

The Efficacy of Different Treatment Options in Cutaneous Sarcoidosis: A Meta-analysis of RCTs

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Cutaneous sarcoidosis is a granulomatous skin disease associated with significant quality-of-life impairment. Evidence from randomized controlled trials (RCTs) guiding systemic or targeted therapy remains limited. To assess the efficacy and safety of pharmacological therapies for cutaneous sarcoidosis in randomized placebo-controlled trials PubMed, the Cochrane Library and ClinicalTrials.gov were searched through January 2025. Eligible studies were RCTs including adults with cutaneous sarcoidosis randomized to pharmacological therapy vs placebo. Outcomes were clinical response, quality of life and serious adverse events. Pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated using random-effects models. Six RCTs including 156 participants were analysed. Pharmacological therapies did not significantly improve clinical response compared with placebo (OR 2.24; 95% CI, 0.73–6.90; $p=0.16$). A nonsignificant trend toward improved quality of life was observed (OR 2.23; 95% CI, 0.87–5.73; $p=0.09$). No significant difference in serious adverse events was found between groups (OR 0.97; 95% CI, 0.48–1.96; $p=0.93$). Risk of bias ranged from moderate to high, with low statistical heterogeneity. Pharmacological add-on therapies showed no significant efficacy beyond corticosteroids but may improve quality of life. Larger, well-designed RCTs are needed.

Key words: cutaneous sarcoidosis; meta-analysis; randomized controlled trials; dermatology; immunomodulation; sarcoidosis; skin; granulomatous disease.

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Sarcoidosis is a potentially multisystem disease characterized by non-caseating granulomas of unknown cause (1, 2). Cutaneous involvement occurs in approximately 25% of patients with systemic sarcoidosis, making the skin one of the most common extrapulmonary organs affected (3, 4). Lesion

SIGNIFICANCE

Sarcoidosis is a systemic, granulomatous disease of unknown cause. Cutaneous involvement, occurring in roughly 25% of systemic cases, often causes disfiguring lesions and significant psychosocial distress. No consensus exists on optimal therapy, and randomized trials are few. We performed a systematic meta-analysis of placebo-controlled trials to evaluate pharmacological treatments for cutaneous sarcoidosis, aiming to clarify their efficacy and safety. This up-to-date synthesis can help guide clinicians in a rare disease with historically limited evidence.

morphology has prognostic significance; erythema nodosum typically indicates acute sarcoidosis with a favourable prognosis (e.g. Löfgren's syndrome), whereas lupus pernio is strongly predictive of chronic, treatment-refractory sarcoidosis and systemic morbidity (5). Cutaneous manifestations may additionally serve as early clinical indicators or prodromal signs of systemic sarcoidosis involvement (1, 6, 7). Persistent disfiguring lesions can influence patient adherence, long-term corticosteroid use and demand for novel therapies. Importantly, cutaneous lesions heavily impact quality of life, causing social stigma, depression and poor DLQI scores (8).

Systemic corticosteroids remain the cornerstone of therapy in cutaneous sarcoidosis; however, long-term use is limited by toxicity, and robust evidence specific to cutaneous disease is lacking. Nonsteroidal agents such as thalidomide, tumour-necrosis-factor- α (TNF- α) inhibitors and combination antibiotics have been used as steroid-sparing therapies (9), but most data derive from small case series or subsets of systemic trials.

The present systematic review and meta-analysis was conducted to synthesise the available randomized, double-blind, placebo-controlled evidence for pharmacological therapies in cutaneous sarcoidosis. Specifically, to assess the efficacy of systemic or targeted interventions on clinical response, health-related quality of life and serious adverse events. By clarifying the strength and certainty of the existing

evidence, we hope to inform clinicians and identify priorities for future research.

MATERIAL AND METHODS

Search strategy

We conducted a systematic review and meta-analysis of double-blind randomized controlled trials (RCTs) evaluating pharmacological therapies for cutaneous sarcoidosis, following Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines. The protocol was registered CRD420261292634.

A comprehensive literature search was performed through January 2025 in PubMed, the Cochrane Library and ClinicalTrials.gov.

