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Clinical and Real-World Implications of IL-17 Receptor Blockade in Plaque Psoriasis and Psoriatic Arthritis With Brodalumab

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ABSTRACT

Brodalumab is a fully human monoclonal antibody targeting interleukin-17 receptor A (IL-17RA), providing a mechanistically distinct approach to inhibiting IL-17–mediated inflammation in plaque psoriasis and psoriatic arthritis (approved in Japan). Across pivotal randomized trials and extensive real-world studies, brodalumab demonstrates a rapid onset of action, high rates of complete skin clearance, durable efficacy, and consistent effectiveness across diverse patient subgroups, while randomized and real-world data also support meaningful improvements in joint outcomes among patients with concomitant psoriatic arthritis. This review synthesizes clinical trial and real-world evidence to contextualize the efficacy, safety, and clinical positioning of IL-17RA blockade with brodalumab across the spectrum of psoriatic disease.

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INTRODUCTION

Psoriasis is a chronic immune-mediated inflammatory disease driven by dysregulated interleukin-17 (IL-17) signaling across skin and musculoskeletal tissues. While upstream IL-23 inhibition and selective IL-17 cytokine blockade have improved outcomes, IL-17 cytokines can arise from IL-23-independent pathways and nonclassical cellular sources, sustaining inflammation despite ligand-specific inhibition and highlighting IL-17 receptor A (IL-17RA) as a central therapeutic target. This review examines clinical trial and real-world evidence for brodalumab, an IL-17RA antagonist, with a focus on efficacy, durability, safety, and its clinical positioning across the spectrum of psoriatic disease.

Role of IL-17 in Plaque Psoriasis and Psoriatic Arthritis: Focus on IL-17 Receptor

The IL-17 cytokine family comprises six structurally related isoforms—IL-17A, IL-17B, IL-17C, IL-17D, IL-17E (IL-25), and IL-17F—that collectively contribute to inflammatory signaling in psoriasis and psoriatic arthritis (PsA).[1] These cytokines are produced by both immune and nonimmune cell populations and signal through heterodimeric receptor complexes that share the IL-17 receptor A (IL-17RA) subunit, creating a central convergence point for downstream inflammatory pathways. In psoriatic disease, IL-17–mediated signaling drives keratinocyte activation, epidermal hyperplasia, neutrophil recruitment, and persistent inflammation across skin and musculoskeletal compartments.[2]

Although IL-23 is a key regulator of Th17 differentiation, IL-17 cytokines can be generated through IL-23-independent pathways, reflecting heterogeneous mechanisms of immune activation.[3] Classical Th17 cells retain the capacity to produce IL-17 in response to alternative inflammatory cues, while innate-like immune populations—including γδT cells, innate lymphoid cells, mast cells, and neutrophils—are enriched in psoriatic skin and synovium and can rapidly release IL-17 independently of IL-23. Histologic analyses further demonstrate that mast cells and neutrophils constitute major sources of IL-17 in lesional skin, whereas IL-17–positive T lymphocytes are relatively sparse, underscoring the importance of nonclassical IL-17–producing cells in tissue-level disease persistence.[4,5]

Transcriptomic studies provide strong support for the clinical relevance of these IL-23-independent pathways. Following IL-17RA blockade, lesional skin demonstrates rapid and broad normalization of the psoriatic transcriptome, with suppression of thousands of aberrantly expressed genes within weeks of treatment.[6] IL-17RA inhibition results in coordinated downregulation of multiple IL-17 ligands, including IL-17A, IL-17F, and IL-17C, and robust suppression of keratinocyte-driven inflammatory gene programs that are not fully normalized by IL-23 inhibition alone.[7,8]

Within this framework, IL-17C and IL-17E represent noncanonical IL-17 family members that are predominantly keratinocyte- and barrier-derived and signal through distinct IL-17RA-containing receptor complexes.[9] IL-17C functions in autocrine and paracrine loops to amplify epidermal inflammatory gene expression, while IL-17E contributes to barrier dysfunction and local immune amplification, both operating independently of IL-23.[10,11] Consequently, selective IL-17 ligand blockade may leave residual IL-17–family activity—particularly keratinocyte-intrinsic inflammatory circuits—intact despite clinical improvement.

Collectively, the convergence of canonical and noncanonical IL-17 signaling, IL-23-independent cytokine production, and keratinocyte-driven transcriptional programs at IL-17RA provides a strong biological rationale for receptor-level inhibition to achieve broader and more complete suppression of inflammation in psoriasis.

