



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE

CURRENT BIOLOGIC THERAPIES FOR MODERATE-TO-SEVERE
HIDRADENITIS SUPPURATIVA: A NARRATIVE REVIEW OF
APPROVED TREATMENTS AND CLINICAL IMPLICATIONS

DOI

[https://doi.org/10.31435/ijitss.3\(51\).2026.6123](https://doi.org/10.31435/ijitss.3(51).2026.6123)

RECEIVED

12 July 2026

ACCEPTED

14 September 2026

PUBLISHED

16 September 2026

LICENSE



The article is licensed under a **Creative Commons Attribution 4.0 International License**.

© The author(s) 2026.

This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

CURRENT BIOLOGIC THERAPIES FOR MODERATE-TO-SEVERE HIDRADENITIS SUPPURATIVA: A NARRATIVE REVIEW OF APPROVED TREATMENTS AND CLINICAL IMPLICATIONS

Kamil A. Wiązania (Corresponding Author, Email: k.wiazania00@gmail.com)

University Clinical Hospital in Opole, 45-401 Opole, Poland

ORCID ID: 0009-0000-5916-0402

Szymon Wróbel

Murcki Hospital, ul. Alfreda Sokółowskiego 2, 40-749 Katowice, Poland

ORCID ID: 0009-0001-3598-9045

Rafał Wilczyński

The Lower Silesian Center for Oncology, Pulmonology and Hematology, pl. Hirszfelda 12, 53-413 Wrocław, Poland

ORCID ID: 0009-0006-8459-4740

Maja Porążko

University Clinical Hospital in Opole, 45-401 Opole, Poland

ORCID ID: 0009-0001-0248-4884

ABSTRACT

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease characterized by recurrent painful nodules, abscesses, and sinus tracts, resulting in substantial impairment of quality of life. Advances in the understanding of HS immunopathogenesis have led to the development of biologic therapies targeting key inflammatory pathways, particularly TNF- α and IL-17. To summarize current evidence regarding approved biologic therapies for moderate-to-severe HS, focusing on their mechanisms of action, efficacy, safety, and clinical applicability. A narrative literature review was conducted using the PubMed/MEDLINE database. Studies published between January 2016 and June 2026 were screened using the terms “hidradenitis suppurativa”, “adalimumab”, “secukinumab”, “bimekizumab”, and names of pivotal clinical trials. Randomized controlled trials, extension studies, real-world evidence reports, and high-quality reviews were included. Data were qualitatively synthesized. Adalimumab, the first approved biologic for HS, demonstrated significant efficacy in the phase III PIONEER I and II trials, with sustained long-term benefits. Secukinumab showed superior clinical response rates compared with placebo in the phase III SUNSHINE and SUNRISE studies. Bimekizumab demonstrated promising efficacy in both phase II and phase III BE HEARD trials, improving disease activity, pain, and quality of life. Overall, biologic therapies targeting TNF- α and IL-17 pathways consistently improved clinical outcomes in patients with moderate-to-severe HS. Adalimumab remains the established first-line biologic agent, while secukinumab and bimekizumab provide effective alternatives for patients with poor response to TNF- α inhibition. Despite encouraging efficacy and safety data, complete disease remission remains difficult to achieve, highlighting the need for further therapeutic advances.

KEYWORDS

Hidradenitis Suppurativa, Adalimumab, Secukinumab, Bimekizumab, Interleukin-17

CITATION

Wiązania, K. A., Wróbel, S., Wilczyński, R., & Porażko, M. (2026). Current biologic therapies for moderate-to-severe hidradenitis suppurativa: A narrative review of approved treatments and clinical implications. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.6123](https://doi.org/10.31435/ijitss.3(51).2026.6123)

COPYRIGHT

© The author(s) 2026. This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

1. Introduction

Hidradenitis suppurativa, also known as acne inversa is an inflammatory disease of the hair follicle unit that usually starts after puberty, characterized by repeated attacks of painful lesions like inflammatory nodules, abscesses, and draining sinuses [1,2]. The prevalence of the condition among the adult population remains unknown, although it is estimated in the general population at 0,4% [3]. There is a female predominance among the HS cases [4].

The pathogenesis of the disease is complicated; there are several contributing factors: follicular occlusion, immune system regulation disorders, chronic inflammation triggered by several cytokines, such as TNF- α and IL-17 [1,2]. There are genetic risk factors, overweight, tobacco use, and hormone changes that may trigger hidradenitis suppurativa [2]. Many patients have developed severe scarring and contractures due to the late diagnosis and insufficient treatment options, resulting in a severely impaired quality of life and even developing psychological problems such as depression and anxiety [5].

For decades, treatment of HS relied mainly on topical therapies, systemic antibiotics, and surgical interventions, frequently providing only temporary relief. The approval of the first biologic agent - adalimumab - marked a turning point in HS management [6,7]. In recent years, the therapeutic landscape has expanded rapidly with the introduction of IL-17 pathway inhibitors [8]. Currently, three biologics are approved for moderate-to-severe HS: adalimumab, secukinumab, and bimekizumab, although new therapies including JAK-inhibitors and IL-23 inhibitors are still in development [7,9].

The purpose of this review is to provide a comprehensive overview of the efficacy, safety, and application of currently approved biologic drugs in the treatment of HS. Specifically, the emphasis will be placed on the clinical practice aspect of using biologics. This review provides a summary of evidence from key clinical trials and current real-world data (2016-2026). Basic information on pathogenesis, epidemiology, and other relevant aspects was obtained from previously published literature.

