T Lymphocyte E Rosette Function During Photochemotherapy (PUVA) of Psoriasis

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E rosette formation by peripheral blood T lymphocytes was studied during photochemotherapy (PUVA) of psoriasis. Prior to PUVA treatment the percentage of E rosette forming cells was found to be markedly decreased in patients with active psoriasis, whereas it was normal in those with inactive skin lesions.

In the initial phase of PUVA therapy (within the first 2 weeks) single PUVA exposure induced the decrease of the percentage of E rosettes in patients with psoriasis, but not in normal controls. This immediate suppression of E rosette formation, which persisted for more than 24 hr, was related to combined 8 MOP + UVA action, but not to 8 MOP or UVA irradiation alone. T lymphocytes reacquired an initial number of E rosette forming cells 48 hr after the last PUVA exposure.

PUVA-induced inhibition of E rosette formation was most prominent in patients with inactive psoriasis in whom the initial percentage of E rosettes before treatment was normal. A lesser degree of decrease of E rosette forming cells after PUVA exposure was observed in patients with active psoriasis and reduced initial percentage of E rosettes.

The suppression of E rosette formation caused by PUVA exposure was related to the presence of psoriatic lesions, since it could not be demonstrated after clearing of skin changes.

Irrespective of preceding activity of the disease, E rosette formation showed a gradual improvement with PUVA therapy. The reduction in the percentage of E rosettes completely disappeared in the fourth week of PUVA treatment, which was correlated with healing of skin lesions. Almost all patients with psoriasis (85%) recovered normal E rosette function up to 1 mo after termination of PUVA treatment.

Patients with psoriasis have been found to have abnormalities in cell-mediated immune response. The frequency and intensity of experimentally induced hypersensitivity to DNCB was significantly lower in the patients than in normal population [1,2]. Decrease in lymphocyte response to nonspecific mitogens (PHA [phytohemagglutinin], Con A [concanavalin A], and PWM [pokeweed mitogen]) was also reported by several authors [3-5]. These abnormalities were not related to the reduction of T cell count. Decreased E rosette formation in patients with psoriasis, previously described by Cormane et al [6] Guilhou et al [4], and Gliński et al [7] was found to be dependent on defective function of T lymphocytes [8]. Furthermore, the reduction in E rosettes and decreased DNCB sensitization in psoriasis was related to the activity of the disease, but not to the extension of skin lesions [1,7]. The suppression of E rosette formation could be restored by in vitro short-term preincubation of psoriatic lymphocytes with Con A, and disappeared during remission of the disease [8].

Finally, the sera of patients with psoriasis were found to contain a factor inhibiting normal T lymphocyte E rosette function, the concentration of which increased within 2 mo of recent relapse of skin lesions [8].

The percentage of E rosette forming cells were also studied in patients with psoriasis receiving PUVA therapy. Cormane et al [9,10] have observed decrease in the number of rosetting lymphocytes in the patients and normal controls after 4 PUVA exposures. The percentage of E rosettes returned to the starting range after 8 PUVA exposures, but was still significantly lower in psoriatics than in normals.

A marked reduction in E rosettes and normal number of EAC rosettes in PUVA-treated patients were reported by Ortonne et al [11]. The initial percentage of E rosette forming cells in those patients was found to be normal. Unfortunately, there were no data concerning clinical state of patients with psoriasis, the number and frequency of PUVA exposures, and the time relation of the E rosette test to the last PUVA irradiation.

The purpose of our paper was to determine a short-term effect of individual PUVA exposure as well as a long-term effect of PUVA therapy on peripheral blood lymphocytes forming E rosettes in patients with psoriasis with varying activity and extent of skin lesions.

MATERIALS AND METHODS

Selection of Patients

58 patients with psoriasis vulgaris receiving PUVA photochemotherapy were studied. Control group consisted of 5 patients treated with PUVA for other purposes (2 urticaria pigmentosa, 2 vitiligo and 1 pre-myocysis fungoides), and 26 normal volunteers. Two clinical criteria were used for classification of patients with psoriasis: 1. activity of the disease, and 2. extent of skin lesions. The activity of cutaneous lesions was defined as: A2—pin-point lesions, small, papules spreading, positive Köbner phenomenon; A1—active spreading peripherally plaque lesions with only occasionally small papules; and AO—skin lesions stationary for a long period of time (more than 3 mo). The extent of skin changes was also graded in terms of surface occupied by psoriatic lesions: B1—less than 10%, B2—10–40%, and B3—more than 40%.

