Phytocannabinoids Derived from *Cannabis sativa* in Psoriasis: Effects on Inflammation and Epidermal Hyperproliferation

Fitocanabinoides Derivados de *Cannabis sativa* na Psoríase: Efeitos Sobre Inflamação e Hiperproliferação Epidérmica

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RESUMO

Objetivo: Analisar criticamente os efeitos dos fitocanabinoides derivados de *Cannabis sativa* sobre a inflamação e a hiperproliferação epidérmica na psoríase. **Método:** Realizou-se revisão integrativa da literatura, com buscas nas bases PubMed/MEDLINE e PubMed Central, complementadas por rastreamento manual das referências dos estudos selecionados, abrangendo publicações de 2016 a 2026, com prioridade para o período de 2021 a 2026 e inclusão excepcional de um estudo seminal anterior. Foram selecionados estudos in vitro, pré-clínicos e clínicos relacionados aos mecanismos moleculares, à resposta inflamatória e à homeostase epidérmica. **Resultados:** O canabidiol, composto mais investigado, modula receptores canabinoides e alvos não clássicos, atenua as vias NF-κB e JAK/STAT, reduz mediadores do eixo IL-23/IL-17 e exerce efeitos antiproliferativos, antioxidantes e favoráveis à diferenciação terminal. Contudo, predominam evidências experimentais, enquanto os estudos clínicos permanecem escassos, heterogêneos e demonstram benefícios discretos. **Conclusão:** Os fitocanabinoides apresentam potencial terapêutico, mas sua aplicação clínica requer ensaios controlados, com formulações padronizadas, biomarcadores teciduais e avaliação prolongada da eficácia e da segurança.

DESCRITORES: Psoríase; Canabinoides; Canabidiol; Inflamação; Proliferação de Células.

ABSTRACT

Objective: To critically analyze the effects of *Cannabis sativa*-derived phytocannabinoids on inflammation and epidermal hyperproliferation in psoriasis. **Method:** An integrative literature review was conducted through searches in the PubMed/MEDLINE and PubMed Central databases, complemented by manual tracking of the reference lists of selected studies, covering publications from 2016 to 2026, prioritizing the 2021-2026 period and exceptionally including an earlier seminal study. In vitro, preclinical, and clinical studies addressing molecular mechanisms, inflammatory responses, and epidermal homeostasis were selected. **Results:** Cannabidiol, the most extensively investigated compound, modulates cannabinoid receptors and non-classical targets, attenuates NF-κB and JAK/STAT signaling, reduces IL-23/IL-17 axis mediators, and exerts antiproliferative, antioxidant, and pro-differentiation effects. However, experimental evidence predominates, while clinical studies remain scarce, heterogeneous, and report only modest benefits. **Conclusion:** Phytocannabinoids show therapeutic potential, but their clinical application requires controlled trials using standardized formulations, tissue biomarkers, and long-term evaluation of efficacy and safety.

DESCRIPTORS: Psoriasis; Cannabinoids; Cannabidiol; Inflammation; Cell Proliferation.

RESUMEN

Objetivo: Analizar críticamente los efectos de los fitocannabinoides derivados de Cannabis sativa sobre la inflamación y la hiperproliferación epidérmica en la psoriasis. **Método:** Se realizó una revisión integradora de la literatura mediante búsquedas en las bases PubMed/MEDLINE y PubMed Central, complementadas con rastreo manual de las referencias de los estudios seleccionados, abarcando publicaciones de 2016 a 2026, priorizando el período 2021-2026 e incluyendo excepcionalmente un estudio seminal anterior. Se seleccionaron estudios in vitro, preclínicos y clínicos sobre mecanismos moleculares, respuesta inflamatoria y homeostasis epidérmica. **Resultados:** El cannabidiol, compuesto más investigado, modula receptores cannabinoides y objetivos no clásicos, atenúa las vías NF-κB y JAK/STAT, reduce mediadores del eje IL-23/IL-17 y ejerce efectos antiproliferativos, antioxidantes y promotores de la diferenciación. Sin embargo, predominan las evidencias experimentales, mientras que los estudios clínicos son escasos, heterogéneos y muestran beneficios modestos. **Conclusión:** Los fitocannabinoides presentan potencial terapéutico, pero su aplicación clínica requiere ensayos controlados, con formulaciones estandarizadas, biomarcadores tisulares y evaluación prolongada de la eficacia y la seguridad.

