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In reply

We thank Lotti and colleagues very much for their interest in our report in the December 2005 issue of the ARCHIVES[1] *and for pointing out the device used in their study.*[2]

It is also very encouraging to learn from their experience that despite the superficial penetration characteristic of shorter UV wavelengths, targeted UV-B can be used to treat palmoplantar psoriasis with prolonged remission. This is probably because large fluences deliverable only by 308-nm excimer laser and targeted light systems and not by conventional phototherapy units were used. With such good response of the palmoplantar lesions, we speculate that these light systems can be used to treat psoriasis at other difficult locations such as the nail and the scalp.

It is exciting that more work involving targeted UV phototherapy is being conducted. We have recently completed our work on topical psoralen and targeted narrowband UV-B and are really looking forward to learning of new developments from Dr Lotti's group and others.

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HIV Eosinophilic Folliculitis in Uganda

We read with interest the article by Toutous-Trellu et al[1] about the beneficial effect of topical tacrolimus therapy for human immunodeficiency virus (HIV)-associated eosinophilic folliculitis (HIV-EF). The usefulness of such treatment has also been mentioned in other case reports.[2,3] Based on the cases presented by Toutous-Trellu and colleagues, however, it is difficult to determine how efficient this treatment is and whether the lesions would have disappeared with the use of other topical medications in association with highly active antiretroviral therapy (HAART) or with HAART alone. The authors mention that HAART alone was insufficient to obtain a remission of HIV-EF, because the symptoms persisted during HAART in the period before the treatment with topical tacrolimus. From the description of the patients, however, it is unclear how long the skin lesions persisted during HAART. It is possible that even without topical tacrolimus therapy, all HIV-EF lesions would have disappeared after prolonged effective HAART. The persistence of HIV-EF lesions during HAART can be explained by 2 phenomena: (1) the skin lesions persisted because of an immune restoration syndrome,[4] or (2) the skin lesions persisted because it generally takes several months of HAART before the HIV viral load drops below a detectable level.

The papular puritic eruption that is frequently observed in patients with HIV infection in Africa[5] is also very resistant to all kinds of topical treatment. Furthermore, within the first months of the initiation of HAART, there is an increase in the number of the lesions, probably because of immune restoration syndrome. In the long run, in our experience in Uganda, the papular puritic eruption and the itching associated with it disappeared in almost all patients during HAART; however, the lesions and symptoms may reappear in the cases in which HAART fails. To demonstrate the degree of efficacy of topical tacrolimus in the treatment of HIV-EF, and maybe also of papular puritic eruption, a randomized clinical trial should be performed to compare this treatment with other treatment strategies in patients with or without HAART.

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In reply

Dr Colebunders and colleagues suggest that HAART alone might be as efficient as HAART and 0.1% tacrolimus therapy in controlling HIV-EF. In our experience, based on 15 cases to date, we have consistently observed accelerated improvement when we have used the combination of HAART and 0.1% tacrolimus compared with HAART alone.[1]

Highly active antiretroviral therapy may control HIV-EF, but it requires a fairly long time to do so, as noted by Colebunders and colleagues. This prolonged persistence of HIV-EF with HAART can be explained by the suboptimal

control of HIV viremia as well as by an immune restoration syndrome. This lag can last several months, during which the patient endures a dermatosis with intense pruritus as well as disfiguring facial lesions. These symptoms may severely affect quality of life and sometimes lead to the patients' experiencing a greater difficulty in accepting and continuing HAART. We would like to emphasize that such a a relatively simple, topical treatment as 0.1% tacrolimus therapy can improve HIV-EF in a relatively short time. Topical tacrolimus therapy often improves pruritus in patients with HIV-EF in less than 1 week, and skin lesions improve in approximately 4 weeks, clearly much faster than with HAART alone. Furthermore, to our knowledge, our article is the first to report on the efficacy of topical tacrolimus therapy for HIV-EF. A few case reports describe the efficacy of topical tacrolimus in Ofuji disease (eosinophilic pustular folliculitis) in patients who do not have HIV,[2-4] *but it is still controversial whether HIV-EF is the same entity as eosinophilic pustular folliculitis in patients without HIV.*[5] *Finally, we agree with Colebunders and colleagues that a randomized clinical trial should be performed in a large cohort of patients to confirm that there is an improvement in the control of HIV-EF with HAART and 0.1% tacrolimus when compared with HAART alone.*