2. Methodology

A narrative literature review was performed using the PubMed/MEDLINE database as the primary source of scientific publications. The main search focused on studies evaluating biologic therapies for hidradenitis suppurativa published between January 2016 and June 2026. Additionally, selected landmark studies and review articles published before 2020 were included to provide historical background and discuss the evolution of biologic treatment strategies.

Search terms included “hidradenitis suppurativa”, “adalimumab”, “secukinumab”, “bimekizumab”, and the names of pivotal clinical trials, including PIONEER, SUNSHINE, SUNRISE, and BE HEARD. Reference lists of relevant articles were also screened to identify additional publications of interest.

Eligible publications included randomized controlled trials, extension studies, open-label studies, real-world evidence reports, systematic reviews, and expert consensus documents. Studies were selected based on their relevance to the efficacy, safety, dosing regimens, mechanisms of action, and clinical use of approved biologic therapies in hidradenitis suppurativa.

Extracted information was qualitatively analyzed and synthesized to provide a comprehensive overview of currently available biologic treatment options. Due to the narrative nature of the review, no formal quality assessment, risk-of-bias evaluation, or quantitative meta-analysis was performed.

3. Results

3.1. Pathophysiology

The pathogenesis of HS is multifactorial and not fully understood yet. However, the process starts from follicular hyperkeratosis and occlusion of the hair follicle infundibulum. The consequences include follicular dilatation, follicular rupture, and the consequent leakage of keratin, bacteria from the follicle into the dermis, leading to intense immune response and formation of abscesses [1,2]. Dysbiosis is common in HS although it is not an infectious disease and it plays an important role in pathogenesis [10].

One of the main aspects of HS is immune dysfunction that affects both innate and adaptive parts of the system. Some of the important pro-inflammatory cytokines take part in this disorder. For instance, tumor necrosis factor- α (TNF- α) serves as an upstream factor and stimulates Th1 and Th17 differentiation [1]. TNF- α also amplifies inflammation and causes tissue damage. Elevated levels of TNF- α have been observed in the HS lesion and the surrounding skin, suggesting the effectiveness of TNF- α inhibitors [1].

The IL-23/Th17/IL-17 pathway is yet another significant factor that has been found to play an important role in the pathology of HS. IL-23 promotes the proliferation and maturation of Th17 lymphocytes that secrete IL-17A and IL-17F. These cytokines are powerful inducers of keratinocyte hyperproliferation and infiltration by neutrophils, alongside the secretion of more pro-inflammatory factors such as S100 proteins, chemokines like CXCL1, CXCL8, and CCL20 [11]. Increased levels of IL-17A and IL-17F have been detected in HS tissue samples [12].

Other important cytokines involved include IL-1 β (which further amplifies the inflammatory loop and neutrophil influx) and IFN- γ [1]. Additional contributing factors include genetic predisposition, dysbiosis of the skin microbiome with biofilm formation, and environmental triggers. Smoking and obesity are major modifiable risk factors; smoking promotes follicular occlusion and oxidative stress, while obesity contributes through mechanical friction, hormonal effects (adipokines), and systemic low-grade inflammation [13].

In summary, the complex interplay between follicular occlusion, microbiome dysbiosis, and exaggerated TNF- α - and IL-17-driven inflammatory responses results in the characteristic clinical picture of painful nodules, abscesses, and chronic suppurating tunnels [1,2]. A deeper understanding of these pathways, particularly the central roles of TNF- α and IL-17, made possible the successful development of targeted biologic therapies discussed in this review [6,7,14,15,16].

3.2. Risk factors

3.2.1. Obesity

Obesity is one of the strongest modifiable risk factors. Excess adipose tissue promotes mechanical friction in intertriginous areas, systemic low-grade inflammation through adipokine dysregulation (especially leptin) [13].

3.2.2 Female sex

The female to male ratio in HS is approximately 3:1 [4]. It is considered that hormonal fluctuations play a major role for female predominance. Disease typically surfaces after puberty and before menopause. Male predominance is more common in oriental countries [4,17]. Menstruation caused worsening of HS symptoms in 76.7%, no change in 22.2%, and improvement in 1.1% [18]. During pregnancy, the distribution between symptoms worsening (34.8%), having no change (28.7%), and improving (36.6%) was relatively even. After menopause, participants typically reported either worsening (39.5%) or no change (44.2%) in HS symptoms [18].

3.2.3. Smoking

Nicotinism is another major risk factor, present in up to 80–90% of patients. Tobacco smoke induces follicular occlusion, oxidative stress, and alters immune responses, significantly worsening disease course and reducing response to therapy [13,19].

3.2.4. Genetics factors

A family history of the condition increases the risk of inheriting the disease, which impacts the way the body's immune and follicular systems interact. The link between HS and familial history has been suspected since the middle of 1980s. The importance of genetic factors was highlighted by some older studies showing that 30%–40% of HS patients reported a family history of HS [20].

3.3. Clinical assessment of HS

The disease is traditionally classified using the Hurley staging system, which divides HS into three stages based on the presence of abscesses, sinus tracts, and scarring. While simple and widely used, Hurley staging is static and less suitable for monitoring treatment response in clinical trials or daily practice [21,22].

More dynamic and validated tools have been developed. The International Hidradenitis Suppurativa Severity Score System (IHS4) is currently considered one of the most reliable scoring systems [22]. It incorporates the number of nodules, abscesses, and draining tunnels, providing a continuous score that better reflects disease activity. Other important tools include the Hidradenitis Suppurativa Clinical Response (HiSCR), which has become the primary efficacy endpoint in biologic trials. HiSCR is defined as at least a 50% reduction in the number of inflammatory nodules and abscesses with no increase in the number of abscesses or draining fistulas [23]. As newer, more effective biologic treatments have emerged, researchers started using higher benchmarks to measure deeper levels of skin clearance [15,24]. They follow the exact same rules regarding no increase in abscesses or draining fistulas, but require a much higher drop in the abscess and nodules (AN) count. HiSCR75 represents a high level of improvement, and major symptom relief ($\geq 75\%$ reduction). HiSCR90 stands for near-complete clearance of active inflammatory lesions, representing optimal disease control ($\geq 90\%$ reduction of symptoms) [15, 24].

