Review paper

A crossroads between dietary habits, alcohol consumption, and smoking in the clinical course of psoriasis: a narrative review

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Abstract

Psoriasis is a chronic autoimmune disease that affects 1–3% of the population. The pathomechanism of psoriasis development is complex, but genetic (non-modifiable) factors play a key role. However, the importance of environmental factors and lifestyle choices, such as the diet, alcohol consumption, and smoking, is increasing. The objective of this review was to analyse the influence of dietary habits, alcohol consumption, and smoking on the clinical course of psoriasis. Stress, a poor diet, alcohol abuse, and smoking can trigger psoriasis or cause its exacerbation. Therefore, in addition to the correct selection of therapy, it is extremely important to educate patients about the impact of these factors on the onset and progression of psoriasis. This literature review confirms that a holistic and multidisciplinary approach is required for patients with psoriasis, further emphasizing Hippocrates' thesis, "Let food be thy medicine, and medicine be thy food".

Key words: psoriasis, lifestyle, eating habits, alcohol, severity.

Introduction

Psoriasis is a chronic autoimmune disease that affects 1–3% of the population, predominantly Caucasians between 20 and 30 years of age, and between 50 and 60 years [1, 2]. The pathomechanism underlying the development of psoriasis is complex, but genetic factors play a key role [3]. Nine different regions of genes implicated in the development of psoriasis (PSORS 1–9) have been located, the most important of which is PSORS 1, located on chromosome 6p21, where the major histocompatibility genes are also present [4].

Activation of interleukin-12/Th1/interferon γ (INF-γ) (IL-12/Th1/IFN-γ) and Th17/interleukin-23 (IL-23) axes is observed, in which IL-12 induces the formation of Th1 lymphocyte subpopulations and IL-23 stimulates the activity of Th17 lymphocyte subpopulations. Moreover, Th17 lymphocyte differentiation induces transforming growth factor β (TGF-β) in the presence of IL-6, while Th17 lymphocytes affect the secretion of interleukin-17 (IL-17) and interleukin-22 (IL-22), which are keratinocyte proliferation simulators [5, 6]. A characteristic feature is the phenomenon of parakeratosis, or accelerated and incomplete keratinization, which is associated with an 8-fold shortening of the cell cycle [7].

The most common clinical form is psoriasis vulgaris, accounting for 90% of cases [8]. It is characterized by the presence of papular lesions on an erythematous base covered with silvery-white scales and located on the scalp (most common), symmetrically on the upright parts of the upper and lower extremities, as well as in the lumbosacral region. The diagnosis of psoriasis is typically made based on the clinical picture [9]. When the diagnosis cannot be made based on the clinical picture and dermoscopy, a skin biopsy from the lesion and histopathological confirmation are indicated [10–12].

The objective of this narrative review was to analyse the influence of dietary habits, alcohol consumption, and smoking on the clinical course of psoriasis.

The importance of environmental factors in the course of psoriasis

Environmental factors can influence both the onset and course of psoriasis and can have a decisive impact

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on the risk of exacerbations [13, 14]. Patients with psoriasis often have a strong sense of the cosmetic defect and social stigma due to their skin lesions, which significantly affects their quality of life and increases their risk of exacerbations [10, 15].

Effects of diet on the clinical course of psoriasis

Although the importance of diet in the clinical course of psoriasis has been evaluated since the 1950s, no clear dietary recommendations have been developed to date [16–18].

The rationale for a reduced-calorie diet in patients with psoriasis is that obesity is a risk factor for the disease [12]. In addition, the concomitant presence of obesity and HLA-Cw*06 increases the risk of psoriasis 35-fold compared to individuals with normal body weight [11, 19]. Studies conducted to date indicate that a body mass index (BMI) of 26–29 kg/m^2 slightly increases the risk of psoriasis, while a body mass index (BMI) > 29 kg/m^2 significantly increases the risk of the disease. At the same time, a reduction in body weight results in an alleviation of symptoms [20, 21]. Hsieh *et al.* showed that reducing caloric intake from food by one third for 4 weeks resulted in a reduction in excessive epidermal proliferation by 45% in a mouse model [22]. The link between weight reduction and relieving the clinical symptoms of psoriasis is most likely due to a reduction in the volume of visceral fat, which consists not only of adipocytes but also a large number of macrophages [23, 24]. Studies indicate that patients with psoriasis have decreased concentrations of adipokines with protective effects, which include adiponectin and omentin, and increased concentrations of leptin, chemerin, visfatin, and resistin [23–28].

