

REVIEW ARTICLE

The role of lifestyle and nutrition in psoriasis: Current status of knowledge and interventions

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Email: cldermct@gmail.com**Abstract**

Extrinsic environmental factors, including patient lifestyle (alcohol intake, smoking, stress, sleep disturbances, and sedentary habit), diet and single nutrients intake may affect psoriasis clinical presentation, severity, and course. All English language articles dealing with psoriasis and lifestyle factors or diet gathered by an extensive PubMed search were carefully examined in order to explore their impact on the disease. Current authoritative knowledge confirms that low-calories, Mediterranean, and protein restricted/vegetarian diets may be beneficial. Psoriatic patients are also recommended to engage regular physical activity, to avoid alcohol intake and to consume fish rich in omega-3 polyunsaturated fatty acids, as well as fruit and vegetables. Prebiotics and probiotics may also provide potential benefit, whereas vitamin D supplementation and gluten-free diet are useful in selected cases only. Changing of dietary and lifestyle habits alone does not replace conventional treatment, but must be considered as an adjuvant. Physicians may play a crucial role, by adequately acknowledging psoriatic patients on the advantages of proper lifestyle and diet habits as well as providing clues to reliable sources of dietary advice.

KEYWORDS

diet, food, lifestyle, nutrients, nutrition, psoriasis

1 | INTRODUCTION

Psoriasis is a multifactorial disease with a complex etiopathogenesis, involving genetic predisposition, as well as immunological and environmental factors. Indeed, lifestyle may affect the clinical presentation, severity, and course of the disease. Psoriasis has recently been regarded as a systemic inflammatory disorder associated with serious comorbidities, such as metabolic syndrome and cardiovascular diseases, all sharing a common underlying chronic inflammatory basis. Data from epidemiological and experimental studies indicate that increased body mass index (BMI) may act as a psoriasis trigger, and waist circumference positively correlates with the disease.¹ These observations suggest that a correct

lifestyle and nutrition may play an important role in the course and outcome of psoriasis.²⁻⁴ Herein, we review current authoritative knowledge on lifestyle and nutrition in psoriasis.

2 | MATERIALS AND METHODS

This study was performed using on the PubMed database (<https://ncbi.nlm.nih.gov/PubMed> last accessed on 30 December 2021) the keys related to "psoriasis" [All Fields] AND "diet" [All Fields] (or "nutrition" [All Fields]), "psoriasis" [All Fields] AND "lifestyle" [All Fields], "psoriasis" [All Fields] AND "alcohol" [All Fields], "psoriasis"

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TABLE 1 Overview of lifestyle and nutrition factors, including specific diets and single nutrients.

Lifestyle	Nutrition
Alcohol	Low calories diet
Smoking	Mediterranean diet
Stress and sleep disturbances	Protein restricted and vegetarian diet
Sedentary habit	Single nutrients
	Omega-3 polyunsaturated fatty acids
	Vitamin B ₁₂
	Vitamin D
	Tryptophan
	Selenium
	Curcumin
	Prebiotics and probiotics
	Caffeine
	Gluten

[All Fields] AND “smoking” [All Fields], “psoriasis” [All Fields] AND “sleep disorders” [All Fields], “psoriasis” [All Fields] AND “physical activity” [All Fields] (or “sport” [All Fields] or “sedentary habit” [All Fields]). According to PRISMA guidelines, all English language articles whose title and/or abstract suggested the association between psoriasis and lifestyle or nutrition (Table 1), including studies on human population as well as research papers considered relevant to the topic, were selected and read entirely. Their references were also examined in order to identify further studies that could be considered in the review, based on the same criteria. Studies identified from the title, abstract, or full text as performed on animals or cells were considered only if relevant to the topic in question. Given the heterogeneity of the studies examined, the decision to carry a narrative review of the literature, rather than a systematic one, was made. The review of available literature was aimed to define the relationship between lifestyle or nutrition factors and psoriasis (current status of knowledge), as well as prospective trials aimed to assess interventional lifestyle and nutritional strategies useful to effectively target psoriasis management (intervention).

