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Research Article

Assessment of IL-32 Serum Levels and Demographic Factors in Psoriasis Patients

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Article Info
Article history:
Received 18 -8-2025
Received in revised form 27-8-2025
Accepted 11-9-2025
Available online 30 -9 -2025
Keywords: Psoriasis, IL-32 serum level, ELISA.

Abstract:

Background: Psoriasis is a chronic immune-mediated inflammatory skin disorder marked by excessive keratinocyte proliferation and T-cell infiltration of the dermis and epidermis. Interleukin-32 (IL-32), a proinflammatory cytokine, has been implicated in psoriasis pathogenesis.

Materials and Methods: A case-control study was conducted on 96 psoriasis patients (41 men, 55 women) and 50 healthy controls (26 men, 24 women) recruited from Imam Hassan Al-Mujtaba Hospital, Karbala. Data on age, gender, height, weight, psoriasis type, and family history were collected via questionnaire. Serum IL-32 levels were measured using ELISA. Statistical analysis used SPSS version 26 with t-tests for mean comparisons and Chi-square tests for categorical variables.

Results: Patients were significantly older, with 36.5% aged 42–54 years and 33.3% aged 55–67 years, compared with a control peak of 36% in the 29–41-year group. Obesity was more prevalent among patients (78.1%). Plaque psoriasis was the dominant clinical type (69%), followed by guttate psoriasis, while nail involvement was rare. Absence of a family history was significantly more common than presence ($p = 0.000$). Serum IL-32 levels were markedly higher in psoriasis patients than in controls.

Conclusion: Psoriasis is significantly associated with older age and higher body-mass index, whereas sex and family history show no meaningful effect. Elevated IL-32 supports its role in the inflammatory milieu and highlights its potential as an immune biomarker and therapeutic target. Its involvement in systemic inflammation may link skin manifestations to comorbidities such as psoriatic arthritis and cardiovascular disease. Future studies should investigate IL-32 isoform-specific expression and evaluate IL-32 levels longitudinally before and after biologic therapy to clarify its value as a treatment-response indicator.

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Peer review under responsibility of Iraqi Academic Scientific Journal and University of Kerbala.

Introduction

Psoriasis is a chronic disease that is mediated through defect in the immune system. It is emerged by a polygenetic trait in addition to environmental factors like trauma, infections or some medications.Underlying pathophysiology influences T-cells and their interplay with dendritic cells and cells responsible for innate immunity such as keratinocytes [1].

Psoriasis reveals a varied frequency across populations worldwide, it found in 2.5% of European populations, 0.05 to 3% in African populations and from 0.1 to 0.5% in Asian ones [2].

Psoriasis is not only expressed as cutaneous lesions, even so it is associated with systemic inflammations and complications like cardiovascular diseases, obesity, and metabolic syndrome [3, 4].

Demographic and structural factors including age, sex, body mass index (BMI), and family history had been proven to affect both clinical manifestations along with severity of psoriasis [5]. High BMI values have been positively associated with augmented disease severity and specific clinical aspects, while family history with this disease is a well-established risk factor for disease onset and recurrence [6].

It's pathogenesis involves a complex interplay between genetic susceptibility, environmental triggers, and dysregulated immunological responses, particularly involving cytokines like TNF-α, IL-17, and IL-23 [7].

Among the emerging cytokines engaged in inflammatory disorders, Interleukin-32 (IL-32) received a lot of attention for its vigorous pro-inflammatory properties. It is produced by numerous immune and non-immune cells, including T cells, natural killer cells, as well as epithelial cells, and is known to trigger the expressions of other cytokines like TNF-α, IL-6, and IL-8 [8].

Al-Shobaili and Rasheed have reported increased IL-32 levels and its isoforms (particularly IL-32α) in peripheral blood mononuclear cells of patients have chronic plaque psoriasis, they proposed a potential role in psoriasis pathogenesis and immune regulations [4].

However, regardless these findings, the exact roles of IL-32 in psoriasis remain underexplored, particularly in relation to its utility as a biomarker for psoriasis activity or severity. Given its capacity to modify both innate and adaptive immunological responses, IL-32 may serve as a valuable biomarker for systemic inflammations in psoriatic patients. Furthermore, grasping its expression patterns in different clinical subtypes of psoriasis could offer insights into disease heterogeneity and immunological disorders [7].

