Available online on 15.8.2025 at http://ajprd.com

Asian Journal of Pharmaceutical Research and Development

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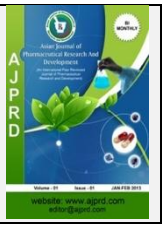

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Review Article

Allicin-Based Transethosomal Gel: A Novel Herbal Approach for Psoriasis Treatment

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A B S T R A C T

Allicin, Garlic is the source of allicin, a bioactive organosulfur molecule that has shown strong antibacterial, anti-inflammatory, and immunomodulatory properties, making it a possible topical treatment for psoriasis. However, because of its limited skin penetration and poor aqueous stability, its therapeutic value is limited. Highly flexible lipid vesicles called transethosomes, which are made of phospholipids, ethanol, and edge activators like sodium cholate, have become sophisticated nanocarrier systems that can improve the transport of drugs through the skin.In this review, the development and usage of transethosomal gels loaded with allicin as a new psoriasis treatment strategy is discussed. The article discusses important formulation parameters, physicochemical characteristics, preparation methods, and advantages over traditional delivery methods. Transethosomal systems have been shown to improve drug entrapment, extend release profiles, increase skin permeability, and improve therapeutic effects throughout the body of current literature. These characteristics highlight how transethosomal gels laden with allicin may help develop herbal-based topical treatments for inflammatory skin conditions like psoriasis.

Key words: Allicin, Transethosomal gel, Psoriasis, Topical drug delivery, Herbal nanocarriers, Sustained release, Skin permeation.

A R T I C L E I N F O: Received 02 Feb 2025; Review Complete 18 April 2025; Accepted 12 June 2025. ; Available online15 August. 2025

Cite this article as:

Kothawade YK, Sonawane D, Sharma Y, Allicin-Based Transethosomal Gel: A Novel Herbal Approach for Psoriasis Treatment, Asian Journal of Pharmaceutical Research and Development. 2025; 13(4):137-144, DOI: http://dx.doi.org/10.22270/ajprd.v13i4.1604

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INTRODUCTION:

Psoriasis is an inflammatory skin condition that is immune-mediated, chronic, and recurrent. It has a major negative impact on sufferers' quality of life.[1] It is mainly identified by erythematous, scaly, and clearly defined plaques that typically develop on the lower back, elbows, knees, and scalp.[2]When the normal 28-day skin cycle is shortened to just 3–7 days, the illness results from an accelerated turnover of keratinocytes.[3]As a result, thick, silvery scales are formed on the surface from the aggregation of immature skin cells.Psoriasis is increasingly understood to be a systemic autoimmune disease with intricate immunological dysregulation, rather than only a skin problem[4] Inflammatory cytokines such as TNF-α, IL-17, and IL-23 are released in excess when dendritic cells and T-helper cells (especially Th1 and Th17) are overactivated, which feeds the cycle of inflammation and skin growth.[5]Disease start and flare-ups are largely influenced by environmental triggers such infections, skin trauma, stress, and specific drugs, as well as genetic predispositions like the HLA-Cw6 allele.[6]The pathophysiology of psoriasis has also been linked to microbial flora, according to recent research.Exacerbations of guttate and scalp psoriasis have been linked to bacterial agents such as Streptococcus pyogenes and Staphylococcus aureus, as well as fungal species like Malassezia.[7,8] Although psoriasis can afflict people of any age, it often manifests between the ages of 15 and 35. Numerous comorbidities, such as psoriatic arthritis, metabolic syndrome, cardiovascular diseases, and psychological stress, are linked to the disease.[9] It is a disease that has significant emotional and psychological effects in addition to medical ones, as the lesions appearance frequently causes social shame, anxiety, and sadness.Effective long-term care of psoriasis is still difficult because it is a chronic and recurrent condition.[10] Novel drug delivery methods, such transethosomal gels, have drawn interest recently due to their potential to increase drug bioavailability,decrease systemic adverse effects, and improve skin penetration all of which

present encouraging prospects for topical psoriasis treatment.[11]