Clinical Trial-Based and Real-World Effectiveness of Brodalumab in Plaque Psoriasis

Pivotal Clinical Trials and Rapid Onset of Efficacy

The efficacy and safety of brodalumab, a human monoclonal antibody that selectively inhibits the interleukin-17 receptor subunit A (IL-17RA), have been demonstrated in multiple pivotal clinical trials. The AMAGINE program (AMAGINE-1, -2, and -3) compared brodalumab with placebo, while AMAGINE-2 and -3 additionally included ustekinumab as an active comparator, in adults with moderate-to-severe plaque psoriasis. Brodalumab was administered at 210 mg at weeks 0, 1, and 2, followed by every two weeks, while ustekinumab followed standard dosing regimens. In AMAGINE-1, brodalumab achieved PASI-75 and PASI-100 in 83.3% and 41.9% of patients, respectively versus 2.7% and 0.5% with placebo at week 12.[12] In AMAGINE-2 and -3, brodalumab demonstrated superior efficacy compared to both placebo and ustekinumab, with PASI-75 response rates at week 12 of 86%, 70%, and 8% (AMAGINE-2) and 85%, 69%, and 6% (AMAGINE-3), respectively.[13] Brodalumab also yielded higher PASI-100 rates versus ustekinumab at week 12 (44% vs 22% in AMAGINE-2 and 37% vs 19% in AMAGINE-3, $P<0.001$), highlighting profound skin clearance.[13] Patients maintained clinical response through 52 weeks, with exploratory analyses showing that 72% of PASI-100 responders at week 12 maintained PASI-100 at week 52,[14] and a post hoc analysis from AMAGINE-1 demonstrated 68.5% maintained PASI-100 over 108 weeks [data on file].

Brodalumab is also characterized by a rapid onset of efficacy. Among select IL-17 inhibitors, 25% of patients achieved PASI-75 in 2.1 weeks with brodalumab, compared with 2.4 weeks for ixekizumab and 3.0 weeks for high-dose secukinumab.[15] Time to 50% improvement in baseline PASI was 1.8 weeks for brodalumab versus 1.9 weeks for ixekizumab and 3 weeks for high-dose secukinumab, with a median time to PASI-75 of 4.2 weeks for brodalumab.[15] Network meta-analyses ranked brodalumab second among psoriasis biologics for PASI-75 at week 4 and highest for PASI-100 at week 4 [data on file]. This rapid clinical effect is attributable to its mechanism as an IL-17RA blocker, which inhibits multiple IL-17 cytokines, including IL-17A, IL-17F, IL-17A/F heterodimer, IL-17C, and IL-17E, resulting in a strong anti-inflammatory effect and early reversal of psoriasis-related gene expression.[16] Systematic reviews and real-world evidence corroborate these findings, showing faster PASI-90 and PASI-100 responses relative to other IL-17 and IL-23 inhibitors, which correlate with improved patient satisfaction and early symptom relief.[17]

Efficacy Across Patient Subgroups

Brodalumab demonstrates consistent efficacy across diverse patient subgroups. PASI response rates were comparable between obese and nonobese patients, with minor differences observed at later time points, indicating that BMI does not impair treatment response.[18] This is further reflected by the need for only a single dosing regimen for brodalumab, regardless of weight, whereas other biologics do require increased dosing. High PASI-75 and PASI-100 rates were observed across patients of different skin-of-color subgroups at weeks 12 and 52, with the strongest maintenance noted in Black patients (70% PASI-75, 60% PASI-100 at week 52).[19] Prior exposure to biologics did not impact efficacy; in post hoc analyses from AMAGINE-2 and -3, 40.9% of biologic-naïve and 39.5% of biologic-experienced patients achieved PASI-100 at week 12 versus 21.1% and 17.0% with ustekinumab, respectively.[20] These findings highlight brodalumab's broad applicability, including in patients with previous biologic failures, and support its use as a first-line or rescue therapy in moderate-to-severe psoriasis.[20] Systematic reviews also show efficacy in difficult-to-treat variants, including scalp, nail, genital, palmoplantar, erythrodermic, and pustular psoriasis.[21]

