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JAMA Dermatology | Review

Dietary Recommendations for Adults With Psoriasis or Psoriatic Arthritis From the Medical Board of the National Psoriasis Foundation
A Systematic Review

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IMPORTANCE Psoriasis is a chronic, inflammatory skin disease and has significant associated morbidity and effect on quality of life. It is important to determine whether dietary interventions help reduce disease severity in patients with psoriatic diseases.

OBJECTIVE To make evidence-based dietary recommendations for adults with psoriasis and/or psoriatic arthritis from the Medical Board of the National Psoriasis Foundation.

EVIDENCE REVIEW We used literature from prior systematic reviews as well as additional primary literature from the MEDLINE database from January 1, 2014, to August 31, 2017, that evaluated the impact of diet on psoriasis. We included observational and interventional studies of patients with psoriasis or psoriatic arthritis. The quality of included studies was assessed using the Newcastle-Ottawa scale for observational studies and the Cochrane Risk of Bias Tool for interventional studies. We made evidence-based dietary recommendations, which were voted on by the National Psoriasis Foundation Medical Board.

FINDINGS We identified 55 studies meeting the inclusion criteria for this review. These studies represent 77 557 unique participants of which 4534 have psoriasis. Based on the literature, we strongly recommend dietary weight reduction with a hypocaloric diet in overweight and obese patients with psoriasis. We weakly recommend a gluten-free diet only in patients who test positive for serologic markers of gluten sensitivity. Based on low-quality data, select foods, nutrients, and dietary patterns may affect psoriasis. For patients with psoriatic arthritis, we weakly recommend vitamin D supplementation and dietary weight reduction with a hypocaloric diet in overweight and obese patients. Dietary interventions should always be used in conjunction with standard medical therapies for psoriasis and psoriatic arthritis.

CONCLUSIONS AND RELEVANCE Adults with psoriasis and/or psoriatic arthritis can supplement their standard medical therapies with dietary interventions to reduce disease severity. These dietary recommendations from the National Psoriasis Foundation Medical Board will help guide clinicians regarding the utility of dietary interventions in adults with psoriatic diseases.

Supplemental content

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Psoriasis is a chronic, inflammatory skin disease that affects 3.2% of US adults[1] and has a substantial effect on quality of life.[2,3] In addition to skin manifestations, psoriasis is associated with many comorbidities including psoriatic arthritis, cardiometabolic disease, gastrointestinal disease, and mood disorders.[2] Various medical therapies exist for psoriatic diseases including topical agents, phototherapy, oral medications, and biologics.[3] Prescription drugs and medical phototherapy have undergone rigorous efficacy and safety evaluation via clinical trials and long-term registries. Regardless of patients' perspectives on established medical therapies, nearly all patients seek to understand how diet affects their psoriatic disease.[4]

Specifically, most patients deem dietary interventions to be relevant to their overall psoriatic disease management; however, diet is rarely discussed during clinical visits.[4,5] Furthermore, studies show that patients with psoriasis are highly motivated to carry out dietary changes because these interventions are perceived to be natural, safe, and patient initiated.[5,6] However, most patients and clinicians lack knowledge on how well dietary interventions work because few articles exist that summarize literature on psoriasis and diet.[4,7-10]

Overall, there is a critical lack of evidence synthesis on the relationship between psoriatic diseases and diet. This literature gap speaks to the need for evidence-based dietary recommendations that are accessible to clinicians and patients. In this article, we present evidence synthesis from a systematic review that answers key questions on the role of dietary interventions in psoriatic diseases. Based on this systematic review, we present evidence-based recommendations from the National Psoriasis Foundation (NPF) Medical Board on the role of dietary interventions in psoriatic diseases.

Key Points

Question What dietary interventions help adult patients with psoriasis and/or psoriatic arthritis reduce their disease severity?

Findings In this systematic review of 55 studies and 4534 patients with psoriasis, we identify the strongest evidence for dietary weight reduction with a hypocaloric diet in overweight and obese patients with psoriasis. The utility of gluten-free diet and supplementation with vitamin D varies by subpopulations of adults with psoriatic diseases, and evidence is low for the utility of specific foods, nutrients, and dietary patterns in reducing psoriatic disease activity.

Meaning Adults with psoriasis and/or psoriatic arthritis can supplement their medical therapies with dietary interventions to reduce disease severity.

Methods

Systematic Review

To explore the current evidence relating to diet and psoriasis, we leveraged prior reviews that used Cochrane Central Register of Controlled Trials in the Cochrane Library, MEDLINE, and EMBASE[4,7-10] and performed additional systematic review synthesizing primary literature from 2014 through 2017 from MEDLINE. For evaluation of additional primary studies from January 1, 2014, to August 31, 2017, we used the following search criteria in MEDLINE: ("*Psoriasis*" [medical subject heading {MeSH}] AND "*Diet, Food, and Nutrition*" [MeSH]) OR "*Psoriasis/diet therapy*" [MeSH]. Through reading of primary literature, we identified additional studies not identified through the MEDLINE search (**Figure**).[5,11,12]

This review included observational and interventional studies (controlled and uncontrolled). The studies must have met the following inclusion criteria: study participants must have psoriasis and/or psoriatic arthritis; studies must use a dietary or nutritional intervention or examine food intake and/or dietary patterns; outcomes must include the development of psoriasis, psoriatic disease severity, or patient-reported effects of diet on disease severity. We excluded case reports, case series, review articles, non-English language studies, and studies exclusively evaluating alcohol as a dietary component.

Two authors (A.R.F. and A.W.A.) read reference abstracts to determine eligibility based on inclusion and exclusion criteria. Selected studies underwent full-text review. Two of us (A.R.F. and A.W.A.) independently extracted the data. We performed quality assessment using the Newcastle-Ottawa scale for observational studies and the Cochrane Risk of Bias Tool for interventional studies. The quality of uncontrolled interventional studies was not assessed.

Recommendation Development

Recommendations were developed using established guidance by Robinson et al[13] to determine the strength of recommendations and the level of supporting evidence. The findings from the systematic review and the proposed recommendations were reviewed and approved by majority vote by the NPF Medical Board.

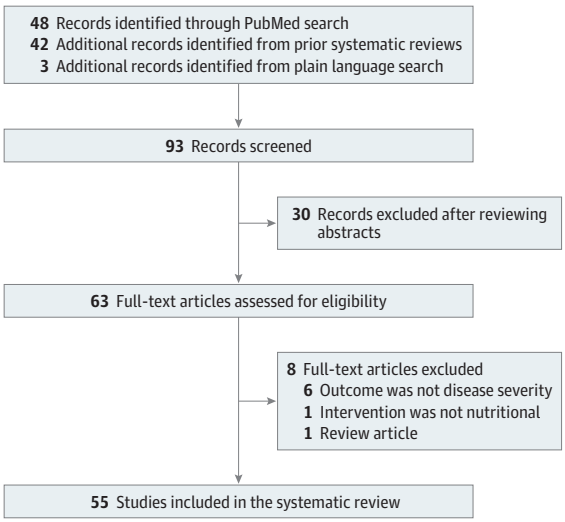
Briefly, level of evidence determines the strength of recommendation[13]:

- Level of evidence "A" refers to high-quality, patient-oriented evidence, which leads to a "strong recommendation" (strength of recommendation 1).
- Level of evidence "B" refers to limited-quality, patient-oriented evidence, which leads to a "weak recommendation" (strength of recommendation 2A).
- Level of evidence "C" refers to low-quality evidence, which leads to a "weak recommendation" (strength of recommendation 2B).

Results of the Systematic Review and NPF Recommendations

Fifty-five studies meet the inclusion criteria for this systematic review (Figure). These studies represent a total of 77 557 unique participants of which 4534 have psoriasis. Although a small number of pediatric patients are included in some studies, the data are insufficient to make recommendations for pediatric psoriasis. Study characteristics and outcome data are summarized in **Table 1**[5,27,29,51,60,61] and **Table 2**.[11,12,14-16,19-28,30,33-41,44-50,52,54-58,62,63,66-75] Quality assessment results for observational studies are included in Table 1; results for interventional studies are reported in the eTable in the Supplement. Dietary recommendations are summarized in **Table 3**.[13-17,19-23,45,60-63]

Figure. Diet and Psoriasis Literature Search and Screening Results



Question 1: Are Gluten-Free Diets Helpful in Psoriasis?

Psoriasis is associated with an increased risk of several autoimmune diseases; this includes a greater than 2-fold increased frequency of celiac disease.[7] Celiac disease is characterized by a small intestinal inflammatory response to dietary gluten. Serum antibodies indicating gluten sensitivity (IgG tissue transglutaminase, IgA endomysial antibody) are used to screen for celiac disease. Definitive diagnosis of celiac disease is made by demonstration of characteristic findings on duodenal biopsy. Of note, controversy exists regarding the existence of gluten sensitivity (characterized by positive serologic markers) in the absence of celiac disease.

Patients with psoriasis have an increased risk of positivity for serologic markers of gluten sensitivity, and higher antibody levels are associated with greater psoriasis severity.[7] Some patients with psoriasis have markers of gluten sensitivity with normal duodenal biopsy results.[7,14] Therefore, some patients with psoriasis seem to have gluten sensitivity in the absence of celiac disease.

Based on the available literature, a gluten-free diet may be helpful in select patients with psoriasis.[14-16] In patients with confirmed celiac disease, a gluten-free diet not only ameliorates gastrointestinal symptoms but also reduces psoriasis severity.[16] In patients who test positive for serologic markers of gluten sensitivity, a gluten-free diet leads to significant improvements in both clinical psoriasis severity and skin biopsy findings after 3 months.[14,15] Histologically, a decrease in the number of inflammatory and proliferating cells is seen in psoriatic plaques.[15] A majority of these patients experience worsening of disease after resuming a regular diet.[14] Additionally, among patients who test positive for serologic markers of gluten sensitivity, a gluten-free diet seems to be beneficial regardless of duodenal biopsy results.[14-16] In contrast, studies show that a gluten-free diet is not helpful in patients with psoriasis who test negative for serologic markers of gluten sensitivity.[14,15]

Recommendation 1: Gluten-Free Diet in Psoriasis

- In adults with psoriasis with confirmed celiac disease, we strongly recommend a gluten-free diet.[16,17]
  - This strong recommendation (strength 1) is based on level A evidence.
- Only in adults with psoriasis who test positive for serologic markers of gluten sensitivity, we recommend a 3-month trial of a gluten-free diet as an adjunctive intervention to standard medical therapies for psoriasis.
- We do not recommend universal screening for serologic markers of gluten sensitivity among adults with psoriatic diseases because of a high rate of false-positive results.[18] Based on recommendations from the American College of Gastroenterology, patients who are candidates for screening include those with a first-degree relative with celiac disease or those with active gastrointestinal symptoms.[17]
  - This weak recommendation (strength 2A) is based on level B evidence; limitations include a lack of randomization, small control groups, no reporting of between-group comparisons, and study-related bias.[14-16]

Question 2: What Is the Association of Dietary Weight Reduction With Psoriasis?

