

The gut microbiota-follicular axis in pilonidal sinus disease: lessons from hidradenitis suppurativa and surgical implications

 Deniz Kütük*,  Gürkan Değirmencioğlu

Department of General Surgery, Ankara Etlik City Hospital, Ankara, Türkiye

Cite this article as: Kütük D, Değirmencioğlu G. The gut microbiota-follicular axis in pilonidal sinus disease: lessons from hidradenitis suppurativa and surgical implications. *J Compr Surg.* 2026;4(4):106-111. doi:10.51271/JOCS-0086

Received: 14.06.2026

Accepted: 18.07.2026

Published: 28.10.2026

ABSTRACT

Pilonidal sinus disease (PSD) and hidradenitis suppurativa (HS) are both members of the follicular occlusion tetrad—a group of chronic inflammatory disorders sharing a common pathogenetic basis of follicular occlusion, subcutaneous tract formation, and recurrent abscess development. While PSD has traditionally been approached as a purely mechanical and anatomical problem, emerging evidence from the HS literature reveals a compelling gut microbiota-skin axis that may be equally operative in PSD. In HS, intestinal dysbiosis, increased intestinal permeability, and elevated systemic lipopolysaccharide levels have been documented through multiple study designs including Mendelian randomization analyses, providing causal evidence that gut microbial imbalance drives systemic immune dysregulation and amplifies local follicular inflammation. Both diseases harbor dysbiotic, anaerobic-predominant, biofilm-forming local microbiota—particularly *Prevotella* and *Finegoldia* species—capable of perpetuating chronic inflammation and resisting surgical eradication. This narrative review synthesizes current evidence on the gut microbiota-follicular axis in HS and PSD, examines the shared immunological and microbial mechanisms underlying both conditions, and proposes that the gut-skin axis insights established in HS are pathobiologically transferable to PSD. Importantly, PSD carries an understudied but emerging association with inflammatory bowel disease, mirroring the established HS-IBD comorbidity and further supporting a shared gut-driven inflammatory pathway. Microbiome-targeted adjunctive strategies—including perioperative probiotic supplementation, biofilm-disrupting local therapies, and dietary modulation—represent rational, evidence-informed approaches to reducing recurrence and postoperative complications in PSD surgery. We propose a structured research agenda for microbiome-integrated prospective PSD cohort studies and call for the first randomized trials of gut-targeted interventions in PSD surgical management. This is a narrative, non-systematic review; direct microbiome data in PSD remain limited, and the proposed framework requires prospective validation.

Keywords: Pilonidal sinus disease, hidradenitis suppurativa, gut microbiota, gut-skin axis, dysbiosis, wound healing

INTRODUCTION

Pilonidal sinus disease (PSD) is a chronic, recurrent inflammatory condition of the sacrococcygeal region affecting predominantly young males in the second and third decades of life, with an estimated annual incidence of 26 per 100,000 persons in high-income countries.^{1,2} Despite a broad repertoire of surgical approaches—from midline excision to off-midline flap reconstructions and minimally invasive endoscopic techniques—recurrence rates following surgical treatment range from 10% to 30%, and a meaningful proportion of patients experience repeated episodes of disease.^{3,4} The dominant explanatory framework for PSD remains mechanical: hair follicle distension, skin maceration in the intergluteal cleft, and forceful hair penetration into subcutaneous tissue drive sinus formation.⁵ Yet this anatomical paradigm fails to explain why certain individuals experience chronic recalcitrant disease despite technically satisfactory surgery, or why systemic inflammatory markers are elevated in a subset of patients.

Hidradenitis suppurativa (HS)—a chronic, debilitating inflammatory disorder of the pilosebaceous unit—shares with PSD a common pathogenetic root: follicular occlusion.^{6,7} Both conditions belong to the follicular occlusion tetrad (FOT), a nosological grouping that includes HS, PSD, acne conglobata, and dissecting cellulitis of the scalp, all of which demonstrate follicular hyperkeratosis, subcutaneous tract formation, anaerobic-dominant microbiota, and high surgical recurrence rates.^{8,9} A 2023 systematic review and meta-analysis confirmed a significantly elevated prevalence of PSD among HS patients compared with unaffected controls, with some authors proposing that PSD may represent a uni-localized anatomical variant of HS.¹⁰

The past decade has seen remarkable advances in understanding the gut microbiota-skin axis in HS. Multiple studies have documented intestinal dysbiosis in HS patients—characterized by reduced microbial diversity, depletion of short-chain fatty acid (SCFA)-producing commensals, and elevated intestinal

*Corresponding Author: Deniz Kütük, denizkutuk01@gmail.com



permeability-with Mendelian randomization analyses providing causal evidence linking specific gut microbial taxa to HS susceptibility.^{11,12} Concurrently, the established comorbidity between HS and inflammatory bowel disease (IBD) points to shared immunological pathways operating at the gut-immune-skin interface.¹³ Crucially, these mechanisms-systemic LPS translocation, SCFA depletion, and Th17/Treg imbalance-are precisely those postulated to impair wound healing, amplify follicular inflammation, and sustain chronic sinus disease.