3 | RESULTS

3.1 | Current status of knowledge: psoriasis and lifestyle

3.1.1 | Alcohol

The prevalence of plaque psoriasis is increased among alcohol addict patients. In addition, alcohol intake is the trigger for initiating, perpetuate the course and increase its psoriasis severity.⁵ At cytokine level, chronic alcohol consumption increases inflammatory markers (such as

tumor necrosis factor alpha [TNF- α], soluble TNF receptor and pro-inflammatory cytokines), CD80 and CD86 expression, and T-cell activation. It also upregulates genes promoting lymphocyte and keratinocyte proliferation,² the latter likely to be related to the skin barrier disruption resulting from ethanol excretion by exocrine glands.² Occasional alcohol consumption may also have an immunosuppressive role, facilitating skin streptococcal and staphylococcal infections that may act as psoriasis triggers. Nevertheless, drinking worsens therapeutic compliance, and chronic alcohol consumption decreases the efficacy and increases the toxicity of systemic antipsoriatic treatments.³

3.1.2 | Interventions

No interventional trials on lifestyle changes dealing with alcohol withdrawal are available so far.

3.2 | Smoking

Studies suggest that smoking is an independent risk factor for psoriasis, either inducing the onset or worsening pre-existing disease. Moreover, smokers with psoriasis are less likely to stop smoking than non-psoriatic smokers.⁶ Pathogenetically, the link is the production of free radicals, which have a deleterious effect through their activation of the mitogen-activated protein kinase (MAPK), nuclear factor-kappa (NF- κ) and Janus kinases-signal transducer and activator of transcription (JAK-STAT) pathways,⁶ the formation of reactive oxygen species (ROS), and the decreased protective antioxidant gene expression, with consequent skin damage.⁷ Nicotine also enhances interleukin-12 (IL-12) production in dendritic cells and activates innate immune system. Finally, smokers psoriatic patients exhibit poor treatment response and reduced adherence to therapy.^{6,7}

3.2.1 | Interventions

No interventional trials on lifestyle changes dealing with smoking cessation are available so far.

3.3 | Stress and sleep disturbances

Psychosocial stress represents a risk factor for psoriasis. The pathogenetic correlation between stress and psoriasis onset and worsening is likely linked to a hypothalamic-pituitary-adrenal axis dysfunction. Stress-responsive psoriatic patients have been found to show higher psoriasis area severity index (PASI) compared to the lesser-responsive counterparts.³ On the other hand, psoriasis itself is a cause of considerable psychosocial morbidity, since it dramatically affects the patient self-image with consequent social avoidance, impairment of interpersonal relationships, and detrimental impact on sexual life and work activity.⁸ Psoriatic patients have, independently of disease severity,

high rates of anxiety and depression and are at risk of suicidal behavior. In addition, they often experience sleep disturbances related to skin symptoms, such as pruritus and pain.⁸

3.3.1 | Interventions

No interventional trials on lifestyle changes dealing with stress coping and sleep disturbance amelioration are available so far.

3.4 | Sedentary habit

It has been suggested that sedentary habit may modify the psoriasis natural course, as well as the incidence of metabolic comorbidities, through epigenomic, metabolic, proinflammatory, and psycho-emotional effects.^{9,10} Psoriasis has been found to be significantly less common in sportive subjects compared to controls. In addition, the number of subjects performing sport activities is significantly lower among psoriatic patients.⁹

3.4.1 | Interventions

Physical activity is reported to be positively associated with improvement of psoriasis, as well as with that of cardiovascular, metabolic, and psychological comorbidities.⁹ A 2019 Cochrane review confirmed that physical activity, along with weight loss, should be considered a standard accompanying treatment, by reducing the oxidative stress and PASI score, especially in over-weight and obese patients receiving biologics or oral/traditional treatments.¹¹ Moreover, a randomized controlled trial (RCT) on 303 overweight or obese patients with moderate-to-severe chronic plaque psoriasis showed that increased physical activity consisting in aerobic exercise for at least 40 min three times a week, associated with a 20-week dietetic intervention, was able to effectively reduce psoriasis severity compared to simple informative counseling about the utility of weight loss for clinical control of psoriatic disease.¹²

3.5 | Current status of knowledge: psoriasis and nutrition

Nutrition is a complex combination of dietary regimens including various types of food and different single nutrients. It is therefore a challenge to discern the effects of single nutrients from that of another when dietary regimens are considered as a whole. Data in the literature are discordant, and most studies on nutrition and psoriasis often show limitations, mainly related to small sample size, lack of case-control trials, short study duration.¹³

