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THE ROLE OF GLP-1 RECEPTOR AGONISTS IN THE TREATMENT OF PSORIASIS: A POTENTIAL THERAPEUTIC APPROACH

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ABSTRACT

Psoriasis is a chronic inflammatory disease with complex pathogenesis. There is growing evidence that excess body fat can lead to systemic inflammation, which exacerbates the symptoms of the disease.
The aim of this study was to review the available scientific literature on the use of GLP-1 receptor agonists in the treatment of psoriasis. Their mechanism of action not only includes better glycaemic control, weight loss and reduction of inflammation, but also has a significant effect on the immune response of T lymphocytes, NK cells and macrophages.
Despite small study groups and a short observation period, the available clinical studies show a reduction in the PASI index and inflammatory markers, with the positive therapeutic effect being most noticeable in patients with obesity and type 2 diabetes.
GLP-1 receptor agonists are a promising therapeutic pathway for the treatment of psoriasis, especially in patients with metabolic disorders. Unfortunately, further long-term clinical trials involving a larger number of subjects are needed to confirm the efficacy and safety of this form of treatment.

KEYWORDS

Psoriasis, GLP-1 Receptor Agonists, PASI Index, Metabolism, Pro-Inflammatory Cytokines

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Introduction

Psoriasis is a chronic, recurrent, inflammatory skin disease affecting approximately 125 million people worldwide, with a prevalence ranging from 0.1% to 8% depending on geographic region [1,2]. The disease is characterized by abnormal keratinocyte proliferation and impaired epidermal differentiation, leading to excessive accumulation of cells within the stratum corneum. Clinically, psoriasis presents as erythematous, round or oval, scaly plaques most commonly located on the extensor surfaces of the limbs (elbows and knees), the scalp, and the lower back; however, any skin surface may be involved [3,4]. Histopathological features include loss of the granular layer, elongation of the dermal papillae with dilated blood vessels, and increased leukocyte infiltration, all of which are associated with activation of immune pathways [3].

The management of psoriasis remains challenging, particularly due to the high prevalence of comorbidities, among which the increased risk of metabolic syndrome is of particular importance, affecting up to one-third of patients [5]. According to current diagnostic criteria, metabolic syndrome is defined by the presence of central obesity or increased waist circumference in combination with at least two additional metabolic abnormalities, including elevated blood pressure, impaired glucose metabolism, or dyslipidemia associated with an increased risk of atherosclerosis. An increasing body of clinical evidence indicates that obesity significantly influences the course of psoriasis. Patients with increased body weight and elevated body mass index (BMI) are at higher risk of developing psoriasis and tend to experience a more severe disease course [6]. Importantly, this relationship appears to be bidirectional, as psoriasis-associated cytokines, such as TNF-α, IL-1β, and IL-6, affect adipose tissue and participate in key mechanisms of triglyceride metabolism and preadipocyte differentiation, thereby promoting the development of obesity [7].

For this reason, growing interest has focused on glucagon-like peptide-1 (GLP-1) receptor agonists, traditionally used in the treatment of obesity and type 2 diabetes mellitus. In addition to promoting weight reduction, these agents exert anti-inflammatory effects that may beneficially influence the course of psoriasis. Consequently, GLP-1 receptor agonists may represent a promising adjunctive therapeutic strategy in patients with psoriasis and coexisting obesity.

GLP-1 Agonists – Mechanism and Immunological Effects

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) constitute a modern group of incretin-based medications, including synthetic GLP-1 analogues that mimic the action of one of the gastrointestinal hormones – GLP-1 [9]. These agents bind to GLP-1 receptors located, among others, in the pancreas, kidneys, nervous system, cardiovascular system, and cells of the immune system. They are characterized by increased enzymatic stability against dipeptidyl peptidase-4 (DPP-4) and slower elimination kinetics compared with endogenous GLP-1, thereby prolonging their pharmacological duration of action [10, 11].

The therapeutic effect of GLP-1 agonists is multidirectional. It includes stimulation of insulin secretion in a manner dependent on postprandial blood glucose concentration, inhibition of glucagon secretion, and delayed gastric emptying. Moreover, these agents reduce appetite through effects on the central nervous system, which promotes body weight reduction. These properties determine their use in the pharmacotherapy of type 2 diabetes, obesity, dyslipidemia, and obesity-related metabolic diseases [9, 10]. GLP-1 receptor agonists demonstrate the ability to modulate numerous pro-inflammatory mechanisms, including glucotoxicity, oxidative stress, recruitment of immune system cells, cytokine production, and lipotoxicity [11].

