



IMMUNE FOLLICULAR GENE EXPRESSION PATTERNS ACROSS THE SEVERITY SPECTRUM OF ALOPECIA AREATA

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ABSTRACT

Alopecia areata (AA) the immune system activates and disrupts the biology of the hair follicle, but it is not clear if there is a consistent change in expression of genes in the scalp as a function of clinical severity. We conducted a secondary analysis of baseline scalp tape-strip RNA expression profiles from 35 subjects (18 healthy controls and 17 AA patients with 4 mild, 7 moderate and 6 severe cases). The gene sets used for interferon/JAK–STAT, cytotoxic T/NK, follicular, and lipid/sebocyte genes were used to calculate expression scores. Patients with AA had higher interferon/JAK–STAT scores, lower follicular scores, and higher cytotoxic T/NK scores when compared to controls. In none of the four scores did the progression of scores across the AA severity groups reach a statistically significant level. In the AA cases, the lipid/sebocyte scores and the cytotoxic T/NK scores were found to be negatively correlated (Spearman's $\rho = -0.713$; adjusted $q = 0.005$). The results showed a clear disease-associated expression pattern, while some of the changes were more ambiguous as they were related to severity. The within-patient association needs to be validated in independent studies using larger severity groups and direct measures of follicular and sebaceous function.

Keyword: Alopecia areata, tape-strip RNA sequencing, gene expression, disease severity, immune pathways

Introduction

Alopecia areata is a non-scarring disorder of hair loss that results from the activity of the immune system against the hair follicle. It is not sufficient to describe it with a single clinical term, as it occurs in different patterns of alopecia from baldness in patches to extensive alopecia of scalp or body hair, with a wide range of biological variability (Akbaba et al., 2025). Inflammation is normally kept in check by a degree of local immune privilege within follicles. If this protection is compromised, pro-inflammatory signaling and the presence of cytotoxic cells are detrimental to the differentiation and growth of follicles (Udović, Šutić, et al., 2024). This is complicated further by genetic susceptibility, which by itself does not account for differences in the extent or in the course of disease (de Souza, 2025). The molecular information obtained directly from the scalp, therefore, could be useful to discriminate between common disease characteristics and individual differences in diseased animals.

Cytotoxic T-cell and natural killer-cell programs as well as signaling pathways like interferon (INF) responsive signaling via the JAK–STAT pathway are prime candidates in alopecia areata. A meta-analysis of studies of lesional scalp expression revealed a reproducible inflammatory profile and alterations in the expression of genes associated with the structure of the follicles (Rivera-Ruiz, Gay-Mimbrera, et al., 2025 A). Analyses of the regulatory networks have also suggested certain immune-related genes as possible markers for disease progression, but a specific gene should not be assumed as a validated marker without independent testing (Xiong et al., 2023). In addition to bulk tissue, single-cell chromatin and transcriptomic profiling of peripheral-blood immune cells have revealed distinct states of multiple immune-cell types in various disease groups (Gay-Mimbrera et al., 2025). Single-cell analysis of the scalp has identified several inflammatory signals in various immune and nonimmune cell types, such as reduced expression of hair-keratin in keratinocytes (Hu et al., 2026). Together these strategies promote an investigation of immune activation and follicular function as linked measurable processes.

But their relationship needs interpretation carefully statistically. The difference between the two groups (patients and unaffected controls) can explain part of the observed strong inverse association between the groups; it is not necessarily valid to describe how immune and follicular program vary within the patients only. Likewise, differences in the molecules from one severity category to another do not necessarily represent a smooth gradient of increasing severity. Activity of the disease can differ among the areas sampled and pathway scores are a composite of numerous genes that do not necessarily change in parallel. Alopecia areata is a disease with clinical and histological features that are distinct from primary cicatricial alopecias, which are also associated with epithelial and immune signals in the affected follicles (Wang et al., 2022) and require disease-specific interpretation. There are also common and subtype-specific pathways that are identified in transcriptomic synthesis of lymphocytic scarring alopecias (Rivera-Ruiz, Ungar, et al., 2025 B).