There is a well-established link between obesity and psoriasis.[2,9] Excess body weight is associated with increased psoriasis incidence, higher psoriasis severity, and reduced response to psoriasis treatments.[2,4,9] These associations may be mediated by the proinflammatory effects of body fat.[4]

Dietary weight reduction refers to weight loss as a result of dietary interventions and not from exercise or surgery. From the literature on dietary weight reduction, we learn that weight reduction with a hypocaloric diet—one that delivers less energy per day than an individual's expenditure—can be helpful in patients with psoriasis who are overweight or obese, defined as a body mass index (BMI, calculated as weight in kilograms divided by height in meters squared) of 25 or more. In these patients, a hypocaloric diet leads to significant improvements in psoriasis severity,[19-22] dermatology quality of life,[21,23] and weight loss,[19-23] compared with patients consuming a regular diet. The duration of beneficial dietary interventions ranges from 16 weeks to 6 months, and improvements in skin disease severity and dermatology quality of life can be maintained after 1 year.[24] The energy content of hypocaloric diets ranges from 800 to 1400 kcal per day.[19-23] Dietary weight reduction is beneficial in conjunction with standard psoriasis treatments including biologic therapy,[19,22,23] cyclosporine,[20-22] methotrexate,[21-23] acitretin,[22] phototherapy,[22] and various topical therapies.[22] Further support comes from 2 recent systematic reviews, 1 with a meta-analysis, that demonstrate significant benefit of weight loss with a hypocaloric diet in overweight and obese patients with psoriasis.[4,9] Uncontrolled data also support a hypocaloric diet in these patients for improvement in skin disease severity, dermatology quality of life, and weight loss.[24-26]

In 1 randomized clinical trial (RCT), after maintaining remission for 12 weeks during methotrexate therapy, patients discontinued methotrexate and were randomized to hypocaloric vs regular diet. The hypocaloric diet group had lower psoriasis severity scores compared with the regular diet group, but this was not statistically significant. Thus, diet alone may not be sufficient to maintain psoriasis remission.[27]

Limited data exist regarding other diets in patients with psoriasis. Ornish and South Beach diets helped patients with psoriasis achieve weight loss in an RCT, but their long-term benefit on psoriasis severity is uncertain.[28]

Table 1. Observational Studies on Dietary Patterns and Dietary Supplements in Psoriasis

Source; Country	Design/ No. of Participants	Participant Characteristics	Measures	Outcomes	Quality[a]
Vitamin D Supplementation					
Merola et al,[51] 2014; United States	Cohort/ 70 437 (502 psoriasis), 973 057 person-years of follow-up	No psoriasis at baseline (1994), female, age 47-74 y, enrolled in Nurses' Health Study	Food frequency questionnaire in 1994, 1998, 2002, 2006 to assess vitamin D intake	No significant association between vitamin D intake (dietary, supplementary, total) and incident psoriasis. RR of psoriasis in patients taking varying levels of vitamin D vs those taking 300-399 IU/d of vitamin D: 0.60 (95% CI, 0.24-1.50) for >1000 IU/d, 1.23 (95% CI, 0.80-1.87) for 800-999 IU/d, 1.05 (95% CI, 0.81-1.36) for 400-799 IU/d, 0.98 (95% CI, 0.71-1.35) for <200 IU/d (P > .99.	5
Supplement Use					
Yousefzadeh et al,[29] 2017; Iran	Case-control/ 138 Psoriasis, 138 controls	Patients with psoriasis: biopsy-confirmed plaque psoriasis, age 20-91 y. Controls: no dermatologic discomfort, age >20 y.	Questionnaire to assess supplement use over 30 d, PASI, DLQI	Patients with psoriasis vs controls: Compared with controls, higher proportion of patients with psoriasis used supplements (72.5% vs 25.4%; P = .01). Among patients with psoriasis: no significant difference in psoriasis severity or dermatology quality of life between patients with psoriasis using and not using supplements or between those using different supplements. Among patients with psoriasis, most common supplements were multivitamins (26.8%), folic acid (21.7%), herbs (12.3%), ω-3/fish oil (10.1%), vitamin E (1.4%).	4
Dietary Patterns					
Barrea et al,[60] 2015; Italy	Case-control/ 62 Psoriasis, 62 controls	Patients with psoriasis: mild-to-severe plaque psoriasis, duration >6 mo, adults, treatment-naive. Controls: healthy subjects matched for sex, age, BMI.	PREDIMED questionnaire measuring adherence to Mediterranean diet, PASI	Patients with psoriasis vs controls: Compared with controls, patients with psoriasis had lower adherence to the Mediterranean diet (P < .001). Compared with controls, a smaller proportion of patients with psoriasis consumed EVOO as main lipid, fruit ≥3 servings/d, fish or seafood ≥3/wk, tree nuts ≥3/wk. Among patients with psoriasis: Lower psoriasis severity was associated with consuming EVOO as main lipid, vegetables ≥2 servings/d, fruits ≥3 servings/d, legumes ≥3/wk, fish or seafood ≥3/wk, tree nuts ≥3/wk, sofrito sauce ≥2/wk (P < .009). Adherence to Mediterranean diet and consuming EVOO were predictors of lower psoriasis severity (P = .007 and P < .001, respectively).	8
Barrea et al,[61] 2015; Italy	Case-control/ 41 Psoriasis, 41 controls	Patients with psoriasis: plaque psoriasis, male, adults, white, treatment-naive. Controls: healthy subjects matched for age, BMI.	7-d food record to measure energy and nutrient intake, PASI	Patients with psoriasis vs controls: Compared with controls, patients with psoriasis had higher consumption of total and simple carbohydrates, total fat, PUFAs, ω-6:ω-3 PUFA ratio, cholesterol; lower consumption of protein, complex carbohydrates, MUFAs, ω-3 PUFAs, fiber (P < .034). Among patients with psoriasis: Higher MUFA consumption was a major predictor of lower psoriasis severity (P < .001). Lower psoriasis severity was associated with lower total energy, saturated fatty acids, total PUFAs, ω-6 PUFAs, ω-6:ω-3 PUFA ratio, simple carbohydrates and with higher ω-3 PUFAs, MUFAs, fiber, complex carbohydrates (P < .007).	8
Afifi et al,[5] 2017; United States	Case-control/ 1206 Psoriasis, 2847 controls	Patients with psoriasis: members of NPF listserv; self-reported severity, 20.9% mild, 42.2% moderate, 36.9% severe; 43.9% with psoriatic arthritis; mean age, 50.4 y; 87.2% white; 79% urban. Controls: NHANES 2009-2010 data set matched for sex, age.	NHANES 2009-2010 dietary screening questionnaire to assess nutrient intake; questionnaire to assess patient-reported skin responses to dietary changes and patients' attitudes regarding diet and psoriasis	Patients with psoriasis vs controls: Compared with controls, patients with psoriasis had lower daily intake of sugar, whole grain fiber, dairy products, calcium (P < .001); higher daily intake of fruits/vegetables/legumes (P = .007). Among patients with psoriasis (psoriasis patient perceptions): Triggers that worsen psoriasis: sugar (13.8%), alcohol (13.6%), tomato (7.4%), gluten (7.2%), dairy (6%). Items that may improve psoriasis: supplements (unspecified) (35.1%), vegetables (26.5%), fruits (21.8%), water (11.2%), fish (9.2%). Dietary reductions associated with patient-reported psoriasis improvement: alcohol (53.8%), gluten (53.4%), nightshades (52.1%), junk foods (50.4%), white flour products (49.9%). Dietary additions associated with patient-reported psoriasis improvement: fish oil/ω-3 (44.6%), vegetables (42.5%), oral vitamin D supplementation (41%), probiotics (40.6%), organic foods (38.4%), fruits (34.6%). Forty percent of patients with psoriasis had tried special diet for psoriasis; patients reported most psoriasis improvement with Pagano, vegan, Paleolithic diet.	4
Del Giglio et al,[27] 2012; Italy	Cross-sectional/ 200	Plaque psoriasis, PASI >10 or PASI >5 but uncontrolled by topical therapy, mean (SD) age, 53 (14) y	Questionnaire to assess opinions on possible dietary approach to psoriasis, BMI	Psoriasis patient perceptions: body weight can influence psoriasis severity (19%), foods can improve psoriasis (38%) (fruits and vegetables 60%, fish 10%), foods can worsen psoriasis (46%) (sausages 20%, dairy products 8%, tomatoes 8%, spicy food and chocolate 7%, fried food 5%), diet regimen can improve psoriasis (62%). Compared with normal weight patients with psoriasis, significantly more overweight/obese patients thought body weight can influence psoriasis severity (P < .003) and diet regimen can improve psoriasis (P < .04).	4

Abbreviations: BMI, body mass index; DLQI, Dermatology Life Quality Index; EVOO, extra virgin olive oil; MUFA, monounsaturated fatty acid; NHANES, National Health and Nutrition Examination Survey; NPF, National Psoriasis Foundation; PASI, Psoriasis Area and Severity Index; PREDIMED, Prevención con Dieta Mediterránea; PUFA, polyunsaturated fatty acid; RR, relative risk.

[a] Quality assessed using Newcastle-Ottawa scale for case-control and cohort studies (maximum score of 9), modified Newcastle-Ottawa scale for cross-sectional studies (maximum score of 8).

Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Gluten-Free Diet					
Michaëlsson et al,[14] 2000; Sweden[a]	Prospective, nonrandomized, controlled	Psoriasis, some with PsA, age 18-70 y, stable psoriasis treatment for at least 1 mo. Group 1: elevated IgA AGA and/or IgG AGA, n = 33. Group 2: normal IgA AGA and IgG AGA, n = 6.	3-mo gluten-free diet followed by 3 mo regular diet. Decrease treatment as tolerated.	Same intervention	Between-group comparisons were not reported. Mean (SD) psoriasis severity (PASI) improved in group 1 from 5.4 (4.5) to 3.6 (3.0) after gluten-free diet ($P < .001$); no improvement in group 2 from 8.9 (6.4) to 10.2 (7.9) ($P = .47$). In group 1, PASI improved in similar proportion of patients with psoriasis with normal and abnormal duodenal biopsy (75% and 67%, respectively). In group 1, 24% of patients decreased their psoriasis treatment during gluten-free diet and 0% increased treatment; in group 2, 0% decreased and 33% increased treatment. After resuming regular diet, 60% of group 1 needed to increase psoriasis treatment due to worsening disease vs 0% of group 2
Michaëlsson et al,[15] 2003; Sweden[a]	Prospective, nonrandomized, controlled	Psoriasis, some with PsA, age 18-70 y, stable psoriasis treatment for at least 1 mo. Group 1: elevated IgA AGA and/or IgG AGA, n = 31. Group 2: normal IgA AGA and IgG AGA, n = 6.	3-mo gluten-free diet. Decrease treatment as tolerated. 3-mm punch biopsy before and after diet.	Same intervention	Between-group comparisons were not reported. Group 1: significant reduction in No. of CD4+ T lymphocytes in involved epidermis ($P = .03$), No. of Ki67+ (proliferating) cells in involved ($P = .008$) and noninvolved ($P = .04$) dermis, % tTG+ area in involved dermis ($P = .008$). Group 1: no significant change in No. of CD4+ T lymphocytes in involved dermis and noninvolved epidermis and dermis, % CD1+ (Langerhans cells) area in involved and noninvolved epidermis and papillary dermis, % CD31/Q-Bend+ (endothelial cells) area in involved and noninvolved dermis, % Ki67+ (proliferating cells) area in involved and noninvolved epidermis, % tTG+ area in non involved dermis. Group 2: no significant changes observed after diet
De Bastiani et al,[16] 2015; Italy	Prospective, uncontrolled	Plaque or guttate psoriasis, some with PsA, age 18-80 y, celiac disease confirmed by histologic analysis, some receiving systemic therapy. N = 9.	6-mo gluten-free diet	None	Uncontrolled data: Gluten-free diet led to significant improvement in psoriasis severity (PASI 50 or PASI 75) in all patients after 3 mo; the level of improvement was maintained in 89% of patients at 6 mo
Dietary Weight Reduction					
Naldi et al,[22] 2014; Italy	Prospective, assessor-blind, randomized, controlled	Plaque psoriasis, PASI >10, age 18-80 y, BMI >25, not achieving clearance with systemic therapy after 4 wk (continued during study). Intervention: n = 151; control: n = 152.	20-wk hypocaloric diet. Encouraged aerobic exercise >40 min 3 times/wk.	Information session on utility of weight reduction for psoriasis control	Compared with controls, hypocaloric diet group had significantly greater reduction in psoriasis severity and body weight. Specifically, median PASI reduction, 48.0% (95% CI, 33.3%-58.3%) hypocaloric diet group vs 25.5% (95% CI, 18.2%-33.3%) controls ($P = .02$). PASI 50, 49.7% (95% CI, 41.7%-57.6%) of hypocaloric diet group vs 34.2% (95% CI, 26.7%-41.7%) of controls ($P = .006$). Median BMI reduction, 3.0% (95% CI, 2.4%-3.9%) hypocaloric diet group vs 1.9% (95% CI, 1.4%-2.3%) controls ($P = .002$)
Di Minno et al,[62] 2014; Italy	Prospective, assessor-blind, randomized, controlled	PsA, age >18 y, BMI >25, failed traditional DMARDs, starting tumor necrosis factor blocker therapy. Intervention: n = 63; control: n = 63.	6-mo hypocaloric diet	Self-managed diet with instruction to eat healthily	Compared with controls, hypocaloric diet group had significantly greater achievement of improved arthritis disease severity and significantly greater weight loss. Specifically, achievement of MDA, 43% of hypocaloric diet group vs 35% of controls (HR, 1.85; 95% CI, 1.02-3.35; $P = .04$). Weight loss >5% more likely in hypocaloric diet group vs controls (HR, 3.23; 95% CI, 1.93-5.39; $P < .001$). Achievement of MDA more likely with greater weight loss regardless of study group
Gisondi et al,[20] 2008; Italy	Prospective, investigator-blind, randomized, controlled	Active plaque psoriasis, PASI ≥10, BSA ≥10%, age ≥18 y, BMI 30-45. Intervention: n = 30; control: n = 31	24-wk hypocaloric diet. Low-dose cyclosporine (2.5 mg/kg daily). Encouraged moderate physical exercise ≥40 min ≥4 times weekly.	No dietary intervention. Same cyclosporine. Same exercise encouragement	Compared with controls, hypocaloric diet group had significantly greater improvement in psoriasis severity and significantly greater weight loss. Specifically, PASI 75, 67% of hypocaloric diet group vs 29% of controls ($P < .001$). PASI 50, 87% of hypocaloric diet group vs 48% of controls ($P < .001$). Mean (SD) PASI at week 24, 2.5 (6.3) hypocaloric diet group vs 8.1 (5.4) controls ($P < .001$). Mean (SD) BSA at week 24, 2.9% (4.4%) hypocaloric diet group vs 7.5% (4.9%) controls ($P < .001$). Mean (SD) weight loss, 7.0 (3.5) kg hypocaloric diet group vs 0.2 (0.9) kg controls ($P < .001$)
Guida et al,[21] 2014; Italy	Prospective, assessor-blind, randomized, controlled	Plaque psoriasis, age ≥18 y, BMI >30, receiving stable systemic treatment for at least 5 mo (continued during study). Intervention: n = 22; control: n = 22.	6-mo hypocaloric, ω-3-enriched diet	No dietary modification	Compared with controls, hypocaloric diet group had lower psoriasis severity (PASI; $P < .05$), better dermatology quality of life (DLQI; $P < .05$), and lower body weight ($P < .05$) after 6 mo. No significant difference between hypocaloric diet and control groups in itch severity (VAS itch score) after 6 mo