Metabolic Effect

The metabolic effect associated with the use of GLP-1 receptor agonists has been confirmed in large randomized trials. A study published in 2024 by Donna H. Ryan et al., involving 17,604 adults with overweight or obesity without diabetes, demonstrated that semaglutide administered over a period of 4 years was associated with a mean body weight reduction of 10.2% and a mean decrease in waist circumference of 7.7 cm. In comparison, the placebo group showed a body weight reduction of 1.5% and a decrease in waist circumference of 1.3 cm [12].

Moreover, an analysis conducted by John P.H. Wilding et al. in 2021 showed that treatment with semaglutide 2.4 mg for 68 weeks in participants with overweight or obesity resulted in a mean body weight reduction of −15.3 kg, compared with −2.6 kg in the placebo group. The treatment group also demonstrated significant improvements in waist circumference (−13.54 cm with semaglutide vs. −4.13 cm with placebo), BMI (−5.54 with semaglutide vs. −0.92 with placebo), as well as systolic and diastolic blood pressure. Benefits of semaglutide therapy were also observed in glycated hemoglobin levels, fasting plasma glucose, C-reactive

protein, and fasting lipid levels, constituting an additional component of the favorable metabolic effects of this drug class [13]. An additional factor in both clinical trials was lifestyle modification among participants.

Metabolic control achieved through GLP-1 receptor agonists translates not only into improvement of the individual components of metabolic syndrome but also into a reduction of systemic inflammation. This effect results primarily from the reduction of visceral adipose tissue, which constitutes an immunologically active source of pro-inflammatory cytokines. A decrease in adipocyte (adipose tissue cell) mass limits adipokine release through an increase in adiponectin levels. This hormone exhibits anti-inflammatory properties by lowering the levels of pro-inflammatory cytokines such as TNF, MCP-1, and IL-6, leading to a reduction of the chronic low-grade inflammation characteristic of obesity [14, 15].

Review of clinical trials of GLP-1 receptor agonists in the treatment of psoriasis.
Improvement in Psoriasis Area and Severity Index (PASI)

The efficacy of liraglutide in reducing psoriatic lesions was observed in a randomized controlled trial conducted by Lin et al. in 2020. The study included 24 patients with psoriasis and type 2 diabetes. Of these, 13 completed the 12-week observation period in the control group, and 11 in the group receiving liraglutide. Patients in the control group received acitretin at a dose of 30–50 mg/day, topical calcipotriol, and continued oral hypoglycemic treatment. In the study group, subcutaneous injections of liraglutide were administered, with gradual dose escalation of 0.6 mg weekly up to a maximum of 1.8 mg per day. The authors of the study noted a significant reduction in disease severity in the liraglutide-treated group – the mean PASI (Psoriasis Area and Severity Index) score decreased from 14.02 ±10.67 to 2.40 ±2.71 ($p < .05$), which corresponds to a reduction in psoriatic lesions of approximately 83%. The change in baseline PASI value was significantly greater than in the control group, in which the PASI score decreased from 13.57 ±5.49 to 7.42 ±3.91 ($p < .05$). Clinically significant, high-level responses such as PASI 50 and PASI 75 occurred much more frequently in the liraglutide-treated group. PASI 50 was achieved by 90.91% of patients and PASI 75 by 72.73%, whereas in the control group these values were 38.46% and 7.69%, respectively [23].

Further evidence supporting the beneficial effect of liraglutide on psoriasis severity derives from a prospective cohort study conducted by Ahern et al. The study included seven patients with psoriasis and type 2 diabetes treated with liraglutide for 10 weeks. A statistically significant reduction in disease severity was observed, with the median Psoriasis Area and Severity Index (PASI) decreasing from 4.8 to 3.0 ($p = 0.03$). Importantly, two patients achieved a PASI reduction exceeding 50%. Clinical improvement was accompanied by a significant improvement in quality of life, as assessed by the Dermatology Life Quality Index (DLQI) [24].

In turn, the efficacy of semaglutide in the treatment of psoriasis was evaluated in a randomized, open-label clinical trial involving 31 patients with obesity, type 2 diabetes, and moderate to severe psoriasis. Participants were randomly assigned to a group receiving semaglutide ($n = 15$) or to a control group continuing metformin monotherapy ($n = 16$). Twenty-eight patients completed the study – 13 in the control group and 15 in the semaglutide group. In patients treated with semaglutide at a dose of 1.0 mg/week for 12 weeks, a marked clinical improvement was demonstrated – the median baseline PASI decreased from 21 (IQR = 19.8) to 10 (IQR = 6; $p = 0.002$). In the control group, only slight improvement was observed: the median PASI decreased from 20.6 (IQR = 8.9) to 15.9 (IQR = 8.7; $p = 0.03$), which emphasizes the therapeutic superiority of semaglutide over standard treatment with metformin. In the analysis of high-level clinical response after 12 weeks of therapy, PASI 90 was achieved in 7 of 15 patients (46%), and PASI 100 in 8% of patients treated with semaglutide, whereas in the control group these values were 7% and 0%, respectively [25].

