as the minimum phototoxic dose always induces erythema. Observation of hypertrichosis of depigmented hairs without perifollicular repigmentation and hypertrichosis of black hairs in the region surrounding the vitiligo lesion in our case question Pavithran's suggestion that the stimulus which activates melanocytes could be responsible for stimulation of the follicular matrix cells. These are two entirely different processes. In any case, it would be interesting to explore further to find out the exact mechanism involved in iatrogenic hypertrichosis, especially due to psoralens. In our case, since there is excessive growth of depigmented hairs, the term hyperleucotrichosis may be preferred as it indicates the correct picture.

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2. Singh G and Lal S : Hypertrichosis and hyperpigmentation with systemic psoralen treatment, Brit J Dermatol, 1967; 79 : 501-502.
3. Pavithran K : Hypertrichosis by topical psoralen, Ind J Dermatol Venereol Leprol, 1984; 50 : 158-160.

Reply

It is quite interesting to note the letter from Dr. RPC Naik. The statements given in the letter with reference numbers 1 and 2 have already been mentioned in my article *Hyper trichosis by topical psoralen.* I thank Dr. Naik for pointing out the mistake of writing histidine instead of thymine with which psoralen forms photo-adducts. The observation of hyperleucotrichosis without perifollicular hypermelanosis, as noted by Dr. Naik is not a point against the postulation put forward by me in the article. The number of melanocytes in the depigmented patches of vitiligo may be normal or sometimes reduced. In some patches, they may even be completely destroyed.[1] Dutta and Mandal have found a poorer repigmentation response in those patches with achromotrichia.[2] It is possible that in the case mentioned by Dr. Naik, the melanocytes in the hair follicles of the patch were destroyed by the immunological process. Hence, psoralen induced stimulus produced hyperleucotrichosis without melanin pigmentation in the patches.

I do agree with Dr. Naik that keratinisation and melanization in the hair follicle are two different processes. But the study by Ridi et al[3] strongly supports my suggestion. They demonstrated marked hyperplastic changes (increased thickness of horny layer, acanthosis and intact hair follicles) in the beta irradiated skin of rats which were fed preliminarily with 8-methoxy psoralen. In the control normal rat skin which was not given 8-MOP; after beta irradiation there was no acanthosis and hyperkeratosis and the hair follicles were destroyed. Though El Mofty has mentioned about the beneficial role of psoralen solution in the treatment of alopecia areata, he has not mentioned hypertrichosis as a complication of topical psoralen therapy of vitiligo. In alopecia areata, psoralen causes regrowth of hairs in an alopecic patch. I do not think this regrowth of hair after treatment can be named as hypertrichosis. The immunopathology of alopecia areata has recently been well studied and it has been postulated that PUVA non-specifically suppresses the immune responses against the hypothetical hair associated antigen. To the best of my knowledge hypertrichosis after topical psoralen has not been reported except the one already cited in the article. I once again thank Dr. Naik for his valuable comments.