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Clothing as Solar Radiation Protection

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Clothing has the ability to protect the skin from incident solar radiation because the fabric from which it is made can reflect, absorb and scatter solar wavelengths. Fabrics differ in their ability to attenuate light in this way because they differ in fiber composition and moisture content, as well as in type and concentration of dye, optical whiteners, or UV-absorbing finishes adsorbed to fibers.

Each fabric must be tested to determine its ability to protect from solar radiation, as this cannot be known from visual observation nor calculated from descriptions of the fabric's composition and structure. The purposes of this chapter are therefore to (a) describe the types of solar radiation protection that can be provided to skin by fabric, (b) to describe the various test methods developed to assess each type of protection, and (c) to indicate possible areas of future research that would be fruitful, especially for photosensitive patients.

Photobiology of the Skin

Virtually all energy required to sustain life comes from the sun; without the sun, life as we know it would be impossible. Sunlight is a form of electromagnetic radiation in the form of ultraviolet (UV), visible, and infrared radiation. As shown in figure 1, most of the emission is in the visible portion of the spectrum (400–700 nm), and roughly 7% of total solar emission is UV between 290 and 400 nm (recall that 1 nm = 10⁻⁹ m).

However, too much sun is not a good thing. Acute or chronic overexposure to sunlight can lead to a variety of deleterious effects. In normal individuals, such acute effects include tanning and erythema (sunburn). Chronic overexposure can lead to connective-tissue damage ('photoaging'), premalignant

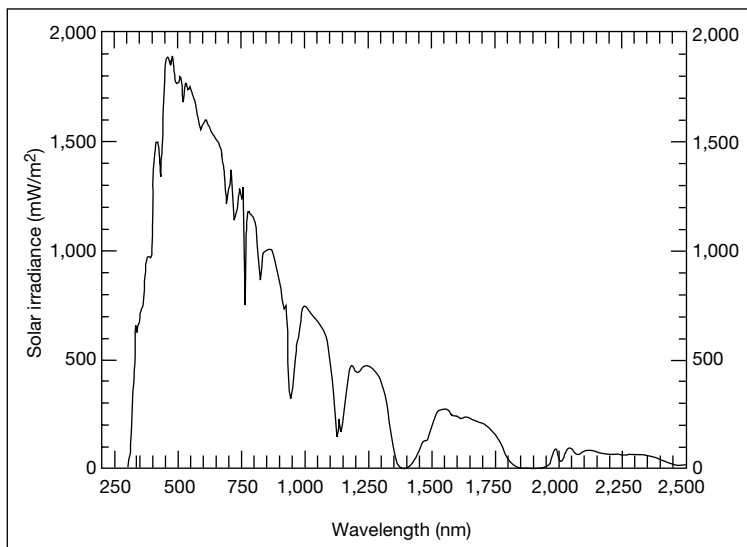


Fig. 1. CIE standard reference spectrum of sunlight. We thank Dr. R.M. Sayre for providing this spectrum.

lesions (e.g. actinic keratosis) and malignancies (basal – or squamous – cell cancer, and perhaps melanoma skin cancer).

For all of these deleterious effects, UV radiation plays a major etiologic role. The UV region constitutes roughly 7% of total solar emission reaching the earth. This region is usually divided into three parts: UVC, UVB and UVA. UVC (<290 nm) is normally not transmitted to the earth because it is absorbed by the stratospheric ozone layer. In contrast, UVB rays (290–320 nm) are only partially absorbed by ozone; a major fraction reaches us especially during the hours close to solar noon, during the summer months, at lower latitudes, and at higher altitudes. Although UVB is a relatively small fraction of total UV radiation (~10% of total UV) it is the major cause of erythema (sunburn), suntanning, photocarcinogenesis and ‘photoaging’. UVA wavelengths (320–400 nm) can also cause erythema and tanning. However, it takes roughly 1,000 times the doses of UVA to produce sunburn and tanning effects equivalent to those of UVB.

