

Article

Not peer-reviewed version

Correlation Between Systemic Inflammation, Metabolic Syndrome, and Quality of Life in Psoriasis Patients

[Maria-Lorena Mustăță](#) , [Carmen-Daniela Neagoe](#) , [Virginia-Maria Rădulescu](#) ^{*} , [Ioana-Gabriela Dragne](#) , [Radu-Cristian Cîmpeanu](#) , [Lucrețiu Radu](#) , [Roxana-Viorela Ahrițculesei](#) , [Dragoș Forțoiu](#) ,
Maria-Cristina Predoi , [Simona-Laura Ianoși](#)

Posted Date: 3 January 2025

doi: 10.20944/preprints202501.0009.v1

Keywords: psoriasis; systemic inflammation; metabolic syndrome; DLQI; PASI; leptin



Preprints.org is a free multidisciplinary platform providing preprint service that is dedicated to making early versions of research outputs permanently available and citable. Preprints posted at Preprints.org appear in Web of Science, Crossref, Google Scholar, Scilit, Europe PMC.

Copyright: This open access article is published under a Creative Commons CC BY 4.0 license, which permit the free download, distribution, and reuse, provided that the author and preprint are cited in any reuse.

Disclaimer/Publisher's Note: The statements, opinions, and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions, or products referred to in the content.

Article

Correlation Between Systemic Inflammation, Metabolic Syndrome, and Quality of Life in Psoriasis Patients

Maria-Lorena Mustață ^{1,†}, Carmen-Daniela Neagoe ², Virginia-Maria Rădulescu ^{3,*},
Ioana-Gabriela Dragne ¹, Radu-Cristian Cîmpeanu ¹, Lucrețiu Radu ^{4,†},
Roxana-Viorela Ahrițculesei ¹, Dragoș Forțofoiu ¹, Maria-Cristina Predoi ⁵
and Simona-Laura Ianoși ⁶

¹ Doctoral School, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania

² Department of Internal Medicine, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania

³ Department of Medical Informatics and Biostatistics, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania

⁴ Department of Hygiene, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania

⁵ Department of Morphology, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania

⁶ Department of Dermatology, Faculty of Medicine, University of Medicine and Pharmacy of Craiova, 200349 Craiova, Romania

* Correspondence: virginia.radulescu@umfcv.ro

† These authors contributed equally to this work.

Abstract: Background/Objectives: Psoriasis is a chronic inflammatory autoimmune disease with important systemic and psychosocial impacts. The association with metabolic syndrome (MS) impairs disease severity and negatively influences patient-reported outcomes, particularly their quality of life as measured by the Dermatology Life Quality Index (DLQI). This study aims to investigate the relationship between systemic inflammation, DLQI scores, and disease severity, focusing on the persistent impact of MS on patient outcomes after one year of treatment. Methods: This retrospective cross-sectional study included 150 psoriasis patients, with 74 also meeting the diagnostic criteria for MS. Clinical and inflammatory markers such as systemic immune-inflammatory index (SII), cytokines (IL-17A, IL-23), leptin, BMI, and triglycerides were analyzed alongside PASI and DLQI scores. Results: Patients with MS had significantly higher PASI and DLQI scores compared to those without MS, reflecting worse disease severity and quality of life ($p < 0.001$). Elevated SII levels were strongly associated with higher DLQI scores ($p < 0.0001$). Despite considerable reductions in PASI scores over one year of treatment, DLQI scores indicated a persistent negative impact of MS on quality of life. Notably, markers of systemic inflammation, such as SII, leptin, and cytokines, correlated positively with both PASI and DLQI scores, highlighting the role of systemic inflammation in disease burden. Conclusions: This study underlines the significant role of systemic inflammation and metabolic comorbidities in amplifying the burden of psoriasis. The persistent impact of MS on quality of life despite clinical improvement underscores the need for comprehensive treatment approaches targeting systemic inflammation, metabolic health, and psychosocial factors to improve long-term outcomes.

Keywords: psoriasis; systemic inflammation; metabolic syndrome; DLQI; PASI; leptin

1. Introduction

Despite the fact that the majority of skin conditions do not directly compromise the lives of patients, they can have a substantial impact on the manner in which patients perceive and interact with their surroundings [1]. There is a misunderstanding of the importance of skin diseases in relation to systemic diseases, a main reason for this misconception being that they do not generally pose a vital risk to patients, although they can be lifelong conditions. This perspective may cause patients to underestimate the complete implications of their diseases. The substantial psychological and social difficulties associated with psoriasis are frequently overlooked. The detrimental psycho-emotional consequences of psoriasis on those who are suffering with the condition have been demonstrated in a multitude of recent research studies [2,3]. Psoriasis typically affects the skin; however, its implications as a systemic disease are evident, particularly regarding the mental health of patients, cardiovascular disease, and metabolic diseases [4,5].

In addition to the obvious skin lesions, the burden of psoriasis extends beyond them, affecting patients' emotional and psychological health, social interactions and economic stability. Recognized as a serious disease by the WHO in 2014, psoriasis is a chronic condition that can persist throughout life, associated with reduced work productivity and significant economic losses [6–9]. In addition, the systemic nature of psoriasis contributes to a range of comorbidities, including psychiatric or psychological, metabolic and cardiovascular disorders, which may ultimately contribute to premature morbidity and mortality [10].

Psoriasis is an autoimmune disease clinically characterized by hyperkeratotic, elevated, clearly defined scaly plaques, typically located on the elbows, knees, scalp and sacral region [9,11]. From a cutaneous perspective, the lesions can cause extreme pruritus, pain, and visible scaling, leading to physical discomfort that causes stigma and limits social interaction and interpersonal interactions [12,13]. The severity of these symptoms is directly proportional to the severity of the skin damage. As a result, psoriasis patients frequently experience feelings of shame, anxiety, or depression [14].

Numerous systemic diseases have been linked to psoriasis; approximately 75% of patients have been demonstrated to have at least one comorbid condition, and many have numerous comorbidities [15–17]. This association is a result of the chronic inflammatory changes and proinflammatory cytokines that are frequently elevated in psoriasis, which induce a continuous inflammatory state. According to the most recent research, the metabolic syndrome is one of the most prevalent comorbidities of psoriasis [18–24]. It is defined by the presence of at least three out of five criteria (abdominal obesity, hypertriglyceridemia, low HDL-cholesterol, hyperglycemia or diabetes mellitus, and hypertension) [25]. The presence of MS in psoriasis patients not only exacerbates clinical symptoms but also contributes to heightened systemic inflammation through mechanisms such as insulin resistance and endothelial dysfunction [26,27].

Psoriasis should be acknowledged as a complex systemic condition, transcending the simplistic classification of a skin condition, due to its significant influence on overall health, including a well-documented association with MS and its related physical and psychosocial complications [28]. Genetic predisposition, common inflammatory pathways, and risk factors are mechanisms underlying the association between psoriasis and MS. Common genetic variants such as FUT2, UBE2L3, CDKAL1, SH2B3, and apolipoprotein E are more frequently found in psoriasis patients, having multiple functions that would explain their dual role in susceptibility to psoriasis and MS [29]. Moreover, common immunological mechanisms involving in particular Th and Th17 cell activation seem to link psoriasis to metabolic comorbidities [30]. The release of inflammatory mediators from psoriatic lesions, such as IL-1, IL-6, IL-17, TNF- α , IFN- α and IFN- γ may have systemic effects that contribute to the atherogenesis process. One finding that reinforces the relationship between the two conditions is the activity of TNF- α and IFN- γ [31]. Extensive research has shown that the inflammatory state characteristic of psoriasis intensifies inflammation in the adipose tissue, which may trigger additional immune responses [32]. In psoriatic adipose tissue, T lymphocytes, dendritic cells, neutrophils, mast cells, and macrophages, all of which are immune cells, play a key role in influencing cardiometabolic health and contribute to the development of obesity and insulin

resistance. The release of adipokines such as chemerin, adiponectin, resistin, visfatin and C-reactive protein by macrophages and T-cells is the link between chronic systemic inflammation and obesity. Patients with psoriasis have elevated levels of these adipokines in the blood, which are thought to contribute significantly to the development of insulin resistance [33,34].

Psoriasis patients' quality of life is profoundly affected, from physical activities and interpersonal relationships to mental health. The psychological impact is amplified by social stigmatization, with a significant number of patients feeling excluded or judged because of the appearance of their skin [35–37]. A meta-analysis of 98 studies and 401,703 patients diagnosed with psoriasis found that they were at least 1.5 times more likely to experience depressive symptoms than those without the condition [38]. Similarly, female psoriasis patients were shown to be at greater risk to depression than their male counterparts, according to a recent systematic review [39].

An essential tool to highlight the degree of impairment and provide insight into the impact of the disease is the Dermatology Life Quality Index (DLQI). This questionnaire consists of ten questions that assess several dimensions of patients' lives, including those related to emotional impact, social interactions and daily activities, with scores ranging from 0 to 30, with a higher value representing a lower quality of life, reflecting the physical, emotional and social burden of the disease [40–42]. However, the DLQI does not appear to explore in detail certain psychological or economic aspects of the disease and is subjective in nature, as it is based on patients' self-reported perception of the impact of their condition. Each patient may perceive his or her own condition differently, which may interfere with responses related to the discomfort created by the disease [43–45].

Along with the quality of life assessment, psoriasis severity is measured using the Psoriasis Area and Severity Index (PASI) score. In general, PASI and DLQI are directly related but the relationship between them is not always linear, in that patients with lower PASI may continue to report a high DLQI even after clinical symptoms have improved, reflecting the persistence of the psychosocial impact of the disease [46–48].

