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Impact of Intermittent Fasting, Keto Diet vs Continuous Caloric Restriction on Psoriasis Severity: A Systematic Review and Meta-Analysis Protocol

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ABSTRACT

Background: Psoriasis is a chronic immune-mediated inflammatory skin disease that is strongly associated with metabolic dysregulation and excess adiposity. Dietary strategies that reduce energy intake either by continuous daily caloric restriction (CCR) or time-limited/periodic intake such as intermittent fasting (IF) or ketogenic diets (Keto) may improve psoriasis severity through weight loss, metabolic improvements, alterations in gut microbiota, and immune-metabolic effects.

Objective: We aimed to compare the therapeutic effectiveness of IF, Keto, and CCR in reducing the severity of psoriasis.

Methods: This study was designed as a systematic review and Meta Analysis. The clinical parameters such as psoriasis extent and outcome measures were evaluated at baseline at different follow up timeframes across studies. Outcome measures were assessed by changes in Psoriasis Area and Severity Index (PASI) scores, quality of life (dermatology life quality index [DLQI]), and metabolic scores.

Results: There was a significant PASI decrease in Keto and CCR cohorts (10-25.6 kg weight loss) compared with IF (0-2kg weight loss). No serious adverse effects were recorded in all interventions.

Limitations: The major limitations were study heterogeneity, lack of head-to-head comparisons, and follow up.

Conclusion: CCR and Keto performed better than IF in managing psoriasis severity, suggesting personalized dietary routines as adjuncts to treatment.

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INTRODUCTION

Psoriasis is a chronic immune-mediated inflammatory skin disease that is strongly associated with metabolic dysregulation and excess adiposity.[1] Adipose tissue dysfunction contributes to systemic inflammation, promoting Th17/IL-17 pathways that drive psoriasis activity.[2] Dietary strategies that reduce energy intake either by continuous daily caloric restriction (CCR) or time-limited/periodic intake such as intermittent fasting (IF) may improve psoriasis severity through weight loss, metabolic improvements, alterations in gut microbiota and immune-metabolic effects (eg, changes in cytokine profiles, autophagy induction, circadian synchronization).[3]

A recent study demonstrated that a 12-week low-calorie diet in psoriatic patients with non-alcoholic fatty liver disease significantly decreased PASI scores, triglycerides, and liver enzymes, and enhanced quality of life.[4] Evidence-based reviews confirm that CCR alleviates symptoms by mitigating metabolic comorbidities, with better pharmacologic response in obese individuals.[5]

Intermittent fasting (IF), including alternate-day fasting, 5:2 diet, or time-restricted feeding, cycles feeding, and fasting periods, potentially offer superior metabolic benefits over CCR by enhancing insulin sensitivity and reducing inflammation.[6] A prospective study on acute time-restricted eating in psoriasis suggests reductions in inflammatory markers.[7] Clinical trials indicate IF attenuates psoriasis-like inflammation, with a phase IIb study reporting PASI reductions at week 28.[8]

The ketogenic diet (Keto), a low-carbohydrate, high-fat regimen promoting ketosis, targets inflammation by limiting glucose availability to immune cells and increasing anti-inflammatory ketones.[9] A metabolomic study found very-low-calorie Keto improved psoriasis symptoms through altered metabolic pathways.[10] A randomized crossover trial in obese PsA patients showed Keto reduced clinical and biochemical inflammation markers, including IL-1β and IL-2, comparable to Mediterranean diet effects.[11]

While individual studies support these diets, head-to-head comparisons of CCR, IF, and Keto on psoriasis severity are lacking, with limited data on effect modifiers such as baseline body mass index (BMI), regimen duration, or adherence. This gap hinders personalized recommendations, especially as dietary patterns influence disease activity.[12] This review synthesizes evidence to determine comparative efficacy, providing clinicians a framework for integrating these interventions in psoriasis management.

Study Objectives

a) To determine whether IF yields greater improvements in psoriasis severity (PASI) than CCR and Keto.
b) To compare IF, Keto, and CCR on patient-reported outcomes (DLQI), metabolic risk markers, and safety/adherence.
c) To explore effect modification by baseline BMI/obesity status, type of IF regimen, concurrent systemic therapy, and intervention duration.

Research Question

Among adults with moderate to severe psoriasis, how does intermittent fasting compare with continuous caloric restriction in reducing disease severity?

METHODS

We conducted this systematic review and meta-analysis to compare the effects of IF, CCR, and Keto on psoriasis severity. The review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA), with registration on PROSPERO.

