

Supplementary Materials for

Neutrophil extracellular traps, B cells, and type I interferons contribute to immune dysregulation in hidradenitis suppurativa

Angel S. Byrd*, Carmelo Carmona-Rivera*, Liam J. O'Neil, Philip M. Carlucci, Cecilia Cisar, Avi Z. Rosenberg, Michelle L. Kerns, Julie A. Caffrey, Stephen M. Milner, Justin M. Sacks, Oluseyi Aliu, Kristen P. Broderick, Jonathan S. Reichner, Lloyd S. Miller, Sewon Kang, William H. Robinson, Ginette A. Okoye, Mariana J. Kaplan*

*Corresponding author. Email: mariana.kaplan@nih.gov (M.J.K.); carmelo.carmona-rivera@nih.gov (C.C.-R.); angel_byrd@alumni.brown.edu (A.S.B.)

Published 4 September 2019, *Sci. Transl. Med.* **11**, eaav5908 (2018)
DOI: 10.1126/scitranslmed.aav5908

The PDF file includes:

- Fig. S1. Analysis of neutrophil stimulation.
- Fig. S2. Analysis of PAD expression and BMI in HS.
- Fig. S3. Type I IFN serum activity is not increased in HS serum.
- Table S1. Correlation analysis of anti-TNF treatment with B cells and autoantibodies.

Other Supplementary Material for this manuscript includes the following:

(available at stm.sciencemag.org/cgi/content/full/11/508/eaav5908/DC1)

Data file S1 (Microsoft Excel format). Primary data.