3.4. Conventional therapies in management of HS - antibiotics and surgery

Although biologic therapies represent a major advance in the treatment of moderate-to-severe hidradenitis suppurativa (HS), antibiotics and surgical interventions remain essential components of a multimodal treatment approach. These conventional modalities are particularly valuable for acute flares, mild disease, localized lesions, and as adjunctive therapy in combination with biologics [1].

3.4.1. Antibiotics

HS exerts both antimicrobial and anti-inflammatory effects. Topical clindamycin 1% is recommended as first-line therapy for mild (Hurley stage I) disease [25,26]. For moderate-to-severe cases, systemic antibiotics are commonly used. The most established regimen is the combination of clindamycin (300 mg b.i.d.) plus rifampicin (600 mg daily) for 10 weeks. This combination has proved beneficial, but with variable results [27,28,29]. Tetracyclines have shown similar clinical outcomes as topical clindamycin in Hurley I stage [26]. Long-term or repeated antibiotic courses should be avoided due to the risk of antimicrobial resistance and limited disease-modifying effect.

3.4.2. Surgery

Surgical interventions have a crucial role, particularly in Hurley stage II and III disease with established sinus tracts and scarring. Procedures range from minimally invasive techniques such as incision and drainage (strictly reserved for acute, painful abscess relief due to high recurrence rates), deroofing (removal of the roof of sinus tracts), to more radical options including wide local excision of all affected tissue [30,31]. Surgery is most effective when performed after optimization of medical therapy, including biologics, which can reduce inflammation preoperatively and improve postoperative healing. Combinations of biologic drugs and surgery were associated with better outcomes in the severity indicators of HS, although the risk of delayed wound healing cannot be avoided [32,33,34].

3.5. Biological treatment of hidradenitis suppurativa

3.5.1. Adalimumab

Adalimumab is a fully human recombinant monoclonal immunoglobulin G1 (IgG1) antibody directed against tumor necrosis factor- α (TNF- α), a key proinflammatory cytokine involved in the pathogenesis of numerous chronic inflammatory disorders. By binding both soluble and membrane-bound TNF- α , adalimumab inhibits downstream inflammatory signaling pathways, thereby reducing leukocyte recruitment, cytokine production, and tissue inflammation [1,2]. The growing understanding of the immunopathogenesis of hidradenitis suppurativa (HS) has highlighted the central role of TNF- α in disease development and progression, providing a strong rationale for TNF- α -targeted therapy. Elevated concentrations of TNF- α have been detected in HS lesions and perilesional skin, where they contribute to chronic inflammation, abscess

formation, tissue destruction, and sinus tract development [1,2,35]. Consequently, adalimumab became the first biologic agent approved for the treatment of moderate-to-severe HS and remains the benchmark against which newer biologic therapies are compared [7].

The clinical efficacy of adalimumab in HS was established through the pivotal phase III PIONEER I and PIONEER II trials, which represented the first large-scale randomized controlled studies evaluating biologic therapy in this disease [6]. These multicenter, double-blind, placebo-controlled trials enrolled more than 600 patients with moderate-to-severe HS who had an inadequate response to conventional systemic therapies. Participants received adalimumab according to an induction regimen followed by maintenance treatment of 40 mg weekly. The primary endpoint was achievement of Hidradenitis Suppurativa Clinical Response (HiSCR) at week 12, defined as at least a 50% reduction in the total abscess and inflammatory nodule count without an increase in abscesses or draining fistulas. In both studies, adalimumab demonstrated significantly higher response rates compared with placebo. In PIONEER I, HiSCR was achieved by 41.8% of patients receiving adalimumab compared with 26.0% of those receiving placebo [6]. In PIONEER II, response rates were even higher, reaching 58.9% and 27.6%, respectively [6]. These findings established adalimumab as the first evidence-based biologic treatment capable of significantly improving disease activity in patients with moderate-to-severe HS.

Beyond reducing inflammatory lesion counts, adalimumab has demonstrated clinically meaningful benefits in several patient-centered outcomes. Patients receiving adalimumab reported greater improvements in pain scores, physical functioning, and overall health-related quality of life. Improvements were also observed in Dermatology Life Quality Index (DLQI) scores and measures of work productivity, suggesting that effective suppression of inflammation translates into substantial psychosocial and functional benefits [36]. These findings are particularly important because HS is associated with high rates of depression, anxiety, social isolation, and occupational impairment [1].

Long-term disease control in HS is challenging due to its chronic, relapsing nature. Integrated analyses of the PIONEER trials and their extension phases demonstrated that continuous weekly adalimumab administration provides sustained HiSCR responses and prolonged disease control, especially in patients achieving a partial response within the first 12 weeks. Conversely, dose reduction or treatment interruption frequently led to a loss of efficacy, while long-term maintenance therapy showed sustained clinical benefits with no new safety signals [37].

The safety profile of adalimumab in HS is generally consistent with that observed in other approved indications such as psoriasis, rheumatoid arthritis, inflammatory bowel disease, and ankylosing spondylitis [14]. The most frequent adverse events include upper respiratory tract infections, headaches, and injection-site reactions. Because TNF-alpha inhibitors increase the risk of serious opportunistic infections and latent tuberculosis reactivation, mandatory pre-treatment screening for TB and viral hepatitis is necessary [14].