The present study aims to estimate levels of IL-32 in psoriasis cases and to explore its possible utility as an immune biomarker reflective of disease activity. Furthermore, the study examines key demographic and variables for psoriasis patients that included: age, sex, BMI, and family history, which may influence immune response and cytokine expression.

Materials and Methods

Study settings and samples collection

The current case-control investigation included 96 psoriasis patients were collected from Imam Hassan Al-Mujtaba Hospital/Karbala province and 50 healthy individuals as a control group, they were selected using a simple random sampling method. The study period extended from December 2024 to March 2025.A questionnaire was given to all patients to document their personal information such as age, sex, height, and weight, type of psoriasis, and the presence or absence of a family history of the disease. Venous blood samples have been collected from all

individuals by standard aseptic techniques. To separate the serum, blood was drawn into red-top vacutainer tubes (without anticoagulant), which allow normal clotting. The samples were let without undisturbed at room temperature for 20–30 mins to make sure complete clot formation, followed by centrifugation at 2000 ×g for ten mins. The produced sera were stored at –20°C until analysis.

Assessment of IL-32 serum Level

Serum levels of IL-32 have been determined using the ELISA technique by following the manufacturer's instructions for used kit (SUNLONG/China). The IL-32 levels have been assessed using ELISA kit (SUNLONG/China; catalog No. SL0994Hu), according to manufacturer's instructions. Briefly, standards and serum specimens have been added to microplate wells pre-coated with anti-IL-32 antibodies. After incubation and wash steps, the biotinylated detection antibodies and streptavidin-HRP conjugated were applied. The reaction was visualized by TMB substrate, and absorbance had been measured at 450nm. Concentrations were evaluated based on standard curve .

Determination of Body mass index

The values body mass index (BMI) was calculated using BMI formula that mentioned below: [9]

BMI = weight (kg) / height^2 (m)^2

The BMI status grouped according to World Health Organization, which classified individuals as Underweight if their BMI values at <18, Normal if their BMI values at 18-24.9, Overweight if their BMI values at 25-29.9 and Obesity if their BMI values at 30-39.9.

Inclusion criteria

All selected individuals must have a confirmed diagnosis of psoriasis without other concomitant dermatological conditions. The cohort is specifically classified into one of the following three clinical subtypes: Plaque, Guttate and Nail.

Exclusion criteria

Subjects with any chronic medical conditions that could potentially confound the analysis or affect immune response were excluded from the current study. These medical conditions includes: Diabetes mellitus (Type 1 and Type 2), malignancies such as any form of diagnosed cancer, hypertension or hypotension (persistently high or low blood pressure), current or recent use of antibiotics.

Ethical approval

Ethical approval has obtained from Karbala Sciences College Ethical Committee. Also, verbal approval has been taken from the patients before taking the sample. During sample collection, health measures and safety had been taken.

Statistical analysis

The data analysis for current investigation was processed using the Statistical Package for the Social Sciences software, version 26 (IBM, SPSS, Chicago, Illinois, USA). Descriptive statistics was performed on the participants' data of each group. Data was analysed for means, and the standard deviation was computed for the continuous variables, whereas frequency was used for computing the qualitative data. The mean of the investigated biomarkers were compared between the studied groups using t-Test; Chi-Square analysis was employed to significant compare between percentages; the results of all hypothesis tests with p-values <0.05 (two-side) were considered to be statistically significant. In addition, SPSS program and Microsoft Excel 2010 program was applied to draw the figures in the present investigation.

Results

Table -1 shows the demographic data for psoriasis patients and the control group. The mean age (46.64±11.877) of the patients was significantly (p=0.0001) higher than that of the healthy controls (36.90±11.578). Both of patients and controls were divided according to their ages into four age categories 16-28y, 29-41y, 42-54, and 55-67. The results of the statistical analysis within the patient group showed that the highest percentage of patients was significantly (p=0.0002) higher within the two older age groups, 42-54 and 55-67 with percent of (36.5% and 33.3%, respectively); while the comparison within the control group showed that the highest percentage was in the age group 29-41 years, at 36%. Additionally, significant differences were found between patients and control comparison in all age categories, except 42-54 age category, (p=0.2041).

Regarding the distribution of study population according to sex, patient's males and females proportions were 42.7% and 57.3%, respectively; while the proportion of males and females in the control group was 52% and 48%, respectively. The results of the statistical analysis didn't reveal any significant differences (p=0.1768) in the distribution of sexes, nor there no any statistical significance for sex in the development of psoriasis.