There are other deleterious effects of sunlight that may result in photosensitivity disorders. These do not ordinarily take place under normal conditions. Some of these may be due to endogenous factors that cause abnormal phototoxic or photoimmunologic reactions to sunlight [1]. Examples of these are polymorphic light eruption (PMLE), solar urticaria, chronic actinic

dermatitis (CAD) or the porphyrias. These present as a wide variety of clinical skin responses.

Other photosensitivity disorders may occur as a result of exogenous factors, factors that are inadvertently introduced when a person has applied a topical medication to his/her skin or who has taken a medication [2]. These medications may cause the skin to elicit a phototoxic or photoallergic response each time the sensitized skin is exposed to solar radiation. An ever-increasing number of drugs and other agents have the ability to induce photosensitivity disorders. Sometimes, inadvertent phototoxicity may occur inadvertently from therapeutic regimens involving photosensitizers (e.g. photochemotherapy or photodynamic therapy).

The photosensitivity responses can, in general, be easily activated by wavelengths that are longer than the wavelengths that cause sunburning, sun-tanning, 'photoaging' and the development of (pre)malignant lesions, all of which are activated predominantly by UVB wavelengths. Exposure to UVA is a major factor in causing people with drug-photosensitized skin and with endogenous photosensitivity conditions to have adverse skin reactions. Exposure to visible rays may be important in some cases of solar urticaria and in the porphyrias.

All of these types of photodamage involve absorption of UV or visible radiation by a key molecule (chromophore), which is ultimately responsible for the elicitation of the observed photobiological effect (e.g. sunburn). Simply put, if no light is absorbed, there is no photochemical or photobiological effect. Therefore the absorption spectrum, which is a wavelength-by-wavelength measure of the probability that light will be absorbed by a given chromophore, is what ultimately determines the wavelength dependence of that event. The structure of the absorbing molecule determines which wavelengths are absorbed, and the intensity of the transition (i.e. the value of the molar extinction coefficient at each wavelength). The ability of light of a given wavelength to 'enact' a given photobiological effect (e.g. sunburn) is proportional to its absorption at that wavelength. The wavelength-dependent 'enactment' of the effect over all relevant wavelengths *in vivo* is referred to as the *action spectrum*. Under 'simple' conditions, the action spectrum is a replica of chromophore absorption spectrum. In skin, nonspecific absorption and scattering of incident radiation by other species alters the spectral distribution of light reaching the chromophore. Thus, the 'shape' of the experimentally measured action spectrum (i.e. the relative 'weight' that each wavelength would have in producing an effect) is generally a significantly distorted version of the chromophore absorption spectrum. Knowledge of the action spectrum for any photosensitivity condition is important from the standpoint of photoprotection, since by definition, this is what delineates the wavelengths that the photosensitive individual must avoid.

Action spectra for erythema [3–5], delayed tanning [3], non-melanoma skin cancer [6], melanoma [7, 8] and elastosis [9] have been published. All of these indicate that UVB wavelengths play a central role in eliciting these responses, with UVA wavelengths usually being 3–4 orders of magnitude less effective (see Setlow and Woodhead [8] however for malignant melanomas). These results are summarized in table 1. Readers interested in additional details are urged to read the original references.

Table 2 summarizes the wavelength ranges for the various photosensitivity disorders. In general, action spectra for these conditions are not as well characterized as are those for the ‘normal’ responses. The term ‘drug photosensitivity’ has been expanded to include chemicals found in household items, cosmetics, manufacturing, agricultural, and recreational activities [2]. Accordingly, the active wavelengths for these responses will vary with the absorption spectrum of the photosensitizer. Often, there is not a unique spectrum for what is nominally referred to as a ‘single’ photosensitivity condition (e.g. PMLE or solar urticaria). In these cases, few narrow-band action spectra are extant, so that the ‘action spectrum’ in this case does not so much represent a structured band as a wavelength region where the responses are preferentially elicited.

