REVIEW ARTICLE

Non-pharmacological Interventions for Psoriasis: An Updated Review of Diet and Exercise

Mohammad Alahmad

Specialist, department of dermatology & venereology, Ministry of health, Jordan

ABSTRACT

Psoriasis, a chronic inflammatory dermatological condition affecting a substantial global population, exerts a notable detrimental impact on individuals' well-being. It is intricately associated with autoimmune and metabolic coexisting conditions, with its severity influenced by multifaceted factors including immune responses, metabolic dynamics, and lifestyle elements. This comprehensive review accentuates the potential ramifications of dietary modifications and physical activity on psoriasis management. Hypocaloric and ketogenic diets exhibit promising outcomes in weight reduction, whereas the Mediterranean diet, characterized by a plant-centric approach and healthful fats, consistently demonstrates efficacy in reducing severity of psoriasis. Gluten-free diet emerged as particularly beneficial for seropositive patients with plaque or palmoplantar manifestations. Exercise emerges as a protective measure against psoriasis onset, supported by robust evidence from large population-based observational studies. Future research mandates a focus on personalized interventions, mechanistic insights, and standardized protocols for a comprehensive psoriasis management strategy. In summation, this review underscores the substantial impact of psoriasis, and the intricate interplay of immune responses, metabolism, and lifestyle. Dietary modifications and exercise hold potential for effective management, necessitating individualized approaches for improved outcomes.

INTRODUCTION

Psoriasis, a chronic inflammatory skin condition, is identified by red plaques covered with scales, primarily found on the extensor surfaces, scalp, and lumbosacral region.[1] According to the 2019 Global Burden of Disease Study, approximately 4.5 million people worldwide are affected by psoriasis.[2] The incidence of this condition varies across different regions, with higher age-standardized rates seen in high-income countries and territories.[2]

Psoriasis has a considerable impact on the quality of life of patients, as indicated by higher scores on the dermatological life quality index (DLQI) when compared to individuals without the condition.[3-6] Factors that predict a lower quality of life for psoriasis patients include a larger affected body surface area and nail changes associated with the condition.[6]

In addition to the cutaneous manifestations, autoimmune and metabolic comorbidities associated with psoriasis contribute to a decline in patients' quality of life.[7,8] Psoriasis is linked to psychiatric disorders, including a higher susceptibility to major depressive disorder.[9-12] Studies have reported a 13.9% prevalence of moderate-to-severe depression in individuals with psoriasis vulgaris.[13] Psoriasis patients also face a 48% increased risk of unspecified anxiety disorder and a 2.51-fold higher risk of developing anxiety symptoms.[14] All in all, the significant impact of psoriasis on patients' quality of life, and its

Correspondence: *Dr Mohammad Alahmad, Specialist, department of dermatology & venereology, Ministry of health, Jordan*

association with debilitating comorbidities necessitate a comprehensive and holistic approach to health in psoriasis patients, including their lifestyle. In accord, this review aims to summarize evidence on the effectiveness of various diets and physical activities on psoriasis severity and patients' perceived quality of life.

The Immunometabolic Basis of Psoriasis:

Understanding the immunometabolic basis of psoriasis is crucial for exploring how diet and exercise might impact its severity. Dysregulated immune responses involving T cells, cytokines, and inflammatory pathways contribute to the characteristic skin lesions.[15] Moreover, metabolic abnormalities, including insulin resistance and obesity, are prevalent in psoriasis patients, highlighting potential links between immune function and metabolism.[15] This evidence is even highlighted in individuals who do not have metabolic syndrome (MetS). Polic et al. showed a significant difference in markers of insulin resistance, including homeostatic model assessment of insulin resistance (HOMA-IR), as well as a homeostatic model assessment of beta cell function (HOMA-β), in patients without MetS and severe form of psoriasis when compared to mild forms of psoriasis.[16]