The association between psoriasis and metabolic syndrome (including abdominal obesity, insulin resistance, type 2 diabetes, atherogenic dyslipidemia, hypertension, and nonalcoholic fatty liver

disease) is confirmed by several studies, with a prevalence ranging from 20% to 50%.^{7,14,15} In addition, psoriatic patients have >50% increased odds of being obese compared to general population.⁷ Also, psoriasis itself could contribute to weight gain, partially because of social isolation, unhealthy dietary habits, reduction of physical activity and, occasionally, therapy with some biologics. The pathophysiological mechanisms linking psoriasis and obesity involve inflammatory pathways and genetic background. In particular the increased visceral fat has a prominent role, since it acts as an endocrine organ with adipocytes releasing a number of pro-inflammatory cytokines, such as TNF- α , IL-1, IL-6, IL-8, IL-23, and IL-17 (whose levels are increased in obese compared to control subjects) and adipocytokines, such as leptin and resistin. This results in chronic Th1 and Th17-mediated immune dysregulation and low-grade inflammation that contributes to the development of insulin resistance, hyperglycemia, dyslipidemia, vascular dysfunction, and non-alcoholic fatty liver disease. Hyperinsulinemia promotes psoriasis worsening or development by facilitating chronic inflammation and angiogenesis, whereas increased TNF- α and IL-6 levels promote epidermal hyperplasia; leptin is also known to induce keratinocyte proliferation. Finally, cytokine dysregulation and inflammation downregulate insulin signaling, leading to insulin resistance and to further increased adipokine expression and obesity. In this way, a vicious circle is established.^{14,16} A proinflammatory diet is generally associated with higher levels of inflammatory markers, as well as higher psoriasis incidence.¹⁷ Among the different dietary regimens, the most credited are low calories, Mediterranean and protein restricted and vegetarian diets.¹⁸ Low calories diet in psoriasis may reverse the vicious circle that links obesity with the release of inflammatory markers and diseases worsening, by promoting weight loss and reducing oxidative stress.¹⁴ Studies performed in the cardiovascular setting have demonstrated that the Mediterranean diet is able to induce in psoriatic patients a reduction in IL-6, vascular adhesion molecule-1 (VCAM-1), intercellular adhesion molecule-1 (ICAM-1) and low density lipoproteins (LDL), and an increase in high density lipoproteins (HDL), all potentially beneficial changes that may reasonably interfere with the metabolic vicious circle causing psoriasis worsening.⁷ However studies based on self-administered questionnaires, such as PREvención con Dieta MEDiterránea (PREDIMED) and MEDiterranean-LITErature (MEDI-LITE), showed a lower adherence to Mediterranean diet among psoriatic patients compared to controls.^{3,7,17} A protein restricted diet may likely suppress systemic inflammation and inhibit angiogenesis, thereby creating an environment in which psoriasis is less easily triggered and is more responsive to therapy. On the other hand consumption of animal proteins is also known to increase serum levels of proinflammatory cytokines (IL-6 and TNF) and markers of inflammation such as C reactive protein (CRP) due to the presence of N-glycolylneuraminic acid (Neu5gc), a potential pro-inflammatory and immunogenic monosaccharide.¹⁸ Finally, a vegetarian diet may downsize the formation of inflammatory eicosanoids detrimental to psoriatic patients through a reduced arachidonic acid (AA) intake. It may also increase cortisol release due to a higher potassium intake,

producing effects on psoriatic plaques similar to those achieved by exogenous synthetic glucocorticoid administration.¹⁹

3.5.1 | Interventions

Low calories diet

Interventional RCTs have unequivocally confirmed the capability of low-calories dietary regimens,³ including the ketogenic diet,⁷ to speed psoriasis improvement. In addition a low calories dietary regimen has been demonstrated to improve the response to systemic treatment with low-dose cyclosporine,²⁰ biologics or phototherapy.²¹