(continued)

Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA) (continued)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Del Giglio et al,[27] 2012; Italy	Prospective, investigator-blind, randomized, controlled	Plaque psoriasis, PASI >10 or >5 and uncontrolled by topical therapy, age ≥18 y, BMI ≥30, receiving methotrexate and obtained PASI 75 for 12 wk. Intervention: n = 22; control: n = 20.	24-wk hypocaloric diet. Methotrexate discontinued. Following diet, 12-wk follow-up without dietary modification.	No dietary modification. Methotrexate discontinued. Same follow-up.	No significant differences between hypocaloric diet and control groups in psoriasis severity (PASI) over 24-week diet or 12-week follow-up. Mean (SD) weight loss at 12 wk, 9% (2.4%) hypocaloric diet group ($P < .05$), maintained at 24 wk ($P < .05$), weight regained at 36 wk; no significant weight change in controls
Kimball et al,[28] 2012; United States	Prospective, assessor-blind, randomized, controlled	Plaque psoriasis, PASI ≥10, age ≥18 y, BMI >25, eligible for phototherapy. Intervention: n = 20; control: n = 10.	12-wk Ornish diet (vegetarian, low fat, n = 10) or South Beach diet (low carbohydrate, n = 10). NB-UVB phototherapy 3 times/wk.	No dietary intervention. Same phototherapy.	No significant differences between Ornish diet, South Beach diet, and control groups in improvement in psoriasis severity. Specifically, PASI 75, 83% of Ornish diet group, 56% of South Beach diet group, 38% of controls ($P = .30$). Mean PASI improvement, 78% Ornish diet group, 72% South Beach diet group, 71% controls. Mean weight loss, 8% Ornish diet group, 7% South Beach diet group, 0% controls ($P < .05$ for each diet group vs controls)
Al-Mutairi and Nour,[19] 2014; Kuwait	Prospective, randomized, controlled	Psoriasis, PASI 20-50, age >18 y, BMI 25-35, receiving biologic therapy. Intervention: n = 131; control: n = 131.	24-wk hypocaloric diet	Usual diet	Compared with controls, hypocaloric diet group had significantly greater reduction in psoriasis severity and body weight. Specifically, PASI 75, 85.9% of hypocaloric diet group vs 59.4% of controls ($P < .001$). Mean (SD) weight change, −13.0 (1.2) kg hypocaloric diet group vs 1.5 (0.5) kg controls ($P < .001$)
Jensen et al,[23] 2013; Denmark	Prospective, randomized, controlled	Plaque psoriasis, age >18 y, BMI >27, stable psoriasis treatment ≥3 mo before study. Intervention: n = 30; control: n = 30.	16-wk hypocaloric diet	Given guidelines for a healthy diet	No significant difference between hypocaloric diet and control groups in psoriasis severity after 16 wk. Specifically, mean between-group difference in PASI, −2.0 (95% CI, −4.1 to 0.1) in favor of hypocaloric diet group ($P = .06$). Compared with controls, hypocaloric diet group had significantly better dermatology quality of life and significantly greater weight loss after 16 wk. Specifically, mean between-group difference in DLQI, −2.0 (95% CI, −3.6 to −0.3) in favor of hypocaloric diet group ($P = .02$). Mean between-group difference in weight loss 15.4 (95% CI, 12.3 to 18.5) kg in favor of hypocaloric diet group ($P < .001$). PASI improvement correlated with weight loss in all participants ($P < .001$)
Ručević et al,[26] 2003; Croatia	Prospective, controlled	Biopsy-confirmed psoriasis inpatients, BSA >30%, duration >10 y, age 40-65 y. Intervention: n = 42; control: n = 40.	4-wk hypocaloric, low-protein diet. No coffee or alcohol.	Standard hospital diet. No alcohol.	Between-group comparisons were not reported. "Significant improvement of clinical findings" in hypocaloric diet group (effect measures not reported). Body weight did not change significantly in either group.
Jensen et al,[24] 2016; Denmark[b]	Prospective, uncontrolled	Plaque psoriasis, age >18 y, BMI >27, stable psoriasis treatment ≥3 mo before study. N = 56.	16-wk hypocaloric diet. Following hypocaloric diet, 48-wk maintenance period with 1 meal and 1 snack daily replaced by hypocaloric diet formula products.	None	Uncontrolled data: Hypocaloric diet resulted in significant improvement in psoriasis severity and dermatology quality of life and significant weight loss after 16 wk; changes in psoriasis severity and dermatology quality of life were maintained after maintenance period. Specifically, mean PASI change, −2.3 (95% CI, −3.1 to −1.5) at week 16, −2.9 (95% CI, −3.9 to −1.9) at week 64. Mean DLQI change, −2.3 (95% CI, −3.2 to −1.4) at week 16 to −1.9 (95% CI, −3.0 to −0.9) at week 64. Mean weight loss, 15.0 (95% CI, 13.4 to 16.6) kg at week 16, 10.1 (95% CI, 8.1 to 12.0) kg at week 64
Roongpisuthipong et al,[25] 2013; Thailand	Prospective, uncontrolled	Plaque psoriasis, age >18 y, BMI ≥30, metabolic syndrome. N = 10.	12-wk hypocaloric diet. Low- or moderate-potency topical steroids.	None	Uncontrolled data: Hypocaloric diet resulted in 30.9% improvement in psoriasis severity (PASI; $P < .05$), 62.5% improvement in dermatology quality of life (DLQI; $P < .01$), and 9.6% weight loss ($P < .01$) after 12 wk
Oral ω-3 Fatty Acid Fish Oil Supplementation					
Kristensen et al,[12] 2018; Denmark	Prospective, double-blind, randomized, placebo-controlled	PsA, age >18 y, not receiving biologics or oral steroids. Intervention: n = 73; control: n = 72.	24-wk supplementation with ω-3 fatty acids: 6 capsules containing 1.5 g EPA, 1.5 g DHA daily	Supplementation with placebo: 6 capsules containing 3 g olive oil daily	No significant differences between ω-3 and control groups in changes in any clinical outcome measures of skin or arthritis disease severity. Compared with ω-6 group, ω-3 group had greater reduction in the use of NSAIDs ($P = .04$) and paracetamol ($P = .04$). Significant reductions in tender joint count, DAS28-CRP, LEI, SPARCC, PASI in ω-3 group; significant reduction in LEI in ω-6 group.
Søyland et al,[35] 1993; Norway	Prospective, double-blind, randomized, placebo-controlled	Plaque psoriasis, BSA >8%, age 19-74 y, no systemic treatment 4 wk before study, topical treatments continued. Intervention: n = 72; control: n = 73.	4-mo supplementation with ω-3 fatty acids: 6 fish oil capsules containing 3.1 g EPA, 1.9 g DHA, vitamin E daily. Reduced dietary saturated fatty acid intake.	Supplementation with ω-6 fatty acids: 6 corn oil capsules with vitamin E daily. Reduced dietary saturated fatty acid intake.	No significant differences between ω-3 and control groups in changes in psoriasis severity. Specifically, mean target plaque score (scaling, erythema, infiltration) changed identically between ω-3 and control groups. Mean PASI did not change significantly in either group. Patient-reported psoriasis severity score (redness, scaling, itching, effect on daily living) did not change significantly in either group.

