Psoriatic Plaques Exhibit Red Autofluorescence that is Due to Protoporphyrin IX

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In evaluating the autofluorescence properties of normal and diseased skin we discovered that psoriatic plaques can emit a distinct red fluorescence when illuminated with UVA or blue light. Using a macrospectrofluorometer equipped with a 442 nm excitation laser, a sharp in vivo fluorescence emission peak around 635 nm could be demonstrated within the plaques of 34 of 75 (45%) patients with psoriasis. This peak was absent from normal appearing skin of psoriatic patients and also from the skin of 66 patients with other dermatologic diseases. A microspectrofluorometer coupled with the same excitation laser was used to obtain emission spectra of separated epidermal sheets and dermis from plaques demonstrating macroscopic red autofluorescence. An emission peak around 635 nm was observed in all three patients thus studied, but only on spectra obtained from the epidermis. Additional spectra of vertical microscopic sections of intact psoriatic skin from five other patients revealed that the peak originated from the stratum corneum. Emission spectra from other microlocations including the mid-epidermis and dermis of psoriatic and normal skin, as well as the stratum corneum of normal skin, failed to demonstrate a 635 nm peak. The excitation and emission fluorescence spectra of acid extracts of psoriatic scale from five patients were all similar to those of protoporphyrin IX in acid solution. High performance liquid chromatography identified the presence of protoporphyrin IX in the acid extracts from psoriatic scale of the same patients. We conclude that native psoriatic plaques can exhibit red autofluorescence that is due to elevated levels of protoporphyrin IX within scales. Key words: psoriasis, spectroscopy. J Invest Dermatol 111:586–591, 1998

The emission of light following absorption of incident photons by chromophores is termed fluorescence. Cutaneous fluorescence can be detected when human skin is illuminated with ultraviolet or visible light, and in the absence of exogenously administered fluorescent compounds this phenomenon is specifically referred to as “autofluorescence” (Anderson and Parrish, 1982; Zeng et al, 1993b). Autofluorescence is believed to originate from endogenous fluorophores, including nicotinamide adenine dinucleotide, elastin, collagen, flavins, porphyrins, and amino acids, although the exact contribution of each of these species to the overall autofluorescence signal emitted by the skin is unclear (Alfano and Kats, 1996). Fluorescence signals originating from the skin are generally of low intensity and can be detected and measured only under conditions of low ambient light.

The ultraviolet A-emitting Wood’s lamp represents a classical application of cutaneous fluorescence for dermatologic diagnosis (Kochevar et al, 1996). Autofluorescence emission images and spectra can be generated and recorded when incident (excitation) light is shone on skin. The use of autofluorescence photography has been described for evaluating treatment response in acne vulgaris (Martin et al, 1973; Lucchina et al, 1996), and fluorescence emission spectroscopy from diseased skin has been studied by ourselves (Zeng et al, 1996) and others (Lohman and Paul, 1988; Sterenborg et al, 1996).

During the course of a study on the autofluorescence spectroscopic properties of various skin disorders, we detected an intense red fluorescence emission peak in the plaques of many patients with psoriasis. Although this red fluorescence was initially recorded during laser-induced in vivo emission spectrofluorometry, we subsequently discovered that it could also be readily visualized using a Wood’s lamp. In this study we sought to evaluate the spectroscopic, microscopic, and biochemical aspects of this heretofore unreported observation in greater detail.

MATERIALS AND METHODS

Patients Seventy-five patients with psoriasis and 66 patients with a variety of other dermatologic disorders were evaluated (18 with actinic keratoses, 12 with warts, 11 with contact dermatitis, four with sebaceous hyperplasia, three with atopic dermatitis, three with porokeratosis, three with rosacea, three with seborrhoeic dermatitis, two with ichthyosis vulgaris, two with discoid lupus erythematosus, and one each with parapsoriasis and squamous cell carcinoma in situ). The racial origins of the psoriatic patients were as follows: 51 Caucasians, 17 Asians, five East Indians, one African, and one Native American; for the nonpsoriatic subjects, the racial distribution was 43 Caucasians, 21 Asians, and three East Indians. All patients were recruited from the outpatient clinics of The Skin Care Centre at the Vancouver Hospital and Health Sciences Centre. The study was approved by the University of British Columbia Clinical Research Ethics Board, and informed consent was obtained from each patient. Noninvasive macrospectrofluorometry was performed on all patients and skin biopsies were taken from 11 patients with psoriasis for detailed microscopic spectral analysis. Stratum corneum and psoriatic scales were obtained by tape stripping from three patients with psoriasis, and scales for biochemical porphyrin analysis were collected from a total of nine patients: five with psoriasis, two with atopic dermatitis, one with ichthyosis vulgaris, and one with an exfoliative drug eruption.

