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Effect of diet and weight loss on the severity of psoriasis

Review

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Abstract

Psoriasis is one of the most common chronic inflammatory skin diseases, constituting a significant health and socioeconomic problem. Despite numerous therapeutic options, the results of treatment often remain insufficient. This may be due to the lack of compliance with medical prescriptions and patients' limited knowledge of their disease. Psoriatic patient's skin well-being is affected by many factors, including lifestyle. The course of the disease is affected by obesity, improper diet, and stimulants. Often these factors coexist. Excessive weight gain in psoriasis can be caused by a decrease in physical activity, caused by feelings of social stigma, coexistence of psoriatic arthritis, depression, and increased alcohol consumption. Several studies have confirmed that the average fat, protein, and calorie content in the diet of a patient with psoriasis are above the recommended norms. On the other hand, adhering to a low calorie, reducing diet results in a clinically significant improvement in the Psoriasis Area Severity Index (PASI) and patients' quality of life (i.e., reduction of Dermatology Life Quality Index [DLQI]). Weight reduction caused by diet and exercise reduces the severity of skin lesions, even in people who have not achieved improvement after general treatment. Therefore, it is important to educate the patient about the nature of the disease at the very beginning of treatment. Patients with moderate to severe forms of the disease are predisposed to the development of cardiovascular diseases, obesity, diabetes, and anxiety. That is why plaque psoriasis requires a comprehensive treatment and a holistic approach to the patient.

Keywords

psoriasis • psoriatic arthritis • lifestyle • diet

1. Introduction

Psoriasis is an inflammatory and chronic skin disease the etiopathogenesis of which is still not completely understood. It is widely considered to be a multifactorial disorder caused by the interaction between inherited susceptibility alleles and environmental risk factors [1]. The best evidence of their roles is the lack of full compliance of psoriasis lesions in monozygotic twins who have the same set of genes, including the genes that predispose to psoriasis. In various studies, this correlation ranges from 35% to 73% [2]. Genetic factors include nine different regions known as Psoriasis Susceptibility (PSORS 1–9), of which PSORS1 is best known, including HLA-Cw6. HLA-Cw6 can present melanocyte specific autoantigen (ADAMTS-like protein 5) to CD8+ T cells and has a high affinity for LL-37, a molecule described as T-cell autoantigen in psoriasis [1, 3]. Although somatic mutations and epigenetic factors may be the cause of incompatibility, this large dissonance strongly emphasizes the significant role of environmental factors in the initiation and pathogenesis of this disease [2]. It is known that environmental factors, such as lifestyle, diet, stimulants, foci of inflammation, some drugs, and psychological factors are involved in provoking the disease. Treatment of psoriasis mainly focuses on the use of general and topical drugs and phototherapy; medical providers often forget about changes in lifestyle, which has a significant impact on the effectiveness of pharmacological treatment. Lack of satisfaction with treatment and insufficient knowledge about the disease can lead patients to alternative medicine. According to a study conducted by the National Psoriasis Foundation, as many as 41% of patients, mainly with mild psoriasis (52.4%) and more often women, use non-conventional methods [4].

This article discusses factors such as diet and weight loss and changes in lifestyle that affect the severity of psoriasis and which

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have a direct impact on the patient and can affect the course of his disease. Therefore, any educational initiatives that increase patient awareness can improve patient satisfaction and outcomes.

2. Weight Reduction

Epidemiological studies regarding the prevalence of psoriasis in the European population show a steady, even two times higher incidence over the last half-century [5]. Civilizational progress affecting changes in diet profile (high-energy diet) and lifestyle (lower physical activity during work and rest) in developed countries leads to an increased incidence of obesity [5–7]. Numerous epidemiological studies confirm the connection between obesity and psoriasis, and obesity is believed to be an independent factor in the development of psoriasis and is associated with a more severe prognosis.