Rescue Efficacy

Brodalumab has demonstrated robust rescue performance in patients with inadequate responses to prior biologics. In AMAGINE-2 and -3, ustekinumab nonresponders who switched to brodalumab 210 mg every two weeks at week 16 achieved higher skin-clearance rates by week 52 than those who continued ustekinumab. PASI 75/90/100 responses were 72.6%, 58.1%, and 36.3% with brodalumab versus 61.7%, 25.5%, and 5.4% with continued ustekinumab, respectively.[22] A matching-adjusted indirect comparison (MAIC) with guselkumab in ustekinumab nonresponders confirmed higher PASI-90 at weeks 12 and 36 and higher PASI-100 at week 36 with brodalumab.[23] Furthermore, in the double-blinded 28-week COBRA trial comparing brodalumab with guselkumab 100 mg in patients who had failed ustekinumab, brodalumab showed numerically higher PASI100 responses at week 16 (53.4% vs 35.9%), earlier separation in time-to-response analyses beginning at week 2, and favorable secondary PASI outcomes, with comparable safety profiles, despite the primary endpoint not reaching statistical significance due to early trial termination.[24] Similar trends were observed in patients failing prior IL-17A inhibitors. In a single-center open-label study of 39 patients who failed secukinumab or ixekizumab (3 months of treatment), PASI 75/90/100 were achieved in 76%, 50%, and 32% of patients, respectively, at week 16.[25] These data support IL-17RA blockade as a unique mechanism for recapturing disease control in treatment-refractory patients.

Retreatment Efficacy

In AMAGINE-1, the study design included a planned withdrawal phase in which patients who achieved a static Physician's Global Assessment (sPGA) score of 0 or 1 at week 12 were eligible to switch from brodalumab 210 mg Q2W to placebo. Patients experiencing disease recurrence (sPGA $\geq$ 3) while in the withdrawal group were eligible for retreatment starting at week 16. Those who retreated with their induction dose of brodalumab 210 mg Q2W achieved robust recapture of response.[26,27] All patients regained PASI-75, with at least 90% achieving this response by week 8 of retreatment. PASI-90 and PASI-100 were recaptured by 96.2% and 83.5% of patients, respectively, with mean times to recapture of 29.7, 44.7, and 55.3 days for PASI-75, PASI-90, and PASI-100.[26] Among patients who achieved PASI-75 during the initial 12 weeks of treatment, 95.2%

recaptured PASI-75 after 6 weeks of retreatment, and 84.4% achieved PASI-100 after 24 weeks.[27] No patients retreated with brodalumab were antidrug antibody (ADA) positive.

Retreatment efficacy may be influenced by the immunogenicity of biologics —particularly the development of antidrug antibodies (ADAs) and neutralizing antibodies (NAbs)—which can emerge during the chronic cycle of treatment withdrawal, flare, and reinitiation.[26,28] Across approved monoclonal antibodies for moderate-to-severe plaque psoriasis, fully human agents generally showed lower ADA rates than humanized, chimeric, or Fab'-fragment antibodies, with infliximab exhibiting the highest incidence. Brodalumab has an exceptionally low immunogenicity, with only 3% of patients developing ADAs, of which none (0%) developed NAbs over the course of 52 weeks.[29] This low immunogenicity may explain the robust recapture rate (>=85% regaining PASI-90) by brodalumab.

Effectiveness of Brodalumab for High-Impact Sites

Psoriasis affecting high-impact and difficult-to-treat sites—including the scalp, nails, palms and soles, and genital region—disproportionately impairs quality of life and is often more refractory to treatment than generalized plaque disease. Achieving clearance in these areas is therefore a critical therapeutic goal. The efficacy of brodalumab in high-impact sites was evaluated in post hoc analyses of the phase 3 AMAGINE-1, -2, and -3 trials, using the Psoriasis Scalp Severity Index (PSSI) for scalp psoriasis through 12 weeks and the Nail Psoriasis Severity Index (NAPSI) for nail psoriasis through 52 weeks, with comparisons to placebo or ustekinumab, respectively.[30] In AMAGINE-1, brodalumab produced rapid and profound scalp clearance, with significant improvements evident by week 2 (67.6% mean improvement rate from baseline PSSI compared to 6.7% in placebo group). Week-12 PSSI75 and PSSI100 response rates of 89.0% and 63.4%, respectively, were significantly greater than placebo (9.5% and 3.2%, respectively). In AMAGINE-2/-3, brodalumab demonstrated progressive and superior nail clearance compared with ustekinumab, with mean NAPSI decreasing to 1.6 by week 52 and substantially higher rates of complete nail clearance (NAPSI 0) at week 52 (63.8% vs 39.1%).

