Journal of Skin Cancers, Conditions, Aesthetics and Reconstructive Surgery

Peer-reviewed and Open Access
Vol.1 No. May 2026
Accepted May 10, 2026

The Cutaneous Mycobiome in the Follicular Occlusion Tetrad: Evidence, Mechanisms, and Clinical Implications: A Systematic Review

Amna Y. Ibrahim, BS[a], Mona Y. Ibrahim, BS[a], Garrett Kraft, BA[a], Harib H. Ezaldein, MD[a]
[a] Miami Dermatology and Mohs Surgery, Department of Dermatologic Surgery Research, 4770 Biscayne Boulevard, Suite 900, Miami, Florida 33137

*** Corresponding author email address**: garrettkraft00@gmail.com

Abstract

The cutaneous mycobiome is an underexplored component of the skin microbiome with potential relevance to chronic inflammatory follicular disorders. While bacterial dysbiosis has been extensively studied, the contribution of fungal organisms to follicular occlusion diseases remains incompletely defined. This narrative review examines available evidence regarding fungal involvement in the follicular occlusion tetrad, including hidradenitis suppurativa, acne conglobata, dissecting cellulitis of the scalp, and pilonidal sinus. Across these conditions, fungi (most commonly Malassezia and Candida species) are detectable within follicular units but are not consistently enriched in lesional skin. Current data suggest that fungi function primarily as secondary colonizers, inflammatory modifiers, or diagnostic mimickers rather than primary pathogenic drivers. Evidence supporting a direct fungal role is most limited in pilonidal sinus disease and more frequently involves clinical mimickers in dissecting cellulitis and acne conglobata. Notably, immune pathways implicated in follicular occlusion disorders, including Th17-mediated and interleukin-17 signaling, also play central roles in antifungal host defense. Therapeutic modulation of these pathways may therefore influence fungal susceptibility. Overall, existing evidence does not support a primary pathogenic role for fungi but highlights their potential relevance in select clinical contexts.

Keywords: Skin mycobiome, Follicular occlusion tetrad, Hidradenitis suppurativa, Fungal dysbiosis

1. Introduction

The skin microbiome plays a critical role in maintaining cutaneous homeostasis and modulating inflammatory skin disease. While bacterial communities have been extensively characterized, comparatively less is known about fungal populations comprising the cutaneous “mycobiome” and their contributions to skin pathology. Representing approximately 10% of the total microbiome, fungal organisms may influence immune activation, particularly in the setting of epidermal barrier disruption, and have been implicated in chronic inflammatory dermatoses such as psoriasis.[1,2]

Beyond classic fungal dermatoses, the potential role of the mycobiome in follicular occlusion disorders remains poorly defined. It is unclear whether fungal populations act as primary contributors to disease pathogenesis, secondary colonizers following follicular disruption, or opportunistic organisms promoted by long-term antibiotic exposure commonly used in these conditions. The follicular occlusion tetrad (hidradenitis suppurativa (HS), acne conglobata, dissecting cellulitis of the scalp, and pilonidal sinus) represents a group of chronic inflammatory disorders unified by follicular obstruction, sinus tract formation, and overlapping clinical features.[3]

This review aims to evaluate existing dermatologic literature to assess evidence supporting a role for the mycobiome in the follicular occlusion tetrad. Specifically, we examine fungal species identified in association with each condition, proposed immunologic and pathogenic mechanisms, and potential clinical relevance.

2. Objectives

The objective of this narrative review is to evaluate existing evidence supporting fungal involvement in the follicular occlusion tetrad and to clarify whether fungi act as primary pathogens, secondary modifiers, or diagnostic mimickers. This review further aims to assess immunologic mechanisms linking fungal colonization to disease activity and to explore potential clinical implications for diagnosis and management.

3. Methods

This narrative review synthesizes peer-reviewed literature examining fungal species, immune interactions, and clinical associations within follicular occlusion disorders. A literature search was conducted across Google Scholar, PubMed, and Embase, with no publication year restrictions to include historical and contemporary studies. The search was initially performed in January 2025 and updated through December 2025 to incorporate recent publications. Search terms combined "mycobiome," "fungi," "Malassezia," "Candida," "fungal microbiome," and each tetrad condition ("hidradenitis suppurativa," "pilonidal sinus," "dissecting cellulitis of the scalp," "acne conglobata"). Reference lists of identified articles were hand-searched for additional relevant studies.

