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Potential therapeutic use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in patients with psoriasis

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ABSTRACT

Introduction and purpose

Psoriasis is a chronic, inflammatory disease affecting around 125 million people worldwide. While it typically affects the skin, psoriasis is characterized by alteration of adaptive and innate immunity with consequences that extend to other organ systems. Metabolic syndrome, and most notably obesity and type 2 diabetes, are known to have higher prevalence in patients with psoriasis compared to the general population. Recent interest has centered on glucagon-like peptide-1 receptor agonists, which offer potential therapeutic benefits by targeting both metabolic pathways and systemic immune dysregulation in psoriasis. This study gathers and synthesizes preclinical and clinical evidence to evaluate the overall efficacy of GLP-1RAs in the management of psoriasis.

Materials and methods

To write this review a search of databases such as PubMed and Google Scholar was conducted using the following terms: psoriasis; glucagon-like peptide-1 (GLP-1); glucagon-like peptide-1 agonists (GLP-1RAs); liraglutide, semaglutide; obesity, type-2 diabetes, metabolic syndrome.

Results

In clinical and epidemiological evaluations, GLP-1 receptor agonists demonstrated significant dermatologic efficacy in psoriasis patients with concurrent type 2 diabetes mellitus or obesity, achieving marked reductions in Psoriasis Area and Severity Index (PASI) scores, Dermatology Life Quality Index (DLQI) scores, and pro-inflammatory biomarkers (IL-6, CRP), alongside reduced long-term cardiovascular events and all-cause mortality.

Conclusions

GLP-1 receptor agonists offer substantial therapeutic value as targeted, dual-action adjuncts for psoriasis patients with concurrent type 2 diabetes mellitus or obesity by effectively mitigating cutaneous severity, systemic inflammation, and cardiometabolic mortality. However, additional high-value randomized-controlled trials are needed to establish their effectiveness in the treatment of non-obese and non-diabetic psoriasis patients.

Keywords

Psoriasis; Glucagon-like Peptide-1 (GLP-1); Glucagon-like Peptide-1 Receptor Agonists

(GLP-1RAs); Liraglutide, Semaglutide; Obesity, Type-2 diabetes, Metabolic Syndrome;

1. Introduction

Psoriasis is a chronic, inflammatory disease of the skin affecting around 125 million people worldwide [1]. Clinical manifestations of psoriasis are heterogenous, and constitute both cutaneous, as well as systemic symptoms [2]. The most common variant of psoriasis is plaque psoriasis, which corresponds to 90% of all cases. It is distinguished by erythematous, pruritic plaques covered with silvery-white scales located most often on the skin of the trunk, the extensor surfaces of the limbs, and the scalp [2, 3].

Etiopathogenesis of psoriasis is complex and mechanisms of inflammation vary between different variants of psoriasis. There is a strong evidence of genetic relevance in terms of its pathogenesis, as well as signs of autoinflammatory perpetuation in some patients [3]. Psoriasis is marked by chronic, sustained inflammation that leads to uncontrolled keratinocyte proliferation and differentiation, neovascularization, and infiltration of the skin by immune cells such as dendritic cells, macrophages, T cells, and neutrophils [3, 4]. Psoriasis is also associated with dysregulated cytokine expression, with increased production of inflammatory cytokines such as TNF-α, IFN-gamma, IL-6, IL-8, IL-17, IL-23 in the skin or synovial membrane [4, 5].

While typical manifestations of psoriasis involve the skin, the disease is known to affect other sites such as the joints or the nails [1, 2]. Around 40% of psoriasis patients develop psoriatic arthritis [1-3]. Psoriasis has also been linked with extracutaneous and extraarticular comorbidities, such as cardiometabolic diseases, chronic kidney disease, gastrointestinal diseases, malignancy, infections, or mood disorders, as well as an increased risk of myocardial infarction, stroke and adverse cardiovascular outcomes [6–9]. Several large epidemiological studies have shown that the risk of those complications increases with the severity of psoriasis [6, 10], while other studies suggest that managing the complications of psoriasis – such as obesity – may improve the treatment outcomes, as well as quality of life of psoriasis patients [11, 12].