Patients with psoriasis had never received PUVA photochemotherapy, and corticosteroids or immunosuppressive drugs systemically before they were included into the study. Prior to PUVA therapy the patients had been only treated externally with tars, corticosteroid ointments, anthralin and salicylic acid.

To obtain comparable results individuals who were extremely sensitive to sunlight have not been included into our experiments.
puVA Treatment

8-methoxypсорalen (8 MOP) (Metoxalen, Westwood Pharm. Inc.) was administered orally in doses of 0.8-0.9 mg/kg body weight 2 hr before UVA irradiation.

UVA light source was a 4-well stand-up cabinet containing 112 60 cm-long fluorescent lamps in a horizontal plane. This source emits a continuous spectrum of irradiation between 320-400 nm wavelength with peak emission at 365 nm. The energy delivered was on average 2.7-3.5 mw/cm² at a distance of 10 cm from the fluorescent lamps.

The patients were irradiated in an alternate day schedule (excluding Sunday) 3 times a week. The dose of UVA used for the first 3 PUVA exposures varied from 2.4 to 2.7 J/cm², and starting from the 4th UVA irradiation a dose was increased to 3.2-3.6 J/cm². Number of PUVA exposures necessary for clearing of psoriatic lesions varied from 12 to 25 (average 15.1) in this group of patients. Total UVA dose received by individual patient was about 35-78 J/cm² (mean 49 J/cm²).

Timing of Separate Experiments

Three different schedules of blood drawing for the determination of E rosette forming cells were used: A. for each patient: 1. before the PUVA therapy; 2. after every 3 PUVA irradiations (once a week) on a day of intermission between 2 consecutive PUVA exposures (3rd and 4th, 6th and 9th, 9th and 10th, etc.; and 3. 1 mo after completion of PUVA therapy.

B. At different times of PUVA treatment (PUVA exposure [1-18]) twice daily: just before oral administration of 8MOP and 2 hr before UVA irradiation, and 1 hr after PUVA exposure.

C. Between two consecutive PUVA exposures (No. 3-4, 4-5, or 5-6): 2 hr before PUVA exposure, 1 hr after PUVA irradiation, 24 hr after PUVA exposure, and 48 hr after PUVA exposure (just before administration of 8 MOP prior to the next PUVA irradiation).

E Rosette Test

Peripheral blood lymphocytes were isolated on a Ficoll-Ronpacon gradient of specific gravity 1.078 by a method described by Boyum [12]. Spontaneous rosette formation with sheep erythrocytes (SRBC) by lymphocytes was studied by modification of the method of Wybran et al [13].

Briefly, SRBC were washed 3 times in Hank's medium and centrifuged at 400 xg for 5 min. 0.2 ml of lymphocyte suspension (4 x 10⁸/ml) was added to 0.2 ml of 0.5% v/v washed SRBC, and 0.2 ml heparinized SRBC-absorbed fetal calf serum. Lymphocyte:erythrocyte ratio was 1:10. The mixture was incubated for 45 min at 37°C, centrifuged at 200 xg for 5 min, and kept overnight at 4°C.

Lymphocytes surrounded by 3 or more erythrocytes were considered as E rosettes. The percentage of lymphocytes forming E rosettes with SRBC was calculated for 400 consecutive lymphocytes.

Tests were done in duplicate for each patient and mean results accepted if the percentage of E rosettes in both samples differed by not more than 5%.

RESULTS

Once a Week Determination of E Rosette Formation During PUVA Treatment

A. in relation to activity of the disease. Prior to PUVA treatment the percentage of E rosette forming cells was found to be decreased in 70% of patients with psoriasis (23 out of 33) compared to normal controls. Most of these cases were distributed into both groups of patients with active skin lesions (A2 and A1). The initiation of PUVA therapy induced the subsequent reduction of starting value of E rosettes in 42% of cases (14 out of 33). This was shown almost exclusively in patients with inactive skin lesions (AO) who were found to have normal initial distribution of E rosette forming cells (62.3%) before onset of PUVA therapy.