Inclusion criteria focused on English-language publications reporting fungal species, immune mechanisms, or clinical associations in these conditions, including clinical studies, case reports, sequencing analyses, and mechanistic reviews. Studies exclusively on bacterial microbiota without fungal components were excluded. No formal risk-of-bias assessment was conducted, consistent with narrative review methodology (Table 1). Key studies are summarized in Table 1. Various proposed fungal roles and colonization sites within the hair follicle exist (Figure 1).

4. Hidradenitis Suppurativa

Hidradenitis suppurativa (HS) is a chronic, inflammatory follicular disorder characterized by recurrent painful nodules, abscesses, and sinus tract formation, most commonly affecting intertriginous skin. Although its pathogenesis is multifactorial, aberrant follicular hyperkeratosis and apocrine unit dysfunction are considered central initiating events, leading to follicular rupture, chronic inflammation, and the development of draining sinus tracts.[4]

Inflammatory signaling in HS is dominated by dysregulated cytokine activity, particularly within the interleukin-1 (IL-1) family. Among these, IL-1β is markedly upregulated and promotes chemokine-driven recruitment of neutrophils and other immune cells, contributing to purulent inflammation.[5] IL-23 is also overexpressed within HS lesions and drives downstream activation of the Th17 pathway. IL-17, a key effector cytokine in Th17-mediated immunity, promotes neutrophil activation and plays a critical role in antifungal host defense, suggesting a mechanistic intersection between HS inflammation and fungal immune responses.[6]

Microbiome dysbiosis is a consistent feature of HS, although its role as a primary driver versus a secondary consequence of disease or antibiotic exposure remains debated. In early-stage HS (Hurley stage I), aerobic organisms such as Staphylococcus lugdunensis and Cutibacterium species are commonly isolated. In contrast, advanced disease (Hurley stages II–III) demonstrates a predominance of strictly anaerobic bacteria, including Porphyromonas and Prevotella, with relative depletion of early-stage commensals.[7] While bacterial communities have been extensively characterized, the fungal component of the HS microbiome has historically received limited attention. This gap is partly attributable to technical challenges in fungal identification, as fungal abundance and diversity vary by anatomic site and require specialized genomic approaches.[8]

A 2022 study provided the first in-depth characterization of the HS-associated mycobiome using high-throughput amplicon sequencing targeting the eukaryotic 18S ribosomal RNA gene.[9] Analysis of lesional, non-lesional, and healthy control skin samples identified Malassezia restricta, Saccharomyces cerevisiae, and Malassezia globosa as the most prevalent fungal species across all sample types. Fungal spores have also been visualized within active HS lesions localized to hair shafts, supporting the physical presence of fungi within diseased follicles.[10] Subsequent reviews by MacGibeny et al. and Rosi et al. highlighted these findings as foundational to emerging fungal research in HS.[11,12] Notably, no statistically significant differences in Malassezia abundance were observed between lesional and control skin, a finding potentially explained by reduced sebaceous gland density and altered pH within HS lesions that may permit fungal persistence without overt overgrowth.[9] Larger studies, particularly those including early-stage HS, are needed to clarify whether fungal alterations precede or follow disease progression.

Clinically, HS shares overlapping features with superficial fungal infections affecting intertriginous regions, including candidiasis and dermatophytosis. However, epidemiologic data do not support a direct association between HS and superficial fungal infection. A population-based study by Lindsø et al. found no increased prevalence of superficial fungal infections among individuals with HS in a cohort of Danish blood donors.[13] This study employed a validated HS questionnaire with high sensitivity and specificity, along with prescription registry data for antifungal medications such as miconazole and clotrimazole. While methodologically robust, limitations included the inability to assess cytokine expression and the exclusion of over-the-counter antifungal use.

The Th17–IL-17 axis represents a shared immunologic pathway linking HS with other inflammatory and autoimmune skin diseases and antifungal immunity.[14] The aryl hydrocarbon receptor (AHR), a transcription factor involved in Th17 differentiation and expressed on immune cells including T and B lymphocytes, has been implicated in chronic inflammatory diseases associated with fungal exposure.[15] These overlapping immune pathways suggest that fungal antigens may act as inflammatory amplifiers in HS rather than primary initiators of disease.