Despite being the reference standard biologic, adalimumab has limitations: primary non-response, secondary loss of efficacy over time, and rare complete remission, leaving residual pain and scarring [37, 38]. This has driven the evolution toward mechanism-based strategies targeting IL-17 and IL-23 pathways [7,9].

In summary, adalimumab remains a milestone and cornerstone in moderate-to-severe HS management, proven to reduce lesion burden and improve quality of life and have had major impact on treatment HS and improvement quality of life for patients [6,14,36].

3.5.2 Bimekizumab

Bimekizumab is a humanized IgG1 monoclonal antibody that selectively neutralizes both interleukin-17A (IL-17A) and interleukin-17F (IL-17F), two key cytokines involved in the inflammatory cascade characteristic for HS and other chronic inflammatory diseases [1,2,11,15]. Unlike other biologic agents targeting only IL-17A, bimekizumab provides dual cytokine inhibition, which may result in more comprehensive suppression of inflammatory pathways [15]. Increasing evidence suggests that the IL-17 pathway plays a central role in HS pathogenesis, with elevated levels of IL-17-producing T-helper (Th17) cells and increased expression of IL-17-related cytokines identified in lesional skin [1,2,11,15]. These findings provided the rationale for investigating IL-17-targeted therapies, including bimekizumab, as potential treatment options for moderate-to-severe disease [15].

The first major evidence supporting the use of bimekizumab in HS came from a multicenter phase II randomized, double-blind, placebo-controlled clinical trial published in JAMA Dermatology in 2021 [39]. The study enrolled 90 patients with moderate-to-severe HS who were randomized to receive bimekizumab (46 patients), placebo (22 patients), or adalimumab (22 patients) as an active reference comparator [39]. Participants treated with bimekizumab received a loading dose of 640 mg followed by 320 mg every two weeks [39]. After 12 weeks of treatment, the primary endpoint, Hidradenitis Suppurativa Clinical Response (HiSCR), was achieved by 57.3% of patients receiving bimekizumab compared with 26.1% in the placebo group [39]. Importantly, more stringent efficacy endpoints also favored bimekizumab. Approximately 46% of treated

patients achieved HiSCR75 and 32% achieved HiSCR90, indicating substantial reductions in inflammatory lesion counts [39]. These response rates were numerically higher than those observed in the adalimumab reference arm [39]. Improvements were also noted in disease severity scores, pain assessments, and dermatology-specific quality-of-life measures [39]. The safety profile was generally favorable, with serious adverse events occurring in only 4% of patients treated with bimekizumab [39]. These findings provided proof-of-concept that simultaneous inhibition of IL-17A and IL-17F may represent an effective therapeutic strategy in HS [39].

The encouraging results of the phase II trial led to the development of the large phase III BE HEARD I and BE HEARD II studies [24]. These identically designed international, randomized, double-blind, placebo-controlled trials enrolled patients with moderate-to-severe HS and evaluated the efficacy and safety of bimekizumab over a 48-week treatment period [24]. Participants received bimekizumab 320 mg every two weeks or every four weeks according to predefined treatment regimens [24]. The primary objective was to assess the proportion of patients achieving HiSCR50 at week 16, with long-term efficacy evaluated through week 48 [24]. The phase III trials confirmed the efficacy observed in earlier studies and demonstrated clinically meaningful improvements across multiple disease domains, including inflammatory lesion burden, pain, and health-related quality of life [24]. The consistency of results across two independent studies substantially strengthened the evidence supporting bimekizumab as a therapeutic option for HS [24].

In summary, bimekizumab represents one of the most promising recent advances in the treatment of moderate-to-severe hidradenitis suppurativa [8,15]. Clinical trials have demonstrated significant improvements in disease activity, pain, and quality of life, while maintaining an acceptable safety profile [15,24,39]. The dual inhibition of IL-17A and IL-17F distinguishes bimekizumab from earlier biologic therapies and may explain its high clinical efficacy [15,24]. As additional long-term and real-world data become available, bimekizumab is likely to play an increasingly important role in the therapeutic management of HS. Longer studies are needed to understand response durability due to limitations of the sample, although current data are encouraging [8,15].

3.5.3. Secukinumab

Secukinumab is a fully human monoclonal antibody that selectively binds to and neutralizes interleukin-17A (IL-17A) [8,16]. By inhibiting the IL-23/Th17/IL-17 axis, secukinumab reduces keratinocyte hyperproliferation, neutrophil recruitment, and chronic inflammation that drive abscess and sinus tract formation in hidradenitis suppurativa (HS) [8].

The pivotal evidence for secukinumab comes from two identical Phase III, multicentre, randomized, double-blind, placebo-controlled trials: SUNSHINE and SUNRISE [16]. These studies enrolled adults with moderate-to-severe HS (Hurley stage II or III) who had an inadequate response to conventional systemic therapy [16]. Patients received subcutaneous secukinumab 300 mg at baseline and weeks 1–4 (loading dose), followed by either every 2 weeks or every 4 weeks maintenance dosing, or placebo [16].

The primary endpoint was the proportion of patients achieving Hidradenitis Suppurativa Clinical Response (HiSCR50) at Week 16 [16]. In the SUNSHINE trial, HiSCR50 was achieved by 45% of patients receiving secukinumab every 2 weeks compared with 34% receiving placebo [16]. In the SUNRISE trial, response rates were 42% for secukinumab every 2 weeks and 46% for secukinumab every 4 weeks versus 31% for placebo [16].