Substantial evidence of inflammatory pathway dysregulation in autoimmune diseases has led to the use of various circulating inflammatory biomarkers such as C-reactive protein, ferritin, and ESR, but their limited diagnostic accuracy has stimulated a large number of investigations to identify more reliable biomarkers. Following the latest research, the systemic immune-inflammatory index (SII) has emerged as a valuable new inflammatory biomarker that has recently been introduced to assess the association between chronic inflammatory state and multiple chronic diseases, including various types of neoplasms, metabolic disorders, and psoriasis [49].

Previous research have extensively investigated the relationship between the clinical severity of psoriasis and quality of life [50,51]. Nonetheless, the combined impact of the metabolic syndrome and the role of inflammation on the quality of life of psoriasis patients has yet to be investigated.

This study aims to explore the relationship between DLQI, PASI and associated comorbidities, particularly metabolic syndrome, in patients with psoriasis. In our research, we analyzed how clinical and inflammatory aspects, such as BMI, triglycerides, glycemia, leptin, SII, and the cytokines IL-17A and IL-23, influence quality of life as measured by DLQI, in psoriasis patients after one year of treatment.

2. Materials and Methods

2.1. Patient Selection

This retrospective cross-sectional study included two groups of patients, 76 of them diagnosed with psoriasis and 74 with both psoriasis and metabolic syndrome. Therefore, a total of 150 patients were enrolled. The study ran for one year, from January 2022 to January 2023. All patients received written informed consent. The study received the approval of the Ethics Committee of the University of Medicine and Pharmacy of Craiova (approval number 195/20.09.2022) and adhered to the principles described in the Declaration of Helsinki (2004).

Inclusion criteria were patients older than 18 years of age with a clinical diagnosis of psoriasis, regardless of its severity, and exclusion criteria were patients with other concomitant autoimmune diseases, severe comorbidities (such as end-stage chronic diseases), patients with a history of alcohol or drug use (which may alter metabolic or inflammatory markers).

Each patient was asked for a complete medical history, including age, residence, occupation, onset, course and duration of the disease. Anthropometric measurements including weight, height, abdominal circumference (AC), and systolic (SBP) and diastolic (DBP) blood pressure were measured for each patient.

BMI was calculated according to the formula: weight in kilograms divided by height in meters squared, and abdominal circumference was measured using a tailor's tape measure, halfway between the costal margin and the upper iliac crest, on the mid axillary line. BMI was categorized according to the criteria proposed by the WHO: underweight < 18.5, normal = 18.5-24.5, overweight = 25.0-29.9 and obese ≥ 30.00 [52].

Normal blood pressure was considered < 120 mmHg (SBP) and < 80 mmHg (DBP), and values between 120-139 mmHg (SBP) and 80-89 mmHg (DBP) were categorized as prehypertensive. All values that exceeded 140 mmHg (SBP) and 90 mmHg (DBP) were considered as hypertension [53].

Laboratory investigations were collected from venous blood from the antecubital vein of each patient: complete blood count, complete lipid profile (total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides), transaminases (ALAT, ASAT), blood glucose, and markers of inflammation, C-reactive protein (CRP), leptin, IL-17 and IL-23. Systemic immune-inflammation (SII) index was calculated using the formula: absolute neutrophil count X (total platelet count/absolute lymphocyte count) ratio. Metabolic syndrome was diagnosed using at least three of the five diagnostic criteria: AC > 80 cm in women and > 94 cm in men; serum triglyceride value > 150 mg/dl (or on lipid-lowering treatment); HDL-cholesterol < 50 mg/dl in women and < 40 mg/dl in men, or on cholesterol-lowering treatment; blood pressure > 130/85 mmHg or on antihypertensive treatment; fasting blood glucose > 100 mg/dl or on blood glucose-lowering treatment.

Assessment of psoriasis severity was performed using the Psoriasis Area and Severity Index (PASI) score, which combines manifestations related to disease severity, such as erythema, induration and desquamation, with the size of the affected area, measured as a percentage. Each region was assigned a score that highlighted the degree of involvement of that region, and a score that recorded the severity of psoriasis. The PASI ranges from 0 to 72, with four areas of the body being assessed: head and neck, upper limbs, trunk and lower limbs, and in each area, the severity index is assessed by signs such as erythema, degree of induration and flaking. Each of these signs is rated on a scale of 1 to 5. Thus, according to the PASI score, psoriasis is categorized into three severity grades: below 10 signifies mild psoriasis, between 10 and 20, moderate psoriasis and above 20, severe psoriasis.

Patients' quality of life was estimated using the DLQI questionnaire which explores some relevant dimensions of the patient's life that may be affected by the disease. The questionnaire comprises 10 questions that assess patients' perceptions of the impact of psoriasis on their quality of life and includes items such as symptoms, emotions or feelings, daily activities, leisure and sport, work or study, social contact and treatment. Based on their performance in the week prior to completing the questionnaire, patients answer these questions, giving scores ranging from 0 to 3: 0 means not at all, 1 - mild, 2 - severe and 3 - very severe. The total DLQI score is 30, with a higher score representing a more impaired quality of life.

2.2. Statistical Analysis

Statistical analyses were performed using the SPSS (Statistical Package for Social Sciences) software, specifically version 26 developed by SPSS Inc. in Chicago, IL, USA. In this study, continuous variables were characterized by their mean and standard deviation (SD), providing a clear summary of the data's central tendency and variability. For categorical and ordinal variables, we utilized frequency distributions and percentages to offer a comprehensive overview of the different categories present in the data.

To evaluate the distribution of the data, we applied the Kolmogorov–Smirnov / Shapiro–Wilk tests, which assess the normality of the dataset. This step was crucial in determining the appropriate statistical tests for further analysis.

For comparing the variables between the study group and the control group, we adopted different statistical methods based on the nature of the data. For data that met the assumptions of normal distribution, we employed the independent t-test. In contrast, when dealing with continuous variables that did not follow a normal distribution, we utilized non-parametric tests, specifically Kendall's tau-b, Mann–Whitney U, and Kruskal–Wallis tests. Additionally, for categorical data, we implemented the chi-square test to examine the relationships between variables.

To conclude our analyses, we considered a p-value of less than 0.05 to be statistically significant, indicating a meaningful difference or correlation in our study results. This threshold helped ensure the robustness of our findings.

3. Results

Following the application of the established inclusion and exclusion criteria, the study group was composed of 150 patients. This cohort included 58 females and 92 males, with ages ranging from 26 to 76 years. The mean age of the participants was 53.85 years, with a standard deviation of ± 11.73 , indicating a broad age distribution among the subjects.

Analysis of the gender distribution revealed that male patients constituted 61.3% of the group, with a mean age of 55.33 years and a standard deviation of ± 12.19 . In comparison, female patients represented 38.7% of the cohort, with a mean age of 51.52 years and a standard deviation of ± 10.64 .

All participants had previously received a diagnosis of psoriasis, a chronic inflammatory skin condition. Significantly, approximately half of these patients—specifically 74 individuals, or 49.33%—also presented with metabolic syndrome (MS), a collection of risk factors that heightens the likelihood of developing cardiovascular disease and other serious health complications. Consequently, the study group was bifurcated into two distinct subgroups: the PSO group, which consisted of patients diagnosed solely with psoriasis, and the PSO–MS group, which included those diagnosed with both psoriasis and metabolic syndrome.

3.1. Analysis of Study Subgroups: PSO Versus PSO–MS

A Mann–Whitney U test was run to determine if there were differences in the ages of patients from both subgroups. Distributions of the ages for PSO and PSO–MS patients were similar, as assessed by visual inspection. The median age for PSO patients (53) was smaller than the median age for PSO–MS patients (55), but the difference was not statistically significantly different, $U = 2628.0$, $z = -0.692$, $p = 0.489$.

The analysis revealed a statistically significant difference in gender distribution between the two subgroups. In the subgroup of patients without metabolic syndrome, gender representation is relatively comparable; however, the subgroup of patients with metabolic syndrome displays a notable contrast. Specifically, within the PSO–MS subgroup, females constitute 27.03% of the patients, while males account for 72.97% of the PSO subgroup ($\chi^2(1) = 8.344$, $p = .004$). Furthermore, it is noteworthy that all patients in the PSO–MS subgroup have hypertension, in comparison to only 5.26% of patients in the PSO group ($\chi^2(1) = 134.818$, $p < 0.001$).

The patients in the PSO subgroup exhibited a median weight of 82.61 kg, which was significantly lower than that of the patients in the PSO–MS subgroup, who demonstrated a median weight of 100.7 kg ($p < 0.001$). Additionally, the abdominal circumference (AC) was notably greater in patients with metabolic syndrome, recording a median value of 109.54 cm, compared to the PSO patients, whose median AC was 91.47 cm ($p < 0.001$). Similar patterns were observed in the analysis of body mass index (BMI); patients in the PSO subgroup without metabolic syndrome had a median BMI of 28.43, significantly lower than the median BMI of 32.71 in patients within the PSO–MS subgroup ($p < 0.001$). These patient characteristics are detailed in Table 1.

Table 1. Main characteristics of the study subgroups.