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Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA) (continued)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Strong and Hamill,[36] 1993; United Kingdom	Prospective, double-blind, randomized, placebo-controlled	Plaque psoriasis inpatients, no prior systemic treatment or phototherapy, no recent topical steroid treatment. Intervention: n = 26; control: n = 25.	7-mo supplementation with ω-3 fatty acids: 12 fish oil/evening primrose oil capsules (Efamol Marine) containing 216 mg EPA, 240 mg DHA daily; started inpatient, continued after discharge	Supplementation with placebo: 12 liquid paraffin capsules daily	No significant differences between ω-3 and control groups in psoriasis severity (percentage area involved, redness, scaling, itch, overall impression) over 7 mo of supplementation
Madland et al,[33] 2006; Norway	Prospective, double-blind, randomized, placebo-controlled	Polyarticular PsA (5 or more swollen joints), no intra-articular glucocorticoids or change in DMARDs for 4 wk before study, NSAIDs and DMARDs continued. Intervention: n = 22; control: n = 21.	14-d supplementation with ω-3 fatty acids: 30 mL seal oil containing 2.4 g EPA, 1.1 g DPA, 2.6 g DHA daily. Following supplementation, 4-wk follow-up without supplementation.	Supplementation with ω-6 fatty acids: 30 mL soy oil daily. Same follow-up	No significant differences between ω-3 and ω-6 groups in clinical outcome measures of skin or arthritis disease severity at the end of supplementation or after the follow-up period. Patient's global assessment VAS improved significantly at week 6 in ω-3 group ($P < .01$). No. of swollen joints decreased at week 6 in ω-3 group ($P < .05$). No. of tender joints decreased at week 6 in both groups ($P < .05$). PASI unchanged in both groups after treatment.
Veale et al,[34] 1994; United Kingdom	Prospective, double-blind, randomized, placebo-controlled	PsA, age 18-76 y, NSAID/paracetamol dose stable for 1 mo before study. Intervention: n = 19; control: n = 19.	9-mo supplementation with ω-3 fatty acids: 12 fish oil/evening primrose oil capsules (Efamol Marine) containing 240 mg EPA, 132 mg DHA, vitamin E daily. Subsequently crossed over to placebo for 3 mo.	12-mo supplementation with placebo: 12 liquid paraffin with vitamin E capsules daily	No significant differences between ω-3 and control groups in arthritis disease severity (grip strength, No. of active joints, Ritchie articular index, duration of morning stiffness, NSAID intake) or skin disease severity (BSA, investigator disease severity VAS, skin itch VAS) after 6, 9, or 12 mo
Bjørneboe et al,[38] 1988; Norway	Prospective, double-blind, randomized, placebo-controlled	Psoriasis, age 17-72 y, no topical steroids 2 mo before study. Intervention: n = 15; control: n = 15.	8-wk supplementation with ω-3 fatty acids: 10 fish oil capsules (MaxEPA) containing 1.8 g EPA, 1.2 g DHA, vitamin A, vitamin E, vitamin D daily	Supplementation with placebo: 10 olive oil capsules daily	Between-group comparisons were not reported. Clinical score (erythema, infiltration, desquamation) and BSA did not change significantly in ω-3 or control groups.
Bittiner et al,[39] 1988; England	Prospective, double-blind, randomized, placebo-controlled	Plaque psoriasis, not receiving systemic treatment, topical treatment continued. Intervention: n = 14; control: n = 14.	12-wk supplementation with ω-3 fatty acids: 10 fish oil capsules (MaxEPA) containing 1.8 g EPA, 1.2 g DHA daily	Supplementation with placebo: 10 olive oil capsules daily	Compared with controls, ω-3 group had significantly greater improvement in itch at week 8 and erythema at weeks 8 and 12 ($P < .05$). No significant differences between groups in improvement in scaling or BSA.
Gupta et al,[37] 1990; United States	Prospective, double-blind, randomized, placebo-controlled	Plaque psoriasis, BSA >10%, adults, no systemic treatment for 4 wk or topical treatment for 2 wk before study. Intervention: n = 10; control: n = 15.	9-wk supplementation with ω-3 fatty acids: 30 fish oil capsules (Max-EPA) containing 5.4 g EPA, 3.6 g DHA daily. Betamethasone diproprionate cream applied twice daily weeks 1-3.	Supplementation with placebo: 30 olive oil capsules daily. Same topical treatment weeks 1-3.	No significant differences between ω-3 and control groups in psoriasis improvement after topical treatment or in time to worsening of psoriasis to pretreatment severity. Specifically, equivalent improvement in psoriasis severity (scale, erythema, thickness, BSA) between groups after 3-wk topical steroid treatment. Mean (SD) time from termination of topical steroid treatment to worsening of psoriasis to pretreatment severity 4.9 (0.5) wk ω-3 group vs 4.5 (0.3) wk controls ($P = .4$).
Gupta et al,[30] 1989; United States	Prospective, double-blind, randomized, placebo-controlled	Psoriasis, BSA 10%-50%, adults, skin type II or III, no systemic treatment for 4 wk or topical treatment for 2 wk before study. Intervention: n = 9; control: n = 11.	15-wk supplementation with ω-3 fatty acids: 20 fish oil capsules (Max-EPA) containing 3.6 g EPA, 2.4 g DHA daily. UVB phototherapy twice weekly weeks 3-11.	Supplementation with placebo: 20 olive oil capsules daily. Same phototherapy weeks 3-11.	Compared with controls, ω-3 group had significantly greater improvement in psoriasis severity. Specifically, change from baseline in scale, −35% ω-3 group vs 9% controls ($P = .008$); erythema, −43% ω-3 group vs 0% controls ($P = .02$); thickness, −48% ω-3 group vs 7% controls ($P = .006$); BSA, −46% ω-3 group vs 32% controls ($P < .001$).

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Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA) (continued)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Danno and Sugie,[66] 1998; Japan	Prospective, open-label, randomized, controlled	Plaque psoriasis, moderate disease, age 25-78 y, topical treatment continued. Intervention: n = 20; control: n = 20.	12-wk supplementation with ω-3 fatty acids: EPA capsules (ReEPA) containing 1.8 g EPA daily. Low-dose etretinate (20 mg daily).	No supplementation. Same etretinate.	Compared with controls, ω-3 group had significantly greater and faster reduction in psoriasis severity. Specifically, ≥75% reduction in clinical score[c] (erythema, induration, desquamation) in 45% of ω-3 group vs 15% of controls ($P < .05$). ≥50% reduction in clinical score in 65% of ω-3 group vs 55% of controls (NS). Mean (SD) duration to 50% reduction in clinical score, 5.1 (1.5) wk ω-3 group vs 7.6 (3.4) wk controls ($P < .05$).
Balbás et al,[67] 2011; Spain	Prospective, open-label, nonrandomized, controlled	Plaque psoriasis, PASI ≤10, age 18-70 y, no systemic/biologic treatment for 4 wk or topical treatment for 2 wk before study. Intervention: n = 15; control: n = 15.	8-wk supplementation with ω-3 fatty acids: 2 Oravex capsules containing 560 mg EPA, 80 mg DHA, 15 mg zinc, 55 µg selenium daily. Topical tacalcitol.	No supplementation. Topical tacalcitol.	Compared with controls, ω-3 group had significantly greater improvement in psoriasis severity, psoriatic nail severity, dermatology quality of life, pruritus, and scalp psoriasis. Specifically, mean PASI reduction, 6.8 ω-3 group vs 3.53 controls ($P < .001$). Mean NAPSI reduction, 1.23 ω-3 group vs 0 controls ($P = .048$). Mean DLQI reduction, 6.7 ω-3 group vs 3.0 controls ($P = .006$). Presence of pruritus reduction, 80% ω-3 group vs 40% controls ($P < .001$). Mean severity of scalp psoriasis reduction, 0.6 ω-3 group vs 0 controls ($P = .004$).
Lassus et al,[68] 1990; Finland	Prospective, uncontrolled	Plaque psoriasis, 43% with PsA, age 23-68 y, not receiving systemic or topical treatment. N = 80.	8-wk supplementation with ω-3 fatty acids: 6 capsules containing 1.12 g EPA, 0.76 g DHA daily	None	Uncontrolled data: Supplementation with ω-3 resulted in significant improvement in skin and arthritis disease severity after 8 wk. Specifically, mean PASI, 3.56 at baseline, 1.98 at 4 wk ($P < .001$), 1.24 at 8 wk ($P < .001$). Mean pruritus, scaling, induration, and erythema scores improved significantly from baseline at weeks 4 and 8 ($P < .01$). Patient-reported severe joint pain in 53% of patients with PsA at baseline, 6% at week 8 ($P < .001$).
Kragballe and Fogh,[69] 1989; Denmark	Prospective, open-label, uncontrolled	Plaque psoriasis, no systemic treatment for 2 mo or topical treatment for 2 wk before study. N = 30.	4-mo supplementation with ω-3 fatty acids: 30 mL fish oil (MaxEPA) containing 5.4 g EPA, 4.8 g DHA, vitamins A, D, and E daily. 100 µg selenium daily. Low-fat diet.	None	Uncontrolled data: Supplementation with ω-3 resulted in "moderate or excellent improvement" in psoriasis severity in 58% of patients, "mild improvement" in 19%, and "no change" in 23% after 4 mo (degree of improvement vs baseline graded at each visit, categories not defined)
Kettler et al,[70] 1988; United States	Prospective, open-label, uncontrolled	Plaque psoriasis (n = 24), generalized pustular psoriasis (n = 1), palmoplantar pustulosis (n = 1), age 19-61 y, psoriasis treatment continued. N = 26.	6-8 wk supplementation with ω-3 fatty acids: 18 fish oil capsules (Max EPA) containing 3.2 g EPA, 2.2 g DHA daily	None	Uncontrolled data: Supplementation with ω-3 resulted in minimal improvement in psoriasis severity in 35% of patients, no change in 43%, and "mild worsening" in 22% after 6-8 wk (minimal improvement is 1 point improvement in erythema, scale, or thickness on 4-point scale)
Kragballe,[71] 1989; Denmark	Prospective, open-label, uncontrolled	Plaque psoriasis, age 12-69 y, no systemic treatment for 2 mo or topical treatment for 2 wk before study. N = 18.	4-mo supplementation with ω-3 and ω-6 fatty acids: 12 capsules (Super Gamma-Oil Marine) containing 540 mg EPA, 360 mg DHA, 3.36 g LA, 960 mg GLA, vitamin E daily. 100 µg selenium daily. Low-fat diet.	None	Uncontrolled data: Supplementation with ω-3 and ω-6 resulted in "moderate or excellent improvement" in psoriasis severity in 59% of patients, "mild improvement" in 23%, and "no change" in 18% after 4 mo (degree of improvement vs baseline graded at each visit, categories not defined)
Ziboh et al,[72] 1986; United States	Prospective, open-label, uncontrolled	Psoriasis, BSA >10%, 13% with PsA, adults, no systemic treatment for 2 mo or topical treatment for 2 wk before study. N = 15.	8 wk supplementation with ω-3 fatty acids: 60-75 g fish oil concentrate (Max-EPA) containing 11-14 g EPA, 7-9 g DHA daily. Exclusion of fats and oils from diet	None	Uncontrolled data: Supplementation with ω-3 resulted in significant reduction in erythema ($P < .02$), scaling ($P < .001$), and thickness ($P < .004$) after 8 wk; BSA unchanged from baseline
Maurice et al,[73] 1987; United Kingdom	Prospective, uncontrolled	Plaque psoriasis, BSA 5%-60%, 20% with PsA, age 25-65 y, long-standing disease requiring systemic treatment. N = 10.	≥6-wk supplementation with ω-3 fatty acids: ≤50 mL fish oil (Max-EPA) containing ≤12 g EPA, unknown quantity DHA daily. Low-fat diet.	None	Uncontrolled data: Supplementation with ω-3 resulted in "slight to moderate improvement" in psoriasis severity (redness, scaling) in 80% of patients after 6 wk; "improvement" was maintained for the duration of treatment