If PSD and HS share not only their follicular pathogenesis but also their systemic immune and microbial dysregulation, then the gut microbiota-skin axis insights established in HS carry direct and transferable implications for PSD-both in understanding its pathophysiology and in designing rational adjunctive therapies. This narrative review aims to: (1) establish the shared pathobiological basis of PSD and HS within the follicular occlusion tetrad framework; (2) synthesize evidence for gut microbiota dysregulation in HS and its mechanistic relevance to follicular inflammation and wound healing; (3) characterize the local microbiome of PSD and its surgical implications; (4) evaluate the emerging PSD-IBD relationship as evidence of a shared gut-driven inflammatory axis; and (5) propose microbiome-targeted adjunctive strategies for PSD surgical management and a structured future research agenda.

PILONIDAL SINUS DISEASE AND HIDRADENITIS SUPPURATIVA: A SHARED PATHOBIOLOGICAL FRAMEWORK

The Follicular Occlusion Tetrad

The concept of the FOT, first consolidated in the 1970s, recognizes that PSD, HS, acne conglobata, and dissecting cellulitis of the scalp share a common initiating event: hyperkeratotic plugging of the follicular infundibulum.⁸ In each condition, follicular occlusion leads to retention of sebaceous secretions, follicular distension, and eventual rupture into the dermis and subcutaneous tissue, triggering a foreign body-type granulomatous response and recruitment of neutrophils, macrophages, and lymphocytes.⁶ The resulting chronic inflammatory cycle perpetuates tract formation, abscess recurrence, and fibrosis. A 2025 histologic and immunohistochemical study comparing PSD and HS tissue microenvironments found comparable expression of cytokeratin 5/6, KLK7, and filaggrin in both conditions,

confirming their shared epithelial and inflammatory signatures at the tissue level.¹⁴

The clinical parallels are equally striking. Both PSD and HS present predominantly in young adults, are associated with obesity, metabolic syndrome, and smoking as modifiable risk factors, and are characterized by high post-surgical recurrence rates driven by factors beyond technical surgical adequacy.^{3,15} A 2025 study in the Journal of the American Academy of Dermatology compared malignant transformation risk between HS and PSD, further cementing their classification as related follicular disorders on a shared biological spectrum.¹⁶

Microbial Overlap Between Pilonidal Sinus Disease (PSD) and HS

Both PSD and HS harbor dysbiotic, anaerobic-predominant local microbiota that differ substantially from adjacent healthy skin. In HS, skin microbiome studies have consistently identified *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Prevotella*, *Porphyromonas*, and anaerobic streptococci as dominant taxa in lesional tissue, with significant enrichment of biofilm-forming organisms.^{17,18} In PSD, 16S rRNA sequencing has similarly revealed a predominance of *Prevotella bivia*, *Fingoldia magna*, *Peptostreptococcus*, and *Bacteroides* species within sinus tracts.^{19,20} The comparative microbiological and clinicopathological features of HS and PSD are summarized in Table. The functional convergence is notable: in both diseases, local dysbiosis is characterized by organisms capable of TLR2/4-mediated immune activation, biofilm formation, and proteolytic extracellular matrix degradation-mechanisms that collectively sustain chronic inflammation and impair wound closure.^{21,22}

Biofilm formation is a particularly important shared feature. In both conditions, polymicrobial biofilms embedded within sinus and fistula tracts exhibit markedly elevated antibiotic tolerance-estimated at 100-1,000-fold higher than planktonic forms-potentially explaining the failure of systemic antibiotic therapy to achieve disease resolution and the high recurrence rates observed after surgical drainage alone.²³ Recognition of this shared biofilm biology has prompted interest in biofilm-disrupting local therapies in both HS and PSD, as detailed in section 5.