DESCRIPTORES: Psoriasis; Cannabinoides; Cannabidiol; Inflamación; Proliferación Celular.

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INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory disease that primarily affects the skin, whose pathophysiology involves a complex interaction between innate immunity, the adaptive immune response, and keratinocytes. Activation of the interleukin-23/T helper 17 cell axis promotes the production of cytokines such as interleukin-17, interleukin-22, and tumor necrosis factor, perpetuating cell recruitment, inflammation, and epidermal hyperproliferation associated with abnormal keratinocyte differentiation [1,2].

In this context, the cutaneous endocannabinoid system plays a role in regulating the immune response, cell proliferation and differentiation, epidermal barrier integrity, and redox homeostasis. Its components include endocannabinoids, metabolic enzymes, CB1 and CB2 cannabinoid receptors, and non-canonical targets such as GPR55, peroxisome proliferator-activated receptors, and TRP family channels. The ability of phytocannabinoids from *Cannabis sativa* to modulate these targets has spurred research into their use in inflammatory skin diseases [3,4].

Cannabidiol is the most extensively studied compound, with experimental results indicating effects on immune cells, pro-inflammatory cytokines, and signaling pathways such as NF-κB, MAPK, and JAK2/STAT3. In a murine model of psoriasis, its administration reduced inflammation, epidermal thickening, and keratinocyte proliferation [5]. Other phytocannabinoids, including cannabigerol, cannabichromene, cannabinol, and tetrahydrocannabivarin, have also demonstrated anti-inflammatory effects in keratinocytes, although indirect and preclinical studies still predominate [6]. In the clinical setting, modest favorable results with topical CBD contrast with the absence of a significant reduction in disease severity following oral administration, highlighting limitations related to the route of administration, formulation, and small sample size [7,8].

In light of this, this review aims to critically analyze the evidence regarding the effects of *Cannabis sativa* phytocannabinoids on inflammation and epidermal hyperproliferation associated with psoriasis, considering molecular mechanisms, preclinical results, clinical applications, and knowledge gaps.

METHOD

This is an integrative literature review with a qualitative approach and a descriptive-analytical nature, designed to critically synthesize the evidence regarding the effects of phytocannabinoids derived from *Cannabis sativa* on inflammation and epidermal hyperproliferation associated with psoriasis. The literature search was conducted in the PubMed/MEDLINE and PubMed Central databases, supplemented by consulting journal websites and manually reviewing the reference lists of the selected studies.

We used Medical Subject Headings (MeSH) terms and free-text terms corresponding to "Psoriasis," "Cannabis," "Cannabinoids," "Cannabidiol," "Phytocannabinoids," "Keratinocytes," "Inflammation," and "Cell Proliferation." The main search strategy was: ("psoriasis" OR "psoriatic") AND ("Cannabis sativa" OR "phytocannabinoid" OR "cannabinoids" OR "cannabidiol" OR "CBD" OR "cannabigerol" OR "CBG" OR "cannabinol" OR "CBN" OR "cannabichromene" OR "CBC" OR "tetrahydrocannabivarin" OR "THCV") AND ("inflammation" OR "cytokines" OR "keratinocytes" OR "proliferation" OR "epidermal hyperplasia" OR "differentiation").