Table (1): The Demographic data of study population

Study groups	Age groups No. (%)				P value
	16-28y	29-41y	42-54	55-67	
Patients (n=96)	8 (8.3%)	21(21.9%)	35 (36.5%)	32 (33.3%)	0.0002*
Control (n=50)	14 (28%)	18 (36%)	13 (26%)	5 (10%)	0.0026*
P value	0.0009*	0.0469*	0.2041^{NS}	0.0005*	
Age (Year) Mean ± S.D					
Patients (n=96)	46.64 ± 11.877				0.0001*
Control (n=50)	36.90 ± 11.578				
sex					
Study groups	Male	Female	Odd (CI 95%)		P value
Patients (n=96)	41 (42.7%)	55 (57.3%)	0.6802 (0.3888 - 1.1899)		0.1768^{NS}
Control (n=50)	26 (52%)	24 (48%)			

*Mean highly significant difference at the 0.05 level
NS: Non-significant p>0.05

In term of distribution of study population according to BMI score. Both patients and controls have been divided according to their BMI values into three groups: normal, overweight, and obesity groups as presented in Figure (1). The percentage (48%) of control individuals who have normal weight was significantly (p=0.0001) higher than that of patients (8.33%), while the percentage of obese patients (78.13%) was significantly (p=0.0002) higher than that of control (38%). On the other hand, no significant (p=0.8474) differences were found in the overweight category when comparing patients and control.

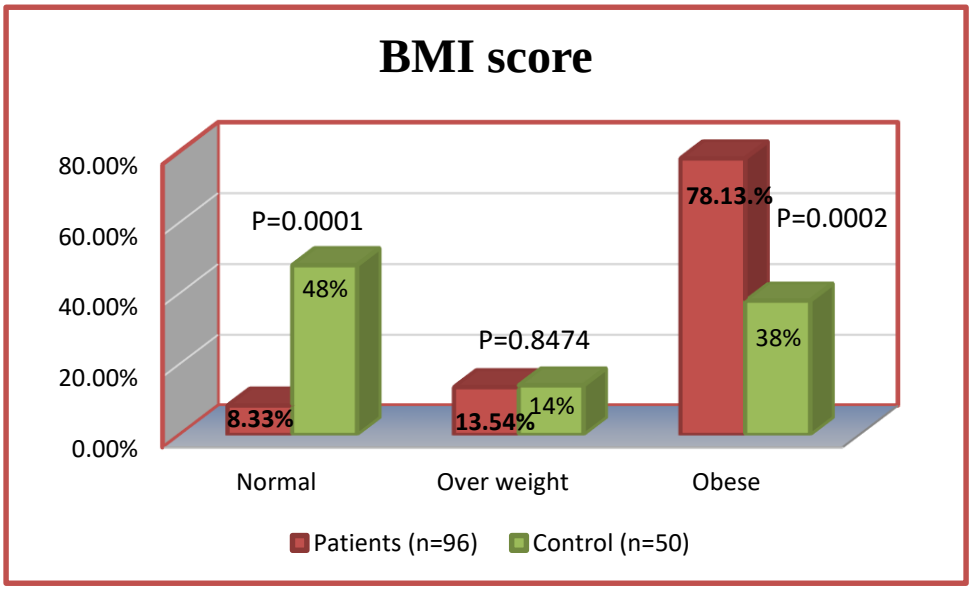

Figure (1): Distribution of BMI score in study population

Figure (2) displays the distribution of psoriasis types among the study patients. Where three main types of psoriasis have been diagnosed, including: plaque, guttate, and nail. This figure indicates that plaque psoriasis was the most common type, accounting for 69% of cases, while nail psoriasis was the least common, accounting for 6%. Guttate psoriasis was intermediate between them, accounting for 25%.