Table. Comparative clinicopathological and microbiological features of HS and PSD within the follicular occlusion tetrad framework

Feature	Hidradenitis suppurativa	Pilonidal sinus disease	Shared?
Pathogenetic basis	Follicular occlusion & rupture	Follicular occlusion & hair penetration	✓ Yes
Classification	Follicular occlusion tetrad	Follicular occlusion tetrad	✓ Yes
Local microbiota	Anaerobes, <i>Staphylococcus</i> , <i>Prevotella</i>	<i>Prevotella bivia</i> , <i>Fingoldia magna</i>	✓ Yes
Biofilm formation	Documented, clinically relevant	Documented, clinically relevant	✓ Yes
Gut-skin axis evidence	Mendelian randomization, cohort data	Hypothesized, no direct data	Partial
Intestinal dysbiosis	↑ Permeability, ↓ diversity (proven)	Unexplored	Extrapolation
Systemic inflammation	↑ TNF-α, IL-1β, IL-17 (systemic)	Local dominant, systemic unclear	Partial
IBD comorbidity	Established (OR 2-4)	Reported, understudied	Partial
Surgical recurrence	High (30-50%)	High (10-30%)	✓ Yes
Probiotic evidence	Emerging case reports	None published	Gap

HS: Hidradenitis suppurativa, PSD: Pilonidal sinus disease, ✓ Yes: Feature confirmed in both conditions, TNF-α: Tumor necrosis factor-alpha, IL-1β: Interleukin-1 beta, IL-17: Interleukin-17, IBD: Inflammatory bowel disease, SCFA: Short-chain fatty acids, OR: Odds ratio, Partial: Confirmed in one, suggested in other, Extrapolation: Mechanistically inferred from HS data, Gap: Unaddressed in PSD literature

GUT MICROBIOTA IN HIDRADENITIS SUPPURATIVA: THE EVIDENCE BASE

Intestinal Dysbiosis in Hidradenitis Suppurativa (HS): Observational Data

Growing evidence demonstrates that HS patients exhibit measurable alterations in gut microbiota composition compared with healthy controls. A 2024 prospective cohort study by Lelonek and Szepietowski¹² assessed fecal microbiome composition in 80 participants (40 HS patients, 40 controls) using next-generation sequencing, identifying significant differences in the relative abundance of specific taxa-including enrichment of pro-inflammatory Proteobacteria and depletion of SCFA-producing Firmicutes-in HS patients compared to controls.²⁴ A subsequent 2025 study by the same group using comprehensive metagenomic analysis identified correlations between specific gut microbial functional pathways, LPS biosynthesis gene abundance, and HS disease severity as measured by Hurley staging.²⁵ These data suggest that gut dysbiosis in HS is not a non-specific epiphenomenon but reflects biologically relevant alterations in taxa and pathways with direct immunological consequences.

A systematic review of skin and gut microbiome alterations in HS (Moreno-Arrones et al.,¹⁷ 2023) identified consistent patterns of reduced gut microbial diversity and decreased abundance of *Faecalibacterium prausnitzii*-a key butyrate producer and anti-inflammatory commensal-across multiple study designs. Reduced *Faecalibacterium* abundance has been independently associated with increased intestinal permeability, systemic LPS translocation, and elevated serum IL-6 and TNF- α levels in inflammatory conditions beyond HS, providing a mechanistic link between gut dysbiosis and systemic immune amplification relevant to all FOT members.²⁶

Causal Evidence: Mendelian Randomization (MR)

Perhaps the most compelling evidence for a causal gut-HS relationship comes from a 2024 two-sample MR study by Liu et al.,¹¹ which used genome-wide association study (GWAS) data to assess causal relationships between specific gut microbial taxa and HS susceptibility. The MR framework-which uses genetic variants as instrumental variables to minimize confounding-identified causal associations between reduced abundance of *Bifidobacterium* and *Lactobacillus* genera and increased HS risk, as well as between elevated *Ruminococcus gnavus* a pro-inflammatory taxon associated with intestinal barrier disruption-and HS susceptibility.¹¹ These findings satisfy Hill's criteria for causality more robustly than observational data alone, and represent the strongest evidence to date that gut microbiome composition is not merely correlated with HS but causally involved in its pathogenesis.