Articles published between 2016 and 2026 were included, with analytical priority given to the period from 2021 to 2026, in English, Portuguese, or Spanish. Clinical trials, observational studies, and *in vivo*, *ex vivo*, and *in vitro* research were considered, as well as reviews used for theoretical contextualization and identification of primary studies. Studies investigating phytocannabinoids in psoriasis or mechanisms directly related to inflammation, proliferation, and differentiation of keratinocytes were eligible. Due to the scarcity of specific evidence on epidermal hyperproliferation, a 2007 study—considered a seminal mechanistic reference on the topic—was exceptionally included.

Duplicate records, conference abstracts, editorials, non-peer-reviewed publications, studies exclusively on synthetic cannabinoids, and studies not directly related to the investigated outcomes were excluded. Research protocols were considered only to characterize ongoing investigations, without including results. After reviewing titles, abstracts, and full-text articles, the following information was extracted from each study: authorship, year, phytocannabinoid studied, study design, experimental model, formulation, mechanisms, outcomes, and limitations. The findings were organized thematically and subjected to a critical synthesis, without a meta-analysis, due to the methodological heterogeneity among the included studies.

Since this is a literature review based on previously published secondary data, without direct contact with humans or animals, the study did not require submission to a Research Ethics Committee.

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RESULTS

Molecular bases of the action of phytocannabinoids on psoriatic skin

In the skin, the endocannabinoid system constitutes a homeostatic network formed by the endocannabinoids anandamide and 2-arachidonoylglycerol, the CB1 and CB2 cannabinoid receptors, and enzymes involved in synthesis and degradation, such as N-acylphosphatidylethanolamine phospholipase D, fatty acid amide hydrolase, diacylglycerol lipase, and monoacylglycerol lipase. These components are present in keratinocytes, fibroblasts, immune cells, and skin appendages, where they regulate proliferation, differentiation, of the epidermal barrier, redox balance, and the production of inflammatory mediators. Phytocannabinoids from *Cannabis sativa* can modify this network through CB1- and CB2-dependent and -independent mechanisms, providing a basis for their investigation in psoriasis [3,4].

Among these compounds, cannabidiol has low direct affinity for classical cannabinoid receptors and exerts pleiotropic effects through transient-potential receptor channels, GPR55, peroxisome proliferator-activated receptors, and the aryl hydrocarbon receptor, in addition to interfering with endocannabinoid metabolism. In keratinocytes and epidermal equivalents, its activation of the aryl hydrocarbon receptor increased the expression of filaggrin and involucrin and induced the antioxidant mediators NRF2 and NQO1, demonstrating that its effects extend beyond classical cannabinoid signaling. However, this experiment did not use psoriatic cells, constituting indirect mechanistic evidence [9] .

Minor phytocannabinoids also exhibit distinct pharmacological profiles. In HaCaT keratinocytes, cannabigerol, cannabichromene, tetrahydrocannabivarin, and cannabigerolic acid differentially modulated the expression or activity of CB1, CB2, GPR55, TRPV1, PPARs, FAAH, and MAGL, as well as the concentrations of endocannabinoids and related lipids [10] . Subsequently, these compounds reduced various inflammatory mediators in lipopolysaccharide-stimulated keratinocytes, involving the endocannabinoid system and the MAPK pathway; however, there was no uniform suppression of all the cytokines evaluated [6] .

Similar results were observed with cannabinol, which increased CB1 and TRPV1 activity, altered the metabolism of anandamide and 2-arachidonoylglycerol, and reduced IL-8, IL-12, and IL-31, while increasing IL-10. The modulation of IL-31 was found to be TRPV1-dependent, reinforcing the importance of non-canonical targets [11] . In another study, standardized *C. sativa* extract and its lipophilic fractions inhibited NF-κB and IL-8 in keratinocytes exposed to TNF-α; CBD, THC, and CBG were among the most active components. Although the findings are consistent with a combined contribution of the phytocomplex, they do not definitively demonstrate synergy among its constituents [12] .

The cellular origin appears to influence these responses. In keratinocytes from patients with psoriasis, CBD showed greater intracellular accumulation and modified phospholipids, endocannabinoid receptors and mediators, but its effects were not uniformly protective, especially after ultraviolet exposure [13] .