Eligibility criteria and data extraction

Eligible studies enrolled adults with cutaneous sarcoidosis and compared an active treatment to placebo. Only published, double-blind RCTs in English or German reporting skin-specific outcomes were included. Trials of systemic sarcoidosis were eligible if cutaneous data were reported separately. Two reviewers independently screened titles, abstracts and full texts. Discrepancies were resolved by consensus between FCT and LB. In case of conflicting interpretation, a third reviewer (MRG) served as deciding vote.

Statistical analysis

The primary outcome was clinical response. Secondary outcomes included quality of life (QoL) and serious adverse events (SAEs). Data were extracted as binary outcomes where possible to calculate odds ratios (ORs) with 95% confidence intervals (CIs). A random-effects model (DerSimonian-Laird method) was used throughout. Forest plots were generated for each outcome; studies with zero events in both arms were excluded from pooled estimates. Heterogeneity was quantified using the I^2 statistic. Publication bias assessment was not performed due to the small number of studies.

Sensitivity and subgroup analyses (by drug class and administration route) were conducted to assess robustness. Full forest plots are provided in Appendix S1.

Pooled effect estimates were calculated using a random-effects model because of anticipated clinical and methodological heterogeneity.

Risk of bias and quality assessment

Risk of bias was assessed by 2 reviewers FCT and LB using Cochrane RoB-2 (domains: randomization, deviations from intended interventions,

missing outcome data, outcome measurement, selective reporting) via signalling questions; disagreements were resolved by consensus or a third reviewer (domain 2 assessed adherence to interventions, domain 3 attrition and completeness of outcome data). Summary of findings tables were generated using the Grading of Recommendations Assessment, Development and Evolution (GRADE) system (Appendix S2). Two reviewers (with consensus and a third adjudicator if needed) rated evidence certainty (risk of bias, inconsistency, indirectness, imprecision, publication bias); pooled odds ratios and assumed baseline risk yielded absolute effects, and explanatory footnotes (a–d) justified any downgrading.

RESULTS

From a total of 181 studies, 6 eligible studies comprising a total of 156 participants were included (**Fig. 1** and Appendix S1: Table SIII). Interventions included infliximab (10), adalimumab (11), golimumab (12), ustekinumab (12), thalidomide (13), a topical mTOR inhibitor (sirolimus) (14) and an oral antimycobacterial combination (CLEAR regimen) (15). All trials permitted concomitant corticosteroids. Details of included and excluded studies are provided in **Table I** and Appendix S1; Table SIII. Heterogeneity analysis revealed moderate variability across studies, with $\tau^2=0.56$ and $I^2=35\%$, suggesting that approximately one-third of the total variability can be attributed to true differences between studies rather than random chance (**Fig. 2**). By contrast, heterogeneity for the secondary outcomes (QoL, SAEs) was $I^2=0\%$, supporting fixed-effect sensitivity analyses for those endpoints.

Included studies

The studies included in this meta-analysis compared the following treatments: adalimumab, infliximab, thalidomide, golimumab, ustekinumab and the CLEAR regimen to placebo and can be found in Table I (10–15). Information on patients' race, ethnicity or country of origin was reported inconsistently across the included RCTs. When available, these data were extracted and are summarized in Table I. Owing to the limited and heterogeneous reporting, subgroup analyses by race, ethnicity or country of origin could not be performed.

Risk of bias assessment

Overall, the quality of evidence was poor, 4 out of 6 studies exhibited a moderate-to-high risk of bias and lacked precision due to small sample sizes (**Table II**). Most studies presented some degree of risk across nearly all domains. This resulted in a high-to-moderate overall risk for all studies, except for 2 (11, 13). In the case of Baughman et al., many critical details were