Keratinocyte overgrowth, an essential hallmark of psoriatic lesions, requires more energy, which is produced by the upregulation of glucose transporter 1 (GLUT1) and other enzymes in the glycolysis pathway.[17,18] In fact, GLUT1 expression levels correlated with psoriasis area and severity index (PASI) scores, and chemical targeting of GLUT1 in keratinocytes was found to decrease imiquimod (IMQ)-induced psoriasiform hyperplasia in animal models.[19,20] Dependence on GLUT1 is also manifested in infiltrating effector CD4+ and CD8+ T-cells, which are decreased when topical pharmacological inhibition of GLUT1 is performed in skin biopsies from psoriasis patients.[20,21] A critical pathway for inflammation and nutrient sensing is the phosphoinositide 3-kinase (PI3K)/Akt/mTOR cascade.[22] Multiple pieces of evidence indicate that elevated mTOR signaling plays a role in the development of psoriasis by increasing keratinocyte overgrowth and releasing inflammatory cytokines.[22] Specifically, mTOR signaling is essential for T helper effector cell differentiation toward Th1, Th2, and Th17.[23] Moreover, exposure of keratinocytes to IL-1β, IL-17A, IL-22 and TNF-α has been shown to activate the mTOR pathway and promote proliferation and differentiation stall.[24,25] The mTOR pathway could serve as a mediator between insulin resistance and psoriasis, as insulin binding to insulin receptor promotes the production of intracellular secondary messengers that activate numerous downstream targets, including mTOR kinase.[26,27]

Overall, our enhanced understanding of skin and T cell immunometabolism, coupled with the interplay between metabolic and inflammatory pathways, has paved the way for further understanding of the role of lifestyle in psoriasis presentation and progression.

Diets and Psoriasis Severity: Hypocaloric Diet and Weight Loss

As previously shown, insulin resistance and metabolic syndrome predicted severe psoriasis, and in line, evidence showed that weight loss improved psoriatic patients' response to topical low-dose cyclosporine therapy and biologic therapy and had a positive impact on patients' PASI

scores.[28-31] Beside the benefits directly drawn from weight loss, ketogenic low-caloric diets are known to reduce the level of insulin and improve insulin resistance.[32]

Castaldo et al revealed that adherence to ketogenic diet for four weeks caused a significant reduction in IL-2 and IL-1β, along with positive improvement in patients' PASI scores, dermatological life quality index (DLQI) scores, and pain and pruritis scores.[33] A single-arm open-label clinical trial with adult patients who are drug-naïve and are overweight or obese supports these findings. Patients underwent a 10 week 2-phase weight loss program, consisting of a 4-week protein-sparing very low-caloric diet (<500 Kcal/d; 1.2g of protein/kg of ideal body weight/d) and a 6-week balanced hypocaloric (25-30 kcal/kg of ideal body weight/d) Mediterranean-like diet. At 10 weeks, 97.3% and 64.9% of patients had PASI scores' improvements that exceeded 50% and 75%, respectively.[34]

More radical approaches such as bariatric surgery was shown to improve psoriasis, with improvement levels directly correlating with post-operative weight loss.[35] An improved response to biologics was also shown in psoriasis patients who undergoing bariatric surgery.[36] Conflicting evidence exists with a retrospective nationwide registry study showing no significant burden in mean PASI after bariatric surgery.[37] Education regarding diet, nutrition and weight loss are important in the prevention and treatment of psoriasis and should be essential part of first-line intervention with the aim of improving patients' outcomes.

Diets and Psoriasis Severity: The Mediterranean Diet

The Mediterranean diet is a primarily a plant-based diet that is characterized by high intake of vegetables, legumes, fruits, nuts, grains, fish, seafood, and extra virgin olive oil.[38] Based on the NutriNet-Santé program in France, a significant inverse relationship was found between the MEDI-LITE, Mediterranean diet adherence survey, score and severe psoriasis.[39] Caso et al revealed an association between adherence to Mediterranean diet and lower disease activity index for psoriatic arthritis (DAPSA), and a positive correlation between DAPSA and body mass index (BMI).[40] The Mediterranean diet was not only linked to reduced psoriasis severity, but also reduced disease risk. For example, lower consumption of olive oil, berry fruits, fish, seafood, tree nuts, and eggs was observed in psoriasis patients when compared to healthy controls.[41]