Modulation of the inflammatory response and clinical implications

Psoriatic inflammation results from the persistent interaction between dendritic cells, Th1 and Th17 lymphocytes, neutrophils, and keratinocytes, driven primarily by the IL-23/IL-17 and TNF-α axes. Studies using cells from patients indicate that cannabidiol (CBD) may interfere at different points in this network. In peripheral blood mononuclear cells from individuals with psoriasis, CBD reduced mediators such as TNF-α, IFN-γ, IL-19, and GM-CSF; limited monocyte migration and dendritic cell maturation; decreased CD86 expression; and promoted the differentiation of macrophages with an M2 profile. However, only four patients with psoriasis were evaluated, limiting the external validity of the results [2] .

In another study using patient-derived cells, CBD decreased IL-17 production in CD3+, Th, and NKT populations and reduced IFN-γ in the CD3+ compartment. The same pattern was not observed in cells from healthy controls, in which some IFN-γ-producing populations increased after exposure to CBD. This discrepancy suggests that immunomodulatory activity depends on the baseline inflammatory state and does not correspond to uniform immunosuppression [14] . Additionally, CBD reduced the formation of extracellular traps by psoriatic neutrophils, a process implicated in the amplification of inflammation and the exposure of autoantigens, although the study was limited to an *ex vivo* setting [15] .

In an imiquimod-induced murine model, topical CBD reduced the clinical score, epidermal thickness, and inflammatory infiltrate. At the molecular level, there was inhibition of IL-23R, JAK2, and STAT3, along with decreased expression of *IL-1β*, *IL-6*, *IL-12β*, *IL-17α*, *IL-22*, and *TNF*. This study provides an experimental link between immunomodulation and improvement in the psoriasiform phenotype; however, the small number of animals and the limitations inherent in the model preclude direct extrapolation to patients [5] .

In the clinical evaluation, a double-blind, controlled trial used a *split-body* design in 51 patients, comparing 2.5% CBD ointment with a placebo over 12 weeks. The local psoriasis severity index favored CBD, with a statistically significant difference throughout the follow-up period, particularly regarding scaling; however, the magnitude of the benefit was modest, and a significant

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proportion of the plaques remained unresponsive. Cases of irritation occurred bilaterally, making it difficult to attribute them to CBD[7] . In a randomized, single-blind, crossover study involving 20 participants, topical formulations containing THC and CBD reduced the PASI score; however, the apparent superior performance of the combination with plant extracts cannot be attributed to phytocannabinoids due to the confounding effect of the multi-herbal formulation [16] .

Favorable results were also reported with a CBD-enriched ointment and a broad-spectrum shampoo. However, the first study included only five patients with psoriasis and had no control group, while the second study included participants with scalp psoriasis and seborrheic dermatitis, used multiple ingredients, and evaluated only 14 days of treatment. Therefore, these findings are highly susceptible to confounding factors, the placebo effect, and regression to the mean [17] .

In contrast, a double-blind, placebo-controlled trial involving 28 patients treated with 60 mg/day of oral CBD did not identify a significant reduction in PASI. Only transient improvements in pruritus and time to fall asleep were observed, with no significant differences in the occurrence of adverse events [8] . The discrepancy between the oral and topical studies may reflect differences in bioavailability, vehicle, concentration, clinical severity, and outcomes, and does not demonstrate definitive superiority of one route of administration over the other. The CanPatch protocol aims to evaluate a transdermal patch composed predominantly of CBD; however, as it has not yet produced results, it should be cited only as ongoing research [18].

Epidermal hyperproliferation, differentiation, and homeostasis

In psoriasis, the persistent interaction between inflammatory cytokines and keratinocytes shortens the epidermal renewal cycle, increases proliferation in the basal and suprabasal layers, and disrupts terminal differentiation. As a result, acanthosis, parakeratosis, and aberrant expression of keratins associated with the hyperproliferative state are observed. Therefore, the restoration of homeostasis depends not only on the reduction of cell division but also on the recovery of differentiation, barrier function, and the balance between cell survival and cell death [19] .