Further, macrophages are a source of a number of pro-inflammatory cytokines, which are responsible for the onset of psoriasis, , as well as other adipokines, such as resistin, visfatin, and retinol-4-binding protein [29, 30]. An increase in the concentration and secretion of TNF-α from visceral adipose tissue is responsible for free fatty acids (FFAs), which, by exerting an activating effect on the Janus kinase pathway and stimulation of Toll-like receptors (TLR-4), contribute to the further development of inflammation and the "vicious circle" mechanism of inflammation [31–33]. FFAs may also exacerbate oxidative stress. An important action of TNF-α is to exert a suppressive effect on the expression of genes encoding enzymes in adipocytes, such as acetyl-CoA carboxylase, lipoprotein lipase, and glycerol phosphate dehydrogenase, which are responsible for the uptake of fatty acids from the blood, resulting in an increase in the concentration of FFAs and triglycerides in the blood [34, 35]. This concept explains the phenomenon of "psoriasis march" and the frequent coexistence of psoriasis with obesity [36].

Low-calorie diet

Serum levels of pro-inflammatory cytokines have been shown to decrease in patients who have experienced weight loss [37]. Patients with psoriasis should consume a diet rich in omega-3 fatty acids that are not synthesized by humans and must be obtained from food. The main representatives of omega-3 acids are eicosapentaenoic acid and docosahexaenoic acid [38]. Omega-3 fatty acids reduce the inflammatory process by affecting the expression of genes that encode proteins crucial to the etiopathogenesis of psoriasis [39]. Inuit people are much less likely to develop psoriasis due to their traditional diet of marine fish, which is an excellent source of eicosapentaenoic acid and docosahexaenoic acid. Similarly, fewer cases of psoriasis have been reported in West Africans, whose diet is also rich in omega-3 fatty acids [32]. Significant improvements in the skin condition and a reduction in lesion severity have been observed in patients with psoriasis who consume omega-3 fatty acids [40]. Gisondi *et al.* showed that weight reduction among obese patients with psoriasis resulting from a low-calorie diet can be a valuable adjunct to traditional pharmacotherapy [41]. Pona *et al.* and Wu *et al.* showed that a low-calorie diet combined with topical or general treatment significantly reduced psoriasis activity and severity index (PASI) and dermatology life quality index values [18, 42]. Al-Mutairi *et al.*, who conducted a randomized prospective study in a group of 262 patients with psoriasis receiving biologic therapy, reported significant reductions in body weight and PASI and Body Surface Area (BSA) values after 24 weeks of follow-up in patients on a low-calorie diet (< 1000 kcal; n = 131) compared to a group of patients whose dietary habits were not changed (control; n = 131) [43]. This finding provides further evidence of the synergistic effect of pharmacotherapy and a reduction diet in patients with psoriasis [43].

The improvement in clinical indicators of psoriasis progression in patients on a low-calorie diet may also be due to a reduced dietary supply of pro-inflammatory arachidonic acid, which, in turn, translates into lower levels of arachidonic acid metabolites, such as leukotriene B4 and prostaglandin 2, and lower CD4+ lymphocyte activity [44].

Gluten-free diet

The National Psoriasis Foundation's 2018 Medical Council indicated that it "weakly recommend[s] a gluten-free diet (GFD) only in patients who test positive for serologic markers of gluten sensitivity" [45]. The first reports of a link between enteropathy, malabsorption syndrome, and psoriasis were published in the 1980s [46]. The common denominator of the three diseases appears to be an increased permeability of the intestinal mucosa in individuals with celiac disease, which affects the increased migration of bacteria acting as superantigens, caus-

ing the onset or exacerbation of psoriatic lesions [47]. Acharya *et al.* conducted a systematic review and meta-analysis of 18 articles that examined the association between celiac disease, GFD, and psoriasis [48]. Only one of the two studies on the prevalence of celiac disease in patients with psoriasis showed a significantly higher prevalence of this dermatosis in patients with gluten intolerance (odds ratio, OR = 1.9, 95% CI = 1.6–2.2 and OR = 1.20, 95% CI = 0.91–1.59 [49]). Two studies also confirmed a significantly higher risk of psoriasis in patients with celiac disease (OR = 1.72, 95% CI = 1.54–1.92 [45] and OR = 1.9, 95% CI = 1.5–2.3 [46]). By contrast, the risk of celiac disease in patients with psoriasis vulgaris or psoriatic arthritis was reported in five [45, 47–50] out of nine [51–54] studies (meta-analysis: OR = 2.16, 95% CI = 1.74–2.69 [48]).