One common feature that does emerge from comparison of tables 1 and 2, however, is the relatively minor role of UVB and the predominance of UVA and/or visible wavelengths in elicitation of photosensitivity conditions. This is in distinct contrast to the action spectra for ‘normal’ responses. This is of much more than academic interest. As we will see, the vast majority of sunscreen and fabric testing uses erythema as the endpoint. This test is insensitive to wavelengths most effective in eliciting photosensitivity responses from susceptible individuals.

Protection against Sunburning

In this section, test methods for determining the ability of fabrics to protect against sunburning are described and compared. Factors influencing protection values are discussed.

Test Approaches

The general approaches to testing fabrics for the ability to prevent sunburning are laboratory testing in vivo and instrumental evaluation in vitro. The quantitative measure of an in vivo determination is the sun protection factor (SPF), and that used to indicate the result obtained instrumentally is the ultra-violet protection factor (UPF).

Table 1. Wavelength dependence of ‘normal’ deleterious responses to sunlight

Response	Wavelengths, nm
Sunburn	290–320 (major), 320–400 (minor) [3–5]
Melanogenesis (delayed tanning)	290–320 (major), 320–400 (minor) [3]
Non-melanoma skin cancer	290–320 (major), ~370 (minor) [6]
Melanoma	UVB (?) [7], UVA (?) [8]
Elastosis	290–320 (major), 320–400 (minor) [9]

‘Minor’ effect 3–4 orders of magnitude less than ‘major’ effect.

Table 2. Wavelength dependence of photosensitivity responses to sunlight

Response	Wavelengths, nm
Drug photosensitivity	Variable, depending on the absorption spectrum of the drug; 320–380 (most), visible (some) [2]
Photosensitivity disorders	UVB (CAD), variable UVA (depending on the sensitizer), visible (some solar urticaria; PMLE) [2]
Porphyrias	UV and visible wavelengths (400–650) [10]
Sensitized photo(chemo)-therapy: PUVA, PDT	(320–380) PUVA [10], (400–700) PDT [11]

In vivo Tests

The *in vivo* tests for the ability of fabric to protect against sunburn are generally a modification of existing methodology given in the Federal Register for sunscreens [12, 13]. Test sites are delineated on the lower backs of at least 10 volunteers. The minimum erythemal dose (MED) from the collimated output of a filtered xenon arc solar simulator is first determined on one set of sites in the absence of fabric. Next, a fabric specimen is placed taut between the solar simulator and the subject’s dorsal surface. A series of seven exposures, centered at the estimated SPF of the given fabric (as estimated from *in vitro* UPF values; see below), are given as per the Final Rule of the current FDA monograph [13]. The incremental series of three exposures are above and the decremental series are below the estimated SPF. For target SPF <8, the intervals are 10% above and below the target SPF; for target SPF between 8 and 15, they are 9%, and for SPF >15, they are 7%. The dose that results in a minimal erythema extending to the borders of irradiation is then used to determine the SPF of the fabric as follows:

$$\text{SPF (in vivo)} = \frac{\text{MED (through test fabric)}}{\text{MED (in the absence of test fabric)}} \quad (1)$$

Since erythema is the explicit measured endpoint in this method, the results of this test only relate to the ability of the fabric to protect against sunburn. The higher the SPF value, the better the fabric's ability to protect against sunburn. The SPF value tells a person how much longer he/she can remain in the sun wearing the fabric to receive the same dose of erythemogenic UV on the fabric-protected portion of the skin as on unprotected skin. Claims cannot be made that the fabric protects the skin in any other way.

Instrumental (in vitro) Methods

Instrumental methods for determining the ability of fabric to protect the skin from sunburning rely on using an instrument to determine the amount of transmittance of each wavelength of UVA and UVB radiation that passes through the fabric. Although the transmittance is measured instrumentally, the action spectrum used to calculate UPF [5] was determined on human subjects.

To evaluate in vitro UPF fabric protection, three wavelength-dependent measurements are necessary: (1) fabric transmission, (2) source spectral distribution, and (3) the action spectrum for erythema (sunburn).

Instruments for measuring fabric transmission include broad-band radiometers, spectroradiometers, or spectrophotometers. The advantages and disadvantages of using each type of instrument are excellently reviewed by Gies et al. [14]. Spectrophotometers with integrating spheres are usually the method of choice for measurement of fabric transmission.

