Racial characteristics of alopecia areata in the United States

Hemin Lee, MD, MPH,[a] Sun Jae Jung, MD, PhD,[b,c] Anisha B. Patel, MD,[d,e] Jordan M. Thompson, MD,[f] Abrar Qureshi, MD, MPH,[f,g,h] and Eunyoung Cho, ScD[f,g,h]
Boston, Massachusetts; Seoul, Korea; Houston, Texas; and Providence, Rhode Island

Background: Epidemiologic studies on the association between race and alopecia areata (AA) are limited.

Objective: To characterize racial differences of AA in the United States.

Methods: Cross-sectional study of self-registered AA patients and noncases in the National Alopecia Areata Registry (NAAR). We evaluated odds of AA and its subtypes for 5 ethnic/racial groups using logistic regression. A sex-stratified analysis and a sensitivity analysis among dermatologist-confirmed cases were also performed.

Results: We identified 9340 AA patients and 2064 noncases. Compared with whites, African Americans had greater odds of AA (odds ratio, 1.77; 95% confidence interval, 1.37-2.28) and Asians had lower odds (odds ratio, 0.40; 95% confidence interval, 0.32-0.50) of AA. The results were consistent in AA subtypes, dermatologist-confirmed cases, and by sex.

Limitations: Residual confounding due to limited number of covariates. Recall or recruitment bias not representative of the entire disease spectrum. Also, outcome misclassification was possible because not all AA cases in the registry were confirmed by dermatologists.

Conclusion: Our findings suggest higher odds of AA in African Americans and lower odds in Asians compared with whites. Future studies examining racial disparity in AA from clinical and genetic perspectives are warranted for a better understanding of the disease pathogenesis. (J Am Acad Dermatol 2020;83:1064-70.)

Key words: alopecia areata; epidemiology; hair disorders; National Alopecia Areata Registry.

Alopecia areata (AA) is a nonscarring hair loss disease characterized by T cell—mediated autoimmunity targeting the hair follicles. The severity of AA ranges from solitary, isolated, coin-sized patches to recurrent patches termed alopecia areata transient (AAT) or alopecia areata persistent (AAP), depending on disease duration.[1] Severe forms of AA can involve the whole scalp as alopecia totalis (AT) or the whole scalp plus body hair as alopecia universalis (AU).

Although most small, solitary alopecia patches resolve spontaneously, widespread and recurrent

From the Division of Pharmacoepidemiology and Pharmacoeconomics, Department of Medicine, Brigham and Women's Hospital, Boston[a]; the Department of Preventive Medicine, Yonsei University College of Medicine, Seoul[b]; the Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston[c]; the Dermatology Department, McGovern Medical School, The University of Texas Health Science Center at Houston[d]; the Department of Pathology, The University of Texas MD Anderson Cancer Center, Houston[e]; the Department of Dermatology, The Warren Alpert Medical School, Brown University, Providence[f]; the Channing Division of Network Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston[g]; and the Department of Epidemiology, Brown University School of Public Health, Providence.[h]

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Reprint requests: Eunyoung Cho, ScD, Box G-D, Department of Dermatology, Warren Alpert Medical School, Brown University, Providence, RI 02912. E-mail: eunyoung_cho@brown.edu.

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cases are often refractory to treatment. Thus, treatment presents numerous challenges in these patients, who often experience considerable physical and psychologic burden of disease. In fact, AA patients report lower health-related quality of life scores compared with patients with other skin conditions, including psoriasis or atopic dermatitis, highlighting the necessity for further research into this complex disease.[2,3]

Currently, little evidence exists on the epidemiology of AA patients in the United States. The first National Health and Nutritional Examination Survey in 1971 to 1974 reported the lifetime risk of AA as 1.7%,[4] and the Rochester Epidemiology Project reported the lifetime AA incidence rate as 1.7% (years 1975-1989)[5] and 2.1% (years 1990-2009).[6] Conventional views held that AA affects both sexes and all race equally,[7] and there have been no genetic or population-level studies stating otherwise. However, in a recent study of AA patients in the Nurses' Health Study (NHS) and Nurses' Health Study II (NHSII)—2 prospective studies of United States women—African American women had higher risk of AA than white women.[8] In the NHS study, AA cases consisted only of self-reported patients, and no information was available on men. Therefore, to better understand racial disparity of AA in the United States, we analyzed cross-sectional data in the National Alopecia Areata Registry (NAAR) and compared the risk of AA between cases and noncases in 5 racial/ethnic groups.