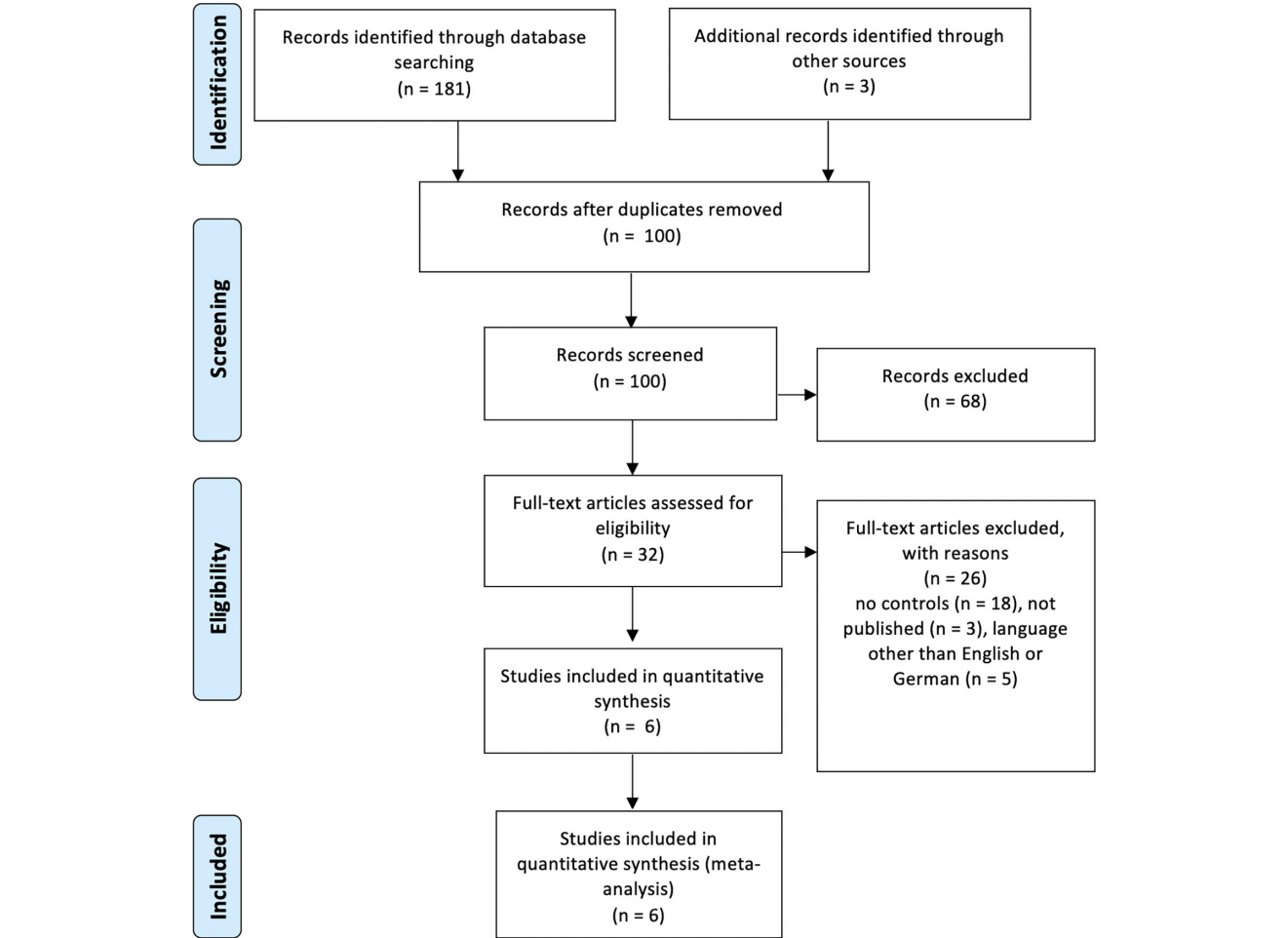


Fig. 1. PRISMA flow chart of the selection process of randomized clinical trials investigating the effect of different pharmacological treatment options for cutaneous sarcoidosis.

Table I. Overview of Included studies. All included studies allowed corticosteroids as background therapy

Study	N=included patients	Treatment	African American	White	Asian	Other	Comment
1 Baughman 2015 (10)	17	Infliximab	10	6	0	1	
2 Drake ^a 2013 (15)	17	CLEAR regime	NR	NR	NR	NR	Levofloxacin, ethambutol, azithromycin, rifampin
3 Droitcourt ^b 2014 (13)	39	Thalidomide	NR	NR	NR	NR	One number was missing in the ClinicalTrials.gov ID
4 Judson ^c 2014 (12)	58	Golimumab, Ustekinumab	NR	NR	NR	NR	
5 Pariser 2013 (11)	15	Adalimumab	14	1	0	0	
6 Redl 2024 (14)	10	Sirolimus	0	9	1	0	

^aDrake et al.: the meta-analysis includes only patients with biopsy proven sarcoidosis.