Therapeutic advances in HS increasingly target inflammatory cytokines central to disease pathogenesis. Tumor necrosis factor-α inhibitors such as adalimumab and infliximab are approved for moderate to severe HS, while IL-17 inhibitors including secukinumab and bimekizumab have more recently expanded treatment options. By suppressing IL-17 signaling, these biologics reduce neutrophil recruitment and keratinocyte activation; however, they may also increase susceptibility to fungal infections, including Candida species and Histoplasma capsulatum, underscoring the clinical relevance of fungal–immune interactions in HS management.[16]

5. Pilonidal Sinus Disease

Pilonidal sinus (PS) disease is a chronic inflammatory condition of the sacrococcygeal region characterized by subcutaneous nodules, abscesses, and sinus tract formation arising from hair follicle involvement. Clinically, PS may present as a solitary cyst, recurrent abscess, or chronic draining sinus, often developing after prolonged local inflammation or follicular rupture.

Proposed pathogenetic mechanisms include aberrant cytokeratin expression within the follicular epithelium, resulting in abnormal follicular proliferation and subsequent immunogenic effects.[17] Clinical overlap between PS and hidradenitis suppurativa has led to speculation that PS may represent a phenotypic variant within the follicular occlusion spectrum. Supporting this hypothesis, one study observed intergluteal involvement in approximately 27% of patients with HS, suggesting shared anatomic and pathogenic features. Microbiologic analysis of PS lesions demonstrates a predominance of mixed bacterial populations, including Actinomyces species (18%), pyogenic streptococci (9%), Corynebacterium species (21%), and anaerobes (40%), particularly Prevotella and Porphyromonas, mirroring bacterial profiles observed in gluteal HS lesions.[18]

Direct evidence implicating fungi in PS pathogenesis remains limited. Cases of actinomycosis involving pilonidal sinus have been reported, with histopathologic examination revealing filamentous gram-positive organisms with fungal-like morphology within infected tissue.[19] Although Actinomyces species are bacterial rather than fungal, their morphologic resemblance highlights diagnostic challenges in chronic follicular sinus disease and underscores the complexity of microbial involvement in PS.

Data specifically addressing the fungal mycobiome in pilonidal sinus disease are sparse. A case series describing nine instances of squamous cell carcinoma arising within long-standing sacrococcygeal pilonidal sinus tracts reported one patient who developed postoperative yeast fungemia following surgical intervention. Despite antifungal therapy, this patient succumbed to septic shock, illustrating the potential for severe opportunistic fungal infection in the setting of chronic disease and surgical disruption.[20] While this observation does not establish causality, it suggests that altered local or systemic immunity in advanced PS may permit fungal invasion under specific circumstances.

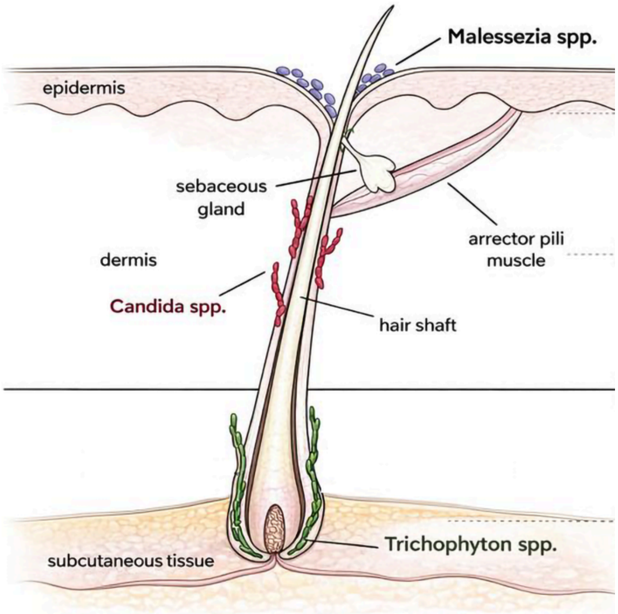

Figure 1. Conceptual schematic of the hair follicle unit illustrating proposed sites of fungal localization discussed across follicular occlusion disorders. The figure is intended to provide anatomic context and does not imply causation.

Additional associations between PS and fungal-related dermatoses have been described. One report noted an increased prevalence of seborrheic dermatitis in a patient with pilonidal cyst disease, raising the possibility of Malassezia overrepresentation in affected individuals, although fungal populations were not directly quantified.[21] At present, the contribution of fungal overgrowth or dysbiosis to pilonidal sinus development remains poorly defined. Existing evidence is largely anecdotal, emphasizing the need for targeted mycobiome analyses to clarify whether fungi play an active role in disease modulation or represent secondary colonizers in chronically inflamed tissue.