CAPSULE SUMMARY

- This study suggests that African Americans have higher odds of alopecia areata compared with whites and that Asians have lower odds of the disease.
- Physicians should be aware of possible racial disparities in alopecia areata. Screening or diagnostic approaches may need to be adjusted for high-risk racial groups.

METHODS

Data source

We analyzed the 2002-2016 NAAR data provided by the National Alopecia Areata Foundation (NAAF). NAAR originated from the Alopecia Areata Registry Biobank and Clinical Trials Network in 2000.[9] The database is a World Wide Web—based registry built to collect biologic samples and epidemiologic data for AA research in the United States, and the establishment of the registry was conducted according to the Declaration of Helsinki Principles. The NAAR data have been managed by NAAF since 2012, and all patients enrolled in the NAAR registered voluntarily, with no restrictions on age, sex, or race/ethnicity for participation.

Participating patients included those who were recruited through academic centers, including The University of Texas MD Anderson Cancer Center, the University of California in San Francisco, the University of Colorado in Denver, the University of Minnesota in Minneapolis, and Columbia University in the New York City. The registry also included noncases (unaffected by AA, had never been given the diagnosis of AA at the time of recruitment, nonblood related to AA cases, and no family history of AA) who were recruited as volunteers through methods listed previously.[1] Noncases were recruited through internet advertisements or at annual NAAF conferences. They also included members of the research team in NAAR or NAAF or unaffected, nonblood-related family members or friends of AA patients.[1] All data were deidentified, and no attempts were made to identify any of the individuals in the registry.

Identification of AA and covariates

NAAR lists case or noncase status of AA based on information entered at the point of registry enrollment. The case status of AA was a mixture of self-reported AA and dermatologist-confirmed AA cases.[9] Among the participants, 1970 were AA patients enrolled through academic institutions after a dermatologist-confirmed diagnosis. Data collected in the registry through surveys included age at enrollment, age at first diagnosis of AA, sex, family history of AA, self-reported race/ethnicity (classified as white, African American, Asian, Hispanic/Latino, and others, which included American Indian and Pacific Islanders), extent of disease (amount of hair loss on scale of 1-5, amount of body hair loss on scale of 1-3, nails involved on scale of 1-3, number of episodes on scale of 1-5), disease subtype (AAT, AAP, AT, AU), and comorbidities, including allergic diseases (asthma, allergic rhinitis, atopic dermatitis, hay fever), autoimmune diseases (lupus, rheumatoid arthritis, juvenile arthritis, Raynaud syndrome, Graves disease, and other thyroid diseases), inflammatory bowel disease (Crohn's disease, ulcerative colitis), and other dermatologic conditions, including psoriasis, vitiligo, and urticaria.

As defined in the data registry, AA patients in the NAAR were classified as AAT if they had disease duration of less than 1 year and AAP if they had

Abbreviations used:

AA:	alopecia areata
AAP:	alopecia areata persistent
AAT:	alopecia areata transient
AT:	alopecia totalis
AU:	alopecia universalis
CI:	confidence interval
NAAF:	National Alopecia Areata Foundation
NAAR:	National Alopecia Areata Registry
NHS:	Nurses' Health Study
NHSII:	Nurses' Health Study II
OR:	odds ratio

disease duration longer than 1 year. AT was defined as total scalp hair loss for more than 1 year, and AU was defined as entire body hair loss. In this study, for analytical purposes, AA subtypes were dichotomized into AAT or AAP and AT or AU. All patients with AA history were classified into only 1 subtype determined at the time of registry entry with no follow-up data.