In active psoriasis (group A1 and A2) the percentage of E rosette forming cells increased gradually between every 3 PUVA irradiations reaching the normal value of rosetting cells after 9 or 12 PUVA exposures respectively (Fig 1). Patients with inactive psoriasis (group AO) showed a marked reduction in E rosettes after 3 PUVA exposures (47.7%), which persisted after 6 PUVA (51.6%), but returned to normal range after 9 PUVA exposures (60.1%).

In the majority of patients the increase in the percentage of E rosette forming cells was correlated with the flattening and disappearing of psoriatic lesions.

There was no decrease of the percentage of E rosettes within the first 2 weeks of PUVA treatment in control groups consisted of 3 normals and the 5 patients receiving photochemotherapy for other purposes.

B. in relation to the extension of skin lesions. Examination after every 3 PUVA exposures showed no significant difference in the percentage of E rosette forming cells between groups of patients with varying extent of skin involvement (B1, B2, and B3) (Fig 2). An initial mean percentage of E rosettes prior to PUVA therapy, and that after 3 PUVA as well as 6 PUVA exposures were markedly decreased. In all these clinical groups E rosette formation after 9, 12, and 15 PUVA irradiations was found to be normal. The reduction of initial percentage of E rosettes after 1 week of PUVA therapy (3 PUVA exposures) was relatively greater in group B1 and B3 than in group B2. However, most of patients with inactive psoriasis was distributed into these 2 groups.

Fig 1. The mean percentage (± SEM) of E rosette forming cells before PUVA therapy and after every 3 PUVA exposures in 33 patients with psoriasis varying with disease activity, and 8 control volunteers (group AO—11 cases; group A1—13 cases, and group A2—9 cases). The E rosette test was performed in each patient on a day of intermission in PUVA photochemotherapy.

Fig 2. The mean percentage (± SEM) of E rosette forming cells before PUVA therapy and after every 3 PUVA exposures in 33 patients with psoriasis varying with extension of skin lesions and 8 control volunteers (group B1—8 cases; group B2—14 cases, and group B3—11 cases). The E rosette test was performed in each patient on a day of intermission in PUVA photochemotherapy.
**E Rosette Formation 1 Month after Completion of PUVA Treatment**

PUVA therapy was effective in all our patients, whose psoriatic lesions cleared within 4–8 weeks and did not relapse for more than 1 mo despite of discontinuation of the treatment. Irrespective of initial activity and extent of psoriatic lesions prior to PUVA 85% of patients (28 out of 33) free of skin lesions reacquired normal E rosette formation up to 1 mo after termination of PUVA therapy in contrast to only 30% of cases showing normal number of E rosettes before PUVA treatment (Table I). The remaining 5 patients were found to have only a slight decrease in E rosettes compared to normal controls, but the percentage of rosetting cells in these cases was much greater than that before starting of PUVA therapy. There was no difference between the mean percentage of E rosette forming cells in patients with psoriasis symptom-free for 1 mo after PUVA treatment and healthy normals.

**Suppression of E Rosette Formation with Single PUVA Exposure**

In patients with inactive psoriasis a reduction of the percentage of E rosette forming cells (47.2%) appeared as early as 1 hr after termination of individual PUVA exposure as compared to 64.1% of E rosettes before PUVA irradiation (Table II). The suppression of E rosette formation was still present after 24 hr (52.0%), whereas after 48 hr just before the subsequent PUVA exposure patients reacquired normal E rosette function (66.8%). Only a slight decrease of percentage of E rosette forming cells from 67.8% before PUVA exposure to 61.2% 1 hr after PUVA exposure was found in healthy normals. The difference between these 2 mean numbers of E rosettes was not statistically significant.

The decrease in the percentage of E rosettes was found in patients with psoriasis receiving 8 MOP followed by irradiation with UVA light (46.4%), but not treated with 8 MOP alone (65.8%) or UVA alone (61.5%) (Table III). Repeated UVA irradiations alone did not induce the reduction of the number of rosetting cells 24 hr after 3 PUVA exposures (62.1%) in contrast to combined 8 MOP + UVA treatment (49.4%).