Glucagon-like peptide-1 (GLP-1) is an incretin hormone secreted by intestinal L cells. Glucagon-like peptide-1 works by binding to GLP-1 receptor (GLP-1R), a G protein-coupled receptor, found on numerous cells in the body – most notably pancreatic β-cells, neuronal cells, cardiovascular cells, gastrointestinal smooth muscle cells and immune cells - such as macrophages, lymphocytes or invariant natural killer T cells (iNKT cells) [13, 14, 15]. While

GLP-1 exerts a broad range of effects – some of which include delaying of the gastric emptying, increasing the feeling of satiety, suppressing the appetite, neuroprotection and enhancement of cognitive function, cardiovascular protection and lowering of the blood pressure – its primary role is to maintain blood glucose homeostasis by promoting glucose-dependent insulin secretion and inhibiting glucagon release [14, 15]. Due to these functions, glucagon-like peptide-1 receptor agonists (GLP-1RAs) are a group of pharmaceuticals broadly used in the treatment of type-2 diabetes and obesity. GLP-1RAs, as opposed to natural GLP-1, are resistant to naturally occurring DPP4-mediated degradation. It is a trait that ensures longer bioavailability of those pharmaceuticals [14, 15].

Beyond their metabolic benefits, GLP-1RAs have also been shown to reduce systemic, as well as tissue-specific inflammation through various mechanisms such as inhibition of NF-κB signaling, reduction of pro-inflammatory cytokines, or modulation of immune cell activity [14, 16, 17]. For instance, a meta-analysis has demonstrated a notable reduction in C-reactive protein (CRP) levels following GLP-1RA therapy in individuals with T2DM, as well as reduced secretion of interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha) by peripheral blood mononuclear cells (PBMC) [14]. As chronic, low-grade inflammation is a hallmark of numerous metabolic, cardiovascular, hepatic, dermatological, and neurodegenerative diseases, the anti-inflammatory effects of GLP-1RAs prompted research into their potential usefulness in treatment of patients affected with autoimmune or inflammatory diseases - especially those also suffering from co-existing metabolic disorders [14-17].

2. Aim

The primary aim of this review is to gather, evaluate and summarize current preclinical and clinical evidence regarding the possible benefits of the use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in the treatment of psoriasis. Through evaluation of findings from in vitro studies, animal models as well as human studies, the article outlines GLP-1RAs' role in managing metabolic comorbidities (obesity, type 2 diabetes) associated with psoriasis. Special attention is given to the anti-inflammatory effect associated with GLP-1RAs use, and its possible role in modulating common immune and inflammatory signaling pathways present in psoriatic skin. The multifaceted mechanism of action of GLP-1RAs may serve to target both cutaneous and systemic manifestations of the disease, as well as play a substantial role in improving the overall quality of life of those patients.

3. Materials and methods

To write this review, a search of databases such as PubMed and Google Scholar was conducted using the following keywords: psoriasis; glucagon-like peptide-1 (GLP-1); glucagon-like peptide-1 agonists (GLP-1RAs); liraglutide, semaglutide; obesity, type-2 diabetes, metabolic syndrome. Reviews, randomized controlled trials, cohort studies, as well as in-vitro and animal model studies were prioritized in order provide a broad evaluation of the underlying mechanisms of action of GLP-1RAs in psoriatic skin, as well as their clinical translation in terms of metabolic and overall improvement of psoriasis patients. Only studies written in English were considered. To ensure that the review reflects the current state of knowledge, only articles published between 2011-2026 were evaluated. In total, this review includes 29 primary research articles and supporting literature.

4. Mechanisms of Action of GLP-1 Receptor Agonists in Psoriasis and Preclinical Evidence for GLP-1 Receptor Agonists in Psoriasis

In vitro studies demonstrate that glucagon-like peptide-1 receptor agonists, such as liraglutide and exendin-4, attenuate psoriasis-related cellular processes through distinct intracellular signaling pathways and immune cell modulations.

A primary mechanism of GLP-1RA anti-inflammatory effect in human keratinocytes may involve activation of the adenosine 5'-monophosphate (AMP)-activated protein kinase (AMPK) phosphorylation pathway [17, 18]. In a 2019 study Yang et al. evaluated effects of liraglutide on cultured human keratinocytes (HaCat cells) [18]. The cells were first incubated for 16 hours with lipopolysaccharide (LPS) to induce inflammatory response, and then treated with liraglutide for 30 minutes. Yang et al. found that the protein expression levels of phospho-IKKα/β S176/S180, phospho-NF-κB p65, phospho-JAK2, phospho-STAT3 and SOCS3, as well as TNF-α and IL-6 levels were significantly increased in HaCat cells incubated with LPS compared to the baseline. Liraglutide administration reversed this LPS-induced inflammatory response in HaCat cells [18]. Additionally, to examine the action of AMPK in LPS-induced inflammation in HaCat cells, AMPK inhibitor Compound C (CC) was added, which impaired liraglutide's ability to suppress phospho-NF-κB p65 and phospho-STAT3. This confirmed that liraglutide decreased the LPS-stimulated protein levels of p-NF-κB p65 and p-STAT3 in an AMPK-dependent manner [18]. Beyond signaling cascades, Yang et al. found that liraglutide significantly reduced HaCat cell viability and proliferation, as well as restrained macrophage migration toward keratinocytes [18].