Another descriptive real-world pilot study evaluated the effectiveness of brodalumab in patients with palmoplantar and genital psoriasis, assessing site-specific disease severity using validated clinician-reported outcome measures, including the palmoplantar Psoriasis Area and Severity Index (ppPASI) and the static Physician Global Assessment of Genitalia (sPGA-G), with longitudinal follow-up through 48 weeks.[31] Brodalumab produced rapid and sustained clearance in both sites, with 83% of patients achieving sPGA-G 0/1 at week 16 and 100% at weeks 24 and 48 for genital psoriasis, and 76.9% achieving ppPASI 75/90/100 at week 16 and 84.6% maintaining these responses at weeks 24 and 48 for palmoplantar psoriasis.

Real-World Effectiveness and Treatment Persistence

Real-world studies reinforce brodalumab's efficacy and safety across multiple populations. The prospective LIBERO study evaluated adults with mild-to-severe plaque psoriasis, demonstrating rapid improvement as early as week 2.[32] Between 77–85% of patients rated their skin as clear or almost clear, and mean DLQI scores improved substantially by week 12. Overall, 73.7% rated brodalumab as quite or very beneficial.[32]

Additional international cohorts corroborate these findings. In Portugal, 126 patients achieved PASI 75/90/100 rates of 83%, 57%, and 29% at week 4, rising to 96%, 93%, and 66% at week 74, with 82.5% drug survival and only mild infectious AEs.[33] In Canada, a retrospective cohort analysis of was conducted on all patients who initiated brodalumab treatment for PsO under the Canadian brodalumab Patient Support Program from July 1, 2018 to June 30, 2022.[34] Of 2482 patients, PASI 90 and PASI 100 responses were 66.1% and 53.2%, respectively, within 3 months, with sustained responses beyond 24 months; one-year treatment persistence was 73.4%. The Canadian Real World Evidence Study of Brodalumab in Plaque Psoriasis to Understand the Impact on Quality of Life and Work Productivity (CARE) also provided numerous insights into the real-world effectiveness of brodalumab in adult patients. Several interim analyses also revealed the following: (1) approximately half of patients on brodalumab achieved PASI 100 by month 6,[34] (2) patients showed comparable improvement across BMI subgroups (nonobese, obese, severely obese) at 6-month follow-up[34]; (3) improvement was also comparable between adults with PsO only and those with PsO and concomitant PsA[34]; (4) brodalumab demonstrated treatment persistence over 6 months with excellent tolerability and no serious adverse events.[34]

In the Czech Republic (BIOREP registry), 273 adults showed predicted drug survival of 92.4% at 6 months and 80.4% at 24 months; 96.5% achieved absolute PASI ≤3 at 12 months and 87.3% achieved DLQI 0/1.[35] In Greece, the BrIDGE study (n=200) reported 42% achieving PASI100 at week 24 and 65% at week 104, with sPGA 0/1 responses of 82.8–92.5% through week 104, mean DLQI improved by 11.4 points, and adherence was 98.9%.[36]

Italian real-world studies further demonstrate rapid, sustained efficacy across subgroups. In a cohort of 299 patients, mean PASI decreased from 15.9 at baseline to 5.4 at week 4 and 0.8 at week 52; PASI 75, 90, and 100 were comparable between bio-naïve and bio-experienced patients, with only modest differences by BMI.[37] In elderly patients (n=69, mean age 70.7 years), brodalumab reduced mean PASI from 15.4 at baseline to 5.2 at week 4 and 0.5 at weeks 36 and 52, with 95.5% achieving PASI-75, 86.4% PASI-90, and 75.8% PASI-100, and DLQI improved from 14.4 to 1.2; AEs were reported in 5.8% and treatment discontinuation occurred in 8.7%.[38]

Real-world data also confirm effectiveness in difficult-to-treat regions. In Germany, 87 patients with nail and scalp involvement achieved PSSI75 at week 12 (93.6%) and NAPSI75 at week 24 (90.3%), with median BSA involvement declining from 14% to 1–1.5% and DLQI improving from 16 to 1–2, sustained over 60 weeks.[39]

Collectively, these data demonstrate that brodalumab delivers rapid, profound, and durable skin clearance across diverse populations, disease severities, prior biologic exposures, elderly patients, and difficult-to-treat sites, with low immunogenicity and high treatment adherence.