Nevertheless, the benefits of the Mediterranean diet in psoriasis cannot be easily traced to a single nutrient. A meta-analysis of 13 randomized controlled trials showed that fish oil supplement did not significantly reduce PASI scores.[42] A more prominent association was established for the intake of fibers, phytosterols, and monounsaturated fatty acids (MUFAs) in psoriasis.[43] The mechanism by which these micronutrients improve psoriasis severity might be mediated via their influence on microbiome position. Fibers including cellulose, hemicellulose, inulin, galactooligosaccharides(GOS), and fructooligosaccharides(FOS) promoted the growth of short-chain fatty acids(SCFAs)-producing bacteria including Bifidobacterium species and *Faecalibacterium Prausantzii*.[44,45] Among SCFAs it is butyrate which was found to promote regulatory T-cell differentiation in mucosal T-cells.[46] Butyrate also activates GPR43

and GPR109a which exert an anti-inflammatory effect.[47] The expression of GPR43 and GPR109a was reduced in keratinocytes and was restored via topical sodium butyrate therapy.[48] Topically applied sodium butyrate led to higher IL-10 and IL-18 levels. While, lowering cytokines that hinder the suppressive activation of Tregs, IL-17, and IL-648. Education the role of the Mediterranean diet and an increase in fiber and healthy fat intake is central to the management of psoriasis and improving patients' outcomes and overall health.

Diets and Psoriasis Severity: The Gluten-Free Diet

Previous studies have shown that 16% of patients with psoriasis vulgaris have IgA and/or IgG antibodies to gliadin.[49] The prevalence of positive anti-tissue transglutaminase antibodies was also higher in psoriasis patients when compared to controls.[50] Treatment of these patients with 3-months of gluten-free diet(GFD) reducing the number of Ki67+ cells in involved dermis, while also decreasing the expression of tissue transglutaminase by 50%.[49] Moreover, Michaëlsson et al reported clinical improvement in 73% of seropositive patients who adhered to GFD for three months.[51] A great improvement in lesions of seropositive patients with palmoplantar psoriasis was also noted on strict adherence to GFD.[52]

A more recent trial by Kolchak et al. found improvement in psoriatic lesions in all gliadin-IgA positive patients, with the decline in PASI and the scale down of pharmaceutical treatment being more pronounced in patients with strong positive gliadin-IgA.[53] The Medical Board of the National Psoriasis Foundation stated "We weakly recommend a gluten-free diet only in patients who test positive for serologic markers of gluten sensitivity".[54] All in all, GFD seems to improve clinical outcomes in psoriasis patients with positive serology. This, along with the increased prevalence of positive serology in psoriasis patients warrant screening for gluten sensitivity in patients with psoriasis, and the use of GFD in the management of psoriasis.

Exercise and Psoriasis Severity

Several large observational cross-sectional and cohort studies showed that patients with psoriasis participated in less physical activity than non-psoriatic patients.[55,56] Low levels of exercise may potentially be a risk factor for psoriasis, but psoriasis itself could be a barrier to physical exercise in psoriasis patients.[57] A survey of 104 patients with severe psoriasis indicated that 40% of these patients avoided sports due to psoriasis alone.[58] Moreover, a cross-sectional study of 378 psoriasis patients showed an inverse correlation between overall quality of life and level of exercise.[59] Furthermore, patients with psoriasis may be less efficient in dissipating heat and have reduced sweating over a two-hour exercise test when compared to healthy controls.[60] Vigorous physical activity was associated with a reduced psoriasis risk in an analysis of the Nurses' Health Study II.[61] Similarly, a Japanese cohort of 487 thousand individuals demonstrated a 13% higher risk for psoriasis in individuals with less than 1 hour of exercise per week.[62]

Overall, conclusive evidence points to the protective impact of exercise in reducing the risk of psoriasis development. However, limited evidence exists on the benefits of exercise and their extent in the management of psoriasis in affected patients. In a study of 303 overweight or obese

patients with PASI scores above 10, those in a 20-week aerobic exercise program and dietary intervention showed a 48% reduction in PASI, compared to a 25.55% reduction in the information counselling group.[63] Apart from this study, there has been a dearth of investigations assessing the efficacy of physical activity in the treatment and control of psoriasis.