Additional evidence of the efficacy of semaglutide comes from a case report of a 74-year-old man with severe plaque psoriasis and type 2 diabetes. Treatment with semaglutide in combination with metformin led to a marked and sustained clinical improvement, which was reflected by a decrease in the PASI from 33.2 at the start of therapy to 8.0 after 4 months and to 2.6 after 10 months of treatment, corresponding to a total PASI reduction of more than 90%. Importantly, a therapeutic response was observed in a patient in whom no improvement had previously been achieved during biological treatment with adalimumab, which underscores the potential role of semaglutide in the treatment of psoriasis resistant to standard therapeutic methods [26].

Table 1. Characteristics of studies evaluating the effects of GLP-1 receptor agonists on PASI in patients with psoriasis

Author (Year)	Study Design	Intervention	Observation Period	Baseline PASI	Final PASI	Key Clinical Response Parameters
Lin et al. (2020)	Randomized controlled trial	Liraglutide (up to 1.8 mg/day)	12 weeks	*M* = 14.02, *SD* = 10.67	*M* = 2.40, *SD* = 2.71	PASI 75 achieved by 72.73% of patients; $p<.05$
Petković-Dabić et al. (2025)	Randomized controlled trial	Semaglutide (1.0 mg/week)	12 weeks	*Mdn* = 21.0, *IQR* = 19.8	*Mdn* = 10.0, *IQR* = 6	PASI 90 achieved by 46% of patients; $p=.002$
Ahern et al. (2012)	Prospective cohort study	Liraglutide (1.2 mg/day)	10 weeks	*Mdn* = 4.8	3.0	PASI 50 achieved by 29.0% of patients; $p=.03$
Costanzo et al. (2021)	Case report	Semaglutide (up to 1.0 mg/week)	10 months	33.2 (severe psoriasis)	2.6	92.2% reduction in PASI from baseline

Note. *M* = mean; *SD* = standard deviation; *Mdn* = median; *IQR* = interquartile range; PASI = Psoriasis Area and Severity Index.

Effects on Pro-Inflammatory Cytokines (IL-6, TNF-α, IL-17)

An increasing number of randomized clinical trials suggest that GLP-1 receptor agonists may exert a direct effect on immune cells expressing GLP-1 receptors, such as T and B lymphocytes and invariant natural killer T (iNKT) cells. Immature lymphocyte subpopulations, as well as the CD4+CD25+ subset, have also been shown to express GLP-1R transcripts, suggesting a role for GLP-1 signaling in lymphocyte maturation and regulatory T cell (Treg) function [11].

Furthermore, GLP-1R expression has been documented in human neutrophils, eosinophils, and monocytes; however, its expression diminishes following differentiation into macrophages [11]. GLP-1 receptor agonists modulate immune response pathways, leading to a reduction in pro-inflammatory cytokines through inhibition of their secretion by macrophages, including IFN-γ, IL-17, IL-2, TNF-β, IL-6, and IL-1β. This effect is associated with decreased local and systemic inflammation, independently of changes in glycemia and body weight [16, 17, 18].

Among the synthetic analogues of endogenous GLP-1 that are particularly relevant in suppressing macrophage-derived pro-inflammatory cytokine secretion are exenatide and exendin-4. Exenatide has been shown to shift macrophage phenotype toward an anti-inflammatory profile by significantly increasing IL-10 levels while reducing TNF-α and IL-1β in cultured human monocytes/macrophages [19]. Additionally, exendin-4 inhibits lipopolysaccharide-induced expression of inflammatory mediators (iNOS, COX-2, PGE2, and NO) and pro-inflammatory cytokines (TNF-α, IL-1β, and IL-6) in RAW264.7 macrophages, and attenuates cellular production of reactive oxygen species (ROS) [20, 21].

The use of GLP-1 receptor agonists contributes to modulation of the immune system both indirectly—through reduction of obesity-associated inflammation—and directly, via interaction with immune cells and subsequent inhibition of cytokine production. As a result, chronic inflammation may be reduced. This is particularly relevant in inflammatory diseases such as psoriasis, in which suppression of excessive IL-6, TNF-α, and IL-17 production may contribute to alleviation of clinical symptoms and reduction of cutaneous inflammatory activity [22].