Statistical methods

We compared the odds of AA among AA patients and noncases across the racial categories using logistic regression with odds ratios (ORs) and 95% confidence intervals (CIs). Participants with no information on age, sex, and race/ethnicity were excluded from the analysis. Multivariate models were adjusted for age at survey (continuous), sex, and comorbidities that were reported to be associated with AA (atopic dermatitis, allergic rhinitis, asthma, hay fever, and thyroid disease, including hypothyroidism and hyperthyroidism).[1] Other comorbid conditions were not adjusted for due to rarity of the diagnosed cases in the registry.

To explore whether there was a racial disparity in the frequency of subtypes of AA, we additionally performed multinomial logistic regression with ORs and 95% CIs. Sensitivity analysis was performed comparing AA cases enrolled through academic institutions (ie, those confirmed by a dermatologist) vs all noncases in the data. In regression analysis, white race and female sex were chosen as reference groups, and stratification by sex was done. All data analysis and statistical processing were performed using SAS 9.4 software (SAS Institute, Inc, Cary, NC).

RESULTS

Characteristics of study population

Information from 12,349 participants was available from the 2000-2016 NAAR data. After excluding 385 participants with no information on age or sex and 560 participants with no information on race/ethnicity, the final study population was 11,404. Among these participants, 2064 participants had no history of lifetime alopecia (noncases), whereas 9340 participants reported at least 1 episode of AA (Fig 1). Participants without a history of AA were older than those with AA history (mean age, 52 vs 43 years) at the time of enrollment, and white was the dominant self-reported race/ethnicity in both groups (79.2% vs 77.4%; Table I). AA-related comorbidities, including atopic dermatitis, asthma, allergic rhinitis, and thyroid diseases, were more prevalent in the AA group.

The unadjusted analysis showed that compared with whites, African Americans had greater odds of AA (OR, 1.92; 95% CI, 1.49-2.47) and Asians had lower odds (OR, 0.44; 95% CI, 0.36-0.54; Table II). The OR of Hispanic/Latino AA patients compared with whites showed no difference in the risk (OR, 1.05; 95% CI, 0.87-1.28). The results were similar in multivariate analysis adjusted for age and sex. Furthermore, in multivariate logistic regression, there was a greater risk in African Americans than in whites of AAT/AAP (OR, 1.93; 95% CI, 1.48-2.52) and AT/AU (OR, 1.57; 95% CI, 1.19-2.06) and a lower risk in Asians of AAT/AAP (OR, 0.46; 95% CI, 0.37-0.59) and AT/AU (OR, 0.32; 95% CI, 0.24-0.42).

These racial differences were consistent by sex, although more significant associations were noted in women, probably due to a much larger sample size and higher statistical power (Table III). The results remained robust in a sensitivity analysis of comparing AA cases confirmed by dermatologists vs all noncases in the registry, albeit statistical significance was only seen in the Asian population due to reduced sample size (n = 1970; Supplementary Table I available at http://dx.doi.org/10.17632/p2hspx8hg5.1). Compared with whites, African Americans had greater odds of AA (OR, 1.34; 95% CI, 0.95-1.88), and Asians had lower odds (OR, 0.40; 95% CI, 0.29-0.56).

DISCUSSION

The present study illustrates that in a cross-sectional registry of AA diagnoses in the United States, the odds of AA differ significantly by race/ethnicity. Higher odds of disease were seen in African Americans compared with whites, whereas the odds were lower in Asians. These results were consistent in multivariate and sex-stratified analyses and in a subgroup analysis according to AA subtype. To our knowledge, this study was the first to explore racial differences in AA subtypes.

Previously, when racial differences in AA were evaluated among 418 (NHS) and 738 (NHSII) self-reported AA cases, a greater lifetime incidence of AA was observed in African American and Hispanic/Latino women compared with white women.[8] In that

