Characterization of the Immune Response in Skin Lesions of Hidradenitis Suppurativa (HS)

Background

- The pathogenesis of Hidradenitis Suppurativa (HS), a chronic inflammatory skin disease, is poorly understood
- Disease-specific autoantibodies, autoantigens, or auto-reactive T cells have not been identified

Research Question

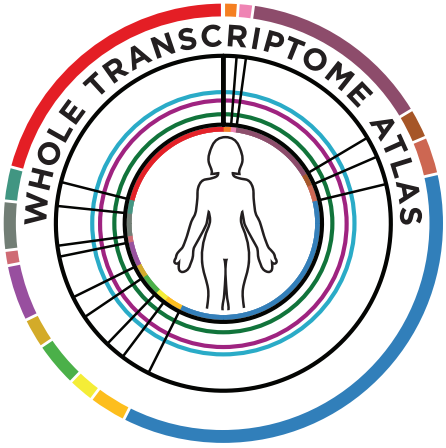
Are B cells prevalent in HS skin lesions and if so, do they form follicular-type clusters? What markers are expressed?

Experimental Setup

Instrument	GeoMx® DSP
Sample Type	FFPE
Tissue Type	Skin
Assay	Human Whole Transcriptome Atlas
Analytes	RNA
Readout	NGS

Why GeoMx?

Digital spatial profiling enabled the transcriptional comparison between B cell clusters and diffuse B cell regions in HS skin legions.



Results & Conclusions

- Comparison of diffuse B cell populations with follicular-type B cell clusters revealed enrichment of transcripts associated with germinal centers: C3, CLU, CR2 (encoding CD21), and FDCSP.
- Skin lesions exhibited downregulation of fibroblast/ECM associated genes such as PDGFRA, DCM, LUM, COL5A1, COL5A2, etc.
- B cells are prominent in HS lesions and some lesions contain ectopic germinal centers.
- An enrichment of transcripts associated with germinal centers was found in the B cell clusters.
- Anti-CEL-producing plasmablasts were detectable within lesional skin.

Macchiarella G et al. Disease Association of Anti-Carboxyethyl Lysine Autoantibodies in Hidradenitis Suppurativa. J Invest Dermatol. 2022 Sep 16;S0022-202X(22)01927-3. ***https://doi.org/10.1016/j.jid.2022.08.051***

For more information, please visit nanostring.com/geomx

NanoString Technologies, Inc.
530 Fairview Avenue North
Seattle, Washington 98109
T (888) 358-6266
F (206) 378-6288
nanostring.com
info@nanostring.com

Sales Contacts
United States **us.sales@nanostring.com**
EMEA: **europe.sales@nanostring.com**
Asia Pacific & Japan **apac.sales@nanostring.com**
Other Regions **info@nanostring.com**

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