Challenges and Future Directions

Despite accumulating evidence highlighting the potential impact of dietary modifications and physical activity on psoriasis severity and patients' quality of life, several challenges remain. The complex interplay between immune responses, metabolism, and lifestyle factors warrants further elucidation. Variability in individual responses to specific diets and exercise regimens calls for personalized approaches. Additionally, long-term adherence to dietary changes and sustained engagement in physical activity present ongoing challenges. Future research should focus on mechanistic insights, standardized intervention protocols, and the integration of lifestyle interventions into comprehensive psoriasis management strategies.

CONCLUSION

In summary, this review underscores psoriasis's significant impact on patients' well-being and its connection to various associated conditions. The interplay of immune responses, metabolism, and lifestyle sheds light on potential mechanisms influencing psoriasis severity. Dietary changes, such as hypocaloric, Mediterranean, and gluten-free diets, hold promise for improving psoriasis outcomes. Furthermore, exercise shows potential as a protective factor against psoriasis, but its treatment role requires further exploration. While progress has been made, challenges like individual variability and sustained adherence persist. A comprehensive approach, encompassing tailored interventions, mechanistic studies, and standardized protocols, is crucial for advancing psoriasis management strategies.

REFERENCES

1. De Rosa G, Mignogna C. The histopathology of psoriasis. Reumatismo 2007; 59 Suppl 1:46-48.
2. Damiani G, Bragazzi NL, Karimkhani Aksut C, et al. The Global, Regional, and National Burden of Psoriasis: Results and Insights From the Global Burden of Disease 2019 Study. Front Med (Lausanne) 2021; 8:743180.
3. Chaptini C, Quinn S, Marshman G. Durable dermatology life quality index improvements in patients on biologics associated with psoriasis areas and severity index: a longitudinal study. Australas J Dermatol 2016; 57:e72-75.
4. Abrouk M, Nakamura M, Zhu TH, Farahnik B, Koo J, Bhutani T. The impact of PASI 75 and PASI 90 on quality of life in moderate to severe psoriasis patients. J Dermatolog Treat 2017; 28:488-91.
5. Mattei PL, Corey KC, Kimball AB. Psoriasis Area Severity Index (PASI) and the Dermatology Life Quality Index (DLQI): the correlation between disease severity and psychological burden in patients treated with biological therapies. J Eur Acad Dermatol Venereol 2014; 28:333-37.
6. Sendrasoa FA, Razanakoto NH, Ratovonjanahary V, et al. Quality of Life in Patients with Psoriasis Seen in the Department of Dermatology, Antananarivo, Madagascar. Biomed Res Int 2020; 2020:9292163.
7. Hao Y, Zhu Y, Zou S, et al. Metabolic Syndrome and Psoriasis: Mechanisms and Future Directions. Frontiers in Immunology. 2021; 12. https://www.frontiersin.org/articles/10.3389/fimmu. 2021.711060.
8. Davidovici BB, Sattar N, Jörg PC, et al. Psoriasis and Systemic Inflammatory Diseases: Potential Mechanistic Links between Skin Disease and Co-Morbid Conditions. J Invest Dermatol 2010; 130:1785-96.
9. Ferreira BIRC, Abreu JLPDC, Reis JPG Dos, Figueiredo AMDC. Psoriasis and Associated