Hidradenitis Suppurativa (HS)-Inflammatory Bowel Disease (IBD) Comorbidity: a Window into the Gut-Follicular Axis

The epidemiological association between HS and IBD is well-established and provides independent clinical evidence for shared gut-immune pathways operating across the FOT. Multiple population-based studies have documented odds ratios of 2.0 to 4.0 for IBD in HS patients compared with unaffected controls, with Crohn's disease showing a particularly strong association-likely reflecting the shared Th17-dominant immune environment, fistula formation

biology, and genetic susceptibility loci (including NOD2 and FUT2).¹³ The HS-IBD association is bidirectional: HS patients have elevated IBD risk, and IBD patients have elevated HS prevalence. Mechanistically, both conditions feature elevated intestinal permeability, dysbiosis-driven Th17 expansion, and tumor necrosis factor-alpha (TNF- α)-mediated mucosal inflammation-explaining why anti-TNF biologics (adalimumab) are effective in both.^{13,27}

The emergence of case series and retrospective data suggesting a PSD-IBD comorbidity-while less well-characterized than the HS-IBD relationship-provides preliminary evidence that the same gut-immune axis may operate in PSD. Given the FOT classification shared by HS and PSD, and the mechanistic plausibility of a shared gut-inflammatory axis, systematic evaluation of IBD prevalence in PSD cohorts is warranted and represents a meaningful research gap.

THE GUT MICROBIOTA-FOLLICULAR AXIS: MECHANISMS TRANSFERABLE TO PSD

Short-chain Fatty Acid (SCFA) Depletion and Systemic Immune Dysregulation

The key mechanistic pathways linking gut dysbiosis to follicular skin disease are illustrated in **Figure**. SCFAs-principally butyrate, propionate, and acetate-are produced by colonic fermentation of dietary fiber by resident commensals including *Faecalibacterium prausnitzii*, *Roseburia*, and *Bifidobacterium* species. SCFAs exert broad immunomodulatory effects: butyrate inhibits histone deacetylase (HDAC) activity, suppresses NF- κ B signaling, promotes regulatory T cell (Treg) differentiation, and reinforces intestinal tight junction integrity.²⁸ In states of gut dysbiosis-characterized by depletion of SCFA-producing taxa-these protective effects are lost, leading to systemic pro-inflammatory skewing. SCFA depletion has been directly associated with impaired keratinocyte proliferation, reduced fibroblast migration velocity, and diminished angiogenic signaling in wound healing models, providing a mechanistic link between gut dysbiosis and delayed cutaneous wound closure.²⁹ In the context of PSD surgery-which requires weeks of open wound healing in a highly contaminated anatomical site-SCFA-driven wound healing impairment could meaningfully contribute to poor outcomes.

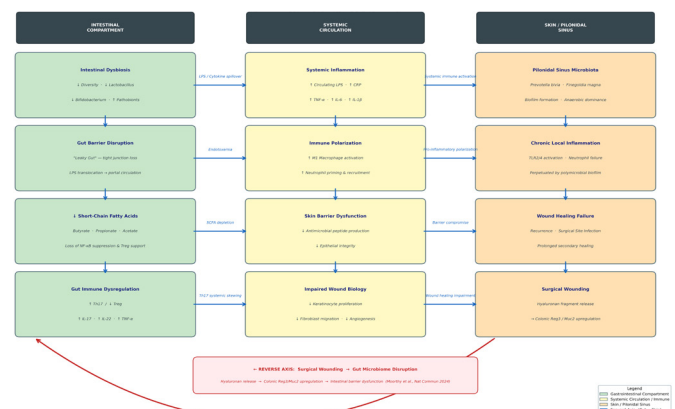


Figure. The gut microbiota-follicular axis in pilonidal sinus disease and hidradenitis suppurativa. The shared follicular occlusion tetrad framework places both conditions within a common pathobiological space

Gut dysbiosis drives systemic immune dysregulation through three parallel pathways: (1) SCFA depletion impairing Treg differentiation and wound healing; (2) intestinal barrier dysfunction and LPS translocation driving systemic TNF- α and IL-6 elevation; and (3) Th17/Treg imbalance amplifying local follicular inflammation. The reverse axis-by which surgical wounding releases hyaluronan fragments that upregulate colonic Reg3/Muc2 and disrupt intestinal microbiome homeostasis-provides a bidirectional link between skin surgery and gut microbial disruption. SCFA: Short-chain fatty acids, LPS: Lipopolysaccharide, SSI: Surgical site infection, IBD: inflammatory bowel disease, TNF- α : Tumor necrosis factor-alpha

Intestinal Permeability and Systemic Lipopolysaccharide (LPS) Translocation

Gut dysbiosis in HS is associated with increased intestinal epithelial permeability and translocation of bacterial LPS into the portal and systemic circulation—a state termed metabolic endotoxemia.³⁰ Circulating LPS activates systemic TLR4 signaling, triggering M1 macrophage polarization, neutrophil priming, and elevated production of TNF- α , IL-1 β , and IL-6. In the context of PSD, this systemic inflammatory tone may amplify local follicular inflammatory cascades, impair the resolution phase of wound healing, and lower the threshold for recurrent abscess formation. Elevated serum LPS levels have been demonstrated in HS patients and correlate with disease severity,³⁰ suggesting that LPS endotoxemia represents a quantifiable and potentially modifiable biomarker of gut-driven systemic inflammation in FOT diseases.