Responses were sustained or continued to improve through Week 52, with more than half of treated patients maintaining HiSCR responses during long-term follow-up [16,40]. Significant improvements were also observed in secondary endpoints, including reduction in abscess and inflammatory nodule (AN) count, pain reduction measured by Numerical Rating Scale (NRS30), draining tunnels, quality of life assessed by the Dermatology Life Quality Index (DLQI), and disease severity measured using the International Hidradenitis Suppurativa Severity Score System (IHS4) [16,40]. Efficacy was generally consistent regardless of prior biologic exposure or concomitant antibiotic therapy [16].

The safety profile of secukinumab in HS was consistent with its established safety profile across other approved indications [8,16,40]. The most commonly reported adverse events up to Week 16 included headache, nasopharyngitis, and mild-to-moderate infections [16]. No new safety signals were identified, and rates of serious adverse events, including serious infections, were low and generally comparable to placebo [16]. Long-term extension analyses confirmed sustained tolerability through at least Week 52 [16,40].

In summary, secukinumab represents an important addition to therapeutic options, offering targeted IL-17A inhibition with sustained efficacy and a favourable safety profile [16,40]. It may be particularly valuable for patients with inadequate response to TNF- α inhibitors or those requiring an alternative mechanism of action [8,40].

4. Discussion

The introduction of biologic therapies have had a major impact on the management of moderate-to-severe hidradenitis suppurativa (HS), moving beyond the limitations of traditional approaches such as prolonged antibiotic courses and repeated surgical interventions. While antibiotics and surgery remain important components of multimodal therapy - particularly for acute flares and established scarring - they often provide only temporary relief and do not address the underlying chronic immune dysregulation. Biologic agents targeting TNF- α (adalimumab) and the IL-17 pathway (secukinumab and bimekizumab) offer a more targeted and sustained control of inflammation.

Adalimumab, the first approved biologic for HS, continues to demonstrate robust efficacy in real-world settings, with network meta-analyses frequently showing the highest or comparable HiSCR responses among approved agents, alongside a well-characterized long-term safety profile. Secukinumab and bimekizumab have expanded therapeutic options by addressing the IL-23/Th17/IL-17 axis, which plays a central role in HS pathogenesis. Both agents achieved statistically significant HiSCR50 rates in phase 3 trials (SUNSHINE/SUNRISE for secukinumab; BE HEARD I/II for bimekizumab) and showed meaningful improvements in pain, quality of life (DLQI), and disease severity (IHS4). Bimekizumab, with its dual IL-17A/F inhibition, may offer numerical advantages in deeper responses (HiSCR75/90) in some analyses, although head-to-head trials are lacking.

Despite these advances, several challenges persist. Response rates rarely exceed 50–60% for HiSCR50, and complete remission remains uncommon. Many patients experience partial responses or require concomitant therapies (antibiotics, surgery, or lifestyle interventions). Factors such as obesity, ongoing smoking, and disease duration negatively influence outcomes, underscoring the importance of early biologic intervention within the “window of opportunity” before irreversible scarring develops. Safety profiles are generally favourable and consistent with known class effects, with infections being the most common adverse events; however, patient selection remains critical.

Real-world evidence is emerging and generally supports trial data, although effectiveness can be lower in heavily pre-treated, more severe populations. The lack of reliable biomarkers for predicting response to specific biologics and optimal treatment sequencing represents a key unmet need. Future directions include combination strategies (biologic + surgery), novel agents (IL-23 inhibitors, JAK inhibitors, small molecules), and personalized approaches based on disease phenotype and comorbidities.

5. Conclusions

Biologic therapies have become the cornerstone of treatment for moderate-to-severe hidradenitis suppurativa. Adalimumab, secukinumab, and bimekizumab provide effective, targeted options that significantly improve clinical outcomes, reduce pain, and enhance quality of life compared with conventional therapies alone. Early initiation of biologics in eligible patients, combined with addressing modifiable risk factors (especially smoking cessation and weight reduction) and appropriate use of surgery, offers the best chance of long-term disease control and prevention of irreversible damage. While important progress has been made, complete remission remains difficult to achieve for many patients, highlighting the need for continued research into novel therapies and personalised treatment algorithms. In daily clinical practice, the choice of biologic should be individualised based on disease severity, comorbidities, prior treatment responses, and patient preferences. Multidisciplinary care involving dermatologists, surgeons, and other specialists is essential for optimal outcomes in this complex disease.

Author Contributions:

Conceptualization: Szymon Wróbel and Kamil Wiązania.; methodology: Rafał Wilczyński and Maja Porażko; software: Szymon Wróbel; validation: Kamil Wiązania and Rafał Wilczyński; formal analysis: Maja Porażko; investigation: Kamil Wiązania and Maja Porażko; resources: Kamil Wiązania and Rafał Wilczyński; data curation: Kamil Wiązania and Maja Porażko; writing—original draft preparatio: Kamil Wiązania and Szymon Wróbel.; writing—review and editin: Rafał Wilczyński and Maja Porażko.; visualization: Kamil Wiązania.; supervision: Szymon Wróbel and Rafał Wilczyński; project administration: Maja Porażko; funding acquisition: Kamil Wiązania. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Conflicts of Interest: No conflicts of interest to declare.

Declaration of the Use of Generative AI and AI-Assisted Technologies in the Writing Process:

During the preparation of this work, the authors used AI tools for the purpose of language editing and grammar correction of the English text to improve readability and linguistic accuracy. After using this tool, the authors reviewed and edited the content as needed and took full responsibility for the substantive content of the publication.