6. Dissecting Cellulitis

Dissecting cellulitis of the scalp (DCS) is a chronic inflammatory follicular disorder characterized by painful nodules, abscesses, and sinus tract formation of the scalp, frequently resulting in scarring alopecia if untreated. Although the precise etiology remains unclear, follicular occlusion followed by intense inflammatory response is thought to underlie disease development. Antibiotic therapy has historically been the cornerstone of treatment, reflecting the presumed contribution of microbial and inflammatory factors.

To date, no definitive fungal etiology has been established for DCS; however, several reports describe fungal infections that clinically mimic its presentation. Shastry et al. reported three pediatric cases initially misdiagnosed as DCS based on nodules and inflammatory plaques, in which fungal cultures ultimately identified Trichophyton species. Following antifungal therapy, all patients achieved complete hair regrowth without scarring, highlighting the importance of distinguishing true DCS from dermatophytosis masquerading as follicular occlusion disease.[22] Similarly, Stein et al. described a 19-year-old patient with inflammatory scalp alopecia resembling DCS, in whom fungal infection was later confirmed and successfully treated with oral and topical antifungals.[23] In this case, Trichophyton tonsurans remained detectable for several weeks after initiation of griseofulvin therapy, with complete clinical resolution observed at six months.[24] Collectively, these reports suggest that fungal infections may act as clinical mimickers of DCS rather than primary drivers of disease.

While immune mechanisms specific to DCS are incompletely characterized, pathogenic overlap with hidradenitis suppurativa provides insight into potential inflammatory pathways. Both conditions exhibit follicular occlusion and chronic inflammation, with evidence implicating tumor necrosis factor–α (TNF-α) signaling in disease activity. The use of TNF-α inhibitors has demonstrated clinical benefit in DCS, supporting a role for cytokine-mediated inflammation in disease progression.[25] Histopathologic studies of DCS lesions reveal lymphoplasmacytic and neutrophilic infiltrates, further reinforcing the contribution of innate and adaptive immune responses.[26] More recently, the Janus kinase (JAK) signaling pathway—previously implicated in HS—has been explored in DCS. In a reported case of a 26-year-old male with refractory disease, treatment with a JAK inhibitor resulted in marked clinical improvement, accompanied by downregulation of proinflammatory cytokines including IL-10, IL-12, and IL-23.[27]

Microbial colonization of the hair follicle may contribute to immune dysregulation in DCS through loss of tolerance to follicular antigens. Early lesions are typically sterile, though secondary bacterial infection may occur as disease progresses. Cultures have demonstrated the presence of coagulase-negative staphylococci, with Staphylococcus aureus isolated from lesions and abscesses in several patients, suggesting a contributory bacterial role in established disease.[28] In contrast, the fungal composition of the healthy scalp is diverse, comprising species from the phyla Ascomycota and Basidiomycota, including Acremonium species, Didymella bryoniae, Cryptococcus liquefaciens, Cryptococcus diffluens, Coniochaeta species, and Rhodotorula species.[29-31] Pediatric scalps demonstrate greater fungal diversity compared to adults aged 20–30 years, with relatively lower abundance of Malassezia species.[32]

Although direct mycobiome analyses in DCS are limited, Malassezia globosa and Malassezia restricta have been reported as predominant species in cases of pseudo–dissecting cellulitis of the scalp.[8,33] Malassezia species are known to influence cutaneous immune responses and have been implicated in inflammatory scalp conditions.[34] Their lipase activity facilitates the release of arachidonic acid, promoting inflammation, while interactions between fungal and bacterial populations may further disrupt microbial homeostasis and amplify inflammatory signaling.[35] In vivo confocal laser scanning microscopy has confirmed the presence of yeasts on the scalp, supporting their physical persistence within the follicular environment.[36] Together, these findings suggest that while fungi are unlikely to initiate DCS, they may contribute to disease modulation in select contexts.