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Red Hairs, Number of Nevi, and Risk of Cutaneous Malignant Melanoma: Results From a Case-Control Study in Italy

Red-haired subjects have increased risk of cutaneous malignant melanoma (CMM). A meta-analysis of 46 epidemiologic studies published before September 2002 reports that red-haired subjects were at higher risk of melanoma than dark-haired ones, the pooled relative risk being 3.64 (95% confidence interval [CI], 2.56-5.37).[1] Higher nevus count is another well-established risk factor for CMM, regardless of hair color; however, Dellavalle et al[2] report that red-haired children had fewer nevi than children with other hair colors (mean count, 2.1 vs 6.1). Thus, the risk of CMM in red-haired subjects seems to be associated with some factor other than nevus count.

Data from a case-control study on CMM based on 542 cases and 539 controls were used to analyze the association between hair color, nevus count, and risk of melanoma. The proportion of red-haired subjects was 1.7% among cases and 0.7% among controls, and in this adult population, all red-haired subjects (n = 13) had freckles. The mean number of melanocytic nevi was significantly lower among red-haired people than among those with other hair colors (6.2 vs 17.4).

Our data confirm that the risk of CMM was higher in red-haired subjects than in subjects with other color hairs, despite the lower number of nevi. Compared with subjects with all other hair colors, red-haired subjects had an odds ratio (OR) of 1.9 (95% CI, 0.5-6.9) after adjustment for sex, age, education, body mass index, solar lentigines, propensity to sunburn, sunburn episodes, and tobacco smoking (adjusted), and an OR of 1.8 (95% CI, 0.5-6.7) after further adjustment for eye and skin color (fully adjusted) (**Table**). We repeated the analysis comparing red-haired subjects with black- and brown-haired subjects only, and the corresponding ORs were 3.0 (95% CI, 0.7-12.7) for the adjusted model and 2.7 (95% CI, 0.6-11.3) for the fully adjusted model (Table).

The population attributable risk[3] for red hair was, however, less than 1% in this Italian population.

Risk of skin cancers, including melanoma, has been associated with several factors related to the so-called red-hair color (RHC) phenotype that implies red hair, fair complexion, inability to tan, and the tendency to freckle. On one side, red hair melanin is characterized by high ratio of pheomelanin to eumelanin,[4] and it is well known that pheomelanin is a risk factor for skin cancer.[5] A British study based on 20 subjects from the United Kingdom, Southeast Asia, and India revised this hypothesis and reported that susceptibility to UV radiation may be determined by factors other than the amount of pheomelanin or the ratio of pheomelanin to eumelanin in skin and hairs.[6] On the other side, some variants of the human melanocortin 1 receptor (*MC1R*) gene that has a crucial role in determining human pigmentation were associated with increased melanoma risk.[7] Three of these

Table. Distribution and Risk of Cutaneous Malignant Melanoma vs Controls by Hair Color

Characteristic	Hair Color		
	Red	Other Than Red	Black or Brown
Cases, No.	9	534	275
Controls, No.	4	533	325
Nevi, mean No. (95% CI)	6.2 (2.3-10.0)	17.4 (15.9-18.9)	15.7 (13.9-17.5)
OR (95% CI)			
Adjusted*	1.9 (0.5-6.9)	Reference	NA
Fully adjusted†	1.8 (0.5-6.7)	Reference	NA
Adjusted*	3.0 (0.7-12.7)	NA	Reference
Fully adjusted†	2.7 (0.6-11.3)	NA	Reference

Abbreviations: CI, confidence interval; NA, not applicable; OR, odds ratio.
*Adjusted for sex, age, education, body mass index, solar lentigines, propensity to sunburn, sunburn episodes, and tobacco smoking.
†Adjusted as above with further adjustment for eye and skin color.