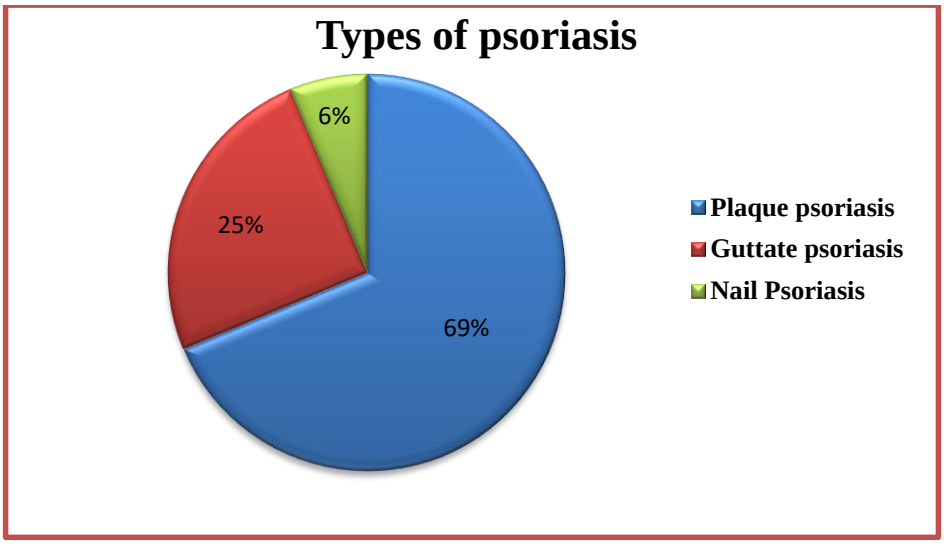

Figure (2): Distribution of psoriasis types in patients

Figure (3) summarizes the relationship between family history and types of psoriasis. The results show that the percentage of patients who do not have a family history of psoriasis is higher than those who have a family history of all types of psoriasis with (p=0.000).

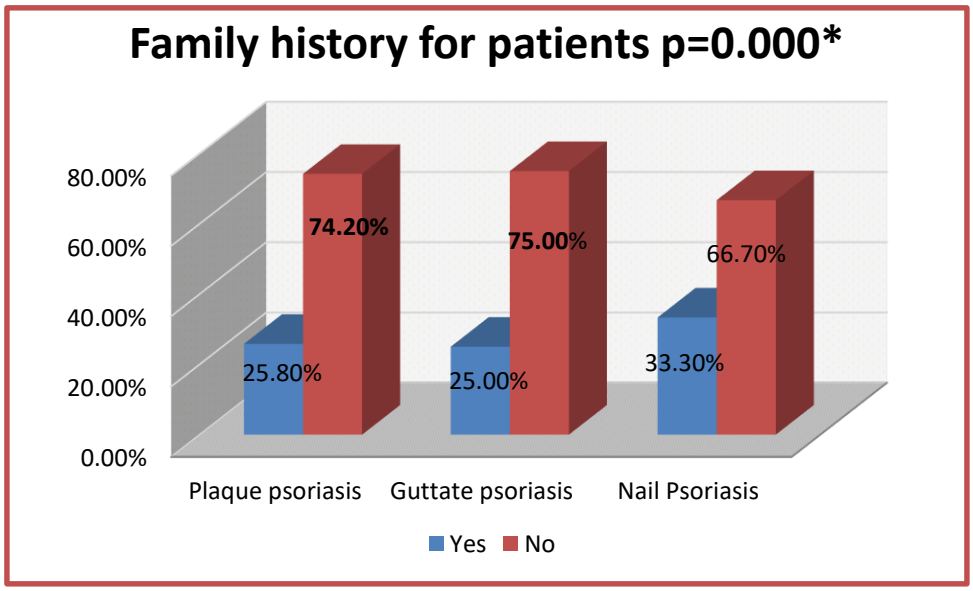

Figure (3): Association of family history with psoriasis types in patients

A comparison of IL-32 levels between patients and control subjects is shown in Table (2). The result of the statistical analysis indicate that IL-32 was significantly higher in psoriasis patients than in healthy individuals at a significance level of (p=0.000).

Table (2): Comparison the Immunological marker between patients and control

Immunological parameter	Mean ± SD		P value
	Patients (n=96)	Control (n=50)	
IL-32	50.1268 ± 13.22231	33.3317 ±18.39517	0.000*

*Significant difference at the 0.05 level by T-test.

The distribution of IL-32 according to psoriasis types is illustrated in Table (3). Although there was a variation between it's levels, the results of the statistical analysis using the ANOVA test did not indicate a significant difference between the types of psoriasis in patients at the probability level of 0.05.

Table (3): Impact of psoriasis types on Immunological parameters in cases.

