

Tape-strip proteomic profiling and biopsy validation highlight inflammatory pathogenesis in seborrheic dermatitis

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BACKGROUND

- Seborrheic dermatitis (SD) is a chronic inflammatory skin disease characterized by erythema, pruritus, and scaling in sebum-rich areas.¹
- Transcriptomic profiling of tape strips has highlighted involvement of the IL-23/Th17 and Th22 pathways and barrier disruption driven by dysregulated lipid metabolism and tight junctional integrity.²
- This study aims to validate the cutaneous molecular signature of SD via proteomics measured using tape strips and full-thickness skin biopsies.

METHODS

- Tape-strips were collected from lesional (LS) and nonlesional (NL) facial skin of 26 adults with mild-to-severe SD (Investigator’s Global Assessment/IGA 2-4) and 12 healthy controls (HC).
- Proteomic analysis was performed on tape strips using Olink and compared to a previously published cutaneous atopic dermatitis (AD) proteome (n=23).³
- Differential expression was defined as |fold change| > 1.5 and false discovery rate (FDR) < 0.05.
- RT-PCR was performed for validation.
- LS and NL skin biopsies were collected from a subset of 8 patients and analyzed using immunofluorescence (IF) and immunohistochemistry (IHC).

RESULTS

Figure 1. Heatmap displaying the relative expression of Th1, Th2, and Th17 immune proteins between healthy controls and the nonlesional (NL) and lesional (LS) skin of seborrheic dermatitis (SD) patients. The table displays the fold changes of pairwise comparisons of individual biomarker expression. + p<0.10, * p<0.05, ** p<0.01, ***<0.001.

	Healthy Controls	Seborrheic Dermatitis		LS v HC	NL v HC	LS v NL
		NL	LS			
Th1						
Th2						
Th17						

ACKNOWLEDGEMENTS

We would like to thank the patients for their participation in this study. This study was funded by Arcutis Biotherapeutics.

Figure 2. Comparisons of proteomic expression between seborrheic dermatitis lesional skin (LS), atopic dermatitis LS, facial skin of healthy controls (HCs), and body skin of HCs. Boxplots represent z-scores of overall immune and pathway-level protein expression from gene set variation analyses. + p<0.10, * p<0.05, ** p<0.01, ***<0.001.