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Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA) (continued)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Kojima et al,[74] 1989; Japan	Prospective, uncontrolled	Psoriasis, age 22-75 y, topical treatment continued. N = 9.	6-mo supplementation with ω-3 fatty acids: 3.6 g EPA daily	None	Uncontrolled data: Supplementation with ω-3 resulted in "clinically significant improvement" in psoriasis severity (erythema, scaling, thickness) in 86% of patients after 6 mo
Intravenous ω-3 Fatty Acid Fish Oil Supplementation					
Mayser et al,[40] 1998; Czech Republic, Germany, Austria, Poland, Slovakia	Prospective, double-blind, randomized, placebo-controlled	Plaque psoriasis inpatients, PASI ≥15, 18% with PsA, age 18-80 y, no systemic treatment for 3 mo or topical treatment for 7 d before study. Intervention: n = 43; control: n = 40.	14-d treatment with ω-3 lipid emulsion (Omegavenous) 100 mL IV twice daily (4.2 g EPA, 4.2 g DHA daily)	Treatment with ω-6 lipid emulsion (Lipovenous) 100 mL IV twice daily	Compared with ω-6 group, ω-3 group had significantly greater reduction in psoriasis severity. Specifically, mean (SD) PASI reduction, 11.2 (9.8) ω-3 group vs 7.5 (8.8) ω-6 group (P = .048). PASI 50, 37% of ω-3 group vs 23% of ω-6 group. Mean (SD) self-assessed severity VAS score[c] reduction, 17.5 (19.3) ω-3 group vs 10.4 (18.4) ω-6 group.
Grimminger et al,[41] 1993; Germany	Prospective, double-blind, randomized, placebo-controlled	Acute guttate psoriasis inpatients, BSA >10%, age 21-65 y, not receiving systemic treatment, no topical treatment for 5 d before study. Intervention: n = 9; control: n = 11.	10-d treatment with ω-3 lipid emulsion (Omegavenous) 50 mL IV twice daily (2.1 g EPA, 2.1 g DHA daily)	Treatment with ω-6 lipid emulsion (Lipovenous) 50 mL IV twice daily	Compared with ω-6 group, ω-3 group had greater improvement in all psoriasis severity measures (erythema, infiltration, desquamation, patient-reported subjective score [daily life impairment, pruritus, burn, pain]) (P < .05). All psoriasis severity measures improved in both groups.
Vitamin D Supplementation					
Siddiqui and Al-Khawajah,[44] 1990; Saudi Arabia	Prospective, double-blind, randomized, placebo-controlled	Biopsy-confirmed psoriasis, PASI >15, adults, no systemic or topical treatment for 4 wk before study. Intervention: n = 20; control: n = 21.	12-wk supplementation with vitamin D: 1 capsule containing 1 μg alfacalcidol daily	Supplementation with placebo: 1 capsule daily	No significant differences between vitamin D and control groups in improvement in psoriasis severity. Specifically, slight improvement (<33% PASI reduction) in 45% of vitamin D group vs 38% of controls. No change or increase in PASI, 55% of vitamin D group vs 62% of controls
Gaál et al,[45] 2009; Hungary	Prospective, open-label, nonrandomized, controlled	PsA, treated with methotrexate 10 mg weekly and NSAIDs for ≥3 mo before study. Intervention: osteopenia, n = 10; control: no osteopenia, n = 9.	6-mo supplementation with vitamin D: 0.5 μg alfacalcidol daily	No supplementation	Between-group comparisons were not reported. Significant improvement in arthritis disease severity (DAS28) in vitamin D group (P = .048); no significant change in controls. No significant improvement in skin disease severity (PASI, patient's global assessment VAS) in either group.
Perez et al,[47] 1996; United States	Prospective, open-label, uncontrolled	Plaque psoriasis or EP, BSA ≥15%, age 19-76 y, not responding to ≥1 standard treatment, no systemic treatment or phototherapy for 30 d or topical treatment for 14 d before study. N = 85.	6-mo to 3-y supplementation with vitamin D: mean (SD) dose 2.1 (0.8) μg calcitriol (Rocaltrol) daily (dose adjusted based on urinary calcium level). Dietary calcium limited to 800 mg daily.	None	Uncontrolled data: Supplementation with vitamin D resulted in significant improvement in psoriasis severity after 6 mo and 3 y. Specifically, mean (SD) PASI, 18.4 (1.0) at baseline, 9.7 (0.8) at month 6, 7.0 (1.3) at year 3 (P < .001). Mean (SD) global severity score (erythema, thickness, scaling), 7.7 (1.2) at baseline, 3.2 (1.0) at year 3 (P < .001). Erythema, scaling, thickness each decreased at month 6 (P < .001) and year 3 (P < .001).
Morimoto et al,[50] 1986; Japan	Prospective, open-label, uncontrolled	Psoriasis, age 23-63 y, all psoriasis treatments stopped before study. N = 17.	6-mo supplementation with vitamin D: 1 μg alfacalcidol (Alfarol) daily	None	Uncontrolled data: Supplementation with vitamin D resulted in effective response in 76% of patients after 6 mo (effective response is "moderate improvement," "marked improvement," or "complete remission" in psoriasis severity)
Smith et al,[48] 1988; United States	Prospective, uncontrolled	Psoriasis, moderate to extensive disease refractory to standard treatment, age 22-63 y, no psoriasis treatment for 2 wk before study. N = 14.	≤1-y supplementation with vitamin D: capsules (Rocaltrol) containing 0.5-2.0 μg calcitriol daily (dose adjusted based on urinary and serum calcium levels). Dietary calcium limited to 800 mg daily.	None	Uncontrolled data: Supplementation with vitamin D resulted in >25% improvement in psoriasis severity (lesion size, erythema, scaling, pruritus) in 71% of patients, >50% improvement in 50%, and >75% improvement in 50%
Huckins et al,[63] 1990; United States	Prospective, open-label, uncontrolled	PsA, age 18-70 y, no change in treatment for 3 mo before or during study (changes in NSAIDs or steroids permitted). N = 10.	6-mo supplementation with vitamin D: 0.5-2.0 μg calcitriol (Rocaltrol) daily (dose adjusted based on urinary and serum calcium levels)	None	Uncontrolled data: Supplementation with vitamin D resulted in significant improvement in tender joint count (P < .01) and physician global assessment of arthritis activity (P < .05) after 6 mo; no significant change in patient global assessment, grip strength, or patient-reported pain and physical activity scores (AIMS)

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Additional studies are needed to determine whether it is the type of diet and/or weight reduction that reduces psoriasis severity, the associated mechanisms, the amount of weight reduction needed for clinical response, how to implement dietary recommendations effectively, and the role of exercise in conjunction with dietary weight reduction.

Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA) (continued)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Finamor et al,[46] 2013; Brazil	Prospective, open-label, uncontrolled	Psoriasis, age >18 y, no change in treatments. N = 9.	6-mo supplementation with vitamin D: 1.75 mL sunflower oil solution containing 35 000 IU cholecalciferol daily. Low-calcium diet with daily hydration ≥2.5 L	None	Uncontrolled data: Supplementation with vitamin D resulted in significant improvement in psoriasis severity (PASI) in all patients after 6 mo (P = .002). Lower psoriasis severity (PASI) was correlated with higher serum vitamin D levels (P = .001)
el-Azhary et al,[75] 1993; United States	Prospective, uncontrolled	Psoriasis, BSA >25%, age 28-65 y, no psoriasis treatment for 4 wk before study (corticosteroid cream not stronger than triamcinolone, 0.05%, permitted). N = 8.	6-mo supplementation with vitamin D: 0.5-2.0 µg calcitriol (Rocaltrol) daily (dose adjusted based on clinical response). Dietary calcium limited to 800 mg daily.	None	Uncontrolled data: Supplementation with vitamin D resulted in "marked improvement" in scale, erythema, and thickness in 13% of patients, "moderate improvement" in 13%, "mild improvement" in 38%, and "no improvement" in 38% after 6 mo; supplementation resulted in "marked improvement" in area of involvement in 13% of patients, "mild improvement" in 13%, and "no improvement" in 75% after 6 mo
Takamoto et al,[49] 1986; Japan	Prospective, uncontrolled	Psoriasis, duration >6 mo, refractory to treatment, age 37-63 y, topical steroids continued. N = 7.	6-mo supplementation with vitamin D: 1 µg alfacalcidol (Alfarol) daily	None	Uncontrolled data: Supplementation with vitamin D resulted in complete remission of psoriasis in 29% of patients, marked improvement in 29%, and no change in 43% after 6 mo (complete remission is complete flattening of plaques, area improved ≥95%; marked improvement is nearly complete flattening of plaques, area improved 50%-90%)
Selenium Supplementation					
Kharaeva et al,[54] 2009; Russia	Prospective, double-blind, randomized, placebo-controlled	Severe EP or PsA inpatients, adults. Intervention: n = 14 (EP), n = 15 (PsA). Control: n = 14 (EP), n = 15 (PsA).	30-d supplementation with selenium: 4 soy lecithin capsules containing 48 µg selenium aspartate, 50 mg coenzyme Q_{10} (ubiquinone acetate), 50 mg vitamin E (α-tocopherol) daily. EP: IV corticosteroids, injections of desensitizers, keratoplastic and corticosteroid ointments. PsA: Oral corticosteroids, desensitizers, NSAIDs, painkillers.	Supplementation with placebo: 4 soy lecithin capsules daily. EP: Same treatment. PsA: Same treatment.	Compared with PsA and EP controls, PsA and EP selenium groups had significantly lower overall psoriasis severity after supplementation. Specifically, mean (SD) severity score[c] (plaque desquamation, plaque hyperemia, plaque inflammation, nail dystrophy, joint pain) at day 30, 1.9 (0.1) PsA selenium group vs 6.8 (0.2) PsA controls (P < .001); 4.0 (0.1) EP selenium group vs 6.2 (0.1) EP controls (P < .05). Mean (SD) PASI at day 30, 16 (6) PsA selenium group vs 29 (10) PsA controls (P value not reported); 19 (4) EP selenium group vs 30 (5) EP controls (P < .05)
Serwin et al,[56] 2006; Poland	Prospective, double-blind, randomized, placebo-controlled	Psoriasis inpatients, age 18-50 y, skin type II or III, nonsmokers for ≥2 y, no selenium supplementation for 6 mo or topical treatment for 1 wk before study. Intervention: n = 19, control: n = 18.	4-wk supplementation with selenium: 2 selenomethionine tablets containing 200 µg selenium daily. NB-UVB phototherapy 5 times weekly. Following treatment, 4-week follow-up without supplementation or phototherapy.	Supplementation with placebo: 2 tablets daily. Same phototherapy. Same follow-up.	No significant differences between selenium and control groups in improvement in psoriasis severity at the end of supplementation or after the follow-up period. Specifically, PASI 75 at wk 4, 47% of selenium group vs 56% of controls (P > .05). PASI 75 at wk 8, 58% of selenium group vs 56% of controls (P > .05). Mean (SD) PASI at baseline, wk 4, wk 8, 13.02 (6.25), 3.59 (2.27), 2.82 (2.48) selenium group vs 12.37 (4.71), 2.34 (1.70), 2.23 (1.49) controls (significant reduction from baseline in both groups; P values not reported).
Serwin et al,[55] 2003; Poland	Prospective, double-blind, randomized, placebo-controlled	Plaque psoriasis, age 18-50 y. Intervention: n = 11; control: n = 11.	4-wk supplementation with selenium: 2 selenomethionine tablets containing 200 µg selenium daily. 5% salicylic acid ointment and 0.1%-0.3% dithranol ointment	Supplementation with placebo: 2 tablets daily. Same topical treatment.	No significant differences between selenium and control groups in proportion of patients with improved psoriasis severity after 2 or 4 wk. Specifically, PASI 50 at wk 2, 54% of selenium group vs 82% of controls (P > .05). PASI 75 at week 4, 45% of selenium group vs 82% of controls (P > .05).

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Table 2. Interventional Studies on the Effects of Gluten-Free Diet, Dietary Weight Reduction, and Dietary Supplements on Psoriasis and Psoriatic Arthritis (PsA) (continued)

Source; Country	Design	Study Population	Intervention	Control	Outcome Summary
Harvima et al,[52] 1993; Finland	Prospective, open, uncontrolled	Plaque psoriasis, age 21-52 y, no systemic treatment for 2 y or topical treatment for 2 wk before study. N = 7.	6-wk supplementation with selenium: 8 selenomethionine-yeast tablets containing 400 μg selenium daily. Following supplementation, 3-mo follow-up without supplementation.	None	Uncontrolled data: Supplementation with selenium resulted in "some improvement" in psoriasis severity in 29% of patients, "slight worsening" in 29%, and "severe worsening" in 14% after 6 wk; "overall clinical condition remained unchanged" after 3-mo follow-up
Vitamin B_{12} Supplementation					
Baker and Comaish,[58] 1962; United Kingdom	Prospective, double-blind, randomized, placebo-controlled	Psoriasis, age >12 y, using various topical treatments. Intervention: n = 36; control: n = 35	3-wk treatment with vitamin B_{12}: 1000 μg vitamin B_{12} IM 5 d weekly. Following treatment, 3 wk follow-up without treatment.	Treatment with placebo: placebo IM 5 d weekly. Same follow-up.	No differences between vitamin B_{12} and control groups in proportion of patients with improved psoriasis severity after 3 or 6 wk (reports that lack of treatment effect is "evident without formal statistical analysis"). Specifically, "substantially improved or clear" in 8% of vitamin B_{12} group vs 14% of controls at week 3; 61% of vitamin B_{12} group vs 60% of controls at week 6
Ruedemann,[57] 1954; United States	Open, uncontrolled	Psoriasis, age 21-68 y. N = 34.	≥10-d treatment with vitamin B_{12}: 1000 μg vitamin B_{12} IM daily. Following treatment, ≥2 mo maintenance period with unspecified dose of vitamin B_{12}.	None	Uncontrolled data: Treatment with vitamin B_{12} resulted in changes in psoriasis severity of "eruption involuted" in 32% of patients, "improved 75%-80%" in 29%, "slowly improving" in 18%, "no results" in 15%, and "slight recurrence after treatment stopped, resumed with good results" in 15%
Micronutrient Supplementation					
Yousefzadeh et al,[11] 2017; Iran	Prospective, double-blind, randomized, controlled	Biopsy-confirmed psoriasis, PASI >10, age 20-50 y, no methotrexate for 2 mo before study. Intervention: n = 17; control: n = 17.	12-wk supplementation with micronutrients: 1 tablet (Immunace) containing folic acid, magnesium, iron, zinc, copper, manganese, selenium, chromium, iodine, vitamins A, D, E, K, C, B_1, B_2, B_3, B_6, B_{12} daily. Methotrexate 7.5-15 mg weekly.	No supplementation. Same methotrexate therapy.	Compared with controls, micronutrient group had significantly greater reduction in psoriasis severity (PASI) after supplementation (P = .045). PASI 75, 65% of micronutrient group vs 35% of controls (P = .08). Mean (SD) PASI improved from 31.80 (10.57) at baseline to 5.50 (3.82) at week 12 micronutrient group (P = .001); from 30.23 (10.87) to 10.86 (9.84) controls (P = .001)

Abbreviations: AGA, antigliadin antibodies; AIMS, Arthritis Impact Measurement Scales; BMI, body mass index, calculated as weight in kilograms divided by height in meters squared; BSA, body surface area; DAS28, 28-joint Disease Activity Score; DAS28-CRP, 28-joint Disease Activity Score (C-reactive protein); DHA, docosahexaenoic acid; DLQI, Dermatology Life Quality Index; DMARDs, disease-modifying antirheumatic drugs; DPA, docosapentaenoic acid; EP, erythrodermic psoriasis; EPA, eicosapentaenoic acid; GLA, γ-linolenic acid; HR, hazard ratio; IM, intramuscular; IV, intravenous; LA, linoleic acid; LEI, Leeds Enthesitis Index; MDA, minimal disease activity; NAPSI, Nail Psoriasis Severity Index; NB-UVB, narrowband UVB; NS, nonsignificant; NSAIDs, nonsteroidal anti-inflammatory drugs; PASI, Psoriasis Area and Severity Index; plus sign, positive; SPARCC, Spondyloarthritis Research Consortium of Canada Enthesitis Index; tTG, tissue transglutaminase; VAS, visual analogue scale.