Parameter	PSO-MS	PSO	<i>p</i> -Value
	Median	Median	
Weight (kg)	100.70	82.61	<0.001
AC (cm)	109.54	91.47	<0.001
BMI (kg/m ²)	32.71	28.43	<0.001
Cholesterol (mg/dL)	238.12	225.70	0.026
Triglycerides (mg/dL)	281.82	138.38	<0.001
Glycaemia (mg/dL)	150.75	94.15	<0.001
Leptin (ng/mL)	982.45	538.96	<0.001
IL-17A (pg/mL)	1426.48	712.46	0.009
IL-23 (pg/mL)	903.77	490.86	0.017
HDL (mg/dl)	39.14	59.14	<0.001

¹ Mann-Whitney U test.

In patients diagnosed with PSO-MS, there was a notable elevation in the levels of cholesterol, triglycerides, and glycemia when compared to individuals with PSO alone. The observed differences in these biochemical parameters were statistically significant, with a *p*-value < 0.001, as illustrated in Table 1. This suggests a potential link between the presence of metabolic syndrome and the disturbance of lipid and glucose metabolism in patients with psoriasis. On the other hand, patients within the PSO-MS subgroup exhibited a reduced concentration of high-density lipoprotein (HDL), which is often regarded as the “good” cholesterol and is crucial for cardiovascular health. These findings underline the importance of monitoring lipid profiles and glucose levels in patients with psoriasis who may also present with metabolic syndrome.

Leptin levels were significantly elevated in patients within the PSO-MS subgroup when compared to those with PSO, with a statistically significant difference observed (*p* < 0.001). Comparative analyses of interleukins IL-17A and IL-23 revealed notable findings; specifically, the PSO-MS group exhibited higher levels for both interleukins. IL-17A demonstrated a statistically significant difference relative to the PSO group (*p* = 0.009). In contrast, IL-23 levels were comparable between the two groups.

3.2. PASI Values Analysis

For all patients, the PASI values were measured before the initiation of PSO treatment. A Mann-Whitney U test was run to determine if there were differences in PASI values between males and females. Distributions of PASI values for males and females were similar, as assessed by visual inspection. The median PASI for males (21.39) was higher than for females (18.80), yet the difference was not statistically significantly different, *U* = 3036.00, *z* = 1.421, *p* = 0.155. A similar test was run to determine whether there were differences in PASI values between patients with and without MS. The analysis of PASI values revealed notable differences between the two patient categories. Specifically, the PSO-MS subgroup demonstrated a higher median PASI value of 22.37, in contrast to the PSO subgroup, which had a median value of 18.46. This difference was found to be statistically significant, as indicated by a *U*=3376.00, *z*= 2.121, and a *p*-value of 0.034.

A Kendall’s tau-b correlation analysis was conducted to examine the relationship between PASI values and the duration between the onset of psoriasis and the commencement of therapy, measured in months. The findings indicated a moderate positive association, with statistical significance reported as *tb* = 0.256, *p* < 0.001. This suggests that a longer interval between the diagnosis of PSO and the initiation of treatment correlates with higher PASI values.

The analyses of the patients participating in this study were conducted regularly, with the PASI data recorded at three-month intervals. This consistent monitoring allowed us to track PASI scores at the start of the study, as well as at three months, six months, and one year.

The results showed that as patients received treatment, the mean PASI score decreased significantly. Specifically, the median PASI scores were as follows: at the initiation of treatment, the median was 20.38; at three months, it was 7.07; at six months, it was 3.67; and at twelve months, it was 2.10.

We applied the Kruskal-Wallis test to assess the differences among the four subgroups of patients categorized by their PASI scores. The results indicated statistically significant differences between these subgroups at various time points, with p-values as follows: PASI at initiation: 0.034; PASI at three months: 0.019; PASI at six months: <0.0001; and PASI at twelve months: <0.0001.

The analysis of the parameters IL-17A, IL-23, baseline leptin levels, and leptin levels at the 12-month mark was conducted, taking into account two patient subgroups (PSO and PSO-MS) as well as the PASI scores recorded during the specified time periods. The findings are succinctly presented in Table 2.

Table 2. Kendall's tau-b correlation between all PASI score and clinical parameters.

Parameter	PASI	Tau-b Coefficient τ_b		<i>p</i> -Value ¹
		PSO	PSO-MS	
IL-17A	3 months	0.083	0.089	<0.0001
	6 months	0.117	-0.006	0.001
	12 months	0.092	0.005	0.001
IL-23	3 months	0.156	0.182	<0.0001
	6 months	0.215	-0.073	0.001
	12 months	0.237	0.026	0.002
Leptin (initial)	3 months	-0.107	0.153	<0.0001
	6 months	0.078	0.047	<0.0001
	12 months	-0.016	0.283	<0.0001
Leptin (12 months)	3 months	-0.004	0.378	0.001
	6 months	0.195	0.064	<0.0001
	12 months	0.067	0.214	0.011

¹ Kendall's tau-b test.

Furthermore, Kendall's tau-b correlations were performed to evaluate the relationship between PASI levels and the clinical parameters detailed in Table 1, encompassing a sample size of 150 participants. The analysis unveiled primarily weak-to-moderate positive associations between the initial PASI index at the commencement of PSO treatment and the parameters listed in Table 1. Notably, most of the tests yielded statistically significant results, as presented in Table 3.

Table 3. Main characteristics of the study subgroups.

Parameter	Tau-b Coefficient τ_b	<i>p</i> -Value ¹
Age (years old)	0.070	0.210
Weight (kg)	0.194	0.001
AC (cm)	0.066	0.244
BMI	0.181	0.001
Cholesterol	0.213	<0.001
Triglycerides	0.217	<0.001
Glycaemia	0.145	0.009
Leptin	0.176	0.002
IL-17A	-0.027	0.630
IL-23	0.143	0.010
HDL	-0.135	0.016

¹ Mann-Whitney U test.

Our research has unveiled significant positive correlations between weight (kg) and PASI values, with a robust Kendall's tau-b of $\tau_b = 0.194$ and a highly significant p-value of $p < 0.001$. Similarly, BMI shows a positive correlation with PASI values, with a substantial $\tau_b = 0.181$ and a p-value of $p < 0.001$. These findings have direct implications for the management of dermatological and metabolic health conditions. Additionally, we identified correlations for cholesterol, triglycerides (TG), glycaemia, leptin, and IL-23, providing a comprehensive understanding of the factors influencing PASI values. Furthermore, lower HDL values were associated with higher PASI values, with a noteworthy $\tau_b = -0.135$ and a p-value of $p = 0.016$. All other parameters were not correlated to PASI values ($p > 0.05$) (Table 3).

3.3. Dermatology Life Quality Index (DLQI) Analysis

Our database confidently includes 150 patients, all classified according to their Dermatology Life Quality Index (DLQI) scores. Of these patients, 9.3% fell into DLQI class 3, 54.7% into DLQI class 4, and 36% into DLQI class 5, with no patients represented in DLQI classes 1 or 2. An analysis of the data by the various DLQI classes indicates the following median scores: patients classified in class 3 exhibited a median DLQI score of 10, those in class 4 demonstrated a median score of 14.63, and patients in class 5 recorded a median score of 24.30.

Statistical analysis revealed significant differences among the DLQI groups, particularly within the PSO and PSO-MS subgroups. These compelling results were obtained through the Mann-Whitney statistical test, yielding $U = 1192.00$, $z = -6.116$, and $p < 0.001$. The p-value, less than 0.001 in this case, indicates that the observed differences among the DLQI groups are statistically significant and not due to random variation.

An evaluation of the quality of life among the study participants revealed that cases were exclusively distributed across DLQI classes 3, 4, and 5, as we said before. A significant majority of the patients, regardless of gender, were classified within DLQI class 4, accounting for 54.70% of the sample. Additionally, 36.00% of patients were categorized in DLQI class 5, while only 9.30% were classified in class 3, as illustrated in Table 4. The statistical analysis conducted to assess the distribution by DLQI class with gender yielded $\chi^2(2) = 2.197$, $p = 0.33$, indicating an absence of statistically significant differences among the categories. Therefore, it can be concluded that gender does not influence the quality of life within the context of this study.

Table 4. Gender distribution to DLQI.

	Class	Gender	Frequency	Age			p-Value ¹
			N (%)	Min	Max	Mean ± Stdev	
DLQI	3	F	4 (6.9)	41	52	46.50±3.17	0.33
		M	10 (10.90)	37	65	53.20±3.24	
	4	F	36 (62.1)	26	68	49.44±1.69	
		M	46 (50)	26	76	56.22±2.11	
	5	F	18 (31.0)	39	72	56.78 ± 2.52	
		M	36 (39.10)	35	74	55.33 ± 1.62	

¹ Chi-square test.

Kendall's tau-b correlations were performed to evaluate the relationship between DLQI and the clinical parameters presented in Table 1 for all 150 participants. The analysis showed weak to moderate associations between the initial DLQI scores at the beginning of psoriasis treatment and the parameters listed in Table 1. Most tests yielded statistically significant results, which are detailed in Table 5.

Table 5. Main characteristics of the study subgroups.

Parameter	Tau-b Coefficient τ_b	p-Value ¹
Age (years old)	0.087	0.187
Weight (kg)	0.166	0.011
CA (cm)	0.125	0.057
BMI	0.190	0.004
Cholesterol	0.220	0.001
Triglycerides	0.383	<0.001
Glycaemia	0.256	<0.001
Leptin	0.266	<0.001
IL-17A	0.116	0.076
IL-23	0.177	0.007
HDL	−0.200	0.002

¹ Kendall’s tau-b test.