Similarly, the incidence of psoriasis among patients with celiac disease appeared to increase according to four [50–53] out of eight [54–57] studies (meta-analysis: odds ratio = 1.8; 95% CI = 1.36–2.38 [48]). In a questionnaire study, Affifi *et al.* showed that 38% of surveyed (n = 1206) patients with psoriasis declared the use of GFD, and another 53.4% (n = 247) indicated an improvement in their clinical condition after eliminating gluten from their diet. In the vast majority of cases, celiac disease occurs in people with other autoimmune diseases. Among patients with psoriasis, a subpopulation with coexisting visceral disease has been observed [15]. A study by Michaëlsson *et al.* showed that in psoriasis patients with serum antigliadin antibodies (AGA), the use of a gluten-free diet improves their clinical status [58].

In a screening study by Michaelsson *et al.,* 16% of 302 patients with psoriasis had elevated titres of IgA-class AGA antibodies [59]. However, other researchers found no statistically significant differences in AGA antibody titres in IgA and IgG classes in patients with psoriasis compared to healthy controls [60]. Moreover, in a study by Zamani *et al*., only 3 of 328 patients with psoriasis had elevated transglutaminase antibodies and anti-endomysial antibodies, which are specific for celiac disease [61].

Further, Kolchak *et al.* showed a significant reduction in PASI values in 56% and 36% of patients with psoriasis who had very high (> 30 U/ml, n = 5) or high (11.5–30.0 U/ml, n = 8) IgA AGA, respectively. Nevertheless, it was not indicated whether these patients suffered from gluten intolerance [62]. De Bastiani *et al.* confirmed the effectiveness of GFD use in patients with psoriasis and coexisting celiac disease, noting during a 3-month follow-up, a reduction in PASI values in 9 patients (n = 2, a reduction in PASI two of at least 50%; n = 5 of at least 75%; n = 1, complete resolution of lesions) [63]. Qiu *et al.* showed that out of 75 antigens in the serum of patients with psoriasis, significantly higher concentrations were recorded only for IgG4 against antigliadin. The concentration of these antibodies positively correlated with the PASI score (r = 0.65, p < 0.001), suggesting that the determination of their levels may be a valuable diagnostic biomarker of psoriasis [64]. Nevertheless, a relatively large number of case-control studies do not indicate an association between GFD and psoriasis severity. Nevertheless, the studies presented have low power (global seroprevalence of celiac disease at 1.4%) [65].

Vegetarian diet

By contrast, the premise of a vegetarian diet is to eschew meat and meat products in favour of large amounts of groats, cereals, legumes, vegetables, fruits, nuts, and mushrooms, reduces the supply of arachidonic acid and its derivatives, prostaglandins and leukotriene B4, which have pro-inflammatory effects [66]. A vegetarian diet benefits weight maintenance, as it results in a lower intake of saturated fatty acids and cholesterol, which is important for patients with psoriasis. It contributes to regulating uric acid, C-reactive protein, and triacylglycerols [67]. People who give up eating meat should introduce a substitute in the form of fish, which supplements omega-3 fatty acids [18]. Elevated markers of lipid peroxidation and oxidized low-density lipoprotein are found in patients with psoriasis, indicating an overproduction of oxygen free radicals in this condition [68]. Thus, people with psoriasis who consume a diet rich in fruits and vegetables, a source of antioxidants, may have less severe psoriatic lesions [69].

Mediterranean diet

The Mediterranean diet, considered a healthy diet, can be a valuable adjunct to the treatment of chronic inflammatory diseases because it is low in saturated fatty acids and trans-fatty acids, and rich in polyunsaturated fatty acids of the omega-3 family, antioxidants and flavonoids [70].

There are many variants of the Mediterranean diet, varying in the quality and quantity of total fat consumed, the type of meat, milk, cheese, fruits and vegetables, and wine [70]. The traditional Mediterranean diet is characterized by a high proportion of olive oil and products of plant origin, such as cereals, fruits, vegetables, legumes, potatoes, nuts and seeds [71]. Fish and poultry are consumed in moderation, and red meat is eaten only a few times a month. Dairy products are mainly consumed in the form of yogurt and cheese [71, 72].

The study evaluated the convergence of the dietary habits of 62 patients with psoriasis with the Mediterranean diet, based on the score obtained from the questionnaire used in the Prevention with Mediterranean Diet study (PREDIMED) [73]. It was assessed that people with psoriasis followed diets significantly less similar to the Mediterranean diet (lower point scores) compared to healthy subjects [73]. In addition, lower levels of similarity to the Mediterranean diet were associated with higher PASI scores and CRP levels [73]. Consumption of olive oil,

fruits, vegetables, legumes, fish and nuts negatively correlated with PASI score [73].