The latest published observational data (753,421 individuals) showed the significant association of higher body mass index (BMI) with psoriasis. An average BMI higher by 1.26 kg/m2 when compared to the control group was observed in adult patients, with an average higher by 1.55 kg/m2 in children. An increase in BMI of 1 kg/m2 was associated with a 4% higher probability of psoriasis, while a higher BMI increases the likelihood of psoriasis by 9% per 1 unit of BMI increase. Overall, a 1 kg/m2 increase in BMI was associated with 4% higher odds of psoriasis. Mendelian randomization analyses provided evidence that higher BMI causally increases the odds of psoriasis (by 9% per 1 unit increase in BMI). Interestingly, it has been shown that the impact of the genetic risk of psoriasis on BMI is small [8]. A meta-analysis of six randomized control studies confirmed that weight reduction as a result of diet or exercise changes results in the reduction of Psoriasis Area Severity Index (PASI) (OR 2.59), and in the case of psoriatic arthritis (PsA), it effectively achieves minimal disease activity (OR 4.20) compared to control [9]. Other studies report that the coexistence of obesity and HLA-CW06 increases the risk of developing psoriasis approximately 35 times compared to lean individuals [10].

Studies have also shown that there is a strong correlation between the severity of psoriasis and obesity. Patients suffering from severe psoriasis were more likely to be obese than patients with moderate to mild psoriasis (66%, 34%, and 14%, respectively) [11, 12].

Visceral adipose tissue, storing excess energy, initially protects against metabolic complications associated with hyperglycemia. However, further accumulation of adipose tissue in obesity and further caloric overload lead to an increase in its volume, not only in the subcutaneous tissue and in the visceral compartment, but also in ectopic tissues, leading to the development of inflammation and insulin resistance [13, 14]. Adipocytes increase their volume more, and more macrophages appear. They initiate inflammation and produce proinflammatory cytokines (TNFα, IL-6, -8, -17, -18, MCP-1), similar to inflammation in psoriasis, and adipokines (adiponectin, omentin, chemerin), which also affect the intensification of disease eruptions [7, 15]. In addition, they participate in the development of dyslipidemia, insulin resistance, and diabetes, leading to the development of cardiovascular diseases. This phenomenon is illustrated by the concept of "psoriatic march," in which systemic inflammation associated with psoriasis, fueled by proinflammatory cytokines and adipokines produced by visceral fat, leads to the development of insulin resistance and damage to endothelial cells. Endothelial dysfunction predisposes to the formation of atherosclerotic plaques and faster development of cardiovascular incidents [7, 15, 16]. Another function of visceral fat is being a reservoir for tissue resident memory cells, triggering a rapid inflammatory response in psoriasis [12, 17, 18].

Recently, data have appeared that intradermal adipocytes release large amounts of antimicrobial peptides during adipogenesis, including cathelicidin. It forms complexes with its own DNA, and through the toll-like receptor 9 (TLR9), it activates plasmacytoid dendritic cells, contributing to the development of psoriatic inflammation [12, 19, 20].

Obesity has a significant impact on psoriasis therapy. It increases the risk of systemic side effects due to the coexistence of obesity-related diseases (nonalcoholic fatty liver disease - NAFLD, hypertension, dyslipidemia, diabetes) and decreases the effectiveness of biological drugs, the dose of which should be adjusted to the patient's weight. This is especially true of TNFα inhibitors (iTNF), because not only lesions, but also visceral fat, is an additional source of TNF. The role of iTNF is to eliminate an excess of TNF from the circulation and psoriatic lesions; therefore, their standard dose may not be sufficient to achieve remission of the disease in obese patients. The study assessing the effect of BMI on the clinical response of systemic treatment in psoriasis showed a lower percentage of patients achieving PASI75 with an increase in BMI: obese OR after 0.7 weeks was 0.73 and after 16 only 0.62 compared to subjects with normal body weight (BMI = 20–24) [21].

A recent meta-analysis of 54 studies (19,372 patients) assessed iTNF efficacy in selected immune-mediated inflammatory diseases (inflammatory bowel diseases, rheumatoid arthritis, spondyloarthropathies, psoriasis, and PsA) in patients, depending on body weight and BMI. The study showed that obese patients had a 60% higher risk of treatment failure (OR 1.60), and an increase in BMI of 1 kg/m2 was associated with a 6.5% enhanced risk of failure [22].

Patients with obesity belong to the "high-needed" group of patients much more often among patients with psoriasis, up to 35.5%. This is severe psoriasis, which occurs when at least two systemic treatments are inappropriate due to a lack of efficacy, intolerance, or contraindications [23]. High BMI is a negative predictor of the systemic therapy efficacy. The dosage of systemic

drugs raises many doubts about whether the dose should be calculated according to actual or appropriate body weight in order to obtain an effective minimum dose as a result.