^bDroitcourt et al.: did not include any details about ethnicity.

^cJudson et al.: the Skin group did not include any details about ethnicity.

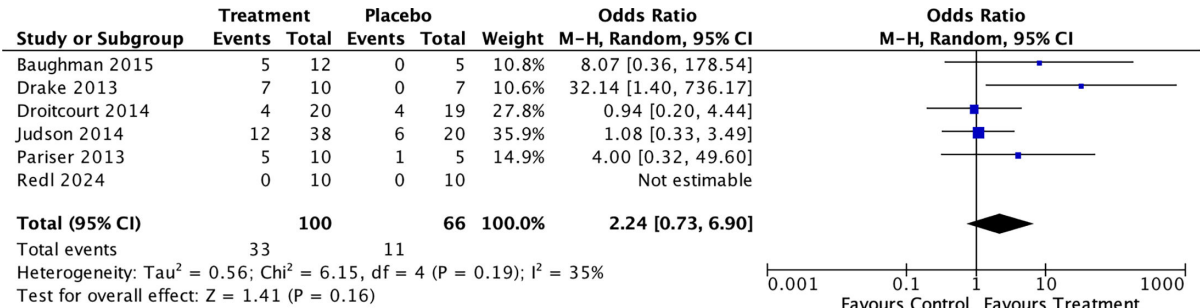


Fig. 2. Forest plot of different treatment effects on the primary endpoint (response as defined in the individual study).

Table II. Summary assessment of the methodological quality of randomized trials included in the systematic review)

Study ID	Risk of bias judgement					Overall
	D1	D2	D3	D4	D5	
Baughman 2015	Some concerns	Some concerns	Low	Low	Some concerns	Some concerns
Drake 2013	Low	Some concerns	High	Low	Low	High
Droitcourt 2014	Low	Low	Low	Low	Low	Low
Judson 2014	Some concerns	Low	High	Low	Low	High
Pariser 2013	Low	Low	Low	Low	Low	Low
Redl 2024	High	Low	High	Some concerns	Low	High

D1-D5: Risk of domains 1-5, D1: Bias arising from the randomization process, D2: Bias due to deviations from intended intervention, D3: Bias due to missing outcome data, D4: Bias in measurement of the outcome, D5: Bias in selection of the reported result.

missing in the available report on ClinicalTrials.gov. In the remaining studies, the high overall risk was mainly attributed to missing outcome data (10, 12, 14, 15).

The bias assessment was conducted independently by 2 reviewers. Across trials, the most frequent sources of potential bias were Domain 1 (randomization and allocation concealment) and Domain 3 (missing outcome data). Redl et al. raised some concerns due to block randomization predictability and missing outcome data in the second treatment period, resulting in an overall "high risk" of bias for that study. Discrepancies arose in the risk of bias evaluation in Domain 1 of Redl et al. (Table II and Appendix S1: Fig. S3) (14).

The certainty of evidence overall using the GRADE GDT pro tool was very low (Appendix S2).

The summary of the risk of bias assessment for each study is shown in Table II.

Assessment of the quality of evidence

The quality was evaluated by 2 authors using the GRADE system. The primary outcome of clinical response, assumed risk with placebo was 167 per 1,000 and the risk with active treatment was 309 per 1,000 (OR 2.24, 95% CI 0.73–6.90), but the certainty of evidence was very low due to risk of bias, heterogeneity, imprecision and potential publication bias. In trials of TNF- α inhibitors vs placebo, the risk with placebo was 233 per 1,000 and with biologic immunomodulators 336 per 1,000 (OR 1.66, 95% CI 0.60–4.62; very-low certainty). For systemic therapies, the risk with placebo

was 196 per 1,000 vs 354 per 1,000 with treatment (OR 2.24, 95% CI 0.73–6.90; very-low certainty). Quality-of-life improvements were observed in 549 per 1,000 treated patients vs 353 per 1,000 in the placebo group (OR 2.23, 95% CI 0.87–5.73; low certainty). Serious adverse events occurred in similar proportions: 134 per 1,000 in the treatment group vs 137 per 1,000 with placebo (OR 0.97, 95% CI 0.48–1.96; very-low certainty). These estimates underline that confidence intervals encompass no effect, and evidence certainty is low to very low across outcomes.

