gender differences suggests similar levels of research productivity at each rank. Nevertheless, despite increasing numbers of women entering dermatology and more female professors at all levels,[1] there are still proportionately fewer female full professors.

The interpretation of these data is not straightforward. It could be attributed to factors such as shifting demographics in dermatology, academic culture, an increased preference for education-focused tracks, mentoring opportunities, and potential gender biases.[3,4] As more women enter the field of academic dermatology, it may be a matter of time before greater numbers of female assistant professors translate to greater numbers of senior female faculty. At the same time, implicit biases disfavoring female academic physicians have been suggested: women report greater difficulty in finding suitable mentors and a reduced sense of belonging in academia.[4]

In this first in-depth analysis of bibliometric indicators, we found that male and female professors of dermatology are achieving similar levels of academic productivity—yet there is lower female representation within the higher academic ranks. With greater numbers of female assistant professors, continuing to enable senior professors to serve as mentors with protected meeting times and mentoring awards may provide support.[5]

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Alopecia areata and poliosis: A retrospective analysis of 258 cases

To the Editor: It is not uncommon to observe white hairs regrown at previous patches of alopecia areata (AA) (Fig 1). However, studies of poliosis in AA are mostly limited to case reports about total or partial whitening with pigmented hair loss. In this study, we aimed to characterize AA patients with poliosis by comparing clinical parameters of AA cases with and without poliosis.

Medical records of 258 patients with AA who visited Wonju Severance Christian Hospital during March 2012-June 2017 were retrospectively assessed. Patients were subdivided into poliosis and

Fig 1. Poliosis in alopecia areata recovery. Multiple alopecia patches in occipital scalp are filled with white hairs.

Table I. Clinical comparison of alopecia areata patients (N = 258) with and without poliosis

Characteristic	Poliosis, n = 13	Nonpoliosis, n = 245	*P* value*	Adjusted *P* value†
Age, y, mean ± SD	47.2 ± 12.4	36.7 ± 16.4	.018	.234
Sex (M:F)				
M, n (%)	8 (61.5)	118 (48.2)	.403	
F, n (%)	5 (38.5)	127 (51.8)		
Onset age, y, mean ± SD	43.2 ± 12.7	35.1 ± 16.6	.058	
Duration, month, mean ± SD	48.9 ± 87.0	20.9 ± 42.1	.288	
Smoking, pack-years, mean ± SD	9.2 ± 11.8	3.8 ± 8.45	.162	
Drinking, bottle-years, mean ± SD	16.2 ± 42.2	115. ± 37.60	.918	
Initial or recurrent, n (%)				
First episode	9 (69.2)	189 (77.1)	.507	
Recurrent	4 (30.8)	56 (22.9)		
Body mass index, kg/m^2, mean ± SD	24.7 ± 5.4	22.5 ± 3.69	.309	
SALT score, n (%)				
≤25	5 (38.5)	178 (73.0)	**.025**	.374
26-50	5 (38.5)	41 (16.7)		
51-75	3 (23.1)	18 (7.3)		
76-100	0 (0)	8 (3.3)		
Comorbidities, n (%)				
Diabetes mellitus	1 (7.7)	3 (1.2)	.188	
Hypertension	3 (23.1)	12 (4.9)	**.032**	**.038**
Dyslipidemia	1 (7.7)	10 (4.1)	.440	
Others‡	3 (23.1)	83 (33.9)	.535	
Alopecia areata family history, n (%)				
Paternal	0 (0)	9 (3.7)	1.000	
Maternal	1 (7.7)	9 (3.7)	.409	
Siblings	1 (7.7)	16 (6.5)	.597	
Offspring	1 (7.7)	1 (0.4)	.098	
Treatment history, n (%)				
Diphenylcyclopropenone	9 (69.2)	91 (37.1)	**.023**	.091
Superficial cryotherapy	3 (23.1)	111 (45.3)	.097	
Oral corticosteroid	0 (0)	18 (7.3)	.382	

Significant values are bolded.
SALT, Severity of Alopecia Tool; *SD*, standard deviation.
*The chi-squared test was used for all comparisons of noncontinuous data (sex, recurrence, SALT score, comorbidities, alopecia areata family history and treatment history) and the Mann-Whitney U test was used for continuous data (age, onset age, duration, smoking history, drinking history, and body mass index).
†Adjusted for age, SALT score, comorbidity of hypertension, and diphenylcyclopropenone treatment history.
‡Other comorbidities include: allergic contact dermatitis, atopic dermatitis, rhinitis, asthma, and urticaria.

nonpoliosis groups and demographic variables (age, sex, body mass index, smoking, and alcohol use); comorbidities; and disease-specific variables (age at onset, duration, recurrence, extent, family history of AA, and treatment history) were compared between the 2 groups.