REFERENCES

1. van Straalen, K. R., Prens, E. P., & Gudjonsson, J. E. (2022). Insights into hidradenitis suppurativa. *The Journal of allergy and clinical immunology*, 149(4), 1150–1161. <https://doi.org/10.1016/j.jaci.2022.02.003>
2. Napolitano, M., Megna, M., Timoshchuk, E. A., Patruno, C., Balato, N., Fabbrocini, G., & Monfrecola, G. (2017). Hidradenitis suppurativa: From pathogenesis to diagnosis and treatment. *Clinical, cosmetic and investigational dermatology*, 10, 105–115. <https://doi.org/10.2147/CCID.S111019>
3. Jfri, A., Nassim, D., O'Brien, E., Gulliver, W., Nikolakis, G., & Zouboulis, C. C. (2021). Prevalence of hidradenitis suppurativa: A systematic review and meta-regression analysis. *JAMA dermatology*, 157(8), 924–931. <https://doi.org/10.1001/jamadermatol.2021.1677>
4. Medianfar, C. E., Fernandez, L. I., Bouazzi, D., & Jemec, G. B. E. (2025). Global hidradenitis suppurativa epidemiology: Exploring themes and gaps beyond europe, australia, and the usa. *Dermatology (Basel, Switzerland)*, 241(Suppl. 1), 159–170. <https://doi.org/10.1159/000542389>
5. Montero-Vilchez, T., Diaz-Calvillo, P., Rodriguez-Pozo, J. A., Cuenca-Barrales, C., Martinez-Lopez, A., Arias-Santiago, S., & Molina-Leyva, A. (2021). The burden of hidradenitis suppurativa signs and symptoms in quality of life: Systematic review and Meta-Analysis. *International journal of environmental research and public health*, 18(13), 6709. <https://doi.org/10.3390/ijerph18136709>
6. Kimball, A. B., Okun, M. M., Williams, D. A., Gottlieb, A. B., Papp, K. A., Zouboulis, C. C., Armstrong, A. W., Kerdel, F., Gold, M. H., Forman, S. B., Korman, N. J., Giamarellos-Bourboulis, E. J., Crowley, J. J., Lynde, C., Regui, Z., Prens, E. P., Alwawi, E., Mostafa, N. M., Pinsky, B., Sundaram, M., ... Jemec, G. B. (2016). Two phase 3 trials of adalimumab for hidradenitis suppurativa. *The New England journal of medicine*, 375(5), 422–434. <https://doi.org/10.1056/NEJMoa1504370>
7. Calabrese, L., Cartocci, A., Rubegni, P., French, L. E., & Kendziora, B. (2026). Efficacy and safety of biologics for hidradenitis suppurativa: A network meta-analysis of phase iii trials. *Journal of the European Academy of Dermatology and Venereology : JEADV*, 40(4), 637–645. <https://doi.org/10.1111/jdv.20617>
8. Pinto Salgueiro, G., Yilmaz, O., Nogueira, M., & Torres, T. (2025). Interleukin-17 inhibitors in the treatment of hidradenitis suppurativa. *BioDrugs : clinical immunotherapeutics, biopharmaceuticals and gene therapy*, 39(1), 53–74. <https://doi.org/10.1007/s40259-024-00687-w>
9. Martora, F., Scalvenzi, M., Ruggiero, A., Potestio, L., Battista, T., & Megna, M. (2023). Hidradenitis suppurativa and jak inhibitors: A review of the published literature. *Medicina (Kaunas, Lithuania)*, 59(4), 801. <https://doi.org/10.3390/medicina59040801>
10. Chopra, D., Arens, R. A., Amornpairoj, W., Lowes, M. A., Tomic-Canic, M., Strbo, N., Lev-Tov, H., & Pastar, I. (2022). Innate immunity and microbial dysbiosis in hidradenitis suppurativa - vicious cycle of chronic inflammation. *Frontiers in immunology*, 13, 960488. <https://doi.org/10.3389/fimmu.2022.960488>
11. Del Duca, E., Morelli, P., Bennardo, L., Di Raimondo, C., & Nisticò, S. P. (2020). Cytokine pathways and investigational target therapies in hidradenitis suppurativa. *International journal of molecular sciences*, 21(22), 8436. <https://doi.org/10.3390/ijms21228436>