The search strategy involved a comprehensive electronic search across MEDLINE (via PubMed), EMBASE, Cochrane CENTRAL, Web of Science, and Scopus. Trial registries, including ClinicalTrials.gov and WHO International Clinical Trials Registry Platform, provided access to unpublished or ongoing studies. Grey literature sources encompassed conference abstracts from the European Academy of Dermatology and Venereology (EADV), American Academy of Dermatology (AAD), and International Society of Dermatology (ISDS), and the National Psoriasis Foundation website. Search terms combined "psoriasis" or "psoriatic arthritis" with intervention-specific phrases: ("intermittent fasting" OR "time-restricted feeding" OR "alternate-day fasting" OR "5:2 diet" OR "Ramadan fasting") OR ("caloric restriction" OR "low-calorie diet" OR "energy-restricted diet") OR ("ketogenic diet" OR "very low-calorie ketogenic diet"). Boolean operators and filters for human studies and trial designs optimized retrieval, with no initial language restrictions applied. Inclusion criteria followed the PICO framework as illustrated below.

Two reviewers independently screened titles and abstracts using Covidence software, advancing eligible records to full-text review, with disagreements resolved via consensus or a third arbitrator. The PRISMA flow diagram illustrated in Figure 1 below documented the process, including reasons for exclusion. Data extraction used standardized excel forms to capture study characteristics, participant details, intervention protocols, and outcomes, with dual extraction minimizing errors. Risk of bias assessment was done using Cochrane Risk of Bias 2.0 (RoB 2) for randomized controlled trials (Figure 2) and ROBINS-I for non-randomized intervention studies and observational cohort studies (Figure 3). Studies with low risk were chosen for higher confidence in findings.

Data Extraction and Management

We used a standardized excel form for data extraction, capturing details such as study design, population, interventions, outcomes (including PASI score and quality of life), duration, and results. Two reviewers independently extracted data, resolving discrepancies through discussion.

Data Analysis

We performed a meta-analysis where studies exhibited sufficient homogeneity, adopting a random-effects model to calculate pooled effect estimates for PASI score reductions and quality of life improvements. A narrative synthesis summarized findings where meta-analysis was not feasible due to variability in study design or outcomes, ensuring all relevant data were represented.

Ethical Considerations

This study relied on publicly available data from published literature. Therefore, ethical approval was not required.

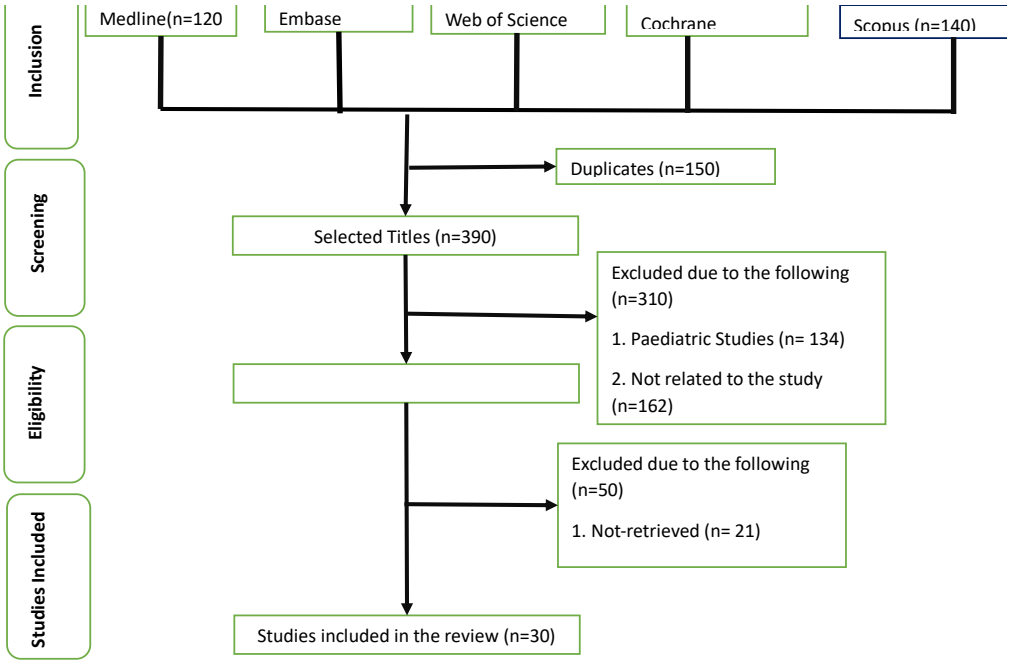
RESULTS

Screening Results and Study Characteristics

Approximately 540 publications were searched from MEDLINE via PubMed (n=120), EMBASE (n=150), Cochrane CENTRAL (n=30), Web of Science (n=100), and Scopus (n=140). After removing duplicates (n=150), 390 titles and abstracts were screened, which were further evaluated following the inclusion and exclusion criteria adopted in our study. After this step, 30 studies[12-42] were included from 80 eligible full-text articles, comprising 4 studies on IF (n=373 patients), 5 studies on Keto (n=74 patients), and 21 studies on CCR (n=1,627 patients).