Th17/Treg Imbalance and Follicular Immunity

Gut dysbiosis drives systemic Th17/Treg imbalance through multiple pathways: reduced butyrate production impairs Treg differentiation from naïve T cells, while dysbiosis-driven segmented filamentous bacteria (SFB) expansion promotes Th17 polarization.³¹ The resulting excess of IL-17A, IL-17F, and IL-22 in the systemic circulation has been demonstrated in both HS and PSD patients compared with controls. IL-17 signaling at the skin level promotes neutrophil recruitment, keratinocyte activation, and amplification of the inflammatory cascade within follicular units.³² In PSD specifically, the anaerobic microbiota of sinus tracts—particularly *Prevotella* species—directly activate TLR2/4 signaling on resident macrophages, which in a Th17-primed systemic environment may result in disproportionate inflammatory amplification and impaired resolution. This mechanistic convergence suggests that gut-driven Th17 excess is not merely a bystander phenomenon in PSD but a pathophysiologically relevant contributor to chronicity.

MICROBIOME-TARGETED THERAPEUTIC STRATEGIES IN PILONIDAL SINUS DISEASE (PSD) SURGERY

Perioperative Probiotic Supplementation

The strongest transferable evidence for microbiome intervention in surgical settings comes from randomized trials in colorectal surgery, where perioperative probiotic supplementation—primarily with *Lactobacillus rhamnosus* GG, *Bifidobacterium longum*, and multi-strain formulations—has consistently demonstrated reductions in surgical site infection (SSI) rates, shortened antibiotic treatment duration, and accelerated wound healing in meta-analyses.^{33,34} The mechanistic basis is well-characterized: probiotics restore SCFA production, reinforce intestinal tight junction integrity, competitively exclude enteric pathogens, and modulate systemic immune polarization toward a Treg-dominant anti-inflammatory phenotype.³⁵ In HS, case reports and small pilot studies have documented symptom improvement and reduced flare frequency with oral probiotic supplementation, consistent with the gut-HS axis model.³⁶ No published trial has yet evaluated perioperative probiotics in PSD surgery, representing the most clinically proximate and methodologically tractable knowledge gap in the field.

Biofilm-Disrupting Local Therapies

Given the documented biofilm dominance in both PSD and HS sinus tracts, systemic antibiotics targeting planktonic bacteria provide inadequate local coverage of the biologically relevant pathogen population. In HS, topical clindamycin and systemic rifampicin combinations—which have anti-biofilm properties in addition to direct antimicrobial activity—are recommended in evidence-based guidelines, with mechanistic rationale grounded in biofilm disruption.¹⁵ For PSD, a prospective randomized trial by Arslan et al.³⁷ demonstrated that triclosan-coated sutures—which provide sustained antimicrobial activity at the wound interface—significantly reduced SSI rates compared with conventional sutures. Antiseptic wound irrigation with dilute hypochlorite or povidone-iodine solutions, and more recently negative pressure wound therapy (NPWT), represent further approaches to reducing local microbial bioburden in the early postoperative period.³⁸ Bacteriophage-based therapies targeting biofilm-embedded *Prevotella* and Staphylococcal species represent an emerging frontier applicable to both HS and PSD, and a comprehensive recent review by Singh et al.³⁹ provides further guidance on the parallel surgical management of these two conditions.

Dietary and Lifestyle Modulation

Dietary modification targeting gut microbiota composition represents a low-risk, accessible adjunctive strategy supported by the HS literature. A Mediterranean-pattern diet—rich in fermentable dietary fiber, polyphenols, and omega-3 fatty acids—has been associated with increased fecal SCFA concentrations, reduced intestinal permeability, and lower systemic inflammatory markers in HS patients.²⁴ Obesity—a shared risk factor for both HS and PSD—is independently associated with gut dysbiosis and reduced microbial diversity, and weight loss has been shown to improve HS outcomes through mechanisms that include microbiome normalization.¹⁵ For PSD, structured preoperative dietary intervention as part of a prehabilitation pathway, targeting gut microbiome optimization prior to surgical wounding, represents a testable and mechanistically rational strategy.