Other forms of heterogeneity can make comparisons of the severity more complicated. Another patient population has shown that age and immunoglobulin E (IgE) status are associated with immune profiles in alopecia areata (Liu et al., 2025). Microbiota patterns on the scalp have been linked to the severity of the disease and systemic inflammatory markers but not shown to drive the changes in the expression pattern (Gómez-Arias et al., 2024). From the studies of the immune microenvironment in androgenetic alopecia, inflammation was also observed in another type of hair loss, which cannot be directly extrapolated to alopecia areata (Charoensuksira et al., 2024). Another reason for interpreting overlapping inflammatory signals in their diagnostic context is with work that has linked frontal fibrosing alopecia with rosacea (Liu et al. 2022). These factors support discrete comparisons and limited assertions as opposed to attempting to make a single pathway the rule for all clinical features.

Another difficulty is sampling method. Tape stripping is less invasive than scalp biopsy, which allows for the extraction of RNA from deeper follicular structures. The original study used to collect the data investigated if the signal in the tape-strip profiles could reflect immune, epithelial, severity, and baricitinib-response signals, and compared them to biopsies (Mochón-Jiménez et al., 2026). It has already published interferon and cytotoxic activation and decreased follicular expression in its findings. Therefore, when conducting a analysis of these profiles, one should respect those results that have already been established and not claim them as discoveries. Its narrower contribution is by adopting consistent baseline tape-strip sampling to further distinguish any case–control differences from association within affected individuals whilst also examining the scores for lipids and sebocytes and cytokines together. A recent multi-dataset prediction study demonstrates the interest shown in the computational signatures of alopecia areata, but association in this small cohort does not suggest predictive performance (Chen et al., 2026). The following analyses are conducted with the publicly deposited, processed expression matrix and clinical data with the aim of achieving three objectives:

1. Compare baseline immune and follicular expression programs between alopecia areata cases and unaffected controls.
2. Assess whether pathway scores exhibit ordered associations across mild, moderate, and severe alopecia areata.
3. Examine within-case relationships among cytotoxic, follicular, and lipid-associated programs while considering clinical heterogeneity.

2. Materials and Methods

2.1 Study Design and Data Source

This analysis used the publicly available alopecia areata transcriptomic files (Monchón-Jiménez et al., 2025). An archive was provided with a gene-by-sample expression matrix, sample metadata, curated gene sets, differential-expression summaries, and an analysis script. There were 15,443 unique gene symbols in the matrix across 60 profiles and 60 sample identifiers in metadata from 44 participants. Since the matrix provided was already normalized and corrected for sequencing batch, its values were directly used. The research question was the expression patterns of baseline immune and follicular status in relation to disease condition and severity.

2.2 Sample Selection and Clinical Groups

To maintain sampling method and treatment status constant, only baseline scalp tape-strip profiles were used in the primary sample. Eighteen normal controls and 17 patients with AA were retained, which included 4 mild, 7 moderate and 6 severe cases of AA. Participation by each included person was only used once in primary comparisons. Two profiles per block were excluded for baseline and ten profiles were excluded for post treatment because of the mixing of sampling method and treatment state. Data on age, sex, and Severity of Alopecia Tool scores were summarized with available data.

2.3 Gene Sets and Pathway Scores

The four pathways were chosen from a set of gene sets in the archive: interferon/JAK–STAT, cytotoxic T/natural killer cells, follicular function and lipid/sebocyte activity. The gene names of both sets were matched to the expression matrix and genes that were not included in the matrix were excluded. For the 35 primary samples, the expression for each gene was normalised by subtracting the sample average and dividing by the sample standard deviation. Each available standardized gene value was averaged to calculate the pathway score for each sample. A higher score does not necessarily mean greater pathway activity, rather, it means greater relative expression in this analysis.

