transaminases, fatigue, and acne. None required treatment cessation or dose modification.

BAA is associated with a high prevalence of anxiety and depressive symptoms.[1-3] Some religions, including Islam, Orthodox Judaism, and Sikhism, require men to grow a full beard.[3] One of our patients regularly competes in beard championships, and the loss of more than 50% of his beard as a result of BAA caused significant distress (Supplemental Figure 1; available at https://data.mendeley.com/datasets/nh2h672rhc/1).

Our results show a strong correlation between the extent of beard and scalp hair regrowth, suggesting that these hair-bearing sites may respond similarly to oral tofacitinib. In contrast to studies of SAA that showed an inverse association between disease duration and treatment response,[4,5] no such association was observed in our BAA cohort. On the contrary, patients with complete beard regrowth had a longer mean disease duration. Patient age, BAA phenotype (patchy or total beard loss), and duration of treatment did not influence the degree of beard response. As with our BAA cohort, age did not predict improvement in Severity of Alopecia Tool score in 90 patients with severe SAA treated with oral tofacitinib.[4]

Our findings suggest that oral tofacitinib might be a promising therapeutic option for patients with BAA. We suggest that prospective clinical trials evaluating oral Janus kinase inhibitors in alopecia areata should seek to include beard assessment in their protocols to determine their effectiveness for the treatment of BAA.

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The correlation of immune status with ultraviolet radiation–associated mutations in cutaneous squamous cell carcinoma: A case-control study

To the Editor: There are an estimated 1 million cases of cutaneous squamous cell carcinoma (SCC) per year in the United States.[1] Ultraviolet (UV) radiation contributes to the pathogenesis of SCC and causes characteristic pyrimidine-pyrimidine dimer mutations.[2] Pickering et al[3] performed whole-exome sequencing of 39 aggressive SCCs and found 65% of mutations to be UVB associated. In addition to UV radiation, immunosuppression increases SCC risk. Patients with organ transplants are 100 times more likely to develop SCC secondary to underlying immunosuppression and toxicity from chemotherapy.[4] However, the pathogenesis of SCC in immunocompromised patients remains to be fully elucidated. We hypothesized that there are fewer UVB-associated mutations in immunocompromised patients than in immunocompetent patients with SCC.

We performed next-generation sequencing using a hotspot mutation panel covering 76 cancer-associated genes (Vela Diagnostics, Singapore) in a cohort of 20 patients with high-risk SCC (Table I). We categorized mutations as being caused by UVA radiation, UVB radiation, reactive oxygen species

Table I. Patient and tumor characteristics by immune status

Status	Average age, y	Sex (n)	Location (n)	AJCC 8 stage (n)	BWH stage (n)	Immunosuppression (n)	Metastasis, % (n/total)
Immunocompetent (n = 12)	70.7	Male (10), female (2)	Cheek (1), ear (5), extremities (1), maxillary/ orbital area (1), nose (1), scalp (2), supraorbital (1)	T1 (2), T3 (6), T4a (2), T4b (2)	T1 (2), T2a (3), T2b (1), T3 (6)	None	50 (6/12)
Immunocompromised (n = 8)	64.4	Male (6), female (2)	Cheek (1), ear (2), extremities (1), eyelid (1), neck (1), scalp (2)	T3 (8)	T2a (3), T2b (3), T3 (2)	Solid organ transplant (5), hematologic malignancy (2), yes: unknown (1)	50 (4/8)

AJCC 8, American Joint Committee on Cancer Staging, eighth edition; *BWH*, Brigham and Women's Hospital.