Of the 258 patients, 13 (5.04%) had poliosis. The average age of the poliosis group was higher than that of nonpoliosis group, and the proportion of patients treated with diphenylcyclopropenone and the proportion of patients with less extensive lesions were higher in the poliosis group than in the nonpolilosis group. However, in the multivariate logistic regression analysis, only hypertension was significantly associated with poliosis (Table I).

Melanocyte-associated T-cell epitopes have been known to act as an autoantigen in AA. White hair is thought to be caused by the interaction of various mechanisms, including dysregulation of signaling pathways and transcription factors in the microenvironment surrounding the hair follicles, including melanocyte stem cells, or by an imbalance in the management of oxidative stress.[1] Thus, poliosis might be promoted in elderly individuals with poor melanocyte reservoir status in AA.

Furthermore, previous studies on the role of the signaling pathway known to be involved in both follicular melanogenesis[2,3] and vascular disease[4,5] might provide clues to the relationship between poliosis and hypertension demonstrated in this study.

There are some limitations to our study, including the use of a small number of patients at a single institution. In addition, because this study was conducted in a specific population, it is necessary to consider genetic differences in the prevalence of AA and comorbidities according to ethnicity. Further studies to evaluate the association of poliosis with treatment or prognosis in other populations and with a larger sample size are needed.

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Response to tofacitinib therapy of eyebrows and eyelashes in alopecia areata

To the Editor: Alopecia areata (AA) can affect any hair-bearing site. The eyebrows, eyelashes, or both are involved in 76% of patients with AA (L. Y. Liu, B. A. King, J. M. Ko, unpublished survey data, 2019). While Janus kinase (JAK) inhibitors have emerged as targeted treatment for AA, studies have focused on scalp hair growth.[1-3] In this study, we evaluated the response of eyebrows and eyelashes in patients with AA treated with the JAK inhibitor tofacitinib.

Table I. Patient demographic and clinical characteristics

Characteristic	Value, N = 119
Sex	
Male	65 (54.6)
Female	54 (45.4)
Age, y, mean ± SD	35.2 ± 15.3
Age <18 y	9 (7.6)
AA with subtotal scalp hair loss	21 (17.6)
AA with scalp, eyebrow involvement but no eyelash involvement	3 (2.5)
AA with scalp, eyelash involvement but no eyebrow involvement	3 (2.5)
AA with scalp, eyelash and eyebrow involvement	15 (12.6)
AA with total scalp hair loss	98 (82.4)
AA with scalp (total), eyebrow involvement but no eyelash involvement	11 (9.2)
AA with scalp (total), eyelash involvement but no eyebrow involvement	1 (0.8)
AA with scalp (total), eyelash and eyebrow involvement	86 (72.3)
Duration of current episode of AA with total scalp hair loss, y, mean ± SD	5.8 ± 6.6
Duration of tofacitinib treatment, mon, mean ± SD	20.8 ± 10.6

Values are n (%), unless otherwise indicated.
AA, Alopecia areata; *SD*, standard deviation.

Records of all patients treated with tofacitinib and evaluated by Dr King from January 2014-April 2018 were identified. The inclusion criteria for this study were AA diagnosis with scalp involvement and eyebrow or eyelash involvement and treatment with tofacitinib for ≥6 months. Treatment responses (absent, partial, or complete) were documented. Multivariate logistic regression models and Kruskal-Wallis rank sum tests were conducted in R statistical package software.

We identified 119 patients (Table I). Of 98 patients with total scalp hair loss, 86 had involvement of both eyebrows and eyelashes. Complete regrowth at all sites was achieved in 16% (19/119) of patients, and complete regrowth of eyebrows and eyelashes and partial growth of scalp hair was achieved in 16% (19/119) of patients (Table II). Complete regrowth of eyebrows was achieved in 34% (41/119) of patients. Out of 21 patients who received intralesional triamcinolone to the eyebrows, 14% (3/21) achieved complete regrowth. Complete regrowth of eyelashes was achieved in 39% (46/119) of patients. Eyelashes