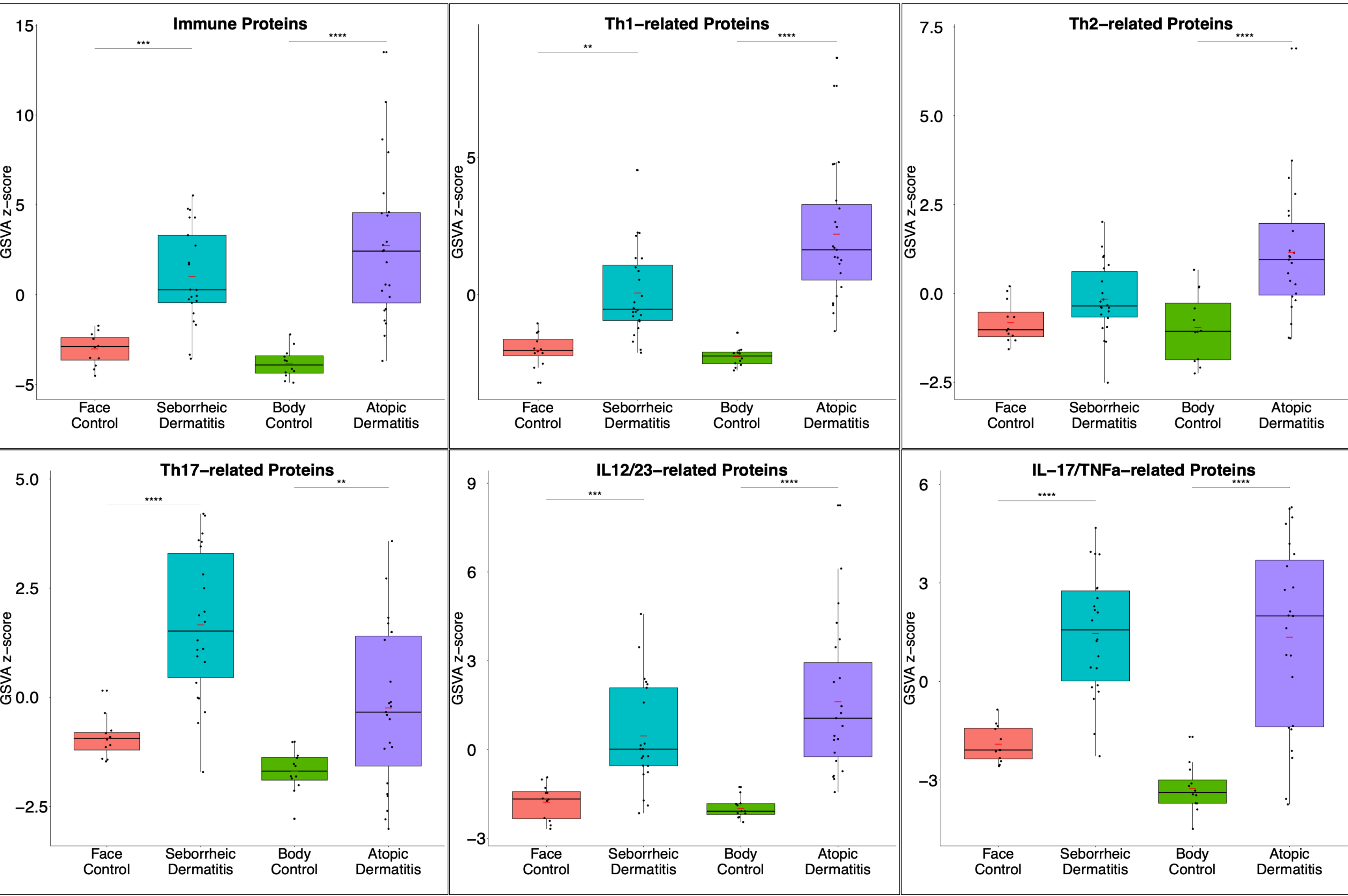
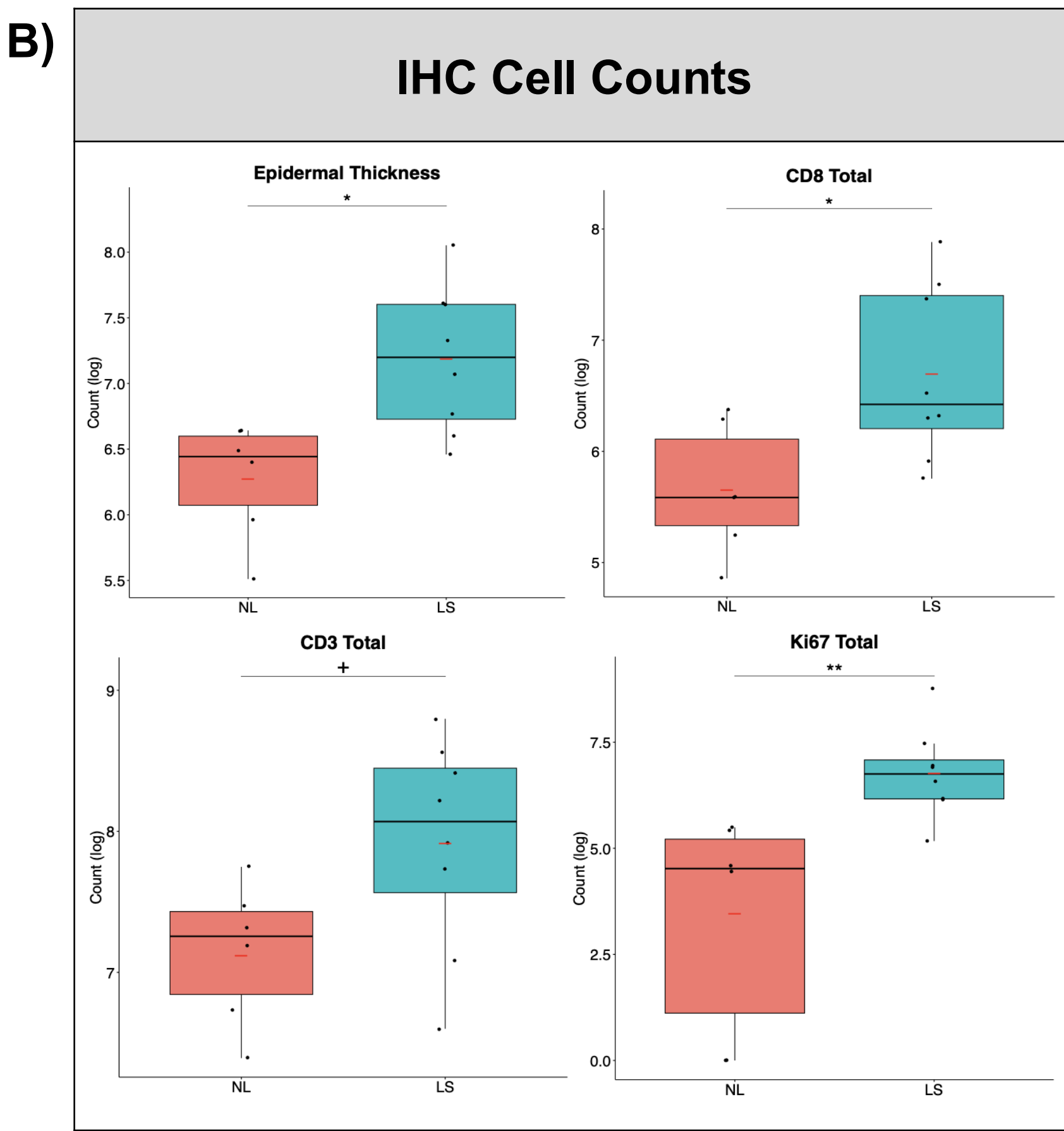


Figure 3. A) Histologic staining and immunohistochemical analysis of CD3, CD8, and Ki67 expression in the nonlesional and lesional skin of participants with seborrheic dermatitis B) Boxplots depicting epidermal, CD3, CD8, and Ki67 cell counts.



CONCLUSIONS

- These data further elucidate and validate the inflammatory pathomechanisms of SD.
- Consistent Th17/Th22 skewing with minimal Th2 involvement reinforces the differences between SD and AD and highlight SD as a primarily Th17-mediated disease.
- Barrier impairment at the protein level supports the multifaceted pathophysiology of SD, which may inform therapeutic targeting.

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