2.4 Group and Severity Comparisons

Characteristics of the groups were presented as counts, age means and SDs, and median severity scores with interquartile ranges of alopecia scores. Kruskal–Wallis tests were used to compare the four pathway scores between controls and mild, moderate and severe cases. A Spearman test was conducted for ordered severity for 17 cases and gene-wise Spearman tests were used to screen for severity associations. A comparison of cases and controls was conducted for 15,443 genes in the Welch tests. Focusing on four group tests, four pathway severity tests, and the two gene-level test families, Benjamini–Hochberg adjustment was applied. Python and pandas, along with SciPy, were used for the analyses; values below 0.05 were deemed significant.

2.5 Pathway Associations and Sensitivity Analysis

Spearman correlations were performed on four pairs of immune–follicular or immune–sebocyte scores for all 35 samples, and separately for 17 cases. One family of the four within-case tests was a Benjamini–Hochberg correction family. The 10,000 resamples and a fixed seed were used to calculate bootstrap percentile intervals for case–control score differences and the strongest within-case correlation. A leave-one-out study was performed to obtain sensitivity estimates for each individual case. For complete cases, severity, age and sex were controlled for in a descriptive rank-based partial correlation. For complete cases, descriptive rank-based partial correlation was used to control for severity, age and sex. Due to the small size of the subgroups and having expression values already processed, relationships were interpreted as associations, not as causal or prognostic.

3. Results

3.1 Sample Characteristics

Total of 35 independent tape-strip profiles (18 controls and 17 cases) is the primary cohort. The four mild cases, seven moderate cases and six severe cases were found in the disease group (Table 1). Of the 17 cases, 12 were women and of the 18 controls, eight were women. Recorded categories have been corroborated by an increase in the Median Severity of Alopecia Tool scores from 3.0 in mild disease to 37.0 in moderate and 86.0 in severe disease. For one mild case, age was not recorded, but for all other primary records age values were recorded. There were no post-treatment or bulk-biopsy samples that went into the next tests (Figure 1).

Table 1. Characteristics of baseline tape-strip samples

Characteristic	Control	Mild	Moderate	Severe
Samples, <i>n</i>	18	4	7	6
Female, <i>n</i>	8	3	4	5
Male, <i>n</i>	10	1	3	1
Age available, <i>n</i>	18	3	7	6
Age, mean $\pm$ SD (years)	47.44 $\pm$ 18.27	36.67 $\pm$ 21.94	49.57 $\pm$ 13.71	39.67 $\pm$ 7.20
SALT score, median (Q1–Q3)	0.00 (0.00–0.00)	3.00 (1.75–4.75)	37.00 (30.00–37.50)	86.00 (64.50–100.00)

SALT = Severity of Alopecia Tool; *SD* = standard deviation.

