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EXPLORING THE POTENTIAL OF GLP-1 RECEPTOR AGONISTS IN PSORIASIS TREATMENT: A REVIEW ARTICLE

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ABSTRACT

Background: Psoriasis is a chronic immune-mediated skin disease associated with systemic inflammation and frequent comorbidities, such as obesity and type 2 diabetes. Its prevalence varies globally, with Poland reporting some of the highest rates in Central and Eastern Europe—approximately 2.9% of adults—compared to 1.5–2.0% in Western European countries. Despite this, Polish populations remain underrepresented in international clinical research. Emerging data suggest that glucagon-like peptide-1 receptor agonists (GLP-1 RAs), originally developed for type 2 diabetes, may offer therapeutic benefits in psoriasis due to their immunomodulatory and metabolic effects.

Objective: This narrative review aims to evaluate the current evidence on the role of GLP-1 RAs in the treatment of psoriasis, focusing on their mechanisms of action, clinical outcomes, and potential relevance in populations with high rates of metabolic comorbidities.

Methods: This article presents a narrative literature review based on a structured search of PubMed, Scopus, and Web of Science databases for articles published between January 2010 and March 2025. Search terms included combinations of "psoriasis," "GLP-1 receptor agonists," "liraglutide," "semaglutide," "inflammation," "IL-17," "obesity," and "metabolic syndrome." Both preclinical and clinical studies were included.

Results: GLP-1 RAs have demonstrated anti-inflammatory properties through modulation of cytokines such as IL-17, IL-23, and TNF-α. Clinical studies and case reports suggest improvements in Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index (DLQI), particularly among patients with obesity or type 2 diabetes. However, evidence remains limited and inconsistent in non-diabetic or glucose-tolerant individuals.

Conclusions: GLP-1 RAs may represent a novel adjunctive strategy for psoriasis treatment, especially in patients with coexisting metabolic disorders. Given the high prevalence of psoriasis and obesity in Poland, further regionally focused, large-scale clinical studies are needed to assess efficacy, safety, and population-specific applicability.

Keywords: psoriasis, GLP-1 receptor agonists, liraglutide, semaglutide, inflammation, IL-17, obesity, and metabolic syndrome.

INTRODUCTION

Psoriasis is a chronic, immune-mediated skin disease affecting approximately 2–3% of the global population. It is characterized by aberrant activation of the IL-23/IL-17 axis, leading to epidermal hyperproliferation and systemic inflammation. The disease is associated not only with cutaneous symptoms but also with significant comorbidities, including obesity, type 2 diabetes, metabolic syndrome, and cardiovascular disease [1, 2].

In Poland, psoriasis affects nearly 3% of adults, placing the country among the most affected in Central and Eastern Europe. Despite this, region-specific studies and therapeutic trials remain scarce. As metabolic comorbidities become increasingly prevalent, new therapeutic approaches are being explored that can simultaneously target inflammatory and metabolic pathways [3].

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), including liraglutide and semaglutide, are widely used in the treatment of type 2 diabetes and obesity. Beyond their metabolic effects, recent studies suggest they may also exert immunomodulatory actions by downregulating pro-inflammatory cytokines such as IL-17 and TNF-α. These properties have sparked growing interest in their potential application in psoriasis management, particularly in patients with metabolic comorbidities [4-6].

AIMS

This narrative review aims to fill a gap in the existing literature on the use of GLP-1 receptor agonists in psoriasis. It focuses on immunological mechanisms, clinical outcomes, and their potential therapeutic role in populations with a high burden of both inflammatory and metabolic disease.

METHODS

Our literature search strategy based on a structured selection conducted in PubMed, Scopus, and Web of Science. The search covered publications from January 2010 to March 2025. The following keywords and their combinations were used: psoriasis, GLP-1 receptor agonists, liraglutide, semaglutide, inflammation, IL-17, obesity, metabolic syndrome, biologic therapy. Titles and abstracts were screened for relevance, and full texts were reviewed to confirm eligibility.

