon (5:00 pm onwards) in summer is more likely to be beneficial to vitamin D health than exposures received during the middle parts of the day as this increases the risk of receiving a greater carcinogenic exposure and can have damaging effects on the photo-production of pre-vitamin D.

Exposures measured in this research showed a clear positive association between SZA and increased UVE exposure. Golfers playing during middle parts of the day are likely to be placed at greater risk for the development of non melanoma skin cancers than those risks quoted in this research which were calculated for casual weekly golfers playing between approximately 3:00 pm and 5:30 pm. Likewise, golfers located in lower latitudes are more likely to experience an increased non melanoma skin cancer risk. The greatest non melanoma skin cancer risk was determined in this research for upper back sites. This site is located in close proximity to the back of the golfer's neck. It is reasonable to expect that upturned collars will reduce the potential for skin cancers to develop to this region of the body and are one measure that could easily be implemented to reduce skin cancer risk on the course. The use of wide-brimmed hats, sunscreen and exposure avoidance where possible are also particularly important strategies that need to be implemented by golfers to further reduce skin cancer risk.

Cloudy or shaded conditions should not be seen as a potential protective barrier for golfers against the harmful effects of solar UVB as scattered and broken cloud cover close to the solar disk can further enhance and increase the diffuse UVB incident at ground level. However, during winter, exposure to a reasonable amount of diffuse UVB irradiance under shade during the mid to late hours of the afternoon could be beneficial for optimising total UVD exposure to golfers with only a minimal risk of attaining an excessive UVE exposure.

The distribution of UV exposure was measured to golfers at three body sites. It was determined that the neck site of a golfer is at greater risk of overexposure than both the vertex

and the forearm as the vertex is often protected by a hat or hair cover and the upper back site, being close to the position of the neck, receives a higher proportion of the ambient UV than the forearm. This may be due to standing over the ball during the golf swing. Sunscreen and shirt collar protection is therefore particularly important for the prevention of skin cancers at this body site for members of the golfing population. Contrasting this to exposures received during the winter months, exposures received to the back of the neck of golfers in southern Queensland are likely to be important for the production of vitamin D₃.

It was determined in this research that exposures to ambient UV during winter on the golf course between 3:00 pm and 5:30 pm could be beneficial for office workers for the production of vitamin D. Optimising weekly exposure periods to late afternoon in the winter months and taking adequate sun protection measures in the summer months are important strategies that golfers can utilise for long term preventative health.

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Tables

Table 1: Body site UVE exposure of golfers expressed relative to ambient UVE. Trials marked with an asterisk were measured during golf rounds which experienced cloud cover between 4 and 8 okta. Damaged dosimeters are marked n/a in the table. Fractions of ambient exposure were not calculated for 15 Feb, 29 Feb and 18 June due to spectroradiometer malfunction on those days. These days are also marked n/a.

Trial	Exposure period	SZA range (°)	Ambient UV (SED)	Golfer 1 (fraction of ambient UV)			Golfer 2 (fraction of ambient UV)		
				Vertex	Upper back	Forearm	Vertex	Upper back	Forearm
1 Feb*	15.00-17.30	41-74	8.5	0.62	0.38	0.19	n/a	0.33	0.17
8 Feb*	15.00-17.05	42-70	4.2	0.55	0.41	0.27	0.64	0.33	0.19
15 Feb	6.00-8.05	58-85	n/a	n/a	n/a	n/a	n/a	n/a	n/a
22 Feb	15.00-17.15	44-74	8.0	0.44	0.29	0.25	0.46	0.34	0.34
29 Feb*	15.00-17.00	46-72	n/a	n/a	n/a	n/a	n/a	n/a	n/a
14 Mar	15.00-17.15	49-79	2.9	1.04	0.86	0.45	1.23	0.74	0.74
9 May*	15.15-17.30	67-94	0.4	0.43	0.21	0.12	0.30	1.30	0.02
16 May*	15.00-17.15	66-92	0.6	0.82	0.44	0.46	0.55	0.53	0.35
13 Jun	15.25-17.25	72-95	0.2	1.14	0.82	0.33	1.12	0.60	0.51
18 Jun*	14.45-17.00	66-90	n/a	n/a	n/a	n/a	n/a	n/a	n/a
18 Jul	14.50-17.10	64-89	0.8	0.51	0.23	0.06	0.32	0.05	n/a
1 Aug	14.55-17.05	62-87	0.8	0.71	0.59	0.18	0.90	0.41	0.61

Table 2: Estimated non melanoma skin cancer risk for casual weekly golfers to the upper back and forearm sites relative to office workers. Annual estimates of indoor, weekend and leave exposure used in this calculation were substituted from the estimates of Vishvakarmen and Wong (25). UVE exposures received by the golfers were assumed to be received in addition to their daily indoor occupational exposure.