A low calories diet resulting in weight loss leads to an improved PASI score, dermatology life quality index (DLQI), and serum lipid levels, as demonstrated by a prospective RCTs in which psoriatic patients with BMI >27 on a low-energy diet (800–1000 kcal/day) for 8 weeks experienced significant clinical improvement, with recurrences after the reintroduction of a normal diet.²⁰ Moreover, aggressive dietary programs consisting of a ketogenic diet, followed by a balanced, hypocaloric, Mediterranean-like diet, have shown promising results, with a metabolic evaluation demonstrating the subsequent adjustment of clinical and biochemical parameters, including reduction of disease severity and of inflammatory markers (such as IL-2 and IL-1 β).^{7,18} Interestingly, Muslim patients have a statistically significant change in PASI and Disease Activity index for Psoriatic Arthritis (DAPSA) score before and after the Ramadan, and this change was found to be less prominent in patients treated with topical therapy compared to those under systemic treatment. During Ramadan patients tend to take systemic drugs after nightfall, it has been assumed that this night schedule may have beneficial effects compared to morning administration, maximizing the effects of antipsoriatic therapy.^{22,23} Obese patients undergoing a low-calories diet, enriched in omega-3 polyunsaturated fatty acids (ω -3 PUFAs) and poor in ω -6 PUFAs, showed improvement of metabolic markers along with a better clinical outcome. These results were also paralleled by a DLQI improvement.²¹ In a RCT, obese psoriatic patients receiving anti TNF- α treatment on a low-calories diet (\leq 1000 kcal per day for 8 weeks) showed an average 84% improvement in mean PASI score compared to 69% of the control group.²

Mediterranean diet

Which nutrients (among others, fish oil and ω -3 PUFAs, vitamin B₁₂, D, A, inositol, and zinc) in Mediterranean diet may play a protective role in psoriasis is difficult to establish. This diet is rich of a range of anti-inflammatory substances such as antioxidants, polyphenols and monounsaturated fats, in the form of extra virgin olive oil (EVOO) which is an important source of monounsaturated fatty acids (MUFAs), that are associated to reduction of coronary heart disease risk and modification of the immune and inflammatory responses. In a cross-sectional case-control observational study, male psoriatic patients compared to a control group and evaluated by a 7-day food-frequency questionnaire had a higher consumption of simple carbohydrates, total fat and n-6/n-3 PUFAs ratio, with a lower intake of protein, complex carbohydrates, n-3 PUFA, and fiber, demonstrating an inverse correlation between psoriasis severity and adherence to the Mediterranean diet.³ A lower psoriasis severity has been correlated to a higher intake of MUFAs

contained in EVOO, a healthier dietary fat compared to saturated fatty acids. Moreover, MUFAs have been used as independent predictor of PASI score, since EVOO contains oleocanthal, a natural anti-inflammatory component whose consumption correlates with lower levels of CRP. Nevertheless, vegetables, that represent a substantial component of Mediterranean diet, are an important source of phytochemicals that reduce cholesterol serum levels and, subsequently, the cardiovascular risk in psoriatic patients.³

Protein restricted and vegetarian diet

In the late 1960s a significant improvement in psoriatic patients placed on low-protein and low-tryptophan diets was reported. Other studies then suggested that protein-restricted diets (vegetarian-, low-energy- low-protein- and taurine-restricted diet) might also improve the disease, in contrast to the “Western diet” rich in processed meat, saturated fats, and sugar.³ A strict vegan diet may considerably enhance treatment response to phototherapy, reducing the number of narrow-band UVB (NB-UVB) sessions needed to achieve psoriatic clearance, likely due to a lower minimum erythemal dose (MED) resulting from the higher intake of furocoumarin-rich food. On this regard, the possible higher risk of adverse events, such as severe erythema, should also be considered.²⁰

3.6 | Single nutrients

3.6.1 | Omega-3 polyunsaturated fatty acids (ω -3 PUFAs)

The ω -3 PUFAs, such as eicosapentanoic acid (EPA), docosahexaenoic acid (DHA), and eicosatetraenoic acid (ETA), are mainly found in oily fish, including mackerel, sardines, herrings, and salmon, where they represent 30%–50% of fatty acids. In psoriatic lesions AA is increased up to 20-fold compared to uninvolved epidermis. Phospholipases A2 and C are required for the release of AA from epidermal lipid pools, and these enzymes are significantly increased in psoriatic skin. The ω -3 PUFAs are bioactive analogues of AA, so their external supplementation causes a metabolic replacement of AA, resulting in a reduced production of IL-1 and TNF- α by monocytes and in a reduced expression of endothelial adhesion molecules such as VCAM-1, E-selectin, and ICAM-1. These changes result in an inhibition of leukocytes ability to migrate across the vascular endothelium and in a mitigation of the inflammatory response.^{21,24} A blockage of Th17 pathway induced by ω -3 PUFAs has also been reported.²⁵