Brodalumab in Psoriatic Arthritis: Clinical and Real-World Evidence

Although brodalumab is not approved for psoriatic arthritis in the United States, it has demonstrated joint and skin efficacy in randomized clinical trials and is approved for PsA in Japan, with additional supportive evidence emerging from post hoc analyses and real-world cohorts. The following section summarizes the totality of evidence evaluating

brodalumab in psoriatic arthritis, including phase III clinical trials, analyses in patients with concomitant PsA, and real-world outcomes from routine clinical practice.

The AMVISION-1 and AMVISION-2 studies were 24-week randomized, double-blind, placebo-controlled phase III trials evaluating brodalumab in adults with psoriatic arthritis.[40] The primary endpoint was ACR20 at week 16 in both trials, with patients randomized to brodalumab or placebo under identical blind conditions. Participants were randomized to receive brodalumab or placebo at weeks 0, 1, and 2 and then every 2 weeks thereafter. Secondary efficacy endpoints included ACR 50/70 and Psoriasis Area and Severity Index (PASI) 75/90/100 response rates. Concomitant NSAIDs, DMARDs (methotrexate, sulfasalazine or leflunomide) and corticosteroids (≤10 mg/day prednisone or equivalent) were permitted, provided the dose was stable for ≥4 weeks prior to initiation of trial treatment. In the pooled analysis of 962 patients with active psoriatic arthritis, brodalumab demonstrated significantly greater joint and skin improvements compared with placebo. At week 16, ACR20 response rates were 47.9% for brodalumab 210 mg, vs 20.9% for placebo, with similar responses maintained at week 24. Brodalumab also produced higher rates of ACR50 and ACR70, as well as greater PASI75, PASI90, and PASI100 skin clearance. Additionally, a significantly larger proportion of patients achieved resolution of dactylitis and enthesitis compared with placebo (all $P<0.01$). Adverse event rates at week 16 were comparable across groups—54.4% for placebo and 54.5% for brodalumab 210 mg—with no new safety signals reported.

In a post hoc analysis of the phase 3 AMAGINE-2 and -3 trials, brodalumab demonstrated significantly higher rates of complete skin clearance (PASI 100), improved symptom severity (PSI), and better quality of life (DLQI 0/1) compared with ustekinumab specifically in patients with moderate-to-severe psoriasis who had concomitant psoriatic arthritis.41 Among these PsA patients, brodalumab more than tripled the odds of achieving PASI 100 (OR 3.15) and substantially increased the likelihood of DLQI 0/1 and PSI ≤8 responses, showing rapid, sustained, and robust benefits for both skin and patient-reported outcomes, while similar advantages were also observed in patients without PsA.

Real-world evidence from European clinical practice supports the effectiveness and tolerability of brodalumab in patients with psoriasis and concomitant psoriatic arthritis (PsA). In a Danish single-center retrospective study of 83 patients treated with brodalumab for psoriasis, 18 patients (21.7%) had PsA, among whom 44.4% achieved complete remission and 11.1% achieved partial remission of joint disease during follow-up, while 27.7% experienced treatment failure; overall drug survival at 52 weeks was 65.7%, and substantial improvements were observed in skin outcomes (65.4% PASI-90, 55.8% PASI-100) and quality of life (mean DLQI reduction from 8.2 to 2.9, with 60.8% achieving DLQI 0/1).[42] Complementary findings were reported from the German Psoriasis Registry PsoBest, which evaluated 227 patients initiating brodalumab in routine care, including 38 patients with concomitant PsA, and demonstrated rapid improvements in both skin and joint symptoms within 3 months that were sustained through 12 months; in the PsA subgroup, mean PsA severity scores decreased from 4.2 to 2.3, tender joint counts declined from 8.6 to 5.5, and meaningful improvements in patient-reported outcomes were observed, with DLQI decreasing from 11.4 to 2.2 and approximately 76.5% of patients achieving DLQI 0/1 at 12 months.[43] Drug persistence in the German cohort was high, with an overall 12-month survival rate of approximately 76% and similar or numerically higher persistence in patients with PsA compared with those without. Collectively, these real-world data demonstrate that brodalumab provides clinically meaningful and sustained improvements in joint symptoms, skin disease, and quality of life in patients with psoriatic arthritis in routine practice, with a safety profile consistent with clinical trial experience.