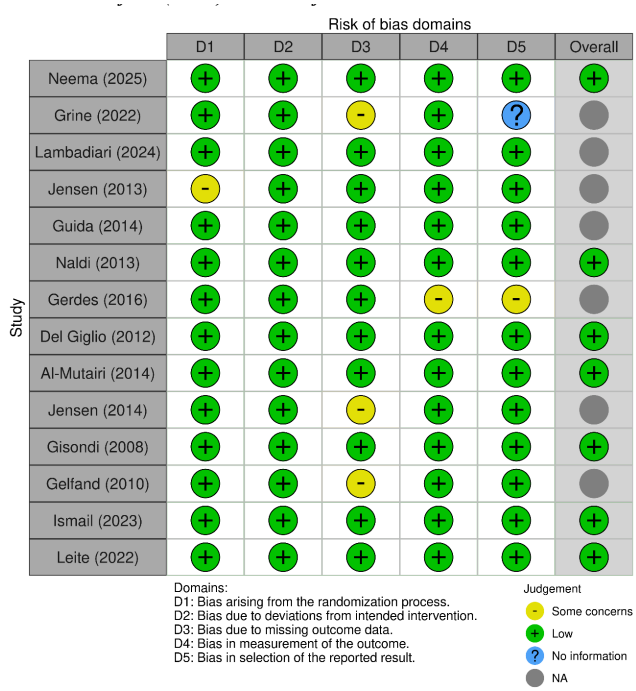
Out of the 30 studies selected, there was no study that directly compared IF with CCR interventions, and thus the necessity for a narrative synthesis. The studies were conducted between 2003 and 2025 and included randomized parallel group trials, single-arm open-label clinical trials, prospective observational studies, and randomized controlled crossover trials, among others. The total sample size across all studies was 2,073 participants, with a pooled mean age of 48.4 years (range 19-70). Gender distribution varied across the intervention and control groups,

FIGURE 1. PRISMA flow chart for selecting articles through databases and registers.



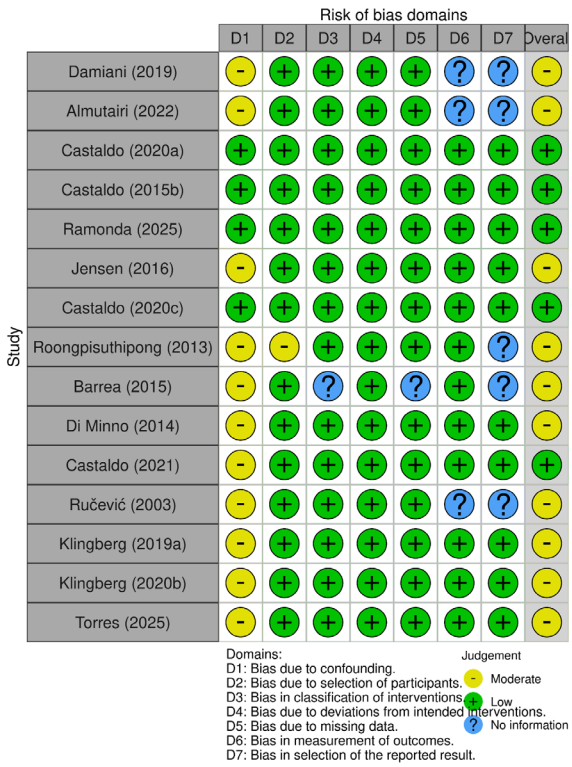
This figure illustrates the PRISMA flow chart used in identifying articles from databases and registers. A total of 540 articles were screened following the inclusion criteria leading to obtaining 30 articles which were used in this study.

FIGURE 2. Cochrane risk of bias (RoB 2) assessment for RCTs.



This figure shows that majority of the RCTs lacked adequate information concerning risk of bias with only 6 RCTs showing low risk of bias.

FIGURE 3. ROBINS-I for non-randomized and observational cohort studies.



This figure shows ROBINS-I for non-randomized and observational cohort studies for the included studies. Majority of the studies had a moderate risk of bias while the rest had low risk of bias, therefore suitable for inclusion in the current study.

averaging 50.1% male and 41.9% female; though some groups showed female predominance.

For the IF studies, baseline characteristics included a pooled mean age of 43.2 years (range 19-61) and 63% male (range 57.4-73.3%). The weighted mean baseline PASI across studies was 8.3 ± 5.0, with significant improvements observed post-intervention. At week 16,[12] PASI reduced to 5.0 ± 2.8, further adjusting to 5.7 ± 2.5 at week 28, while other studies reported a 1-month PASI of 3.1 ± 1.6, reflecting a -0.87 average reduction (P<0.0001 to P=0.001).[13] DLQI, reported only in one study,[12] decreased from 11.9 ± 4.3 at baseline to 4.9 ± 2.3 at week 16 and 5.1 ± 1.3 at week 28. Weight remained stable, with the IF group in some studies[13] showing a baseline of 67.4 kg and 67.1 kg at week 28 (change -0.3 kg), while others reported no significant BMI change (baseline 31.6 ± 5.2).[13] High sensitivity C-reactive protein (hsCRP) dropped from 4.4 ± 4.1 mg/L at baseline to 2.0 ± 2.3 at week 16 and 2.3 ± 2.0 at 28 weeks, pointing to a reduced inflammation.[12] Adverse effects were mild, with some studies in this group recording hunger, fatigue, and constipation (n=28/121), and no serious events reported across all studies.[12-15] Some limitations were noted in this IF group such as inconsistent follow-up durations

TABLE 1.