Moreover, data from the entire group were analyzed to determine the correlation between DLQI and the initial PASI score. A moderate positive correlation was found between these two variables, with a τ_b value of 0.563 and a p-value of less than 0.0001. Additionally, the correlation between DLQI and PASI values at the one-year mark was also examined. It revealed a weak correlation; however, there were statistically significant differences between the groups, as indicated by a p-value of less than 0.0001.

Table 6. Kruskal Wallis test for DLQI score and PASI.

PASI	Median		p-Value ¹
	PSO	PSO-MS	
0 months	18.461	22.368	<0.0001
3 months	5.729	8.457	0.003
6 months	2.326	5.068	0.004
12 months	0.979	3.262	0.001

¹ Kruskal Wallis test.

The data collected from patients regarding DLQI scores, PASI scores, and the PSO and PSO-SM subgroups were further analyzed using the Kruskal-Wallis test. The results indicated statistically significant differences among all categories, as shown by the p-values presented in Table 6.

Analyzing the distribution of cases according to SII in relation to their belonging to DLQI classes, it was observed that 54.70% of cases were in the 4th DLQI class, 36.00% were cases in the 5th DLQI class and only 9.30% were cases in the 3rd DLQI class (Table 7).

Table 7. SII distribution to DLQI.

DLQI	DLQI		SII		<i>p</i> -Value ¹
	Class	Number of cases N(%)	Lower critically risk N(%)	Higher critically risk N(%)	
3	14 (9.30%)	12 (8.00%)	2 (1.30%)	0.0001	
4	82 (54.70%)	40 (26.70%)	42 (28.00%)		

5	54 (36.00%)	15 (10.00%)	39 (26.00%)	
---	-------------	-------------	-------------	--

It can be seen that the cases in class 3 DLQI were predominantly cases with low SII (8%) and those with high SII were only 1.30%. The situation changes as the DLQI increases. If for DLQI 4 the distribution of cases suffers a difference of about 2% of the cases, at DLQI 5 the cases with high SII are twice as many as those with SII below the risk value.

Furthermore, from a statistical point of view, it was found that there are significant differences between the categories of SII cases relative to the DLQI classes, obtaining a p-value < 0.0001 when applying the Chi-square test. A moderate positive correlation was also found between categories (p=0.322).

4. Discussion

A wide variety of studies characterize the impact of psoriasis on quality of life, most of them reinforcing the perception that the psycho-emotional impact of psoriasis is as important as its physical consequences, contributing equally to the overall morbidity of the disease [54–56]. The indicators of severity of psoriasis are clinical manifestations and the total body surface area affected, but the assessment of clinical severity can sometimes be difficult because physical symptoms do not always indicate the clinical severity of the disease [47,48,57]. Although some patients may have a fairly small involved body surface area, the location and appearance of the lesions can be a burden on participation in daily activities, weighing heavily on emotions and self-image. For example, a patient whose psoriasis is classified as mild psoriasis, with lesions located in an easily visible area (such as on the hands or elbows), is likely to be more socially and psychologically affected than a patient with the same lesions, but located in a less visible area (for example, on the abdomen) [58,59].

Psoriasis is a complex, immune-mediated skin condition that manifests clinically as a consequence of a complex interaction between the nervous, immune, and cutaneous systems. This interaction is referred to as the nervous-skin-immune system chain or the neuro-immune-cutaneous system which is characterized by a close collaboration between keratinocytes, neuropeptide mediators, and immune system cells, and its activation contributes to a state of continuous inflammation [60,61]. Psychological stress is a recognized trigger and aggravating factor in psoriasis, primarily due to the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which generates the release of cortisol and catecholamines that can have proinflammatory effects by dysregulating cortisol function in the skin [62–65].

Psoriasis is mediated by T lymphocytes, in particular Th1 and Th17, and dendritic cells that are activated and elevated in skin lesions and migrate and release proinflammatory cytokines such as IL-1 and IL-6, but also TNF-a, which favor keratinocyte proliferation and inflammation. Several studies have reported elevated levels of proinflammatory cytokines, CRP, and TNF-a among patients suffering from major depressive disorders [66].

The results of this study underscore the profound impact of psoriasis on patients’ quality of life, especially in the presence of metabolic comorbidities. In order to manage psoriasis efficiently, an in-depth understanding of its complex nature and the comorbidities present is imperative. The aim of this research was to better understand how the metabolic syndrome affects the quality of life of psoriasis patients. Comparative analysis between the PSO and PSO-MS groups revealed significant differences in both disease severity as expressed by PASI and quality of life as evidenced by DLQI.

The differences between the PSO and PSO-MS groups highlight the combined burden of metabolic syndrome among psoriasis patients. The components of the metabolic syndrome, such as obesity and dyslipidemia, are not only diagnostic markers, but actively contribute to systemic inflammation and disease severity [67,68].

The proinflammatory status that metabolic syndrome promotes, given its pathophysiology, exacerbates the severity of psoriasis. Psoriasis and metabolic syndrome both exhibit chronic systemic inflammation, which is a distinguishing feature of both conditions [69,70]. Its components, which

include obesity, hypertension, dyslipidemia, and insulin resistance, exacerbate cardiovascular risk, complicate disease management, and impact the quality of life of patients [71]. In comparison to individuals without metabolic syndrome, patients with both psoriasis and metabolic syndrome are at an increased risk of cardiovascular events, including stroke and myocardial infarction, according to numerous studies [68–72]. The metabolic abnormalities in MS, when combined with the systemic inflammation present in psoriasis, synergistically increase the cardiovascular risk in these patients. This is accompanied by a lower quality of life, which is translated into poor physical and mental health [73].

Patients in the PSO-MS group, as expected, had a higher degree of hypertension than those in the PSO group, where only 5.26% of them had hypertension ($p < 0.001$). Endothelial dysfunction is the main mechanism linking chronic inflammation in metabolic syndrome and psoriasis to cardiovascular risk [70]. Due to proinflammatory cytokines, such as IL-17, IL-23, and TNF- α existent in patients with psoriasis and MS, endothelial cell function is destroyed [74]. The bioavailability of nitric oxide, an essential molecule produced by endothelial cells which facilitates vasodilatation, is disturbed, thereby increasing vascular resistance and developing arterial stiffness. Chronic inflammation contributes to changes in the structure of the arterial walls, including collagen deposition and loss of elasticity, thus contributing to arterial hypertension [75,76].

In addition, oxidative stress exacerbates endothelial dysfunction. Common in MS due to obesity and dyslipidemia, oxidative stress produces reactive oxygen species (ROS) that degrade nitric oxide and facilitate the formation of peroxynitrite, a toxic compound that damages endothelial cells [76,77]. Inflammation and lipid abnormalities promote endothelial activation which is characterized by an increase in the adhesion molecules ICAM-1 and VCAM-1, which facilitates the recruitment of monocytes to the endothelium, initiating and accelerating the development of atherosclerosis [78].

In line with previous findings, the lipid profile and weight were more elevated in patients with PSO-MS, which is in concordance with the defining features of MS. These findings align with the diagnostic criteria and emphasize its role in exacerbating the inflammatory and metabolic dysregulations observed in our study.

Results also indicate a significant gender disparity, with a higher prevalence of MS among male patients. These findings are supported by previous research linking MS to sex-specific inflammatory and metabolic responses. A cross-sectional study of 44715 patients using The Health Improvement Network (THIN) database showed a higher prevalence of male patients among those with psoriasis and metabolic syndrome [79]. Moreover, the positive correlation between weight and PASI ($p < 0.001$) highlights the role of obesity as a significant factor influencing the severity of psoriasis. Obesity contributes to a pro-inflammatory state that exacerbates psoriasis by secreting adipokines, such as leptin and resistin, which stimulate Th17 and Th1 cells, thereby increasing IL-17 and TNF- α cytokines, key factors involved in the pathogenesis of psoriasis [80,81]. In addition, elevated levels of cholesterol, triglycerides, glycemia were also positively correlated with PASI score, emphasizing the impact of dyslipidemia and metabolic syndrome on the severity of psoriasis [82].

A notable clinical implication of this study is the persistent impact of MS on patient-reported outcomes, as measured by DLQI scores, even after one year of treatment. In terms of PASI scores, at baseline, patients in the PSO-MS group had higher values than those in the PSO group, reflecting the greater severity of disease in the presence of metabolic syndrome. Over one year of treatment, PASI decreased in both subgroups, but the PSO-MS patients maintained higher scores in each of the analyzed intervals (3, 6 and 12 months), emphasizing that MS not only worsens systemic inflammation, but also slows the response to treatment, as has been shown in other studies [83]. On the other hand, DLQI scores indicated persistent impairment despite improvement in PASI. Although PASI had significant decreases over time, patients in the PSO-MS group reported higher DLQI scores compared with the PSO subgroup, suggesting exacerbation of the psychosocial burden of psoriasis complicated by MS, most likely due to persistent systemic inflammation and its effects on patients' perception of their condition. Even after one year of treatment, DLQI remains statistically significant in the PSO-MS group. This highlights the disconnect between clinical improvement and

quality of life as perceived by patients, particularly those with various associated comorbidities, especially metabolic syndrome.

The DLQI scores were exclusively distributed in classes 3 to 5, suggesting moderate to severe impairment; more than half of the patients (54.7%) were part of class 4, while 36% were in class 5, highlighting the substantial impact of the disease even after treatment. These particular results from our study highlight the need for further research exploring the clinical, psychosocial and metabolic factors that shape the interaction between PASI and DLQI. The majority of existing studies focus on the relationship between PASI and DLQI in isolation, without exploring in depth the factors that contribute to the incongruence between these scores [84]. There is a lack of longitudinal studies that follow the evolution between PASI and DLQI over the long term in patients with psoriasis and metabolic comorbidities, and that examine whether changes in clinical severity, regarding the clinical improvement of psoriasis, might lead to proportional improvements in quality of life.