Psychiatric Disorders: A Systematic Review on Etiopathogenesis and Clinical Correlation. J Clin Aesthet Dermatol 2016; 9:36-43.
10. Dalgard FJ, Gieler U, Tomas-Aragones L, et al. The psychological burden of skin diseases: a cross-sectional multicenter study among dermatological out-patients in 13 European countries. J Invest Dermatol 2015; 135:984-91.
11. Jensen P, Ahlehoff O, Egeberg A, Gislason G, Hansen PR, Skov L. Psoriasis and New-onset Depression: A Danish Nationwide Cohort Study. Acta Derm Venereol 2016; 96:39-42.
12. Kurd SK, Troxel AB, Crits-Christoph P, Gelfand JM. The risk of depression, anxiety, and suicidality in patients with psoriasis: a population-based cohort study. Arch Dermatol 2010; 146:891-95.
13. Tian Z, Huang Y, Yue T, et al. A Chinese cross-sectional study on depression and anxiety symptoms in patients with psoriasis vulgaris. Psychol Health Med 2019; 24:269-80.
14. Jalenques I, Bourlot F, Martinez E, et al. Prevalence and Odds of Anxiety Disorders and Anxiety Symptoms in Children and Adults with Psoriasis: Systematic Review and Meta-analysis. Acta Derm Venereol 2022; 102: adv00769.
15. Sarandi E, Krueger-Krasagakis S, Tsoukalas D, et al. Psoriasis immunometabolism: progress on metabolic biomarkers and targeted therapy. Front Mol Biosci 2023; 10: 1201912.
16. Polic MV, Miskulin M, Smolic M, et al. Psoriasis Severity-A Risk Factor of Insulin Resistance Independent of Metabolic Syndrome. Int J Environ Res Pub Health 2018; 15. DOI:10.3390/IJERPH15071486.
17. Cibrian D, de la Fuente H, Sánchez-Madrid F. Metabolic Pathways That Control Skin Homeostasis and Inflammation. Trends Mol Med 2020; 26:975-86.
18. Liu YZ, Xu MY, Dai XY, et al. Pyruvate Kinase M2 Mediates Glycolysis Contributes to Psoriasis by Promoting Keratinocyte Proliferation. Front Pharmacol 2021; 12. DOI:10.3389/FPHAR.2021.765790/FULL.
19. Nestle FO, Kaplan DH, Barker J. Psoriasis. N Engl J Med 2009; 361:496-509.
20. Zhang Z, Zi Z, Lee EE, et al. Differential glucose requirement in skin homeostasis and injury identifies a therapeutic target for psoriasis. Nat Med 2018; 24:617-27.
21. Almeida L, Lochner M, Berod L, Sparwasser T. Metabolic pathways in T cell activation and lineage differentiation. Semin Immunol 2016; 28:514-24.
22. Buerger C. Epidermal mTORC1 Signaling Contributes to the Pathogenesis of Psoriasis and Could Serve as a Therapeutic Target. Front Immunol 2018; 9: 2786.
23. Chi H. Regulation and function of mTOR signalling in T cell fate decisions. Nature Reviews Immunology 2012; 12:325-38.
24. Patel AB, Tsilioni I, Weng Z, Theoharides TC. TNF stimulates IL-6, CXCL8 and VEGF secretion from human keratinocytes via activation of mTOR, inhibited by tetramethoxyluteolin. Exp Dermatol 2018; 27:135-43.
25. Mitra A, Raychaudhuri SK, Raychaudhuri SP. IL-22 induced cell proliferation is regulated by PI3K/Akt/mTOR signaling cascade. Cytokine 2012; 60:38-42.
26. Ardestani A, Lupse B, Kido Y, Leibowitz G, Maedler K. mTORC1 Signaling: A Double-Edged Sword in Diabetic β Cells. Cell Metab 2018; 27:314-31.
27. Guillén C, Benito M. MTORC1 overactivation as a key aging factor in the progression to type 2 diabetes mellitus. Front Endocrinol (Lausanne) 2018; 9:412239.
28. Naldi L, Conti A, Cazzaniga S, et al. Diet and physical exercise in psoriasis: a randomized controlled trial. Brit J Dermatol 2014; 170:634-42.
29. Alotaibi HA. Effects of Weight Loss on Psoriasis: A Review of Clinical Trials. Cureus 2018; 10. DOI: 10.7759/CUREUS.3491.
30. Guida B, Napoleone A, Trio R, et al. Energy-restricted, n-3 polyunsaturated fatty acids-rich diet improves the clinical response to immuno-modulating drugs in obese patients with plaque-type psoriasis: a randomized control clinical trial. Clin Nutr 2014; 33:399-405.
31. Al-Mutairi N, Nour T. The effect of weight reduction on treatment outcomes in obese patients with psoriasis on biologic therapy: a randomized controlled prospective trial. Expert Opin Biol Ther 2014; 14:749-56.
32. Yuan X, Wang J, Yang S, et al. Effect of the ketogenic diet on glycemic control, insulin resistance, and lipid metabolism in patients with T2DM: a systematic review and meta-analysis. Nutr Diabetes 2020; 10. DOI:10.1038/S41387-020-00142-Z.
33. Castaldo G, Pagano I, Grimaldi M, et al. Effect of Very-Low-Calorie Ketogenic Diet on Psoriasis Patients: A Nuclear Magnetic Resonance-Based Metabolomic Study. J Proteome Res 2021; 20:1509-21.
34. Castaldo G, Rastrelli L, Galdo G, Molettieri P, Rotondi Aufiero F, Cereda E. Aggressive weight-loss program with a ketogenic induction phase for the treatment of