3.6.2 | Interventions

Oral supplementation with EPA and DHA induces a mild to moderate clinical improvement in psoriatic skin, associated with an increase of ω -3 PUFAs levels in serum lipids and a reduction in leukotriene B₄, along with an increase of the less potent mediator leukotriene B₅. A meta-analysis including 10 studies with a total of 560 participants showed that oral supplementation with ω -3 PUFAs as monotherapy

(>1800 mg/day) was correlated with a better psoriasis outcome in term of reduction of PASI score, erythema, and scaling.²⁴ Recently, a beneficial role of ETA dietary intake has also been suggested based on a phone survey including recall dietary interviews from 15,733 participants.²⁶ However, the effect on itching, desquamation, infiltration, and percent total body surface area (BSA) remains to be precisely assessed, and therefore the role of ω -3 PUFAs supplementation remains controversial.²⁴ Whether ETA supplementation may be more beneficial than that with EPA and DHA also remains to be defined.²⁶

Vitamin B12

It is mainly found in shellfish, liver, fortified cereals, and red meat. It plays a role in nucleic acid synthesis, likely influencing psoriasis by its immunomodulatory effects on T lymphocytes and cytokines. Association of vitamin B12 deficiency with psoriasis has been reported. However, a recent meta-analysis and systematic review including 24 studies has shown no difference between serum basal level of vitamin B12 in psoriatic patients and controls.²⁷

3.6.3 | Interventions

There is discordance on the beneficial role of supplementation of vitamin B12 in psoriatic patients,² and no well-designed trials have recently been performed.

Vitamin D

It is found in cod liver, oil, oily fish, mushrooms, and fortified cereals. A great quota of vitamin D is synthesized by the skin after exposure to UVB radiation. Successively, it undergoes hydroxylation by the liver, forming 25-hydroxyvitamin D or 25(OH)D₃, an inert form of the vitamin, and another hydroxylation in the kidney, where the biologically active form of vitamin D, calcitriol or 1,25(OH)D₃, is synthesized. Vitamin D, binding the vitamin D receptor, regulates keratinocytes differentiation and proliferation, the inflammatory response, and the cutaneous immune system function. It also increases the synthesis of keratins (K1 and K10), involucrin, transglutaminase, loricrin, and filaggrin in the stratum spinosum and regulates the synthesis of glycosylceramides essential for epidermal skin barrier function. These actions are due to the ability of vitamin D to regulate intracellular calcium level, through induction of the calcium receptor, and the phospholipase C enzymes. A role of vitamin D in psoriasis has been suggested, although the mechanism of action is not fully elucidated. A recent meta-analysis evaluating D₃ serum levels in 571 psoriatic patients found lower serum levels compared to control subjects, with a significant negative correlation with psoriasis severity.²⁸

3.6.4 | Interventions

The beneficial effects of dietary intake of vitamin D or oral vitamin D supplementation is still debated, with some studies reporting improvement and others, including double blind RCTs, no significant response.²⁹ Therefore, current recommendations do not support its

administration in psoriatic individuals with normal vitamin D serum levels.³⁰

Tryptophan

It is an essential aminoacid contained principally in red meat, dairy products, shellfish, and soya. Human body is unable to synthesize tryptophan so it is necessary the assumption from the outside. Tryptophan effects on psoriasis remain controversial, although the conflicting results may be related to the presence of two different enzymes, namely L-kynureninase (KYNU) and indoleamine 2,3 dioxygenase (IDO), that play a pro-inflammatory and immunosuppressive role, respectively. In particular, KYNU has been reported to correlate with disease severity and inflammation, its concentration in psoriatic skin being progressively reduced after treatment.²¹ Another possible hypothesis may be related to serotonin, an important neurotransmitter synthesized from tryptophan that enhances psoriasis development through the activation of keratinocytes proliferation, epidermal turnover, and T cell function, as confirmed by its higher epidermal expression in psoriatic skin than controls.³¹