Alcohol consumption, addiction, and association with psoriasis

According to available statistics, more than 10% of the adult population in Poland has consumed alcohol in a harmful way at some point in their lives [74]. The main reasons for not attempting or discontinuing alcohol treatment are psychological barriers in addicts, who are ashamed of their disease, fear being associated with a facility, and may face social ostracism [75]. It should be emphasized that alcohol consumption, especially in a risky manner, significantly restricts possible therapeutic options for psoriasis [8, 76]. For example, aztrethin and methotrexate may also have hepatotoxic effects, limiting their usefulness in the treatment of psoriasis in patients with chronic alcohol abuse [77]. The exacerbation of psoriasis in alcoholics appears to be associated with more frequent mechanical trauma, a greater susceptibility to infections, and the exertion of adverse changes in the differentiation of specific T-lymphocyte subpopulations that disrupt the immune response [78]. Alcohol can both initiate and exacerbate inflammation by stimulating lymphocyte proliferation and the production of pro-inflammatory cytokines [79]. A study by Gerdes *et al.* of 1203 patients hospitalized for psoriasis found an association between the severity of psoriatic lesions expressed on the PASI scale and alcohol consumption [80].

Brenaut *et al.* reported that patients with psoriasis consumed alcohol more frequently and in greater amounts compared to those without dermatosis [81]. Poikolainen *et al.* reported that men with psoriasis consumed an average of 42.9 g of alcohol per day, whereas healthy volunteers drank an average of 21 g of alcohol per day. By contrast, the average annual frequency of alcoholic beverage consumption was 61.6 g in the psoriasis group and 42.6 g in the control group [82]. In subsequent observations, Poikolainen *et al.* observed that in women, the incidence of psoriasis was significantly associated with alcohol consumption, smoking, and the occurrence of negative life events, but the degree of skin surface involvement was only significantly associated with alcohol consumption [83].

Wolk *et al.* also observed a statistically significant association between alcohol intake and the onset of psoriasis in men but not in women. In men, the odds ratio was 3.4 for 5–19 drinks per month and 3.1 for more than 20 drinks per month [84]. Svanström *et al.* also observed that alcohol consumption among patients with psoriasis was significantly above average and concluded that alcohol consumption is associated with a more severe and frequent incidence of psoriasis. They also emphasized that in patients with psoriasis who abuse alcohol, skin lesions are located primarily on the face, groin, knee, and elbow flexures, and acral localization [85, 86].

Smoking and psoriasis

Nicotine is absorbed both through the alveoli in the lungs and through the skin and intestinal mucosa. Nicotine receptors, through which nicotinism effects on the human body are exerted, are located in the central nervous system, as well as in the adrenal medulla, keratinocytes, and cells of the immune system [87]. Patients with psoriasis often declare current or previous nicotine addictions. Smoking may not only worsen the course of already existing psoriasis but also increase the risk of disease [88]. Fortes *et al.* showed that patients who smoked more than 20 cigarettes per day had twice the risk of developing severe psoriasis and a reduced likelihood of periods of remission compared to those who smoked less than 10 cigarettes per day [88]. By contrast, in a retrospective study of patients with psoriasis vulgaris and psoriatic arthritis, Kinahan *et al.* observed no effect of cigarette smoking on clinical responses to general treatment [89]. Nevertheless, it is important to remember that smoking is a risk factor for the development of psoriasis; it is positively associated with the disease at the population level but negatively associated with patients with psoriasis, a phenomenon referred to as the "smoking paradox" in psoriasis [87, 89].

It is important to note that many studies have not confirmed a worse clinical response to biological treatment in patients with psoriasis who smoked cigarettes compared to those who were not smokers [88–94]. Zhou *et al.* pointed out that the inconclusive association between smoking and psoriasis progression and response to treatment is due to the relatively small number of studies on the relationship between psoriasis and cigarette smoking [94].

Conclusions

Psoriasis is a dermatosis that affects 1–3% of the adult population. It has a complex etiopathogenesis that is becoming increasingly well understood. The risk of onset and clinical course of psoriasis is influenced by factors beyond our control (genetic factors) and can be modified to reduce the risk of the disease and possible exacerbations. Therefore, in addition to the correct selection of therapy, it is extremely important to educate patients about the impact of alcohol consumption, smoking, stress, physical activity, and a well-balanced diet on the onset and progression of psoriasis. This literature review confirms that a holistic and multidisciplinary approach is required when treating patients with psoriasis. It seems that the best complement to pharmacotherapy is a well-balanced diet, attention to a healthy lifestyle, and avoidance of common stimulants.

Of course, this review of the literature does not fully address the topic, but it does highlight the validity of Hippocrates' thesis, "Let food be thy medicine, and medicine be thy food" [95].

Conflict of interest

The authors declare no conflict of interest.