Long-Term Safety Profile

Overview of Adverse Events

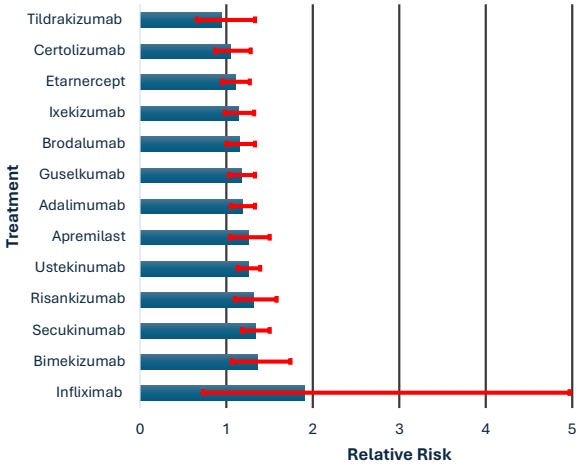
In the 8-year postmarketing setting, the pattern and frequency of commonly reported adverse events (AEs) with brodalumab were consistent with those described in the prescribing information based on pooled clinical trial data. In controlled trials, the most common AEs reported with brodalumab included arthralgia (~6%), headache (~4%), fatigue (~3%), injection-site reactions (~3%), diarrhea (~3%), and myalgia (~2%).[29] In real-world postmarketing use, these same events remained the most frequently reported but occurred at substantially lower crude reporting rates, including arthralgia (2.58 per 100 patients), headache (1.04 per 100 patients), fatigue (1.03 per 100 patients), injection-site reactions (0.80 per 100 patients), myalgia (0.73 per 100 patients), and diarrhea (0.65 per 100 patients) [data on file]. Importantly, few new cases were reported since the 7-year analysis (eg, 6 new cases of arthralgia and ≤4 new cases for other common AEs), indicating no evidence of increasing incidence or emerging safety signals with extended exposure. Rare events, such as neutropenia and tinea infections, were infrequently reported (0.02 per 100 patients each), further supporting the long-term stability of the brodalumab safety profile.

Similarly, adverse events of special interest (AESIs) observed in the 8-year postmarketing setting were consistent with those reported in the prescribing information derived from clinical trial data. In pooled trials, serious infections occurred in approximately 1–2% of patients, depression-related events in ~1%, malignancies in <1%, and inflammatory bowel disease (IBD) events were rare (<0.1%), with no increased risk of major adverse cardiovascular events (MACE) identified. In real-world postmarketing use, serious infections were reported at a crude rate of approximately 2.1 per 100 patients, while depression and malignancy were reported at rates of 1.10 and 1.13 per 100 patients, respectively. Adjudicated MACE were uncommon (0.25 per 100 patients), fungal infections were infrequent (0.18 per 100 patients), and IBD was rare (0.05 per 100 patients), supporting the absence of emerging or cumulative AESI safety signals with long-term brodalumab exposure.

Infection Risk and Opportunistic Infections

Biologic therapies have transformed the management of psoriasis by targeting key immune pathways; however, their immunomodulatory effects raise important considerations regarding infection risk. A recent network meta-analysis evaluating infection and serious infection rates during placebo-controlled clinical trials found that brodalumab had the lowest relative risk among IL-17 inhibitors, ranking 10th out of 14 systemic agents in psoriasis and 7th out of 18 agents in psoriatic arthritis (Figure 1),[44] findings that are consistent with the low rates of serious infection observed in long-term postmarketing analyses. Among infectious complications, fungal infections warrant particular attention, as IL-17 inhibition is associated with an increased risk of mucocutaneous candidiasis; however, susceptibility varies by underlying disease,