A recent study [47], which included an analysis of data from four phase-3 clinical trials, investigated the relationship between changes in PASI and DLQI scores in patients with moderate to severe psoriasis and showed that reduction in psoriasis severity was associated with significant improvements in patients' quality of life, as reflected by DLQI scores. In our study, patients with associated metabolic syndrome, although they had an improvement in psoriasis as evidenced by their PASI score, their quality of life was poorer compared to patients in the PSO group without other comorbidities, reflecting the psychosocial impact that metabolic syndrome exerts on patients. Although there is a limited amount of data in this area, most indicate a correlation between MS and a decrease in quality of life, particularly in patients who also suffer from depression [85–87].

Metabolic syndrome amplifies the negative perception of patients' health. The symptoms associated with metabolic syndrome, in particular the high risk of cardiovascular disease, contribute to a heightened sense of vulnerability, with patients often perceiving these signs as indicators of a general deterioration in health [88].

Aesthetic factors and social pressure also play an essential role. Abdominal obesity, a common component of the metabolic syndrome, can in itself attract social stigmatization, which can amplify feelings of inadequacy and devaluation, contributing to low self-confidence [89].

Moreover, correlation analyses between DLQI and metabolic parameters, including BMI, triglycerides, and glycemia, reinforce the role of MS as a factor in reducing the quality of life among psoriasis patients. For example, their higher values were associated with higher DLQI scores. Similarly, HDL-cholesterol was negatively correlated with DLQI, suggesting that dyslipidemia contributes to both disease severity and lower quality of life. A recent cross-sectional study of 41 patients (25 women and 16 men, mean age 43.12 ± 12.39 years and mean BMI 28.17 ± 5.34 kg/m²), found that low HDL levels and elevated triglyceride and cholesterol values were correlated with reduced physical activity capacity and lower scores on quality of life assessments, including physical and social functioning [90].

Unlike Czarnecka et al. [91], our findings suggest that higher DLQI scores were associated with an increase in markers of systemic inflammation, such as leptin and IL-23, implying a link between BMI-driven inflammation and quality of life deterioration. This could indicate that in our cohort, the psychosocial burden of psoriasis as reflected by DLQI is directly influenced by systemic inflammatory pathways more than by BMI alone.

As we evaluated the connection between quality of life and systemic immune-inflammatory index (SII), we established a strong association. Notably, only 1.3% of the patients who were in DLQI class 3, indicative of a moderate quality of life, exhibited a higher SII risk, while the majority of the 8% in this subgroup had a low SII risk. At the same time, 26% of patients in DLQI class 5, which is characterized by a significant impairment in quality of life, were classified as having a high SII risk, indicating a strong correlation between amplified systemic inflammation and psychosocial burden. These results underline that as systemic inflammation increases, the quality of life deteriorates.

Similar to the findings of Gambichler et al. [92] and Cozma et al. [93], which emphasize the close links between SII, disease severity as measured by PASI score, and the degree of psychoemotional

impairment of life as measured by the DLQI score, our study uniquely categorizes DLQI classes and correlates them with specific SII risk levels. This approach provides deeper insight into how systemic inflammation may exacerbate not only clinical symptoms but also psychosocial challenges. Moreover, Hagino et al. [94] underlined in their study the potential of targeting systemic inflammation to improve patient outcomes, which is consistent with our study.

5. Conclusions

This study highlights the complex interaction between systemic inflammation, metabolic syndrome and quality of life in patients with psoriasis. While treatment for psoriasis have markedly reduced clinical severity, as reflected in the PASI scores, the DLQI score highlights a persistent impairment in quality of life, particularly in patients with MS. The disconnect between PASI improvements and DLQI scores underlines the profound psychosocial and systemic burden exerted by MS.

The main strength of this study lies in the in-depth analysis of inflammatory biomarkers, such as leptin, IL-23 and SII, alongside PASI and DLQI scores, providing a multidimensional view of the disease burden. By stratifying the results according to DLQI classes and correlating them with markers of systemic inflammation, this research offers new insights into the interplay between clinical severity, systemic comorbidities and psychosocial well-being.

However, it is important to consider the limitations of the study. Due to the retrospective design of this cross-sectional investigation, causal relationships cannot be definitively established. Subjectivity may be introduced by reliance on patient-reported outcomes, such as DLQI, which could potentially affect the assessment of quality of life. The complex interplay between PASI, DLQI and systemic inflammatory markers warrants further investigation in future longitudinal studies that will include a wide range of patient demographics.

Author Contributions: Conceptualization, M.-L.M. and C.-D.N.; Data curation, V.M.-R.; Formal analysis, L.R.; Investigation, M.-L.M.; Methodology, S.-L.I.; Project administration, L.R.; Resources, R.-V.A. and R.-C.C.; Software, V.-M.R.; Supervision, S.-L.I.; Validation, C.-D.N. and S.-L.I.; Visualization, I.-G.D. and D.F.; Writing – review & editing, M.-C.P. All authors have read and agreed to the published version of the manuscript.

Funding: The Article Processing Charges were funded by the University of Medicine and Pharmacy of Craiova, Romania.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board (or Ethics Committee) of the University of Medicine and Pharmacy of Craiova (approval number 195/20.09.2022).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The authors declare that the data of this research are available from the corresponding authors upon reasonable request.

Acknowledgments: This study is part of the PhD thesis of Maria-Lorena Mustață from the University of Medicine and Pharmacy of Craiova, Craiova, Romania.

Conflicts of Interest: The authors declare no conflicts of interest

References

1. Szabó, C. Psychotrauma and the Skin. *Journal of the European Academy of Dermatology and Venereology* 2020, 34 (12), 2689–2690. <https://doi.org/10.1111/jdv.17030>
2. Kouris, A.; Platsidaki, E.; Kouskousis, C.; Christodoulou, C. Psychological Parameters of Psoriasis. *Psychiatriki* 2017, 28 (1), 54–59. <https://doi.org/10.22365/jpsych.2017.281.54>.
3. C. Malsawmtluangi; Paul, N.; Panda, S. Emotional Turbulence Perceived by Psoriasis Patients. *International journal of science and healthcare research* 2023, 8 (2), 562–567. <https://doi.org/10.52403/ijshr.20230276>.

4. Singh, S.; Taylor, C.; Kornmehl, H.; Armstrong, A. W. Psoriasis and Suicidality: A Systematic Review and Meta-Analysis. *Journal of the American Academy of Dermatology* 2017, 77 (3), 425-440.e2. <https://doi.org/10.1016/j.jaad.2017.05.019>
5. Tampa, M.; Sarbu, M.-I.; Mitran, M.-I.; Mitran, C.-I.; Matei, C.; Georgescu, S.-R. The Pathophysiological Mechanisms and the Quest for Biomarkers in Psoriasis, a Stress-Related Skin Disease. *Disease Markers* 2018, 2018, 1–14. <https://doi.org/10.1155/2018/5823684>
6. Lee, S.; Xie, L.; Wang, Y.; Vaidya, N.; Baser, O. Comorbidity and Economic Burden among Moderate-to-Severe Psoriasis and/or Psoriatic Arthritis Patients in the US Department of Defense Population. *Journal of Medical Economics* 2018, 21 (6), 564–570. <https://doi.org/10.1080/13696998.2018.1431921>
7. Lopes, N.; Dias, L.L.S.; Azulay-Abulafia, L. et al. Humanistic and Economic Impact of Moderate to Severe Plaque Psoriasis in Brazil. *Adv Ther* 36, 2849–2865 (2019). <https://doi.org/10.1007/s12325-019-01049-7>
8. Wu, Ying, Douglas Mills, and Mohan Bala. "Impact of psoriasis on patients' work and productivity: a retrospective, matched case-control analysis." *American journal of clinical dermatology* 10 (2009): 407-410. <https://doi.org/10.1016/j.jaad.2007.07.023>
9. <https://doi.org/10.1016/j.jaad.2007.07.023>
10. <https://www.frontiersin.org/journals/medicine/articles/10.3389/fmed.2021.743180/full>
11. Reich, K. The Concept of Psoriasis as a Systemic Inflammation: Implications for Disease Management. *Journal of the European Academy of Dermatology and Venereology* 2012, 26, 3–11. <https://doi.org/10.1111/j.1468-3083.2011.04410.x>
12. Elewski, B.; Alexis, A. F.; Lebwohl, M.; Stein Gold, L.; Pariser, D.; Del Rosso, J.; Yosipovitch, G. Itch: An Under-Recognized Problem in Psoriasis. *Journal of the European Academy of Dermatology and Venereology* 2019, 33 (8), 1465–1476. <https://doi.org/10.1111/jdv.15450>
13. Pithadia, D. J.; Reynolds, K. A.; Lee, E. B.; Wu, J. J. Psoriasis-Associated Itch: Etiology, Assessment, Impact, and Management. *Journal of Dermatological Treatment* 2019, 31 (1), 18–26. <https://doi.org/10.1080/09546634.2019.1572865>
14. Gloria-Beatrice Wintermann; Bierling, A.; Eva M.J. Peters; Abraham, S.; Beissert, S.; Weidner, K. Psychosocial Stress Affects the Change of Mental Distress under Dermatological Treatment—a Prospective Cohort Study in Patients with Psoriasis. *Stress and Health* 2023, 40 (1). <https://doi.org/10.1002/smi.3263>
15. Lebwohl, M. G.; Kavanaugh, A.; Armstrong, A. W.; Van Voorhees, A. S. US Perspectives in the Management of Psoriasis and Psoriatic Arthritis: Patient and Physician Results from the Population-Based Multinational Assessment of Psoriasis and Psoriatic Arthritis (MAPP) Survey. *American Journal of Clinical Dermatology* 2015, 17 (1), 87–97. <https://doi.org/10.1007/s40257-015-0169-x>
16. Takeshita, J.; Grewal, S.; Langan, S. M.; Mehta, N. N.; Ogdie, A.; Van Voorhees, A. S.; Gelfand, J. M. Psoriasis and Comorbid Diseases. *Journal of the American Academy of Dermatology* 2017, 76 (3), 377–390. <https://doi.org/10.1016/j.jaad.2016.07.064>
17. Bu, J.; Ding, R.; Zhou, L.; Chen, X.; Shen, E. Epidemiology of Psoriasis and Comorbid Diseases: A Narrative Review. *Frontiers in Immunology* 2022, 13 (880201). <https://doi.org/10.3389/fimmu.2022.880201>. – de pus si pt depresie/anxietate
18. 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 Feb; 37(2): 146–155.
19. Gisondi P, Tessari G, Conti A et al. Prevalence of metabolic syndrome in patients with psoriasis: a hospital-based case-control study. *Br J Dermatol* 2007 Jul; 157(1): 68–73.
20. Chen Y-J, Wu C-Y, Shen J-L et al. Psoriasis independently associated with hyperleptinemia contributing to metabolic syndrome. *Arch Dermatol* 2008 Dec; 144(12): 1571–1575.
21. Augustin M, Reich K, Glaeske G, Schaefer I, Radtke M. Co-morbidity and age-related prevalence of psoriasis: analysis of health insurance data in Germany. *Acta Derm Venereol* 2010 Mar; 90(2): 147– 151.
22. Parisi R, Symmons DPM, Griffiths CEM, Ashcroft DM. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. *J Invest Dermatol* 2013 Feb; 133(2): 377–385.
23. Souza, C. S.; de Castro, C. C. S.; Carneiro, F. R. O.; Pinto, J. M. N.; Fabricio, L. H. Z.; Azulay-Abulafia, L.; Romiti, R.; Cestari, T. F.; Suzuki, C. E.; Biegun, P. M.; Guedes, L. S.; Oyafuso, L. K. M. Metabolic Syndrome and Psoriatic Arthritis among Patients with Psoriasis Vulgaris: Quality of Life and Prevalence. *The Journal of Dermatology* 2018, 46 (1), 3–10. <https://doi.org/10.1111/1346-8138.14706>

