number T32-AR-007465 and receives partial salary support through a Pfizer Fellowship grant to the Trustees of the University of Pennsylvania.

Conflicts of interest: None disclosed.

Reprints not available from the authors.

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https://doi.org/10.1016/j.jaad.2019.05.091

Increased risk of alopecia areata for people with hidradenitis suppurativa in a cross-sectional study

To the Editor: Hidradenitis suppurativa (HS) is a chronic inflammatory disease with hair follicle abnormalities and associations with autoimmune and inflammatory diseases[1]; alopecia areata (AA) is an autoimmune disorder of the hair follicle.[2] Because both disorders involve hair follicles, understanding the relationship between these 2 diseases may help elucidate the pathogenesis of each. In a Korean study, AA was more common in patients with HS than in patients without HS (adjusted odds ratio, 1.35).[3] Because HS is distinctly different in the Asian population, we investigated the relationship between HS and AA in a US sample.

A cross-sectional study was performed by using the MarketScan Commercial Claims database (Truven Health Analytics, Ann Arbor, MI). No institutional review board approval was needed. Inclusion criteria were age 12 years and older with continuous enrollment for the period of January 1, 2005, through December 31, 2014. The HS group included individuals with 2 separate HS-related claims in an 18-month period. A control group was created by randomly selecting individuals (10:1) who were never diagnosed with HS, pilonidal cyst, acne conglobata, or dissecting scalp cellulitis over the same interval after matching for age and sex. Risk ratios (RRs) were investigated for any alopecia and then specifically for AA and lichen planopilaris. Potential confounding factors, including hypothyroidism and Down syndrome, were identified and included in the chi-squared tests. RRs and confidence intervals were calculated by using Altman's methods and significance was set at P of .05 or less.

Table I. Characteristics of the study groups

Patient characteristics	HS group (n = 3 645)	Control group (n = 36 450)
Age, mean ± SD	34.4 ± 14.2	36.8 ± 13.4
Female sex, n (%)	2789 (76.52)	27 294 (74.88)
Urban/suburban, n (%)	3064 (84.07)	29 783 (81.71)
Region, n (%)		
Northeast	382 (10.48)	4312 (11.83)
North Central	862 (23.65)	9386 (25.75)
South	2148 (58.93)	19 041 (52.24)
West	232 (6.36)	3273 (8.98)
Unknown	21 (0.58)	437 (1.20)

SD, Standard deviation.

A total of 40 095 patients were identified: 3645 patients with HS and 36 450 matched control individuals (Table I). When compared with the control group, the HS group had significantly higher risk of any AA (RR = 2.22) including alopecia totalis or universalis (RR = 2.17) (Table II). We also identified an increased risk of lichen planopilaris (RR = 1.54). HS was also associated with hypothyroidism (RR = 1.17) and Down syndrome (RR = 11.46); however, after controlling for these potential confounding factors, the relative risk for AA was still elevated (RR = 2.09; 95% confidence interval, 1.69-3.20; $P < .001$). The findings of this study are limited by a lack of clinical outcome data, temporal association, and detection bias for skin conditions.

Our findings are consistent with those of Lee et al[3] and showed an increased risk of AA in a US-based HS sample. As currently understood, AA is caused by the loss of immune privilege at the hair follicle either because of local hair follicle epithelium disruption or a dysregulated immune response.[2] HS pathogenesis involves hair follicle disruption and a sustained dysregulated immune response.[4] Both AA and HS likely involve an inciting event at the hair follicle that leads to subsequent inflammation. In support of this hypothesis, the lesions of patients with HS and AA have considerable overlap in the expression of key inflammatory cytokines, including tumor necrosis

Table II. Relative risk of selected conditions for individuals with HS versus those without HS

Diagnosis	ICD-9 codes	HS group (n = 3645), n (%)	Control group (n = 36 450), n (%)	Relative risk (95% CI)	*P* value
Any type of alopecia areata	704.01	47 (1.29)	213 (0.58)	1.99 (1.61-3.03)	<.001
Alopecia capitis totalis/universalis	704.09	28 (0.77)	129 (0.35)	2.17 (1.44-3.27)	<.001
Lichen planopilaris	697.0	33 (0.91)	214 (0.59)	1.54 (1.07-2.23)	.021
Hypothyroidism	244.9	751 (20.6)	6425 (17.63)	1.17 (1.08-1.27)	<.001
Down syndrome	758.0	47 (1.29)	41 (0.11)	11.46 (7.53-17.45)	<.001

CI, Confidence interval; *ICD-9*, International Classification of Diseases, 9th revision.

factor α, interleukin 17, interferons, chemokine ligands 9 and 10, granzyme B, and others.[4,5] The similarities in the gene expression profiles of patients with HS and AA may explain why AA is more common in patients with HS. The results of this study encourage more research to investigate the molecular, genetic, and environmental factors contributing to this increased risk relationship between AA and HS. Clinicians should also be aware of the increased risk of AA in people with HS.

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Funding sources: None.

Disclosure: Dr Kirby received honoraria from AbbVie and led sponsored studies for AbbVie, Incyte, InflaRx, and UCB. She is also a consultant for Incyte and ChemoCentryx. Drs Horissian, Maczuga, and Nelson have no conflicts of interest to declare.

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https://doi.org/10.1016/j.jaad.2019.05.093

Publication productivity of authors of psoriasis clinical practice guidelines with and without ties to industry

To the Editor: A recent Journal of the American Academy of Dermatology article explored whether excluding experts with ties to industry might limit the expertise of clinical practice guideline (CPG) panels.[1] The study compared publication productivity, a surrogate for expertise, between authors of CPGs with and without industry ties.[1] Authors of CPGs without industry ties reportedly had comparable levels of publication productivity as authors with industry ties.[1] This result seemed surprising. We expected that for at least psoriasis, a condition for which treatments (a priority of CPG development) have evolved rapidly, authors with industry ties would have greater expertise (with their greater participation in industry-funded clinical trials of new drugs). We tested this hypothesis using the publication productivity method.

Psoriasis CPGs from the American Academy of Dermatology, National Psoriasis Foundation, and British Association of Dermatology were analyzed, and all listed authors were categorized as either authors with or without self-reported industry ties. For members of the American Academy of Dermatology and National Psoriasis Foundation CPGs, the ties to industry were also assessed by using the OpenPaymentsData.cms.gov website. Publication productivity was determined by calculating an author's h-index and g-index using Harzing's Publish or Perish software for the year in which the CPG was published and for years 1945-2018, excluding guideline publications from