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The distribution of biologically effective UV spectral irradiances received on a manikin face that cause erythema and skin cancer

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ABSTRACT

Solar ultraviolet (UV) radiation is a major cause of erythema and skin cancer in humans and the face is one of the highest risk sites. Biologically effective UV irradiation (UVBE) is wavelength-dependent, and risk assessment has been demonstrated based on the value of the received UV radiation. Therefore, this study measured the face skin exposure to UV spectral irradiance using a spectroradiometer and a head manikin, which were weighted by action spectra to calculate the UVBE that causes erythema ($UVBE_{ery}$), non-melanoma ($UVBE_{non\text{-}mel}$), human squamous cell cancer ($UVBE_{h\text{-}SCC}$), and DNA damage ($UVBE_{DNA\text{-}d}$). We determined that the biologically effective UVB and UVA irradiances on clear sky days had peak values at 65–73° SEA (8–9 UVI) and 55–68° SEA (6–7 UVI), respectively. In the 10–30° SEA range, the highly skin-damaging wavelengths were all observed at 300 nm. However, in the 30–60°, 60–81°, and 10–81° SEA ranges, the highly skin damaging wavelengths were 300 nm, 304 nm and 300 nm for $UVBE_{ery}$, respectively; 304 nm, 306 nm and 304 nm for $UVBE_{non\text{-}mel}$, respectively; all 305 nm for $UVBE_{h\text{-}SCC}$, and two small peaks at 302 nm and 312 nm for $UVBE_{DNA\text{-}d}$.

1. Introduction

Solar ultraviolet (UV) radiation is a major cause of erythema and skin cancer in humans, which is supported by sufficient experimental and epidemiological evidence [1–11]. Recently, the reduction of stratospheric ozone has led to elevated ultraviolet radiation levels at the Earth's surface [12]. A 10% reduction in ozone could lead to as much as a 15–20% increase in UV exposure depending on the biological process. Several studies have shown that the increase in the solar spectrum reaching the Earth's surface through the decrease in stratospheric ozone may increase the incidence of skin damage [13–21]. According to estimates provided by the "United Nations Environment Programmer" (UNEP), a decrease in stratospheric ozone of 10% each year will further increase the 304,500 cases of skin cancer that occur each year worldwide [22]. People should become more aware of the risk of skin exposure to solar UV radiation.

Epidemiological evidence that is relevant to the effects of UV on cancer risk in humans is derived primarily from studies of the effects of sun exposure and cancer risk. The most direct evidence of the carcinogenicity of UV radiation in humans should come, in principle, from observing the effects of personal exposure to sun. In practice, it is difficult to measure personal sun exposure accurately. Additionally, skin cancer occurs most frequently in the most highly exposed areas and correlates with the degree of outdoor exposure. The face is a high-risk area that is two to four times more sensitive than the limbs [23–25]. Accurately measuring the UV radiation that reaches the face is important for exposure assessment. A few studies have carried out using manikins to better simulate the face UV exposure for humans [26–30]. The benefit of using manikins was that it can reflect the effects of the surrounding anatomic sites to the monitored site. So we used a head manikin to accurately simulate the exposure of the face to UV radiation by the sun and assess the risk of face exposure to UV irradiance.

An action spectrum is a graph of the reciprocal of the radiant exposure required to produce a given effect at each wavelength. All the data in these curves are normalized to the datum at the most efficacious wavelength(s). By summing the biologically effective irradiance over the exposure period, the biologically effective radiant exposure ($J\,m^{-2}$ effective) can be calculated. Describing the relationship of exposure (dose) to skin damage (erythema and skin cancer) requires the availability of a biological hazard function or action spectrum for skin damage. The action spectrum gives the relative biological response of damage caused by UV radiation at different wavelengths; therefore, it is specific for a certain effect.

Erythema is an acute injury caused by UV radiation that appears up to 8 h after exposure to UV radiation. The relative effectiveness

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of the different wavelengths to induce erythema is expressed as an erythema action spectrum. The action spectrum adopted by the International Commission on Illumination (CIE) in human skin was proposed in 1987 and was based on the statistical analysis of the results of eight published studies of the determination of minimal erythema doses in groups of normal subjects. The erythema action spectrum is represented by relatively simple functions over three clearly defined spectral regions encompassing the wavelength interval from 250 to 400 nm. The most erythemogenic wavelengths are in the 250–290 nm range, and a decrease in effectiveness is observed as the wavelengths increase [31]. The erythemal (sunburn effective) action spectrum of the human skin weighted with the integral over the spectral UV irradiance on a horizontal plane in $W\,m^{-2}$ and multiplied by the constant $40\,W^{-1}\,m^2$ was used to calculate the Global Solar UV index (UVI) [31–33]. To promote solar UV protection, the UVI was developed as a tool to visualize the amount of harmful radiation and to encourage people to use sun protection [34].

Skin cancer is chronic skin damage caused by UV radiation. For UV induced skin cancer, the CIE 2006 has shown a standard which proposed the adoption of an action spectrum (weighting function) derived from experimental laboratory data [35–38] and modified to estimate the non-melanoma tumor response in human skin [39]. This action spectrum has a peak at 299 nm (at effectiveness 1) and constant value in the wavelength range 340–400 nm (at effectiveness 0.000394). In 1993, one research measured the wavelength dependency of skin cancer induced by UV in hairless mice and represented this in an action spectrum [40]. The action spectrum for humans have estimated in 1994 [37] by correcting for differences in epidermal transmission between mice and humans. The action spectra for UV-induced squamous cell cancer (SCC) have two peaks at 293 nm and 380 nm for hairless mice, and two peaks at 299 nm and 381 nm for humans. The valleys of the action spectra for UV-induced SCC were both at 352 nm for hairless mice and humans.