RESULTS OF FINDINGS

Specifically, we present the mechanisms of action of GLP-1 in psoriasis and the observed clinical effects based on a literature review.

PSORIASIS

Psoriasis is a long-term inflammatory condition that primarily affects the skin and joints, though it can involve multiple systems in the body [7]. The WHO defines psoriasis as a chronic, painful, and debilitating noncommunicable disease (NCD) characterized by disfiguring skin lesions, for which there is no known cure. The condition significantly impacts patients' quality of life [8]. The prevalence of psoriasis is known in approximately 20% of countries, ranging from 0.14% in East Asia to 1.99% in Australasia. Its distribution varies geographically and is closely associated with ethnicity. It is most common among Caucasians in Central and Western Europe (1.83% and 1.92%, respectively) and North America (1.50%). Data on the incidence of psoriasis are limited, with most reports originating from high-income countries, particularly in Europe and North America. The disease can onset at any age, but typically shows two peaks: between 30–39 years and 60–69 years for men, and 18–29 years and 50–59 years for women. Psoriasis is also becoming more prevalent in children, with rates of 0.13% in infants (0–2 years) and 0.67% in adolescents (14–18 years), indicating it is relatively uncommon in the pediatric population. There is a trend of decreasing incidence, potentially due to improved diagnosis and early treatment, though patients with psoriasis still have a reduced life expectancy due to comorbidities associated with the disease. The prevalence is similar between sexes, though men tend to experience more severe forms of psoriasis and are more likely to receive systemic biological therapies than women [9]. Genetics play a significant role in the development of this disease [10]. Psoriasis has a notable genetic component, with over 20% of patients reporting a family history of the condition. Key factors involved in the susceptibility and development of psoriasis include interleukins (ILs), human leukocyte antigens (HLAs), genes associated with the nuclear factor kappa B (NF-κB) pathway, interferons (IFNs), and those in the Janus kinase (JAK)-signal transducer and activator of transcription (STAT) pathway. Genetic predisposition to psoriasis is often linked to immune system dysregulation, which is influenced by specific high-risk genetic variants [11]. Current research indicates that psoriasis is a T-cell-driven disorder characterized by an overproduction of IL-17, which is triggered by IL-23 stimulation. The IL-23/IL-17 axis plays a central role in shaping the inflammatory response seen in psoriasis, driving a continuous cycle of pro-inflammatory activity. This leads to the activation and proliferation of keratinocytes, which contribute to the formation of psoriatic lesions [12]. Psoriasis can manifest in various forms, including plaque, flexural, guttate, pustular, or erythrodermic types [10]. The diagnosis is mainly based on clinical evaluation, and a skin biopsy is rarely necessary [7]. Psoriasis treatments encompass a range of therapeutic options, including topical agents such as vitamin D analogues and corticosteroids, as well as phototherapy using narrowband ultraviolet B (NB-UVB) or psoralen and ultraviolet A (PUVA) radiation. Systemic therapies include

traditional treatments like methotrexate, ciclosporin, and acitretin, as well as biologic therapies targeting tumor necrosis factor (TNF), interleukin (IL)-17, and IL-23 inhibitors. Additionally, small molecule inhibitors such as dimethyl fumarate and apremilast are also used in the management of psoriasis [10]. Psoriasis is associated with psoriatic arthritis, malignancy, inflammatory bowel disease, increased rates of cardiometabolic (stroke, hypertension, myocardial infarction), hepatic and psychological comorbidity [7, 9, 10, 13]. Obesity is prevalent among individuals with psoriasis, especially those with severe disease, and is often accompanied by other metabolic conditions such as diabetes. These comorbidities are associated with ongoing systemic inflammation, which increases the risk of cardiovascular diseases and related complications. Research suggests that obesity-related inflammation may play a role in the development or worsening of psoriasis. Additionally, obese patients with psoriasis tend to have more difficult treatment responses and face a higher likelihood of developing conditions like dyslipidemia, hypertension, and diabetes. As such, obesity represents a significant challenge in the management of psoriasis [4].