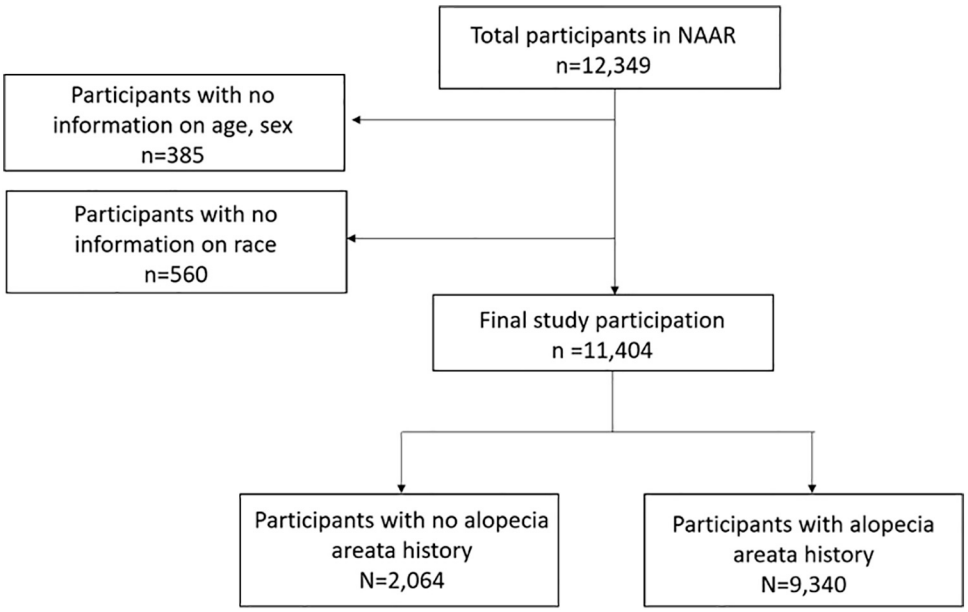

Fig 1. Flowchart for selection of participants from the National Alopecia Areata Registry (*NAAR*) cohort for an analysis of association between self-reported race and alopecia areata risk between years 2000 and 2016.

Table I. Characteristics of alopecia areata patients and noncases enrolled in the National Alopecia Areata Registry from 2000-2016

Variable	Noncases (n = 2,064)	Alopecia areata (n = 9,340)	*P* value
Age, mean (SD), y	51.6 (18.1)	42.52 (18.8)	<.01
Sex, No. (%)			<.01
Female	1283 (62.2)	6695 (71.7)	
Male	781 (37.8)	2645 (28.3)	
Race/ethnicity, No. (%)			<.01
White	1634 (79.2)	7227 (77.4)	
African American	71 (3.4)	602 (6.5)	
Asian	146 (7.1)	283 (3.0)	
Hispanic/Latino	131 (6.4)	610 (6.5)	
Others*	82 (4.0)	618 (6.6)	
History of			
Atopic dermatitis, No. (%)	189 (9.2)	1613 (17.3)	<.01
Asthma, No. (%)	199 (9.6)	1247 (13.4)	<.01
Allergic rhinitis, No. (%)	387 (18.8)	1782 (19.1)	.73
Thyroid disease, No. (%)	137 (6.6)	858 (9.2)	<.01

No., Number, *SD*, standard deviation.
*Includes American Indians and Pacific Islanders.

study, the number of Asian participants was limited and included as part of the "other" race/ethnic group with American Indians, Native Hawaiians, and Pacific Islanders. No statistically significant disparity in the incidence of AA in was identified in this group.

We were able to classify race/ethnicity with more granularity in our study by using a detailed classification. Our study's finding of increased odds of AA in African Americans was consistent with those from the NHS and NHSII.[8] In the NHS/NHSII, one of the explanations for differing incidence by racial groups was diagnostic bias due to patchy hair loss appearing more apparent in the nonwhite populations. This hypothesis, however, was not supported by the lower odds of AA seen in Asians in our study. Because only self-reported AA cases were analyzed in the NHS/NHSII, misclassification of AA cases was one of the limitations and a potential explanation for

Table II. Odds ratios (*ORs*) and 95% confidence intervals (*CIs*) of ethnic/racial groups and ever-diagnosis of alopecia areata (*AA*) and AA subtypes in the National Alopecia Areata Registry from 2000-2016