Reduction of Inflammatory Markers Under the Influence of GLP-1 Receptor Agonists

In a clinical study conducted by Buysschaert et al. (2014) involving seven patients with psoriasis and type 2 diabetes mellitus, treatment with a glucagon-like peptide-1 (GLP-1) analog demonstrated beneficial effects on the course of both conditions. After 16-20 weeks of GLP-1 therapy, a reduction in γδ T cells was documented in six patients. Administration of the GLP-1 analog improved the clinical status of psoriasis, and this beneficial outcome was associated with a decreased number of γδ T cells in the skin and reduced IL-17 expression [27].

The impact of liraglutide on the clinical course of psoriasis in patients with concomitant type 2 diabetes was documented in a 2022 clinical study by Lu Lin et al. In a group of 24 patients, 12 weeks of liraglutide therapy resulted in a significant reduction in the expression of IL-23, IL-17, and TNF-α. Moreover, IL-23 and IL-17 levels differed significantly between the control and treatment groups. In contrast, the difference in TNF-α expression between the groups was not clinically significant [28].

Furthermore, in patients with psoriasis, type 2 diabetes, and obesity, treatment with GLP-1 receptor agonists led to a reduction in both the number of cutaneous γδ T cells and IL-17 mRNA expression in psoriatic plaques. The effectiveness of GLP-1 receptor agonists is presumably related to improvements in adipose tissue dysfunction, thereby reducing an important source of adipocytokines that may contribute to the development and persistence of psoriasis [29].

In a 2023 clinical report by A.E. Malavazos et al., a 50-year-old Caucasian woman with psoriasis (since 2011), obesity, and type 2 diabetes was treated with semaglutide. The initial dose was 0.25 mg weekly for the first four weeks, followed by 0.5 mg weekly, and after 16 weeks, a maintenance dose of 1 mg weekly was established and continued until the end of the study. After 10 months of therapy, a significant reduction in C-reactive protein levels (-88.4%) and IL-6 levels (-30.6%) compared with baseline values was observed [28].

In another clinical study conducted by Hogan et al. (2011), the effect of liraglutide on invariant natural killer T (iNKT) cells was evaluated in patients with psoriasis. These cells are responsible for the production of proinflammatory cytokines such as IFN-γ and IL-17. Two female patients were included in the study. The first, aged 55, with obesity (BMI 31.3 kg/m²), type 2 diabetes, and psoriasis, was treated with liraglutide at a dose of 1.2 mg weekly. The second, aged 51, with obesity (BMI 34.5 kg/m²), type 2 diabetes, and psoriasis, received 1.8 mg weekly. After six weeks of therapy, the percentage of iNKT cells in psoriatic plaque biopsies decreased from 2.16% to 0.07% of total lymphocytes in the first patient and from 0.32% to 0.00% in the second. The reduction in iNKT cells following liraglutide treatment was associated with decreased levels of proinflammatory cytokines (IFN-γ and IL-17) in both women. These findings suggest an indirect effect of liraglutide in reducing proinflammatory cytokines during GLP-1 therapy [29].

In a 2012 clinical study by Buysschaert et al., the effect of exenatide - one of the GLP-1 receptor agonists - on psoriasis, overweight, and type 2 diabetes was evaluated. The study involved a 61-year-old man with a 30-year history of psoriasis, 14-year history of type 2 diabetes, and concomitant hypertension. His BMI was 25.5 kg/m². At baseline in 2008, his Psoriasis Area and Severity Index (PASI) score was 11 and C-reactive protein (CRP) level was 0.22 mg/dL. Treatment with exenatide was initiated at 5 µg twice daily. After 12 months, in addition to weight reduction and clinical improvement of psoriasis (PASI approximately 3), laboratory tests showed a significant decrease in CRP to 0.03 mg/dL [30].

Weight Reduction as an Indirect Contributing Factor

Adipose tissue in obese patients with psoriasis produces proinflammatory adipokines and promotes systemic inflammation, which plays a central role in both psoriasis and its associated comorbidities [31]. Weight loss reduces adipose tissue mass, thereby indirectly decreasing the activation of inflammatory mediators that exacerbate psoriasis.