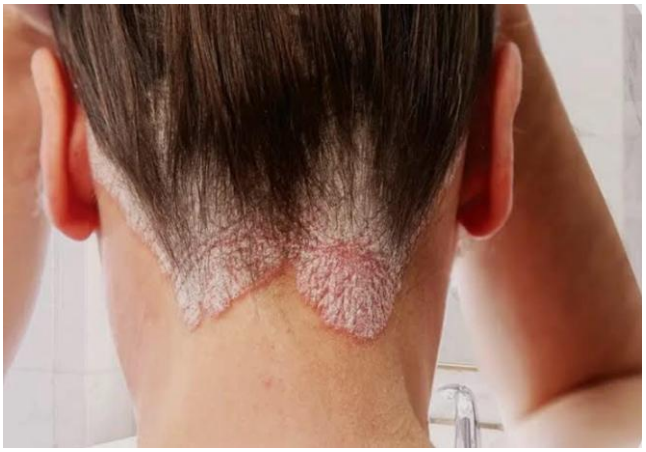

Figure 1: Psoriasis

Etiological Agent:

Psoriasis is not caused by a single infectious agent but can be triggered or exacerbated by certain microbial infections. Among these, **Streptococcus pyogenes** is strongly associated with guttate psoriasis, especially following throat infections.[12] Similarly, **Staphylococcus aureus**, commonly found on the skin, may contribute to disease flares by promoting local inflammation and immune activation. These bacteria act as triggering factors, especially in genetically predisposed individuals, by activating immune pathways that lead to psoriatic plaque formation.[13]

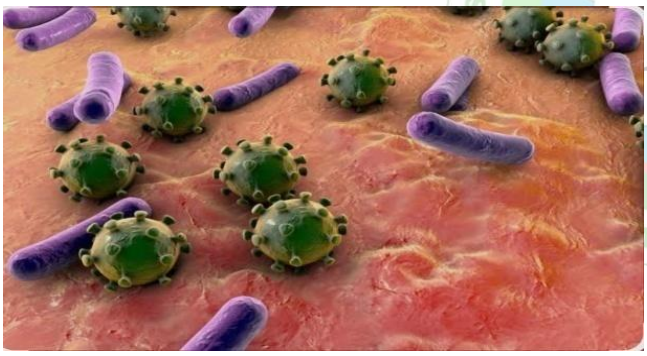

Figure 2*: Streptococcus pyogenes*

Pathophysiology of Psoriasis:

The pathophysiology psoriasis is a chronic inflammatory skin condition that is mediated by the immune system and involves a mix of environmental stimuli and genetic predisposition.[14]Genes that raise the likelihood of aberrant immunological responses, including those in the IL-23/Th17 signaling pathway and HLA-Cw6, are strongly associated with the illness. Dendritic cells in the skin are activated by environmental variables such as infections, skin injuries, and emotional stress. These cells then excite T-helper cells, specifically Th17, Th1, and Th22.[15] These immune cells cause inflammation and aberrant keratinocyte proliferation by releasing pro-inflammatory cytokines such as interleukin-17 (IL-17), IL-22, and tumor necrosis factor-alpha (TNF-α).The epidermal turnover time is shortened from the typical 28–30 days to only 3–5 days due to this immunological activity. Immature keratinocytes thus build up on the surface to create thick, erythematous plaques that are covered with silvery-white scales.[16] Furthermore, elevated vascular endothelial growth factor (VEGF) stimulates dermal angiogenesis, resulting in the Auspitz sign, which is characterized by redness and pinpoint hemorrhage upon scale removal.[17] Guttate psoriasis is known to be triggered by Streptococcus pyogenes, particularly in younger people, and changes in the skin and gut microbiota may exacerbate the disease's symptoms.[18] A persistent inflammatory cycle between immune cells and keratinocytes maintains the chronic nature of psoriasis and has emerged as a key target for biologic treatments like IL-17 and IL-23 inhibitors.[19]

Global Epidemiology and Burden of Psoriasis:

Globally, psoriasis affects roughly 2-3% of people, with prevalence and severity varying by area. [20]Although prevalence rates are lower in some regions of Asia and Africa, they are higher in high-income nations, especially in Europe and North America, where they can reach as high as 4.5%. Although it can strike at any age, the disease usually manifests in two stages: early onset (15–25 years) and late onset (50–60 years). It affects both genders equally.The effects of psoriasis are not limited to the skin.[29]In addition to the higher risks of depression, type 2 diabetes, metabolic syndrome, and cardiovascular disease, patients frequently develop psoriatic arthritis (30% of cases).[30] In addition to raising healthcare expenses, these comorbidities have a major negative influence on quality of life. Psoriasis has significant physical, psychological, and social ramifications and is recognized by the World Health Organization (WHO) as a major non-communicable illness.[31] Inadequate access to efficient treatment exacerbates the worldwide disease load, particularly in low- and middle-income nations.[32]

Global Control Programs and Future Directions in Psoriasis Management:

Psoriasis is now globally recognized as a chronic, non-communicable, and immune-mediated disease with substantial physical, emotional, and social impact.[33] In **2016**, the **World Health Organization (WHO)** released the Global Report on Psoriasis, emphasizing the need for improved awareness, timely diagnosis, access to treatment, and the integration of psoriasis care into national health policies.[34] This landmark report urged member countries to reduce stigma, enhance education, and ensure universal healthcare support for psoriasis patients.Globally, organizations such as the National Psoriasis Foundation (USA), International Federation of Psoriasis Associations (IFPA), and various dermatological societies have initiated community-based support programs, clinical research collaborations, and awareness campaigns.[35,36] Some countries, especially in Europe and North America, have integrated psoriasis management within their universal healthcare frameworks, offering biologics and advanced therapies as part of standard treatment regimens.[37]

Future Directions:

- **Advanced Topical Delivery Systems**: Incorporation of vesicular drug carriers like transethosomes, ethosomes, and liposomes is gaining attention for improving dermal penetration and drug retention, especially for phytoconstituents like Allicin.[38]

- **Biologics and Immunotherapy**: Monoclonal antibodies targeting specific cytokines (e.g., IL-17, IL-23, TNF-α) are increasingly used for moderate to severe cases.[39]
- **Personalized Medicine**: Genetic profiling and patient-specific treatment plans are being explored to enhance therapeutic outcomes.[40]
- **Plant-based Therapeutics**: Research into natural compounds such as garlic-derived Allicin shows potential in reducing inflammation and improving skin regeneration with minimal side effects.[41]
- **Digital Health and Teledermatology**: Future programs are expected to integrate telemedicine for early diagnosis and remote management of psoriasis, particularly in underserved areas.[42]
- **Policy and Education**: Expansion of educational campaigns to reduce stigma, promote early intervention, and empower patients through awareness is a key global goal.[43]

Diagnostic Approches for Psoriasis:

Psoriasis is mainly diagnosed clinically by observing well-defined, red plaques with silvery-white scales, commonly on the scalp, elbows, and knees. Key signs include the Auspitz sign and Koebner phenomenon.[44] A thorough medical history, including triggers and family history, supports the diagnosis. In unclear cases, a skin biopsy confirms features like parakeratosis and acanthosis. Tools like the Psoriasis Area and Severity Index (PASI) help assess disease severity.[45] Dermatoscopy aids in differentiating psoriasis from similar conditions, and imaging may be used in cases with suspected psoriatic arthritis. Blood tests are rarely needed but may help rule out other conditions or assess systemic involvement.[46]

Introduction of Allicin:

Garlic (***Allium sativum***) has a sulfur-containing bioactive component called **allicin,** which has antibacterial, anti-inflammatory, and antioxidant qualities.[47]Alliin is transformed into allicin by the enzyme alliinase, which is released when garlic is crushed. This substance is beneficial for both internal and external infections since it works against a variety of microorganisms, such as Staphylococcus aureus, E. coli, Candida albicans, and even viruses like influenza.[48]The anti-inflammatory properties of allicin are especially significant in psoriasis. By blocking pro-inflammatory cytokines like TNF-α, IL-6, and IL-1β, which are essential to the pathophysiology of psoriasis, it alters immune responses.

Its antioxidant qualities also aid in lowering oxidative stress, which is a major contributor to skin damage and inflammation. [49] Allicin's instability and low bioavailability, which cause it to break down rapidly in the presence of heat, air, or digestion, restrict its clinical application.[50]Advanced drug delivery methods like liposomes and transethosomes are being investigated in an effort to get around these restrictions.Particularly when applied topically to inflammatory skin conditions, these systems aid in stabilizing allicin and improving its absorption.

Allicin's combined antibacterial, anti-inflammatory, and antioxidant properties make it a promising natural medicinal agent for the treatment of psoriasis.[51] For chronic inflammatory skin disorders, developing reliable and bioavailable allicin delivery systems could offer a useful substitute for traditional treatments.[52]

Figure 3: Garlic (***Allium sativum***)

- **Botanical Name**: Allium sativum L.
- **Family**: Amaryllidaceae (formerly Liliaceae)
- **Part Used**: Bulb (clove)
- **Major Active Compound**: Allicin (formed from Alliin by enzyme Alliinase upon crushing garlic).
- **Phytochemical Class**: Organosulfur compound (Thiosulfinate group).
- **Physical Nature**: Colorless,oily liquid with pungent odor.[53,54]

Therapeutic potential for Allicin:

Allium sativum, or garlic, has a sulfur-rich substance called allicin, which has antibacterial, anti-inflammatory, and antioxidant qualities.[55] It helps treat psoriasis because it inhibits pro-inflammatory cytokines like TNF-α, IL-6, and IL-1β. Its limited bioavailability and volatility, however, restrict its application. Its stability, penetration, and therapeutic efficacy for topical application in skin conditions are improved by recent developments such as transethosomal gels.[56]

IntroductionTransethosomal Gel:

Transethosomes are a sophisticated family of lipid-based nanocarriers that are particularly helpful for topical medication delivery because they enhance the absorption and transport of active pharmaceutical ingredients (APIs) through the skin.[57]The increasing need for more effective and efficient methods of delivering bioactive substances, including antibiotics, antifungal agents, and anti-inflammatory medications, across the epidermal barrier has led to the development of these novel delivery systems. Transethosomes are elastic vesicular structures that improve the skin penetration of hydrophilic and lipophilic compounds by combining the advantageous qualities of liposomes, ethanol, and surfactants.[58]

The skin's protective barrier, especially the stratum corneum (the outermost layer of the skin), which acts as a natural defensive mechanism against the penetration of exogenous substances, presents the fundamental issue of topical medication administration. Many medicinal substances cannot pass through this barrier, which restricts their ability to reach the deeper layers of the skin where they are most required. [59]Conventional liposomes and creams, among other drug delivery methods, frequently fall short of this barrier. With their distinct composition and structure, transethosomes provide a way to overcome this difficulty by increasing the

skin's permeability, which enables the active substances to reach deeper layers of the skin and start working therapeutically.[60]

Structure& combination of Transethosome:

1. **Phospholipids:** Form the vesicle bilayer help encapsulate the drug and maintain stability.
2. **Ethanol**: Enhances skin penetration by disrupting the stratum corneum.
3. **Surfactants**: Stabilize vesicles, reduce surface tension, improve skin adhesion, and control drug release.[61,62]

Mechanism of Action:

Transethosomes enhance skin drug delivery through a combination of flexible lipid bilayers, ethanol, and surfactants.

1. **Lipid Composition & Elasticity**: Phospholipids provide elasticity, allowing vesicles to deform and penetrate the stratum corneum, reaching deeper skin layers.[63]
2. **Ethanol**: Acts as a penetration enhancer by disrupting skin lipids, improving permeability and solubilizing hydrophobic drugs.
3. **Surfactants**: Stabilize vesicles, reduce surface tension, and support better skin adhesion and drug delivery.
4. **Controlled Release**: Once inside the skin, transethosomes provide sustained drug release, enhancing therapeutic efficacy and reducing dosing frequency.[64,65]

A new class of lipid-based vesicular system called a transethosome was created to improve the way active pharmaceutical ingredients (APIs) are delivered through the skin. A special structure that combines the flexibility of liposomes with the penetration-enhancing qualities of ethanol and surfactants is the basis of their mode of action.[66]These elements give transethosomes the ability to efficiently penetrate deeper skin layers and go past the stratum corneum, the skin's outermost barrier. Transethosomes are a promising treatment option for a number of dermatological diseases because they enable more effective drug delivery. Transethosomes' mode of action is covered in this section, along with the benefits that make them a good choice for transdermal medication delivery.[67]

Advantages of Transethosomes:

1. **Enhanced Skin Penetration**: Ethanol and surfactants improve permeability through the stratum corneum.
2. **High Drug Loading**: Suitable for both hydrophilic and lipophilic drugs.
3. **Improved Drug Stability**: Protects active ingredients from degradation.
4. **Sustained and Controlled Release**: Prolongs therapeutic effect and reduces dosing frequency.
5. **Non-Invasive Delivery**: Offers a painless alternative to injections.
6. **Flexible Vesicles**: Can deform and pass through narrow skin pores and tight junctions.
7. **Reduced Side Effects**: Targeted delivery minimizes systemic exposure.
8. **Versatile Application**: Useful in treating various skin diseases like psoriasis, eczema, and fungal infections.[68,69]