**Effect of Single PUVA Exposure on Psoriatic Lymphocytes at Different Times of Photochemotherapy**

The comparison of E rosette test before and 1 hr after PUVA exposure revealed that the percentage of E rosette forming cells was significantly reduced, up to 32% of lymphocytes, in 20 out of 29 patients with psoriasis studied at different times of PUVA therapy (Table IV). The marked decrease of about one third of lymphocytes forming E rosettes (45.7%) within initial phase of therapy (1–5 PUVA exposures) was found in all patients, who previously had the normal percentage of E rosette forming cells (67.1%). In patients with active psoriasis with reduced initial percentage of E rosette forming cells (42.0%) only about 15% of lymphocytes were inhibited in their E rosette function by single PUVA exposure (35.7%). This was demonstrated in 5 of 9 these patients 1 hr after PUVA exposure in the first 2 weeks of PUVA treatment.

After further irradiations (7–18 PUVA exposures) 5 of 10 cases who showed a marked decrease in E rosettes were found to have persistent elevated psoriatic lesions. No reduction in E rosettes was observed in those almost free of skin lesions.

**DISCUSSION**

The present data implies that at least 2 distinct phenomena involving T lymphocyte subpopulations compete with each other during PUVA treatment of psoriasis, immediate suppression of T cells after PUVA and a gradual improvement in the T cell function with therapy.

Individual PUVA exposure immediately induced the suppression of T lymphocyte E rosette formation in patients with psoriasis, but not in normal controls. This lasted for more than 24 hr after irradiation, whereas before subsequent PUVA exposure 48 hr later T lymphocytes reacquired their normal function. These findings are in agreement with communications of Cormane et al [9,10] and Ortonne et al [11] who reported on

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**Table I. E rosette forming cells in 33 patients with psoriasis before and after PUVA photochemotherapy**

<table>
<thead>
<tr>
<th>Clinical state</th>
<th>No. of patients</th>
<th>% E Rosette forming lymphocytes before PUVA treatment</th>
<th>% E Rosette forming lymphocytes 1 mo after PUVA treatment</th>
<th>% patients with Statistical difference decrease* (t-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Range</td>
<td>Mean</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>52.5a</td>
<td>16.5</td>
<td>30.5–82.5</td>
</tr>
<tr>
<td>Activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>11</td>
<td>59.8a</td>
<td>7.8</td>
<td>47–70</td>
</tr>
<tr>
<td>A1</td>
<td>13</td>
<td>51.0a</td>
<td>13.6</td>
<td>30.5–82.5</td>
</tr>
<tr>
<td>A2</td>
<td>9</td>
<td>44.8b</td>
<td>7.4</td>
<td>31–55</td>
</tr>
<tr>
<td>Extention</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B1</td>
<td>8</td>
<td>57.3a</td>
<td>8.4</td>
<td>47–70</td>
</tr>
<tr>
<td>B2</td>
<td>14</td>
<td>47.4b</td>
<td>10.2</td>
<td>30.5–64</td>
</tr>
<tr>
<td>B3</td>
<td>11</td>
<td>55.5a</td>
<td>11.0</td>
<td>30.5–68</td>
</tr>
<tr>
<td>Normal</td>
<td>20</td>
<td>68.5a</td>
<td>5.7</td>
<td>58.5–80.5</td>
</tr>
</tbody>
</table>

*a* More than 2 SD below the mean percentage of E rosette forming cells in normal controls.

*b* Significantly different from normal/controls (t-test), *a* = p < 0.001, *b* = p < 0.002.

