THERAPY OF PSORIASIS BY TAR PHOTOSENSITIZATION

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For the first time since Goeckerman suggested the therapeutic potential of tar photosensitization of psoriatic skin, this mechanism has been shown to be effective in treating generalized psoriasis vulgaris. Long treatment times, need for special high-intensity sources, the presence of skin pain, and absence of information about long-term effects makes this treatment impractical at present.

For more than 50 years, the combination of tar and ultraviolet (UV) light has been the customary major treatment for generalized psoriasis. Tar is applied to the skin prior to UV exposure because, ever since Goeckerman first described this treatment in 1925 [1], it has been assumed that tar-induced photosensitization is an important aspect of the therapy [2]. This proposed mechanism must now be reconsidered for the following reasons. The erythema action spectrum of tar has been shown to lie within the UV-A range (320-400 nm) [3,4]. However, erythema from light sources most commonly used in the Goeckerman regimen is due to the more energetic, short-wavelength UV component (UV-B, 290-320 nm) [5,6]. Thus photosensitizing doses of UV-A are never attained with such sources because exposures are limited by UV-B erythemogenesis.

Recently, high-intensity UV-A fluorescent lamps have become available. Compared with conventional sources, the UV-B output is relatively insignificant. With these lamps it is now possible to deliver photosensitizing doses of UV-A without being limited by the UV-B energy. As part of a continuing reevaluation of the Goeckerman regimen, this study examined the use of photosensitizing doses of UV-A in the treatment of 5 patients with generalized psoriasis. Using the bilateral comparison technique [7], the response was compared with the Goeckerman treatment in the same patients. Estar, a recently-introduced tar product, was used as previously described [8].

MATERIALS AND METHODS

Five patients with stable generalized psoriasis covering more than 30% of their body surface were hospitalized to insure complete adherence to the protocol and permit careful observation at least once daily. No systemic medications or other topical medications were permitted. The minimal erythema dose (MED) to UV-B and the minimal phototoxic dose (MPD) of UV-A to tar-treated skin was measured for each patient. Estar was applied to the skin twice each day and removed with soap and water 2 hr before the single daily UV exposure. One side was exposed to UV-B and the opposite side to UV-A. Opaque sheets and midline tapes were used to insure no inadvertent double exposure of the contralateral side. Because UV-B exposures were brief, the UV-B side was usually treated first. Patients were treated for 20 consecutive days or until markedly improved.

UV-A Source (Lie-Down Unit)

The UV-A treatment system is a horizontal planar array of 50, four ft-long, specially designed fluorescent UV-A lamps (GTE Sylvania, Lighting Products Group). A 0.005-inch-thickness mylar sheet was placed beneath the lamps to eliminate wavelengths shorter than 318 nm. This system produced a UV-A irradiance of 9.0 mw/cm² at 10 cm beneath the mylar (320-400 nm, IL783), with some visible illumination, and immeasurably low UV-B irradiance. Horizontal uniformity of UV-A irradiance was ± 5%, in a plane 10 cm from the mylar, and ± 10% to a distance of 25 cm from the mylar, over the central region of the lamp array, where the patients were placed.

Dosimetry

Exposure doses were initially based on the erythema testing (MED and MPD) and subsequently on clinical judgment of the dose needed to maintain a minimal but definite erythema of normal skin.

RESULTS

In all patients the 2 sides were equally improved. The Table shows the number of treatments, UV dose at final treatment, and cumulative exposure dose for each patient. Patients 3 and 4 were essentially clear and were discharged for maintenance therapy. Patients 1 and 5 were markedly improved but still had some evidence of psoriasis at 20 treatments. Patient 2 was much improved after 14 treatments but refused to continue the inconvenient and complicated inpatient protocol.

While UV-B exposure times ranged from 30 sec to 17 min, UV-A exposures required up to 1 hr or more to both front and back of one lateral side. Interruptions to obtain relief from the smarting reaction (see below) caused further increase in treatment time. Tanning was the same on both sides.

SIDE EFFECTS

The lateral side of both breasts of one patient became red and tender. One patient had transient discomfort on a small site on the UV-A side, a third patient on the UV-B side. All responded to shielding for 1-to-3 days. The most bothersome side effect was a burning or smarting sensation during exposure to UV-A. This phenomenon, previously described in pitch workers [9], consisted of an unpleasant burning pain coming on at various intervals after commencing UV-A and limited to exposed skin. All patients experienced this at some stage during their course. At times it was severe and frequently treatments had to be interrupted. The smarting sensation would subside within seconds of stopping irradiation. No discomfort occurred during UV-B exposures.

DISCUSSION

Because individual variables are minimized, the bilateral comparison technique is a practical method for careful comparison of variables of treatment in a small number of patients.
Using this technique, we applied tar to patients with generalized psoriasis and compared repeated erythrogenic exposure doses of UV from 2 artificial sources. Psoriasis was improved equally whether treated with UV-A or UV-B.

This observation could be explained if the tar was the sole therapeutic agent [10] and UV had no effect [11]. Clinical experience and studies with tar alone suggest that this is not the case. Also, in these patients we observed that areas treated with tar but not exposed to UV improved only minimally. Treatment in the prone position does not allow exposure of certain intertriginous sites and these sites showed no erythema or pigmentation, and only minimal improvement of psoriasis.

Of course, it is possible that, in tar-sensitized skin, the action spectrum for the therapy of psoriasis is not the same as the action spectrum for erythema [3,4]. This hypothesis would be extremely difficult to prove. It is more likely that both therapeutic efficacy and erythrogenesis result from phototoxicity whether due to UV alone or UV plus sensitizer (tar, psoralen).

Measurements of these patients’ MED suggest that erythema on the side treated with tar and UV-B resulted from UV-B alone. The facts that repeated erythrogenic doses of UV-B are known to be beneficial to psoriasis [12,13] and that several studies suggest UV-B is the active wavelength in tar-UV therapy [14-16], raise interesting questions about the exact role of tar in Goeckerman therapy, as popularly used, with light sources whose spectral distribution is such that erythrogenesis is primarily from UV-B.

As was demonstrated daily by these 5 patients, erythrogenesis on the tar-UV-A-treated side was closely related to tar phototoxicity. The range of UV-A exposure doses used here is not adequate to cause erythema in normal unsensitized skin [17,18]. Studies in our laboratory using this light source and others have shown that it requires more than twice the UV-A dose used here to cause minimal erythema of persons of equivalent complexion [18]. Because radiation of wavelengths less than 320 nm made up less than 10^-5% of the radiation, erythema could not possibly result from these shorter wavelengths.

Suberythrogenic UV-A exposure doses of tar-treated skin have not been successful in clearing psoriasis [14,16]. The larger and more phototoxic doses used here led to improvement in all 5 patients. The range of UV-A exposures used in this study (6-31 J/cm^2) is not therapeutic to psoriasis [19,20; unpublished data] but daily exposure to much larger doses (100 to 300 J/cm^2) of UV-A alone is both erythrogenic to normal skin and leads to improvement of psoriasis vulgaris [21].

This paper documents for the first time the successful treatment of psoriasis by tar phototoxicity. This observation has theoretical interest but clearly the tar-UV-A treatment cannot be considered a practical option. Treatment times, using commonly-available light sources are too long. Higher intensity sources would merely increase the frequency and severity of smarting reactions. Finally, the unknown long-term effects of high doses of deeply-penetrating UV-A remain a concern.

### References