			Ever-diagnosis of AA			Diagnosis of AAT/AAP		Diagnosis of AT/AU	
Race/ethnicity	Noncases (n = 2064)	AA (n = 9340)	Crude OR (95% CI)	Age-, sex-adjusted OR (95% CI)	Multivariate adjusted OR* (95% CI)	Crude OR (95% CI)†	Multivariate adjusted OR† (95% CI)	Crude OR (95% CI)†	Multivariate adjusted OR (95% CI)†
White	1634	7227	1 [Referent]	1 [Referent]	1 [Referent]	1 [Referent]	1 [Referent]	1 [Referent]	1 [Referent]
African American	71	602	1.92 (1.49-2.47)	1.69 (1.31-2.18)	1.77 (1.37-2.28)	2.15 (1.66-2.80)	1.93 (1.48-2.52)	1.63 (1.25-2.15)	1.57 (1.19-2.06)
Asian	146	283	0.44 (0.36-0.54)	0.39 (0.32-0.49)	0.40 (0.32-0.50)	0.52 (0.41-0.65)	0.46 (0.37-0.59)	0.34 (0.26-0.45)	0.32 (0.24-0.42)
Hispanic/Latino	131	610	1.05 (0.87-1.28)	0.86 (0.70-1.05)	0.90 (0.74-1.11)	1.28 (1.04-1.57)	1.07 (0.87-1.32)	0.78 (0.62-0.98)	0.70 (0.55-0.88)
Others‡	82	618	1.70 (1.35-3.16)	1.26 (0.99-1.60)	1.27 (0.99-1.62)	1.89 (1.48-2.42)	1.37 (1.07-1.77)	1.48 (1.15-1.92)	1.15 (0.89-1.50)

AAP, Alopecia areata persistent; *AAT*, alopecia areata transient; *AT*, alopecia totalis; *AU*, alopecia universalis.
*Multivariate models were adjusted for age at cohort entry (continuous), sex, and comorbidities, including atopic dermatitis, asthma, allergic rhinitis, and thyroid diseases.
†Multinomial regression compares AAT/AAP or AT/AU disease status to noncases.
‡Includes American Indians and Pacific Islanders.

higher AA risk in African Americans than in whites. Other forms of alopecia that frequently arise in African Americans, such as traction alopecia and central centrifugal cicatricial alopecia, may also have contributed to the positive findings.[10] However, when we limited AA cases to those confirmed by a dermatologist, the positive association observed in African Americans remained similar, although somewhat attenuated and nonsignificant due to a much smaller sample size.

An intricate interplay between genetic and environmental factors may account for the racial differences. Pathogenesis of AA is at times linked with autoimmunity by its strong association with human leukocyte antigen class II alleles.[11] Human leukocyte antigen class II alleles are also associated with lupus pathogenesis,[12] and with frequent overlap between lupus and AA patients, it has been suggested that the 2 diseases may share similar pathophysiology.[13] Current epidemiologic data on systemic lupus erythematosus in the United States reveals higher disease incidence in African Americans, Asians, and other races than in whites.[14] Our hypothesis also stemmed from interrelating racial trends of lupus incidence with AA.

Yet the "Hispanic paradox" is at times referenced among lupus epidemiology studies in the United States, and similar "paradoxic" finding may also be present in alopecia. The Hispanic paradox explains that even though Hispanic/Latino group seems to have higher risk of lupus than other race and ethnic groups, when demographics and other clinical factors are adjusted for, Hispanic and Asian lupus patients' mortality is lower than that of African Americans, whites, or Native Americans.[15] A similar potential protective mechanism against AA not yet examined could exist in the Asian population. Further exploration of differing risk of disease by race/ethnicity at both a clinical and genetic level will be crucial in our understanding of the disease pathogenesis.

Also, the impact of race/ethnicity on AA was consistent by sex, while we observed more significant associations in women than in men, potentially due to the larger sample size of women. Future registry data comprising more male patients and longitudinal follow-up may further help to shed light on the presence of a true sex difference.