Inclusion and Exclusion Criteria

Category	Inclusion Criteria	Exclusion Criteria
Population	Human studies including adults (≥18 years) with clinician-diagnosed psoriasis (any subtype, preferably moderate-to-severe, e.g., PASI ≥10).	Pediatric studies (<18 years).
Study Design	Randomized controlled trials (including cluster and crossover), quasi-randomized trials, non-randomized controlled trials, and controlled prospective cohort studies; controlled before-after studies with a clear comparator.	Animal, in vitro, or mechanistic-only studies; case reports, case series, narrative reviews, editorials, or commentaries without original data; uncontrolled pre-post studies without a comparator.
Intervention	Clearly described IF regimens (e.g., timing, fasting windows, frequency, duration) or CCR regimens (daily calorie target or predefined kcal deficit); studies reported sufficient protocol detail to classify as IF or CCR.	Studies where major systemic psoriasis therapy initiated or substantially changed during the intervention without stratified reporting (unless sensitivity analyses isolated dietary effects); bariatric surgery or pharmacologic weight-loss as primary exposure (unless dietary regimen isolated from surgical/pharmacologic effects).
Comparator	CCR, usual care/no-diet control, or alternative dietary interventions allowing direct or indirect comparison with IF.	--
Outcomes	Reported at least one prespecified outcome (PASI, PASI-75, DLQI, weight/BMI, or biomarkers).	--
Duration	Minimum follow-up of ≥4 weeks from intervention start; longer follow-up preferred for sustained effect assessment.	--
Language	Studies published in English (translations sought for highly relevant non-English studies if resources allowed).	--

This table illustrates the inclusion and exclusion criteria adopted in selecting articles which were analysed for this study.

TABLE 2.

Follow up Results Across IF Studies

Parameter	Baseline	16 Weeks	28 Weeks	1 Month	Notes
PASI	8.3 ± 5.0	5.0 ± 2.8	5.7 ± 2.5	3.1 ± 1.6	Weighted mean; 1-month data from Ramadan studies (n=229); 16/28-week data from Neema (n=60)
DLQI	11.9 ± 4.3	4.9 ± 2.3	5.1 ± 1.3	Not reported	Only from Neema[12] (n=60)
Weight (kg)	67.4	Not reported	67.1	No significant change	Neema[13] baseline/28-week (n=60); Ramadan studies report stable BMI (n=229)
hsCRP (mg/L)	4.4 ± 4.1	2.0 ± 2.3	2.3 ± 2.0	Not reported	Only from Neema[12] (n=60)
IL-6 (pg/ml)	6.6 ± 5.6	3.6 ± 2.8	4.0 ± 3.1	Not reported	Only from Neema[12] (n=60)
PASI50 Achievement	--	78.3%	54.4%	Not reported	Neema[12] at 16/28 weeks (n=60); Ramadan studies lack PASI50 data
Adverse Effects	None	None	None	Mild (hunger, fatigue, constipation)	Reported in Ramadan studies[13-14] (n=229); none serious across all

This table shows the follow-up data across the included studies. The participants were followed up after 16 weeks, 28 weeks and 1 month. In most of the studies, there were no reported adverse outcomes.

TABLE 3.

Reported KD pooled Outcomes at Baseline and On Follow-Up Across the Studies			
Parameter	**Baseline (Pooled)**	**Post-Follow-Up (Pooled)**	**Notes**
PASI	15.2 (range 0-37)	~5.3 (64% reduction)	Weighted mean; greatest reductions in severe cases,[16,17,19,21] minimal in mild PsA21
DLQI	13.3 ± 4.0	0.8 ± 1.3	From Studies,[16-19] significant QoL improvement
Weight (kg)	~76.5	~66.3 (~10.9% loss)	Consistent across all[16-21] BMI reductions paralleled weight loss
CRP/IL-6	Elevated (e.g., IL-6 baseline in Study 6)	Reduced (e.g., IL-6 -55.6% in KD)	From Studies,[16,18,20,21] reduced inflammation overall
Other Biomarkers	High cytokines, insulin resistance	Improved lipids, glucose, HOMA-IR	Metabolic normalization in Studies[16,17,19,21,22]
PASI-75 Achievement	--	~65%	From Studies,[16, 17, 19] not reported in others
Adverse Effects	--	Mild (8-27% incidence)	No serious AEs; high compliance

This table shows the reported pooled outcomes on baseline and after follow-up. There were significant PASI changes, weight reduction across studies and no serious adverse events.

TABLE 4.