Serious adverse events

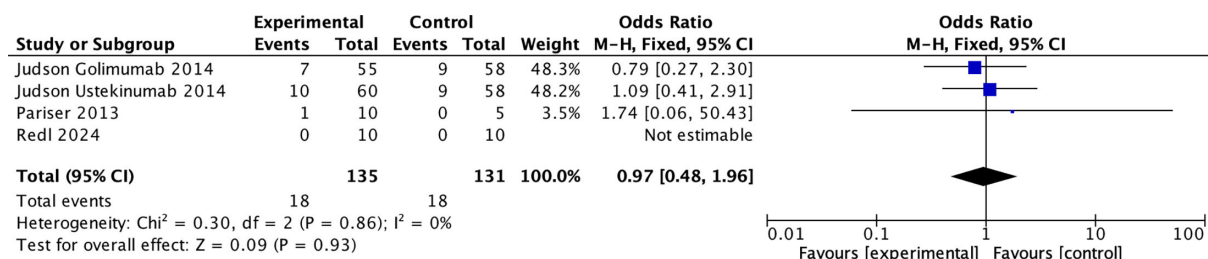
A total of 135 patients in the intervention arms and 131 in the control arms were included in this analysis. Pooled analysis demonstrated no significant difference in the risk of SAEs between treatment and control groups (Fig. 3). Reported SAEs included pneumonia requiring hospitalization (11) and various infections, respiratory and cardiac disorders (12). In the study by Droitcourt et al. (13), selected patients in the thalidomide group experienced multiorgan symptoms such as abdominal pain, dizziness, leg oedema, rash and dyspnoea; one placebo-treated patient developed sensorimotor neuropathy confirmed by electromyography. No SAEs were reported in the study by Redl et al. (14). These findings indicate that treatment did not significantly increase the risk of SAEs, as the pooled estimate was close to 1 and nonsignificant. However, confidence intervals remain wide, particularly in Pariser et al. (11), limiting the precision of these estimates.

The low event rates further limit definitive conclusions, although many of the investigated medications, such as systemic sirolimus, have been in clinical use for extended periods with substantial post-marketing safety data available (16, 17).

Quality of life

Quality of life was measured using DLQI, Skindex-29 and the Sarcoidosis Health Questionnaire; improvement was defined as a ≥ 4 -point reduction, based on established minimal clinically important differences (details and psychometric rationale in Appendix S3).

The impact of treatment on health-related quality of life (QoL) was evaluated using a dichotomous

**Fig. 3. Forest plot of the secondary endpoint, serious adverse events in cutaneous sarcoidosis.**

approach across 3 studies. The pooled risk ratio for improvement in QoL was 2.23, suggesting a trend toward improvement in the intervention groups, though statistical significance was not reached (Fig. 4). These findings indicate that while treatment may enhance QoL, the wide confidence intervals and dichotomization of continuous scores limit interpretability. Future studies should prioritize standardized QoL reporting with continuous effect estimates to facilitate more precise assessments.

Sensitivity analysis

A leave-one-out sensitivity analysis to assess the robustness of our primary findings was performed. Excluding each study sequentially yielded pooled effect estimates (Odds Ratios [OR]) ranging from 1.39 (95% CI, 0.60–3.24; without Drake 2013) to 3.73 (95% CI, 0.78–17.83; without Judson 2014 Appendix S1; Figs S6–S11) (12, 15). None of the sensitivity analyses substantially altered the overall conclusion, as all remained statistically nonsignificant (*p* values ranging from 0.10 to 0.44). Heterogeneity across analyses was generally low to moderate (*I*²=0%–47%).

Additional subgroup analyses were conducted to explore potential differences in treatment effect by therapeutic class and route of administration.