chronic plaque psoriasis: A proof-of-concept, single-arm, open-label clinical trial. Nutrition 2020; 74. DOI:10.1016/J.NUT.2020.110757.
35. Romero-Talamás H, Aminian A, Corcelles R, Fernandez AP, Schauer PR, Brethauer S. Psoriasis improvement after bariatric surgery. Surg Obesity Rel Dis 2014; 10:1155-59.
36. Chen YY, Lan CCE. Improved therapeutic response to biologic treatment after bariatric surgery: Experience from an obese patient with psoriasis. Dermatologica Sinica 2020; 38:51.
37. Laskowski M, Schiöler L, Ottosson J, et al. Impact of Bariatric Surgery on Moderate to Severe Psoriasis: A Retrospective Nationwide Registry Study. Acta Derm Venereol 2021; 101.
38. Schwingshackl L, Morze J, Hoffmann G. Mediterranean diet and health status: Active ingredients and pharmacological mechanisms. Br J Pharmacol 2020; 177: 1241-57.
39. Phan C, Touvier M, Kesse-Guyot E, et al. Association Between Mediterranean Anti-inflammatory Dietary Profile and Severity of Psoriasis: Results From the NutriNet-Santé Cohort. JAMA Dermatol 2018; 154: 1017-24.
40. Caso F, Navarini L, Carubbi F, et al. Mediterranean diet and Psoriatic Arthritis activity: a multicenter cross-sectional study. Rheumatol Int 2020; 40:951-58.
41. Ingkapairoj K, Chularojanamontri L, Chaiyabutr C, Silpa-archa N, Wongpraparut C, Bunyaratavej S. Dietary habits and perceptions of psoriatic patients: Mediterranean versus Asian diets. J Dermatolog Treat 2022; 33:2290-96.
42. Yang SJ, Chi CC. Effects of fish oil supplement on psoriasis: a meta-analysis of randomized controlled trials. BMC Complement Altern Med 2019; 19: 354.
43. Madden SK, Flanagan KL, Jones G. How lifestyle factors and their associated pathogenetic mechanisms impact psoriasis. Clin Nutr 2020; 39:1026-40.
44. Scott KP, Martin JC, Duncan SH, Flint HJ. Prebiotic stimulation of human colonic butyrate-producing bacteria and bifidobacteria, in vitro. FEMS Microbiol Ecol 2014; 87:30-40.
45. Fagundes RR, Bourgonje AR, Saeed A, et al. Inulin-grown Faecalibacterium prausnitzii cross-feeds fructose to the human intestinal epithelium. Gut Microbes 2021; 13.
46. Furusawa Y, Obata Y, Fukuda S, et al. Commensal microbe-derived butyrate induces the differentiation of colonic regulatory T cells. Nature 2013 504:7480 2013; 504:446-50.
47. Cheng D, Xu JH, Li JY, et al. Butyrate ameliorated-NLRC3 protects the intestinal barrier in a GPR43-dependent manner. Exp Cell Res 2018; 368:101-10.
48. Krejner A, Bruhs A, Mrowietz U, et al. Decreased expression of G-protein-coupled receptors GPR43 and GPR109a in psoriatic skin can be restored by topical application of sodium butyrate. Arch Dermatol Res 2018; 310:751-58.