GLUCAGON-LIKE-PEPTIDE-1 RECEPTOR AGONISTS

The glucagon-like peptide-1 (GLP-1) receptor (GLP-1R) is a key member of the G protein-coupled receptor (GPCR) family, primarily found on the surface of various cells throughout the body. It binds to GLP-1, a hormone that plays a vital role in regulating blood glucose levels, lipid metabolism, and other important physiological functions. GLP-1 receptor agonists (GLP-1RAs) have become a focal point in medical research due to their novel mechanisms of action, significant therapeutic efficacy, and broad potential for treating various conditions. Initially developed for managing type 2 diabetes mellitus (T2DM), GLP-1RAs are now being explored for their benefits in a wide range of diseases. Research findings indicate that GLP-1 receptor agonists (GLP-1RAs) could be a highly promising treatment option for psoriasis patients with comorbid diabetes or obesity, providing both cardiovascular benefits and potential improvements in psoriatic symptoms [13]. GLP-1RAs promote weight loss, protect islet β-cells, enhance β-cell proliferation, and have minimal side effects, making them an effective treatment for T2DM. Beyond their role in diabetes management, these drugs have shown promise in treating a variety of other conditions due to their broad biological effects. GLP-1Rs are widely distributed across tissues, including the pancreas, and their activation has been linked to reducing neuroinflammation, promoting nerve regeneration, improving cardiovascular function, suppressing appetite, delaying gastric emptying, and regulating lipid metabolism. Recent studies have highlighted the potential of GLP-1RAs in areas such as neuroprotection, cardiovascular protection, anti-infective measures, and metabolic regulation. Their applications are expanding to include the treatment of obesity, non-alcoholic fatty liver disease (NAFLD), neurodegenerative disorders, musculoskeletal inflammation, and even certain types of cancer. Additionally, there is increasing interest in understanding the relationship between GLP-1RAs and tumorigenesis in patients with T2DM, as well as their effects on cancer development and prognosis [14, 15].

GLUCAGON-LIKE-PEPTIDE-1 RECEPTOR AGONISTS AND PSORIASIS

Many recent studies have also provided information on possible positive effects of GLP-1RAs treatment on psoriasis management. In patients with psoriasis and type 2 diabetes treated with GLP-1RAs, such as liraglutide or semaglutide, significant improvements in psoriasis severity were observed. Notably, these improvements occurred shortly after treatment began, even before achieving glycemic control or weight loss, suggesting that the benefits may be due to the immunomodulatory effects of GLP-1RAs. GLP-1 analogues have become recognized not only for their effectiveness in promoting weight loss but also for their anti-inflammatory properties, which can help reduce the systemic inflammation often seen in obesity and contribute to psoriasis improvement. A meta-analysis of clinical trials found that liraglutide significantly reduced the Psoriasis Area and Severity Index (PASI) in diabetic patients, with a notable decrease in psoriasis severity. Liraglutide was also shown to improve the Dermatology Life Quality Index (DLQI) and decrease levels of pro-inflammatory cytokines such as IL-17, IL-23, and TNF-α, which are implicated in both psoriasis and obesity. Similarly, semaglutide led to a 19% reduction in PASI and significant improvements in DLQI scores in diabetic patients with psoriasis. While most studies on GLP-1 analogues in psoriasis have focused on diabetic patients, further research is needed to explore their effects in obese patients without diabetes, in order to better understand their broader therapeutic potential for treating psoriasis across different populations [4, 5]. In a research no effect of GLP-1 in psoriasis management was found in in glucose-tolerant patients [16].