Therapeutic strategies for DCS primarily target inflammation and follicular hyperkeratosis. Oral antibiotics such as doxycycline, azithromycin, and dapsone are commonly used for their anti-inflammatory effects. Retinoids, particularly isotretinoin, are effective due to their antiproliferative and immunomodulatory properties. Biologic therapies targeting TNF-α and IL-12/IL-23/IL-17 pathways, including adalimumab, infliximab, secukinumab, guselkumab, and risankizumab, have been employed in refractory cases, reflecting shared inflammatory pathways with other follicular occlusion disorders.[37]

7. Acne Conglobata

Acne conglobata (AC) is a severe, chronic form of inflammatory acne characterized by nodules, abscesses, cysts, and interconnected sinus tracts. The condition is uncommon but highly morbid, often resulting in scarring and significant psychosocial burden. Bacterial dysbiosis is a well-recognized component of AC pathogenesis, with Cutibacterium avidum and Cutibacterium acnes implicated through altered phylotype distribution, particularly dominance of C. acnes phylotype IA1. Additional bacterial contributors include Staphylococcus epidermidis, which may further influence follicular inflammation.[38]

The immune landscape of AC shares similarities with other follicular occlusion disorders, including neutrophilic and lymphocytic infiltration and dysregulated inflammatory signaling. Tumor necrosis factor–α (TNF-α) has been proposed as a key mediator of inflammation in AC, supporting parallels with hidradenitis suppurativa and related conditions.[39] Genetic contributions have also been described, including mutations in components of the γ-secretase complex such as NCSTN, which may disrupt follicular keratinization and epidermal differentiation. Polymorphisms in the toll-like receptor 4 (TLR4) gene have been associated with acne severity, although some variants may confer relative protection against AC. Additionally, mutations in proline-serine-threonine phosphatase–interacting protein 1 (PSTPIP1) have been identified in AC, with responsiveness to interleukin-1 inhibition highlighting potential therapeutic targets.[40]

Evidence implicating fungi in AC is limited but notable. A 1971 case report described a 17-year-old male with refractory AC in whom Candida albicans was cultured from drusen-like material expressed from pustules.[41] The author proposed that prolonged antibiotic therapy disrupted bacterial equilibrium, permitting secondary Candida overgrowth within hair follicles and surrounding tissue. The condition, termed “acne conglobata candidosa,” responded to prolonged antifungal therapy with bis-phenyl-(2-chlorophenyl)-1-imidazolyl-methane, resulting in near-complete clinical resolution. Although this observation was not subsequently expanded into larger studies, it raises the possibility that fungal overgrowth may complicate AC in the setting of antibiotic-induced dysbiosis.

Beyond isolated case reports, fungal species—particularly Malassezia—have been increasingly implicated in acneiform disorders more broadly. A 2018 study analyzing acne patients with suspected fungal involvement identified Candida species and highlighted the presence of lipase genes shared by both Candida and Malassezia, suggesting a potential mechanistic contribution to follicular inflammation.[42] Another study involving 32 patients isolated Malassezia yeasts from acne vulgaris comedones, identifying M. restricta, M. globosa, and M. furfur as predominant species.[43] These findings support the concept that Malassezia folliculitis and acne may coexist, overlap, or clinically mimic one another, complicating diagnosis. Indeed, Malassezia folliculitis is frequently misdiagnosed as acne vulgaris or steroid-induced acne due to similar clinical morphology and may, in severe cases, resemble acne conglobata.[44]

Standard treatment of AC relies on systemic therapy aimed at suppressing inflammation and follicular hyperkeratosis. Oral isotretinoin is commonly employed, often in combination with systemic corticosteroids to mitigate inflammatory flares. Dapsone serves as an alternative in refractory cases, and combination therapy with isotretinoin has demonstrated clinical benefit. Additional systemic agents, including colchicine and cyclosporine, may be considered in resistant disease. Targeted biologic therapies, particularly TNF-α antagonists such as adalimumab, have shown efficacy in severe AC, further underscoring shared inflammatory pathways across the follicular occlusion spectrum.[40]

8. Conclusion

This narrative review highlights emerging evidence suggesting that fungal organisms may contribute to the follicular occlusion tetrad, comprising hidradenitis suppurativa, acne conglobata, dissecting cellulitis of the scalp, and pilonidal sinus. While these disorders are classically defined by follicular occlusion, chronic inflammation, and bacterial dysbiosis, accumulating data indicate that components of the cutaneous mycobiome may act as modulators of disease activity rather than primary pathogenic drivers.

Across the tetrad, fungal species such as Malassezia and Candida are most consistently identified in association with follicular disease, though evidence varies substantially by condition. In hidradenitis suppurativa, mycobiome analyses demonstrate fungal persistence within lesional and non-lesional skin without clear overrepresentation, suggesting a potential role in immune amplification rather than disease initiation. Recent studies published between 2004 and 2025 continue to underscore the limited and conflicting evidence for fungal primacy in HS, with reduced lesional fungal prevalence in some cases and persistent calls for larger mycobiome-focused analyses.[45,46]

In acne conglobata and dissecting cellulitis of the scalp, fungi more commonly emerge as clinical mimickers or secondary colonizers in the setting of antibiotic exposure or follicular disruption. Evidence linking fungi to pilonidal sinus remains limited and largely anecdotal, underscoring the need for caution in attributing pathogenic significance.