Supplementary Materials

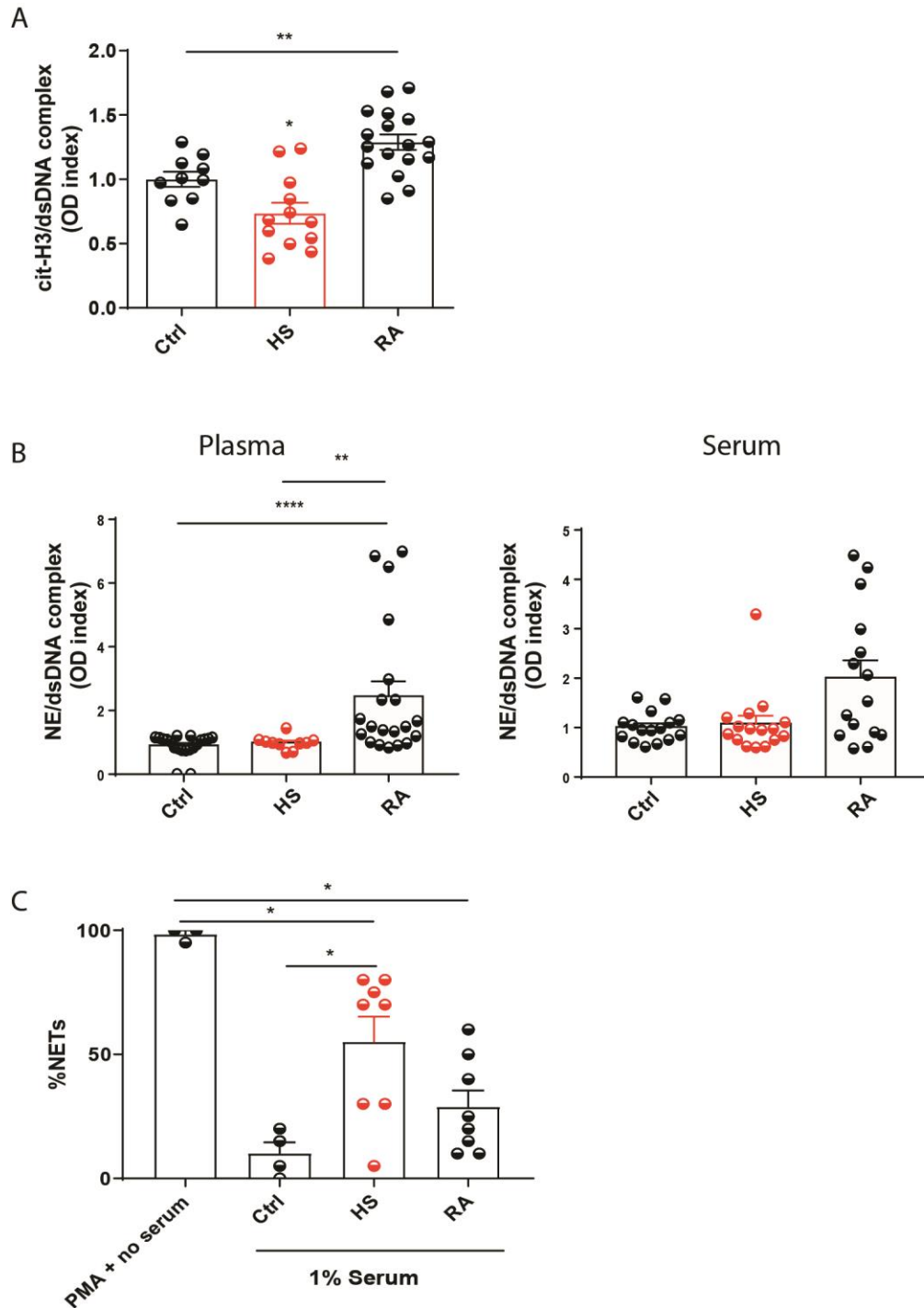


Fig. S1. Analysis of neutrophil stimulation. (A) Control (Ctrl; n=10), HS (n=12) and RA (n=17) sera were analyzed for the presence of NETs complexes (cit-histone H3-DNA

complexes) according to Material and Methods. Results are the mean \pm SEM, $*p < 0.05$, $**p < 0.01$, Mann-Whitney *U* test analysis was used. **(B)** Control (Ctrl; n=20), HS (n=10) and RA (n=20) plasma and (Ctrl; n=16), HS (n=16) and RA (n=15) sera were analyzed for the presence of neutrophil elastase-DNA complexes according to Material and Methods. Results are the mean \pm SEM, $**p < 0.01$, $***p < 0.001$, Mann-Whitney *U* test analysis was used. **(C)** Control (Ctrl; n=4), HS (n=8) and RA (n=8) sera were analyzed for their capacity to degrade PMA-generated NETs from control neutrophils according to Material and Methods. PMA + no serum was used as reference. Results are the mean \pm SEM, $*p < 0.05$, Mann-Whitney *U* test analysis was used.