CCR group Outcomes at Baseline and Post-Follow Up			
Parameter	**Baseline (Pooled)**	**Post-Follow-Up (Pooled)**	**Notes**
PASI	~12.5 (range 5-19.8)	~6.3 (50% reduction)	Weighted mean; varies by study design (e.g., 77% with VLCKD, 84% with biologics)
DLQI	~13.5 (range 5.1-19.2)	~5.5 (2-13 points improvement)	Significant QoL gains in most studies
Weight (kg)	~80.0 (variable)	(8.5 kg loss)	Greater in LED/VLED (10-18.7 kg); BMI reduced 3-6 units
CRP (mg/L)	~4.0 (variable)	~2.0 (reduced)	Notable drops in studies reporting
Other Biomarkers	Elevated glucose, lipids, insulin resistance	Improved (glucose, lipids, HOMA-IR)	Enhanced metabolic and PsA activity (MDA 39-54%)
PASI-75 Achievement	--	29-86% (intervention)	Higher with adjunct therapies vs. controls
Adverse Effects	--	Mild (hunger, fatigue)	Low dropouts (0-11%); no serious AEs

The table shows the group outcomes at baseline and after follow up. There was a significant reduction in biomarkers across studies with only mild hunger and fatigue reported in few studies and no adverse effects.

(1 month vs 28 weeks) and missing biomarker data in some studies, affecting comprehensive pooling.

In the Keto group, the pooled mean age was 46.9 years across the studies. The weighted baseline PASI across studies was approximately 15.2 (ranging from 0 in mild PsA cases to 37 in severe psoriasis). Post-intervention, pooled PASI reductions averaged 64% while DLQI improved significantly, where reported from 15.9 to 0 (-13.4 points). Weight loss was consistent, averaging 10.2 kg while BMI decreased with an average of 4.2. Inflammatory biomarkers improved: reduced CRP, IL-6, IL-17, TNF-α (IL-6 -55.6% in Keto vs -20.56% Mediterranean); reduced IL-2/IL-1β. Metabolic parameters enhanced recorded improved outcomes, including glucose, lipids, insulin resistance, and cardiovascular risk. Adverse effects were mild, with high compliance and low dropouts. Table 3 below summarizes these Keto outcomes.

For the CCR cohort, the weighted baseline PASI was 12.5 (range mild to severe). Post-intervention, pooled PASI reductions averaged 50% (eg, 77% in very-low-calorie ketogenic diet (VLCKD) hybrids, 84% with biologics, 30-48% in others), with PASI-75 in 29-86% of intervention groups vs controls. DLQI improved by 2-13 points across studies, enhancing QoL. Weight loss averaged 8.5 kg or 5-16% (greater in LED/VLED: 10-18.7 kg), reducing BMI by 3 to 6 units. Biomarkers showed CRP drops (4 to 2 mg/L), improved lipids, glucose, insulin resistance and PsA activity (MDA 39-54%). There were mild adverse effects recorded across studies such as hunger, headaches, and fatigue.

Across all groups, weight loss correlated with PASI improvement, with Keto and CCR showing greater reductions (up to 25.6 kg) than IF (stable to 2 kg). No serious adverse effects were reported across interventions, with mild issues (eg, hunger, fatigue) noted in 18% to 27% of participants where documented.

DISCUSSION

Dietary interventions represent a promising adjunct in psoriasis therapy, targeting the interplay between metabolic syndrome and immune dysregulation.[43] The observed superiority of Keto and CCR over IF in reducing disease severity underscores the role of substantial weight loss and ketosis in modulating inflammatory pathways, such asTh17/IL-17 signaling, which drive psoriatic pathogenesis.[44]This aligns with the immunometabolic hypothesis, where adipose-derived cytokines exacerbate skin inflammation, and caloric deficits mitigate this via reduced adipokine secretion.[45]

The modest efficacy of IF, often limited to adjunctive settings, can be explained from its reliance on time-restricted feeding without enforced caloric deficits, leading to minimal metabolic shifts compared with Keto or CCR. For instance, while IF enhances autophagy and circadian alignment, potentially dampening IL-6 and hsCRP, its inconsistent impact on weight suggests insufficient disruption of the obesity-psoriasis axis. This is corroborated by recent evidence indicating that IF's benefits in autoimmune conditions are dose-dependent on fasting duration, with shorter protocols yielding suboptimal anti-inflammatory effects.[46] In contrast, Keto's induction of beta-hydroxybutyrate suppresses NLRP3 inflammasome activation, offering a mechanistic edge in psoriasis, where inflammasome hyperactivity perpetuates keratinocyte proliferation.[47]

CCR's improved outcomes can be linked to sustained energy deficits, highlighting its accessibility for obese patients as it facilitates endothelial function improvements and cardiovascular risk reduction.[48] However, adherence challenges in real-world settings may limit long-term efficacy, as evidenced by partial weight regain in extended follow-ups.

