- Yao Y, Jørgensen AR, Thomsen SF. Work productivity and activity impairment in patients with hidradenitis suppurativa: a cross-sectional study. *Int J Dermatol*. 2020;59(3):333-340.
- Sandhu VK, Shah M, Piguet V, Alavi A. The impact of Hidradenitis suppurativa on work productivity and activity impairment. Br J Dermatol. 2020;182(5):1288-1290.

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Serum cytokine levels in patients with hidradenitis suppurativa vary with race



To the Editor: Hidradenitis suppurativa (HS) is a chronic condition characterized by painful nodules and abscesses in intertriginous areas that can lead to disfigurement. The presumed immunopathogenesis of HS involves an exaggerated response to ruptured follicles. Aberrant cytokine expression has been demonstrated in the serum of patients with HS, specifically, increased serum tumor necrosis factor (TNF)- α , interleukin (IL)-17, and IL-1 β . Although biologics targeting TNF- α and IL-1 have shown success, the quest for additional treatment options remains ongoing.

In this institutional review board—approved study, we aimed to characterize serum cytokine profiles in HS. Adults with HS and healthy age-, sex-, and race-matched control individuals were recruited from a tertiary care dermatology clinic in New Orleans, Louisiana. Individuals with active infection, cancer, or other autoimmune disease were excluded. Additionally, those with use of systemic medication (systemic antibiotics, antiandrogen therapy, immunosuppressants, immunomodulators) within 1 month of sample collection, or biologics within 6 months, were excluded.

Demographic information and blood samples were collected. Twenty-five cytokines were quantified in duplicate using a multiplex assay (Millipore Human TH17 Magnetic Bead Panel [Millipore, Burlington, MA] and Luminex xMAP technology [Luminex, Austin, TX]).

Demographics were analyzed using a 2-tailed t test for continuous variables and chi-square and Fisher's exact tests for noncontinuous variables. Mann-Whitney and Kruskal-Wallis tests were used when evaluating cytokine levels. For data below the detection limit, a value of 0 was used. A P value of \leq .05 was considered significant.

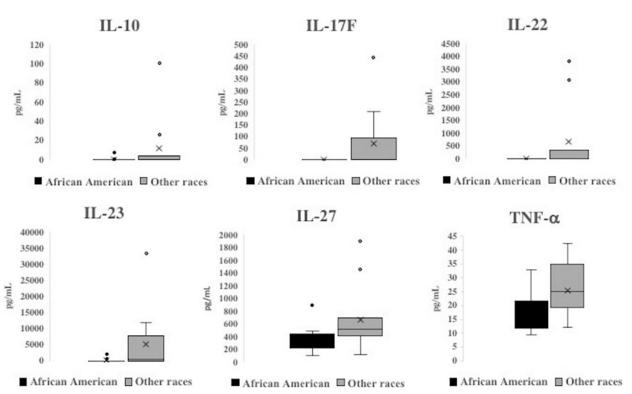


Fig 1. Cytokine levels in African American patients versus those of other races. A comparison of serum concentrations of TNF- α , IL-22, IL-23, IL-17F, IL-27, and IL-10 in African American patients (n = 16) and those of other races (n = 11) with hidradenitis suppurativa. *IL*, Interleukin; *TNF*, tumor necrosis factor.

In total, 27 patients with HS and 9 control individuals were included. There were no statistical differences between the cohorts based on sex, age, race, or body mass index. The HS cohort was majority female (92.6%), African American race (59.3%), with an average age of 37.3 ± 14.5 years and average body mass index (BMI) in the obese range of $35.5 \pm 8.2 \, \text{kg/m}^2$. The control group was also majority female (88.9%) and of African American race (66.7%), with an average age of 37.1 ± 13.3 years and average BMI in the obese range (32.9 \pm 6.8 kg/m²).

No statistically significant differences were found in cytokine levels for patients with HS versus control individuals. Additionally, multivariate analysis of variance failed to show variation based on BMI, smoking status, disease severity, or duration. Upon subgroup analysis by race, the following 6 cytokines were lower in African American patients with HS (n = 16) compared to other races (n = 11): TNF- α (P = .01), IL-22 (P = .05), IL-23 (P = .01), IL-17F (P = .01), IL-27 (P = .05), and IL-10 (P = .03) (see Fig 1).

African American race has been associated with increased HS prevalence, treatment resistance, and increased risk of other fibroproliferative disorders. This risk is thought to be due to evolutionary gene selection protecting against helminth infections. Previously demonstrated racial variation in inflammatory genes may in part explain differences in susceptibility and outcomes. Further genetic studies are needed to evaluate this relationship.

A limitation of this study includes the small sample size. Larger studies evaluating the immunopathogenesis of HS are needed. Furthermore, whether variation in cytokine levels among races in patients with HS contributes to treatment outcomes is yet to be determined. At the least, race should be reported in studies evaluating the etiology and treatment of HS. Whenever possible, efforts should be made to include a racially diverse cohort in HS research so that results can be translated to real-world clinical practice.

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Reprints not available from the authors.

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REFERENCES

- Matusiak L, Bieniek A, Szepietowski JC. Increased serum tumour necrosis factor-alpha in hidradenitis suppurativa patients: is there a basis for treatment with anti-tumour necrosis factor-alpha agents? Acta Derm Venereol. 2009;89: 601-603.
- 2. Matusiak L, Szczech J, Bieniek A, et al. Increased interleukin (II)-17 serum levels in patients with hidradenitis suppurativa: implications for treatment with anti-II-17 Agents. *J Am Acad Dermatol*. 2017;76:670-675.
- Jimenez-Gallo D, de la Varga-Martinez R, Ossorio-Garcia L, et al. The clinical significance of increased serum proinflammatory cytokines, C-reactive protein, and erythrocyte sedimentation rate in patients with hidradenitis suppurativa. *Mediators Inflamm*. 2017;2017:2450401.
- Garg A, Kirby JS, Lavian J, et al. Sex- and age-adjusted population analysis of prevalence estimates for hidradenitis suppurativa in the United States. *JAMA Dermatol.* 2017;153(8): 760-764.
- Van Dyke AL, Cotea ML, Wenzlaffa AS, et al. Cytokine SNPs: comparison of allele frequencies by race and implications for future studies. Cytokine. 2009;46(2):236-244.

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Real-world experience of adalimumab in the treatment of hidradenitis suppurativa



To the Editor: Adalimumab, a fully human IgG monoclonal antibody that targets tumor necrosis factor- α , is the only approved drug to treat moderate-severe hidradenitis suppurativa (HS).^{1,2}