ical conditions, though otherwise resembling those of Hansen's disease, are not associated with nerve damage. It may be that this very frequent consignment by various authors, of long recognized observations important in the etiology of HD to different parts of the same paper, is due to our unwillingness to visualize spatial events measurable in cu μm and below, in a time frame measured in weeks, months, and years. As a result, we tend to ignore the fact that in tuberculoid lesions microscopic axoplasmic damage and granuloma formation are so closely associated in time and place as not to exclude the possibility that the former may precede and cause the latter, rather than the reverse [Corcos, M. G. Molecular biology of HD; the case for the involvement of a transferable plasmid. The Star 40:6 and 41:16 (1981–1982)].

So long as observations and experimental results are misinterpreted by their division into the logic-tight compartments to which I have elsewhere referred ([1]), so long will each one continue to add to the already substantial fabric of misunderstanding.

There is much forgetting to be done. At the time of this writing it is beginning to look as if molecular biological and biotechnological methods are becoming incompatible with "leprosy," the "lepra" cell, "lepromin," "lepra" reactions, "leprosy" patients, and "*Mycobacterium leprae.*" These older words were in use long before there had been any investigations into the relationship between bacterial genetics and the molecular pathology of higher plants and animals. Would it not be better to substitute words more in keeping with the newer technology for the older ones, rather than to attempt to fit terms that may have outlived their scientific as well as their social usefulness, into a new and hopefully advancing field of knowledge? Failure to do this could be instrumental in slowing the advance, in opening a larger "can of worms," in perpetuating further uncertainty and more confusing literature, and even perhaps in increasing the refinement of wrong structural models ([9]).

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Photoactivated Topical 8-Methoxypsoralen in Repigmentation of a Tuberculoid Leprosy Lesion

To the Editor:

We have reported a case of tuberculoid leprosy in which repigmentation was accelerated by use of systemic 8-methoxypsoralen followed by exposure to sunlight (PUVASOL) ([2]).

An 18-year-old female with a tuberculoid leprosy lesion over the extensor aspect of the right elbow was treated with 600 mg of rifampin once a month for 6 months and 100 mg of dapsone daily for 2 years. Treatment was then discontinued and she was kept on 3 monthly follow up. During her second follow-up visit she complained of persistence of hypopigmentation. She was advised to take 20 mg of 8-methoxypsora-

len followed after 2 hr by exposure to sunlight for 15 min. She was unable to tolerate the treatment, and the treatment was changed to topical PUVASOL after 1 week. The 8-methoxypsoralen solution (0.75%) was applied topically, and she was advised to expose the lesion to sunlight for 2 min 1 hr after the topical application. The lesion was subsequently cleaned with soap and water.

The lesion showed mild repigmentation at the end of 1 month and significant repigmentation at the end of 3 months. Topical therapy was discontinued and pigmentation was seen to persist 3 months later. Unlike vitiligo where repigmentation is usually follicular, pigmentation was diffuse.

PUVA is an accepted mode of therapy in vitiligo and acts possibly by a) increasing the number of melanocytes, b) hypertrophy of melanocytes, c) increasing the arborization of dendrites, d) increasing the size of melanosomes, e) stimulating tyrosinase activity and promoting new tyrosinase formation, and f) enhanced migration of activated melanocytes from skin appendage ([1]). The last modality probably does not play an important role in repigmentation of tuberculoid leprosy since the lesions show alopecia and are anhydrotic. This is also possibly the reason for the pigmentation being diffuse instead of follicular, as seen in vitiligo.

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A Family with Histoid Leprosy

To the Editor:

We recently saw a family in which eight members were suffering from histoid leprosy and two had borderline tuberculoid (BT) leprosy. The occurrence of leprosy in several members of a family is not uncommon, but involvement of many members with the same type of leprosy is not usual. Moreover in this family three generations of the same family were involved. This incited us to bring this family to the attention of our colleagues working in the field of leprosy who might encounter similar cases.

The index case, a 75-year-old male, was a known case of histoid leprosy registered with our clinic at Benghazi, Libya. He had 7 sons and 5 daughters; 1 son (37 years old) and 1 daughter (age 35) had histoid leprosy. They both came voluntarily to the clinic because of their skin lesions. The son's wife was also found to have histoid leprosy, having been discovered on active clinical examination of the family contacts. A positive history of consanguinity was found between them, she being his first cousin's sister. The couple had 8 sons and 2 daughters; 2 sons aged 14 years and 9 years, respectively, were found to have histoid leprosy. On examination of the wife's other family contacts, one of her uncles (40 years old) was found to have histoid leprosy.

The daughter of the index case had 5 sons and 3 daughters; 1 son (age 14) had histoid leprosy, 2 daughters (18 and 10 years old, respectively) were found to have BT leprosy. The clinical findings were confirmed by histopathology.

The description of this family supports the view that heredity plays an important role in the transmission of leprosy. Occurrence of the same type of leprosy in many members of the same family leads one to