Limitations

The lack of direct comparisons in our review emphasizes the need for randomized trials integrating biomarkers like gut microbiota profiles, which mediate diet-skin interactions.[49] Emerging data suggest that personalized interventions such as factoring baseline BMI and PsA presence can optimize responses, with Keto being suitable in metabolic syndrome-dominant cases.[50]

Safety profiles across interventions were favorable, with only moderate gastrointestinal effects reported, thus supporting the applicability of integrating these outcomes into guidelines. Nonetheless, nutritional deficiencies in restrictive regimens warrant monitoring, particularly in vulnerable populations. Future research should advance head-to-head comparisons to delineate distinct effects, potentially revolutionizing non-pharmacologic psoriasis management amid rising biologic concerns.[51]

CONCLUSION

This study demonstrated that Keto and CCR significantly reduced psoriasis severity, primarily through substantial weight loss and metabolic improvements, while IF offered modest benefits, particularly as an adjunct therapy. No significant side effects were observed in all the interventions, indicating their safety in psoriasis management. The results validate the use of personalized nutritional strategies in treatment protocols for the management of psoriasis patients.

DISCLOSURES

The authors have no conflicts of interest to declare.

REFERENCES

1. Chan JJ. Psoriasis: an update on topical and systemic therapies. *Aust Prescr.* 2025;48:87-92. doi: 10.18773/austprescr.2025.026
2. Egeberg A, Gisondi P, et al. The role of the interleukin-23/Th17 pathway in cardiometabolic comorbidity associated with psoriasis. *J Eur Acad Dermatol Venereol.* 2020;34(8):1695-1706.
3. Simancas-Racines D, Román-Galeano NM, Verde L, et al. Targeting cytokine. https://doi.org/10.1111/jdv.16273. Accessed November 21, 2025.
4. Ismail AMA, Saad AE, Draz RS. Effect of low-calorie diet on psoriasis severity index, triglycerides, liver enzymes, and quality of life in psoriatic patients with non-alcoholic fatty liver disease. *Reumatologia.* 2023;61(2):116.
5. Lambadiari V, Katsimbri P, Kountouri A, et al. The effect of a ketogenic diet versus Mediterranean diet on clinical and biochemical markers of inflammation in patients with obesity and psoriatic arthritis: a randomized crossover trial. *Int J Mol Sci.* 2024;25:2475.
6. Dysregulation in psoriasis: the role of dietary interventions in modulating the immune response. *Int J Mol Sci.* 2025;26:2895.
7. Romero Arocha S, Han K, Huffstutler R, et al. Effects of acute time-restricted eating on inflammation in individuals with psoriasis: protocol for a case-control, prospective study. *JMIR Res Protoc.* 2025;14:e74999. doi: 10.2196/74999
8. Romero Arocha S, Han K, Huffstutler RD, et al. Effects of acute time-restricted eating on inflammation in individuals with psoriasis: protocol for a case-control, prospective study. *JMIR Res Protoc.* 2025;14:e74999.
9. Malinowska D, Żendzian-Piotrowska M. Ketogenic diet: a review of composition diversity, mechanism of action and clinical application. *J Nutr Metab.* 2024;2024:6666171. doi:10.1155/2024/6666171
10. Simancas-Racines D, Román-Galeano NM, Verde L, et al. Targeting cytokine dysregulation in psoriasis: the role of dietary interventions in modulating the immune response. *Int J Mol Sci.* 2025;26:2895.
11. Zukierski K, Jarych W, Michalak K, et al. The role of diet in psoriasis management: a focus on Mediterranean, ketogenic, and plant-based dietary approaches: review. *Qual Sport.* 2025;42:61162.
12. Neema S, Vausdevan B, Misra P, et al. Efficacy of intermittent fasting in the management of chronic plaque psoriasis: a phase IIb clinical trial. *Indian Dermatol Online J.* 2025;16:389-396. doi: 10.4103/idoj.idoj_635_24
13. Damiani G, Watad A, Bridgewood C, et al. The impact of Ramadan fasting on the reduction of PASI score in moderate-to-severe psoriatic patients: a real-life multicenter study. *Nutrients.* 2019;11(2):277. doi: 10.3390/nu11020277. PMID: 30691245
14. Almutairi N, Shaaban D. Clinical implications of intermittent Ramadan fasting on stable plaque psoriasis: a prospective observational study. *Postepy Dermatol Alergol.* 2022;39(2):368-374. doi: 10.5114/ada.2021.107098. PMID: 35645682
15. Grine L, Hilhorst N, Michels N, et al. The effects of modified intermittent fasting in psoriasis (MANGO): protocol for a two-arm pilot randomized controlled open cross-over study. *JMIR Res Protoc.* 2022;11(2):e26405. doi: 10.2196/26405. PMID: 35195533

