ABSTRACT BOOK ABSTRACTS



A new ERA for global Dermatology 10 - 15 JUNE 2019 MILAN, ITALY

ACNE, ROSACEA, AND RELATED DISORDERS (INCLUDING HIDRADENITIS SUPPURATIVA)

IDENTIFICATION OF AUTOANTIBODIES IN PATIENTS WITH HIDRADENITIS SUPPURATIVA USING PHIP-SEQ

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Introduction: Hidradenitis suppurativa (HS) is a chronic, debilitating inflammatory disease that disproportionally affects African American females. It is characterized by recurrent painful draining nodules, fistulas, and scarring in intertriginous regions. Recent studies have shown the role of immune dysregulation in HS pathogenesis. Though appreciated in other autoimmune diseases, autoantibody-mediated immune dysregulation has yet to be appreciated in HS. Thus, we sought to characterize autoreactivity in patients with HS using a high-throughput phage immunoprecipitation sequencing (PhIP-Seq) screening technique.

Objective: To provide an unbiased, proteomic-scale assessment of autoreactivities contained within the serum of HS patients by screening the human peptidome.

Materials and Methods: Sera samples were collected from 17 distinct HS patients after informed consent was obtained and under an approved Institutional Review Board protocol. Patient demographics were as follows: 13 females, 4 males, 15 blacks, 2 whites, and average age of 38.5 years. The phage immunoprecipitation and sequencing library was prepared and HS sera samples were compared to a control population of 519 healthy samples from the NIH Vaccine Research Center (VRC) clinical trials. Sequencing data was generated from PhIP-Seq experiments and analyzed on python and R software.

Results: The following candidate autoantigens, in order of decreasing hit frequency, were noted to have significantly increased autoreactivity in HS patients when compared to controls: sperm-associated antigen 1 (SPAG1), cyclic AMP-responsive element-binding protein 3-like protein 3 (CREB3L3), filaggrin-2 (FLG2), and A-kinase anchor protein 13 (AKAP13).











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Conclusions: In this novel application of PhIP-Seq, we identified candidate antigens not previously associated with HS. Our preliminary findings suggest that autoantibodies to these antigens may play a role in HS pathogenesis. Understanding the role of autoantibodies in HS may one day enable development of personalized therapies.



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