[a] These 2 articles report different outcome measures from the same study.

[b] Follow-up of subset of patients from Jensen et al,[23] 2013; intervention group entered maintenance period, control group first crossed over to 16-week hypocaloric diet then entered maintenance period.

[c] Higher score corresponds to greater disease severity.

Weight reduction should always be recommended for overweight patients because it has beneficial effects on other health outcomes including reduction of cardiometabolic and certain cancer risks. At this time, data are lacking on dietary weight reduction in normal-weight patients with psoriasis.

Recommendation 2: Dietary Weight Reduction in Psoriasis

- In overweight or obese adults with psoriasis (BMI, ≥25), we strongly recommend dietary weight reduction with a hypocaloric diet as an adjunctive intervention to standard medical therapies for psoriasis.
- This strong recommendation (strength 1) is based on level A evidence, with consistent results among included studies and agreement with systematic reviews and meta-analysis.[4,9,19-23]

Question 3: Are Any Dietary Supplements Helpful in Psoriasis?

Dietary supplements are used by a significantly higher proportion of patients with psoriasis than those without psoriasis.[29] Many patients with psoriasis believe that supplementation can improve their disease severity.[5] Patients most frequently report improvement from supplementation with fish oil and vitamin D.[5] However, patients should consult with a health care practitioner

Table 3. Dietary Recommendations for Psoriasis and Psoriatic Arthritis

Recommended or Not	Recommendation	Strength of Recommendation	Level of Evidence[a]	Source[b]
Psoriasis				
1. Gluten-free diet				
✓✓✓	In adults with psoriasis with confirmed celiac disease, we strongly recommend a gluten-free diet.	1	A	Rubio-Tapia et al,[17] 2013
✓✓	Only in adults with psoriasis who test positive for serologic markers of gluten sensitivity, we recommend a 3-mo trial of a gluten-free diet as an adjunctive intervention to standard medical therapies for psoriasis.	2A	B	De Bastiani et al,[16] 2015; Michaëlsson et al,[15] 2003; Michaëlsson et al,[14] 2000
X	We do not recommend universal screening for serologic markers of gluten sensitivity among adults with psoriatic diseases because of a high rate of false-positive results. Based on recommendations from the American College of Gastroenterology, patients who are candidates for screening include those with a first-degree relative with celiac disease or those with active gastrointestinal symptoms.			
2. Dietary weight reduction				
✓✓✓	In overweight or obese adults with psoriasis (BMI ≥25), we strongly recommend dietary weight reduction with a hypocaloric diet as an adjunctive intervention to standard medical therapies for psoriasis.	1	A	Al-Mutairi and Nour,[19] 2014; Gisondi et al,[20] 2008; Guida et al,[21] 2014; Jensen et al,[23] 2013; Naldi et al,[22] 2014
3a. Fish oil supplementation				
X	We do not recommend oral fish oil supplementation for treatment of psoriasis in adults because oral fish oil was not effective at the examined doses and durations.			
…	Because evidence is limited regarding the effectiveness of intravenous fish oil supplementation on psoriasis in adults, a recommendation cannot be made at this time.			
3b. Vitamin D supplementation				
X	We do not recommend oral vitamin D supplementation for prevention or treatment of psoriasis in adults with normal vitamin D levels.			
3c. Selenium supplementation				
X	We do not recommend selenium supplementation for treatment of plaque psoriasis in adults.			
…	Because evidence is limited regarding the effectiveness of selenium supplementation on erythrodermic psoriasis in adults, a recommendation cannot be made at this time.			
3d. Vitamin B_{12} supplementation				
X	We do not recommend vitamin B_{12} supplementation for treatment of psoriasis in adults.			
3e. Micronutrient supplementation				
…	Because evidence is limited regarding the effectiveness of combination micronutrient supplementation on psoriasis in adults, a recommendation cannot be made at this time.			
4. Specific foods, nutrients, or dietary patterns				
✓	Based on low-quality evidence, adults with psoriasis may consider a trial of a Mediterranean diet and consuming extra virgin olive oil as the main culinary lipid, ≥2 servings of vegetables daily, ≥3 servings of fruits daily, legumes ≥3 times weekly, fish or seafood ≥3 times weekly, tree nuts ≥3 times weekly, or sofrito sauce (tomatoes, onions, garlic, olive oil) ≥2 times weekly.	2B	C	Barrea et al,[60] 2015
✓	Based on low-quality evidence, adults with psoriasis may consider a trial of consuming more ω-3 PUFAs, monounsaturated fatty acids, fiber, or complex carbohydrates and consuming less total energy, saturated fatty acids, total PUFAs, ω-6 PUFAs, ω-6:ω-3 PUFA ratio, or simple carbohydrates.	2B	C	Barrea et al,[61] 2015
Psoriatic Arthritis				
5. Dietary interventions				
✓✓	In overweight or obese adults with psoriatic arthritis (BMI ≥25), we recommend dietary weight reduction with a hypocaloric diet as an adjunctive intervention to standard medical therapies for psoriatic arthritis.	2A	B	Di Minno et al,[62] 2014

(continued)

Table 3. Dietary Recommendations for Psoriasis and Psoriatic Arthritis (continued)

Recommended or Not	Recommendation	Strength of Recommendation	Level of Evidence[a]	Source[b]
✓✓	In adults with psoriatic arthritis, we recommend a trial of oral vitamin D supplementation (0.5 μg alfacalcidol or 0.5-2.0 μg calcitriol daily) as an adjunctive intervention to standard medical therapies for psoriatic arthritis.	2A	B	Gaál et al,[45] 2009; Huckins et al,[63] 1990
…	Because evidence is limited regarding the effectiveness of selenium supplementation on psoriatic arthritis in adults, a recommendation cannot be made at this time.			
X	We do not recommend fish oil supplementation for treatment of psoriatic arthritis in adults.			

Abbreviations: BMI, body mass index, calculated as weight in kilograms divided by height in meters squared; check mark, positive recommendation, with strength of recommendation indicated by number of check marks; ellipses, neither positive nor negative recommendation; X, negative recommendation.

[a] Level of evidence determines the grade of recommendation.[13] Level of evidence "A" refers to high-quality, patient-oriented evidence, which leads to a "strong recommendation" (strength of recommendation 1). Level of evidence "B" refers to limited-quality, patient-oriented evidence, which leads to a "weak recommendation" (strength of recommendation 2A). Level of evidence "C" refers to low-quality evidence, which leads to a "weak recommendation" (strength of recommendation 2B).

[b] Table exclusively includes sources from the highest level of evidence supporting the corresponding strength of recommendation.

before taking supplements because many supplements have adverse effects or contraindications.

ω-3 Fatty Acids (Fish Oil)

Polyunsaturated fatty acids (PUFAs), a type of dietary fatty acids, include ω-3 and ω-6 PUFAs. The ω-6 PUFAs arachidonic acid and linoleic acid are metabolized into leukotriene B4 (LTB4); LTB4 is an inflammatory mediator that is elevated in psoriatic plaques.[30,31] In contrast, metabolites of ω-3 PUFAs oppose the pro-inflammatory effects of LTB4.[32]

Whereas the typical Western diet has an ω-6:ω-3 ratio of 15-20:1, an ideal ratio of 1.8:1 is recommended by an international panel of experts.[31] This ratio is similar to that found throughout human history. Notably, lower psoriasis incidence is seen in regions with a higher relative intake of ω-3 PUFAs.[31] Oil derived from cold-water fish is rich in ω-3 PUFAs, including eicosapentaenoic acid (EPA) and docosapentaenoic acid (DHA).[30]

Studies evaluating the use of oral fish oil supplementation in psoriasis show conflicting results. Accurately assessing study quality is paramount when trial results differ and wheremeta-analyses are not possible due to heterogeneity of outcomes. Double-blind RCTs are particularly helpful in minimizing assessment bias. Among 9 double-blind RCTs,[12,30,33-39] oral supplementation with fish oil was shown to be ineffective in reducing psoriasis severity in 7 of 9 trials; these data are based on 449 patients enrolled in trials showing no benefit and 48 patients in trials showing benefit. The duration of supplementation in these trials ranged from 14 days to 7 months, and the daily dosage of EPA and DHA varied from 0.216 to 5.4 g and 0.132 to 3.6 g, respectively.

However, with regard to intravenous fish oil supplementation, 2 double-blind RCTs with a pooled population of 103 patients showed beneficial effects in reducing psoriasis severity.[40,41] The duration of supplementation in these trials ranged from 10 to 14 days, and the daily intravenous dosage of both EPA and DHA varied from 2.1 to 4.2 g.

Recommendation 3a: Fish Oil Supplementation in Psoriasis

- We do not recommend oral fish oil supplementation for treatment of psoriasis in adults because oral fish oil was not effective at the examined doses and durations.
- Because evidence is limited regarding the effectiveness of intravenous fish oil supplementation on psoriasis in adults, a recommendation cannot be made at this time.

Vitamin D

Vitamin D can be given in different forms including cholecalciferol, alfacalcidol, and calcitriol.[8] While topical vitamin D is well established as an effective treatment option in psoriasis, the benefit of oral supplementation is less certain. Vitamin D deficiency is common in patients with psoriasis,[42] and lower levels of serum vitamin D are associated with higher psoriasis severity.[43] However, data are lacking regarding the effects of vitamin D supplementation on psoriasis severity in vitamin D–deficient patients.

Overall, high-quality evidence is lacking to support the use of vitamin D supplementation in psoriasis. Controlled studies show no benefit of vitamin D supplementation for improving skin disease severity over a period of 3 to 6 months.[44,45] In comparison, some studies show that improvement in skin disease severity can be seen after vitamin D supplementation over a period of 6 months or longer in patients with plaque or erythrodermic psoriasis.[46-50] However, this is based entirely on uncontrolled data. In addition, to date, there is no clear evidence to support one form of vitamin D over another.

In individuals without a history of psoriasis, vitamin D intake appears to have no impact on the development of psoriasis.[51]

Recommendation 3b: Vitamin D Supplementation in Psoriasis

- We do not recommend oral vitamin D supplementation for prevention or treatment of psoriasis in adults with normal vitamin D levels.