The most frequently cited direct evidence regarding phytocannabinoids and keratinocyte proliferation remains the study [20] , included in this review as a historical exception to the time frame.

In human hyperproliferative keratinocytes immortalized by HPV-16, Δ^9-tetrahydrocannabinol (THC), cannabidiol (CBD), cannabinol (CBN), and cannabigerol (CBG) inhibited proliferation in a concentration-dependent manner, with IC_{50} values ranging from approximately 2.0 to 2.9 μM. The cell integrity assays employed by the authors indicated that the effect was not simply due to cytotoxicity. Furthermore, CB1, CB2, and TRPV1 antagonists did not reverse the inhibition, suggesting a predominance of mechanisms not mediated by classical cannabinoid receptors [20] . More recent findings confirm that the antiproliferative effect is concentration- and experimental model-dependent. In a supplementary analysis conducted with HaCaT keratinocytes, CBD reduced BrdU incorporation only at concentrations above 15 μM, with an IC_{50} close to 20 μM; lower concentrations did not significantly alter DNA synthesis [2] .

In the murine model of imiquimod-induced psoriasis, topical application of 0.01% or 0.1% CBD reduced epidermal thickness, keratosis, and hyperproliferation, alongside improvements in PASI-like scores. These effects were associated with the inhibition of IL-23R/JAK2/STAT3 signaling and a reduction in the expression of mediators such as *Il1b*, *Il6*, *Il17a*, *Il22,* and *Tnf*. Since STAT3 is involved in both the cytokine response and the proliferation and survival of keratinocytes, its inhibition provides a mechanistic link between the control of inflammation and the normalization of epidermal architecture [5] .

In addition to limiting proliferation, CBD may promote terminal differentiation. In human keratinocytes and three-dimensional epidermal models, one of the studies reviewed [9] demonstrated that CBD activated the aryl hydrocarbon receptor (AhR), increased the expression of filaggrin and involucrin, and elevated the OVOL1/OVOL2 transcriptional ratio. NRF2, NQO1, CYP1A1, and the AhR repressor were also induced, while the epidermal models exhibited a more pronounced granular layer. Pharmacological blockade or silencing of AhR attenuated these effects, supporting the involvement of the AhR–OVOL1–filaggrin and AhR–NRF2–NQO1 pathways. These findings suggest that CBD may act as a pro-differentiation and antioxidant modulator, and not merely as a cytostatic agent. However, the experiments did not use psoriatic keratinocytes or equivalents exposed to the IL-17/IL-22/TNF-α microenvironment, making it impossible to assert that the same response occurs in psoriatic plaques [9] .

Studies using primary keratinocytes from patients add a metabolic dimension to epidermal homeostasis. CBD modified phosphatidylcholines, phosphatidylserines, ether-linked phosphoethanolamines, and sphingomyelins in UVB-irradiated psoriatic keratinocytes, producing changes related to membrane organization and, under certain conditions, to pro-apoptotic signaling [21] . In another study, its effects on phospholipids, lipid peroxidation, and surface charge differed between healthy and psoriatic cells and also depending on exposure to UVA or UVB. Although some parameters approached the values observed in control cells, other changes were more pronounced in patients'

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cells [22] . Additionally, CBD reduced metalloproteinase activity and partially normalized the MMP/TIMP ratio and the expression of angiogenic factors in psoriatic keratinocytes irradiated with UVB [23] .

The formulation also influences epidermal exposure. In mice, lipid nanoparticles containing CBD reduced the PASI score more significantly than the free CBD emulsion, nanoparticles without CBD, and the untreated group. Nanoparticles labeled with coumarin-6 showed localized penetration of approximately 100 µm in rat skin [24] . However, the distribution of the fluorescent marker does not necessarily correspond to the cutaneous pharmacokinetics of CBD, and the lipids in the formulation themselves may modify the skin barrier and the inflammatory response.