16. Castaldo G, Rastrelli L, Galdo G, et al. Aggressive weight-loss program with a ketogenic induction phase for the treatment of chronic plaque psoriasis: a proof-of-concept, single-arm, open-label clinical trial. *Nutrition.* 2020;74:110757. doi: 10.1016/j.nut.2020.110757. PMID: 32222582.
17. Castaldo G, Galdo G, Rotondi Aufiero F, et al. Very low-calorie ketogenic diet may allow restoring response to systemic therapy in relapsing plaque psoriasis. *Obesity Res Clin Pract.* 2016;10(3):348-352.
18. Castaldo G, Pagano I, Grimaldi M, et al. Effect of very-low-calorie ketogenic diet on psoriasis patients: a nuclear magnetic resonance-based metabolomic study. *J Proteome Res.* 2020;20(3):1509-1521.
19. Barrea L, Megna M, Cacciapuoti S, et al. Very low-calorie ketogenic diet (VLCKD) in patients with psoriasis and obesity: an update for dermatologists and nutritionists. *Crit Rev Food Sci Nutr.* 2022;62(2):398-414. doi: 10.1080/10408398.2020.1818053. PMID: 32969257.
20. Ramonda R, Ometto F, Striani G, et al. Ketogenic diet improves disease activity and cardiovascular risk in psoriatic arthritis: a proof of concept study. *PLoS ONE.* 2025;20(4):e0321140.
21. Lambadiari V, Katsimbri P, Kountouri A, et al. The effect of a ketogenic diet versus Mediterranean diet on clinical and biochemical markers of inflammation in patients with obesity and psoriatic arthritis: a randomized crossover trial. *Int J Mol Sci.* 2024;25(5):2475. doi: 10.3390/ijms25052475. PMID: 38473723
22. Jensen P, Robin C, Claus Z, et al. Long-term effects of weight reduction on the severity of psoriasis in a cohort derived from a randomized trial: a prospective observational follow-up study. *Am J Clin Nutr.* 2016;104(2):259-265.
23. Jensen P, Zachariae C, Christensen R, et al. Effect of weight loss on the severity of psoriasis: a randomized clinical study. *JAMA Dermatol.* 2013;149(7):795-801. doi: 10.1001/jamadermatol.2013.722. PMID: 23752669.
24. Guida B, Napoleone A, Trio R, et al. Energy-restricted, n-3 polyunsaturated fatty acids-rich diet improves the clinical response to immuno-modulating drugs in obese patients with plaque-type psoriasis: a randomized control clinical trial. *Clin Nutr.* 2014;33(3):399-405. doi: 10.1016/j.clnu.2013.09.010. PMID: 24120032.
25. Castaldo G, Rastrelli L, Galdo G, et al. Aggressive weight-loss program with a ketogenic induction phase for the treatment of chronic plaque psoriasis: a proof-of-concept, single-arm, open-label clinical trial. *Nutrition.* 2020;74:110757.
26. Campanati A, Molinelli E, Ganzetti G, et al. The effect of low-carbohydrates calorie-restricted diet on visceral adipose tissue and metabolic status in psoriasis patients receiving TNF-alpha inhibitors: results of an open label controlled, prospective, clinical study. *J Dermatolog Treat.* 2017;28(3):206-212. doi: 10.1080/09546634.2016.1214666. PMID: 27552000.
27. Naldi L, Conti A, Cazzaniga S, et al. Diet and physical exercise in psoriasis: a randomized controlled trial. *Br J Dermatol.* 2014;170(3):634-642. doi: 10.1111/bjd.12735. PMID: 24641585.
28. Gerdes S, Dethlefs B, Personke Y, et al. Online weight-loss coaching for patients with psoriasis: results of a pilot study. *Br J Dermatol.* 2016;174(3):674-676. doi: 10.1111/bjd.14187. PMID: 26399576.
29. Roongpisuthipong W, Pongpudpunth M, Roongpisuthipong C, et al. The effect of weight loss in obese patients with chronic stable plaque-type psoriasis. *Dermatol Res Pract.* 2013;2013:795932. doi: 10.1155/2013/795932. PMID: 24159327
30. Del Giglio M, Gisondi P, Tessari G, et al. Weight reduction alone may not be sufficient to maintain disease remission in obese patients with psoriasis: a randomized, investigator-blinded study. *Dermatology.* 2012;224(1):31-37. doi: 10.1159/000335566. PMID: 22456343.
31. Al-Mutairi N, Nour T. The effect of weight reduction on treatment outcomes in obese patients with psoriasis on biologic therapy: a randomized controlled prospective trial. *Expert Opin Biol Ther.* 2014;14(6):749-756. doi: 10.1517/14712598.2014.900541. PMID: 24661040.
32. Jensen P, Zachariae C, Christensen R, et al. Effect of weight loss on the cardiovascular risk profile of obese patients with psoriasis. *Acta Derm Venereol.* 2014;94(6):691-694. doi: 10.2340/00015555-1824. PMID: 24556829.