FIGURE 1. Risk of Infection for Various PsO Treatments compared to Placebo. Data sourced from Chiu et al., *Ther Adv Chronic Dis.* 2023;14:1–11.[44]



treatment duration, dose, and the specific IL-17 inhibitor used[45] Reported incidences of candidiasis range from 0–3.5% for ixekizumab, 0.3–7% for brodalumab, 1.4–13.5% for secukinumab, and 1.9–21.2% for bimekizumab.[46,47] Brodalumab, which blocks the IL-17 receptor A and thereby inhibits signaling from multiple IL-17 cytokines, has generally demonstrated low rates of candidiasis, potentially reflecting residual IL-17 cytokine signaling between dosing cycles that may help preserve antifungal host defenses.[48] However, targeted dual cytokine inhibition or selective IL-17A blockade has been associated with higher rates of mucocutaneous fungal infections.[49] Consistent with these observations, a 2025 real-world analysis of nonsponsored, open-label, and real-world studies reported no serious fungal infections with brodalumab and a low overall fungal infection rate of 0.16 per 100 patients.[50] Collectively, these findings indicate that while IL-17 inhibitors carry a theoretical risk of fungal infections, brodalumab, as an IL-17RA blocker, demonstrates a consistently low incidence of candidiasis across clinical trials and real-world studies, supporting a favorable long-term infection safety profile.

Malignancy Risk

Across long-term postmarketing surveillance and real-world observational studies, malignancies associated with brodalumab were reported infrequently and without evidence of an emerging safety signal. In the 8-year US postmarketing pharmacovigilance analysis, malignancy was reported at a low crude reporting rate of approximately 1.13 per 100 patients, with no indication of increasing incidence over time (data on file). Consistent findings were observed across nonsponsored open-label and real-world studies, including a large German real-world cohort with over 600 patients included in the safety analysis, in which malignancies were uncommon and no clustering by cancer type was observed over short- and long-term follow-up.[50] Reported malignancies across studies were rare and heterogeneous, consisting of isolated cases without consistent attribution to brodalumab, including sporadic solid tumors such as leiomyosarcoma and colon cancer.[50] Large observational and pharmacovigilance analyses did not identify an increased risk of malignancy with brodalumab compared with other biologic therapies used in psoriasis. Collectively, these data indicate that malignancy events reported with brodalumab occur at low rates and are consistent with background expectations in patients with moderate-to-severe psoriasis, supporting a stable long-term malignancy safety profile.

Depression, Suicidal Ideation, and Suicidal Behavior

Patients with psoriasis have a well-established increased baseline risk of depression and suicidal ideation and behavior (SIB) compared with the general population, reflecting the cumulative burden of chronic disease, stigma, functional impairment, and psychosocial stressors.[51] Meta-analyses show approximately two-fold higher odds of suicidal ideation (OR 2.05; 95% CI 1.54–2.74) and a significantly increased risk of suicidal behaviors overall (OR 1.26; 95% CI 1.13–1.40), including higher odds of suicide attempts (OR 1.32) and completed suicide (OR 1.20).[52] These risks are observed independent of systemic therapy, underscoring the need to contextualize neuropsychiatric events during biologic treatment within this background risk

Psychiatric adverse events and SIB associated with brodalumab were evaluated across five psoriasis clinical trials, including placebo-controlled and active-controlled phases, encompassing 4,464 patients and 9,161.8 patient-years of exposure.[53] During the 12-week placebo-controlled period, no completed suicides occurred in any treatment group, and rates of depression and other psychiatric adverse events were comparable among brodalumab, placebo, and ustekinumab. Through 52 weeks, follow-up–adjusted incidence rates of overall SIB were 0.20 per 100 patient-years with brodalumab and 0.60 per 100 patient-years with ustekinumab, with overlapping confidence intervals, indicating no imbalance during controlled study periods.[53]

Across the full brodalumab clinical program, four completed suicides were initially reported, of which three were confirmed after independent adjudication, corresponding to a follow-up–adjusted incidence rate of 0.04 per 100 patient-years.[53] All confirmed cases occurred in men aged 39–59 years with substantial pre-existing psychiatric disease and/or major psychosocial stressors, including financial hardship, legal difficulties, social isolation, or ongoing treatment for depression and anxiety. One additional fatal event was adjudicated as indeterminate, involving mixed opiate toxicity in the context of antidepressant and benzodiazepine use, and was not classified as a confirmed suicide. No temporal clustering around treatment initiation, dose-response relationship, or increase following randomized withdrawal of brodalumab was observed.[53] In light of these findings, brodalumab is available in the United States through a Risk Evaluation and Mitigation Strategy (REMS) program, which requires prescriber certification and patient counseling to ensure appropriate risk–benefit assessment, particularly in individuals with a history of depression or suicidality.[29]