There are more and more promising reports about the impact of bariatric surgery on the reduction of PASI in the psoriatic patients with obesity. Hossler et al. reported significant postoperative reduction of skin lesions in 60% of patients treated by bariatric surgery [24]. The Danish analysis of almost 14,000 cases highlights the beneficial role of the gastric bypass method. It was associated with a significantly reduced risk and improved prognosis of psoriasis and PsA, whereas gastric banding was not. It is probably related to the postoperative differences in the secretion of gastric hormones' direct anti-inflammatory effects on gut microbiota, which modulate inflammation [25] (Fig. 1).

Numerous studies have shown an association between obesity and PsA. Obesity, present in 45% of patients with PsA, increases the risk of arthritis in psoriasis [26], is associated with higher disease activity and worse response to treatment, and also obviously worsens physical fitness, contributing to the development of disability [27–30]. In interventional studies published to date assessing the effect of weight loss on the course of arthritis, a significant improvement in joint disease and tendon attachment activity has been observed [31, 32]. In the Klingberg et al. study, ACR 20, 50, and 70 improved by 51%, 34%, and 7%, respectively [31], which is particularly significant compared to randomized studies of the effect of iTNF on PsA activity, where improvement in ACR 20 and 50 were observed in 50%–60% and 30%–40%, respectively [33, 34]. The mechanism leading to the occurrence of PsA in obese people is not completely understood. It seems that in addition to the immunological factors described above, the impact of microtrauma associated with overload is particularly important in the area of the lumbar spine and lower limbs [35]. In addition, local inflammation in adipose tissue also presents intra-articularly and has an impact in the vicinity of tendon attachments [36].

Recommendations for the patient: weight reduction, cooperation with a dietitian.

3. Diet and Dietary Supplements

Diet, along with infection and stress, is one of the very important elements affecting the course of psoriasis, although it is still underestimated and not sufficiently researched. Some patients eagerly use diet and methods proposed by alternative medicine due to the lack of satisfaction with the effects of treatment obtained only by pharmacotherapy [63].

The first study that confirmed the impact of diet on the course of the disease was conducted in the US as part of the National Health and Nutrition Examination Survey (NHANES). Diet



Fig. 1. The patient with over 20-year-old history of plaque psoriasis
A) Condition before sleeve gastrectomy, treated with methotrexate 25mg / week. (PASI: 30.4, DLQI: 24)
B) Condition after sleeve gastrectomy, treated with methotrexate 10mg / week. (PASI: 1.2, DLQI: 4)