The solar spectral distribution is usually representative of a 'noonday' solar spectrum. Often, a reference spectrum is used, although investigators may decide to measure the local solar spectrum under ambient conditions. Transmittance values, in general, depend on the portion of the solar spectrum used.

The erythemal action spectrum is not ordinarily measured by each individual investigator, rather, the established Commission Internationale d'Eclairage (CIE) erythema (hazard) spectrum is used [5].

To begin the UPF calculations, risk estimates for unprotected skin are obtained by multiplying the solar spectral source with the CIE erythema action spectrum at each wavelength interval, $\Delta\lambda$, and summing the response over all relevant wavelengths. In the spectrometer, transmittance data are normally collected at 2- or 5-nm intervals, usually between 290 and 400 nm.

$$\text{Risk}_{\text{unprotected}} = \sum S(\lambda) \cdot A(\lambda) \cdot \Delta\lambda \quad (2)$$

where $S(\lambda)$ is the source spectrum in $\text{W} \cdot \text{m}^2 \cdot \text{nm}^{-1}$, $A(\lambda)$ is the action spectrum for the measured response (dimensionless) and $\Delta\lambda$ is the bandwidth in nm, determined by the experimental conditions of the measurement. Risk estimates for fabric-protected skin are obtained by multiplying the risk at each

wavelength by the transmittance of the fabric at each wavelength, $T(\lambda)$, and summing over the same wavelengths:

$$\text{Risk}_{\text{protected}} = \Sigma \cdot S(\lambda) \cdot A(\lambda) \cdot T(\lambda) \cdot \Delta\lambda \quad (3)$$

Then, risk estimates for fabric-protected skin are obtained by multiplying the risk at each wavelength by the transmittance through the fabric at each wavelength, $T(\lambda)$, and summing over the same wavelengths:

Finally UPF is calculated as:

$$\text{UPF} = \text{Risk}_{\text{unprotected}} / \text{Risk}_{\text{protected}} = \frac{\Sigma \cdot S(\lambda) \cdot A(\lambda) \cdot \Delta\lambda}{\Sigma \cdot S(\lambda) \cdot A(\lambda) \cdot T(\lambda) \cdot \Delta\lambda} \quad (4)$$

Clearly, the calculation of UPF depends not only on the spectral distribution used, and the scanning interval used to obtain the transmittance values, but also on the choice of action spectrum.

UPF values indicate the ability of fabric to protect the skin it covers from sunburning. The wavelengths incident on the fabric during the test are limited to those in the UVA and UVB ranges (there is no visible or infrared). An erythema action spectra is used to weight the transmittance data. The higher the UPF of the fabric, the better is its ability to protect the skin lying under it. UPF indicates how much longer a person can stay in the sun with the fabric covering skin, as compared with uncovered skin to obtain same erythema response.

Because fabric transmission, in general, varies significantly with wavelength over the UV and visible spectra, knowledge about UPF is not a good indicator of the ability of a fabric to protect skin that is photosensitive to wavelengths outside of the UV range. Likewise, UPF, calculated using the McKinlay-Diffey erythema action spectrum, is not a good indicator of the ability of the fabric to protect skin from photosensitive individuals because the appropriate action spectrum for photosensitivity has not been used in this calculation.

Comparison of SPF vs. UPF Values

Theoretically, the UPF and SPF values for any fabric should be the same, given the same incident spectral distribution on the fabric specimen. However, this has not proven to be the case.

Gies et al. [15] reported very good agreement between in vitro UPF and in vivo SPF for 16 fabrics with protection factors ranging between 10 and 200 (SPF > 50 was scored as '50+'). These workers reported only a single misclassification of protection category. On the other hand, Menzies et al. [16] reported that in 5 out of 6 cases in vivo SPF was less than in vitro UPF if the human testing was 'on skin'. Better agreement between SPF and UPF was

obtained when the human testing was performed 2 mm ‘off skin’ (8 mm in one case). Greenoak and Pailthorpe [17] measured 22 fabrics, and in 21 cases obtained a SPF/UPF ratio significantly less than unity.