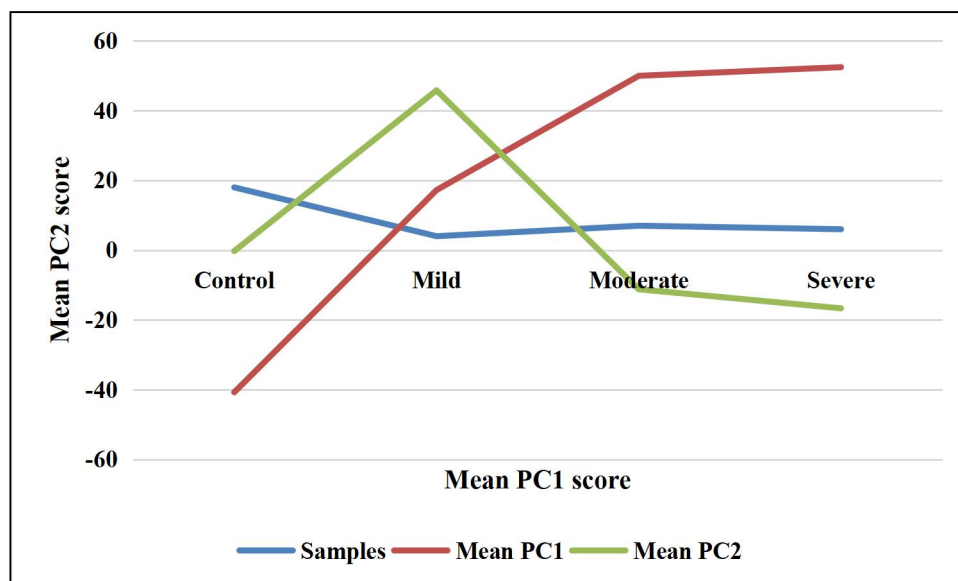


Figure 1. Expression profile map

3.2 Pathway Scores Across Clinical Groups

Correction was required and the between group differences for interferon/JAK – STAT, cytotoxic T/natural killer and follicular pathway scores were significant after correction in four tests. The immune score was generally higher for cases than controls, whereas the follicular score was generally lower for cases than controls. There was no significant difference between the groups in terms of lipid/sebocyte score. However, the four pathway scores did not show a significant ordered mild to severe trend following correction in the 17 cases; and the follicular trend was not a progressive decrease but a progressive increase (Table 2). The between-group results primarily differentiate between diseased and healthy scalps and should not be interpreted as evidence for molecular progression with disease severity (Figure 2).

Table 2. Pathway scores and tests of clinical-group differences

Pathway (genes available)	Cont rol mean ± SD	Mild mea n ± SD	Moder ate mean ± SD	Seve re mea n ± SD	Case– control difference (95% CI)	Gro up <i>H</i>	Group <i>p</i>	Group adjust ed <i>p</i>	Case- only severi ty <i>p</i>	Severi ty adjust ed <i>p</i>
IFN/JAK– STAT (128)	–0.31 4 0.448	0.63 2 0.63 4	0.243 ± 0.189	0.23 8 0.32 1	0.647 (0.389 to 0.924)	14.5 71	0.0022	0.0030	–0.29 2	0.511

Cytotoxic	−0.60	0.75	0.589 ±	0.63	1.255	22.9	0.0000	0.0001	−0.16	0.530
T/NK (17)	9 ±	4 ±	0.409	8 ±	(0.934 to	89	41	62	4	
	0.621	0.25		0.40	1.591)					
		0		2						
Follicular	0.332	−0.7	−0.252	−0.1	−0.683	14.7	0.0020	0.0030	0.449	0.282
(28)	±	90 ±	± 0.368	75 ±	(−0.993 to	66				
	0.439	0.59		0.46	−0.388)					
		1		6						
Lipid/seboc	−0.04	0.26	0.005 ±	−0.0	0.098	3.06	0.383	0.383	−0.18	0.530
yte (60)	8 ±	4 ±	0.546	39 ±	(−0.166 to	0			6	
	0.231	0.33		0.57	0.343)					
		9		6						

Scores are means of standardized gene expression values. Case-control differences compare all 17 cases with 18 controls; confidence intervals are bootstrap percentile intervals. Group tests include all four groups. Severity correlations include cases only. Adjusted values use separate four-test correction families.