Selenium

Selenium is an essential nutrient that can affect the immune system.[52] Decreased selenium levels can be seen in patients with psoriasis,[52] and low selenium concentrations may be associated with longer disease duration.[53]

Based on the available literature, selenium supplementation has varying results in patients with different subtypes of psoriasis.[52,54-56]

Specifically, in 1 small study (n = 28) evaluating inpatients with erythrodermic psoriasis, selenium supplementation with a combination of 48 µg selenium aspartate, 50 mg coenzyme Q_{10} (ubiquinone acetate), and 50 mg vitamin E (α-tocopherol) daily seemed to be beneficial in conjunction with conventional therapy.[54]

However, selenomethionine supplementation provides no benefit in patients with plaque psoriasis either as monotherapy or in combination with phototherapy or topical therapy.[52,55,56]

Recommendation 3c: Selenium Supplementation in Psoriasis

- We do not recommend selenium supplementation for treatment of plaque psoriasis in adults.
- Because evidence is limited regarding the effectiveness of selenium supplementation on erythrodermic psoriasis in adults, a recommendation cannot be made at this time.

Vitamin B_{12}

Based on the available literature, Vitamin B_{12} supplementation appears to be ineffective as a therapeutic intervention in psoriasis.[57,58] When compared with patients treated with placebo, intramuscular high-dose vitamin B_{12} was shown to be ineffective.[58]

Recommendation 3d: Vitamin B_{12} Supplementation in Psoriasis

- We do not recommend vitamin B_{12} supplementation for treatment of psoriasis in adults.

Micronutrients

Combination micronutrient supplementation may be beneficial in plaque psoriasis. Specifically, compared with patients with psoriasis treated with low-dose methotrexate alone, those receiving supplementation with a combination of vitamins and minerals (folic acid, magnesium, iron, zinc, copper, manganese, selenium, chromium, iodine, and vitamins A, D, E, K, C, B_1, B_2, B_3, B_6, and B_{12}) for 3 months in conjunction with low-dose methotrexate had significantly greater improvement in psoriasis severity.[11] However, this is based on 1 small study (n = 34).

Recommendation 3e: Micronutrient Supplementation in Psoriasis

- Because evidence is limited regarding the effectiveness of combination micronutrient supplementation on psoriasis in adults, a recommendation cannot be made at this time.

Question 4: Are There Any Specific Foods, Nutrients, or Dietary Patterns That Are Helpful or Harmful in Psoriasis?

A large proportion of patients with psoriasis report having tried a special diet for their psoriasis,[5] and a majority of patients believe that diet regimen can improve psoriasis.[27] Although multiple observational studies exist evaluating the dietary patterns of patients with psoriasis,[5,59-61] data demonstrating a relationship with disease severity is limited. In general, the dietary patterns of patients with psoriasis appear to be different from those of patients without psoriasis (Table 1). The contribution of certain foods, nutrients, and dietary patterns to psoriasis severity varies, which leads to the following recommendation.[60,61] Additional data from patient experiences help inform this topic[5,27]; however, these observations are exploratory and subject to reporting bias.

Recommendation 4: Specific Foods, Nutrients, or Dietary Patterns in Psoriasis

- Based on low-quality evidence, adults with psoriasis may consider a trial of a Mediterranean diet and consuming extra virgin olive oil as the main culinary lipid, at least 2 servings of vegetables daily, at least 3 servings of fruits daily, legumes at least 3 times weekly, fish or seafood at least 3 times weekly, tree nuts at least 3 times weekly, or sofrito sauce (tomatoes, onions, garlic, olive oil) at least 2 times weekly.[60]
- Based on low-quality evidence, adults with psoriasis may consider a trial of consuming more ω-3 PUFAs, monounsaturated fatty acids, fiber, or complex carbohydrates and consuming less total energy, saturated fatty acids, total PUFAs, ω-6 PUFAs, ω-6:ω-3 PUFA ratio, or simple carbohydrates.[61]
  - This weak recommendation (strength 2B) is based on level C evidence.[60,61]

Question 5: Are Any of These Dietary Interventions Useful for Patients With Psoriatic Arthritis?

Psoriatic arthritis affects approximately one-third of patients with psoriasis.[2] Determining the effects of dietary interventions on psoriatic arthritis severity is important for overall disease management.

Gluten-Free Diet

All of the studies on gluten-free diet include patients with psoriatic arthritis but do not assess arthritis disease severity or report outcomes separately in these patients.[14-16] Therefore, it remains unknown whether gluten-free diet can help improve psoriatic arthritis severity.

Dietary Weight Reduction

Dietary weight reduction with a hypocaloric diet seems to be helpful in overweight and obese (BMI, ≥25) patients with psoriatic arthritis. Specifically, patients starting treatment with tumor necrosis factor blockers (etanercept, adalimumab, or infliximab) are significantly more likely to achieve minimal arthritis disease activity and to experience weight loss after a 6-month hypocaloric diet than after a regular diet.[62]

Dietary Supplements

Whereas oral vitamin D supplementation seems to be ineffective in improving skin disease severity, it may be beneficial in psoriatic arthritis. Specifically, uncontrolled studies show that 0.5 µg alfacalcidol or 0.5 to 2.0 µg calcitriol daily for 6 months leads to significant improvement in arthritis disease severity.[45,63]

In 1 small study (n = 30) evaluating inpatients with psoriatic arthritis, a combination of 48 µg selenium aspartate, 50 mg coenzyme Q_{10} (ubiquinone acetate), and 50 mg vitamin E (α-tocopherol) daily seemed to be beneficial in conjunction with conventional therapy.[54]

Based on the literature, we determine fish oil supplementation to be ineffective in psoriatic arthritis. Compared with control patients, patients supplementing with fish oil do not experience significantly greater improvement in arthritis disease severity.[12,33,34]

There are no data on vitamin B_{12} or combination micronutrient supplementation in psoriatic arthritis.

Specific Foods, Nutrients, or Dietary Patterns

There are no data to support specific foods, nutrients, or dietary patterns in psoriatic arthritis.

Recommendation 5: Dietary Interventions in Psoriatic Arthritis

- In overweight or obese adults with psoriatic arthritis (BMI, ≥25), we recommend dietary weight reduction with a hypocaloric diet as an adjunctive intervention to standard medical therapies for psoriatic arthritis.
- In adults with psoriatic arthritis, we recommend a trial of oral vitamin D supplementation (0.5 µg alfacalcidol or 0.5-2.0 µg calcitriol daily) as an adjunctive intervention to standard medical therapies for psoriatic arthritis.
- Because evidence is limited regarding the effectiveness of selenium supplementation on psoriatic arthritis in adults, a recommendation cannot be made at this time.
- We do not recommend fish oil supplementation for treatment of psoriatic arthritis in adults.
  - These weak recommendations (strength 2A) are based on level B evidence; limitations include a lack of randomization,[45] uncontrolled data,[45,63] and study-related bias.[45]

Discussion

In this article, we made evidence-based recommendations on the impact of diet in adults with psoriatic diseases. We highlight 3 concepts important for interpreting these recommendations. First, it is universally important to continue regular medical treatment for psoriatic diseases. We do not recommend relying on dietary interventions as the sole source of treatment. Second, dietary interventions can be associated with adverse effects and contraindications. Third, these dietary recommendations may have impact beyond patients' skin and joints and may affect one's general health.[5,64,65]

The findings of this systematic review are dependent on the strength of the primary literature. Some studies are not randomized, blinded, or controlled, which may introduce bias. There is heterogeneity among studies with regard to populations studied, interventions used, and outcomes measured.

Conclusions

Dietary interventions, when implemented, should be used in conjunction with standard medical therapies for psoriatic diseases. We strongly recommend dietary weight reduction with a hypocaloric diet in overweight and obese patients with psoriasis. We weakly recommend a gluten-free diet only in patients who test positive for serologic markers of gluten sensitivity. Based on low-quality data, select foods, nutrients, and dietary patterns may affect psoriasis. For patients with psoriatic arthritis, we weakly recommend vitamin D supplementation and dietary weight reduction with a hypocaloric diet in overweight and obese patients.

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REFERENCES

1. Rachakonda TD, Schupp CW, Armstrong AW. Psoriasis prevalence among adults in the United States. *J Am Acad Dermatol*. 2014;70(3):512-516.

2. Takeshita J, Grewal S, Langan SM, et al. Psoriasis and comorbid diseases: epidemiology. *J Am Acad Dermatol*. 2017;76(3):377-390.

3. Blauvelt A, Armstrong AW, Krueger GG. Essential truths for the care and management of moderate-to-severe psoriasis. *J Drugs Dermatol*. 2015;14(8):805-812.

4. Debbaneh M, Millsop JW, Bhatia BK, Koo J, Liao W. Diet and psoriasis, part I: impact of weight loss interventions. *J Am Acad Dermatol*. 2014;71(1):133-140.

5. Afifi L, Danesh MJ, Lee KM, et al. Dietary behaviors in psoriasis: patient-reported outcomes from a US national survey. *Dermatol Ther (Heidelb)*. 2017;7(2):227-242.

6. Magin PJ, Adams J, Heading GS, Pond DC, Smith W. Complementary and alternative medicine therapies in acne, psoriasis, and atopic eczema: results of a qualitative study of patients' experiences and perceptions. *J Altern Complement Med*. 2006;12(5):451-457.

7. Bhatia BK, Millsop JW, Debbaneh M, Koo J, Linos E, Liao W. Diet and psoriasis, part II: celiac disease and role of a gluten-free diet. *J Am Acad Dermatol*. 2014;71(2):350-358.

8. Millsop JW, Bhatia BK, Debbaneh M, Koo J, Liao W. Diet and psoriasis, part III: role of nutritional supplements. *J Am Acad Dermatol*. 2014;71(3):561-569.

9. Upala S, Sanguankeo A. Effect of lifestyle weight loss intervention on disease severity in patients with psoriasis: a systematic review and meta-analysis. *Int J Obes (Lond)*. 2015;39(8):1197-1202.

10. Upala S, Yong WC, Theparee T, Sanguankeo A. Effect of omega-3 fatty acids on disease severity in patients with psoriasis: a systematic review. *Int J Rheum Dis*. 2017;20(4):442-450.

11. Yousefzadeh H, Jabbari Azad F, Banihashemi M, Rastin M, Mahmoudi M. Evaluation of psoriasis severity and inflammatory responses under concomitant treatment with methotrexate plus micronutrients for psoriasis vulgaris: a randomized double blind trial. *Acta Dermatovenerol Alp Pannonica Adriat*. 2017;26(1):3-9.

12. Kristensen S, Schmidt EB, Schlemmer A, Rasmussen C, Johansen MB, Christensen JH. Beneficial effect of n-3 polyunsaturated fatty acids on inflammation and analgesic use in psoriatic arthritis: a randomized, double blind, placebo-controlled trial. *Scand J Rheumatol*. 2018;47(1):27-36.

13. Robinson JK, Dellavalle RP, Bigby M, Callen JP. Systematic reviews: grading recommendations and evidence quality. *Arch Dermatol*. 2008;144(1):97-99.

14. Michaëlsson G, Gerdén B, Hagforsen E, et al. Psoriasis patients with antibodies to gliadin can be improved by a gluten-free diet. *Br J Dermatol*. 2000;142(1):44-51.

15. Michaëlsson G, Ahs S, Hammarström I, Lundin IP, Hagforsen E. Gluten-free diet in psoriasis patients with antibodies to gliadin results in decreased expression of tissue transglutaminase and fewer Ki67+ cells in the dermis. *Acta Derm Venereol*. 2003;83(6):425-429.

16. De Bastiani R, Gabrielli M, Lora L, et al. Association between coeliac disease and psoriasis: Italian primary care multicentre study. *Dermatology*. 2015;230(2):156-160.

17. Rubio-Tapia A, Hill ID, Kelly CP, Calderwood AH, Murray JA; American College of Gastroenterology. ACG clinical guidelines: diagnosis and management of celiac disease. *Am J Gastroenterol*. 2013;108(5):656-676.

18. Benson BC, Mulder CJ, Laczek JT. Anti-gliadin antibodies identify celiac patients overlooked by tissue transglutaminase antibodies. *Hawaii J Med Public Health*. 2013;72(9)(suppl 4):14-17.

19. 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.

20. Gisondi P, Del Giglio M, Di Francesco V, Zamboni M, Girolomoni G. 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 Nutr*. 2008;88(5):1242-1247.

21. Guida B, Napoleone A, Trio R, et al. Energy-restricted, n-3 polyunsaturated fatty acids-rich diet improves the clinical response to immunomodulating drugs in obese patients with plaque-type psoriasis: a randomized control clinical trial. *Clin Nutr*. 2014;33(3):399-405.