Importantly, many immune pathways implicated in follicular occlusion disorders, including Th17-mediated signaling, IL-17 activity, and aryl hydrocarbon receptor–dependent responses, also play central roles in antifungal immunity. Therapeutic strategies targeting these pathways may therefore influence fungal colonization and susceptibility to opportunistic infection, highlighting the clinical relevance of understanding fungal–host interactions in this disease spectrum.

Despite growing interest in the cutaneous mycobiome, significant knowledge gaps remain. Current evidence is limited by small sample sizes, heterogeneous methodologies, and a reliance on case reports or indirect associations. Future studies employing standardized sequencing techniques, longitudinal sampling, and disease-stage stratification are needed to clarify whether fungal dysbiosis contributes meaningfully to disease progression or represents a secondary consequence of chronic inflammation and antimicrobial therapy. Elucidating the role of the mycobiome may inform more comprehensive diagnostic approaches and support the rational integration of antifungal strategies as adjuncts in the management of follicular occlusion disorders.

Acknowledgments

None

Artificial Intelligence Disclosure

Authors of this paper ensure that the manuscript complies with the Journal's strict prohibition on generative artificial intelligence in both text and clinical imagery.

References

1. Belvoncikova, P., Splichalova, P., Videnska, P. and Gardlik, R. (2022). The human mycobiome: colonization, composition and the role in health and disease. Journal of Fungi, 8, p. 1046.
2. El-Jurdi, N. and Ghannoum, M.A. (2017). The mycobiome: impact on health and disease states. Microbiology Spectrum, 5.
3. Vasanth, V. and Chandrashekar, B.S. (2014). Follicular occlusion tetrad. Indian Dermatology Online Journal, 5, pp. 491–493.