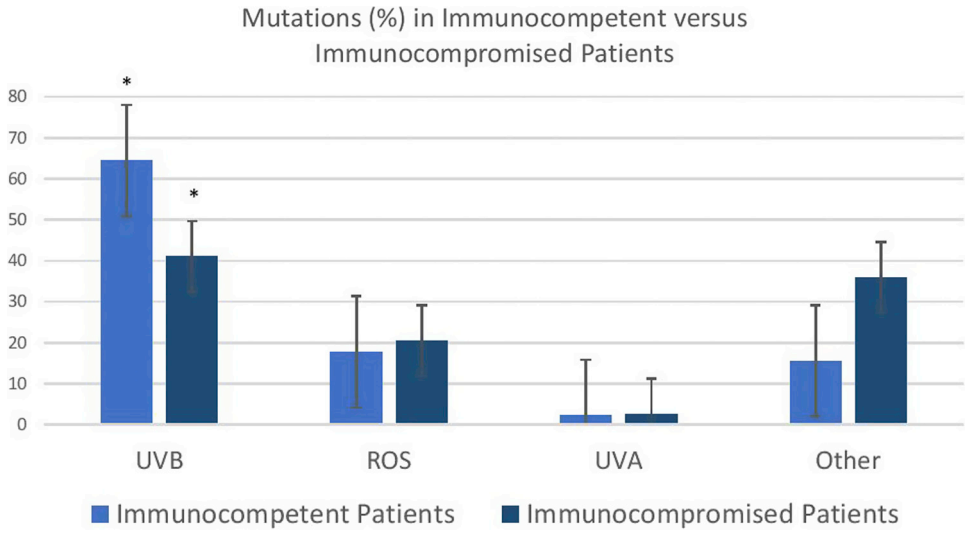

Fig 1. The distribution of mutation types by immune status.

(ROS) (thought to be secondary to UVA damage, likely due to deeper penetration in the skin), or other based on methods by Agar et al.[5] Exploring the pathogenesis of SCC development by stratifying for UVA, UVB, and ROS mutations provides insight into the mechanism of SCC development in immunosuppressed patients.

We found that 64.4% of mutations in immunocompetent patients were UVB associated, consistent with the literature (Fig 1). However, UVB mutations accounted for only 41.0% of mutations in immunocompromised patients; this was significantly different ($P = .04$) (Fig 1). In contrast to the literature suggesting that fewer mutations in SCCs arise in immunocompromised patients, the number of mutations per patient was not significantly different between immunocompetent and immunocompromised patients (3.75 vs 4.88, respectively; $P > .05$). This may be due to the use of a targeted panel with a limited number of genes. In a separate analysis, the percentage of UV mutations was also examined for tumors in different anatomic locations (Supplemental Table I; available at https://doi.org/10.17632/gv9yh6nk5r.1). The proportion of both UVB and UVA/ROS mutations was significantly different based on tumor location, with tumors in high-risk areas (ear, lip, periorbital region, and nose; n = 11) having more UVA/ROS mutations (30.77% vs 11.62%, $P = .03$) and fewer UVB mutations (41.03% vs 67.44%, $P = .02$) than tumors in medium- and low-risk area (n = 9).

These findings affirm our hypothesis that UVB radiation may contribute less to the pathogenesis of SCC in immunocompromised patients. Perhaps less

UV radiation is required to be carcinogenic in immunocompromised patients secondary to their underlying immunosuppression. The literature surrounding SCC in immunocompromised patients also points to a permissive microenvironment to explain these findings. Furthermore, the significantly higher contribution of UVA and ROS and lesser contribution of UVB to SCCs in high-risk areas of the face may be due to the thinner epidermal layer and shorter distance that UVA has to travel to become carcinogenic. Because these anatomic areas are somewhat equally sun exposed, there also appears to be an inherent risk in certain locations due to patterns of vascularization and lymphatic drainage. Further studies are warranted to examine additional mutational differences between SCC in these groups of patients.

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Patients with hidradenitis suppurativa may suffer from neuropathic pain: A Finnish multicenter study

To the Editor: Patients with hidradenitis suppurativa (HS) have a diminished quality of life (QoL), and pain being a major contributor to poor QoL.[1,2] A recent study reported that pain was the only contributor for decreased QoL if the severity of disease was excluded.[3] Despite the immense impact of pain in patients with HS, there is lack of studies that more closely analyze the pain in these patients.

This multicenter study was conducted in Finland. Patients who were diagnosed with HS at least 6 months before the study period were retrospectively identified. Pain intensity and type were evaluated during the study visit using the visual analog scale (VAS) and the pain*DETECT* questionnaire. The Dermatology Life Quality Index questionnaire and Beck's Depression Inventory were used to evaluate the patients' QoL and the severity of depression. Methods are described in detail in additional materials (available at Mendeley (DOI:10.17632/rrw9wmmnyb.2).

The study included 92 patients. Patient characteristics are presented in Table S1 (available at Mendeley, DOI:10.17632/rrw9wmmnyb.2). In pain*DETECT*, 31.5% of patients were defined as having suspicion of neuropathic pain (NeP, "NeP positive"), 41.3% as having no NeP ("NeP negative"), and 27.2% were classified as having unclear results (Table I). Most patients reporting moderate to severe pain by VAS were also NeP positive (Table I). The percentage of patients in different pain groups stratified by disease severity is provided in Table I.

Patients reporting NeP had more psychiatric comorbidities (13 of 29 [44.8%]), such as depression and sleep disorders, compared with patients in the NeP-negative (9 of 38 [23.7%]) or NeP-unclear (6 of 25 [24.0%]) groups, but this finding was not statistically significant. No other differences were found