22. Naldi L, Conti A, Cazzaniga S, et al; Psoriasis Emilia Romagna Study Group. Diet and physical exercise in psoriasis: a randomized controlled trial. *Br J Dermatol*. 2014;170(3):634-642.

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.

24. Jensen P, Christensen R, Zachariae C, 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.

25. Roongpisuthipong W, Pongpudpunth M, Roongpisuthipong C, Rajatanavin N. The effect of weight loss in obese patients with chronic stable plaque-type psoriasis. *Dermatol Res Pract*. 2013;2013:795932.

26. Ručević I, Perl A, Barišić-Druško V, Adam-Perl M. The role of the low energy diet in psoriasis vulgaris treatment. *Coll Antropol*. 2003;27(suppl 1):41-48.

27. Del Giglio M, Gisondi P, Tessari G, Girolomoni G. 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.

28. Kimball AB, Alavian C, Alora-Palli M, Bagel J. Weight loss in obese patients with psoriasis can be successfully achieved during a course of phototherapy. *J Eur Acad Dermatol Venereol*. 2012;26(12):1582-1584.

29. Yousefzadeh H, Mahmoudi M, Banihashemi M, Rastin M, Azad FJ. Investigation of dietary supplements prevalence as complementary therapy: comparison between hospitalized psoriasis patients and non-psoriasis patients, correlation with disease severity and quality of life. *Complement Ther Med*. 2017;33:65-71.

30. Gupta AK, Ellis CN, Tellner DC, Anderson TF, Voorhees JJ. Double-blind, placebo-controlled study to evaluate the efficacy of fish oil and low-dose UVB in the treatment of psoriasis. *Br J Dermatol*. 1989;120(6):801-807.

31. Logan AC. Omega-3, omega-6 and psoriasis: a different view. *Int J Dermatol*. 2005;44(6):527-528.

32. Lands WE. Biochemistry and physiology of n-3 fatty acids. *FASEB J*. 1992;6(8):2530-2536.

33. Madland TM, Björkkjaer T, Brunborg LA, Fröyland L, Berstad A, Brun JG. Subjective improvement in patients with psoriatic arthritis after short-term oral treatment with seal oil: a pilot study with double blind comparison to soy oil. *J Rheumatol*. 2006;33(2):307-310.

34. Veale DJ, Torley HI, Richards IM, et al. A double-blind placebo controlled trial of Efamol Marine on skin and joint symptoms of psoriatic arthritis. *Br J Rheumatol*. 1994;33(10):954-958.

35. Søyland E, Funk J, Rajka G, et al. Effect of dietary supplementation with very-long-chain n-3 fatty acids in patients with psoriasis. *N Engl J Med*. 1993;328(25):1812-1816.

36. Strong AMM, Hamill E. The effect of combined fish oil and evening primrose oil (Efamol Marine) on the remission phase of psoriasis: a 7-month double-blind randomized placebo-controlled trial. *J Dermatolog Treat*. 1993;4(1):33-36.

37. Gupta AK, Ellis CN, Goldfarb MT, Hamilton TA, Voorhees JJ. The role of fish oil in psoriasis: a randomized, double-blind, placebo-controlled study to evaluate the effect of fish oil and topical corticosteroid therapy in psoriasis. *Int J Dermatol*. 1990;29(8):591-595.

38. Bjørneboe A, Smith AK, Bjørneboe GE, Thune PO, Drevon CA. Effect of dietary supplementation with n-3 fatty acids on clinical manifestations of psoriasis. *Br J Dermatol*. 1988;118(1):77-83.

39. Bittiner SB, Tucker WF, Cartwright I, Bleehen SS. A double-blind, randomised, placebo-controlled trial of fish oil in psoriasis. *Lancet*. 1988;1(8582):378-380.

40. Mayser P, Mrowietz U, Arenberger P, et al. Omega-3 fatty acid-based lipid infusion in patients with chronic plaque psoriasis: results of a double-blind, randomized, placebo-controlled, multicenter trial. *J Am Acad Dermatol*. 1998;38(4):539-547.

41. Grimminger F, Mayser P, Papavassilis C, et al. A double-blind, randomized, placebo-controlled trial of n-3 fatty acid based lipid infusion in acute, extended guttate psoriasis: rapid improvement of clinical manifestations and changes in neutrophil leukotriene profile. *Clin Investig*. 1993;71(8):634-643.

42. Orgaz-Molina J, Buendía-Eisman A, Arrabal-Polo MA, Ruiz JC, Arias-Santiago S. Deficiency of serum concentration of 25-hydroxyvitamin D in psoriatic patients: a case-control study. *J Am Acad Dermatol*. 2012;67(5):931-938.

43. Ricceri F, Pescitelli L, Tripo L, Prignano F. Deficiency of serum concentration of 25-hydroxyvitamin D correlates with severity of disease in chronic plaque psoriasis. *J Am Acad Dermatol*. 2013;68(3):511-512.

44. Siddiqui MA, Al-Khawajah MM. Vitamin D3 and psoriasis: a randomized double-blind placebo-controlled study. *J Dermatolog Treat*. 1990;1(5):243-245.

45. Gaál J, Lakos G, Szodoray P, et al. Immunological and clinical effects of alphacalcidol in patients with psoriatic arthropathy: results of an open, follow-up pilot study. *Acta Derm Venereol*. 2009;89(2):140-144.

46. Finamor DC, Sinigaglia-Coimbra R, Neves LC, et al. A pilot study assessing the effect of prolonged administration of high daily doses of vitamin D on the clinical course of vitiligo and psoriasis. *Dermatoendocrinol*. 2013;5(1):222-234.

47. Perez A, Raab R, Chen TC, Turner A, Holick MF. Safety and efficacy of oral calcitriol (1,25-dihydroxyvitamin D3) for the treatment of psoriasis. *Br J Dermatol*. 1996;134(6):1070-1078.

48. Smith EL, Pincus SH, Donovan L, Holick MF. A novel approach for the evaluation and treatment of psoriasis: oral or topical use of 1,25-dihydroxyvitamin D3 can be a safe and effective therapy for psoriasis. *J Am Acad Dermatol*. 1988;19(3):516-528.

49. Takamoto S, Onishi T, Morimoto S, et al. Effect of 1 alpha-hydroxycholecalciferol on psoriasis vulgaris: a pilot study. *Calcif Tissue Int*. 1986;39(6):360-364.

50. Morimoto S, Yoshikawa K, Kozuka T, et al. Treatment of psoriasis vulgaris by oral administration of 1 alpha-hydroxyvitamin D3—open-design study. *Calcif Tissue Int*. 1986;39(3):209-212.

51. Merola JF, Han J, Li T, Qureshi AA. No association between vitamin D intake and incident psoriasis among US women. *Arch Dermatol Res*. 2014;306(3):305-307.

52. Harvima RJ, Jägerroos H, Kajander EO, et al. Screening of effects of selenomethionine-enriched yeast supplementation on various immunological and chemical parameters of skin and blood in psoriatic patients. *Acta Derm Venereol*. 1993;73(2):88-91.

53. Serwin AB, Waşowicz W, Gromadzińska J, Chodynicka B. Selenium status in psoriasis and its relationship with alcohol consumption. *Biol Trace Elem Res*. 2002;89(2):127-137.

54. Kharaeva Z, Gostova E, De Luca C, Raskovic D, Korkina L. Clinical and biochemical effects of coenzyme Q(10), vitamin E, and selenium supplementation to psoriasis patients. *Nutrition*. 2009;25(3):295-302.

55. Serwin AB, Mysliwiec H, Hukalowicz K, Porebski P, Borawska M, Chodynicka B. Soluble tumor necrosis factor-alpha receptor type 1 during selenium supplementation in psoriasis patients. *Nutrition*. 2003;19(10):847-850.

56. Serwin AB, Wasowicz W, Chodynicka B. Selenium supplementation, soluble tumor necrosis factor-alpha receptor type 1, and C-reactive protein during psoriasis therapy with narrowband ultraviolet B. *Nutrition*. 2006;22(9):860-864.

57. Ruedemann R Jr. Treatment of psoriasis with large doses of vitamin B12, 1,100 micrograms per cubic centimeter; preliminary clinical report. *AMA Arch Derm Syphilol*. 1954;69(6):738-739.

58. Baker H, Comaish JS. Is vitamin B12 of value in psoriasis? *Br Med J*. 1962;2(5321):1729-1730.

59. Johnson JA, Ma C, Kanada KN, Armstrong AW. Diet and nutrition in psoriasis: analysis of the National Health and Nutrition Examination Survey (NHANES) in the United States. *J Eur Acad Dermatol Venereol*. 2014;28(3):327-332.

60. 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.

61. Barrea L, Macchia PE, Tarantino G, et al. Nutrition: a key environmental dietary factor in clinical severity and cardio-metabolic risk in psoriatic male patients evaluated by 7-day food-frequency questionnaire. *J Transl Med*. 2015;13:303.

62. Di Minno MN, Peluso R, Iervolino S, Russolillo A, Lupoli R, Scarpa R; CaRRDs Study Group. Weight loss and achievement of minimal disease activity in patients with psoriatic arthritis starting treatment with tumour necrosis factor α blockers. *Ann Rheum Dis*. 2014;73(6):1157-1162.

63. Huckins D, Felson DT, Holick M. Treatment of psoriatic arthritis with oral 1,25-dihydroxyvitamin D3: a pilot study. *Arthritis Rheum*. 1990;33(11):1723-1727.

64. Kristensen S, Schmidt EB, Schlemmer A, et al. The effect of marine n-3 polyunsaturated fatty acids on cardiac autonomic and hemodynamic function in patients with psoriatic arthritis: a randomised, double-blind, placebo-controlled trial. *Lipids Health Dis*. 2016;15(1):216.

65. 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.

66. Danno K, Sugie N. Combination therapy with low-dose etretinate and eicosapentaenoic acid for psoriasis vulgaris. *J Dermatol*. 1998;25(11):703-705.

67. Balbás GM, Regaña MS, Millet PU. Study on the use of omega-3 fatty acids as a therapeutic supplement in treatment of psoriasis. *Clin Cosmet Investig Dermatol*. 2011;4:73-77.

68. Lassus A, Dahlgren AL, Halpern MJ, Santalahti J, Happonen HP. Effects of dietary supplementation with polyunsaturated ethyl ester lipids (Angiosan) in patients with psoriasis and psoriatic arthritis. *J Int Med Res*. 1990;18(1):68-73.

69. Kragballe K, Fogh K. A low-fat diet supplemented with dietary fish oil (Max-EPA) results in improvement of psoriasis and in formation of leukotriene B5. *Acta Derm Venereol*. 1989;69(1):23-28.

70. Kettler AH, Baughn RE, Orengo IF, Black H, Wolf JE Jr. The effect of dietary fish oil supplementation on psoriasis: improvement in a patient with pustular psoriasis. *J Am Acad Dermatol*. 1988;18(6):1267-1273.

71. Kragballe K. Dietary supplementation with a combination of n-3 and n-6 fatty acids (super gamma-oil marine) improves psoriasis. *Acta Derm Venereol*. 1989;69(3):265-268.

72. Ziboh VA, Cohen KA, Ellis CN, et al. Effects of dietary supplementation of fish oil on neutrophil and epidermal fatty acids: modulation of clinical course of psoriatic subjects. *Arch Dermatol*. 1986;122(11):1277-1282.

73. Maurice PD, Allen BR, Barkley AS, Cockbill SR, Stammers J, Bather PC. The effects of dietary supplementation with fish oil in patients with psoriasis. *Br J Dermatol*. 1987;117(5):599-606.

74. Kojima T, Terano T, Tanabe E, Okamoto S, Tamura Y, Yoshida S. Effect of highly purified eicosapentaenoic acid on psoriasis. *J Am Acad Dermatol*. 1989;21(1):150-151.

75. el-Azhary RA, Peters MS, Pittelkow MR, Kao PC, Muller SA. Efficacy of vitamin D3 derivatives in the treatment of psoriasis vulgaris: a preliminary report. *Mayo Clin Proc*. 1993;68(9):835-841.