24. Gisondi, P.; Fostini, A. C.; Fossà, I.; Girolomoni, G.; Targher, G. Psoriasis and the Metabolic Syndrome. *Clinics in Dermatology* 2018, 36 (1), 21–28. <https://doi.org/10.1016/j.clindermatol.2017.09.005>.
25. Huang, P. L. A Comprehensive Definition for Metabolic Syndrome. *Disease Models & Mechanisms* 2009, 2 (5-6), 231–237. <https://doi.org/10.1242/dmm.001180>.
26. Samburskaya, O. V.; S. Yu. Kalinchenko; Batkaeva, N. V. BIOCHEMICAL PATHWAYS of METABOLIC DISORDERS in PSORIASIS. *Juvenis scientia* 2021, 7 (6), 6–16. https://doi.org/10.32415/jscentia_2021_7_6_6-16.
27. Chan, W.; Yew, Y.; Theng, C.; Liew, C.; Oon, H. Prevalence of Metabolic Syndrome in Patients with Psoriasis: A Cross-Sectional Study in Singapore. *Singapore Medical Journal* 2020, 194–199. <https://doi.org/10.11622/smedj.2019152>.
28. Korman, N. J. Management of Psoriasis as a Systemic Disease: What Is the Evidence? *British Journal of Dermatology* 2019, 182 (4), 840–848. <https://doi.org/10.1111/bjd.18245>.
29. Patrick, M. T.; Stuart, P. E.; Zhang, H.; Zhao, Q.; Yin, X.; He, K.; Zhou, X.; Mehta, N. N.; Voorhees, J. J.; Boehnke, M.; Gudjonsson, J. E.; Nair, R. P.; Handelman, S. K.; Elder, J. T.; Liu, D. J.; Tsoi, L. C. Causal Relationship and Shared Genetic Loci between Psoriasis and Type 2 Diabetes through Trans-Disease Meta-Analysis. *Journal of Investigative Dermatology* 2021, 141 (6), 1493–1502. <https://doi.org/10.1016/j.jid.2020.11.025>.
30. Gisondi, P.; Bellinato, F.; Girolomoni, G.; Albanesi, C. Pathogenesis of Chronic Plaque Psoriasis and Its Intersection with Cardio-Metabolic Comorbidities. *Frontiers in Pharmacology* 2020, 11. <https://doi.org/10.3389/fphar.2020.00117>.
31. Mehta, N. N.; Teague, H. L.; Swindell, W. R.; Baumer, Y.; Nicole Leanne Ward; Xing, X.; Baugous, B.; Johnston, A.; Joshi, A. A.; Silverman, J.; Barnes, D. H.; Wolterink, L.; Nair, R. P.; Stuart, P. E.; Playford, M.; Voorhees, J. J.; Sarkar, M. K.; Elder, J. T.; Gallagher, K.; Ganesh, S. K. IFN- γ and TNF- α Synergism May Provide a Link between Psoriasis and Inflammatory Atherogenesis. *Scientific Reports* 2017, 7 (1). <https://doi.org/10.1038/s41598-017-14365-1>
32. Rose, S.; Stansky, E.; Dagur, P. K.; Samsel, L.; Weiner, E.; Amir Jahanshad; Doveikis, J.; Naik, H. B.; Playford, M. P.; J Philip McCoy; Mehta, N. N. Characterization of Immune Cells in Psoriatic Adipose Tissue. *Journal of Translational Medicine* 2014, 12 (1). <https://doi.org/10.1186/s12967-014-0258-2>.
33. Gisondi, P.; Lora, V.; Bonauguri, C.; Russo, A.; Lippi, G.; Girolomoni, G. Serum Chemerin Is Increased in Patients with Chronic Plaque Psoriasis and Normalizes Following Treatment with Infliximab. *British Journal of Dermatology* 2013, 168 (4), 749–755. <https://doi.org/10.1111/bjd.12118>
34. Davidovici, B. B.; Sattar, N.; Jörg, P. C.; Puig, L.; Emery, P.; Barker, J. N.; van de Kerkhof, P.; Stähle, M.; Nestle, F. O.; Girolomoni, G.; Krueger, J. G. Psoriasis and Systemic Inflammatory Diseases: Potential Mechanistic Links between Skin Disease and Co-Morbid Conditions. *Journal of Investigative Dermatology* 2010, 130 (7), 1785–1796. <https://doi.org/10.1038/jid.2010.103>.
35. Blackstone B, Patel R, Bewley A. Assessing and Improving Psychological Well-Being in Psoriasis: Considerations for the Clinician. *Psoriasis (Auckl)*. 2022;12:25-33 <https://doi.org/10.2147/PTT.S328447>
36. Patel N, Nadkarni A, Cardwell LA, et al. Psoriasis, Depression, and Inflammatory Overlap: A Review. *Am J Clin Dermatol*. 2017;18(5):613-620. doi:10.1007/s40257-017-0279-8
37. Pistorio ML, Moretta T, Musumeci ML, et al. Impact of Attachment Style and Temperament Traits on the Quality of Life of Patients with Psoriasis. *Behav Sci (Basel)*. 2024;14(6):434. Published 2024 May 22. doi:10.3390/bs14060434
38. Dowlatshahi, E. A.; Wakkee, M.; Arends, L. R.; Nijsten, T. The Prevalence and Odds of Depressive Symptoms and Clinical Depression in Psoriasis Patients: A Systematic Review and Meta-Analysis. *Journal of Investigative Dermatology* 2014, 134 (6), 1542–1551. <https://doi.org/10.1038/jid.2013.508>
39. Adesanya, E. I.; Matthewman, J.; Schonmann, Y.; Hayes, J. F.; Henderson, A.; Mathur, R.; Mulick, A. R.; Smith, C. H.; Langan, S. M.; Mansfield, K. E. Factors Associated with Depression, Anxiety and Severe Mental Illness among Adults with Atopic Eczema or Psoriasis: A Systematic Review and Meta-Analysis. *British Journal of Dermatology* 2023, 188 (4), 460–470. <https://doi.org/10.1093/bjd/ljac132>