Immunological parameters	Psoriasis type for patients Mean ± Sd.			*P value*
	Plaque psoriasis (n=66)	Guttate psoriasis (n=24)	Nail Psoriasis (n=6)	
IL-32	49.8843 ± 13.7442	51.5937 ± 13.45484	46.9275 ± 3.14368	0.720^{NS}

NS: Non-significant difference at the 0.05 level by one way ANOVA test.

Discussion:

The age distribution in present study demonstrated a significant prevalence of psoriasis among older age groups (both 42-54 and 55-67y), as found in Table (1). This finding agrees with previous investigations that revealed a higher prevalence of psoriasis in middle-aged and elderly patients [10, 11].

This high prevalence of psoriasis among older patients can be attributed age-related immune system changes, such as escalated systemic inflammations and changed immune responses, which could lead to autoimmune diseases like psoriasis; this opinion supported by Yu *et al.*, who revealed the roles of chronic inflammations and immune decline in exacerbating autoimmune disease like psoriasis [12].

In addition, most individuals at this age may suffer from chronic diseases such as diabetes, high blood pressure, and obesity, which may cause a higher incidence of psoriasis in these groups in particular. Kimball *et al.*, stated that multiple and age-appropriate comorbidities are related with psoriasis and could influence psoriasis treatments options [13].

Besides, some lifestyle and environmental factors, such as smoking, prolonged

exposure to stress, and infections, may present a further justify for increasing prevalence of psoriasis in older individuals, because these triggers accumulate by time. Saavedra *et al.* emphasized the impacts of lifestyle factors and accumulated environmental exposures over time [14].

However, Parisi noted a bimodal age distribution, with peaks in early 30s and 60s [15].

These differences between studies may be due to variations in study population, geographic factors, and environmental exposures of psoriasis patients.

As for sex distribution, the current study demonstrated a higher prevalence of psoriasis among females (57.3%) than males (42.7%), though this difference isn't significant. This finding contrasts with previous finding of Lubrano, which revealed a higher prevalence in males. This variation may be as a result of genetic predispositions, hormonal variations, or cultural variance in healthcare-seek behavior [16].

According to present finding, obesity is deeming as a significant risk factor for psoriasis, where the prevalence of obesity among psoriasis cases is notably higher than healthy individuals, as demonstrated in figure (1). Our finding agrees with research conducted by Vata *et al.,* who referred to an association between psoriasis and obesity, they are stated that psoriasis is related to obesity in a two-way manner, as each could precipitate in developing of the other [17].

Various adipose tissues-secreted adipokines that were revealed to be increased in obese psoriasis individuals, showing similar mechanisms of action for those underlying pathogenesis of psoriasis. Excess body weight could affect not only the healing response in psoriasis, but also the adverse event, leading to minimize patient's compliance. Where the inflammatory status associated with obesity characterized by increased levels of TNF-alpha and IL-6, could contribute to pathogenesis of psoriasis [18].

However, Alizadeh *et al.*, stated that the association between BMI and psoriasis severity isn't straightforward [19].

Paroutoglou *et al.,* stated that while obesity may exacerbate psoriasis, the exact mechanisms and extent of this association remain unclear [20].

Plaque psoriasis, which accounting for 69% of cases in current study (figure-2), is widely known as a predominant form for its association with genetic predisposition as well as environmental triggers like stress and infections [21].

The present study is in line with previous investigations, which reported that Plaque psoriasis being most common type of psoriasis, accounting for about 70%-90% of cases [21, 22]. Plaque psoriasis is characterized by raise, inflame, and scaly patches of skin, often enveloped with silvery-white scales; they are typically manifest on elbows, knees, scalp, and lower back, but they could present anywhere on the body [23].

The present finding of 25% prevalence rate of Guttate psoriasis agrees with Telfer *et al.*, who revealed that 26% of psoriasis patients had acute guttate psoriasis [22]. Guttate psoriasis, a distinct type of psoriasis, it is more common in children and adolescents [24]. It is typically associated with streptococcal infections and is more prevalent in younger population (Leung *et al.,* 2023). Nail psoriasis, the least common type at 5%, is often underdiagnosed or overlooked in clinical settings, as its symptoms could be mistaken for other nail diseases or may arise with no skin involvement [25].

