Sir,
Ultraviolet radiation (UVR) induces isomerization of trans-urocanic acid (UCA), a natural component of human skin, into cis-UCA, which has been shown to suppress both delayed hypersensitivity and contact hypersensitivity (1).

An autoantigen-specific T-cell-mediated mechanism has been proposed in both chronic cutaneous lupus erythematosus, presenting mostly as discoid lupus erythematosus (DLE), and polymorphous light eruption (PLE) (2, 3). A subnormal epidermal UCA content might theoretically lead to disease exacerbation of T-cell-mediated disorders due to less cis-UCA-mediated suppression of cell-mediated autoimmune processes. To study this possibility, we examined the epidermal UCA contents of patients with DLE and compared the results with data obtained from PLE patients and control persons.

PATIENTS AND METHODS

Patients
DLE patients (7 males, 9 females, mean age 44 years, age range 17–71 years), PLE patients (3 males, 9 females, mean age 46 years, age range 29–64 years) and non-photosensitive healthy individuals (10 males, 14 females, mean age 45 years, age range 17–71 years) were included in the study. The diagnoses were based on clinical, histological (4) and serological findings. All DLE patients had been or were on antimalarial medication (either chloroquine 250 mg daily or hydroxychloroquine 300 mg daily). Eleven DLE patients were sampled while using antimalarial medication and 12 DLE patients while not using medication. Thus, 7 patients were sampled twice, with or without medication. The use of medication had lasted from 1 to 48 weeks and the abstinence for at least 5 weeks. In all PLE patients, the PLE rash had occurred during the preceding summer. None of the PLE patients was on antimalarial medication during or for at least 6 months before the study. Eight of 16 DLE patients presented with a history of PLE type skin lesions, a coincidence recently noted to be rather common in DLE (5).

Sampling and UCA analysis
In all patients and control persons, samples were collected from buttock skin. In the DLE patients, the back of the hand and in the PLE patients, healthy-looking extensor forearm skin, previously affected by PLE rash, was also examined. The back of the hand and the forearm were sampled in control patients in 19 and 12 cases, respectively. Eleven DLE patients and 12 DLE patients while not using medication. Thus, 7 patients were sampled twice, with or without medication. The use of medication had lasted from 1 to 48 weeks and the abstinence for at least 5 weeks. In all PLE patients, the PLE rash had occurred during the preceding summer. None of the PLE patients was on antimalarial medication during or for at least 6 months before the study. Eight of 16 DLE patients presented with a history of PLE type skin lesions, a coincidence recently noted to be rather common in DLE (5).

Statistical analysis
As the data were not normally distributed, the Mann–Whitney U test was used for comparison between groups. Statistical analysis was performed with Statistica® for Windows (Version 5.1, StatSoft, Inc, Tulsa, Okla, USA). Values of \( p < 0.05 \) were considered statistically significant.

RESULTS

The median cis-UCA content, but not the total UCA content, in UVR-protected buttock skin, was significantly lower in non-medicated DLE patients than in control persons and in PLE patients (Fig. 1). The cis-UCA results of the DLE patients with a history of PLE did not differ from those without such history.

In DLE patients with antimalarial medication, numerically higher cis- and total UCA contents in buttock skin were found, compared with the patients without medication, but the differences did not reach statistical significance.

The cis- or total UCA contents of DLE patients in sun-exposed skin, i.e. the back of the hand during the sunny season, did not differ significantly from those of control persons. However, the median cis-UCA content, but not the median total UCA content, in the back of the hand was significantly higher during the sunny season than in the winter, both in DLE patients (\( p=0.00007 \) and control persons.
Chloramphenicol Induced Acute Generalized Exanthematous Pustulosis Proved by Patch Test and Systemic Provocation

Sir,
Acute generalized exanthematous pustulosis (AGEP) is characterized by sudden onset of high fever, generalized scarlatiniform erythema covered by numerous non-follicular small superficial sterile pustules, blood leukocytosis with neutrophilia, and acute evolution (1–2). The main causative agent is drugs, but chloramphenicol has been rarely implicated (3).

Patch tests were performed in several cases of AGEP and results showing eczematous or pustular reaction were considered positive (2). Systemic provocation proved the cause in 1 case sensitive to isoniazid (4).

We here report a case of AGEP in which chloramphenicol was shown to be the cause by both patch test and oral provocation with a lowered dose.

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References

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