In humans, the correlation between clinical improvement and epidermal normalization remains indirect. A *split-body* trial involving 51 participants and 108 pairs of patches found a small reduction in the local severity score after 12 weeks of treatment with a 2.5% CBD ointment, primarily in the scaling component [7] . In contrast, a trial involving 28 participants did not observe a significant reduction in PASI with oral CBD compared to placebo [8] .

DISCUSSION

Taken together, the reviewed findings support the biological plausibility of phytocannabinoids, particularly cannabidiol, as modulators of key processes in psoriasis. The convergence of mechanisms dependent on and independent of CB1 and CB2 receptors—including GPR55, peroxisome proliferator-activated receptors, TRP channels, and the aryl hydrocarbon receptor—suggests that the potential therapeutic effect is not limited to classical cannabinoid signaling but involves a broader regulatory network already implicated in skin homeostasis [3,4,9,10,12,15] .

From an inflammatory perspective, the reduction in mediators of the IL-23/IL-17 axis and the inhibition of the NF-κB and JAK2/STAT3 pathways observed in different models align with the main therapeutic targets already validated for psoriasis, which reinforces the mechanistic consistency of the findings [1,2,5,14] . However, the immunomodulatory response was not uniform: the effect was predominant in cells from patients with psoriasis and was not reproduced—or was even reversed—in cells from healthy individuals, suggesting that the action of CBD depends on the tissue's baseline inflammatory state, rather than on nonspecific immune suppression [14] . This context-dependence has important implications for extrapolating findings obtained from non-psoriatic cell lines.

In the epidermal compartment, the described antiproliferative and pro-differentiation effects reinforce the hypothesis that phytocannabinoids act on two pillars of psoriasis pathophysiology—excessive proliferation and abnormal differentiation—and not merely on the immune response. However, this evidence depends heavily on the concentration used, the cell line, and the presence or absence of concomitant inflammatory stimulation, which makes it difficult to define a safe and effective therapeutic window [9,20,21,22,23].

From a clinical perspective, the contrast between the results obtained with topical and oral CBD is one of the most relevant findings of this review. While the topical formulation was associated with a statistically significant, albeit modest, benefit regarding scaling, the oral route did not demonstrate a reduction in PASI, although it did improve secondary outcomes such as pruritus and sleep [7,8] . This discrepancy likely reflects differences in pharmacokinetics and bioavailability between the routes of administration, in addition to the high methodological heterogeneity among the available clinical studies: small sample sizes, the absence of a control group in some study designs, polyherbal formulations with confounding effects, and short follow-up periods [6,16,17,18] .

These methodological limitations, coupled with the predominance of *in vitro* and preclinical evidence over clinical studies, restrict the external validity of the findings and preclude, at this stage, any recommendation for the routine use of phytocannabinoids in the treatment of psoriasis. Furthermore, minor compounds such as cannabigerol, cannabichromene, cannabinol, and tetrahydrocannabivarin remain substantially less studied than cannabidiol, representing a significant gap for future research [6,11] . The absence of standardized formulations, validated tissue biomarkers, and consistent pharmacokinetic data therefore represents a major obstacle to the clinical translation of this evidence.

CONCLUSION

The findings of this review indicate that phytocannabinoids derived from *Cannabis sativa*, especially cannabidiol, have biological plausibility for modulating key processes in the pathophysiology of psoriasis, acting on both the inflammatory response and the hyperproliferation and abnormal differentiation of keratinocytes.

However, this evidence comes primarily from *in vitro* studies and animal models, and the few available clinical trials show modest and inconsistent benefits between the topical and oral routes. Thus, phytocannabinoids are promising candidates for further research, but the current evidence does not support their routine incorporation into the treatment of psoriasis.

It is recommended that larger clinical trials be conducted, using standardized formulations, tissue biomarkers, pharmacokinetic assessment, and long-term follow-up, capable of establishing with greater certainty the efficacy, safety, and therapeutic applicability of this class of compounds.

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