However, our study conflicts with study of Rajashekar *et al.,* which demonstrated a higher prevalence of nail psoriasis ranging between (10-78%) in their cohort study, their higher prevalence may be due to the

inclusion of patients with psoriatic arthritis, where nail implication is more common [26]. The variations in prevalence proportions among studies can occur due to variances in study population, diagnostic criteria, and environmental factors. These contradictions underscored the significance of considering demographic and methodological variations when interpreting the prevalence rates.

The previous feedback that patients have no family history of psoriasis exhibited higher prevalence rates in all types of psoriasis support with existing finding (figure-3); while genetic predisposition is an important factor in psoriasis development, the environmental determinants and lifestyle factors also have a vital role in sporadic cases. Investigations have revealed that patients with no family history may develop psoriasis responding to external factors such as stress, infection, and metabolic disorders [27].

Interestingly, the investigation had highlighted variances in psoriasis severity and onset between familial and sporadic patients; where familial cases is often corresponding with earlier onset and more severe symptoms, while sporadic cases may appear later in life and with mild symptoms [28]. This distinction underlines the multifactorial nature of psoriasis, where both genetic and non-genetic factors interact to affect psoriasis expression.

The present findings highlighted significant alterations in cytokine levels in psoriasis patients compared with controls. Increased IL-32 levels underscored its impacts as pro-inflammatory factor in disease's pathogenesis. The present result agrees with previous investigation that reported increased IL-32 levels in lesion psoriatic skin, and highlighted its potential role in inflammations [29]. Also, our study aligns with the study of Al-Shobaili and Rasheed, which revealed that IL-32 are significantly increased in psoriasis patients, sharing in the inflammatory responses through Th1 and Th17 pathways [4].

The non-significant differences in IL-32 levels among the psoriasis types may be due to common mechanisms in the inflammation process across psoriasis types, which may lead to increased levels of these cytokines in all psoriasis patients, regardless of the type of psoriasis they have, whether it is Plaque psoriasis, Guttate psoriasis, or Nail psoriasis. Psoriasis is a multifactorial autoimmune condition that characterized by immune dysregulation, and immunological markers like IL-32, IL-38, and IL-39 playing critical roles in these inflammatory processes, but may not clearly vary according to clinical type [30]. Wallimann and Schenk emphasized that IL-32 acts as a pro-inflammatory cytokine in several inflammatory disorders, including psoriasis, regardless its particular correlation with subtypes [31].

Conclusion:

The present case-control investigation revealed a statistically significant association between increased IL-32 levels and presence of psoriasis. The strong association between obesity and psoriasis that indicated by the markedly increased percent of obese patients boosts the role of metabolic status in modulating inflammatory pathways. Also, the present study showed that plaque psoriasis emerged as the most prevalent clinical type. Remarkably, a high percent of patients lacked a family history with psoriasis, proposing that while genetic predisposition is substantial, non-genetic factors may do outstanding role in expressing this disease in some populations. Importantly, the significant increase of IL-32 levels in psoriasis patients than healthy individuals reinforces its involvement in the inflammatory milieu of implication. Future studies must investigate IL-32 isoform-

specific expression and their mechanistic impacts in psoriasis subtypes. Longitudinal studies evaluating IL-32 before and after biologic therapy may clarify its utility as a therapeutic response indicator.

References

[1] Griffiths, Christopher E. M., April W. Armstrong, Johann E. Gudjonsson, and Jonathan N. W. N. Barker. (2021). "Psoriasis." The Lancet 397(10281): 1301–1315.

[2] Furue, Masutaka, and Takafumi Kadono. (2016). "Psoriasis: Behind the Scenes." Journal of Dermatology 43(1): 4–8.

[3] Naldi, Luigi, Liliane Chatenoud, Dennis Linder, Anna Belloni Fortina, Andrea Peserico, Anna Rosa Virgili, and Carlo La Vecchia. (2005). "Cigarette Smoking, Body Mass Index, and Stressful Life Events as Risk Factors for Psoriasis: Results from an Italian Case–Control Study." Journal of Investigative Dermatology 125(1): 61–67.

[4] Al-Shobaili, Hani A., and Zafar Rasheed. (2018). "Elevated Gene Expression of Interleukin-32 Isoforms Alpha, Beta, Gamma, and Delta in the Peripheral Blood of Chronic Psoriatic Patients." Diseases 6(1): 21.