33. Gisondi P, Del Giglio M, Di Francesco V, et al. Weight loss improves the response of obese patients with moderate-to-severe chronic plaque psoriasis to low-dose cyclosporine therapy: a randomized, controlled, investigator-blinded clinical trial. *Am J Clin Nutri.* 2008;88(5):1242-1247. https://doi.org/10.3945/ajcn.2008.26427
34. Barrea L, Balato N, Di Somma C, et al. Nutrition and psoriasis: is there any association between the severity of the disease and adherence to the Mediterranean diet? *J Transl Med.* 2015;13:18. doi: 10.1186/s12967-014-0372-1. PMID: 25622660
35. di Minno MN, Peluso R, Iervolino S, et al. Obesity and the prediction of minimal disease activity: a prospective study in psoriatic arthritis. *Arthritis Care Res (Hoboken).* 2013;65(1):141-147. doi: 10.1002/acr.21711. PMID: 22514189.
36. Gelfand JM, Abuabara K. Diet and weight loss as a treatment for psoriasis. *Arch Dermatol.* 2010;146(5):544-546. doi: 10.1001/archdermatol.2010.92. PMID: 20479304
37. Ramonda R, Ometto F, Striani G, et al. Ketogenic diet improves disease activity and cardiovascular risk in psoriatic arthritis: a proof of concept study. *PLoS One.* 2025;20(4):e0321140. doi:10.1371/journal.pone.0321140
38. Ismail AMA, Hamed DE. Erectile dysfunction and metabolic syndrome components in obese men with psoriasis: response to a 12-week randomized controlled lifestyle modification program (exercise with diet restriction). *Ir J Med Sci.* 2024;193(1):523-529. doi: 10.1007/s11845-023-03412-8. PMID: 37258850
39. Rucevic I, Stefanic M, Tokic S, et al. Lack of association of vitamin D receptor gene 3'-haplotypes with psoriasis in Croatian patients. *J Dermatol.* 2012;39(1):58-62. doi: 10.1111/j.1346-8138.2011.01296.x. PMID: 21951018.
40. Klingberg E, Bilberg A, Björkman S, et al. Weight loss improves disease activity in patients with psoriatic arthritis and obesity: an interventional study. *Arthritis Res Ther.* 2019;21(1):17. doi: 10.1186/s13075-019-1810-5. PMID: 30635024
41. Klingberg E, Björkman S, Eliasson B, et al. Weight loss is associated with sustained improvement of disease activity and cardiovascular risk factors in patients with psoriatic arthritis and obesity: a prospective intervention study with two years of follow-up. *Arthritis Res Ther.* 2020;22(1):254. doi:10.1186/s13075-020-02350-5
42. Torres L, Jonsson CA, Eliasson B, et al. A six-month weight loss intervention is associated with significant changes in serum biomarkers related to inflammation, bone and cartilage metabolism in obese patients with psoriatic arthritis and matched controls. *BMC Rheumatol.* 2025;9(1):58. doi:10.1186/s41927-025-00511-0
43. Leite BF, Morimoto MA, Gomes CMF, et al. Dietetic intervention in psoriatic arthritis: the DIETA trial. *Adv Rheumatol.* 2022;62(1):12. doi: 10.1186/s42358-022-00243-6. PMID: 35387686.
44. Okamura Y, Miyanishi H, Kinoshita M, et al. A defective interleukin-17 receptor A1 causes weight loss and intestinal metabolism-related gene downregulation in Japanese medaka, Oryzias latipes. *Sci Rep.* 2021;11(1):12099. doi:10.1038/s41598-021-91534-3
45. Chehimi M, Vidal H, Eljaafari A. Pathogenic role of IL-17-producing immune cells in obesity, and related inflammatory diseases. *J Clin Med.* 2017;6(7):68. doi:10.3390/jcm6070068
46. DiSpirito JR, Mathis D. Immunological contributions to adipose tissue homeostasis. *Semin Immunol.* 2015;27(5):315-321. doi:10.1016/j.smim.2015.10.005
47. Hansen B, Sánchez-Castro M, Schintgen L, et al. The impact of fasting and caloric restriction on rheumatoid arthritis in humans: a narrative review. *Clin Nutr.* 2025;49:178-186. doi:10.1016/j.clnu.2025.04.025
48. Jiang Z, Yin X, Wang M, et al. Effects of ketogenic diet on neuroinflammation in neurodegenerative diseases. *Aging Dis.* 2022;13(4):1146-1165. doi:10.14336/AD.2021.1217
49. Hays A, Soleimanifard S, Schär M, et al. Coronary endothelial function is directly related to extent of weight loss in obese patients. *J Cardiovasc Magn Reson.* 2013;15(Suppl 1):O44. doi:10.1186/1532-429X-15-S1-O44
50. Chai J, Deng F, Li Y, et al. Editorial: the gut-skin axis: interaction of gut microbiome and skin diseases. *Front Microbiol.* 2024;15:1427770. doi:10.3389/fmicb.2024.1427770
51. Okube OT, Kimani S, Mirie W. Community-based lifestyle intervention improves metabolic syndrome and related markers among Kenyan adults. *J Diabetes Metab Disord.* 2022;21(1):607-621. doi:10.1007/s40200-022-01023-1

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