Several studies have yielded some insight into the reasons for the observed disparities. Menzies et al. [16] found, in contrast to his results on the six tested fabrics, that UPF values obtained from standardized neutral density thin film meshes were in good agreement with the in vivo SPF values. They attributed the lack of agreement between fabric UPF and SPF values to nonuniformity of fabric transmission (‘hole effect’).

Ravishankar and Diffey [18] found that the fabric SPF was anatomic-site dependent, and was consistently higher than the corresponding UPF measured by standard methodology. They attributed this result to the observation that standard UPF testing is done with a collimated light source held normal to the fabric; when the lamp is rotated in an arc around the sensor, UPF increases with increasing angle of incidence from the normal. This is due to the longer path length that the light must travel through the fabric. Ravishankar and Diffey [18] argue that in practice, this situation is closer to what would happen in the ‘real world’ since the sun is a diffuse source. This latter view is supported by the work of Moehrle and Garbe [19]. Using the sun in conjunction with cosine-corrected dosimeters attached to the exposure site, they found that SPF values were higher than those obtained from companion measurements using conventional collimated sources.

Clearly, this is a complicated area with many opposing experimental parameters. Much more systematic study is needed to separate the individual effects of each parameter.

Standardized Test Methods

Currently there are standard test methods for Australia/New Zealand (AS/NZS 4399 [20]), the USA (AATCC TM 183 [21]) and Great Britain (BS 7914 [22]), respectively. All of these describe the procedures for determining the transmittance of UV radiation through fabric and describe how the UPF of the fabric tested is to be calculated. While all calculations are based on the formulas given above, the individual standard methods differ from each other in regard to scanning intervals, positioning of the fabric specimens in the instruments, and in other details that can affect the transmittance values and calculated UPF of the fabric. In addition, the erythral action spectrum designated is not the same for each standard; it is either based on Albuquerque NM spectral distribution or on Melbourne Australia spectral distribution.

While standard-setting organizations realize that there are many factors that influence the transmittance measurements, these have not yet been

included in the standards. Two major factors are fabric moisture content and tension state. It is known that the UV transmittance of a wetted fabric specimen can be significantly lower than that of that same specimen when 'dry' (i.e. at moisture regain water content). Cotton fabrics and other cellulosic fabrics that can absorb large amounts of moisture have the greatest differences in wet (saturated) and dry UPF values and polyester and manufactured synthetic-fiber fabrics have far lower differences between wet and dry UPF values. Currently there is no standardized procedure for ensuring uniform moisture content of the fabric presented to the instrument. It is expected that such procedures will be forthcoming, as some standard-setting organizations would like to determine the lowest UPF of the fabric.

The tension state of the fabric specimen can also dramatically affect the transmittance of UV through the fabric and therefore the UPF value calculated from that data. 'Tension state' refers to whether the fabric is in a stretched or relaxed state at the time a transmittance measurement is taken. Fabrics that have the greatest potential difference between UPFs measured while they are not at all tensioned (stretched) and at full tension are those called 'stretch fabrics' in the marketplace. Many of these contain a fiber called 'spandex' (elastane). Minor changes in tension state have a dramatic influence on UV transmittance through the fabric. Tensioning tends to create larger 'holes' in the fabric, through which there is no material to either reflect or absorb UV radiation incident on the fabric surface.

There is also interest in defining the 'use' state of the fabric when it is submitted for UV transmittance testing. These interests arise primarily with labeling concerns. There is interest in labeling of UV-protective textiles with a UPF value that reflects the lowest protection the fabric will provide during its useful life. Normal wear and laundering of fabric, its exposure to UV radiation and/or chlorinated water during use may decrease the ability of the fabric to protect. ASTM has a standardized practice (ASTM D 6544 [23]) that specifies that fabric must be laundered 50 times, exposed to 100 AATCC fading units of simulated sunlight. This is roughly equivalent to the amount of sunlight during a 2-year period. If the fabric is intended for swimwear, it must be exposed to chlorinated pool water prior to UV transmittance testing.