FUTURE RESEARCH AGENDA

The framework proposed in this review generates several high-priority research questions for the PSD field. First, fecal microbiome profiling should be incorporated into prospective PSD surgical cohort studies, comparing alpha- and beta-diversity, SCFA-producing taxa abundance, and intestinal permeability biomarkers (zonulin, fecal calprotectin) between primary and recurrent disease, and correlating these with postoperative outcomes. Second, the reverse skin-to-gut axis—demonstrated experimentally by Moorthy et al.,⁴⁰ who showed that dermal injury releases hyaluronan fragments that upregulate colonic Reg3/Muc2 and disrupt intestinal microbiome homeostasis—should be prospectively evaluated in PSD surgical cohorts through pre- and postoperative paired fecal sampling. Third, the PSD-IBD comorbidity should be systematically evaluated in large administrative database studies, paralleling the epidemiological methodology used to establish the HS-IBD relationship. Fourth, a multicenter randomized controlled trial evaluating perioperative probiotic supplementation in PSD surgery—with primary endpoints of wound healing duration and 12-month

recurrence, and secondary endpoints of SSI rate and fecal SCFA concentrations-is both feasible and urgently needed. Fifth, comparative microbiome profiling studies between HS and PSD using standardized tissue sampling and sequencing protocols would test the hypothesis of shared local dysbiotic signatures and identify common therapeutic targets. Sixth, Mendelian randomization analyses analogous to those conducted in HS should be performed using available PSD GWAS data to evaluate causal gut-PSD microbial associations.

Methodological standardization across future PSD microbiome studies is essential. Specimen collection protocols should specify anatomical sampling sites (sinus tract epithelium versus adjacent skin), storage conditions (RNAlater or immediate freezing at -80°C), DNA extraction methods, and sequencing depth targets. The establishment of multicenter PSD registries with embedded biobanking infrastructure would enable adequately powered microbiome-outcome association studies that single-center retrospective designs cannot provide.

CONCLUSION

Pilonidal sinus disease and hidradenitis suppurativa occupy a shared pathobiological space within the follicular occlusion tetrad, united by common mechanisms of follicular occlusion, dysbiotic local microbiota, biofilm-mediated chronicity, and high surgical recurrence. The robust evidence base linking gut microbiota dysregulation to HS through intestinal permeability, SCFA depletion, Th17 immune skewing, and causal Mendelian randomization data provides a well-evidenced framework that is mechanistically transferable to PSD. The emerging HS-IBD and PSD-IBD comorbidities further support the existence of a shared gut-driven inflammatory axis operative across the FOT. By reframing PSD within this gut-follicular axis framework-rather than as a purely mechanical anatomical problem-this review opens new avenues for understanding treatment failure and recurrence, and identifies microbiome-targeted adjunctive strategies as rational candidates for clinical investigation in PSD surgery.

The surgical community managing PSD has an opportunity to learn from the dermatological experience with HS and to accelerate microbiome research in its own field. Prospective cohort studies with integrated microbiome profiling, rigorous epidemiological evaluation of the PSD-IBD comorbidity, and the first randomized trials of perioperative probiotic supplementation in PSD surgery represent achievable near-term priorities. Ultimately, a microbiome-informed surgical approach to PSD-combining anatomical correction with systemic immune optimization-may represent the next frontier in reducing the disease's unacceptably high recurrence burden. As a narrative, non-systematic review, the present work is limited by the absence of direct microbiome data in PSD and the extrapolatory nature of the proposed gut-follicular axis framework; prospective validation in dedicated PSD cohorts is required before clinical translation.

ETHICAL DECLARATIONS

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: DK; Design: DK; Control: GD; Data Collection and/or Processing: GD; Analysis and/or Interpretation: DK; Literature Review: DK, GD; Writing the Article: DK, GD; Critical Review: DK, GD.

Editor Note

Gürkan Değirmencioglu is a member of the Editorial Board of this journal. He was not involved in the editor assignment, reviewer selection, peer review process, or editorial decision for this manuscript. The peer review process was managed independently by another editor.