In a 2013 clinical study by Jensen et al., the effect of weight reduction on plaque psoriasis was investigated in 60 participants with BMI >27 kg/m² and diagnosed psoriasis. Patients were divided into two groups of 30 each. The intervention group (Low-Energy Diet, LED) underwent a 16-week intensive weight-loss program, while the control group received standard dietary counseling. During the first eight weeks, the LED group followed a low-calorie diet of 800-1000 kcal/day. For the following eight weeks, participants consumed a diet with a maximum energy intake of 1200 kcal/day. Meanwhile, the control group adhered to a standard healthy diet according to national recommendations.

After 16 weeks, body weight and PASI scores were assessed in both groups. The intervention group achieved a mean weight loss of 15.8 kg, compared with 0.4 kg in the control group. The PASI score decreased by an average of 2.3 points in the intervention group and by 0.3 points in the control group. The significant reductions in body weight and PASI in the LED group indicate that treatment of overweight and obesity substantially improves the clinical course of plaque psoriasis [32].

Roongpisuthipong et al. (2013) conducted a study involving 10 patients with plaque psoriasis and obesity (mean BMI >35.2 kg/m²). The mean baseline PASI score was 5.72. Participants followed a very-low-calorie diet for 12 weeks. After this period, mean body fat mass was reduced by 17.9%, and the PASI score decreased by an average of 30.9% [33].

An additional analysis by Castaldo et al. (2019) evaluated the impact of weight reduction on plaque psoriasis in 37 patients with BMI >25 kg/m². During the first four weeks, patients followed a very-low-calorie ketogenic diet (VLCKD) providing no more than 500 kcal per day. For the subsequent six weeks, the diet was changed to a calorie-restricted Mediterranean diet. After 10 weeks, the average weight loss was 10.6 kg, and the PASI score decreased by an average of 10.6 points. The study demonstrated a significant reduction in inflammatory parameters, resulting in marked improvement in psoriasis severity [34].

Risk and challenges

One of the main problems encountered in assessing the role of GLP-1 receptor agonists in the treatment of psoriasis is the lack of large, randomized controlled trials. Due to the small sample size - a maximum of 31 participants - the therapeutic results obtained are unable to reflect the complexity and variability observed in the general population. Inappropriate selection of the study group may also adversely affect the final results. It is necessary to carefully define the study population in terms of BMI, insulin resistance, and the presence of type 2 diabetes, given the varying effects of GLP-1 analogues in these patient groups [35,36].

Another problem is the lack of long-term studies. In an 8-week study, a significant reduction in PASI scores was observed in the liraglutide-treated group. However, ending the observation after such a short period makes it impossible to fully assess the therapeutic effect, especially in the context of the long-term action of GLP-1 analogues[37]. Available studies typically last between 8 and 16 weeks, which significantly limits the ability to draw reliable conclusions and make precise therapeutic recommendations [38,39].

Limitation that we may encounter when analyzing available clinical trials is also the inappropriate selection of participants, resulting from recruitment conducted only in one clinical facility, as well as the lack of appropriate control groups [40,41].

The small number of clinical trials involving large patient populations and characterized by long follow-up periods limits the ability to formulate recommendations for the treatment of psoriasis with GLP-1 drugs. For this reason, it is advisable to conduct further clinical trials that will include larger numbers of subjects and have a significantly longer duration than those mentioned above [42,43].

Conclusions

Glucagon-like peptide-1 receptor agonists exert well-documented metabolic effects, including body weight reduction, decreased waist circumference, and improvement in other metabolic parameters. In addition, they modulate the immune system both indirectly - by reducing the chronic inflammatory state associated with obesity - and directly through their effects on immune cells.

The immunomodulatory properties of GLP-1 receptor agonists, such as liraglutide and semaglutide, are associated with reduced secretion of proinflammatory mediators (IL-17, IL-6, TNF-α) and decreased C-reactive protein levels, which may lead to clinical improvement in patients with psoriasis. These mechanisms suggest a beneficial role of these agents in psoriasis therapy, including alleviation of symptoms and reduction in PASI scores.

However, it should be emphasized that the number of studies evaluating the impact of GLP-1 receptor agonists in this context remains limited. This is due to the small number of available studies, small and heterogeneous study populations, and relatively short follow-up periods. Despite promising short-term outcomes, larger-scale studies with longer follow-up are necessary to establish robust and evidence-based therapeutic recommendations.

Methodology

The above work is a review and was written in December 2025, based on studies published in PubMed between 2010 and 2024. We focused mainly on clinical trials, observational studies and other review papers. They described the effect of GLP-1 agonists on the course of psoriasis, taking into account their metabolic impact. We evaluated the publications in terms of the size of the study groups, the observation period, the concentration of inflammatory markers and changes in the PASI score.

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