REVIEW OF CLINICAL EVIDENCE: GLP-1 RECEPTOR AGONISTS IN PSORIASIS

Numerous reports suggest that treatment with GLP-1 agonists can significantly improve psoriasis symptoms. While numerous clinical observations support the potential benefits of GLP-1 receptor agonists in psoriasis, many studies remain limited in design or scale. For instance, Lin et al. (2022) conducted a randomized controlled trial involving patients with type 2 diabetes and psoriasis treated with liraglutide, demonstrating significant PASI and DLQI improvement alongside reductions in IL-17 and IL-23. Similarly, Nicolau et al. (2023) reported notable clinical and histological improvement in obese patients treated with liraglutide 3 mg over three months, independent of weight loss. However, a randomized placebo-controlled study by Faurschou et al. (2015) in glucose-tolerant patients failed to show any benefit of liraglutide, suggesting that therapeutic effects may depend on underlying metabolic status. Overall, the available evidence is promising but heterogeneous, emphasizing the need for larger, well-controlled clinical trials that clearly define inclusion criteria, patient profiles, and endpoints related to psoriasis severity, systemic inflammation, and metabolic comorbidities.

The elevated levels of GLP-1 receptors observed in psoriatic plaques are likely attributed to the infiltration of immune

cells. This phenomenon may help clarify why GLP-1 receptor agonists have a therapeutic effect on psoriasis patients [17]. A study observed that treating psoriasis with a GLP-1 RA, an incretin-mimetic, led to a noticeable reduction in disease severity within a few days, independent of blood sugar levels. After six weeks of GLP-1 analogue therapy, patients showed significant improvement in psoriasis symptoms. This was accompanied by changes in iNKT cell distribution, with higher levels in the bloodstream and fewer in psoriatic plaques. Laboratory tests confirmed that iNKT cells express GLP-1 receptors and that GLP-1 inhibits their cytokine secretion in a dose-dependent manner, while not affecting their ability to perform cytolytic functions [18]. A different study confirmed that GLP-1-RA are associated with a notable decrease in the risk of all-cause mortality and immune-mediated inflammatory diseases among individuals with type 2 diabetes and coexisting immune-mediated conditions. The results mirror those seen in patients without immune-mediated inflammatory disorders [19]. In another study, significant improvement in psoriatic skin lesions was observed in patients with type 2 diabetes following treatment with liraglutide. This effect appeared to be associated with a reduction in key inflammatory markers, including IL-17, IL-23, and TNF-α, highlighting the potential role of liraglutide in modulating inflammatory processes in psoriasis [20]. It has been also demonstrated that GLP-1RA therapy, such as liraglutide, can alleviate the severity of psoriasis in patients with coexisting type 2 diabetes. Notably, this improvement appears to occur independently of changes in body weight or glycemic control [21]. GLP-1RAs have shown effectiveness in patients unresponsive to other treatments, particularly those with obesity who may also gain additional advantages from this therapy [22]. Research has demonstrated that a three-month course of liraglutide 3 mg is both effective and safe for reducing weight and improving psoriatic lesions in individuals with obesity and psoriasis. Importantly, the observed improvements in skin lesions occurred independently of weight loss, suggesting avenues for further exploration [23]. A case report detailed a 59-year-old man with psoriasis and type 2 diabetes who showed noticeable improvement in his skin condition shortly after beginning liraglutide treatment. Symptoms like itching and scaling decreased quickly, with further progress over three months. Notably, the patient had previously achieved good diabetes control without any effect on his psoriasis, and the improvement with liraglutide began before weight loss occurred. This suggests a possible direct anti-inflammatory effect of liraglutide, alongside indirect benefits from managing comorbidities such as obesity [24]. Adverse effects can occasionally occur with liraglutide, as highlighted in a case report describing a rare generalized skin reaction. The vesicular rash appeared approximately two and a half weeks after starting liraglutide and resolved gradually within two weeks after discontinuation. Such reactions are uncommon, but dermatologists should be aware of the potential for liraglutide-related cutaneous side effects [25]. While these findings are encouraging, there is a clear need for further research into the effects of GLP-1RAs on psoriatic arthritis (PsA). Although weight loss and reduced systemic inflammation are likely to benefit both psoriasis and PsA, specific studies investigating the direct impact of GLP-1 therapies on PsA are currently limited. Understanding how these agents influence joint inflammation and disease progression could provide critical insights and expand their therapeutic applications. GLP-1RAs represent a promising avenue for psoriasis management, offering benefits that extend beyond metabolic improvements. Their ability to directly modulate immune responses, combined with their metabolic advantages, positions them as valuable therapeutic options for patients with psoriasis, particularly those with coexisting obesity or type 2 diabetes. However, further research is needed to fully understand their mechanisms of action and to establish their long-term safety and efficacy in broader patient populations.