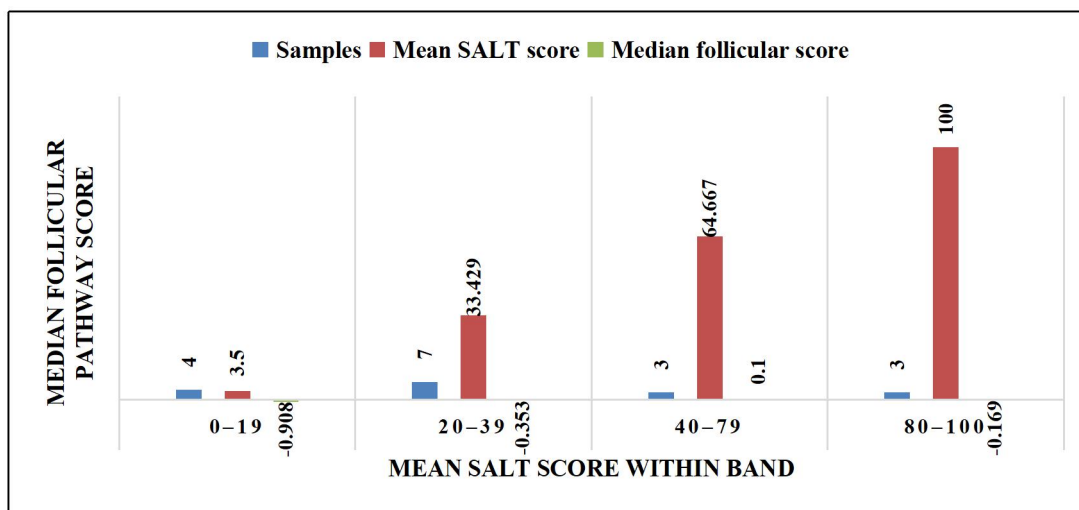


Figure 2. Follicular expression across SALT score bands

3.3 Gene-Level Expression Differences

At a false-discovery rate of < 0.05, 5,159 of 15,443 genes were found to be higher in the cases and 1,894 lower. Of the 15,443 genes, 5,159 were found to be higher in the cases and 1,894 lower, when comparing the genes with the 18 controls using Welch comparisons at a false-discovery rate of < 0.05. Table 3 does not provide a diagnostic panel and only includes genes from the pre-specified pathways. Genes that were

expressed at higher levels were the cytotoxic genes (GZMA, GZMM) while the genes that were expressed at lower levels were the follicular genes (DSG4, KRT86). Gene differences: Provided a batch corrected matrix of log-scale gene differences. No discoveries were found in case-only severity tests when the severity was adjusted, the lowest adjusted value being 0.420 (Figure 3).

Table 3. Selected gene expression differences between cases and controls

Gene	Prespecified pathway	Mean difference: cases minus controls	<i>p</i> value	Adjusted <i>p</i>
GZMA	Cytotoxic T/NK	4.744	1.39×10^{-10}	3.06×10^{-7}
GZMM	Cytotoxic T/NK	3.829	4.28×10^{-9}	9.99×10^{-7}
CXCR6	Cytotoxic T/NK	3.605	1.26×10^{-7}	7.75×10^{-6}
IFI44L	IFN/JAK-STAT	4.326	1.90×10^{-7}	9.73×10^{-6}
LHX2	Follicular	-2.388	0.000012	0.000212
DSG4	Follicular	-4.123	0.000026	0.000371
KRT86	Follicular	-4.709	0.000036	0.000483
KRT85	Follicular	-3.742	0.000303	0.002513

Mean differences are on the supplied processed log-scale expression values. Adjusted values account for all 15,443 gene-level case-control tests, not just the eight displayed genes.

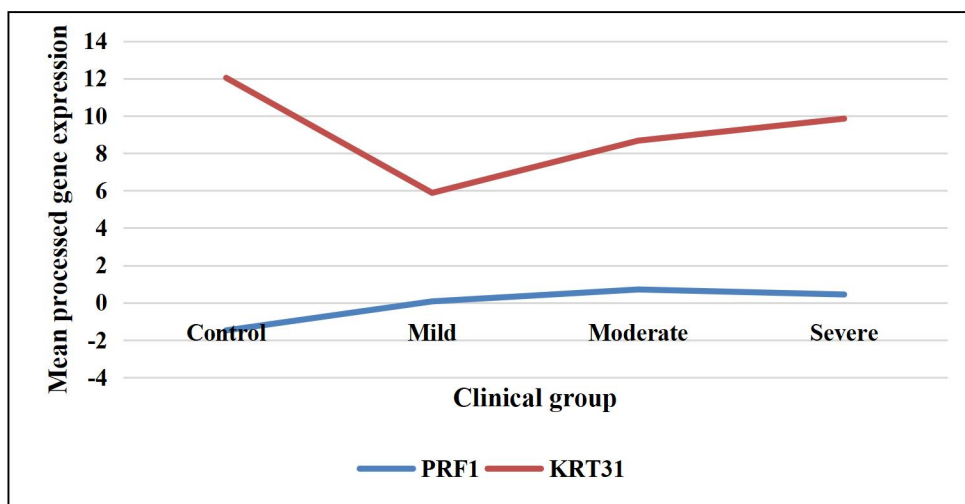


Figure 3. Expression of two illustrative genes

3.4 Relationships Between Pathway Scores

Immune and follicular scores were inversely related in all 35 samples, with significant attenuation within the cases as shown in Table 4. In cases, the lipid/sebocyte score remained inversely associated with the cytotoxic score ($\rho = -0.713$; adjusted $p = 0.005$; 95% CI for the bootstrap: -0.870 to -0.399). The leave-one-out estimates were between -0.738 and -0.656 . The descriptive partial rank correlation over severity, age and sex for the 16 complete cases was found to be -0.801 . This within case relationship (Figure 4) is limited in interpretation due to the small sample size and lack of independent validation.