REFERENCES

- Duman K, Girgin M, Harlak A. Prevalence of sacrococcygeal pilonidal disease in Turkey. *Asian J Surg.* 2017;40(5):434-437. doi:10.1016/j.asjsur.2015.10.005
- Hodges RM. Pilonidal sinus. *Boston Med Surg J.* 1880;103:485-486.
- Wu P, Zhang Y, Wang S, Fan Z. Progress in the surgical treatment of sacrococcygeal pilonidal sinus: a review. *Int J Surg.* 2023;109(7):2388-2403. doi:10.1097/JS9.0000000000000481
- van Ramshorst GH, Huurman EA, Smeenk RM, et al. A nationwide snapshot study on outcomes one year after surgery for chronic pilonidal sinus disease. *Colorectal Dis.* 2024;26(6):1198-1209. doi:10.1111/codi.16959
- Karydakis GE. Easy and successful treatment of pilonidal sinus after explanation of its causative process. *Aust N Z J Surg.* 1992;62(5):385-389. doi:10.1111/j.1445-2197.1992.tb07208.x
- Zouboulis CC, Benhadou F, Byrd AS, et al. What causes hidradenitis suppurativa? 15 years after. *Exp Dermatol.* 2020;29(12):1154-1170. doi:10.1111/exd.14214
- Scala E, Cacciapuoti S, Garzorz-Stark N, et al. Hidradenitis suppurativa: where we are and where we are going. *Cells.* 2021;10(8):2094. doi:10.3390/cells10082094
- Jansen I, Altmeyer P, Piewig G. The follicular occlusion triad (hidradenitis suppurativa, acne conglobata and dissecting cellulitis of the scalp). *J Eur Acad Dermatol Venereol.* 2001;15(2):111-114. doi:10.1046/j.1468-3083.2001.00209.x
- Scheinfeld N, Sundaram M. Hidradenitis suppurativa and follicular occlusion syndrome: where is the pathogenetic link? *Clin Dermatol.* 2023;41(5):484-493. doi:10.1016/j.clindermatol.2023.08.012
- Wark KJL, Der Sarkissian SA, Tatian A, Woods J, Cains GD. The association between pilonidal sinus disease and hidradenitis suppurativa: a systematic review and meta-analysis. *Br J Dermatol.* 2023;188(5):673-675. doi:10.1093/bjd/ljac166
- Liu Y, Liu H, Li Z. Causal relationship between gut microbiota and hidradenitis suppurativa: a two-sample Mendelian randomization study. *Front Microbiol.* 2024;15:1355113. doi:10.3389/fmicb.2024.1355113
- Lelonek E, Szepietowski JC. Insights into gut microbiome composition in hidradenitis suppurativa: a comprehensive examination of dietary habits and environmental influences. *Nutrients.* 2024;16(11):1776. doi:10.3390/nu16111776
- Kokolakis G, Wolk K, Bartsch T, et al. Delayed diagnosis of hidradenitis suppurativa and its effect on patients and the healthcare system. *Dermatology.* 2020;236(5):421-430. doi:10.1159/000503023
- Karadag AS, Ozlu E, Akdeniz N, et al. Comparison of the microenvironments between pilonidal sinus disease and hidradenitis suppurativa. *Biotech Histochem.* 2025;100(5):1-10. doi:10.1080/10520295.2025.2518582
- Hendricks AJ, Hsiao JL, Lowes MA, Shi VY. A comparison of international management guidelines for hidradenitis suppurativa. *Dermatology.* 2021;237(1):81-96. doi:10.1159/000503605
- Khalid N, Yildiz H, Maverakis E, et al. Malignancy risk differs between hidradenitis suppurativa and pilonidal sinus disease. *J Am Acad Dermatol.* 2025;S0190-9622(25)00164-1. doi:10.1016/j.jaad.2025.01.036
- Moreno-Arrones OM, Serrano-Villar S, Fuentesláz V, et al. Skin and gut microbiome in hidradenitis suppurativa: a systematic review. *Biomedicine.* 2023;11(8):2277. doi:10.3390/biomedicine11082277