12. Yao, Y., & Thomsen, S. F. (2017). The role of interleukin-17 in the pathogenesis of hidradenitis suppurativa. *Dermatology online journal*, 23(7), 13030/qt8rw2j9zv. <https://doi.org/10.5070/D3237035729>
13. Elzawawi, K. E., Elmakaty, I., Habibullah, M., Ahmed, M. B., Al Lahham, S., Al Harami, S., Albasti, H., & Alsherawi, A. (2024). Hidradenitis suppurativa and its association with obesity, smoking, and diabetes mellitus: A systematic review and meta-analysis. *International wound journal*, 21(9), e70035. <https://doi.org/10.1111/iwj.70035>
14. Kim, E. S., Garnock-Jones, K. P., & Keam, S. J. (2016). Adalimumab: A review in hidradenitis suppurativa. *American journal of clinical dermatology*, 17(5), 545–552. <https://doi.org/10.1007/s40257-016-0220-6>
15. Martora, F., & Megna, M. (2025). Bimekizumab for hidradenitis suppurativa from trials to real life: A review of the published literature. *Clinical, cosmetic and investigational dermatology*, 18, 3511–3519. <https://doi.org/10.2147/CCID.S565686>
16. Kimball, A. B., Jemec, G. B. E., Alavi, A., Reguiat, Z., Gottlieb, A. B., Bechara, F. G., Paul, C., Giamarellos-Bourboulis, E. J., Villani, A. P., Schwinn, A., Ruëff, F., Pillay Ramaya, L., Reich, A., Lobo, I., Sinclair, R., Passeron, T., Martorell, A., Mendes-Bastos, P., Kokolakis, G., Becherel, P. A., ... Muscianisi, E. (2023). Secukinumab in moderate-to-severe hidradenitis suppurativa (sunshine and sunrise): Week 16 and week 52 results of two identical, multicentre, randomised, placebo-controlled, double-blind phase 3 trials. *Lancet (London, England)*, 401(10378), 747–761. [https://doi.org/10.1016/S0140-6736\(23\)00022-3](https://doi.org/10.1016/S0140-6736(23)00022-3)
17. Chandran, N. S., Lee, J. H., & Kurokawa, I. (2021). Hidradenitis suppurativa in South-East asia and east asia. *Experimental dermatology*, 30 Suppl 1, 23–26. <https://doi.org/10.1111/exd.14340>
18. Fernandez, J. M., Hendricks, A. J., Thompson, A. M., Mata, E. M., Collier, E. K., Grogan, T. R., Shi, V. Y., & Hsiao, J. L. (2020). Menses, pregnancy, delivery, and menopause in hidradenitis suppurativa: A patient survey. *International journal of women's dermatology*, 6(5), 368–371. <https://doi.org/10.1016/j.ijwd.2020.07.002>
19. Poulsen, A. H., Zhang, D. G., Nielsen, V. W., Maul, J. T., Ring, H. C., Thyssen, J. P., Egeberg, A., Holgersen, N., & Thomsen, S. F. (2026). Impact of smoking on disease severity and treatment outcomes in hidradenitis suppurativa. *Journal of the European Academy of Dermatology and Venereology : JEADV*, 10.1111/jdv.70344. Advance online publication. <https://doi.org/10.1111/jdv.70344>
20. Fitzsimmons, J. S., & Guilbert, P. R. (1985). A family study of hidradenitis suppurativa. *Journal of medical genetics*, 22(5), 367–373. <https://doi.org/10.1136/jmg.22.5.367>
21. Zouboulis, C. C., Del Marmol, V., Mrowietz, U., Prens, E. P., Tzellos, T., & Jemec, G. B. (2015). Hidradenitis Suppurativa/Acne inversa: Criteria for diagnosis, severity assessment, classification and disease evaluation. *Dermatology (Basel, Switzerland)*, 231(2), 184–190. <https://doi.org/10.1159/000431175>
22. Zouboulis, C. C., Tzellos, T., Kyrgidis, A., Jemec, G. B. E., Bechara, F. G., Giamarellos-Bourboulis, E. J., Ingram, J. R., Kanni, T., Karagiannidis, I., Martorell, A., Matusiak, Ł., Pinter, A., Prens, E. P., Presser, D., Schneider-Burrus, S., von Stebut, E., Szepietowski, J. C., van der Zee, H. H., Wilden, S. M., Sabat, R., ... European Hidradenitis Suppurativa Foundation Investigator Group (2017). Development and validation of the international hidradenitis suppurativa severity score system (IHS4), a novel dynamic scoring system to assess its severity. *The British journal of dermatology*, 177(5), 1401–1409. <https://doi.org/10.1111/bjd.15748>
23. Kimball, A. B., Jemec, G. B., Yang, M., Kageleiry, A., Signorovitch, J. E., Okun, M. M., Gu, Y., Wang, K., Mulani, P., & Sundaram, M. (2014). Assessing the validity, responsiveness and meaningfulness of the hidradenitis suppurativa clinical response (HiSCR) as the clinical endpoint for hidradenitis suppurativa treatment. *The British journal of dermatology*, 171(6), 1434–1442. <https://doi.org/10.1111/bjd.13270>
24. Kimball, A. B., Jemec, G. B. E., Sayed, C. J., Kirby, J. S., Prens, E., Ingram, J. R., Garg, A., Gottlieb, A. B., Szepietowski, J. C., Bechara, F. G., Giamarellos-Bourboulis, E. J., Fujita, H., Roller, R., Joshi, P., Dokhe, P., Muller, E., Peterson, L., Madden, C., Bari, M., & Zouboulis, C. C. (2024). Efficacy and safety of bimekizumab in patients with moderate-to-severe hidradenitis suppurativa (be heard i and be heard ii): Two 48-week, randomised, double-blind, placebo-controlled, multicentre phase 3 trials. *Lancet (London, England)*, 403(10443), 2504–2519. [https://doi.org/10.1016/S0140-6736\(24\)00101-6](https://doi.org/10.1016/S0140-6736(24)00101-6)
25. Clemmensen O. J. (1983). Topical treatment of hidradenitis suppurativa with clindamycin. *International journal of dermatology*, 22(5), 325–328. <https://doi.org/10.1111/j.1365-4362.1983.tb02150.x>
26. Jemec, G. B., & Wendelboe, P. (1998). Topical clindamycin versus systemic tetracycline in the treatment of hidradenitis suppurativa. *Journal of the American Academy of Dermatology*, 39(6), 971–974. [https://doi.org/10.1016/s0190-9622\(98\)70272-5](https://doi.org/10.1016/s0190-9622(98)70272-5)
27. Gener, G., Canoui-Poitrine, F., Revuz, J. E., Faye, O., Poli, F., Gabison, G., Pouget, F., Viallette, C., Wolkenstein, P., & Bastuji-Garin, S. (2009). Combination therapy with clindamycin and rifampicin for hidradenitis suppurativa: A series of 116 consecutive patients. *Dermatology (Basel, Switzerland)*, 219(2), 148–154. <https://doi.org/10.1159/000228334>