Table 1. Relationship between systemic psoriasis treatment and obesity

Drug	Result
Methotrexate	• The consequence of obesity is NAFLD → an increase in MTX hepatotoxicity and the risk of liver fibrosis during therapy (monitoring this risk using elastography) [37, 38]. • In contrast, MTX reduces the risk of cardiovascular disease (low doses with folic acid! to prevent the formation of atherogenic homocysteine) [39].
Cyclosporine	• Cs-A is lipophilic and binds to lipoproteins → atherogenic dyslipidemia in obesity → disruption of pharmacokinetics [39, 40]. • High BMI limits drug distribution → an increase in toxicity (which is why the dose is calculated due to the patient's appropriate body weight) [41]. • However, the increase in creatinine levels was higher in the group of the patients who received the weight-adjusted dose than in the group on the new, weight-independent regimen [42]. • Obesity as a risk factor for hypertension increases the probability of its development and nephrotoxicity during therapy [39, 40]. • A weight loss of 5-10% improves the effects of Cs-A treatment in the obese; a low calories diet can be a complement to therapy [43].
Acitretin	• Hypercholesterolemia with ↓ HDL and hypertriglyceridemia are a common side effects in patients treated with acitretin, especially associated with diabetes, obesity or alcohol addiction [15, 39]. • Disorders of lipid and glucose metabolism during treatment were transient and were not associated with changes in BMI, concentration of adipokines associated with obesity (TNF-α, resistin, adiponectin) [44].
Phototherapy	• Obesity does not affect the effectiveness of PUVA, because the dose of psoralen is calculated according to body weight. • However, patients may be more exposed to developing erythema or burns due to their closer distance to the light source [37].
Biologics	• Increased body weight requires a change in the dosage of biological drugs (ustekinumab, infliximab). Excessive body weight increases the volume of distribution, which results in a decrease in drug concentration [45]. • In obesity, TNFα production is increased. The role of iTNF is to eliminate its excess from the circulation and psoriatic lesions, which is why these drugs may be less effective in obese patients: - the percentage of patients with PASI 75 decreases with increasing BMI, regardless of gender, severity of lesions and prior treatment [21]. - only a 160mg loading dose followed by 80 mg of adalimumab causes effective disease remission in obese psoriasis patients [46]. - lower body weight is a predictor of long-term results of iTNF treatment efficacy [47]. • During iTNF therapy, an increase in body weight was observed, on average 2-3 kg after 6 months of treatment [38, 48], however iTNF may improve insulin sensitivity causing hypoglycemia particularly in patients receiving insulin [49]. • In contrast to infliximab, ustekinumab does not cause ↑ BMI in treated patients [50]. • Secukinumab - no BMI changes during treatment and no difference in PASI reduction depending on BMI [51], but the effectiveness is lower in patients with BMI> 30. • Ixekizumab is more effective in overweight patients, regardless of BMI, even with primary or secondary resistance to secukinumab [52, 53]. • Tildrakizumab - safety and efficacy of treatment does not depend on coexisting metabolic syndrome and obesity [54]. • Risankizumab - there is no significant difference in PASI reduction depending on BMI, even >30, although blood levels in patients >100 kg are 30% lower than in patients with lower body weight, but they still are within the therapeutic range [55]. • Guselkumab is not reported to increase body weight and showed sustained superior efficacy, regardless of bodyweight [56].
Small molecules	• Apremilast → PDE4 inhibition → slight weight loss during therapy [57], about 20% of patients reduce body weight by 0.5% due to [58]: - Increase in energy expenditure, - Improvement of glucose metabolism, - Increase in metformin activity, - Lipolysis and regulation of white adipose tissue [37, 49]. • Tofacitinib - after treatment, an increase in HDL-C, but also LDL-C, total cholesterol, ApoB and ApoA-I (decrease in cholesterol catabolism), improvement of markers of HDL antiatherosclerosis function [59, 60]. • Fumaric acid esters (FAE) - the positive effect on cardiovascular and metabolic diseases through their anti-inflammatory and antioxidant properties (endothelial protection, reduction of insulin resistance, decrease of C-reactive protein, and increase of adiponectin) [61, 62].

and nutritional status in patients with psoriasis were assessed in comparison with the control population, stating a positive correlation between BMI, increased vitamin A and α-carotene concentration, and negative correlation with the consumption of simple sugars and the occurrence of psoriasis [64]. Similar results were obtained in Italian studies assessing untreated men with psoriasis compared to a group of healthy men of the same age, who were BMI-matched. A 7-day diet, BMI, glucose concentration, lipid profile, CRP, HOMA-IR, visceral obesity index (VAI), and fatty liver index (FLI) were analyzed. It turned out that patients with psoriasis consumed more simple carbohydrates, fat, polyunsaturated fatty acids (PUFA), but with an unfavorable n-6 to n-3 ratio. Protein intake, complex carbohydrates, monounsaturated fatty acids (MUFA), n-3 PUFA and fiber was less in patients than in the control group. In addition, PASI was correlated with BMI, glucose and lipid profile values, and liver function. The amount of consumed MUFA was associated with lower PASI ($P < 0.001$) [65]. The influence of the diet on the course of psoriasis was observed by psoriasis patients themselves. The most common reported disease aggravating factors were sugar (13.8%), alcohol (13.6%), tomatoes (7.4%), gluten (7.2%), and dairy products (6%), and less often meat, bread, beer, wine, eggs, and spicy dishes, while improvement was observed after introducing dietary supplements (35.1%), vegetables (26.5%), fruit (21.8%), water (11.2%), and fish (9.2%) to the diet [66]. Incorrect dietary habits correlate not only with the occurrence of obesity, but also with a more severe course of psoriasis and a decrease in the quality of life [67].

An incorrect diet of patients with psoriasis may result from concomitant eating disorders, which secondarily result in the development of obesity and metabolic syndrome. More often in this group, attacks of binge eating (BED) are observed [68]. It can be caused by anxiety and depression, which more often affect patients with psoriasis, which causes incorrect eating habits [68, 69].

