the epidennis. This would support the hypothesis of Shiohara et al. that HLA-DR-positive keratinocytes may stimulate T cells to release additional lymphokines, such as interleukin 2, which can increase skin damage in lichenoid diseases.

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Pigmented purpura-like eruption progressing to mycosis fungoides

To the Editor: The cases of patients with a pigmented purpura-like eruption that progressed to mycosis fungoides, reported by Barnhill and Braverman (J AM ACAD DERMATOL 1988;19:25-31), is an important reminder of the need for long-term follow-up of these patients. The authors may be interested to learn that their patients are not the first with this association to be described in the American literature. Farrington1 reported the first case of lichen aureus in the American literature and subsequently corresponded with Waisman and Waisman2 that this patient with lichen aureus progressed to mycosis fungoides.

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REFERENCES

Incidence of malignancy in patients with generalized pruritus

To the Editor: With reference to the editorial by Lober on the need to evaluate patients with generalized pruritus for malignancy (J AM ACAD DERMATOL 1988;19:350-2), I report our data from the Department of Dermatology at the University of Bari, Italy. Of 447 patients with idiopathic pruritus observed for a period of 18 years, malignancy developed in only 3 (one case each of Hodgkin's disease, lymphoblastic lymphoma, and laryngeal cancer). Therefore the incidence of malignancy in our patients despite thorough investigation is considerably lower than that reported by Lober.

Franco Rantuccio, MD
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Reply

To the Editor: I have reviewed Dr. Rantuccio's letter concerning my editorial (J AM ACAD DERMATOL 1988;19:350-2) and have found that his series of 447 patients is, to the best of my knowledge, the largest such group reported in the literature to date. It is worth noting that the incidence of malignancy in his series was less than 1% and that in two of the three patients in whom malignancy developed, the malignancies were hematopoietic.

Additional details of his study would be valuable to know. Were the patients studied prospectively or retrospectively? What were the criteria for inclusion of a patient in the study? Exactly what did Rantuccio's "thorough investigation" entail? Did any patients who entered the study group drop out because of death or other reasons? Before we can fully evaluate the merit of his study, these and other questions must be answered. I encourage Dr. Rantuccio to report his findings more completely.

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Audiologic disturbances in vitiligo

To the Editor: The interesting article by Tosti et al. (J AM ACAD DERMATOL 1987;17:230-3) led us to search for possible sensorial hearing defects in our group of patients with vitiligo. Human melanocytes, whose embryonic origin is from the neural crest, are located in various sites: epidermis, hair follicles, dermis, mucous membranes, leptomeninges, eye (uveal tract and retinal pigment epithelium), and inner ear (in the cochlea, wall of the modiolus, spiral lamina, Reissner's membrane, stria vascularis; in the vestibular system, sacculus, utricle, ampullae).

The function of the skin melanin is well documented; it acts as a filter to protect cellular DNA from injurious solar ultraviolet radiation. In extracutaneous tissues (except in the eye, where it plays an important role in the normal function of neural retina4) its function is unclear. Melanocytes in the inner ear may prevent damage to the neuroepithelium caused by environmental factors.5,6 Such damage might result in hearing loss.

Vitiligo may be accompanied by ocular disturbances7-8 as well as by auditory abnormalities.9,10 The pathogenesis of these associated defects, which could indicate that vitiligo is a systemic disease of melanocytes, is unknown. Direct evidence of melanocyte alterations in the inner ear in vitiligo patients has not been reported. If melanocytes of the inner ear do in fact function to prevent
Table I. Features of four vitiligo patients with hypoacusis

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)/Sex</th>
<th>Duration (yr)</th>
<th>Extent</th>
<th>Ear affected</th>
<th>Frequency (Hz)</th>
<th>Hearing loss (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15/F</td>
<td>4</td>
<td>General</td>
<td>Right</td>
<td>4000</td>
<td>55</td>
</tr>
<tr>
<td>2</td>
<td>21/F</td>
<td>15</td>
<td>General</td>
<td>Both</td>
<td>4000 (right), 4000 (left)</td>
<td>35 (right), 50 (left)</td>
</tr>
<tr>
<td>3</td>
<td>23/F</td>
<td>9</td>
<td>General</td>
<td>Both</td>
<td>2000</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>41/M</td>
<td>5</td>
<td>Acral</td>
<td>Both</td>
<td>4000 (right), 4000 (left)</td>
<td>60 (right), 40 (left)</td>
</tr>
</tbody>
</table>

hearing loss, their possible involvement in vitiligo may be evidenced by audiometric analysis.

We searched for an audiologic defect in a group of 50 patients with vitiligo. They were selected from 93 vitiligo outpatients. Subjects older than 50 years or those exposed to factors known to cause hypoacusis were excluded, as were those affected by congenital hypoacusis or other auditory diseases. The group included 24 male and 26 female patients, aged from 10 to 50 years, (median 33.28 years). Duration of disease ranged from 1 to 37 years, (median 11.48 years). Forty patients were affected by generalized vitiligo, five by localized vitiligo, and five by acral vitiligo. All had normal results of biohumoral examinations. To perform the audiometric test, we used an audiometer (Amplaid 208, Amplus Corp., Boulder, Colo.), and the test was performed in a silent cabin (Ampliphon type G5, Milan, Italy). The pure-tone thresholds were estimated for each ear at the frequencies of 125 to 8000 Hz for air conduction and 250 to 4000 Hz for bone conduction. Fifty healthy persons without vitiligo were matched for sex and age and were chosen as controls with the use of the exclusion criteria described herein.

In our study four (8%) vitiligo patients and one (2%) control subject showed minimal audiometric alterations (Table I). Three patients with hypoacusis and vitiligo had generalized vitiligo; one had acral vitiligo, and five by acral vitiligo. All had normal results of biohumoral examinations. To perform the audiometric test, we used an audiometer (Amplaid 208, Amplus Corp., Boulder, Colo.), and the test was performed in a silent cabin (Ampliphon type G5, Milan, Italy). The pure-tone thresholds were estimated for each ear at the frequencies of 125 to 8000 Hz for air conduction and 250 to 4000 Hz for bone conduction. Fifty healthy persons without vitiligo were matched for sex and age and were chosen as controls with the use of the exclusion criteria described herein.

In our study four (8%) vitiligo patients and one (2%) control subject showed minimal audiometric alterations (Table I). Three patients with hypoacusis and vitiligo had generalized vitiligo; one had acral vitiligo; three were females; one was male; three were younger than 25 years of age; one was 41 years old. Statistical analysis showed that the result was not significant when compared with the control group (p > 0.05).

Our results contrast with the previous report 10 and suggest that there is no proof of involvement of ear melanocytes in vitiligo. Even if melanocytes were damaged, hearing function may not be compromised. Thus the role of melanocytes in the inner ear is unclear.

Although ocular disturbances are well documented in vitiligo, audiologic abnormalities are not to be considered an aspect of the disease.

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REFERENCES

Severe reaction to diphenhydramine

To the Editor: The excellent CME article by Grekin and Auletta, “Local Anesthesia in Dermatologic Surgery” (J AM ACAD DERMATOL 1988;19:599-614), contains one point that deserves comment. Diphenhydramine at a concentration of 50 mg/ml is recommended as an alter­native to lidocaine for local anesthesia in patients allergic to lidocaine. I report a severe reaction presumably trig­gered by diphenhydramine used in this manner.

Case report. A 45-year-old woman had a subungual tumor of