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**M**icrospectrofluorometry

In *situ* macroscopic autofluorescence spectra from the skin were recorded using a computerized fluorescence spectrophotometer system (Zeng et al., 1993b). The light source for fluorescence excitation was a 442 nm helium-cadmium laser (He-Cd, Omnichrome, Chino, CA), and this was directed to a spectrometer (PC 1000, Ocean Optics, Dunedin, FL) and a personal computer. The excitation laser light was conducted to the skin through a bundle of 6 × 200 µm optical fibers. Autofluorescence emitted by the skin was collected with a 200 µm optical fiber and transmitted to the spectrometer for analysis. Using this set up each spectrum represented the fluorescence collected from an area of skin measuring ≈3 mm in diameter. A total of 16 separate psoriatic lesions were assayed with macroscopic spectrofluorometry, including plaques on the limbs (60), trunk (25), face (3), buttocks (three), scalp (two), palms (six), soles (one), as well as four psoriatic nails and two unspecified sites. For each lesion examined, microspectrofluorometry was performed on psoriatic skin, as well as on perilesional normal-appearing skin from the same body region to facilitate comparison of spectra. Patients had no topical medications on their skin at the time of spectra acquisition. Peak analysis was performed with the GRAMS/386 Software (Galactic Industries, Salem, NH).

Skin biopsies, epidermal sheet isolation, and tape-stripplings

Three millimeter punch biopsies from psoriatic plaques were performed on eight patients who demonstrated a macroscopic autofluorescence emission peak above 600 nm within their psorias; normal skin was also biopsied in six of these patients. Additional biopsies were taken from lesional skin in three other patients with psoriasis, all of whom did not demonstrate a red emission peak. One patient with parapsoriasis, and another patient with squamous cell carcinoma *in situ* (both of whom had no macroscopic emission peak above 600 nm) were also biopsied. All specimens except for three of the eight psoriatic biopsies that demonstrated a peak above 600 nm were snap frozen in liquid nitrogen-cooled isopentane. Twenty micrometer vertical sections were then cut perpendicular to the skin surface. Care was taken to minimize exposure of the tissue to ambient light. The sections were placed unfixed on glass slides for microspectrofluorometric analysis. The remaining three psoriatic specimens were separated into epidermis and dermis by overnight incubation in the dark at 4°C in Hank’s buffer solution containing 0.25% trypsin (Madsen et al., 1991). The following morning, the epidermis was mechanically separated from the dermis, mounted directly on glass slides, and frozen at –80°C until analyzed by microspectrofluorometry. The dermis was frozen in liquid nitrogen-cooled isopentane, from which 20 µm thick frozen sections were taken for analysis. Tape-stripped scales were obtained from three patients with psoriasis (all of whom also demonstrated red fluorescence emission peaks on macroscopic-spectrofluorometry) for microspectrofluorometry analysis by applying standard Scotch tape (3M) to psoriatic skin for 5 s. This procedure was repeated three times with the same piece of tape that was subsequently attached to a glass slide for analysis.

Microspectrofluorometry

The skin biopsies, isolated epidermal sheets, and tape-stripplings were analyzed using a microspectrophotometer (Zeng et al., 1993a). A 442 Hz-Cd laser was connected to an inverted Nikon microscope (Diaphot model, Nikon, Mississauga, ON) equipped with a 400 µm microtens-tipped optical fiber. A 480 nm long pass filter (Omega, Brattleboro, VT) was placed after the objective. Another optical fiber was used to collect light from a specific microlocation on the slide for transmission to an optical multichannel spectral analyzer (OMAIII, EG & G, Trenton, NJ), which was calibrated with a mercury lamp. This apparatus enabled acquisition of autofluorescence emission spectra from specific microlocations as small as 24 µm. For intact whole skin and isolated dermis, microspectrofluorometry was carried out on vertical tissue sections (i.e., sections cut perpendicular to the skin surface), whereas isolated epidermal sheets and scales collected by tape-stripping were analyzed in a horizontal orientation. At least two spectra each were recorded from the stratum corneum, mid-epidermis, lower epidermis, and dermis of psoriatic, diseased nonpsoriatic, and normal skin for the whole skin biopsy specimens. For the stratum corneum, mid-epidermis, lower epidermis, and dermis of psoriatic, diseased nonpsoriatic, and normal skin for the whole skin biopsy specimens. For the slides were placed on glass slides for analysis. Using this set up each spectrum represented the fluorescence collected from an area of skin measuring ≈3 mm in diameter. A total of 16 separate psoriatic lesions were assayed with macroscopic spectrofluorometry, including plaques on the limbs (60), trunk (25), face (3), buttocks (three), scalp (two), palms (six), soles (one), as well as four psoriatic nails and two unspecified sites. For each lesion examined, microspectrofluorometry was performed on psoriatic skin, as well as on perilesional normal-appearing skin from the same body region to facilitate comparison of spectra. Patients had no topical medications on their skin at the time of spectra acquisition. Peak analysis was performed with the GRAMS/386 Software (Galactic Industries, Salem, NH).