References

1. AlQassimi S, AlBrashdi S, Galadari H, Hashim MJ. Global burden of psoriasis–comparison of regional and global epidemiology, 1990 to 2017. Int J Dermatol 2020; 59: 566-71.
2. van Vugt LJ, van den Reek JM, Hannink G, et al. Association of HLA-C* 06: 02 status with differential response to ustekinumab in patients with psoriasis: a systematic review and meta-analysis. JAMA Dermatol 2019; 155: 708-15.
3. Pollock RA, Abji F, Gladman DD. Epigenetics of psoriatic disease: a systematic review and critical appraisal. J Autoimmun 2017; 78: 29-38.
4. Wiśniewski A, Matusiak L, Szczerkowska-Dobosz A, et al The association of ERAP1 and ERAP2 single nucleotide polymorphisms and their haplotypes with psoriasis vulgaris is dependent on the presence or absence of the HLA-C* 06: 02 allele and age at disease onset. Hum Immunol 2018; 79: 109-16.
5. Cornaghi L, Gagliano N, Preis FB, et al. Inside-out and outside-in organotypic normal human skin culture: JAK-STAT pathway is activated after pro-inflammatory psoriatic cytokine exposure. Tissue Cell 2022; 74: 101675.
6. Baliwag J, Barnes DH, Johnston A. Cytokines in psoriasis. Cytokine 2015; 73: 342-50.
7. Zaniewski P, Flisiak I, Chodynicka B. Rola czynnika wzrostu śródbłonka naczyniowego w patogenezie luszczycy. Przegl Dermatol 2010; 97: 406-12.
8. Reich A, Adamski Z, Chodorowska G, et al. Psoriasis. Diagnostic and therapeutic recommendations of the Polish Dermatological Society. Part 1. Dermatol Rev 2020; 107: 92-108.
9. Pogorzelska-Antkowiak A. Diagnostyka łuszczycy za pomocą dermoskopu i mikroskopu konfokalnego. Forum Dermatologicum 2019; 5: 11-3.
10. Roque Ferreira B, Pio-Abreu JL, Reis JP, Figueiredo A. Analysis of the prevalence of mental disorders in psoriasis: the relevance of psychiatric assessment in dermatology. Psychiatr Danub 2017; 29: 401-6.
11. Nowowiejska J, Baran A, Flisiak I. Aberrations in lipid expression and metabolism in psoriasis. Int J Mol Sci 2021; 22: 6561.
12. Kunz M, Simon JC, Saalbach A. Psoriasis: obesity and fatty acids. Front Immunol 2019; 10: 1807.
13. Kamiya K, Kishimoto M, Sugai J, et al. Risk factors for the development of psoriasis. Int J Mol Sci 2019; 20: 4347.
14. Lakuta P, Marcinkiewicz K, Bergler-Czop B, et al. Associations between site of skin lesions and depression, social anxiety, body-related emotions, and feelings of stigmatization in psoriasis patients. Adv Dermatol Allergol 2018; 35: 60-6.
15. Sagi L, Trau H. The Koebner phenomenon. Clin Dermatol 2011; 29: 231-6.
16. Duarte G, Barbosa LO, Rosa MEA. The management of psoriasis through diet. Psoriasis Targets Ther 2012; 2: 45-53.
17. Wolters M. Diet and psoriasis: experimental data and clinical evidence. Br J Dermatol 2005; 153: 706-14.
18. Garbicz J, Całyniuk B, Górski M, et al. Nutritional therapy in persons suffering from psoriasis. Nutrients 2022; 14: 119.
19. Owczarczyk-Saczonek A, Purzycka-Bohdan D, Nedoszytko B, et al. Pathogenesis of psoriasis in the "omic" era. Part III. Metabolic disorders, metabolomics, nutrigenomics in psoriasis in psoriasis. Adv Dermatol Allergol 2020; 37: 452-67.