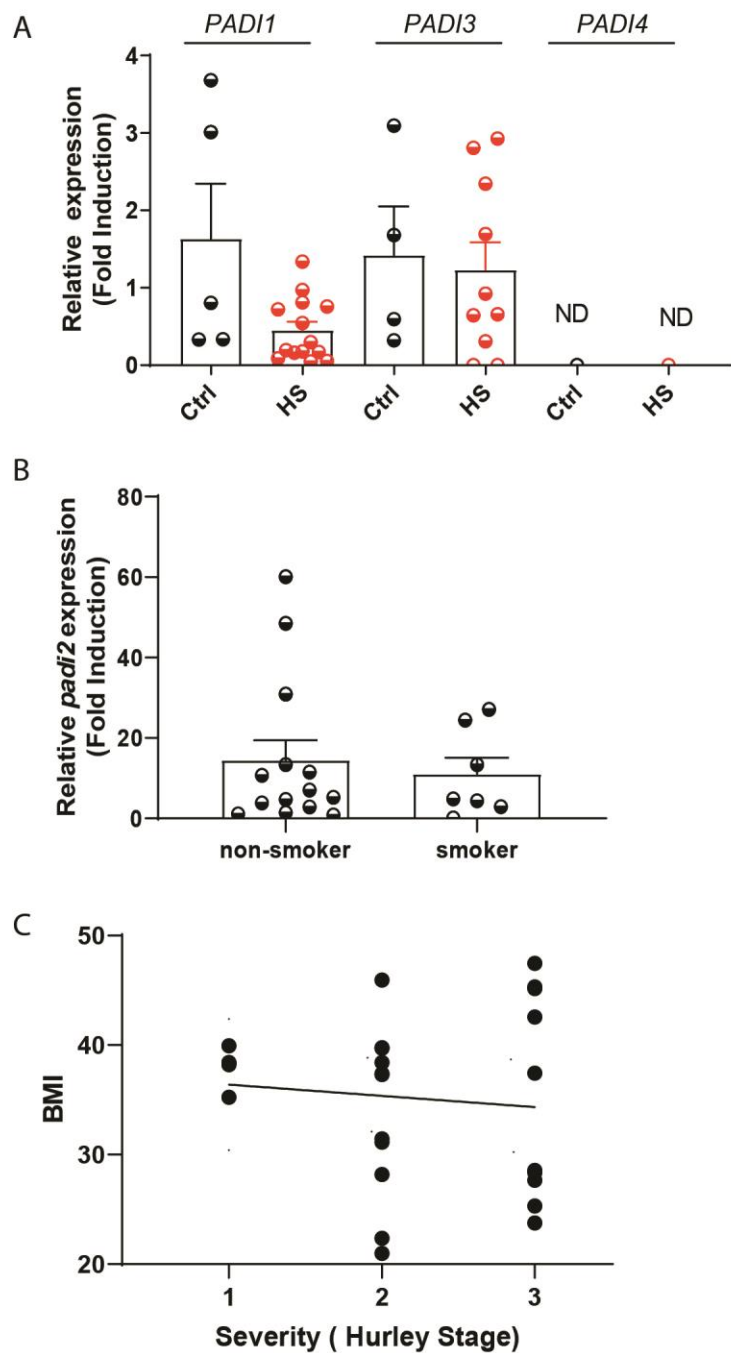


Fig. S2. Analysis of PAD expression and BMI in HS. (A) Control (Ctrl; n=4-5) and HS (n=10-14) skin were analyzed for the presence of *PADI1* and *PADI3* expression according to Material and Methods. Results are reported as relative gene expression (fold induction) and are the

mean +/- SEM. ND, not detected. **(B)** Smoker (Ctrl; n=7) and non-smoker (n=14) HS patients were analyzed for the presence of *PADI2* expression according to Material and Methods. Results are reported as relative gene expression (fold induction) and are the mean +/- SEM. **(C)** Correlation between Body mass index (BMI) and disease severity (Hurley stage) (Stage 1 n=3; Stage II n= 8; Stage III n=8). Mann-Whitney *U* test analysis was used.

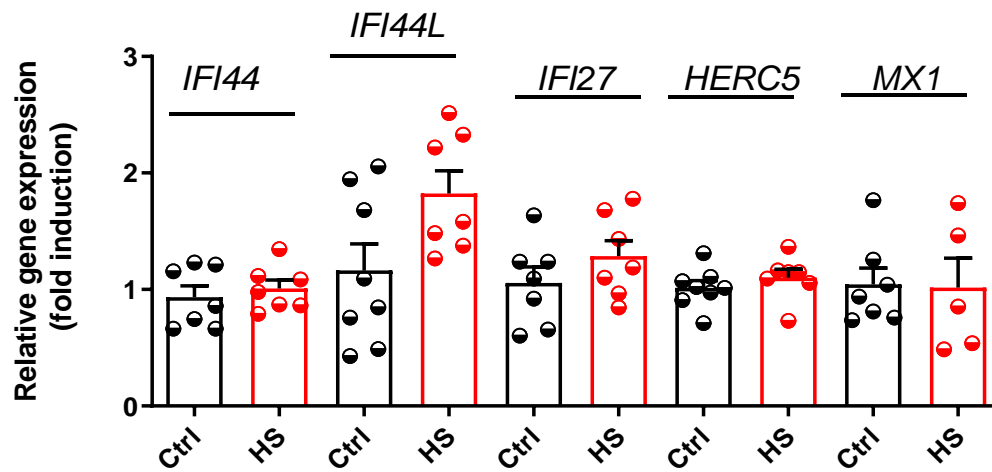


Fig. S3. Type I IFN serum activity is not increased in HS serum. Quantitative PCR for type I IFN inducible genes was performed on HeLa cells incubated with either control (n=7) or HS (n=7) sera for 6 hours according to Material and Methods. Mann-Whitney *U* test analysis was used.

Table S1. Correlation analysis of anti-TNF treatment with B cells and autoantibodies.

Linear regression was performed to determine the influence of anti-TNF treatment on B cell activation, IgG production, and presence of autoantibodies.

	anti-TNF treatment	
	R	p value
B cells	0.3019	0.3374
activated B cells	0.1753	0.5814
plasma cells	0.05449	0.7055
IgG	0.4036	0.2494
autoantibodies	0.008467	0.7544