Rapid industrial development also leads to a change in eating habits, such as the dominance of a diet high in fat, salt, and sugar, and with excessive calorie intake, which results in obesity and metabolic syndrome [6]. According to the United States National Institutes of Health Panel, the ω-3 to ω-6 ratio in the diet should be 1:1.8, and the American diet provides ω-3 to ω-6 like 1:15–20 [70]. That is why it is worth considering increasing the supply of some nutrients and supplements as a support for psoriasis therapy. ω-3 PUFAs reduce the production of TNFα, IL-1β, and IL-1α and demonstrate a significant anti-inflammatory effect, in contrast to ω-6 acids, which have an pro-inflammatory effect [71]. Moreover, PUFAs are substrates for the production of prostaglandins and leukotrienes, which have pro- or anti-inflammatory properties, depending on the PUFA from into which they were made. The product of ω-6 is highly pro-inflammatory leukotriene B4, which is involved in the activation of neutrophils [72].

Recent studies emphasize that saturated fatty acids (SFAs) can directly activate inflammasomes in keratinocytes and macrophages and induce IL-17 production by Tγδ cells, while ω-3 PUFAs inhibit Th17 differentiation [73]. Therefore, limiting the consumption of dairy or meat and replacing them with PUFAs is rational.

After analyzing many diets, the Mediterranean diet is acknowledged as optimal, not only in psoriasis, but also in other diseases associated with cardiovascular disease in developed countries. It is based on: (1) high consumption of the highest quality extra virgin olive oil, fruit, vegetables, legumes, cereals, fish, and seafood; (2) low consumption of dairy products, meat, and derivatives; and (3) consuming moderate amounts of alcohol, such as wine, during a meal. It is a significant source of antioxidants and MUFA with anti-inflammatory properties that are associated with lower cardiovascular risk [74].

Eicosanoids are lipid intermediates that form a link between immune cells and dietary fatty acids through their ability to regulate inflammation [75]. These are oxidized 20-carbon PUFA derivatives such as arachidonic acid and eicosapentaenoic acid (EPA). The increased incorporation of EPA and docosahexaenoic acid (DHA) into the membranes results in lesser arachidonic acid eicosanoids production, and DHA has an inhibitory effect on antigen-presenting cells in the epidermis [72, 76]. However, the consequences of a Western diet mean that eicosanoids are mainly produced from arachidonic acid substrates [77], which results in increased inflammation [78]. Arachidonic acid has a particularly strong pro-inflammatory effect due to three activated CH2 groups, which makes it prone to oxidative attack and more likely to form free radicals. Eicosanoids such as prostaglandins and leukotrienes develop a variable structure depending on the PUFA from which they were made [72]. This potentially explains the mechanism by which arachidonic acid promotes inflammation, while EPA soothes it [72].

The use of a gluten-free diet raises many doubts. The gluten-free diet has been growing in popularity since the 1990s, inter alia, thanks to the research of Michaëlson et al. [79]. The best-known work assessed the effects of a 3-month gluten-free diet in patients with psoriasis with positive anti-gliadin (AGA-positive) and negative anti-gliadin (AGA-negative) antibodies. Reduction of psoriatic lesions was found in AGA-positive patients after a gluten-free diet, and there was no effect in AGA-negative patients. Returning to the gluten-containing diet again made the lesions worse in the first group. However, AGA-positivity is common and often persistent in the older population, which has been exposed to gluten for decades. AGAs have low specificity and should not be used to diagnose celiac disease (CD) [80]. A recent meta-analysis of 18 studies found a significant association for CD in patients with psoriasis (OR 2.16) and psoriasis in patients

with CD (OR 1.8). Moreover, the CD patients were at 1.7 times higher risk of developing new-onset psoriasis [81]. This may confirm the role of gut-skin-axis, in which inflammatory changes in the intestinal microbiome cause an increase in permeability, lead to a local and systemic immune response, and stimulate psoriatic skin inflammation [82]. However, the latest Nurses' Health Study II study, including 85,185 psoriatic female patients and 85,324 with PsA, showed no significant effect of increased gluten intake on psoriasis (HR 1.15) and PsA (HR 1.12) [83]. Therefore, a gluten-free diet should only be used on patients with confirmed CD. Adults with a positive serological gluten sensitivity test should use 3 months' gluten-free diet (GFD) to confirm its effectiveness [84]. Patients' dyspeptic complaints should not be ignored and, if necessary, diagnostic tests (tissue transglutaminase antibodies, IgA endomysial antibody, endoscopic examination) should be performed. However, screening for hypersensitivity to gluten in psoriasis is not recommended due to the high percentage of false positives. Based on the recommendations of the American College of Gastroenterology, it is advisable to perform these tests in first-degree relatives with celiac disease or in patients with active gastrointestinal symptoms [84].