The myriad of factors that may significantly influence the observed UPF value of a given fabric in a complicated way reinforces the enormity of the task confronting any organization that seeks to set standards of fabric protection. The question is always begged as to the 'right' or 'most realistic' criteria for assessing UV fabric protection to consumers. Although not a simple problem, legal, medical and commercial concerns for proper labeling continue to push for a 'best solution'.

Protection Against Other Photobiological Endpoints

Dermatologists have noted that fabric protects the skin from sunlight effects other than sunburn. Bech-Thomsen et al. [24] for example concluded after observing a single patient with xeroderma pigmentosum (XP) that there is a direct relation between UV transmission of clothes and the appearance of skin tumors. There was a marked difference in the patient's skin condition after she was instructed to wear leather or denim shirts that transmit little UV. Further, after UVA-blocking film was applied to the windows of the patient's house and car, clinical manifestations of XP decreased. O'Quinn and Wagner [25] studied a patient who worked outdoors and who always wore cowboy shirts. These shirts have a double layer of fabric in the yoke and a single layer in the body of the shirt. O'Quinn and Wagner observed a marked reduction in the number of skin cancers in the areas of that patient's skin covered by the yoke as compared with that covered by the body of the shirt.

UV-Induced Skin Cancer

Menter et al. [26] compared the ability of a fabric with SPF >30 with a fabric with SPF 6.5 to prevent protected skin from producing skin tumors under solar radiation conditions. Using a rapid tumor induction technique [26], they exposed Sk-1 hairless albino mice whose skin was protected by the test fabrics to solar-simulating UV irradiation at a dose regimen 7-fold higher than that used to produce squamous cell cancer (SCC) in unprotected hairless mice. Under the latter conditions, the fabric with SPF >30 completely protected against (pre)malignant lesions, whereas squamous cell carcinoma was produced in mice protected with 6.5 SPF fabric to the same extent as in unprotected (positive) control mice. This study demonstrated that wearing clothing per se does not necessarily guarantee protection against skin cancer. Although SPF values are not exact indicators of skin cancer protection, it is apparent that 'high SPF' fabrics provide better protection than do 'low SPF' fabrics. Because of the high SPF of the latter fabric, there was no attempt to quantitatively evaluate protection against solar-induced tumors.

UV-induced DNA damage elicits an overexpression of p53 protein. Berne et al. [27] suggested that this could be used as a short-term 'surrogate endpoint' that would be suitable for assessing sunscreen and fabric protection against skin cancer in humans. These workers studied the effect on p53 expression of a topical sunscreen and of a blue denim fabric in chronically-exposed human skin after sun exposure during the summer. In one group, 7 individuals spent 5- to 9-week summer holidays at various resort areas. Every morning, they applied the SPF 15 sunscreen on a defined area on one dorsal forearm. A second group of 11 individuals on similar 5- to 10-week summer holidays kept one forearm

constantly covered with a blue denim fabric (SPF 1700) attached with an occlusive dressing (SPF 20). In both groups, the other forearm served as untreated control. Biopsies from protected and unprotected sites from both groups were stained with an immunostain that recognizes both wild-type and mutated forms of p53 protein. The number of immunopositive keratinocytes was significantly lower in those skin areas protected by either sunscreen or denim fabric than in the unprotected skin areas. There appeared to be different degrees of sun protection for the sunscreen, occlusive dressing, and the denim fabric. The results suggested that UV protection against p53 expression reaches a 'plateau' at high SPF, after which additional shielding offers only marginal protection.