Another proposed mechanism involves C1q/TNF-related protein 3 (CTRP3) mediated inhibition of STAT3 phosphorylation [19]. A 2022 study by Xue et al. outlines that the application of a GLP-1RA, exendin-4, in imiquimod-induced mouse model restored C1q/TNF-related protein 3 expression in keratinocytes, which in turn inhibited STAT3 phosphorylation via lysosomal-associated membrane protein 1 (LAMP1) and alleviated the psoriatic symptoms in both obese, as well as normal-diet mice [19].

Other possible mechanisms of action involve downregulation of major, psoriasis-associated inflammatory cytokines, which include IL-17, IL-22, IL-23 and TNF-α, as demonstrated by Chen et al. in a 2021 study examining the use of liraglutide in obese, diabetic mice with psoriasiform skin [20], as well as GLP-1RA-mediated modulation in activity and distribution of innate immune cells such as invariant natural killer T (iNKT) cells [13].

Together, these findings indicate that GLP-1RAs mitigate psoriatic inflammation by activating AMPK [17, 18], reducing keratinocyte viability [18], dampening macrophage migration [18], restoring CTRP3 expression [19], and inhibiting cytokine secretion by iNKT cells in a dose-dependent manner [13].

5. Clinical Evidence for GLP-1 Receptor Agonists Use in Psoriasis

In clinical populations with concurrent type 2 diabetes mellitus (T2DM) or obesity, GLP-1RAs consistently demonstrate dermatologic and quality-of-life benefits [21–23].

In a randomized-controlled trial Lin et al. evaluated liraglutide at 1.8 mg daily in a cohort of 25 psoriasis patients with T2DM [21]. Over 12 weeks, liraglutide induced a marked improvement in the Dermatology Life Quality Index (DLQI) from 22.00 ± 5.85 to 3.82 ± 3.60 ($p < .05$), which constituted a significant difference compared with the control group ($p < .05$). Lin et al. also reported that the treatment was associated with decreased expression of pro-inflammatory cytokines such as IL-17, IL-23 and TNF-α in the affected skin tissue [21]. Similarly, in an open-label randomized trial, Petković-Dabić et al. investigated semaglutide in obese T2DM patients on background metformin [22]. At 12 weeks, semaglutide significantly reduced median PASI scores from 21.0 (IQR = 19.8) to 10.0 (IQR = 6; $p = 0.002$) and median DLQI from 14.0 (IQR = 5) to 4.0 (IQR = 4; $p = 0.002$). These clinical gains correlated with significant decreases in serum IL-6, C-reactive protein, BMI, and low-density cholesterol (LDL) [22]. Danielle Bar et al. further assessed GLP-1 analogs in obese psoriasis patients with a BMI of 27 or higher, finding that patients treated with GLP-1 analogs achieved an Investigator's Global Assessment

score of 0 or 1 (clear or almost clear) at significantly higher rates than non-treated controls (44.4% versus 12.1%, p <0.001) [23]. Greater weight loss correlated with a faster cutaneous response, and combining GLP-1 therapy with biologics yielded superior clear-skin rates [23].

Despite success in metabolic cohorts, GLP-1RA efficacy appears contingent on baseline metabolic dysfunction. A randomized, placebo-controlled trial by A. Faurschou et al. tested liraglutide over 8 weeks in non-diabetic, glucose-tolerant patients with plaque psoriasis [24]. Although liraglutide produced significant weight loss compared to placebo (4.7 ± 2.5 kg compared to 1.6 ± 2.7 kg) (P = 0.014), it failed to induce significant changes in PASI (-2.6 ± 2.1 compared to -1.3 ± 2.4) (P = 0.228), DLQI (-2.5 ± 4.4 compared to -3.7 ± 4.8) (p = 0.564), or high-sensitivity C-reactive protein [24]. This indicates that GLP-1RA use in glucose-tolerant individuals may be insufficient for rapid psoriatic plaque clearance, however considering overall positive effect of GLP-1RAs on metabolic health and weight loss, further high quality randomized placebo-controlled studies are needed to examine their efficacy in this group of patients.

Beyond cutaneous metrics, real-world comparative evidence highlights substantial systemic benefits for psoriasis patients receiving GLP-1RAs [25, 26]. Olbrich et al. analyzed 3,048 propensity-matched psoriasis patients with T2DM or obesity over two years, finding that GLP-1RA therapy was associated with a 78% reduction in all-cause mortality (hazard ratio (HR) 0.219, 95% confidence interval (CI) 0.123-0.391; p < 0.001) and a 44% reduction in major adverse cardiovascular events (HR 0.561, 95% CI 0.442-0.714; p < 0.001) compared to alternative antidiabetic or antiobesity medications, alongside reduced risks of alcohol and substance abuse [25]. Similarly, Ma et al. confirmed long-term cardioprotection in a propensity-matched cohort of 41,873 GLP-1RA-treated psoriasis patients, demonstrating significantly reduced odds of cerebrovascular disease, heart failure, atherosclerosis and coronary interventions at 5, 10, and 20 years [26].

