As far as lichenoid tissue reactions are concerned, the phenomenon of transepidermal elimination has only occasionally been reported. In 1981, Bardach (7) published a case of perforating lichen nitidus in a 8-year-old boy. Hanau & Sengel (8) described the first case of perforating lichen planus in 1984. In both reports the extruded material seemed to be formed by hyaline bodies, as in our case.

The clinicopathologic findings in the present case suggest that clusters of necrotic keratinocytes are pushed up during the epidermal remodelling, following the acute phase of lichenoid reaction, until they are eliminated. The extrusion of necrotic material may be a mechanism of healing of lichenoid eruptions. In view of this report we believe that LS should be added to the list of dermatoses in which transepidermal elimination has been demonstrated.

REFERENCES

1. Muller SA, Winkelmann RK. Lichen striatus. Clinical and histologic study with emphasis on vesicular aspects. JAMA 1963; 183: 206–208.
2. Stewart WM, Lauret PH, Pietrini P, Thomine E. Lichen striatus. Critères histologiques. Ann Dermatol Venereol 1977; 104: 132–135.
3. Charles CR, Johnson BL, Robinson TA. Lichen striatus. A clinical, histologic and electron microscopic study of an unusual case. J Cutan Pathol 1974; 1: 265–274.
4. Staricco R. Lichen striatus. Arch Dermatol 1959; 79: 311–324.
5. Woo TY, Rasmussen JE. Disorders of transepidermal elimination. Part 1. Int J Dermatol 1985; 24: 267–279.
6. Woo TY, Rasmussen JE. Op. cit. Part 2. Int J Dermatol 1985; 24: 337–348.
7. Bardach H. Perforating lichen nitidus. J Cutan Pathol 1981; 8: 111–116.
8. Hanau D, Sengel D. Perforating lichen planus. J Cutan Pathol 1984; 11: 176–178.

## Polymorphous Light Eruption: Eliciting and Inhibiting Wavelengths

BERNHARD PRZYBILLA, ATTILA GALOSI, MARLIES HEPPELER, THOMAS RUZICKA and JOHANNES RING

*Dermatologische Klinik und Poliklinik of the Ludwig-Maximilians-University, Munich, Federal Republic of Germany*

Przybilla B, Galosi A, Heppeler M, Ruzicka T, Ring J. Polymorphous light eruption: Eliciting and inhibiting wavelengths. Acta Derm Venereol (Stockh) 1988; 68: 173–176.

In 38 patients with polymorphous light eruption (PLE) photoprovocation tests were performed by applying 100 J/cm$^2$ of UV-A to the extensor side of the upper arm on three consecutive days. Test sites were divided into four areas by covering the patients' skin with Schott glass filters WG 320, WG 335, WG 360, and GG 385 (or GG 395). In 17 patients typical skin lesions could be provoked in at least one test field. 10 patients reacted either at all test sites or at shorter wavelengths (simultaneously higher doses) only. In the remaining 7 patients test reactions did not occur underneath filters with lower cut-off points, whereas skin lesions were induced by radiation deprived of the shorter wavelengths passing through these filters. This observation suggests an inhibitory effect of shorter wavelengths in these cases. (Received June 25, 1987.)

B. Przybilla, Dermatologische Klinik und Poliklinik der Ludwig-Maximilians-Universität, Frauenlobstr. 9–11, D-8000 München 2, FRG.

Polymorphous light eruption (PLE) is a common skin disease affecting up to 10% of the population (1). Characteristically, skin lesions develop after intensive irradiation in skin

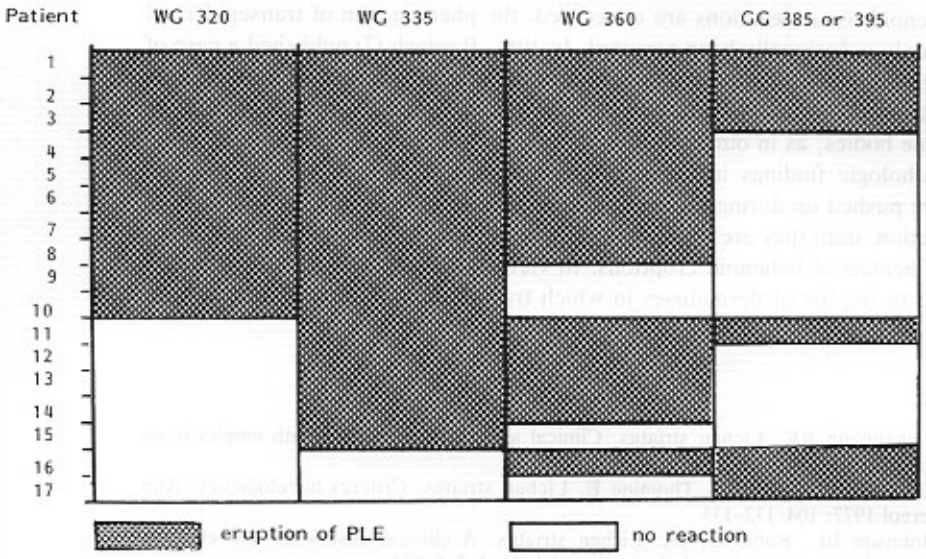


*Fig. 1.* Photoprovocation (3×100 J/cm$^2$ UVA unfiltered initial dose) in 17 patients with polymorphic light eruption: pattern of skin reactions beneath various filters.