Table 4. Associations between immune and follicular or sebocyte pathway scores

Pathway pair	All samples, n	All-sample ρ	All-sample p	Cases only, n	Case-only ρ	Case-only p	Case-only adjusted p
IFN/JAK–STAT × follicular	35	−0.712	0.00000161	17	−0.081	0.758	0.758
Cytotoxic T/NK × follicular	35	−0.798	0.00000000976	17	−0.478	0.0523	0.105
IFN/JAK–STAT × lipid/sebocyte	35	0.166	0.340	17	0.176	0.498	0.664
Cytotoxic T/NK × lipid/sebocyte	35	0.008	0.966	17	−0.713	0.00131	0.00523

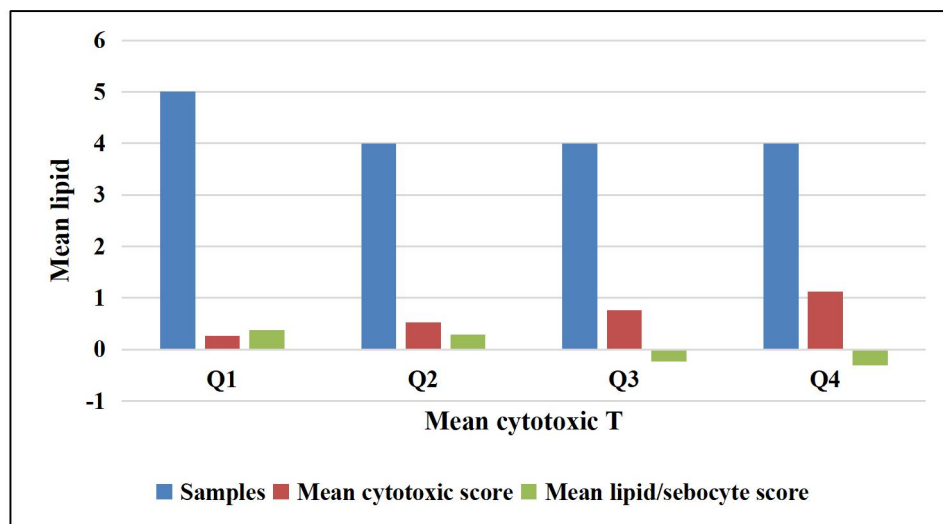


Figure 4. Cytotoxic and lipid/sebocyte scores among cases

4. Discussion

Distinctive molecular differences between alopecia areata (AA) and normal scalp. Baseline scores of interferon/JAK–STAT and cytotoxic T/NK expression were higher in patients with AA than in follicular expression. The direction of these differences aligns with reports of immune activation and decreased expression of hair related genes in AA skin (Kim et al., 2024). Immune and epithelial changes were also reported in the accompanying study which analysed the same data from the tape-strip (Mochón-Jiménez et al., 2026). Therefore, the present analysis should be interpreted as a secondary analysis of these data only, and not as a confirmation. Its contribution is to highlight the overall disease–control differences, in comparison with the patterns within the AA group, which were less consistent.

A negative correlation between immune and follicular scores was found for all 35 baseline tape-strip samples. This correlation is in line with the idea that inflammation and disturbance of follicular programs happen simultaneously in AA (Fitzhugh et al., 2025). This inverse correlations were reduced when analysis was limited to 17 AA cases. The pooled relationship was strong and represents the separation between patients and controls, and cannot be interpreted as an indicator of immune activation predicting immune suppression in any given patient. Similarly, no difference in the four pathway scores was found between mild and severe AA, and this progression was not statistically significant in the present analysis. This is different than the molecular patterns described in the accompanying publication (Mochón-Jiménez et al., 2026) associated with the severity. The disagreement may be attributed to differences in sample selection, pathway scoring, and/or statistical models. The current analysis has limited power to detect a gradient in severity as there are only four mild cases.

