or UVB pretreatment (UVB + IPL vs UVB only; 1.13-fold decrease; Fig 1, *B*). Thus, IPL treatment appears to be able to modify UVB-induced activation of AP-1 transcription.

We next investigated whether IPL treatment may also alter the expression of MMP-1 in vitro. As shown in Fig 1, *C*, MMP-1 expression by fibroblasts was increased after UVB treatment (1.56-fold, compared with control), a finding that is in accordance with previous reports,[1,2] while IPL treatment decreased the MMP-1 expression level by 11.47-fold (vs control), which is conistent with the report by Luo et al.[4]

In summary, we have provided limited evidence for the possible molecular mechanisms that govern the photorejuvenation effect of IPL treatment by demonstrating that IPL may down-regulate the AP-1 expression enhanced by UVB. More investigation will be necessary to solidify the relevance of IPL's influence on AP-1 expression to downstream signaling events and its photorejuvenation effects.

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Melanocytes: A possible autoimmune target in alopecia areata

To the Editor: Alopecia areata (AA) is thought to be an autoimmune disease directed against the hair follicle that results in hair loss. Observationally, AA tends to respond to immunosuppressive therapy and occurs in patients with other autoimmune phenomena, such as autoimmune thyroiditis and vitiligo. Further support comes from animal models. In a severe combined immunodeficiency (SCID) mouse model (used because these mice will not reject human lymphocytes or skin), investigators have shown that as T lymphocytes disappear from engrafted AA-affected skin, hair growth ensues.[1] Moreover, if T cells from AA patients are stimulated in vitro with hair follicle antigens and then injected into the engrafted skin on the immunodeficient mice, immunologic hair loss similar to that seen in AA ensues.[2] More specifically, incubating T cells with only melanocytes from hair follicles from patients with AA will result in hair loss in the SCID mouse model.[3] Both $CD4^+$ and $CD8^+$ T cells have been reported to be active in this process.[2] Even though an autoimmune process is likely the pathogenic mechanism of AA, the exact autoimmune target antigen has yet to be defined. Given the clinical observation that AA seems to target pigmented hairs more then lighter or graying hairs and that nonpigmented hairs regrow first in areas of alopecia, melanocytes and melanocytic proteins have been investigated as potential autoantigens.

We sought to strengthen the evidence that the melanocyte may be one of the autoimmune targets in AA by using immunoperoxidase stains to assess melanocyte density in patients with active disease. Scalp samples were obtained from clinically active areas of 18 patients with AA and stained with MART-1. Microphthalmia transcription factor was also used, but proved less reliable because of nonspecific nuclear staining of follicular epithelium. The number of melanocytes present and the presence or absence of dendritic processes was compared to five control subjects. Samples were given scores from trace to 3+ depending on the number of melanocytes identified. Thirteen of 18 patients (72%) had only trace to 1+ staining for melanocytes, usually with limited dendritic processes (Fig 1). Three of 18 had 2+ staining, and two had 3+ staining. Melanocytes were identified in follicular epithelium at all phases (anagen, telogen, and catagen). A 3+ level of staining was present in all control specimens (5/5) (Fig 2). Inflammation in the AA patients ranged from early peribulbar

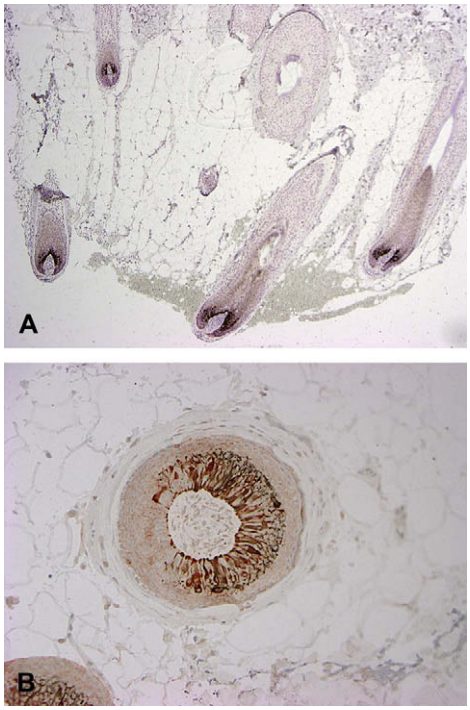

Fig 1. Melan-A immunoperoxidase stains, normal scalp controls. Note the intense staining of melanocytes directly above the dermal papilla. (Original magnification: **A**, ×40; **B**, ×200.)

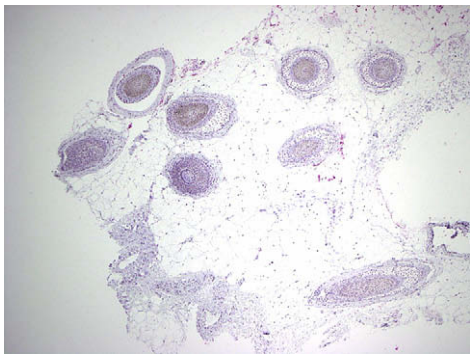

Fig 2. Melan-A immunoperoxidase stains, alopecia areata patient. Note the paucity of staining for melanocytes. Melanocytes were present in the interfollicular epidermal basal layer. (Original magnification: ×40.)

inflammation to end stage with minimal inflammation, and the results were similar regardless of the stage of disease.

Our study shows there are a decreased number of follicular melanocytes in AA. The study does not clarify whether melanocytes are decreased in number because of autoimmune attack or because of rapid hair cycling. Further research will focus on making this distinction.

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Instilled bimatoprost ophthalmic solution in patients with eyelash alopecia areata

To the Editor: No treatments exist to stimulate the regrowth of eyelashes in patients with alopecia areata (AA). Bimatoprost (Lumigan; Allergan, Inc, Irvine, CA), a prostanoid F2alfa receptor agonist, is well established as an intraocular pressure (IOP)—lowering agent for glaucoma. Glaucoma patients treated with bimatoprost[1,2] have noticed longer, darker, and thicker eyelashes. This raises the question whether bimatoprost can also promote eyelash growth in patients with AA. One patient with AA was reported to grow eyelashes after cutaneous eyelid application of latanoprost, a prostaglandin analogue.[3] The objective of our 16-week, open-label, prospective study was to assess the safety and efficacy of instilled bimatoprost ophthalmic solution in promoting eyelash growth in patients with AA.

Patients 18 years of age or older with AA were eligible to participate if they had at least 50% bilateral eyelash loss for a period of longer than 6 months (Table I). Exclusion criteria included