[5] Sobhan, Mohammadreza, and Mahmoud Farshchian. (2017). "Associations between Body Mass Index and Severity of Psoriasis." Clinical, Cosmetic and Investigational Dermatology 10: 493–498. https://doi.org/10.2147/CCID.S147236

[6] Takeshita, Junko, Sungat Grewal, Sinead M. Langan, Nehal N. Mehta, Alexis Ogdie, Abby S. Van Voorhees, and Joel M. Gelfand. (2017). "Psoriasis and Comorbid Diseases: Epidemiology." Journal of the American Academy of Dermatology 76(3): 377–390.

[7] Sora, Anna, Tony Hangan, Sergiu Ioachim Chirila, Leonard Gurgas, Mihaela Botnarciuc, Lavinia Carmen Daba, Ana Maria Cretu, Ionut Burlacu, Mihaela Zamfirescu, Adina Petcu, Adrian Cosmin Rosca, Ramona Mihaela Stoicescu, and Lucian Cristian Petcu. (2025). "Salivary Interleukins as Non-Invasive Biomarkers for Psoriasis: Advances and Challenges in Diagnosis and Monitoring." Medicina 61(7): 1180. https://doi.org/10.3390/medicina61071180

[8] Zhou, Yaqin, and Ying Zhu. (2015). "Important Role of the IL-32 Inflammatory Network in the Host Response against Viral Infection." Viruses 7(6): 3116–3129. https://doi.org/10.3390/v7062762

[9] Jensen, Michael D., Donna H. Ryan, and Caroline M. Apovian. (2013). "AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults." Circulation 129(25): 102–138.

[10] Fernandez-Torres, Rosa M., Susana Paradela, and Eduardo Fonseca. (2012). "Psoriasis in Patients Older than 65 Years: A Comparative Study with Younger Adult Psoriatic Patients." Journal of Nutrition, Health & Aging 16(6): 586–591.

[11] El-Komy, Mohamed H. M., Heba Mashaly, Khadiga S. Sayed, Vanessa Hafez, Marwa S. El-Mesidy, Eman R. Said, and Hoda Rasheed. (2020). "Clinical and Epidemiologic Features of Psoriasis Patients in an Egyptian Medical Center." JAAD International 1(2): 81–90.

[12] Yu, Weiru, Yifei Yu, Siyuan Sun, Chenxu Lu, Jianan Zhai, Yumei Lei, Feirong Bai, Ran Wang, and Juan Chen. (2024). "Immune Alterations with

Aging: Mechanisms and Intervention Strategies." Nutrients 16(22): 3830. https://doi.org/10.3390/nu16223830

[13] Kimball, Alexa B., Craig Leonardi, Mona Stahle, Wayne Gulliver, Marc Chevrier, Shahram Fakharzadeh, and the PSOLAR Steering Committee. (2014). "Demography, Baseline Disease Characteristics and Treatment History of Patients with Psoriasis Enrolled in a Multicentre, Prospective, Disease-Based Registry (PSOLAR)." British Journal of Dermatology 171(1): 137–147. https://doi.org/10.1111/bjd.13013

[14] Saavedra, Danay, Ana Laura Añé-Kourí, Nir Barzilai, Calogero Caruso, Kyung-Hyun Cho, Luigi Fontana, Claudio Franceschi, Daniela Frasca, Nuris Ledón, Laura J. Niedernhofer, Karla Pereira, Paul D. Robbins, Alexa Silva, Gisela M. Suarez, Wim Vanden Berghe, Thomas von Zglinicki, Graham Pawelec, and Agustín Lage. (2023). "Aging and Chronic Inflammation: Highlights from a Multidisciplinary Workshop." Immunity & Ageing 20(1): 25. https://doi.org/10.1186/s12979-023-00352-w

[15] Parisi, Rosa, Ireny Y. K. Iskandar, Evangelos Kontopantelis, Matthias Augustin, Christopher E. M. Griffiths, and Darren M. Ashcroft. (2020). "National, Regional, and Worldwide Epidemiology of Psoriasis: Systematic Analysis and Modelling Study." BMJ 369: m1590. https://doi.org/10.1136/bmj.m1590

[16] Lubrano, Ennio, Silvia Scriffignano, Mauro Fatica, Paola Triggianese, Paola Conigliaro, Fabio Massimo Perrotta, and Maria Sole Chimenti. (2023). "Psoriatic Arthritis in Males and Females: Differences and Similarities." Rheumatology and Therapy 10(3): 589–599.