Conversely, Chiang et al. emulated a target trial comparing GLP-1RA initiation against DPP-4 inhibitors in T2DM patients. While GLP-1RAs were associated with a reduced risk of autoimmune blistering diseases, initiation was unexpectedly linked to a higher relative risk of incident psoriasis (HR 1.19, 95% CI 1.11-1.28) [27].

In conclusion, based on current clinical evidence GLP-1RAs display strong clinical efficacy in resolving psoriatic lesions, improving quality of life, and mitigating cardiometabolic

comorbidities in patients with concurrent T2DM or obesity [21-23, 25, 26]. However additional research is needed in order to determine their effects in non-obese and non-diabetic patients.

6. Discussion

While these collective findings highlight the therapeutic promise of GLP-1RAs, several notable methodological and clinical limitations restrict quality of the evidence.

Firstly, a major limitation is the significant heterogeneity in study designs and patient populations across the literature. Small sample sizes and short follow-up periods - typically ranging from 8 to 12 weeks in prospective clinical trials such as those by Lin et al. [21], Petković-Dabić et al. [22], and Faurschou et al. [24] - hinder the evaluation of long-term dermatologic outcomes, relapse rates, and durable disease remission following treatment discontinuation. Furthermore, open-label designs in several of the small clinical studies introduce potential observer and reporting bias regarding subjective metrics like DLQI and PASI scores [22].

Secondly, substantial evidence demonstrates that weight loss and glycemic control serve as critical adjuncts in mitigating psoriasis severity [11, 12]. Adipose tissue is known to secrete pro-inflammatory cytokines like TNF-α and IL-6 that exacerbate cutaneous lesions [10]. Structured low-energy dietary interventions have been shown to yield significant reductions in body weight, Psoriasis Area and Severity Index (PASI) scores, and Dermatology Life Quality Index (DLQI) metrics [11, 12]. Concurrently, hyperglycemia nearly doubles flare-up risks in severe disease [28], whereas antidiabetic therapies like pioglitazone improve lesion clearance [29]. While both dietary strategies, as well as GLP-1RAs use appear to offer positive results in the treatment of psoriasis patients, head-to-head randomized trials comparing dietary weight-loss interventions to GLP-1RA therapy for reductions in Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index (DLQI) scores are currently lacking. Thus, in studies evaluating obese or diabetic cohorts, such as those by Danielle Bar et al. [23], marked body weight reduction serves as a major confounding variable, making it difficult to isolate direct GLP-1RAs' immuno-modulatory effects from the well-documented anti-inflammatory benefits of fat mass reduction. Additionally, the lack of efficacy of GLP-1RA treatment observed by A. Faurschou et al. [24] in normoglycemic individuals with psoriasis could mean that isolated immunomodulatory effect of GLP-1RAs is insufficient in the treatment of psoriasis patients unburdened by concomitant cardiometabolic diseases. Finally, many patients in these observational studies were receiving concomitant anti-psoriatic treatments, including topical

agents, conventional DMARDs, or biologics, which introduces synergistic or masking effects [23, 25, 26]. Thus, rigorous, randomized-controlled trials isolating non-metabolically impaired cohorts are necessary to clarify whether GLP-1RAs possess independent therapeutic efficacy for cutaneous psoriasis, or if their observed skin improvements depend fundamentally on underlying metabolic restoration.

7. Conclusions

The synthesized evidence highlights GLP-1 receptor agonists as a promising therapeutic strategy at the intersection of metabolic and inflammatory disease management in psoriasis.

In preclinical studies, GLP-1RAs have been shown to suppress key psoriatic pathways—reducing keratinocyte hyperproliferation and downregulating pro-inflammatory cascades (e.g., NF-κB, STAT3, IL-17, TNF-α) via AMPK activation and immune cell modulation. Clinically, these cellular mechanisms translate into marked improvements in cutaneous lesions (PASI) and quality of life (DLQI), alongside significant reductions in all-cause mortality and major adverse cardiovascular events (MACE). However, these therapeutic gains are thus far mostly documented in patients with co-existing type 2 diabetes or obesity.

Ultimately, further high-quality randomized controlled trials are required to define GLP-1RAs' long-term position in therapy of psoriasis, potential synergistic use with other therapies, and efficacy across non-diabetic populations.

Declarations

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