CLINICAL AND OBSERVATIONAL EVIDENCE

Several clinical observations and case studies have reported improvements in psoriasis symptoms in patients treated with GLP-1 receptor agonists (GLP-1 RAs), particularly among individuals with metabolic comorbidities such as obesity and type 2 diabetes. These effects include reductions in PASI and DLQI scores, as well as decreases in systemic inflammatory markers such as C-reactive protein (CRP), IL-17, and TNF-α. However, the available data are heterogeneous in study design, sample size, and outcome reporting.

Table 1. Studies Investigating the Effects of GLP-1 Receptor Agonists on Psoriasis

Study	Year	Design / Type	Drug	Population	Main Findings	Notes
Faurschou et al.	2015	RCT (placebo-controlled)	Liraglutide	Non-diabetic patients with psoriasis	No significant PASI improvement	Possible lack of effect in glucose-tolerant subjects
Lin et al.	2022	RCT	Liraglutide	Psoriasis + T2DM	PASI and DLQI significantly improved; ↓IL-17, ↓IL-23	Improvement correlated with metabolic markers
Nicolau et al.	2023	Case series	Liraglutide	Obese patients with	Clinical and histological improvement	Effect partially independent

				psoriasis	after 3 months	of weight loss
Al-Janabi et al.	2019	Observational	Liraglutide	Diabetic patients	Reported improvements in skin symptoms and CRP	Non-controlled study
Animal studies	—	Preclinical	GLP-1 RAs	Murine psoriasis models	↓TNF-α, ↓IL-17, ↓keratinocyte proliferation	Mechanism-focused

PROPOSED MECHANISMS OF ACTION

The beneficial effects of GLP-1 RAs in psoriasis are thought to result from both immunomodulatory and metabolic pathways. Key mechanisms include inhibition of Th17 cells, reduction of pro-inflammatory cytokines (IL-17, IL-23, TNF-α), improved insulin sensitivity, and weight loss. These effects are particularly relevant in patients with obesity-associated or metabolically driven psoriasis.

Table 2. Proposed Mechanisms of GLP-1 Receptor Agonists in Psoriasis

Pathogenic Target	Effect of GLP-1 RAs	Mechanism	References
IL-17	↓ Expression	Inhibition of Th17 differentiation	Faurschou et al., 2015; Lin et al., 2022
IL-23	↓ Expression	Reduced dendritic cell activation	Lin et al., 2022
TNF-α	↓ Serum levels	Systemic anti-inflammatory effect	Preclinical studies
Th17 cells	↓ Activation	Indirect immune modulation	Animal models
CRP	↓ Concentration	Reduced systemic inflammation	Al-Janabi et al., 2019
Insulin resistance	↓ HOMA-IR	Improved insulin sensitivity	Nicolau et al., 2023
BMI	↓ Body weight	Appetite regulation and metabolic shift	Multiple studies