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**Table II. E rosette formation after individual PUVA exposure in 7 patients with inactive psoriasis during photochemotherapy (3rd–5th PUVA exposure) and 7 normal controls (1st–2nd PUVA exposure)**

<table>
<thead>
<tr>
<th>Time of examination</th>
<th>Psoriasis (7)</th>
<th>Normal (7)</th>
<th>Statistical difference from control (t-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Range</td>
</tr>
<tr>
<td>1. Before PUVA</td>
<td>64.1</td>
<td>4.9</td>
<td>57–70</td>
</tr>
<tr>
<td>After PUVA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 hr</td>
<td>47.2a</td>
<td>7.5</td>
<td>36–55</td>
</tr>
<tr>
<td>3. 24 hr</td>
<td>52.0b</td>
<td>6.5</td>
<td>44–61</td>
</tr>
<tr>
<td>4. 48 hr</td>
<td>66.8</td>
<td>4.8</td>
<td>62–76</td>
</tr>
</tbody>
</table>

*a,b* significantly different from group 1 (t-test), *a* = p < 0.002, and *b* = p < 0.01.
TABLE III. The percentage of E rosette forming cells after oral administration of 8 MOP alone, UVA irradiation alone, and combined application of 8 MOP + UVA in patients with psoriasis

<table>
<thead>
<tr>
<th>Inactive psoriasis</th>
<th>% E rosette forming cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before mean ± SD</td>
<td>1 hr after 1st application mean ± SD</td>
</tr>
<tr>
<td>1. 8 MOP (6)</td>
<td>62.2 ± 7.8</td>
</tr>
<tr>
<td>2. UVA (4)</td>
<td>66.0 ± 6.5</td>
</tr>
<tr>
<td>3. 8 MOP&lt;sup&gt;+&lt;/sup&gt; UVA (5)</td>
<td>60.6 ± 6.3</td>
</tr>
</tbody>
</table>

<sup>a</sup> 3 hr after 8 MOP.
<sup>b</sup> 1 hr after UVA.
<sup>c</sup> Group 3 significantly different from group 1 (p < 0.005), and group 2 (p < 0.05) (t-test).

TABLE IV. Immediate effect of individual PUVA exposure on peripheral blood lymphocytes of 29 patients with psoriasis at different periods of PUVA therapy

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of PUVA exposure</th>
<th>% E rosette forming cells</th>
<th>Patients with decrease&lt;sup&gt;c&lt;/sup&gt;</th>
<th>Absolute % reduction of E rosettes</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inactive psoriasis (10)</td>
<td>1-5</td>
<td>67.1 ± 8.8</td>
<td>45.7 ± 8.5</td>
<td>10/10</td>
<td>-12 -32</td>
</tr>
<tr>
<td>Active psoriasis (9)</td>
<td>1-5</td>
<td>42.0 ± 6.8</td>
<td>35.7 ± 8.4</td>
<td>5/9</td>
<td>+2 -14.5</td>
</tr>
<tr>
<td>Further phase of PUVA, disease activity undefined&lt;sup&gt;d&lt;/sup&gt; (10)</td>
<td>7-18</td>
<td>58.8 ± 13.8</td>
<td>66.7 ± 14.4</td>
<td>5/10</td>
<td>+14 -15</td>
</tr>
<tr>
<td>Total (29)</td>
<td>1-18</td>
<td>56.5 ± 14.4</td>
<td>46.4 ± 13.6</td>
<td>20/29</td>
<td>+14 -32</td>
</tr>
</tbody>
</table>

<sup>a</sup> More than 5% of lymphocytes below an initial percentage of E rosettes before PUVA irradiation.
<sup>b</sup> Actual activity of skin lesions impossible to assess precisely with regard to clinical improvement—before treatment mostly active lesions.

The depression in E rosette number in patients receiving PUVA therapy. The inhibitory effect of PUVA exposure on peripheral blood lymphocytes of psoriatic patients was due to combined 8 MOP and UVA action. 8 MOP alone and UVA light alone had no influence on the relative number of rosetting cells.

Since immediate suppression of E rosette formation with PUVA exposure occurred usually in the initial phase of the treatment (within the first 2 weeks) or after further irradiations only in patients whose skin lesions were relatively resistant, it seemed to be related to the presence of psoriatic lesions of both active and inactive type.

In active psoriasis in respect of diminished initial number of E rosettes an additional reduction of the percentage induced by PUVA was relatively small. In contrast, the immediate suppression was found to be responsible in patients with inactive psoriasis for extreme reduction of rosetting T cells within initial phase of PUVA therapy from normal level prior to the treatment to the range comparable to that of patients with active lesions.