**Fluorescence photography**

For clinical fluorescence photography, a Nikon F-601 camera with a 60 mm micro AF lens set at f/2.8 was used. Psoriasis plaques were illuminated in the dark with a Wood’s lamp (UVP, Upland CA, Model B 100 AP) and photographs were taken using a ASA 1600 Fujichrome film and exposure times ranging between 1/40 s and 1/4 s. For fluorescence microscopy, 20 µm sections of skin biopsies from psoriatic and normal skin were examined and photographed with a Nikon fluorescence photomicroscope (same model as used for microspectrofluorometry) equipped with epi-illumination from a mercury lamp and a 425 ± 25 nm band pass filter (Omega, Brattleboro, VT) between the lamp and the objective. A 590 nm long pass filter (Oriel, Stratford, CT) was placed between the specimen and the camera.

**Porphyrin extraction and analysis**

Scales were collected with 3–6 mm Fox dermal curettes from patients whose psoriatic plaques exhibited a red emission peak on macroscopic-spectrofluorometry. Scales from nonpsoriatic control subjects who did not demonstrate macroscopic red autofluorescence in lesional skin were also collected from two patients with atopic dermatitis, one patient with ichthyosis vulgaris, and one patient with an exfoliative drug eruption. Scales were added to 3 M HCl and incubated with constant shaking at room temperature for 30 min. The mixtures were then centrifuged at 3000 rpm for 10 min and the supernatants collected. An LS-5 Perkin-Elmer fluorometer was used to obtain excitation and emission spectra for the acid extracts from five psoriasis and four control specimens (Puckel et al., 1991). To generate excitation spectra, the acid extracts were scanned with light from 350 to 440 nm while monitoring fluorescence emission at 601 nm, whereas for emission spectra recorded from 550 to 700 nm, a fixed excitation of 403 nm was used. A Hewlett-Packard high performance liquid chromatograph (HPLC, model HP Series II 1090) was used to separate and identify any porphyrins present in scale extracts. Supernatant acid extracts were filtered and injected onto a C18 reversed phase column. The porphyrins were eluted with a linear gradient of 100% solvent A (10:90 by volume mixture of acetonitrile and 1 M ammonium acetate per liter buffered at pH 5.16) to 90% Solvent B (10:90 by volume mixture of acetonitrile:methanol) that was reached over 17 min followed by another 8 min of elution with 90% solvent B. The flow rate was 1.5 ml per min. Porphyrins were detected with a Hewlett-Packard fluorescence detector (model 1046 A) with the excitation set at 401 nm and the emission recorded above 550 nm. Porphyrin standards (Porphyrin Products, Logan, UT) diluted in 3 M HCl were also eluted using the same parameters.

**RESULTS**

**Psoriasis exhibits an autofluorescence emission peak around 635 nm**

Fluorescence emission spectra from normal skin of patients with psoriasis showed an increase in fluorescence intensity from 470 nm to about 520 nm followed by a monotonic decrease from 520 nm to the beginning of the infrared (Fig 1). In 34 of 75 (45%) psoriatic patients evaluated, the fluorescence emission spectrum of psoriatic skin exhibited a narrow peak around 635 nm (Fig 1), which was absent on all spectra from normal appearing skin of the same psoriatic patients. Some patients exhibiting the peak had used no treatment for their psoriasis for more than 3 mo. The mean wavelength of the peak was 634 ± 2 nm (mean ± SD) for 46 spectra. In other respects, the remainder of the fluorescence emission spectra of psoriatic skin was not significantly different from those obtained from normal skin. When the spectra were analyzed by taking the ratio of psoriasis to normal skin fluorescence, the ratio spectra were essentially flat apart from a peak around 635 nm (data not shown). The 635 nm peak was absent from all 66 emission spectra that were obtained under the same conditions from patients with other skin diseases. If the laser excitation light was shone on the skin for more than 5–15 s before recording a spectrum, the 635 nm peak decreased in intensity and a new peak appeared at 675 ± 5 nm (mean ± SD from 23 spectra). This 675 nm signal was not present on normal skin of psoriatic patients, nor on the lesional or normal skin of nonpsoriatic subjects.