4. Alikhan, A., Lynch, P.J. and Eisen, D.B. (2009). Hidradenitis suppurativa: a comprehensive review. Journal of the American Academy of Dermatology, 60, pp. 539–561.
5. Witte-Händel, E., Wolk, K., Tsaousi, A., et al. (2019). The IL-1 pathway is hyperactive in hidradenitis suppurativa and contributes to skin infiltration and destruction. Journal of Investigative Dermatology, 139, pp. 1294–1305.
6. Melchor, J., Prajapati, S., Pichardo, R.O. and Feldman, S.R. (2024). Cytokine-mediated molecular pathophysiology of hidradenitis suppurativa: a narrative review. Skin Appendage Disorders, 10, pp. 172–179.
7. Zouboulis, C.C., Benhadou, F., Byrd, A.S., et al. (2020). What causes hidradenitis suppurativa? 15 years after. Experimental Dermatology, 29, pp. 1154–1170.
8. Findley, K., Oh, J., Yang, J., et al. (2013). Topographic diversity of fungal and bacterial communities in human skin. Nature, 498, pp. 367–370.
9. Ring, H.C., Thorsen, J., Fuursted, K., et al. (2022). Amplicon sequencing demonstrates comparable follicular mycobiomes in patients with hidradenitis suppurativa compared with healthy controls. Journal of the European Academy of Dermatology and Venereology, 36, pp. e580–e583.
10. Byrd, A.S., Carmona-Rivera, C., Kerns, M.L., et al. (2017). Role of fungi and extracellular matrix in hidradenitis suppurativa. Journal of Investigative Dermatology, 137, p. S112.
11. MacGibeny, M.A., Adjei, S., Pyle, H., et al. (2024). Alterations in the skin microbiome in dermatologic diseases and with external exposures: CME part 2. Journal of the American Academy of Dermatology.
12. Rosi, E., Guerra, P., Silvi, G., et al. (2023). Consistency of bacterial triggers in the pathogenesis of hidradenitis suppurativa. Vaccines, 11, p. 179.
13. Lindsø Andersen, P., Kjaersgaard Andersen, R., Jemec, G.B.E., et al. (2021). Superficial fungal infections and patients with hidradenitis suppurativa: a study under the Danish Blood Donor Study. Clinical and Experimental Dermatology, 46, pp. 571–573.
14. Yidana, D.B. (2021). Hidradenitis suppurativa: the role of interleukin-17, the aryl hydrocarbon receptor and the link to a possible fungal aetiology. Medical Hypotheses, 149, p. 110530.
15. Sabatino, J.J. Jr and Zamvil, S.S. (2017). Aryl hydrocarbon receptor activity may serve as a surrogate marker for MS disease activity. Neurology: Neuroimmunology & Neuroinflammation, 4, p. e366.
16. Li, X., Lau, S.K. and Woo, P.C. (2020). Fungal infection risks associated with the use of cytokine antagonists and immune checkpoint inhibitors. Experimental Biology and Medicine, 245, pp. 1104–1114.
17. Kurokawa, I., Nishijima, S., Suzuki, K., et al. (2002). Cytokeratin expression in pilonidal sinus. British Journal of Dermatology, 146, pp. 409–413.
18. Guet-Revillet, H., Delage, M., Riverain-Gillet, É., et al. (2020). The microbiology of suppurative pilonidal sinus disease: another link with hidradenitis suppurativa. Experimental Dermatology, 29, pp. 27–28.
19. Oh, H.B., Abdul Malik, M.H. and Keh, C.H. (2015). Pilonidal abscess associated with primary actinomycosis. Annals of Coloproctology, 31, pp. 243–245.
20. Soria Rivas, A., Bea-Ardebol, S., Vida Navas, E., et al. (2023). Treatment of metastatic squamous cell carcinoma arising in sacrococcygeal pilonidal sinus: a case report series. Frontiers in Medicine, 10, p. 1248894.
21. Kaba, M. (2024). New factors affecting wound healing and recurrence after pilonidal sinus surgery in adolescents: seborrheic dermatitis and psychiatric co-occurring conditions. International Wound Journal, 21, p. e14404.
22. Shastry, J., Ciliberto, H. and Davis, D.M. (2018). Tinea capitis mimicking dissecting cellulitis in three children. Pediatric Dermatology, 35, pp. e79–e83.
23. Stein, L.L., Adams, E.G. and Holcomb, K.Z. (2013). Inflammatory tinea capitis mimicking dissecting cellulitis in a postpubertal male: a case report and review of the literature. Mycoses, 56, pp. 596–600.
24. Twersky, J.M. and Sheth, A.P. (2005). Tinea capitis mimicking dissecting cellulitis: a distinct variant. International Journal of Dermatology, 44, pp. 412–414.
25. Martin-García, R.F. and Rullán, J.M. (2015). Refractory dissecting cellulitis of the scalp successfully controlled with adalimumab. Puerto Rico Health Sciences Journal, 34, pp. 102–104.
26. Federico, A., Rossi, A., Caro, G., et al. (2021). Are dissecting cellulitis and hidradenitis suppurativa different diseases? Clinics in Dermatology, 39, pp. 496–499.
27. Islam, Z., Toker, M., Gandhi, I.M., Sher, A. and Campton, K. (2024). Improvement of recalcitrant dissecting cellulitis of the scalp after a trial of upadacitinib. Cureus, 16, p. e52377.
28. Georgala, S., Korfitis, C., Ioannidou, D., et al. (2008). Dissecting cellulitis of the scalp treated with rifampicin and isotretinoin. Cutis, 82, pp. 195–198.
29. Xu, Z., Wang, Z., Yuan, C., et al. (2016). Dandruff is associated with the conjoined interactions between host and microorganisms. Scientific Reports, 6, p. 24877.
30. Jo, J.H., Deming, C., Kennedy, E.A., et al. (2016). Diverse human skin fungal communities in children converge in adulthood. Journal of Investigative Dermatology, 136, pp. 2356–2363.
31. Wang, L., Clavaud, C., Bar-Hen, A., et al. (2015). Characterization of the major bacterial–fungal populations colonizing dandruff scalps in Shanghai, China shows microbial disequilibrium. Experimental Dermatology, 24, pp. 398–400.
32. Park, H.K., Ha, M.H., Park, S.G., et al. (2012). Characterization of the fungal microbiota (mycobiome) in healthy and dandruff-afflicted human scalps. PLoS One, 7, p. e32847.
33. Polak-Witka, K., Rudnicka, L., Blume-Peytavi, U. and Vogt, A. (2020). The role of the microbiome in scalp hair follicle biology and disease. Experimental Dermatology, 29, pp. 286–294.
34. Miteva, M. (2021). Dissecting cellulitis of the scalp. In Miteva, M. (Ed.), Hair Pathology with Trichoscopic Correlations (pp. 135–141). Boca Raton: CRC Press.
35. Mishra, K., Bukavina, L. and Ghannoum, M. (2021). Symbiosis and dysbiosis of the human mycobiome. Frontiers in Microbiology, 12, p. 636131.
36. Meyer, L.E., Otberg, N., Tietz, H.J., Sterry, W. and Lademann, J. (2005). In vivo imaging of Malassezia yeasts on human skin using confocal laser scanning microscopy. Laser Physics Letters, 2, pp. 148–152.
37. Masson, R., Jeong, C.Y., Ma, E., et al. (2023). Treatments for dissecting cellulitis of the scalp: a