20. Bremmer S, Van Voorhees AS, Hsu S, et al. Obesity and psoriasis: from the Medical Board of the National Psoriasis Foundation. J Am Acad Dermatol 2010; 63: 1058-69.
21. Uzun L, Kutlu R, Ataseven A, Aydemir FHY. Total oxidant capacity, total antioxidant capacity, ischemic modified albumin, microRNA levels, and their relationship with psoriasis area and severity index. Indian J Dermatol Venereol Leprol 2023; 89: 501-9.
22. Hsieh EA, Chai CM, de Lumen BO, et al. Dynamics of keratinocytes in vivo using HO labeling: a sensitive marker of epidermal proliferation state. J Invest Dermatol 2004; 123: 530-6.
23. Mahyoodeen NG, Crowther NJ, Pillay L, et al. Relationship of visceral fat and adipokines with cardiometabolic diseases in psoriasis. Acta Derm Venereol 2019; 99: 1218-23.
24. Sajja A, Abdelrahman KM, Reddy AS, et al. Chronic inflammation in psoriasis promotes visceral adiposity associated with noncalcified coronary burden over time. JCI Insight 2020; 5: e142534.
25. Kong Y, Zhang S, Wu R, et al. New insights into different adipokines in linking the pathophysiology of obesity and psoriasis. Lipids Health Dis 2019; 18: 171.
26. Kielbowski K, Bakinowska E, Ostrowski P, et al. The role of adipokines in the pathogenesis of psoriasis. Int J Mol Sci 2023; 24: 6390.
27. Wong Y, Nakamizo S, Tan KJ, Kabashima K. An update on the role of adipose tissues in psoriasis. Front Immunol 2019; 10: 1507.
28. Pasierb A, Pietrzak R, Rykowski P, et al. Serum concentrations of adipokines in psoriasis and cardiometabolic risk. Dermatol Rev 2019; 106: 16-24.
29. Kuraitis D, Rosenthal N, Boh E, McBurney E. Macrophages in dermatology: pathogenic roles and targeted therapeutics. Arch Dermatol Res 2022; 314: 133-40.
30. Nakai K. Multiple roles of macrophage in skin. J Dermatol Sci 2021; 104: 2-10.
31. Cnop M. Fatty acids and glucolipotoxicity in the pathogenesis of type 2 diabetes. Biochem Soc Trans 2008; 36: 348-52.
32. Logan AC. Omega-3, omega-6 and psoriasis: a different view. Int J Dermatol 2005; 44: 527-8.
33. Li M, van Esch BC, Wagenaar GT, et al. Pro-and anti-inflammatory effects of short chain fatty acids on immune and endothelial cells. Eur J Pharmacol 2018; 831: 52-9.
34. Yu S, Cheng Y, Zhang L, et al. Treatment with adipose tissue-derived mesenchymal stem cells exerts anti-diabetic effects, improves long-term complications, and attenuates inflammation in type 2 diabetic rats. Stem Cell Res Ther 2019; 10: 333.
35. Takatani N, Taya D, Katsuki A, et al. Identification of paracentrone in fucoxanthin-fed mice and anti-inflammatory effect against lipopolysaccharide-stimulated macrophages and adipocytes. Mol Nutr Food Res 2021; 65: 2000405.
36. Furue M, Kadono T. "Inflammatory skin march" in atopic dermatitis and psoriasis. Inflamm Res 2017; 66: 833-42.
37. Katsimbri P, Korakas E, Kountouri A, et al. The effect of antioxidant and anti-inflammatory capacity of diet on psoriasis and psoriatic arthritis phenotype: nutrition as therapeutic tool? Antioxidants 2021; 10: 157.
38. Honda T, Kabashima K. Current understanding of the role of dietary lipids in the pathophysiology of psoriasis. J Dermatol Sci 2019; 94: 314-20.