Body Site	Estimated Annual Exposure (SED)	Non melanoma skin cancer risk compared to office workers	
		BCC risk	SCC risk
Golfer forearm	817	1.11	1.15
Golfer upper back	839	1.16	1.22

List of Figures



Figure 1: Dosimeter placement sites for the study were located on the forearm, upper back and vertex. Green arrows highlight dosimeter placement locations.

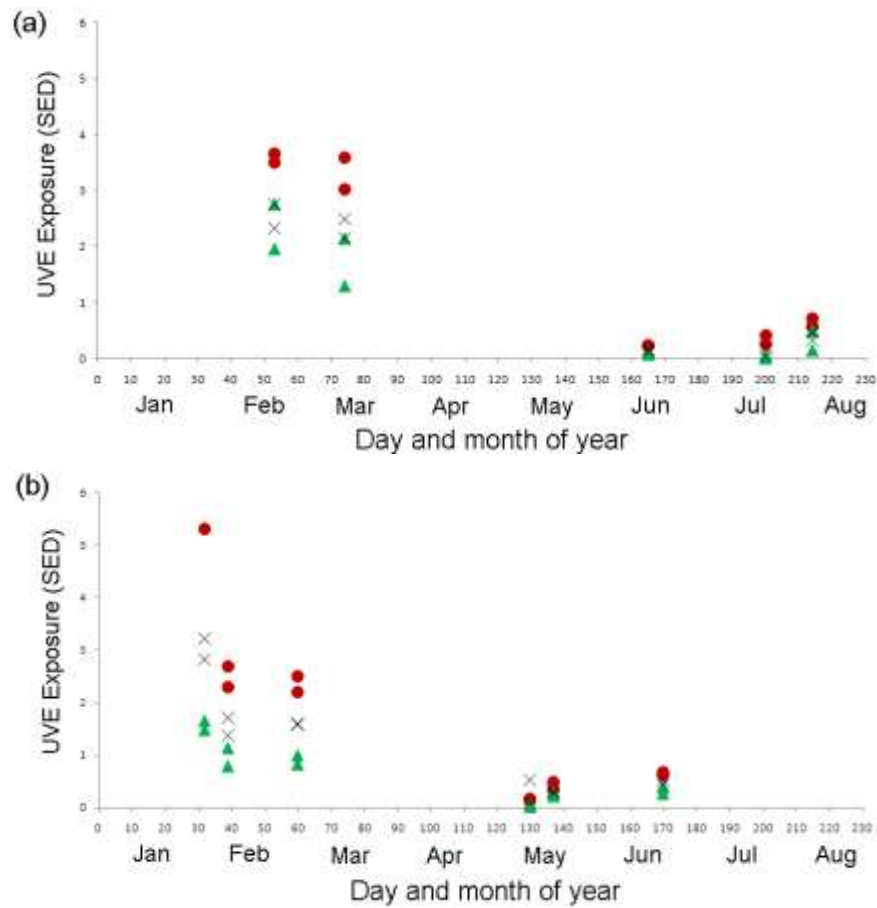


Figure 2: UVE exposure measured between February and August 2008 to forearm (triangles), upper back (crosses), and vertex (circles) body sites. UVE exposures presented in (a) were measured under low cloud cover conditions (< 4 okta). UVE exposures presented in (b) were measured on days which experienced cloud cover between 4 and 8 okta.

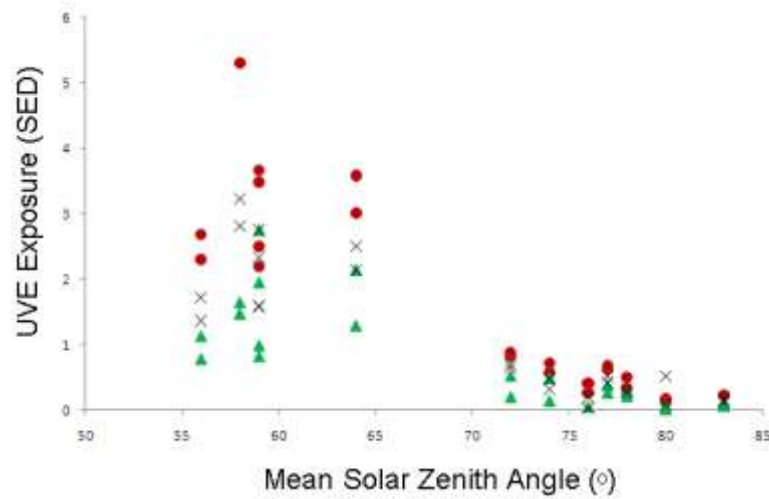


Figure 3: The dependence of forearm (triangles), upper back (crosses), and vertex (circles) UVE body site exposure of golfers on SZA. Rounds of golf played during winter, late afternoon and early morning experience a larger mean SZA, reducing UVE exposure by increasing the UV absorbing atmospheric path. Playing golf near midday would significantly increase the UVE exposure from the exposures measured in this research which were recorded during the early morning and late afternoon.