[17] Vata, Dan, Bogdan Marian Tarcau, Ioana Adriana Popescu, Ioana Alina Halip, Adriana Ionela Patrascu, Dragos-Florin Gheuca Solovastru, Madalina Mocanu, Petronela Cristina Chiriac, and Laura Gheuca Solovastru. (2023). "Update on Obesity in Psoriasis Patients." Life (Basel, Switzerland) 13(10): 1947.

[18] Wirth, Theo, Nathalie Balandraud, Laurent Boyer, Pierre Lafforgue, and Thao Pham. (2022). "Biomarkers in Psoriatic Arthritis: A Meta-Analysis and Systematic Review." Frontiers in Immunology 13: 1054539.

[19] Alizadeh, Narges, Seyyede Zeinab Azimi, and Somayeh Hoseinzadeh. (2021). "Body Mass Index and Severity of Psoriasis: A Cross-Sectional Study." Iranian Journal of Dermatology 24(1): 46–52.

[20] Paroutoglou, Kyriaki, Evangelia Papadavid, Georgios S. Christodoulatos, and Maria Dalamaga. (2020). "Deciphering the Association Between Psoriasis and Obesity: Current Evidence and Treatment Considerations." Current Obesity Reports 9: 165–178.

[21] World Health Organization. (2016). Global Report on Psoriasis. World Health Organization, Geneva. https://www.who.int/publications/i/item/9789241565189

[22] Telfer, Nicholas R., Robert J. G. Chalmers, Kathleen Whale, and Geoffrey Colman. (1992). "The Role of Streptococcal Infection in the Initiation of Guttate Psoriasis." Archives of Dermatology 128(1): 39–42.

[23] Griffiths, Christopher E. M., and Jonathan N. W. N. Barker. (2007). "Pathogenesis and Clinical Features of Psoriasis." The Lancet 370(9583): 263–271.

[24] Galili, Eran, Shir Rubinstein Levy, Ido Tzanani, Oz Segal, Anna Lyakhovitsky,

Aviv Barzilai, and Sharon Baum. (2023). "New-Onset Guttate Psoriasis: A Long-Term Follow-Up Study." Dermatology (Basel, Switzerland) 239(2): 188–194.

[25] Kaeley, Gurjit S., Lihi Eder, Sibel Zehra Aydin, Phoebe Rich, and Catherine J. Bakewell. (2021). "Nail Psoriasis: Diagnosis, Assessment, Treatment Options, and Unmet Clinical Needs." The Journal of Rheumatology 48(8): 1208–1220.

[26] Rajashekar, Shreya, Seethalakshmi Ganga Vellaisamy, Navakumar Manickam, Kannan Gopalan, and Muthusamy Kandasamy. (2018). "Prevalence of Nail Changes in Patients with Psoriasis and Correlation of NAPSI (Nail Area Psoriasis Severity Index) with BSA (Body Surface Area)." IP Indian Journal of Clinical and Experimental Dermatology 4(2): 116–122.

[27] Basko-Plluska, Juliana L., and Vesna Petronic-Rosic. (2012). "Psoriasis: Epidemiology, Natural History, and Differential Diagnosis." Psoriasis: Targets and Therapy 2: 67–76.

[28] Cao, Lin, Lu Lu, Yu Yuying, Zhou Hong, and Bin Lin. (2024). "Clinical Characteristics of Patients with a Family History of Psoriasis: An Observational Epidemiological Study in Chinese Han Population." Frontiers in Medicine 11: 1455953.

[29] Kempuraj, Duraisamy, Pio Conti, Magdalini Vasiadi, Konstantinos-Dionysios Alysandratos, Michael Tagen, Demetrios Kalogeromitros, and Theoharis C. Theoharides. (2010). "IL-32 Is Increased Along with Tryptase in Lesional Psoriatic Skin and Is Up-Regulated by Substance P in Human Mast Cells." European Journal of Dermatology 20(6): 865–867.

[30] Vičić, Marijana, and Marija Kaštelan, Ines Brajac, Vlatka Sotošek, and Larisa Prpić Massari. (2021). "Current Concepts of Psoriasis Immunopathogenesis." International Journal of Molecular Sciences 22(21): 11574.

[31] Wallimann, Alexandra, and Mirjam Schenk. (2023). "IL-32 as a Potential Biomarker and Therapeutic Target in Skin Inflammation." Frontiers in Immunology 14: 1264236.