Chronic inflammation in psoriatic lesions also affects the formation of oxygen-free radicals (ROS) and superoxide anion, leading to "oxidative stress." It is an imbalance between the amount of ROS and antioxidants at the cellular level (α and β-carotene, ascorbic acid, α-tocopherol, flavonoids) [85,86]. This leads to lipid peroxidation and the formation of oxidized low-density lipoprotein- LDL, which also strongly stimulates atherosclerotic processes. Free radicals also cause damage to endothelial cells, increasing the permeability of small vessels and enabling the migration of inflammatory cells, which facilitates the development of inflammation in psoriasis [86]. Psoriasis patients had significantly reduced oxidative capacity, decreased catalase activity, glutathione peroxidase and selenium levels, and these disorders correlated with the severity of skin lesions [87,88]. Therefore, supplementation with organo-selenium compounds, such as thionine and selenocysteine, may be useful in the treatment of psoriasis by supporting an antioxidative defense [89,90]. However, when supplementing with selenium, it should be remembered for its toxicity at doses above 400 mg/dL, manifested in nausea, vomiting, nail changes, loss of energy, and irritability. Natural sources of selenium are Brazil nuts, garlic, legumes, and yeast [91]. In addition to selenium supplementation, it seems beneficial to use other antioxidants such as coenzyme Q10 and vitamin E, especially in patients with severe forms of psoriasis [92].

The popularity of vitamin D3 supplementation has raised hope for its potentially beneficial effect in the treatment of psoriasis. While the local effect of its derivatives is well proven (slowing of keratinocyte proliferation, improvement of differentiation and apoptosis, inhibition of T cell proliferation, Tregs induction),

Table 2. Analysis of the effectiveness of diets used in psoriasis [84, 98, 99]

Diet	Mechanism of action	Outcome summary
Low-calorie diet	• Reduction of visceral fat, a source of pro-inflammatory cytokines and adipokines, • Decrease in supply of arachidonic acid, a source of pro-inflammatory LTB4 • ↓CD4+ lymphocyte activity and increase in anti-inflammatory IL-4 concentration, • ↓free radicals (the role of oxidative stress) • Reduction of keratinocyte proliferation [100] • ↓supply of sulfur (red meat) → ↓hydrogen sulfide → undamaged gut mucosa is impermeable to bacteria, that can't activate the Toll-like and NF-kB inflammatory pathway receptors [101]	• Improvement of skin lesions (PASI reduction) and faster achievement of minimal PsA activity [102, 103] • Improving the metabolic profile (↓ of cholesterol, LDL, serum triglycerides) [104]
A very low-calorie ketogenic diet (VLCKD)	• Rapid ↓ visceral fat volume → ↓ production of pro-inflammatory mediators	• Reduction of pruritus, improvement of skin lesions (PASI reduction) and DLQI [105]
Mediterranean diet	• Anti-inflammatory action (MUFAs, oleic acid, polyphenoles, antioxidant agents, and the inhibitor of cyclooxygenase-2, resveratrol) • Dietary fiber favoring beneficial effects on gut microbiota and improve the gut immune responses	• Patients adhering to diet achieved PASI reduction faster compared to non-adherents [106, 107] • Improving the metabolic profile (lipid profile) [108]
Vegetarian diet	• ↓ supply of arachidonic acid → ↓ B4 leukotriene production and ↑ anti-inflammatory IL-4 • ↓ restoration of normal neutrophil activity (↓ lactoferrin, a marker of their activity).	• During fasting, joint pain in PsA decreases • Reduction of skin lesions in psoriasis and pustular psoriasis of the feet [109]
Gluten-free diet GFD	• Psoriasis susceptibility loci overlap with CD • The role of gut-skin axis (activation of Th1 and Th17)	• CDs are more common in psoriasis, and psoriasis is more common in CDs, and the CD patients were at 1.7 times higher risk of developing new onset psoriasis [81] • BUT: There is no effect of increased gluten intake on psoriasis [83]