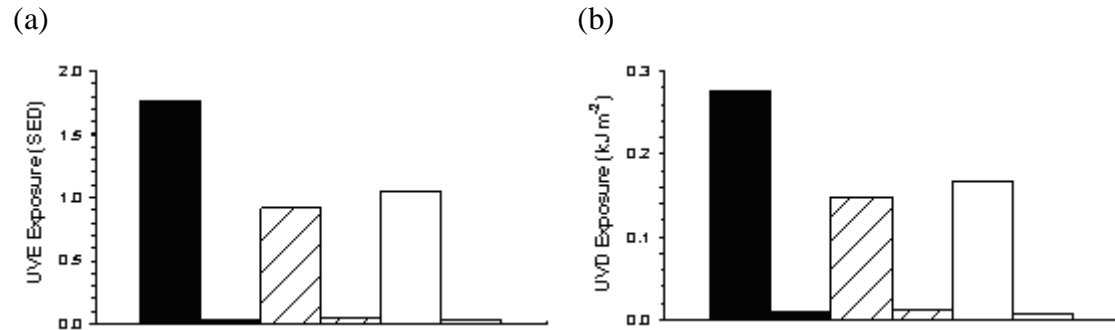


Figure 4: (a) Averaged UVE for each anatomical site received over the measurement campaign under all atmospheric conditions. (b) Averaged UVD for each anatomical site received over the measurement campaign under all atmospheric conditions. By proportion, UVD exposures were greater to the office workers compared to the golfers, highlighting the greater sensitivity of human skin to UVD compared with UVE. Vertex (dark bars), forearm (hatched bars) and upper back (light bars) exposures are shown for both golfers and office workers where the lower exposures were received by the officer workers.

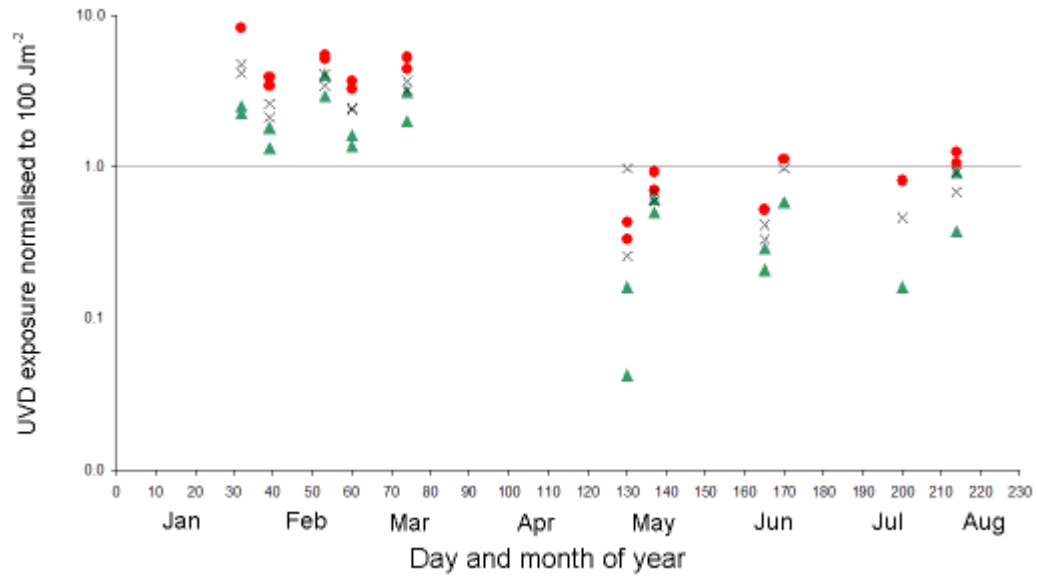


Figure 5: UVD exposures measured at forearm (triangles), upper back (crosses) and vertex sites (circles) in the February to August study period. Exposures are normalised to 100 Jm⁻² UVD.