**Figure 1. Macroscopic emission peak at 635 nm on psoriatic skin.**

Macroscopic-spectrofluorometric emission spectra were recorded *in vivo* after excitation with a 442 nm laser. Gray curve, normal skin of a psoriatic patient; black curve, psoriatic skin, a.u., arbitrary units.

**Fluorescence emission spectra from normal skin of patients**

Figure 1. Emission peak on normal skin.
Psoriasis exhibits visible red autofluorescence on Wood’s lamp examination

After identifying the 635 nm emission within psoriatic plaques on macrospectrofluorometry, we subsequently observed that bright red fluorescence could be readily seen on psoriatic skin illuminated with Wood’s lamp (Fig 2). The distribution pattern of fluorescence could be heterogeneous within a plaque as shown in Fig 2(b). Plaques with thicker scaling were found to display more intense fluorescence, whereas within a given patient some plaques did not exhibit any visible red fluorescence at all (not shown). Although the number or proportion of psoriatic plaques per patient exhibiting red fluorescence was not systematically recorded or evaluated, it appeared to be more frequently seen on the trunk than on the limbs. Red fluorescence within plaques was present both in patients undergoing UVB phototherapy and in patients using no light treatments. This fluorescence was not detected on normal skin of psoriatic patients. When superficial scales from a psoriatic plaque were gently removed and placed either on the patient’s own normal skin or on the normal skin of an individual without psoriasis, red fluorescence from the scales could be demonstrated (data not shown).

The red autofluorescence of psoriasis originates from the stratum corneum

Bright red fluorescence was observed in the stratum corneum of psoriasis but was absent from the dermis and other layers of the epidermis (Fig 3). The microscopic emission spectrum of isolated epidermal sheets from normal-appearing skin of patients with psoriasis showed a sharp increase in autofluorescence around 475 nm followed by a maximum around 525 nm and a gradual decrease at higher wavelengths (not shown). In contrast, the emission spectra of isolated psoriatic epidermal sheets demonstrated a narrow peak around 635 nm in all three patients studied; the 635 nm peak was absent from the corresponding isolated dermal sections (not shown). Microspectrofluorometry was performed on vertical sections of whole skin biopsies from five psoriatic patients to localize the epidermal component responsible for the 635 nm fluorescence. Figure 4(a) shows an intense emission peak around 635 nm that was detected only in the stratum corneum of psoriatic skin. The wavelength of this emission peak was 637.4 ± 1.5 (mean ± SD) for 38 spectra from six patients. A lower intensity peak at 673 ± 5 nm (mean ± SD; 16 spectra from five patients) on the emission spectrum of psoriatic stratum corneum...
Figure 5. Emission spectrum of acid extracts from psoriatic scales is similar to the emission spectrum obtained with protoporphyrin IX. (a) emission spectrum of psoriatic scale extract; (b) emission spectrum of protoporphyrin IX. a.u., arbitrary units.

(Fig 4a) appeared when laser light from the microspectrofluorometer was shone at the same location while recording repeated spectra (data not shown); as the intensity of this peak increased there was a corresponding decrease in the intensity of the 635 nm peak. The primary 635 nm peak was absent from other microlocations, including the mid-epidermis and dermis of psoriatic and normal skin and the stratum corneum of normal skin (Fig 4b–d). The peak was also absent from the stratum corneum of three psoriatic patients who failed to demonstrate a macroscopic 635 nm signal as well as from the biopsies of psoriasis and squamous cell carcinoma in situ (one each).

Microspectrofluorometry of tape-strippings from psoriatic plaques showed a distinct peak around 635 nm that was present on tape with scales and absent on control tape without scales thereby further confirming the stratum corneum origin of the peak (not shown).