Counts by subgroup were as follows: TNF- α inhibitors (3 comparisons: infliximab, adalimumab, golimumab) vs non-TNF agents (3 comparisons: thalidomide, ustekinumab, CLEAR regimen, Appendix S1; Fig. S13); by route, systemic treatments (6 active-vs-placebo comparisons across 5 studies) vs nonsystemic (1 study: topical sirolimus, Appendix S1; Fig. S14) and immunomodulatory agents (thalidomide, infliximab, adalimumab) vs nonimmunomodulatory interventions (Golimumab, sirolimus, CLEAR regime, Appendix S1; Fig. S12). In Judson et al. (12), the golimumab and ustekinumab arms were analysed as separate active-placebo comparisons; to avoid unit-of-analysis errors with a shared placebo, the placebo group was allocated proportionally across comparisons.

Analysis of studies evaluating TNF- α inhibitors (adalimumab, infliximab, golimumab) demonstrated no significant treatment effect compared to placebo (*Z*=0.97, *p*=0.33), with minimal heterogeneity across studies (*I*²=1%), suggesting a consistent but

nonsignificant effect within this drug class (Appendix S1; Fig. S13).

DISCUSSION

To our knowledge, this is the first meta-analysis investigating the efficacy of immunotherapy beyond corticosteroids on clinical outcomes in cutaneous sarcoidosis. Although the broad confidence intervals may suggest a possible benefit of immunomodulatory therapy over placebo, the nonsignificant *p* value reflects the limited power and variability of available data. Heterogeneity was moderate for the primary endpoint (*I*²=35%) and negligible for quality-of-life and serious adverse event endpoints (*I*²=0%), underscoring the importance of interpreting the pooled estimates cautiously.

Unlike pulmonary and cardiac sarcoidosis, which determine morbidity and mortality, cutaneous sarcoidosis rarely threatens life but carries a substantial psychosocial burden. Shared decision-making is thus critical: patients with nonfacial lesions may elect to delay treatment when physical symptoms are minimal, whereas distress about cosmetic appearance or pruritus may prompt therapy (18). Conversely, patients with lupus pernio often require aggressive treatment, yet residual depigmentation and scarring can cause ongoing psychological distress (18). These considerations emphasize that in future clinical trials health-related quality-of-life measures (e.g. DLQI, Skindex-29, Sarcoidosis Health Questionnaire) should be prioritized alongside clinician-reported outcomes.

The available evidence for pharmacologic treatment of cutaneous sarcoidosis is limited to a handful of small, heterogeneous RCTs with varying outcome definitions (10–15). High or unclear risk of bias was common because of inadequate concealment, incomplete outcome reporting or concomitant steroid use. Heterogeneity across studies prevented meaningful subgroup analyses, and the overall precision of pooled estimates was low. Unfortunately, publication bias could not be assessed reliably. Furthermore, the lack of standardized quality-of-life instruments limits the comparability of patient-reported outcomes. Only a limited number of manuscripts reported patients’ race, ethnicity or country of origin, and the terminology

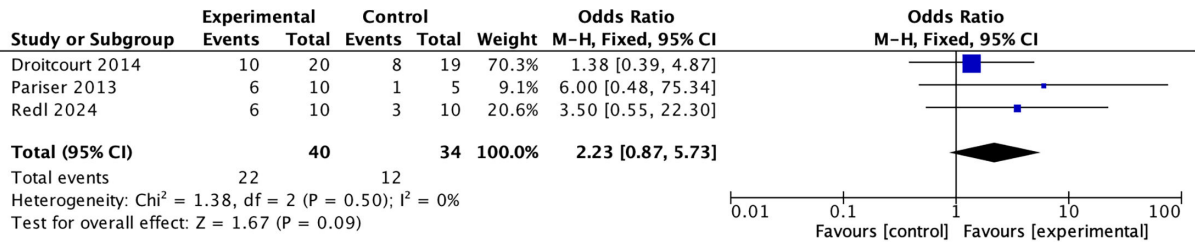


Fig. 4. Forest plot of the treatment effect on the secondary endpoint, quality of life in cutaneous sarcoidosis.

used across studies was heterogeneous. Consequently, subgroup analyses based on these variables were not feasible in the present review. This represents an important limitation, as differences in genetic ancestry, environmental exposures, healthcare access, disease phenotype and other social or biological factors may contribute to variation in treatment response. Future prospective trials in this rare dermatologic condition should therefore collect and report race, ethnicity or country of origin using predefined and, where possible, self-reported categories to enable assessment of therapeutic effects across relevant subpopulations. These limitations underline the need for larger, well-designed RCTs with standardized outcome measures, prespecified steroid tapering protocols and longer follow-up to assess the durability of response and relapse rates.