areas which have been protected from UV exposure for a prolonged period of time (2). Under laboratory conditions elicitation of skin lesions of PLE can be achieved in 50 to 90% of the cases (2, 3). There are somewhat contradictory results regarding the action spectrum of PLE: elicitation by UVB, UVA, and longer wavelength regions of the spectrum has been reported (for review see 2). From recent investigations it can be concluded that in the majority of patients the action spectrum lies within the UVA region with a few individuals reacting to UVB exposure (4, 5). We present here preliminary data on the action spectrum of PLE within the UVA region which indicate an inhibitory effect of shorter wavelengths in some patients.

## PATIENTS AND METHODS

A total of 38 patients presenting with a history of PLE were investigated. Photoprovocation tests were performed by applying 100 J/cm$^2$ UVA to the extensor side of the upper arm on three consecutive days using a radiation source free of UVB (UVASUN 5000) (6). Irradiance was 53 mW/cm$^2$ at a source-to-skin distance of 40 cm. The test site was divided into four fields measuring 5×5 cm each by applying to the patients' skin the four Schott glass filters WG 320, WG 335, WG 360, and GG 385 (or GG 395 in some patients). At the thickness used, these filters transmitted 50% of radiation at 320 nm, 335 nm, 360 nm, and 380 nm (or 390 nm), respectively, while wavelengths shorter than 280 nm, 300 nm, 320 nm, and 340 nm (350 nm) were virtually cut off. Irradiance measured beneath these filters was 94.0%, 93.7%, 80.0%, 31.3%, or 16.4% of the initial intensity.

## RESULTS

In 17 of the 38 patients typical skin lesions were provoked in at least one test area (Fig. 1). Three patients exhibited positive reactions in all test fields. There were 7 patients who reacted only to higher doses as well as shorter wavelengths, while failing to exhibit positive results at the sites covered with GG 385 (GG 395) (5 patients) or GG 385 (or GG 395) and WG 360 (2 patients). In the remaining 7 patients test reactions did not occur

beneath WG 320 (5 patients), WG 320 and WG 335 (1 patient), or WG 320, WG 335, and WG 360 (1 patient), whereas skin lesions were induced by irradiation deprived of the shorter wavelength regions passing through these filters.

## DISCUSSION

Successful experimental elicitation of PLE depends on the use of the appropriate wavelengths. According to our results not only UVB- or UVA-provoked conditions have to be differentiated, but an even more complex spectrum interaction within the UVA region has to be considered for a subset of patients. Shorter wavelengths passing through the filter WG 320 (in some cases also WG 335 and WG 360) seem to inhibit reactions found beneath filters cutting off radiation at longer wavelengths. Another explanation might be that this effect is due to differences in the UV doses applied. However, the amount of radiation reaching the skin surface beneath the filter WG 320 was only 0.9 J/cm$^2$ higher than beneath the filter WG 335. Although an inhibitory effect of higher doses cannot be excluded, this seems rather unlikely. Therefore, this effect is most probably attributable to interactions of different wavelength regions. It was remarkable that in most of the patients with PLE inhibited by shorter wavelengths a slight increase of 0.3% of the total unfiltered UV dose was sufficient to achieve this effect. One has to assume that a small dose within the appropriate wavelength region matching the action spectrum of some unknown photobiological process alters the clinical response. As long as the etiopathogenetic basis of PLE itself has not been elucidated, one can only speculate on the mechanism of the inhibitory action of shorter wavelengths. One possibility could be that a photosensitizing compound may be inactivated by irradiation with an appropriate spectrum.

Spectral interactions have also been observed in cellular systems irradiated at different wavelengths in vitro (7, 8, 9). In recent studies an inhibition of solar urticaria by pre-irradiation with wavelengths longer than the action spectrum has been reported (10, 11, 12), and we have observed an inhibition of UVA-induced photopatch test reactions in some patients pre-irradiated with low non-erythematogenic doses of UVB (13).

These findings raise the possibility that in some patients with PLE, UVB avoidance achieved, e.g. by conventional sunscreens or by window glass, is not even inefficient in the prevention of the eruption, but rather may deteriorate the condition by removing an inhibiting action of UVB. Furthermore, the findings presented seem to make PLE even more polymorphic than it is now on clinical and histological grounds.