PSORIASIS TREATMENT

Topical treatments are commonly used for mild to moderate psoriasis and moderate to severe cases typically require systemic therapies. Treatment choices are also influenced by the presence of comorbidities, such as psoriatic arthritis [26]. Recent developments in topical therapies, such as microneedles and nanoparticle-based delivery systems, show potential for enhancing drug delivery and improving the efficacy of treatments for psoriatic plaques [27]. Salicylic acid can be combined with other topical treatments, including corticosteroids and calcineurin inhibitors, to enhance their absorption into thick, localized psoriatic plaques [28]. Additionally, emollients can be used to moisturize and hydrate the skin, while keratolytic agents, such as urea, help remove dead skin cells and promote skin renewal [29]. Treatment options for psoriasis range from topical therapies, such as vitamin D analogs and corticosteroids, to phototherapy methods, including narrowband ultraviolet B (NB-UVB) and psoralen with ultraviolet A (PUVA) [10]. NB-UVB phototherapy is a first-line treatment for extensive plaque psoriasis, offering greater efficacy than broadband UVB and a safer profile compared to PUVA. Standard NB-UVB therapy typically involves three sessions per week for a minimum of three months [30]. Systemic treatments include traditional options like methotrexate, ciclosporin, and acitretin, as well as small molecule inhibitors like dimethyl fumarate and apremilast [10]. Despite the potential risk of hepatotoxicity, methotrexate (MTX) seems to offer the most favorable balance of safety and efficacy among systemic agents when considering serious adverse events [31]. Greater focus has been placed on creating noninvasive methods, such as imaging techniques and serum biomarker tests, to detect early liver fibrosis and reduce reliance on liver biopsies [32].