DNA damage caused by UV radiation is an initiator and key mediator in the development of skin cancers [41–47]. One significant UV-induced DNA lesion is the cyclobutyl pyrimidine dimer, formed between adjacent pyrimidines on the same DNA strand [48]. ENVIRONMENTAL HEALTH CRITERIA 160 (EHC160) has shown an action spectra for cyclobutane pyrimidine dimer formation in epidermal DNA to shown the relative biological response of DNA damage and this action spectrum was determined by one research in 1989 [48]. The peak of this action spectrum is near 300 nm and decreases rapidly at both longer and shorter wavelengths.

To accurately assess the risk of facial skin damage because of exposure to solar UV irradiance, the present study measured the facial skin exposure to UV spectral irradiance using a spectroradiometer and a head manikin, with regards to the characteristics of the facial anatomic structure. The measured UV spectral data were weighted by the erythema [31], non-melanoma [36], human-SCC [37] and pyrimidine photo-dimerization [48] action spectra to calculate the biologically effective UV irradiation (UVBE) that causes erythema ($UVBE_{ery}$), non-melanoma ($UVBE_{non\text{-}mel}$), human squamous cell cancer ($UVBE_{h\text{-}SCC}$), and DNA damage ($UVBE_{DNA\text{-}d}$), respectively. The diurnal variation of facial skin exposure to UVA and UVB waveband biologically effective irradiance at different SEAs and the biologically highly effective wavelengths for erythema, non-melanoma, human-SCC and DNA damage were determined in this study.

2. Materials and methods

2.1. Experimental materials

The experimental materials consisted of a head manikin, a middle shelf, and a turntable base that rotated at a constant speed from top to bottom. The total height of the manikin system was approximately 170 cm. The distance between the three anatomic measurement sites, including the cheek, nose, forehead and ground were approximately 155 cm, 155 cm and 165 cm, respectively. A computer and a computer-controlled fiber-optic (FO) spectrometer with two detectors were placed on the shelf to measure the UV spectral irradiance intensity (unit: $\mu W\,cm^{-2}\,nm^{-1}$). One detector was placed at the vertex of the manikin's head, and the other detector was placed on the anatomic measurement sites cheek (Fig. 1A), nose (Fig. 1B), forehead (Fig. 1C), where hold the plane tangent to the anatomic measurement sites at the most anterior point. For the actual anatomic measurement sites of cheek, the distance between cheek and the Lower eyelid of the head manikin is ~2 cm (Fig. 1D-L5) and the distance between cheek and the Nasal septum of the head manikin is ~4.5 cm (Fig. 1D-L6); For the actual anatomic measurement sites of nose was the tip of the nose; For the actual anatomic measurement sites of forehead, the distance between forehead and the vertex of the head manikin is ~5 cm (Fig. 1D-L1), the distance between forehead and the connection of two eyebrow ridges is ~2.5 cm (Fig. 1D-L2) and the distance between forehead and the right and left of the head manikin are ~7.5 cm (Fig. 1D-L3, L4). The detectors simultaneously recorded the UV radiation levels at the corresponding horizontal ambient and anatomic measurement sites.

2.2. Spectrometer and equipment calibration

The UV spectral irradiance was measured with a dual-channel miniature FO spectrometer (AvaSpec, 2048 × 14-2-USB2, Netherlands) with high UV sensitivity and high quantum efficiency. The design of the spectrometer is based on the AvaBench-75 symmetrical Czerny-Turner design with a 2048-pixel CCD detector array, making it particularly suitable for low-light, high-resolution applications. The full-width-at-half-maximum (FWHM) resolution of the spectrometer is 2.0 nm, and the resolution for stray light is less than 0.1%. The signal-to-noise ratio is 500 dB. The spectrometer has a slit size of 200 μm and a diffraction grating with 1200 lines per mm. The USB2 interface has ultrafast data sampling at 450 spectra s^{-1}, with data transfer at 2.24 ms. The two FO connectors can work synchronously. The detector has a cosine corrector (CC-UV/VIS) with an active area of 3.9 mm, which allows it to accept light from a 180° angle. The spectrometer was configured and radiometrically calibrated for absolute irradiance measurements over a range of 200–400 nm. Before the experiment, the fiber optic spectrometer was calibrated by the National Physical Laboratory GB (NPL).

2.3. UV radiation measurement and experimental methods

The measurements were performed in May in the town of Dou Men near Shao Xing city (30.09°N, 120.60°E, altitude 553 m), in the province of Zhejiang, China. The measurements were recorded on May 27 and 30, 2010 (summer) from 06:30 to 18:00 China Standard Time (CST) (solar noon at approximately 12:00 CST). The cheek and horizontal ambient irradiance measurements were performed on May 27, 2010. The nose, forehead and corresponding horizontal ambient irradiance measurements were performed on May 30, 2010. The mean air pollution index (API) of the measurement days in Shao Xing was approximately 62 for both days. The approximate midday maximum SEAs for the measurement days were both approximately 82°. The total column ozone amounts for May 27 and 30, 2010 were 276 Dobson Units (DU) and 326 DU, respectively (from National Aeronautics and Space Administration, NASA; http://ozoneaq.gsfc.nasa.gov/ozone_overhead_current_v8.md).

The monitoring location was on the roof of a hospital. In all cases, the roofs had unobstructed fields of view. The experimental