The variation between patients could also be due to biological variation other than clinical severity. The molecular profile of AA may be influenced by associated immune features as revealed by the activation of the OX40 receptor in the skin and systemic circulation in both atopic and non-atopic backgrounds (Kim et al., 2024). The relationship between comorbidities of atopy and autoimmunity and susceptibility to AA has been investigated by genetic studies (Herrera-Rivero et al., 2026). There has also been an association study of immune-regulatory polymorphisms with susceptibility in a Jordanian patient population (Alghamdi et al., 2024). These studies serve as context for heterogeneity but are different measures because they measure other phenomena—hereditary variants or comorbidities can not be deduced from expression scores in a scalp sample. A meta-analysis report showed that several immune-mediated diseases were more common in persons with AA, further emphasizing the importance of considering comorbidities in future AA expression studies (Krajewski et al., 2025).

There are some interesting findings within the case which require further investigation. There was no significant difference between the AA and control samples for the lipid/sebocyte score, but the higher the cytotoxic T/NK score in the AA group, the lower the lipid/sebocyte score. A simple comparison of diagnostic groups may obscure an association that is evident among patients. It is an association based on expression, and not a direct measure of sebum production, sebaceous gland function or immune-cell

abundance. Immunological pathways that are common to both the AA and COVID-19 analyses have also been identified through computational analysis, demonstrating that an immune expression signature is insufficient to define a specific disease mechanism (Lu et al., 2025). Androgenetic alopecia has been studied with regard to interactions between immune and nonimmune processes (Xiao et al., 2025). Spatial and single-cell research in primary cicatricial alopecia also shows the presence of inflammation of the follicles, but with a different clinical and pathological trend (Chen et al., 2025).

There are a number of limitations to interpretation. Processed, batch-corrected expression data and a small case group were used for the analysis, which led to a pathway score that is dependent on the gene sets selected and the pathway scoring method used. Immune and follicular signals may be different because of differences in sampled cell populations. The difference between the available metadata and the accompanying publication was found to be 60 profiles versus 61 profiles, respectively; this should be addressed before submission (Mochón-Jiménez et al., 2026). This baseline analysis did not include treatment response, nor does the observed association provide any information about the timing or cause of follicular disruption. Future studies involving the within case results should employ an independent sample to assess the results, document pertinent comorbidities and correlate tape strip expression with follicular and sebaceous biology parameters. These findings confirm a disease–control contrast that is reproducible for the time being, but the relationship between pathway expression and clinical severity remains unclear.

5. Conclusion

In the severity-focused analysis a clear molecular difference was identified between alopecia areata (AA) and healthy scalp. Baseline tape-strip samples showed higher interferon/JAK–STAT and expression scores for patients with AA compared to controls, while expression scores for the follicles were lower in patients than in controls. But the four pathway scores analysed did not show any significant trend from mild to severe disease in the 17 AA cases. The data thus confirm an expression pattern that is associated with the disease but do not provide evidence for a correlation with clinical severity. Inside the AA group, a lower lipid/sebocyte score was correlated with higher scores of the cytotoxic T/NK cells. There are indications that there may be a link between local immune activity and expression of sebaceous related genes which warrants further investigation. The measurements are based on RNA expression and, therefore, do not indicate whether immune activity impacts the function of the sebaceous glands, or whether there is any biological relationship between them. They've added to the molecular understanding of AA by differentiating between differences between patients and controls and variation among patients with AA. Due to the relatively low number of cases, particularly the mild group, conclusions about severity are limited. The next step would be to validate the pathway scores and the association between the cytotoxic–lipid in an independent, larger cohort of patients with uniform severity assessment and direct measures of follicular and sebaceous biology.

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