Systemic treatment for plaque psoriasis involves two key phases: an induction phase, typically lasting up to 16 weeks (or extended to 24 weeks based on the drug and dosage), and a maintenance phase, which begins after the induction period [33]. Advancements in treatments for moderate to severe plaque psoriasis include biologics targeting tumor necrosis factor-alpha (TNF-α), interleukin-12/23 (IL-12/23, p40), interleukin-17 (IL-17), and interleukin-23 (IL-23, p19), as well as an oral phosphodiesterase 4 inhibitor [34]. When selecting a biologic drug, certain factors must be taken into account. TNF-α blockers may be unsuitable or require caution in patients with conditions such as latent tuberculosis, severe heart failure, demyelinating diseases, or alopecia areata. Conversely, IL-17 inhibitors are relatively contraindicated in cases of inflammatory bowel diseases like Crohn's disease, where anti-TNF-α monoclonal antibodies may be a more appropriate choice due to their efficacy in treating both conditions [35]. Moreover, TNF-α inhibitors not only reduce insulin resistance but may also lower the risk of diabetes and cardiovascular co-morbidities in patients. This therapy could improve outcomes by addressing both psoriasis and related co-morbidities [36]. TNF-α inhibitors can lead to the development of antidrug antibodies and are recommended to be used in combination with methotrexate [37]. Various hypoglycemic agents, including not only GLP-1RAs but also thiazolidinediones, DPP-4 inhibitors, and biguanides, have been shown to significantly reduce the Psoriasis Area and Severity Index (PASI) score from baseline. Their antipsoriatic effects may be attributed not only to their glucose-lowering properties but also to mechanisms such as inhibiting keratinocyte hyperproliferation, promoting the expression of differentiation markers, suppressing inflammatory immune responses, and interfering with calcium channels and MAPK signaling pathways [38]. Metformin is considered safe for psoriasis and shows strong therapeutic benefits in patients with comorbidities like diabetes, metabolic syndrome, and obesity. However, its efficacy as monotherapy in psoriasis without comorbidities remains unclear, highlighting the need for larger clinical trials [39]. Metformin's ability to suppress TNF-α- and IL-17A-induced inflammatory responses in keratinocytes indicates its potential as an immunomodulatory treatment for psoriasis patients with type 2 diabetes [40]. Additionally, statins, commonly prescribed for hyperlipidemia, have demonstrated benefits in psoriasis by reducing disease severity, cardiovascular risk, and improving the efficacy of topical corticosteroids. These effects are linked to immune modulation, such as inhibiting leukocyte adhesion and lowering inflammatory markers like CRP, TNF-α, IL-1, and IL-6 [41]. Psoriasis severity, progression, and symptoms can be influenced by various external factors, such as dietary habits, nutrient intake, and lifestyle choices, including smoking, alcohol use, stress, lack of sleep, and a sedentary lifestyle [42]. Adopting lifestyle changes can enhance both disease management and overall quality of life [43]. Dietary interventions can improve psoriasis severity in specific groups. For example, low-calorie diets are effective for individuals with obesity or overweight, while gluten-free diets benefit those with celiac disease or gluten sensitivity. Furthermore, anti-inflammatory diets, such as the Mediterranean diet, may help manage psoriasis by influencing the inflammatory pathways associated with the disease [44]. Vitamin D plays a crucial role in the proliferation and maturation of keratinocytes, making it a valuable therapeutic option for psoriasis. Given the widespread prevalence of vitamin D deficiency, patients who are not using topical vitamin D analogs may benefit from supplementation [45]. In addition to dietary changes, the most well-supported complementary and alternative treatments for psoriasis include indigo naturalis, curcumin, fish oil supplementation, mindfulness practices [46]. Weight loss has been shown to improve psoriasis and reduce the risk of its onset in individuals with obesity. Addressing obesity may therefore play a role in lessening the impact of psoriasis both individually and socially. Lifestyle changes, such as diet and exercise, lead to modest improvements in psoriasis, while significant weight loss through bariatric surgery, especially gastric bypass, may prevent its development in obese patients [47]. Exercise may reduce psoriasis severity partly by lowering excess adipose tissue, which is often associated with obesity. A reduction in fat mass leads to a decrease in the release of inflammatory cytokines that contribute to psoriasis development. This decrease not only limits the factors driving chronic plaque psoriasis but also helps alleviate systemic low-grade inflammation [48]. Psoriasis patients often face significant sleep disturbances and metabolic issues linked to unhealthy lifestyles. Sleep disorders can disrupt skin homeostasis and worsen psoriasis outcomes. Factors like alcohol, smoking, pollution, infections, and vaccines may further influence disease progression and sleep quality. Clinicians should include sleep assessments in psoriasis management [49]. Moreover, research indicates that psychiatric conditions are common in individuals with psoriasis, with prevalence rates ranging from 24% to 90%. However, the link between psoriasis and mental disorders is often overlooked in clinical practice, leading to underdiagnosis. Psychiatric evaluations for these patients could offer significant therapeutic benefits and improve overall care [50].

CONCLUSIONS

GLP-1 receptor agonists (GLP-1 RAs) represent a promising therapeutic option for patients with psoriasis, particularly those with coexisting obesity or type 2 diabetes. Beyond their established metabolic effects, these agents demonstrate immunomodulatory activity through suppression of key inflammatory pathways, including IL-17 and TNF-α, which are central to psoriasis pathogenesis.

Incorporating GLP-1 RAs into the management of psoriasis may provide a dual therapeutic advantage—targeting both chronic inflammation and metabolic dysfunction. This aligns with the broader goal of integrated, patient-centered care, addressing not only cutaneous disease but also the systemic burden and overall quality of life in individuals living with psoriasis.

From a clinical perspective, GLP-1 receptor agonists may be considered in psoriasis patients with metabolic comorbidities, particularly obesity or type 2 diabetes, where conventional treatments have limited efficacy or tolerability. Clinicians should monitor metabolic parameters alongside skin symptoms and be aware of the potential immunological benefits of GLP-1 RAs. Until large trials are available, off-label use should be guided by individual risk–benefit

assessment and patient-specific factors.

DISCLOSURES

CONFLICTS OF INTEREST

Authors have no conflict of interest to declare.

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AUTHORS' CONTRIBUTIONS

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