39. Venter C, Eyerich S, Sarin T, Klatt KC. Nutrition and the immune system: a complicated tango. Nutrients 2020; 12: 818.
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: 539-47.
41. Gisondi P, Bellinato F, Girolomoni G, Albanesi C. Pathogenesis of chronic plaque psoriasis and its intersection with cardio-metabolic comorbidities. Front Pharmacol 2020; 11: 117.
42. Wu AG, Weinberg JM. The impact of diet on psoriasis [published correction appears in Cutis. 2019 Oct;104(4):241]. Cutis 2019; 104 (2S): 7-10.
43. Al-Mutairi N, Al-Farag S, Al-Mutairi A, Al-Shiltawy M. Comorbidities associated with psoriasis: an experience from the Middle East. J Dermatol 2010; 37: 146-55.
44. Chung M, Bartholomew E, Yeroushalmi S, et al. Dietary intervention and supplements in the management of psoriasis: current perspectives. Psoriasis Targets Ther 2022; 12: 151-76.
45. Ford AR, Siegel M, Bagel J, et al. Dietary recommendations for adults with psoriasis or psoriatic arthritis from the medical board of the National Psoriasis Foundation: a systematic review. JAMA Dermatol 2018; 154: 934-50.
46. Hendel L, Hendel J, Johnsen A, Gudmand-Höyer E. Intestinal function and methotrexate absorption in psoriatic patients. Clin Exp Dermatol 1982; 7: 491-8.
47. De Pessemier B, Grine L, Debaere M, et al. Gut–skin axis: current knowledge of the interrelationship between microbial dysbiosis and skin conditions. Microorganisms 2021; 9: 353.
48. Acharya P, Mathur M. Association between psoriasis and celiac disease: a systematic review and meta-analysis. J Am Acad Dermatol 2020; 82: 1376-85.
49. Egeberg A, Griffiths CEM, Mallbris L, et al. The association between psoriasis and coeliac disease. Br J Dermatol 2017; 177: e329-30.
50. Iqbal T, Zaidi MA, Wells GA, Karsh J. Celiac disease arthropathy and autoimmunity study. J Gastroenterol Hepatol 2013; 28: 99-105.
51. Grode L, Bech BH, Jensen TM, et al. Prevalence, incidence, and autoimmune comorbidities of celiac disease: a nationwide, population-based study in Denmark from 1977 to 2016. Eur J Gastroenterol Hepatol 2018; 30: 83-91.
52. Ludvigsson JF, Lindelöf B, Zingone F, Ciacci C. Psoriasis in a nationwide cohort study of patients with celiac disease. J Invest Dermatol 2011; 131: 2010-6.
53. Assa A, Frenkel-Nir Y, Tzur D, et al. Large population study shows that adolescents with celiac disease have an increased risk of multiple autoimmune and nonautoimmune comorbidities. Acta Paediatr 1992 2017; 106: 967-72.
54. Collin P, Reunala T, Pukkala E, et al. Coeliac disease--associated disorders and survival. Gut 1994; 35: 1215-8.
55. Ventura A, Magazzù G, Greco L. Duration of exposure to gluten and risk for autoimmune disorders in patients with celiac disease. SIGEP Study Group for Autoimmune Disorders in Celiac Disease. Gastroenterology 1999; 117: 297-303.
56. Sategna Guidetti C, Solerio E, Scaglione N, et al. Duration of gluten exposure in adult coeliac disease does not correlate with the risk for autoimmune disorders. Gut 2001; 49: 502-5.
57. Bibbò S, Pes GM, Usai-Satta P, et al. Chronic autoimmune disorders are increased in coeliac disease: a case-control study. Medicine 2017; 96: e8562.
58. Michaëlsson G, Åhs S, Hammarström I, et al. 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: 425-9.
59. Michaelsson G, Gerden B, Ottosson M, et al. Patients with psoriasis often have increased serum levels of IgA antibodies to gliadin. Br J Dermatol 1993; 129: 667-73.
60. Sultan SJ, Ahmad QM, Sultan ST. Antigliadin antibodies in psoriasis. Australas J Dermatol 2010; 51: 238-42.
61. Zamani F, Alizadeh S, Amiri A, et al. Psoriasis and coeliac disease: is there any relationship? Acta Derm Venereol 2010; 90: 295-6.
62. Kolchak NA, Tetarnikova MK, Theodoropoulou MS, et al. Prevalence of antigliadin IgA antibodies in psoriasis vulgaris and response of seropositive patients to a gluten-free diet. J Multidiscip Healthc 2018; 11: 13-9.
63. De Bastiani R, Gabrielli M, Lora L, et al. Association between coeliac disease and psoriasis: Italian primary care multicentre study. Dermatology 2015; 230: 156-60.
64. Qiu J, Yuan Y, Li Y, et al. Discovery of IgG4 anti-gliadin autoantibody as a potential biomarker of psoriasis using an autoantigen array. Proteomics Clin Appl 2020; 14: e1800114.
65. Singh P, Arora A, Strand TA, et al. Global prevalence of celiac disease: systematic review and meta-analysis. Clin Gastroenterol Hepatol 2018; 16: 823-36.e2.
66. Musumeci ML, Nasca MR, Boscaglia S, Micali G. The role of lifestyle and nutrition in psoriasis: crrent status of knowledge and interventions. Dermatol Ther 2022; 35: e15685.
67. Baran A, Kiluk P, Myśliwiec H, Flisiak I. The role of lipids in psoriasis. Dermatol Rev Dermatol 2017; 104: 619-35.
68. Wroński A, Gęgotek A, Skrzydlewska E. Protein adducts with lipid peroxidation products in patients with psoriasis. Redox Biol 2023; 63: 102729.
69. Koeder C, Perez-Cueto FJ. Vegan nutrition: a preliminary guide for health professionals. Crit Rev Food Sci Nutr 2022: 1-38. doi: 10.1080/10408398.2022.2107997.
70. Caso F, Navarini L, Carubbi F, et al. Mediterranean diet and psoriatic arthritis activity: a multicenter cross-sectional study. Rheumatol Int 2020; 40: 951-8.
71. Roncero-Ramos I, Rangel-Zuñiga OA, Lopez-Moreno J, et al. Mediterranean diet, glucose homeostasis, and inflammasome genetic variants: the CORDIOPREV study. Mol Nutr Food Res 2018; 62: e1700960.
72. Dominguez LJ, Veronese N, Di Bella G, et al. Mediterranean diet in the management and prevention of obesity. Exp Gerontol 2023; 174: 112121.
73. 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.
74. Moskalewicz J, Wciórka J, Stokwiszewski J, et al. The prevalence of common mental disorders in the population of adult Poles by sex and age structure–an EZOP Poland study. Psychiatr Pol 2015; 49: 15-27.
75. Opora R, Breska R. Charakterystyka pacjentów przerywających stacjonarną terapię uzależnień uzyskana na podstawie analizy dokumentów medycznych. Stud Paedagog Ignatiana 2018; 21: 199-218.
76. Reich A, Adamski Z, Chodorowska G, et al. Psoriasis. Diagnostic and therapeutic recommendations of the Polish Dermatological Society. Part 2. Dermatol Rev 2020; 107: 110-37.
77. de Fatima Santos Paim de Oliveira M, de Oliveira Rocha B, Duarte GV. Psoriasis: classical and emerging comorbidities. An Bras Dermatol 2015; 90: 9-20.