The gradual improvement of E rosette formation with PUVA therapy was found in patients with both active and inactive psoriatic lesions in whom lymphocyte subpopulations were followed up once a week on a day after every 3 PUVA exposures. At this time the immediate suppression of T cells caused by preceding PUVA exposure, if present, should be still demonstrated because it lasts for more than 24 hr. T lymphocyte rosetting function became normal in the third week of PUVA treatment which was correlated to flattening of skin lesions.

Further irradiations (12-15 PUVA exposures) did not induce the immediate suppression of E rosette formation in patients with clinical improvement or in those almost free of skin lesions. T lymphocyte quantitation in this phase of PUVA and 1 mo after completion of the treatment remained normal in almost all cases irrespectively of initial disease activity and extent of psoriatic lesions. This is in agreement with our previous data on the restoration of T cell function in the remission of psoriasis [7, 8].

The mechanism of reduction in the percentage of T lymphocytes forming E rosettes during PUVA treatment is not clear. *In vitro* irradiation of peripheral blood lymphocytes with PUVA was found to inhibit lymphocyte transformation to nonspecific mitogens [14], and spontaneous incorporation of tritiated thymidine [15]. However, it is not clear if this effect could be due to either inhibition of DNA synthesis by 8 MOP-DNA photoaduct formation, or increased cell death in the phototoxic reaction.

The immediate inhibitory effect of PUVA exposure on T cells could be related to: 1. the irradiation of recirculating lymphocytes in cutaneous infiltrates and capillaries with a PUVA dose adequate to inhibit T lymphocyte function (direct mechanism), and/or (2) the release of inhibiting factors (i.e., immune complexes) from skin lesions into the circulation (indirect mechanism).

Close relation of the PUVA-induced depression of E rosette formation to the presence of skin lesions may argue for both an indirect mechanism of T cell inhibition and direct suppression of the function of recirculating lymphocytes. It is quite clear that indirect inhibition related to released factors should disappear with clearing of psoriatic lesions. Similarly, the absence of direct immediate suppression of T cells in intermediate phase of PUVA treatment might be explained by the disappearance of lymphocyte infiltration [16] and reduced blood flow through skin capillaries in healing cutaneous lesions [17] as well as an adequate protection against PUVA by hyperpigmented skin.

It should be stressed that PUVA appeared to be unable to suppress E rosette formation of more than one half of T lymphocytes in patients with psoriasis. This could be also explained by direct or indirect mechanism of PUVA action. Not all lymphocytes recirculating through the skin could receive UVA dose adequate to inhibit their rosetting function. However, in active psoriasis in contrast to inactive psoriasis the number of T cells which are sensitive to PUVA irradiation is relatively low despite of similar distribution of skin lesions. In active psoriasis T lymphocytes might be already inhibited with underlying unknown factors (perhaps previously described blocking factors in psoriatic sera [8]. The defect of E rosette formation in these patients could be unblocked in *vitro* by preincubation of lymphocytes with Con A [8].

PUVA exposure may provoke the release of inhibiting factors from skin lesions, which subsequently inhibit E rosette formation, but only of T cell subpopulation exhibiting affinity to those factors. In inactive psoriasis T lymphocytes sensitive to the action of blocking factors might be previously unaffected, so the greater number of T cells than in active psoriasis could
be suppressed after release of inhibiting factors stimulated by PUVA irradiation.

The alternative mechanism of PUVA-induced inhibition of T cell function remains to be elucidated.

REFERENCES


Corrected Date for the International Symposium on the Treatment of Psoriasis and Psoriatic Arthritis

This international symposium had been previously scheduled for February 1979 and will now be held from November 18–25, 1979. Speakers include Drs. T. J. Ryan, F. Sagher, N. Hjorth, and H. Maibach. The official language of the Symposium is English. The sessions will take place in Tel Aviv and the Dead Sea. Information can be obtained by writing or cabling N. D. Yahalom, 44 Ibn Gvirol, Tel Aviv, Israel. The meeting is sponsored by the Dermatology Department of the Hadassah Hospital, The Israeli Dermatologic Society, and the International Psoriasis Treatment Center.