40. Mazzotti, E.; Barbaranelli, C.; Picardi, A.; Abeni, D.; Pasquini, P. Psychometric Properties of the Dermatology Life Quality Index (DLQI) in 900 Italian Patients with Psoriasis. *Acta Dermato-Venereologica* 2005, 85 (5), 409–413. <https://doi.org/10.1080/00015550510032832>.
41. FINLAY, A. Y.; KHAN, G. K. Dermatology Life Quality Index (DLQI)-a Simple Practical Measure for Routine Clinical Use. *Clinical and Experimental Dermatology* 1994, 19 (3), 210–216. <https://doi.org/10.1111/j.1365-2230.1994.tb01167.x>.
42. Lewis, V.; Finlay, A. Y. 10 Years Experience of the Dermatology Life Quality Index (DLQI). *Journal of Investigative Dermatology Symposium Proceedings* 2004, 9 (2), 169–180. <https://doi.org/10.1111/j.1087-0024.2004.09113.x>.
43. Bulat, V.; Šitum, M.; Delaš Aždajić, M.; Lovrić, I.; Dediol, I. Study on the Impact of Psoriasis on Quality of Life: Psychological, Social and Financial Implications. *Psychiatria Danubina* 2020, 32 (Suppl 4), 553–561.
44. Kimball, A. B.; Jacobson, C.; Weiss, S.; Vreeland, M. G.; Wu, Y. The Psychosocial Burden of Psoriasis. *American Journal of Clinical Dermatology* 2005, 6 (6), 383–392. <https://doi.org/10.2165/00128071-200506060-00005>.
45. Ljosaa, T. M.; Bondevik, H.; Halvorsen, J. A.; Carr, E.; Wahl, A. K. The Complex Experience of Psoriasis Related Skin Pain: A Qualitative Study. *Scandinavian Journal of Pain* 2020, 0 (0). <https://doi.org/10.1515/sjpain-2019-0158>.
46. Mattei, P. L.; Corey, K. C.; Kimball, A. B. 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. *Journal of the European Academy of Dermatology and Venereology* 2013, 28 (3), 333–337. <https://doi.org/10.1111/jdv.12106>.
47. Houghton, K.; Patil, D.; Gomez, B.; Feldman, S. R. Correlation between Change in Psoriasis Area and Severity Index and Dermatology Life Quality Index in Patients with Psoriasis: Pooled Analysis from Four Phase 3 Clinical Trials of Secukinumab. *Dermatology and Therapy* 2021, 11 (4), 1373–1384. <https://doi.org/10.1007/s13555-021-00564-2>.
48. Mueller, S. M.; Itin, P. H.; Navarini, A. A.; Goldust, M.; Brandt, O.; Griffiths, C. E. M.; Kleyn, C. E. The Relationship between PASI and DLQI with Itch, Stress, and Depression: Do We Need Additional Decision-Making Tools in Psoriasis? *Dermatologic Therapy* 2020, 33 (3). <https://doi.org/10.1111/dth.13276>.
49. Hu, B.; Yang, X.-R.; Xu, Y.; Sun, Y.-F.; Sun, C.; Guo, W.; Zhang, X.; Wang, W.-M.; Qiu, S.-J.; Zhou, J.; Fan, J. Systemic Immune-Inflammation Index Predicts Prognosis of Patients after Curative Resection for Hepatocellular Carcinoma. *Clinical Cancer Research* 2014, 20 (23), 6212–6222. <https://doi.org/10.1158/1078-0432.CCR-14-0442>.
50. Martínez-Ortega, J. M.; Nogueras, P.; Muñoz-Negro, J. E.; Gutiérrez-Rojas, L.; González-Domenech, P.; Gurpegui, M. Quality of Life, Anxiety and Depressive Symptoms in Patients with Psoriasis: A Case-Control Study. *Journal of Psychosomatic Research* 2019, 124, 109780. <https://doi.org/10.1016/j.jpsychores.2019.109780>.
51. Ahmad Fuat, M. S.; Mat Yudin, Z.; Muhammad, J.; Mohd Zin, F. Quality of Life and Its Associated Factors among Patients with Psoriasis in a Semi-Urban Northeast Malaysia. *IJERPH* 2022, 19 (18), 11578. <https://doi.org/10.3390/ijerph191811578>.
52. WHO Expert Consultation. Appropriate Body-Mass Index for Asian Populations and Its Implications for Policy and Intervention Strategies. *The Lancet* 2004, 363 (9403), 157–163. [https://doi.org/10.1016/s0140-6736\(03\)15268-3](https://doi.org/10.1016/s0140-6736(03)15268-3).
53. John William McEvoy; McCarthy, C. P.; Rosa Maria Bruno; Brouwers, S.; Canavan, M. D.; Ceconi, C.; Ruxandra Maria Christodorescu; Daskalopoulou, S. S.; Ferro, C. J.; Gerdt, E.; Hanssen, H.; Harris, J.; Lauder, L.; McManus, R. J.; Molloy, G. J.; Rahimi, K.; Regitz-Zagrosek, V.; Gian Paolo Rossi; Else Charlotte Sandset; Scheenaerts, B. 2024 ESC Guidelines for the Management of Elevated Blood Pressure and Hypertension. *European Heart Journal* 2024, 45 (38). <https://doi.org/10.1093/eurheartj/ehae178>.
54. Devrimci-Ozguven, H.; Kundakci, N.; Kumbasar, H.; Boyvat, A. The Depression, Anxiety, Life Satisfaction and Affective Expression Levels in Psoriasis Patients. *Journal of the European Academy of Dermatology and Venereology* 2000, 14 (4), 267–271. <https://doi.org/10.1046/j.1468-3083.2000.00085.x>.

55. García-Sánchez, L.; Montiel-Jarquín, Á. J.; Vázquez-Cruz, E.; May-Salazar, A.; Gutiérrez-Gabriel, I.; Loría-Castellanos, J. Quality of Life in Patients with Psoriasis. *Gaceta medica de Mexico* 2017, 153 (2), 185–189.
56. Geale, K.; Henriksson, M.; Schmitt-Egenolf, M. How Is Disease Severity Associated with Quality of Life in Psoriasis Patients? Evidence from a Longitudinal Population-Based Study in Sweden. *Health and Quality of Life Outcomes* 2017, 15 (1). <https://doi.org/10.1186/s12955-017-0721-x>.
57. Golpour, M.; Hosseini, S. H.; Khademloo, M.; Ghasemi, M.; Ebadi, A.; Koohkan, F.; Shahmohammadi, S. Depression and Anxiety Disorders among Patients with Psoriasis: A Hospital-Based Case-Control Study. *Dermatology Research and Practice* 2012, 2012. <https://doi.org/10.1155/2012/381905>.
58. Zachariae, R.; Zachariae, H.; Blomqvist, K.; Davidsson, S.; Molin, L.; MØrk, C.; Sigurgeirsson, B. Quality of Life in 6497 Nordic Patients with Psoriasis. *British Journal of Dermatology* 2002, 146 (6), 1006–1016. <https://doi.org/10.1046/j.1365-2133.2002.04742.x>.
59. Fortune, D. G.; Richards, H. L.; Main, C. J.; Griffiths, C. E. M. What Patients with Psoriasis Believe about Their Condition. *Journal of the American Academy of Dermatology* 1998, 39 (2), 196–201. [https://doi.org/10.1016/s0190-9622\(98\)70074-x](https://doi.org/10.1016/s0190-9622(98)70074-x).
60. Tang, J.; Zhao, S.; Shi, H.; Li, X.; Ran, L.; Cao, J.; He, Y. Effects on Peripheral and Central Nervous System of Key Inflammatory Intercellular Signalling Peptides and Proteins in Psoriasis. *Experimental Dermatology* 2024, 33 (5). <https://doi.org/10.1111/exd.15104>.
61. Riøl-Blanco, L.; Ordovas-Montanes, J.; Perro, M.; Naval, E.; Thiriout, A.; Alvarez, D.; Paust, S.; Wood, J. N.; von Andrian, U. H. Nociceptive Sensory Neurons Drive Interleukin-23-Mediated Psoriasiform Skin Inflammation. *Nature* 2014, 510 (7503), 157–161. <https://doi.org/10.1038/nature13199>.
62. Marek-Jozefowicz, L.; Czajkowski, R.; Borkowska, A.; Nedoszytko, B.; Żmijewski, M. A.; Cabała, W. J.; Slominski, A. T. The Brain–Skin Axis in Psoriasis—Psychological, Psychiatric, Hormonal, and Dermatological Aspects. *International Journal of Molecular Sciences* 2022, 23 (2), 669. <https://doi.org/10.3390/ijms23020669>.
63. Hunter, H. J. A.; Griffiths, C. E. M.; Kley, C. E. Does Psychosocial Stress Play a Role in the Exacerbation of Psoriasis? *British Journal of Dermatology* 2013, 169 (5), 965–974. <https://doi.org/10.1111/bjd.12478>.
64. Hall, J. M. F.; Cruser, desAnge; Podawiltz, A.; Mummert, D. I.; Jones, H.; Mummert, M. E. Psychological Stress and the Cutaneous Immune Response: Roles of the HPA Axis and the Sympathetic Nervous System in Atopic Dermatitis and Psoriasis. *Dermatology Research and Practice* 2012, 2012, 1–11. <https://doi.org/10.1155/2012/403908>.
65. Harvima, I. T.; Nilsson, G. Stress, the Neuroendocrine System and Mast Cells: Current Understanding of Their Role in Psoriasis. *Expert Review of Clinical Immunology* 2012, 8 (3), 235–241. <https://doi.org/10.1586/eci.12.1>.
66. Connor, C. J.; Liu, V.; Fiedorowicz, J. G. Exploring the Physiological Link between Psoriasis and Mood Disorders. *Dermatology Research and Practice* 2015, 2015. <https://doi.org/10.1155/2015/409637>.
67. Reddy, P.; Lent-Schochet, D.; Ramakrishnan, N.; McLaughlin, M.; Jialal, I. Metabolic Syndrome Is an Inflammatory Disorder: A Conspiracy between Adipose Tissue and Phagocytes. *Clinica Chimica Acta* 2019, 496, 35–44. <https://doi.org/10.1016/j.cca.2019.06.019>.
68. Elks, C. M.; Francis, J. Central Adiposity, Systemic Inflammation, and the Metabolic Syndrome. *Current Hypertension Reports* 2010, 12 (2), 99–104. <https://doi.org/10.1007/s11906-010-0096-4>.
69. Xu, T.; Zhang, Y.-H. Association of Psoriasis with Stroke and Myocardial Infarction: Meta-Analysis of Cohort Studies. *British Journal of Dermatology* 2012, 167 (6), 1345–1350. <https://doi.org/10.1111/bjd.12002>.
70. Masson, W.; Lobo, M.; Molinero, G. Psoriasis and Cardiovascular Risk: A Comprehensive Review. *Advances in Therapy* 2020, 37 (5), 2017–2033. <https://doi.org/10.1007/s12325-020-01346-6>.
71. Mosca, M.; Hong, J.; Hader, E.; Hakimi, M.; Brownstone, N.; Liao, W.; Bhutani, T. Psoriasis and Cardiometabolic Comorbidities: An Evaluation of the Impact of Systemic Treatments in Randomized Clinical Trials. *Dermatology and Therapy* 2021, 11 (5), 1497–1520. <https://doi.org/10.1007/s13555-021-00590-0>.
72. Ryan, C.; Kirby, B. Psoriasis Is a Systemic Disease with Multiple Cardiovascular and Metabolic Comorbidities. *Dermatologic Clinics* 2015, 33 (1), 41–55. <https://doi.org/10.1016/j.det.2014.09.004>.