Protoporphyrin IX is present in psoriatic scale extracts The emission spectra from each of five psoriatic scale acid extracts showed two broad peaks around 602 nm and 660 nm (Fig 5a). The spectrum of protoporphyrin IX in HCl was similar with two emission peaks at 602 nm and 658 nm (Fig 5b). The excitation spectra of the five psoriatic scale extracts were also similar to that of protoporphyrin IX. HPLC elution profiles of acid extracts from psoriatic scales revealed a major peak at 22.5 min for all five patients studied (Fig 6a). The retention time of this peak corresponded to that of protoporphyrin IX (Fig 6b). There were also minor peaks that were not of sufficient intensity to be integrated (Fig 6a), and none of these matched the retention times of the porphyrin standards except for one sample in which a peak eluting at around 9 min was close to the retention time of uroporphyrin. In another patient scale sample, a small signal that did not match any of the retention times for the porphyrin standards was present at 13.7 min. No porphyrin peaks were observed on the excitation and emission spectra or HPLC chromatographs of acid extracts from the scales taken from the patients with atopic dermatitis, ichthyosis, and exfoliative drug eruption (not shown).

DISCUSSION

Porphyrins and porphyrinogens are biologically ubiquitous intermediates in the biosynthetic pathway of heme, and are present in all cells capable of heme synthesis. Using in vivo fluorescence spectroscopy, microscopy, and HPLC, we have demonstrated that elevated levels of porphyrins can occur in psoriatic plaques as compared with normal or diseased nonpsoriatic skin. The porphyrins are present at levels sufficient to permit their direct visualization under Wood’s lamp illumination as red fluorescence. Punctate red fluorescence on the nose and forehead under Wood’s lamp was reported as early as 1927 (Bommer, 1927), and has been linked to the presence of porphyrins generated by Propionibacterium acnes (Cornelius and Ludwig, 1967; Lee et al, 1978; McGinley et al, 1980; Johnsson et al, 1987; Konig et al, 1994; Lucchina et al, 1996). The presence of red skin autofluorescence at the center of experimentally produced or grafted tumors has been reported for rats (Gougerot and Patte, 1939; Policard, 1924; Rochese, 1954), mice (Konig et al, 1989), rabbits (Ghadially and Neish, 1960), and chemically induced squamous cell carcinoma in the cheek pouch of hamsters (Harris and Werkhaven, 1987). In the latter model, microscopic
examination revealed that the red fluorescence was restricted to the "surface keratin layer" (Harris and Werkhazen, 1987). Similar red autofluorescence has also been reported for human oral and oropharyngeal squamous cell carcinoma (Harris and Werkhazen, 1987; Konig et al., 1994; Dhintra et al., 1996), oral mucosa dysplasia (Ingrams et al., 1997), as well as normal human tongue (Harris and Werkhazen, 1987).

In studies where macrospectroscopic autofluorescence measurements were performed, the emission peak was centered around 636–640 nm (Konig et al., 1994; Dhintra et al., 1996). There is no mention of psoriasis or psoriatic skin. Low levels of porphyrins have been shown to be detected by our HPLC technique cannot be ruled out, but protoporphyrin IX cannot be detected in normal skin of nonpsoriatic skin or psoriatic skin due to the presence of protoporphyrin IX.

Because porphyrins generate intense red fluorescence when excited with light around 400 nm, spectrofluorometric techniques are ideal for detecting and measuring porphyrins in the skin. Macrospectrofluorometry has been successfully used to detect and monitor cutaneous porphyrins noninvasively in patients receiving exogenous porphyrins for photodynamic therapy (Rhodes et al., 1997; Stringer et al., 1996).

We found that macrospectrofluorometric emission spectra from psoriatic patients were similar to those described for normal human skin in previous studies (Zeng et al, 1993b), except for a unique emission peak around 635 nm that is present only in psoriatic plaques. This wavelength corresponds to the in vivo emission peak of cutaneous protoporphyrin IX generated by topical application of the exogenous porphyrin precursor 5-aminolevulinic acid to the skin (Goff et al., 1992; Stringer et al., 1996; Rhodes et al., 1997). The light-induced 675 nm peak we observed in psoriasis also corresponds to that of the major excitable photoprotect of protoporphyrin IX (Charlesworth and Truscott, 1993).

It is possible that our macrospectrofluorometer may not have been sensitive enough to detect lower, yet still abnormally elevated levels of porphyrins that may have been present in the normal skin of psoriatic patients or in the affected skin of patients with nonpsoriatic disorders. Although we have not compared the intensity of the endogenous peak to the peak observed after topical application of ALA, our results demonstrate that it is important to account for baseline levels of porphyrin in psoriasis when evaluating local protoporphyrin IX induction induced by the administration of exogenous 5-aminolevulinic acid (Stringer et al., 1996).