Further analyses demonstrated that SIB events occurred predominantly among patients with known risk factors. Follow-up–adjusted incidence rates of SIB were markedly higher among patients with a history of depression (1.42 per 100 patient-years) or prior suicidality (3.21 per 100 patient-years) compared with those without such histories (0.21 and 0.20 per 100 patient-years, respectively).[53] No increase in SIB was observed following randomized withdrawal of brodalumab, and depressive and anxiety symptoms assessed using the Hospital Anxiety and Depression Scale showed greater improvement with brodalumab than placebo during controlled treatment.[53]

Postmarketing and real-world data provide additional context. In an 8-year U.S. postmarketing safety analysis (2017–2025) involving 5,654 patients, depression was reported at a crude rate of approximately 1.10 per 100 patients, and no completed suicides were reported during the observation period.[50] These crude reporting rates are comparable to,

TABLE 1.

Total Completed Suicides, Total Patients Prescribed, and Suicides Per Total Patients Prescribed For All Indications For Each Biologic

Biologic name	Completed suicides since approval	Total patients prescribed	Corresponding date for total patients prescribed	Suicides per total patients prescribed
Tildrakizumab	0	n/a	n/a	n/a
Risankizumab	0	n/a	n/a	n/a
Brodalumab	1*	20,871	July 31, 2021	4.79×10^{-5}
Ixekizumab	4	175,000	February 28, 2021	2.29×10^{-5}
Guselkumab	4	n/a	n/a	n/a
Certolizumab	12	n/a	n/a	n/a
Ustekinumab	12	n/a	n/a	n/a
Secukinumab	17	>500,000†	December 23, 2021	3.20×10^{-5}†
Etanercept	62	n/a	n/a	n/a
Infliximab	84	3,100,000	August 01, 2020	2.61×10^{-5}
Adalimumab	175	>1,400,000†	January 01, 2020	1.04×10^{-4}†

Table sourced from: Yeroushalmi S, Chung M, Bartholomew E, Hakimi M, Koo J. Examining worldwide postmarketing suicides from biologics used for psoriasis with a focus on brodalumab: A cross-sectional analysis using the FAERS database. *JAAD Int.* 2022;9:119–121.[54]

or lower than, background psoriasis estimates, in which depression is common and SIB incidence ranges from approximately 0.09–0.54 per 100 patient-years [Kurd et al, 2010; Pompili et al, 2016]. Large global pharmacovigilance analyses similarly report very low absolute numbers of completed suicides among patients prescribed brodalumab, with rates comparable to other biologic therapies used for psoriasis (Table 1).[54] Taken together, the totality of clinical trial, postmarketing, and real-world evidence does not support a causal association between brodalumab treatment and increased risk of depression or suicidal behavior.

DISCUSSION

Brodalumab demonstrates a distinct and clinically meaningful role in the management of moderate-to-severe plaque psoriasis, supported by converging evidence from randomized clinical trials, long-term extension studies, and extensive real-world experience. Across the AMAGINE program, brodalumab achieved rapid onset of action and high rates of complete and near-complete skin clearance, with PASI 90 and PASI 100 responses observed early and sustained over time. These findings are consistently reproduced in real-world cohorts, which show durable effectiveness, high treatment persistence, and meaningful improvements in quality of life across diverse patient populations.

A defining feature of brodalumab is its unique mechanism as a selective interleukin-17 receptor A (IL-17RA) antagonist, which inhibits signaling from multiple IL-17 family cytokines rather than IL-17A alone. This broader pathway blockade likely contributes to its rapid clinical response, depth of clearance, and ability to recapture disease control in patients with prior biologic failure, including those who have failed other IL-17 inhibitors. Clinically, this positions brodalumab as an effective option for biologic-naïve patients requiring fast disease control as well as for those with refractory or difficult-to-treat disease.

Efficacy has been shown to be consistent across key subgroups, including patients with obesity, skin of color, elderly individuals, and those with high-impact anatomic involvement such as the scalp, nails, palms, soles, and genital region. The long-term safety profile of brodalumab has remained stable, with low rates of serious infection, malignancy, inflammatory bowel disease, and major adverse cardiovascular events. When contextualized against the elevated baseline risk inherent to psoriasis, the totality of evidence does not support a causal association with psychiatric adverse events, with ongoing risk mitigation ensured through the REMS program. Collectively, these data support brodalumab as a differentiated, effective, and durable therapeutic option within the evolving psoriasis treatment landscape.

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