28. Mendonça, C. O., & Griffiths, C. E. (2006). Clindamycin and rifampicin combination therapy for hidradenitis suppurativa. *The British journal of dermatology*, 154(5), 977–978. <https://doi.org/10.1111/j.1365-2133.2006.07155.x>
29. van der Zee, H. H., Boer, J., Prens, E. P., & Jemec, G. B. (2009). The effect of combined treatment with oral clindamycin and oral rifampicin in patients with hidradenitis suppurativa. *Dermatology (Basel, Switzerland)*, 219(2), 143–147. <https://doi.org/10.1159/000228337>
30. van der Zee, H. H., Prens, E. P., & Boer, J. (2010). Deroofing: A tissue-saving surgical technique for the treatment of mild to moderate hidradenitis suppurativa lesions. *Journal of the American Academy of Dermatology*, 63(3), 475–480. <https://doi.org/10.1016/j.jaad.2009.12.018>
31. Ovadjia, Z. N., Zugaj, M., Jacobs, W., van der Horst, C. M. A. M., & Lapid, O. (2021). Recurrence rates following reconstruction strategies after wide excision of hidradenitis suppurativa: A systematic review and meta-analysis. *Dermatologic surgery : official publication for American Society for Dermatologic Surgery [et al.]*, 47(4), e106–e110. <https://doi.org/10.1097/DSS.0000000000002815>
32. Prens, L. M., Huizinga, J., Janse, I. C., & Horváth, B. (2019). Surgical outcomes and the impact of major surgery on quality of life, activity impairment and sexual health in hidradenitis suppurativa patients: A prospective single centre study. *Journal of the European Academy of Dermatology and Venereology : JEADV*, 33(10), 1941–1946. <https://doi.org/10.1111/jdv.15706>
33. DeFazio, M. V., Economides, J. M., King, K. S., Han, K. D., Shanmugam, V. K., Attinger, C. E., & Evans, K. K. (2016). Outcomes after combined radical resection and targeted biologic therapy for the management of recalcitrant hidradenitis suppurativa. *Annals of plastic surgery*, 77(2), 217–222. <https://doi.org/10.1097/SAP.0000000000000584>
34. Aarts, P., van Huijstee, J. C., van der Zee, H. H., van Doorn, M. B. A., van Straalen, K. R., & Prens, E. P. (2023). Adalimumab in conjunction with surgery compared with adalimumab monotherapy for hidradenitis suppurativa: A randomized controlled trial in a real-world setting. *Journal of the American Academy of Dermatology*, 89(4), 677–684. <https://doi.org/10.1016/j.jaad.2023.04.034>
35. van der Zee, H. H., de Ruiter, L., van den Broecke, D. G., Dik, W. A., Laman, J. D., & Prens, E. P. (2011). Elevated levels of tumour necrosis factor (TNF)- α , interleukin (IL)-1 β and IL-10 in hidradenitis suppurativa skin: A rationale for targeting TNF- α and IL-1 β . *The British journal of dermatology*, 164(6), 1292–1298. <https://doi.org/10.1111/j.1365-2133.2011.10254.x>
36. Kimball, A. B., Sundaram, M., Shields, A. L., Hudgens, S., Okun, M., Foley, C., & Ganguli, A. (2018). Adalimumab alleviates skin pain in patients with moderate-to-severe hidradenitis suppurativa: Secondary efficacy results from the pioneer i and pioneer ii randomized controlled trials. *Journal of the American Academy of Dermatology*, 79(6), 1141–1143. <https://doi.org/10.1016/j.jaad.2018.05.015>
37. Zouboulis, C. C., Okun, M. M., Prens, E. P., Gniadecki, R., Foley, P. A., Lynde, C., Weisman, J., Gu, Y., Williams, D. A., & Jemec, G. B. E. (2019). Long-term adalimumab efficacy in patients with moderate-to-severe hidradenitis suppurativa/acne inversa: 3-year results of a phase 3 open-label extension study. *Journal of the American Academy of Dermatology*, 80(1), 60–69.e2. <https://doi.org/10.1016/j.jaad.2018.05.040>
38. Gulliver, W., Alavi, A., Wiseman, M. C., Gooderham, M. J., Rao, J., Alam, M. S., Papp, K. A., Desjardins, O., & Jean, C. (2021). Real-world effectiveness of adalimumab in patients with moderate-to-severe hidradenitis suppurativa: The 1-year solace study. *Journal of the European Academy of Dermatology and Venereology : JEADV*, 35(12), 2431–2439. <https://doi.org/10.1111/jdv.17598>
39. Glatt, S., Jemec, G. B. E., Forman, S., Sayed, C., Schmieder, G., Weisman, J., Roller, R., Seegobin, S., Baeten, D., Ionescu, L., Zouboulis, C. C., & Shaw, S. (2021). Efficacy and safety of bimekizumab in moderate to severe hidradenitis suppurativa: A phase 2, double-blind, Placebo-Controlled randomized clinical trial. *JAMA dermatology*, 157(11), 1279–1288. <https://doi.org/10.1001/jamadermatol.2021.2905>
40. Kimball, A. B., Bechara, F. G., Badat, A., Giamarellos-Bourboulis, E. J., Gottlieb, A. B., Jemec, G. B. E., Reguiai, Z., Villani, A. P., Alarcon, I., Bansal, A., Gasperoni, F., Martin, R., Paguet, B., Uhlmann, L., Zouater, H., Ravichandran, S., & Alavi, A. (2025). Long-term efficacy and safety of secukinumab in patients with moderate-to-severe hidradenitis suppurativa: Week 104 results from the sunshine and sunrise extension trial. *The British journal of dermatology*, 192(4), 629–640. <https://doi.org/10.1093/bjd/ljae469>