Our study has multiple strengths. Our analysis comprises a large sample size representing diverse geographic areas in the United States. Most of the participants in the registry were white, but compared with other similar works, the present study includes greater proportions of African American, Asian, and Latino/Hispanic participants.[8] Detailed classification

Table III. Multivariable-adjusted* odds ratios (*ORs*) and 95% confidence intervals (*CIs*) of alopecia areata (*AA*) in the National Alopecia Areata Registry from 2000-2016

	Males					Females				
Ethnicity/race	Noncases (n = 781)	AA (n = 2,645)	Ever-diagnosis of AA, OR (95% CI)	AAT/AAP, OR (95% CI)†	AT/AU, OR (95% CI)†	Non-cases (n = 1,283)	AA (n = 6,695)	Ever-diagnosis of AA, OR (95% CI)	AAT/AAP, OR (95% CI)†	AT/AU, OR (95% CI)†
White	622	2087	1 [Referent]	1 [Referent]	1 [Referent]	1012	5140	1 [Referent]	1 [Referent]	1 [Referent]
African American	21	102	1.36 (0.83-2.24)	1.74 (1.04-2.91)	0.96 (0.54-1.68)	50	500	1.93 (1.42-2.60)	2.03 (1.49-2.77)	1.79 (1.30-2.47)
Asian	50	99	0.53 (0.37-0.77)	0.69 (0.46-1.02)	0.37 (0.23-0.58)	96	184	0.34 (0.26-0.44)	0.37 (0.28-0.50)	0.29 (0.21-0.41)
Hispanic/Latino	55	185	0.83 (0.60-1.14)	1.04 (0.74-1.48)	0.58 (0.40-0.86)	76	425	0.96 (0.74-1.24)	1.09 (0.84-1.43)	0.77 (0.57-1.03)
Others‡	33	172	0.99 (0.66-1.48)	1.06 (0.69-1.62)	0.92 (0.59-1.42)	49	446	1.45 (1.07-1.97)	1.56 (1.14-2.15)	1.29 (0.93-1.81)

AAP, Alopecia areata persistent; *AAT*, alopecia areata transient; *AT*, alopecia totalis; *AU*, alopecia universalis.
*Multivariate models were adjusted for age at cohort entry (continuous), sex, and comorbidities, including atopic dermatitis, asthma, allergic rhinitis, and thyroid diseases.
†Multinomial regression was performed comparing AAT/AAP or AT/AU disease status to noncases.
‡Includes American Indians and Pacific Islanders.

of AA patients into AAT, AAP, AT, and AU also enabled subtype analysis with multinomial regression.

Nevertheless, we were unable to overcome the limitations rooting from inherent features of registry data. Because the invitation to participate in the registry was voluntary and not a randomized process, characteristics of AA patients enrolled in the registry may differ compared with those who were not enrolled, perhaps by disease severity or duration. Mild cases of AA often undergo spontaneous remission; thereby, the registry can be susceptible to selection bias from participants with refractory AA more willing to join a patient-centered research community.

In addition, because of a large amount of missing data (incomplete entry at survey or registry level), we were not able to incorporate the onset age of AA and comorbidities for additional adjustment in the multivariate analysis. We also could not account for misclassification of race/ethnicity because it was self-reported, and noncases in the registry may not be the most appropriate comparison to AA cases, partly judging from their large age differences.

Lastly, with a limited number of covariates in the registry, we could not account for all known and unknown confounders of the association, which may impact our point estimates. Still, our study reveals an important association between race/ethnicity and AA in men and women across diverse geographic regions in the United States in both self-reported AA and clinician-confirmed AA populations.

CONCLUSION

In a large AA registry database in the United States, we found significantly higher odds of AA in African Americans compared with whites and lower odds of AA in Asians than in whites. Our findings raise a different perspective from the conventional view that AA does not differ by race/ethnicity. These racial disparities may arise from a complex interplay of differences in the disease risk, screening frequency, and diagnostic bias.[16] AA is a chronic, recurrent disease that has profound economic and psychologic impact on individuals.[17] Therefore, if true differences in the disease risk exist, efforts to efficiently deliver care with early, accurate diagnosis and assistance on long-term psychologic stress in the high-risk racial group will be needed. Failing to mitigate these disparities may place an additional burden on health care delivery in the future.

Additional studies are needed to validate our results along with continued investigation for causes of racial differences and targeted screening of groups vulnerable to AA. We suggest that studies with

improved adjustment for confounders and emphasis on the treatment outcome with longitudinal design will help us clarify whether there are persistent differences in AA burden throughout all stages of the disease.

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