49. Michaëlsson G, Åhs S, Hammarström I, Lundin IP, Hagforsen E. Gluten-free diet in psoriasis patients with antibodies to gliadin results in decreased expression of tissue transglutaminase and fewer Ki67+ cells in the dermis. Acta Derm Venereol 2003; 83:425-29.
50. De Bastiani R, Gabrielli M, Lora L, et al. Association between coeliac disease and psoriasis: Italian primary care multicentre study. Dermatol 2015; 230:156-60.
51. Michaëlsson G, Gerdén B, Hagforsen E, et al. Psoriasis patients with antibodies to gliadin can be improved by a gluten-free diet. Brit J Dermatol 2000; 142:44-51.
52. Michaëlsson G, Kristjánsson G, Pihl Lundin I, Hagforsen E. Palmoplantar pustulosis and gluten sensitivity: a study of serum antibodies against gliadin and tissue transglutaminase, the duodenal mucosa and effects of gluten-free diet. Brit J Dermatol 2007; 156:659-66.
53. Kolchak NA, Tetarnikova MK, Theodoropoulou MS, Michalopoulou AP, Theodoropoulos DS. Prevalence of antigliadin IgA antibodies in psoriasis vulgaris and response of seropositive patients to a gluten-free diet. J Multidiscip Health 2018; 11: 13.
54. 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.
55. Schwarz PEH, Pinter A, Melzer N, Barteczek P, Reinhardt M. ERAPSO: Revealing the High Burden of Obesity in German Psoriasis Patients. Dermatol Ther (Heidelb) 2019; 9: 579-87.
56. Wilson PB. Prevalence of weight loss attempts and behaviors used by individuals with psoriasis in the United States population. J Dermatolog Treat 2017; 28: 515-19.
57. Yeroushalmi S, Hakimi M, Chung M, Bartholomew E, Bhutani T, Liao W. Psoriasis and Exercise: A Review. Psoriasis: Targets and Therapy 2022; 12: 189.
58. RAMSAY B, O'REAGAN M. A survey of the social and psychological effects of psoriasis. Brit J Dermatol

1988; 118: 195-201.
59. Khan JM, Rathore MU, Tahir M, Abbasi T. Dermatology Life Quality Index In Patients Of Psoriasis And Its Correlation With Severity Of Disease. J Ayub Med Coll Abbottabad 2020; 32: 64-67.
60. LEIBOWITZ E, SEIDMAN DS, LAOR A, SHAPIRO Y, EPSTEIN Y. Are psoriatic patients at risk of heat intolerance? Brit J Dermatol 1991; 124: 439-42.
61. Frankel HC, Han J, Li T, Qureshi AA. The association between physical activity and the risk of incident psoriasis. Arch Dermatol 2012; 148: 918-24.
62. Goto H, Nakatani E, Yagi H, Moriki M, Sano Y, Miyachi Y. Late-onset development of psoriasis in Japan: a population-based cohort study. JAAD Int 2020; 2: 51-61.
63. Naldi L, Conti A, Cazzaniga S, et al. Diet and physical exercise in psoriasis: a randomized controlled trial. Brit J Dermatol 2014; 170: 634-42.