Table 3. Diet recommendations for patients with psoriasis

YES	• Dietary weight reduction in patients with BMI>25 • ω-3 PUFAs (oily fish: sardines, salmon, mackerel, herring, tuna and vegetable oils: linseed, rapeseed, soybean, corn oil) - wild fish (not farmed), • Antioxidants such as selenium (oat, brown rice, pumpkin seeds, poultry, fish), coenzyme Q, vitamin E per day and polyphenols in natural pigments (dark grapes, blueberries, black elderberries, cranberries), • Only in case of gluten intolerance, glute-free diet is recommended (elimination of wheat, rye, barley and oats).
NO	• Red meat, animal fats and offal • Restriction of dairy products (SFA) • Nightshade vegetables (tomatoes, paprika, chili-pepper, eggplant, pepper) • All types of alcohol should be avoided (except for a glass of red wine containing resveratrol) • Highly processed foods containing preservatives and fried foods (damage of essential non-saturated fatty acids in high temperature and formation of harmful and carcinogenic compounds: acrolein and acrylamide) • Excessive consumption of products containing caffeine and spicy food is not recommended

despite initial admiration for the effectiveness of its supplementation, many doubts now appear [94]. In fact, a meta-analysis involving 571 patients showed that 25(OH)D serum levels are lower in patients with psoriasis, and that there is a small but statistically significant negative correlation between its level and the severity of skin lesions [94]. However, a 14-year study of 70,437 women in the US showed no significant association between vitamin D intake and the risk of psoriasis [95]. In case of vitamin D3 deficiency, supplementation should be considered. This is especially important because of the strong interaction between psoriasis and metabolic syndrome, which is associated with an increased cardiovascular risk. Data confirming the beneficial effects of vitamin D3 in psoriasis remain controversial; its supplementation is recommended only in cases of deficiency [84, 93]. In contrast, in patients with PsA, oral supplementation (0.5 µg alphacalcidol or 0.5–2.0 µg calcitriol daily) is recommended as a supplement to standard therapy [84].

Other products that appear to have a beneficial effect on plaque psoriasis include garlic (due to its natural antibiotic properties and lipoxygenase inhibition), milk thistle (which inhibits leukotrienes and improves liver function, which affects the reduction of endotoxins in blood, present in the cell walls of intestinal bacteria), and licorice (which works favorably by inhibiting the conversion of cortisol to inactive cortisone) [96].

A summary of research on supplements and the role of diet in psoriasis is a recent study analysis (1990–2018) that confirms that weight reduction combined with a healthy lifestyle is indicated for patients with moderate to severe disease, causing a significant reduction in the PASI. In contrast, oral vitamin D supplementation is only recommended if vitamin D is deficient, and selenium and omega 3 appear to be beneficial for patients with psoriasis [97].

4. Conclusions

The problem of obesity is more often observed in patients with psoriasis. It is necessary to lead a healthy lifestyle, maintain proper body weight and proper eating habits, and exercise to improve the quality of the patient's life and the course of the disease.

Abbreviations

BMI - body mass index, **CD -** coeliac disease, **CRP -** C-reactive protein, **DHA -** docosahexaenoic acid, **DLQI -** Dermatology Life Quality Index, **EPA -** eicosapentaenoic acid, **FLI -** fatty liver index, **GFD -** gluten-free diet, **IL –** interleukin, **iTNF -** tumor necrosis factor α inhibitors, **LL-37 -** 37- residue human antibacterial peptide, **LTB4 -** leukotriene B4, **MCP -** monocyte chemoattractant protein, **MUFA -** monounsaturated fatty acids, **OR -** odds ratio, **PASI -** Psoriasis Area Severity Index, **PsA -** psoriatic arthritis, **PUFA -** polyunsaturated fatty acids, **ROS -** oxygen free radicals, **SFAs -** saturated fatty acids, **TLR -** Toll-like receptor, **TNFα -** tumor necrosis factor α, **VAI -** visceral obesity index.

Authors' Contribution

A.O.-S.: research concept and design, supervising the project, acquisition of data, data analysis and interpretation; writing (original draft preparation); **M.K.-F.**: acquisition of data, data analysis and interpretation; writing (original draft preparation); **M.K.-W.**: acquisition of data, writing (original draft preparation); **J.S.**: supervising the project, acquisition of data, writing (original draft preparation).

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Conflict of Interest

The authors have no potential conflicts of interest to declare.

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