73. Wu, J. J.; Kavanaugh, A.; Lebwohl, M. G.; Gniadecki, R.; Merola, J. F. Psoriasis and Metabolic Syndrome: Implications for the Management and Treatment of Psoriasis. *Journal of the European Academy of Dermatology and Venereology* 2022, 36 (6), 797–806. <https://doi.org/10.1111/jdv.18044>.
74. Hu, S.; Lan, C.-C. E. Psoriasis and Cardiovascular Comorbidities: Focusing on Severe Vascular Events, Cardiovascular Risk Factors and Implications for Treatment. *International Journal of Molecular Sciences* 2017, 18 (10), 2211. <https://doi.org/10.3390/ijms18102211>.
75. Hao, Y.; Zhu, Y.; Zou, S.; Zhou, P.; Hu, Y.; Zhao, Q.; Gu, L.; Zhang, H.; Wang, Z.; Li, J. Metabolic Syndrome and Psoriasis: Mechanisms and Future Directions. *Frontiers in Immunology* 2021, 12, 711060. <https://doi.org/10.3389/fimmu.2021.711060>.
76. Panagiota Anyfanti; Margouta, A.; Goulas, K.; Gavrilaki, M.; Lazaridou, E.; Aikaterini Patsatsi; Eugenia Gkaliagkousi. Endothelial Dysfunction in Psoriasis: An Updated Review. *Frontiers in Medicine* 2022, 9. <https://doi.org/10.3389/fmed.2022.864185>.
77. Ito, F.; Sono, Y.; Ito, T. Measurement and Clinical Significance of Lipid Peroxidation as a Biomarker of Oxidative Stress: Oxidative Stress in Diabetes, Atherosclerosis, and Chronic Inflammation. *Antioxidants* 2019, 8 (3), 72. <https://doi.org/10.3390/antiox8030072>.
78. Kang, H.; Li, X.; Xiong, K.; Song, Z.; Tian, J.; Wen, Y.; Sun, A.; Deng, X. The Entry and Egress of Monocytes in Atherosclerosis: A Biochemical and Biomechanical Driven Process. *Cardiovascular Therapeutics* 2021, 2021, 1–17. <https://doi.org/10.1155/2021/6642927>.
79. Langan, S. M.; Seminara, N. M.; Shin, D. B.; Troxel, A. B.; Kimmel, S. E.; Mehta, N. N.; Margolis, D. J.; Gelfand, J. M. Prevalence of Metabolic Syndrome in Patients with Psoriasis: A Population-Based Study in the United Kingdom. *Journal of Investigative Dermatology* 2012, 132 (3), 556–562. <https://doi.org/10.1038/jid.2011.365>.
80. Liakou, A.; Zouboulis, C. Links and Risks Associated with Psoriasis and Metabolic Syndrome. *Psoriasis: Targets and Therapy* 2015, 125. <https://doi.org/10.2147/ptt.s54089>.
81. Orloff, J. N.; Kaminetsky, J. R.; Aziz, M. Psoriasis and Obesity: A Review of the Current Literature. *SKIN The Journal of Cutaneous Medicine* 2018. <https://doi.org/10.25251/skin.2.6.4>.
82. Nakhwa, Y. C.; Rashmi, R.; Basavaraj, K. H. Dyslipidemia in Psoriasis: A Case Controlled Study. *International Scholarly Research Notices* 2014, 2014, 1–5. <https://doi.org/10.1155/2014/729157>.
83. Gisondi, P.; Del Giglio, M.; Girolomoni, G. Considerations for Systemic Treatment of Psoriasis in Obese Patients. *Am J Clin Dermatol* 2016, 17 (6), 609–615. <https://doi.org/10.1007/s40257-016-0211-7>.
84. Yang, J.; Hu, K.; Li, X.; Hu, J.; Tan, M.; Zhang, M.; Chen, J.; Kuang, Y. Psoriatic Foot Involvement Is the Most Significant Contributor to the Inconsistency Between PASI and DLQI: A Retrospective Study from China. *CCID* 2023, Volume 16, 443–451. <https://doi.org/10.2147/CCID.S396997>.
85. Räikkönen, K.; Matthews, K. A.; Kuller, L. H. Depressive Symptoms and Stressful Life Events Predict Metabolic Syndrome Among Middle-Aged Women. *Diabetes Care* 2007, 30 (4), 872–877. <https://doi.org/10.2337/dc06-1857>.
86. Katano, S.; Nakamura, Y.; Nakamura, A.; Suzukamo, Y.; Murakami, Y.; Tanaka, T.; Okayama, A.; Miura, K.; Okamura, T.; Fukuhara, S.; Ueshima, H. Relationship between Health-Related Quality of Life and Clustering of Metabolic Syndrome Diagnostic Components. *Qual Life Res* 2012, 21 (7), 1165–1170. <https://doi.org/10.1007/s11136-011-0029-y>.
87. Ford, E. S.; Li, C. Metabolic Syndrome and Health-Related Quality of Life among U.S. Adults. *Annals of Epidemiology* 2008, 18 (3), 165–171. <https://doi.org/10.1016/j.annepidem.2007.10.009>.
88. Lin, Y.-H.; Chang, H.-T.; Tseng, Y.-H.; Chen, H.-S.; Chiang, S.-C.; Chen, T.-J.; Hwang, S.-J. Changes in Metabolic Syndrome Affect the Health-Related Quality of Life of Community-Dwelling Adults. *Sci Rep* 2021, 11 (1), 20267. <https://doi.org/10.1038/s41598-021-99767-y>.
89. Pearl, R. L.; Wadden, T. A.; Hopkins, C. M.; Shaw, J. A.; Hayes, M. R.; Bakizada, Z. M.; Alfaris, N.; Chao, A. M.; Pinkasavage, E.; Berkowitz, R. I.; Alamuddin, N. Association between Weight Bias Internalization and Metabolic Syndrome among Treatment-Seeking Individuals with Obesity. *Obesity (Silver Spring)* 2017, 25 (2), 317–322. <https://doi.org/10.1002/oby.21716>.
90. Ozdemir, F.; Vardar Yagli, N.; Saglam, M.; Uyaroglu, O. A.; Calik Basaran, N.; Durusu Tanriover, M. Relationship between Blood Lipid Panel with Body Mass, Smoke Exposure, Dyspnea, Physical Activity,

- and Quality of Life in Patients with Dyslipidemia. *European Journal of Preventive Cardiology* 2024, 31 (Supplement_1), zwae175.054. <https://doi.org/10.1093/eurjpc/zwae175.054>.
91. Czarnecka, A.; Zabłotna, M.; Purzycka-Bohdan, D.; Nowicki, R. J.; Szczerkowska-Dobosz, A. An Observational Study of 147 Psoriasis Patients: Overweightness and Obesity as a Significant Clinical Factors Correlated with Psoriasis. *Medicina* 2023, 59 (11), 2006. <https://doi.org/10.3390/medicina59112006>.
 92. Gambichler, T.; Würfel, L.; Abu Rached, N.; Mansour, R.; Bechara, F. G.; Scheel, C. H. Systemic Immune-inflammation Biomarkers in Psoriasis Patients under Interleukin 17A -inhibitor Treatment. *Acad Dermatol Venereol* 2023, 37 (7). <https://doi.org/10.1111/jdv.18895>.
 93. Cozma, E. C.; Găman, M.-A.; Orzan, O.; Hamed, K.-V.; Voiculescu, V. M.; Găman, A.-M. Oxidative Stress and Inflammation Levels in a Population of Eastern European Naïve Versus Treated Psoriasis Patients. *Cureus* 2023. <https://doi.org/10.7759/cureus.48177>.
 94. Hagino, T.; Saeki, H.; Fujimoto, E.; Kanda, N. Long-Term Effectiveness and Safety of Deucravacitinib in Psoriasis: A 52-Week Real-World Study of Genital, Scalp, and Nail Lesions. *Clinical and Experimental Dermatology* 2024, llae530. <https://doi.org/10.1093/ced/llae530>

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.