18. Johnson CE, Naik HB. Microbiome perturbations in hidradenitis suppurativa. *Dermatol Clin.* 2025;43(2):193-202. doi:10.1016/j.det.2024.12.005
19. Banerjee T, Singh A, Anurag, Pal S, Basu S. Emergence of unusual microorganisms in microflora of pilonidal sinuses: a multiple case series. *J Wound Ostomy Continence Nurs.* 2023;50(1):26-30. doi:10.1097/WON.0000000000000934
20. Wolcott RD, Hanson JD, Rees EJ, et al. Analysis of the chronic wound microbiota of 2,963 patients by 16S rDNA pyrosequencing. *Wound Repair Regen.* 2016;24(1):163-174. doi:10.1111/wrr.12370
21. Hajishengallis G, Darveau RP, Curtis MA. The keystone-pathogen hypothesis. *Nat Rev Microbiol.* 2012;10(10):717-725. doi:10.1038/nrmicro2873
22. Murphy EC, Frick IM. Gram-positive anaerobic cocci-commensals and opportunistic pathogens. *FEMS Microbiol Rev.* 2013;37(4):520-553. doi:10.1111/1574-6976.12005
23. Malone M, Bjarnsholt T, McBain AJ, et al. The prevalence of biofilms in chronic wounds: a systematic review and meta-analysis of published data. *J Wound Care.* 2017;26(1):20-25. doi:10.12968/jowc.2017.26.1.20
24. Lelonek E, Krajewski PK, Szepietowski JC. Gut microbiome correlations in hidradenitis suppurativa patients. *J Clin Med.* 2025;14(14):5074. doi:10.3390/jcm14145074
25. Nikolakis G, Zouboulis CC, Stratigos A, et al. Unveiling the microbiome's role in hidradenitis suppurativa: a comprehensive review of pathogenetic mechanisms. *Int J Mol Sci.* 2025;26(19):9876.
26. Ney LM, Wipplinger M, Grossmann M, Engert N, Wegner VD, Mosig AS. Short chain fatty acids: key regulators of the local and systemic immune response in inflammatory diseases and infections. *Open Biol.* 2023;13(3):230014. doi:10.1098/rsob.230014
27. Rennoll SA, Weaver CT, Elson CO. Mucosal immunity in inflammatory bowel disease and hidradenitis suppurativa: shared immunological pathways. *J Crohns Colitis.* 2024;18(3):340-352. doi:10.1093/ecco-jcc/jjad148
28. Chai J, Zhuang Y, Cui K, Bi Y, Zhang N. Gut microbiota dysbiosis, oxidative stress, inflammation, and epigenetic alterations in metabolic diseases. *Antioxidants (Basel).* 2024;13(8):985. doi:10.3390/antiox13080985
29. Mihail MM, Bălăceanu-Gurău B, Ion A, et al. Host-microbiome crosstalk in chronic wound healing. *Int J Mol Sci.* 2024;25(9):4629. doi:10.3390/ijms25094629
30. De Pessemer B, Grine L, Debaere M, Maes A, Paetzold B, Callewaert C. Gut-skin axis: current knowledge of the interrelationship between microbial dysbiosis and skin conditions. *Microorganisms.* 2021;9(2):353. doi:10.3390/microorganisms9020353
31. Sinha S, Lin G, Ferenczi K. The skin microbiome and the gut-skin axis. *Clin Dermatol.* 2021;39(5):829-839. doi:10.1016/j.clindermatol.2021.05.008
32. Piazzesi A, Lamminmäki A, Lehtimäki J. Modulations of the skin microbiome in skin disorders: a narrative review from a wound care perspective. *Int Wound J.* 2024;21(1):e70087. doi:10.1111/iwj.70087
33. Wan B, Zhang X, Xia Z, Jiang Y. Effect of perioperative probiotic supplementation on postoperative infection complications and inflammatory response in colorectal cancer: systematic review and meta-analysis. *Front Oncol.* 2022;12:845597. doi:10.3389/fonc.2022.845597
34. Liu Z, Qin H, Yang Z, et al. Randomised clinical trial: the effects of perioperative probiotic treatment on barrier function and post-operative infectious complications in colorectal cancer surgery-a double-blind study. *Aliment Pharmacol Ther.* 2011;33(1):50-63. doi:10.1111/j.1365-2036.2010.04492.x
35. Gao T, Wang X, Li Y, Ren F. The role of probiotics in skin health and related gut-skin axis: a review. *Front Microbiol.* 2023;14:1221672. doi:10.3389/fmicb.2023.1221672
36. Nikolakis G, Liakou AI, Bonovas S, et al. Oral probiotic supplementation in patients with hidradenitis suppurativa: results of a case series. *Acta Derm Venereol.* 2023;103:adv12173. doi:10.2340/actadv.v103.12173
37. Arslan NC, Atasoy G, Altintas T, Terzi C. Effect of triclosan-coated sutures on surgical site infections in pilonidal disease: prospective randomized study. *Int J Colorectal Dis.* 2018;33(10):1445-1452. doi:10.1007/s00384-018-3101-6
38. Huurman EA, Galema HA, de Raaff CAL, et al. Non-excisional techniques for the treatment of intergluteal pilonidal sinus disease: a systematic review. *Tech Coloproctol.* 2023;27(11):1191-1200. doi:10.1007/s10151-023-02842-5
39. Singh S, Desai K, Gillern S. Management of pilonidal disease and hidradenitis suppurativa. *Surg Clin North Am.* 2024;104(3):503-515. doi:10.1016/j.suc.2023.12.003
40. Moorthy AN, Stacy D, Jaber A, et al. Dermal injury drives a skin to gut axis that disrupts the intestinal microbiome and intestinal immune homeostasis in mice. *Nat Commun.* 2024;15(1):3009. doi:10.1038/s41467-024-47342-4