3.6.5 | Interventions

The Turkish diet is rich in this aminoacid and some studies confirm its beneficial role in psoriatic patients, with exacerbation of the disease at reinstatement of the normal diet, while others suggest a low-tryptophan diet to be beneficial.²¹ Intake of serotonin reuptake inhibitor antidepressants has been found to decrease the need of systemic treatment in psoriatic patients. Whether this effect is related to interference with serotonin signaling pathways or to a direct biological anti-inflammatory effect, or to other mechanisms is unknown.³¹

Selenium

Good alimentary sources of selenium are Brazil nuts, sunflowers seeds and fish (tuna, halibut, sardines, flounder, salmon). Selenium food content depends on the concentration of selenium in the soil where the crops are grown. It is essential for the normal functioning of glutathione peroxidase, an important antioxidant enzyme with protective effects against UV induced oxidative DNA damage and harmful environmental factors in skin cells. In addition, selenium enhances immune function and has anti-proliferative properties through an inhibitory effect on DNA synthesis.³²

3.6.6 | Interventions

Although several pilot studies and open trials showed that selenium levels may be particularly low in patients with prolonged disease duration, the role of selenium in psoriasis remains controversial. Currently, there are no studies available assessing the role of selenium individually rather than in association with other nutrients. Therefore, no reliable conclusions may be drawn regarding its role in promoting psoriasis improvement.^{3,21,33}

Curcumin

It is the primary and most active constituent of turmeric, a spice derived from the rhizome of the plant *Curcuma longa* that has been used for centuries by the Middle East and Asian populations as an ingredient of the famous curry sauce, as a natural dye, and as a therapeutic agent. Its anti-inflammatory properties are linked to its ability to block both a serine/threonine-specific protein kinase (PhK) and TNF- α . It has also been demonstrated in vitro to reduce interferon gamma (IFN γ), IL-17 and IL-22 cytokine production in psoriasis, down-regulating in a dose-dependent manner the immune response.³⁴

3.6.7 | Interventions

Interesting results, paralleled by a significant reduction of IL-22 serum levels, were obtained in psoriatic patients treated with topical steroids plus oral curcumin (2 g/day) compared to those taking oral placebo.³ However, further well-controlled clinical trials are required to confirm these findings.

Prebiotics and probiotics

The gastrointestinal microbiota (GM) plays an important role in many different body districts. It specifically induces the development of the immune system, produces secondary bile acids, facilitates vitamins synthesis, and maintains the bacterial homeostasis avoiding intestinal infections.³⁵ An alteration of the GM, namely dysbiosis, may impair these functions. Many studies deal particularly with lipopolysaccharide (LPS) and psoriasis. LPS is an endotoxin belonging to Gram-negative bacteria, that bind receptors like pathogen-associated molecular patterns (PAMPs) and Toll-like receptors (TLRs), stimulating production of pro-inflammatory cytokines and developing a chronic low-grade inflammatory state, able to promote psoriasis development.³ Elevated levels of LPS-binding protein have been reported in psoriatic patients, probably due to a diet based on excessive consumption of fat.³ Therefore, the use of probiotics and prebiotics may have a beneficial role in psoriasis by reducing LPS production. In addition, probiotics may act by enhancing carbohydrate digestion by gut microbiota, with consequent increase of the production of short-chain fatty acids, which in turn promote T cell differentiation into regulatory T (Treg) cells, leading to a decrease of the levels of IL-23/Th17 axis-related inflammatory cytokines.³⁶⁻³⁸ Food containing prebiotics useful to psoriatic patients include chicory root, dandelion greens, artichoke, onion, garlic, leeks, and asparagus. Another possible option to control the intestinal dysbiosis is the low-Fermentable Oligosaccharides, Disaccharides, Monosaccharides and Polyols (FODMAPs) diet, a diet with fermentable foodstuffs (onion, garlic, wheat, legumes) restriction able to reduce gas production in the gut.³

3.6.8 | Interventions

Multiple observations point to a promising role of probiotics and prebiotics.³⁹ Bile acid deficiency is reported to correlate with LPS

translocation in the bloodstream and with psoriasis. A study of 800 psoriasis patients divided in two groups, treated for 1–8 weeks with oral bile acid (dehydrocholic acid) supplementation or with conventional treatment, showed that 57.9% of patients treated with bile acid had no relapse after 2 years, compared to 6% of patients from the other group.³ Another RCT showed a reduction of the PASI of up to 75% in the probiotic group (vs. 41.9% in the placebo group) and a lower risk of relapse at a 6-month follow up.³⁹