systematic review and treatment algorithm. Dermatology and Therapy, 13, pp. 2487–2526.

38. Dréno, B., Dagnelie, M.A., Khammari, A. and Corvec, S. (2020). The skin microbiome: a new actor in inflammatory acne. American Journal of Clinical Dermatology, 21, pp. 18–24.
39. Shirakawa, M., Uramoto, K. and Harada, F.A. (2006). Treatment of acne conglobata with infliximab. Journal of the American Academy of Dermatology, 55, pp. 344–346.
40. Canpolat, F., Kurmuş, G.I. and Gönül, M. (2017). Acne conglobata. Romanian Journal of Clinical and Experimental Dermatology, 2, pp. 68–73.
41. Engelhardt, A.W. (1971). Acne conglobata candidosa. Mykosen, 14, pp. 1–7.
42. Omran, A.N. and Mansori, A.G. (2018). Pathogenic yeasts recovered from acne vulgaris: molecular characterization and antifungal susceptibility pattern. Indian Journal of Dermatology, 63, pp. 386–390.
43. Kang, S.H. and Kim, H.U. (1999). The isolation of Malassezia yeasts in the comedones of acne vulgaris. Korean Journal of Medical Mycology, 4, pp. 33–39.
44. Malgotra, V. and Singh, H. (2021). Malassezia (Pityrosporum) folliculitis masquerading as recalcitrant acne. Cureus, 13, p. e13534.
45. Darwiche, S., Gacesa, R., Ferraro, R.B., et al. (2025). Prevalence of skin fungi markedly declines in the lesions of two patients with moderate hidradenitis suppurativa. Microbial Pathogenesis, 206, p. 107778.
46. Johnson, C.E. and Naik, H.B. (2025). Microbiome perturbations in hidradenitis suppurativa. Dermatologic Clinics, 43, pp. 193–202.

Author, year	No. of patients	Diagnosis	Article type	Gender, n (%)	Age (years)	Treatment	Fungal findings
Ring et al., 2022 [9]	30	Hidradenitis suppurativa	Original article	19 female (63%), 11 male (37%)	Mean 46.9 ± 14	None reported	*Malassezia restricta*, *Saccharomyces cerevisiae*, *Malassezia globosa*
Oh et al., 2015 [19]	1	Pilonidal disease	Case report	Female	25	Oral amoxicillin–clavulanate	Filamentous gram-positive organisms described as fungal-like in the original report
Rivas et al., 2023 [20]	1	Pilonidal sinus	Case report	Male	53	Radiotherapy and antifungals	Yeast fungemia
Omran et al., 2018 [42]	70	Acne*	Original article	49 female (70%), 21 male (30%)	10–30	Triazole class	*Candida parapsilosis*, *C. krusei*, *C. lusitaniae*, *C. kefyr*
Kang et al., 1999 [43]	32	Acne*	Original article	Not reported	Not reported	Not reported	*Malassezia* folliculitis, *M. restricta*, *M. globosa*, *M. furfur*
Engelhardt, 1971 [41]	1	Acne conglobata (candidosa)**	Case report	Male	17	Bis-phenyl-(2-chlorophenyl)-1-imidazolyl-methane	*Candida albicans*
Findley et al., 2013 [8]	10 (subset of larger cohort)	Pseudo–dissecting cellulitis of the scalp***	Case series	Not reported	18–40	Not reported	*Malassezia restricta*, *Malassezia globosa*
Darwiche et al., 2025 [45]	2	Hidradenitis suppurativa	Case series	Female (100%)	27, 30	Not reported	Markedly declined prevalence of skin fungi in lesions

Table 1. Summary of studies reporting fungal findings in follicular occlusion disorders

*Includes acne vulgaris with suspected fungal involvement.
**Term used by the original author to describe Candida-associated acne conglobata.
***Described as pseudo–dissecting cellulitis in the original study.