It should be stressed that approximately half of our patients did not demonstrate red psoriatic autofluorescence, even with thorough evaluation by Wood's lamp and multiple site macrospectrofluorometry. If the protoporphyrin IX is of microbial origin, this heterogeneity could be related to variations in the microenvironment present on different plaques both within and between individuals. These variations could create microenvironments that are more or less optimal for microbial protoporphyrin IX production.

The ability of the microspectrofluorometer to obtain emission spectra from specific sites on tissue sections (Zeng et al., 1993a) revealed that the 635 nm peak originated specifically from the stratum corneum.

Fluorescence spectral analysis of psoriatic scale extracts were similar to the typical spectral pattern of porphyrins in acidic solution (Kappas et al., 1989). This pattern was absent in the scales of all five nonporphyrin control subjects studied, suggesting either the absence of porphyrins in the scales of these patients or the presence of porphyrins at concentrations below the sensitivity of our analytical method. Normal skin was not evaluated as it was not possible to amass a sufficient quantity of normal skin from psoriatic patients or in the affected skin of patients with nonpsoriatic dermatoses.

The origin of the protoporphyrin IX within the scales of psoriatic plaques remains unknown. Possibilities include in situ synthesis within the stratum corneum by cells or microorganisms, synthesis by metabolically active epidermal or dermal cells with subsequent accumulation in the stratum corneum, synthesis at a distant site within the body with accumulation in the stratum corneum via the blood supply, or direct topical application of exogenous porphyrins or porphyrin precursors to the skin by patients. The absence of a 635 nm microspectrofluorometric signal within the dermis and epidermis suggests that protoporphyrin IX does not reach the stratum corneum from the epidermis or dermis, although it cannot be excluded that dermal or epidermal porphyrin levels may have been too low to be detected by our microspectrofluorometer. Some of our patients were using either topical treatments (corticosteroids, calcipotriol, tar) or ultraviolet B phototherapy, or no treatment at all. None were using topical preparations that would be suspected of containing either porphyrins or porphyrin precursors, although it is possible that certain topical formulations could have enhanced the synthesis of porphyrins in psoriatic plaques. Bacteria synthesize porphyrins, and red fluorescence of colonies is used as an aid for identifying certain types of bacteria. For example, P. acnes produces protoporphyrin IX in vitro (Kjeldstad et al., 1984) and has been implicated as the source of clinical fluorescence observed in acne (Cornelius and Ludwig, 1967; McGinley et al., 1980). We were able to isolate P. acnes by culture from psoriatic scales of one patient and observed red fluorescence of these colonies under Wood's lamp (data not shown); however, other psoriatic patients who presented a 635 nm peak failed to grow P. acnes, and in other cases P. acnes could be isolated from normal appearing skin. Polymorphonuclear neutrophils can infiltrate the psoriatic epidermis in numbers sufficient to form microabscesses. Although the pattern of red fluorescence seen on micrographs (Fig 3) does not suggest a focal accumulation of protoporphyrin IX in microabscesses, it cannot be ruled out that protoporphyrin IX present in the stratum corneum somehow originates from polymorphonuclear neutrophils. Further studies including pretreatment of plaques with topical antibiotics may help determine the origin of the protoporphyrin IX present in psoriatic scale.

Blue and red light can activate protoporphyrin IX and both have been used to treat psoriasis following topical application of 5-aminolevulinic acid to induce protoporphyrin IX synthesis (Boehncke et al., 1994). It remains to be determined whether visible light can improve psoriasis in the presence of elevated endogenous protoporphyrin IX within scales. Sunlight exposure can improve psoriasis, and although this has been largely attributed to the effects of UVB and UVA photons, visible light may also play a role, possibly via photodynamic activation of protoporphyrin IX within psoriatic plaques. The observed higher frequency of red autofluorescence on truncal versus extremity plaques may have been due to photobleaching of protoporphyrin IX on sites that sustain greater exposure to ambient indoor and outdoor light.

In conclusion, we have demonstrated that red autofluorescence can be observed in psoriasis due to the presence of protoporphyrin IX within psoriatic scales. This phenomenon appears to be somewhat unique to psoriatic skin because it could not be demonstrated on normal skin of psoriatic patients or on the skin of patients with other noninfectious dermatoses.

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