## REFERENCES

1. Morison WL, Stern RS. Polymorphous light eruption: a common reaction uncommonly recognized. Acta Derm Venereol (Stockh) 1982; 62: 237–240.
2. Hölzle E, Plewig G, Hofmann C, Roser-Maaß E. Polymorphous light eruption. Experimental reproduction of skin lesions. J Am Acad Dermatol 1982; 7: 111–125.
3. Ortel B, Tanew A, Wolff K, Hönigsmann H. Polymorphous light eruption: action spectrum and photoprotection. J Am Acad Dermatol 1986; 14: 748–753.
4. Jansén C. The polymorphic phototest reaction. Arch Dermatol 1982; 118: 638–642.
5. Plewig G, Hölzle E, Roser-Maaß E, Hofmann C. Photoallergy. In: Ring J, Burg G, eds. New trends in allergy. Berlin: Springer, 1981: 152–169.
6. Mutzhas MF, Hölzle E, Hofmann C, Plewig G. A new apparatus with high radiation energy between 320–460 nm: physical description and dermatological applications. J Invest Dermatol 1981; 76: 42–47.
7. Suzuki F, Han A, Hill CK, Elkind MM. Suppressive action of near-ultraviolet light on ouabain resistance induced by far-ultraviolet light in Chinese hamster cells. Mutat Res 1983; 119: 399–406.
8. Tyrell RM. Mutagenic action of monochromatic UV radiation in the solar range on human cells. Mutat Res 1984; 129: 103–110.

9. Tyrell RM, Werfelli P, Moraes EC. Lethal action of ultraviolet and visible (blue-violet) radiations at defined wavelengths on human lymphoblastoid cells: action spectra and interaction sites. Photochem Photobiol 1984; 39: 183–189.
10. Hasei K, Ichihashi M. Solar urticaria. Determinations of action and inhibition spectra. Arch Dermatol 1982; 118: 346–350.
11. Horio T, Yoshioka A, Okamoto H. Production and inhibition of solar urticaria by visible light exposure. J Am Acad Dermatol 1984; 11: 1094–1099.
12. Ichihashi M, Hasei K, Hayashibe K. Solar urticaria. Further studies on the role of inhibition spectra. Arch Dermatol 1985; 121: 503–507.
13. Przybilla B, Ring J, Schwab U, Galosi A, Dorn M, Braun-Falco O. Photosensibilisierende Eigenschaften nichtsteroidaler Antirheumatika in Photopatch-Test. Hautarzt 1987; 38: 18–25.

## Persistent Light Reaction

### *Successful Treatment with Cyclosporin A*

PETER DUSCHET, THOMAS SCHWARZ, GREGOR OPPOLZER and FRITZ GSCHNAIT

*Department of Dermatology, Hospital Vienna-Lainz, Austria*

Duschet P, Schwarz T, Oppolzer G, Gschnait F. Persistent light reaction: Successful treatment with cyclosporin A. Acta Derm Venereol (Stockh) 1988; 68: 176–178.

A 53-year-old male patient who had suffered for several years from severe persistent light reaction possibly due to tribromsalan photosensitivity was treated with cyclosporin A after long-term low-dose administration of corticosteroids which had to be discontinued. PUVA therapy was impracticable due to the extraordinarily high UVA sensitivity. When cyclosporin A blood concentrations between 100 and 200 ng/ml were reached, the patient was nearly free from symptoms; the excellent clinical response was also documented by phototesting performed prior to and during therapy. Cyclosporin A may be a valuable therapeutic alternative to systemic corticosteroids for severe cases of persistent light reaction which cannot be controlled by photoprotective measures. (Received July 27, 1987.)

P. Duschet, Department of Dermatology and Venereology, Hospital Vienna-Lainz, Wolkersbergenstr. 1, A-1130 Vienna, Austria.

Photoallergic contact dermatitis is common and may be caused by a variety of chemical agents such as coumarins, para-aminobenzoic acid, benzocain, musk ambrette and halogenated phenolic compounds (1). In most instances, the skin eruptions clear when exposure to the sensitizer is discontinued; in a minority of patients, however, persistent photosensitivity develops even if contact with the eliciting agent is carefully avoided (1, 2).

Persistent light reaction (PLR) (3) represents one of the most disabling disorders in photomedicine. Various therapeutic attempts have been performed in patients with persistent light reaction, with moderate success such as topical and systemic corticosteroids, 'sun blockers' and $\beta$-carotene (4). Oral photochemotherapy (PUVA), though offering high photoprotection due to melanin hyperpigmentation (4, 5, 6), fails if patients have an extremely low UVA threshold and thus cannot tolerate low-dose UVA after photosensitization with psoralens. Skin biopsies of patients with PLR show a predominance of Leu-3a-positive cells (i.e. T-helper cells) (7) whose activity can be successfully inhibited by systemic administration of cyclosporin A (8).