All included trials permitted concurrent glucocorticoid therapy, and most did not standardize dosing or tapering. Cox et al. note that maintaining patients on stable doses of steroids and other immunosuppressants may blunt the ability to detect the additive benefits of investigational therapies and negatively impact quality of life due to increased pill burden (19).

Emerging targeted therapies, such as JAK inhibitors and mTOR inhibitors, are supported by case reports and small open-label studies, but their safety profiles (infection, cytopenias, oedema) warrant caution (20–22).

A prospective series of 5 patients with pulmonary sarcoidosis treated with tofacitinib reported improvement in all 3 evaluated participants, each of whom was able to taper systemic glucocorticoids (22). These observations highlight both the therapeutic potential of JAK inhibition; however, we must be cautious not to extrapolate from small uncontrolled trials. Robust, placebo-controlled trials are needed before these off-label treatments can be recommended.

Recent case reports and small series suggest that targeted therapies may hold promise for refractory cutaneous sarcoidosis. A 65-year-old woman with longstanding cutaneous sarcoidosis achieved complete clearance of her lesions after 6 months of tofacitinib 5 mg twice daily, with improvement noted within 2 weeks (21). A 2024 case report described a patient with recalcitrant lupus pernio who showed near-complete resolution of lesions after 6 months of upadacitinib 15 mg daily; discontinuation led to relapse within 6 weeks, but restarting upadacitinib again cleared the disease and maintained remission for more than 18 months (23). These reports are consistent with a pooled review of 11 case series (21 patients total), where systemic JAK-inhibitor therapy achieved complete responses in approximately 83% of cutaneous cases and partial responses in the remainder (21). Additionally, a recent

review of sirolimus therapy summarised that systemic mTOR inhibition led to clinical and histological improvement in about 70% of patients with persistent cutaneous sarcoidosis (24). While these uncontrolled observations highlight therapeutic potential, their very small sample sizes and susceptibility to publication bias mean that targeted agents should not yet be considered standard care. Controlled trials are urgently needed to confirm efficacy and assess long-term safety.

Future trials should incorporate dose-tapering strategies to ensure comparable baseline therapy (25, 26). Head-to-head comparisons of TNF- α inhibitors and formal evaluation of targeted therapies (mTOR inhibitors, JAK inhibitors, IL-17/IL-23 blockers, anti-IL-6 agents) are essential to refine treatment algorithms (27, 28). Investigations should also assess long-term safety, given reports of infection, cytopenias, hyperlipidaemia and venous thromboembolism associated with JAK inhibitors and peripheral oedema, diarrhoea and bone-marrow toxicity with sirolimus (20). Finally, biomarker development (e.g. serum sIL-2R levels, PET-based granuloma activity, gene expression signatures) and precision medicine approaches may help identify subgroups most likely to benefit from specific therapies. Addressing these gaps will move the field toward evidence-based, patient-centred management of cutaneous sarcoidosis.

Conclusion

Evidence from randomized trials suggests that the addition of steroid-sparing agents may improve cutaneous lesions in sarcoidosis, but the certainty of evidence is low, and confidence intervals are wide. No intervention demonstrated a statistically significant increase in clinical response compared with placebo, but there was a trend for improved quality of life. Given the small sample sizes and methodological limitations of existing studies, definitive conclusions about efficacy cannot yet be drawn. Future research should prioritize multicentre, blinded RCTs with sufficient power, harmonized endpoints and robust assessment of patient-reported outcomes. Until such data are available, clinicians should individualize therapy based on disease severity, comorbidities and patient preferences.

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Data Availability: The data that support the findings of this study are available from the corresponding author upon reasonable request.

The authors have no conflicts of interest to declare.

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