Blocking of Vitamin D Photosynthesis

During exposure to sunlight, epidermal 7-dehydrocholesterol (7-DHC) undergoes photolysis to pre-vitamin D₃ in a reaction activated by UVB radiation. Pre-vitamin D₃ forms vitamin D₃ in a thermal process. Matsuoka et al. [28] measured circulating concentrations of vitamin D₃ as a measure of UVB exposure in human subjects. Whole body exposure at suberythemal doses produced significant elevation in serum vitamin D₃ in unclothed subjects. This could be attenuated or prevented altogether by seasonal clothing. Some concern was expressed that prolonged extensive fabric coverage, as takes place in cultures in which covering is maximal, could result in vitamin D deficiency, and this was taken as a possible deleterious consequence of fabric protection. Therefore, a reasonable amount of care ought to be taken when dealing with individuals who, for some reason, must rigorously avoid sun exposure.

Phototoxicity following Photodynamic Therapy (PDT)

Administration of 5-aminolevulinic acid (ALA), a normal metabolite in porphyrin metabolism, leads to production of the potent photosensitizer protoporphyrin IX. Protoporphyrin IX preferentially localizes in tumor tissue; subsequent irradiation with visible light is toxic to the tissue. Therefore, ALA-induced photosensitization is often used in PDT. Menter et al. [29] tested two fabrics for their ability to protect skin sensitized by topical administration of ALA onto tape-stripped skin in hairless mice. In this study, the same two fabrics used in their previous carcinogenesis study [26] were used.

The action spectrum for ALA sensitization lies throughout the visible range [11, 30, 31]. The response to ALA photosensitization may therefore be looked on as an indicator of virtually all visible wavelengths reaching the skin. To this end, Menter et al. [28] used tungsten-halogen source that emitted a continuous visible spectrum from 380 to 800 nm. The minimum phototoxic dose (MPD) was determined on one set of hairless mice in the absence of fabric. Multiples of the MPD were then given to mice through each fabric. In the case

of the SPF >30 fabric, an upper limit for the protection factor was estimated to be 25–30. The protection afforded by the SPF 6.5 fabric against visible wavelengths was <2.5. The latter differences can be explained from the transmission properties of the fabrics. The SPF 6.5 fabric was treated with a UV-absorbing coating. This gave the fabric added protection against erythemogenic wavelengths, but not visible wavelengths, where the PDT response was active. The SPF >30 fabric had better blocking of visible as well as UV wavelengths, but even here, protection was somewhat better in the UV. This underscores the salient conclusion that fabrics do not, in general, act merely as neutral scatterers of impinging radiation.

Summary and Conclusions

The sun is essential for life. Yet, sunlight can also be a source of such deleterious effects as sunburn, and suntanning, as well as premalignant and malignant lesions. These may all occur in individuals with normal responses to sunlight. In addition, there exist a variety of ‘abnormal’ photosensitivity responses to sunlight that may result from either endogenous imbalances (e.g. the porphyrias) or from added exogenous factors (e.g. drug photosensitivity). The ‘normal’ responses to sunlight, by and large, are produced preferentially by UVB (290–320 nm), with minor contribution by UVA (320–400 nm) wavelengths. In contrast, the ‘abnormal’ photosensitivity responses are, for the most part, elicited predominantly by long UVA and, in some cases, visible light.

In the last 20 years or so, considerable attention has been paid to the use of fabrics as photoprotective materials. The vast majority of work in this area has been concerned with fabric protection against sunburn. In addition to *in vivo* measurement of fabric SPF, *in vitro* evaluation of fabric UPF has been carried out in numerous laboratories around the world. The UPF is estimated from the wavelength-dependent transmission of the fabric, the solar UV spectrum and the erythral action spectrum over the wavelength region 290–400 nm. Depending on the fabric, UPF values range from 2 to several thousand. More recently, it has become clear that such environmental influences as laundering, solarization, humidity, wetting and degree of stretching may play a major role in fabric protection. Protection also may be altered by addition of dyes, UV absorbers and fluorescent whitening agents.

To date, there have been relatively few studies of fabric protection for endpoints other than sunburn erythema. Yet, many fabrics that provide good protection against sunburn may provide inadequate protection against photosensitization by intrinsic or extrinsic absorbing molecules or against (pre)malignant lesions. Future work should explicitly address the efficacy of

protective fabrics against photosensitivity diseases that are activated by long UVA or visible wavelengths.

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