Caffeine

It is an alkaloid (methylxanthine) and an important constituent of coffee whose effects on the immune system are still unclear. It has antioxidant properties that may help quelling inflammation. It may also halt psoriasis progression by increasing intracellular cAMP levels that are found to be reduced in cutaneous leukocytes of psoriatic patients. On the other hand, caffeine seems to have pro-inflammatory effects when administered in rats in the presence of an acute inflammatory process, increasing tissue damage as evidenced by increased mRNA levels of TNF- α , TNF- β , lymphotoxin- β , IL-6, and IFN- γ in the spleen and increased IFN- γ in peripheral blood. Its mechanism of action may also be related to its ability to bind adenosine receptors, due to a structural similarity.⁴⁰ Clinical studies have also demonstrated conflicting results concerning the role of coffee in autoimmune diseases. On one hand, coffee increases the risk of developing some diseases, such as rheumatoid arthritis and type 1 diabetes mellitus. In contrast, it seems to exert a protective role against multiple sclerosis, primary sclerosing cholangitis as well as psoriasis.⁴¹

3.6.9 | Interventions

In a survey on 64 patients with psoriasis and psoriatic arthritis evaluating the consumption of coffee (low intake, <120 mg/day; moderate intake, 120–180 mg/day; high intake, >180 mg/day) during therapy with methotrexate, caffeine consumption has not been found to affect methotrexate dosage requirements in psoriatic patients.⁴⁰ In a prospective cohort study, no association between coffee, caffeine intake and risk of psoriasis was demonstrated.⁴² However, a more recent investigation identified a reduced PASI score in coffee consumers.⁴³

Gluten

A clear association between celiac disease (CD) and psoriasis has not been established yet and remains still controversial. The link between psoriasis and celiac disease may be related to the sharing of the same genetic susceptibility, namely, an involvement of some genes (TNFAIP3, RUNX3, ELMO1, ZMIZ1, ETS1, SH2B3, SOCS1, and UBE2L3) implicated in the regulation of innate and adaptive Th1 immune responses. An additional hypothesis considers a role of altered intestinal permeability present in both conditions and resulting in a reduced vitamin D uptake.⁷ An evaluation of antigliadin antibodies (AGA) levels in 300 subjects (100 with psoriasis, 100 with both psoriasis and psoriatic arthritis, and 100 healthy controls) found no

difference in the presence of abnormal AGA and no correlations between AGA positivity and psoriasis severity, joint involvement, or age of onset of psoriasis or arthritis.⁷ However, in a Swedish cohort study comparing individuals with CD to controls on their propensity for psoriasis, patients with CD had a statistically significant increased risk of psoriasis (absolute risk of 135/100.000 person-years) both before and after CD diagnosis, with 42% of all psoriasis attributed to the underlying CD.⁴⁴ A large meta-analysis confirmed a positive correlation between psoriasis, CD, and CD markers, showing that psoriatic patients have 2.2-fold risk of having CD and 2.4-fold increased risk of elevated levels of AGA compared to controls. A positive correlation between CD antibody positivity and severity of psoriasis or psoriatic arthritis, that not necessarily corresponded to a biopsy-confirmed diagnosis of CD, was also observed, suggesting that psoriasis may be associated with gluten sensitivity but not necessarily with gluten enteropathy.⁴⁵ These conclusions concur with those from another recent systematic review⁴¹ and with those from a study⁴⁶ in which the prevalence of AGA antibody was found to be significantly higher among psoriatic patients without CD or non-celiac gluten sensitivity. In accordance with these considerations, a gluten-free diet has been suggested to be potentially beneficial in celiac antibody positive psoriatic patients.