78. Adışen E, Uzun S, Erduran F, Gürer MA. Prevalence of smoking, alcohol consumption and metabolic syndrome in patients with psoriasis. An Bras Dermatol 2018; 93: 205-11.
79. Rehm J, Gmel Sr GE, Gmel G, et al. The relationship between different dimensions of alcohol use and the burden of disease – an update. Addiction 2017; 112: 968-1001.
80. Gerdes S, Zahl VA, Weichenthal M, Mrowietz U. Smoking and alcohol intake in severely affected patients with psoriasis in Germany. Dermatology 2010; 220: 38-43.
81. Brenaut E, Horreau C, Pouplard C, et al. Alcohol consumption and psoriasis: a systematic literature review. J Eur Acad Dermatol Venereol 2013; 27: 30-5.
82. Poikolainen K, Reunala T, Karvonen J, et al. Alcohol intake: a risk factor for psoriasis in young and middle aged men? BMJ 1990; 300: 780-3.
83. Poikolainen K, Reunala T, Karvonen J. Smoking, alcohol and life events related to psoriasis among women. Br J Dermatol 1994; 130: 473-7.
84. Wolk K, Mallbris L, Larsson P, et al. Excessive body weight and smoking associates with a high risk of onset of plaque psoriasis. Acta Derm Venereol 2009; 89: 492-7.
85. Svanström C, Lonne-Rahm SB, Nordlind K. Psoriasis and alcohol. Psoriasis 2019; 9: 75-9.
86. O'Connor R, Schneller LM, Felicione NJ, et al. Evolution of tobacco products: recent history and future directions. Tob Control 2022; 31: 175-82.
87. Fortes C, Mastroeni S, Leffondré K, et al. Relationship between smoking and the clinical severity of psoriasis. Arch Dermatol 2005; 141: 1580-4.
88. Naldi L. Psoriasis and smoking: links and risks. Psoriasis 2016; 6: 65-71.
89. Kinahan CE, Mazloom S, Fernandez AP. Impact of smoking on response to systemic treatment in patients with psoriasis: a retrospective case-control study. Br J Dermatol 2015; 172: 428-36.
90. Pezzolo E, Naldi L. The relationship between smoking, psoriasis and psoriatic arthritis. Expert Rev Clin Immunol 2019; 15: 41-8.
91. Menter A, Gordon KB, Leonardi CL, et al. Efficacy and safety of adalimumab across subgroups of patients with moderate to severe psoriasis. J Am Acad Dermatol 2010; 63: 448-56.
92. Tillack C, Ehmann LM, Friedrich M, et al. Anti-TNF antibody-induced psoriasiform skin lesions in patients with inflammatory bowel disease are characterised by interferon-γ-expressing Th1 cells and IL-17A/IL-22-expressing Th17 cells and respond to anti-IL-12/IL-23 antibody treatment. Gut 2014; 63: 567-77.
93. Anzengruber F, Augustin M, Radtke MA, et al. Smoking does not alter the therapy response to systemic anti-psoriatic therapies: a two-country, multi-center, prospective, non-interventional study. Acta Derm Venereol 2019; 99: 871-7.
94. Zhou H, Wu R, Kong Y, et al. Impact of smoking on psoriasis risk and treatment efficacy: a meta-analysis. J Int Med Res 2020; 48: 0300060520964024.
95. Witkamp RF, van Norren K. Let thy food be thy medicine ... when possible. Eur J Pharmacol 2018; 836: 102-14.