3.6.10 | Interventions

Studies evaluating AGA-positive and AGA-negative patients with psoriasis after a 3-month gluten free diet (GFD), followed by a resumption of the normal diet for the same time period, demonstrated a statistically significant reduction in the mean PASI score only in AGA-positive patients with elevated AGA levels. In addition, reduction in Ki67 positive cells and tissue transglutaminase-tTG expression in dermis was observed.⁴⁵ Subsequently, other studies confirmed the finding of a sustained improvement of PASI score in psoriatic patients with high IgA-AGA levels (even with a normal biopsy) on a GFD.^{45,46} Although large RCTs have not yet been performed, and a recent study evaluating 85,185 women found no association between gluten intake and incidence of psoriasis,⁴⁷ AGA testing may be useful to identify psoriatic patients likely to benefit from GFD, three-month GFD in adults with psoriasis and positive serum markers for gluten sensitivity is recommended.^{7,44}

4 | DISCUSSION AND CONCLUSIONS

Lifestyle and nutrition may play an important role in the outcome of psoriasis. Current knowledge indicates that alcohol and smoking should be avoided to prevent disease exacerbation, as well as psychosocial stress. Physical activity has a non-secondary role on the general approach of psoriasis and its associated comorbidities, and weight loss may lead to psoriasis improvement and enhance the efficacy of systemic treatments.^{7,11} Undoubtedly, a healthy lifestyle should also be based on a proper dietary program combining calories

restriction with the preferential intake of food with high antioxidant properties, such as fish rich in ω -3 PUFAs, fruits and vegetables, avoiding that with a potential proinflammatory action.^{7,11,21,48,49} Oral vitamin D supplementation and gluten-free diet are only recommended for patients with documented hypovitaminosis and increased serum AGA antibody levels, respectively,^{7,49} whereas prebiotics and probiotics, also regarded as potentially beneficial in psoriasis, represent a new developing area of investigation. Promising investigations also point to a role of dietary interventions in the modification of aberrant miRNA expression in psoriasis, since bioactive compounds may lead to psoriasis improvement by proficiently affecting microRNA expression.⁵⁰

However, despite the relevance of data about the relationship between diet and psoriasis, changing nutritional habits alone does not replace conventional treatment, but must be considered as an adjuvant. Further evidence-based controlled studies are needed to compare the effects of alcohol abstinence and smoke cessation, of different exercise programmes (e.g., walking, jogging, or other sport activities), as well as the effects of diet combined with systemic treatments, or the benefits/disadvantages obtained from other different types of diets (including Asian diets) and restrictions, specific food, or nutritional supplements.⁵¹ It is also unclear if dietary intervention with or without an exercise programme may be effectively useful in non-obese psoriatic subjects.

Both lifestyle and diet are among the most debated topics on the internet. In real life one of the most frequent concerns from patients is whether specific dietary regimens may help in improving psoriasis and therefore serve as an alternative approach.^{3,52} Interestingly, a “Google” search on “diet and dermatology”, including psoriasis, acne and eczema, showed that most of the information provided was unfounded or misleading, with only 30% educational webpages, 30% being promotional and 40% handled by self-proclaimed experts.¹³ Also, an in-depth search on YouTube found that nutrition/diet were among the most addressed topics (25%), and that nearly two-thirds of psoriasis-related videos were misleading or even dangerous (52% and 11%, respectively), and generally of low quality as unrelated to reliable medical sources.⁵³ Therefore, in order to induce proper dietary healthy habits in psoriatic patients,⁵⁴ there is a need to provide correct internet and social media information regarding diet and psoriasis, based on authoritative scientific literature, and to discredit wrong and unfounded recommendations.^{13,55}

Undoubtedly, physicians should not only adequately inform psoriatic patients regarding proper dietary habits, but they should also provide clues to reliable sources of information, considering that internet and social media platforms have the potential to significantly influence patient education, often with unverified information.⁵⁵

AUTHOR CONTRIBUTIONS

All authors have contributed to the design of the manuscript, to the acquisition, analysis, and interpretation of data, to the writing of the manuscript, to its revision for intellectual content, and have read and approved its final version.

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CONFLICT OF INTEREST

All authors have no conflicts of interest or funding sources to declare regarding design, implementation, analysis, and interpretation of the data in this article. Maria Letizia Musumeci has previously served as advisory board member and consultant, and has received speaker's honoraria and fees for her participation to clinical trials for Abbvie, Almirall, Biogen, Eli-Lilly, Janssen Cilag, Leo Pharma, and Novartis.

DATA AVAILABILITY STATEMENT

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

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