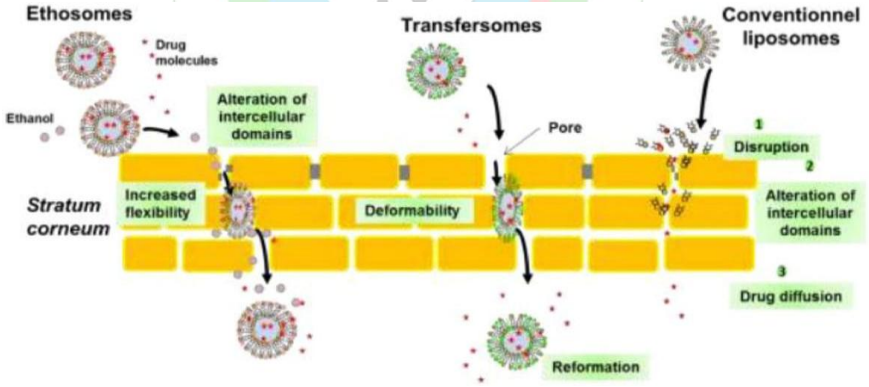

Figure 4: Mechanism of Transethosomes

Materials Used for Preparation of Allicin-Loaded Transethosomal Gel:

1. **Phospholipid 90G (Lipoid):** Serves as the primary lipid constituent that forms the transethosome bilayer. Improves allicin stability and encapsulation
2. **Sodium Cholate**: Enhances vesicle flexibility and skin penetration by acting as an edge activator (surfactant).
3. **Allicin**: The potent natural ingredient that has antioxidant, antibacterial, and anti-inflammatory qualities. useful for treating psoriasis.
4. **Carbopol 934**: Transethosomal solution is transformed into a gel for simple topical application using this gelling agent.
5. **Polyethylene Glycol (PEG)**: Increases formulation spreadability and hydrates skin by acting as a humectant and solubilizer.
6. **Triethanolamine**: Used to balance the pH of the gel composition and neutralize carbopol.
7. **Ethanol**: A penetration enhancer that improves medication delivery through the skin by upsetting the stratum corneum.
8. **Distilled water**: Used as a carrier and solvent in the manufacture of gel and transethosome.[70,71]

Preparation of Transethosomes:

Transethosomes were prepared by the **cold technique**. To guarantee that the lipid component was completely dissolved, the necessary quantity of Phospholipid 90G was first dissolved in ethanol. Sodium cholate was added to this lipid

solution as a surfactant to help transethosomal vesicles develop. In order to accomplish successful medication encapsulation within the vesicles, the active ingredient, allicin, was then added to the mixture. After the medicine was added, the liquid was gently stirred to achieve homogeneity.The lipid mixture was then combined with an aqueous phase while being continuously stirred to complete the hydration process, which encouraged the development of the vesicular system. To improve the homogeneity of the transethosomal formulation and decrease vesicle size, the resultant suspension was sonicated.

A sealed container was used to hold the prepared transethosomal suspension after it had cooled. After the formulation's stability, entrapment effectiveness, and vesicle size were further assessed, it was added to a gel base for topical administration. Lastly, the formulation's effectiveness as a treatment, drug release, and skin penetration were assessed.[72]

Evaluation Parameters of Allicin-Loaded Transethosomal Gel:

Physical Appearance and Homogeneity: The gel's color, clarity, texture, phase separation, and air bubbles were all visually inspected. After stirring, samples were taken from various locations to evaluate homogeneity by looking for clumps and smoothness. Three observations (n=3) were made. [73]

Viscosity: A Brookfield viscometer (Ametek, USA) was used to measure the gel's viscosity at $25 \pm 1°C$. The rheological behavior of the gel was evaluated at several rotating rates after choosing an appropriate spindle. To guarantee consistency, the results were measured in triplicate (n=3) and recorded in centipoises (cP).[74]

pH Measurement: A calibrated digital pH meter (Mettler Toledo, India) was used to measure the pH. Ten milliliters of distilled water were used to dissolve a part of the gel, which was then leftto equilibrate for thirty minutes at 25 ± 1°C. Each measurement of the stabilized pH was made three times (n=3).[75]

Spreadability: To evaluate spreadability, a 50 g weight was placed on the top slide and 1 g of gel was sandwiched between two glass slides. The spread's diameter was measured in millimeters after a minute. To ensure consistency and repeatability, this process was carried out three times.

Drug Content Uniformity: To analyze the drug content, 1 g of gel was dissolved in 100 mL of ethanol while being constantly stirred, and then the mixture was filtered. A UV-Visible spectrophotometer (Shimadzu UV 1900, Japan) was used to evaluate the filtrate at 224 nm, and a standard calibration curve was used to determine the allicin content. Three separate measurements were made.[76]

In-vitro Drug Release Study: A Franz diffusion cell apparatus fitted with a synthetic cellulose membrane (molecular weight cutoff 12–14 kDa) placed between the donor and receptor compartments was used to assess the in-vitro release profile of the allicin-loaded transethosomal gel. While the receptor chamber was filled with phosphate-buffered saline (PBS, pH 7.4) containing 1% sodium amide, a 1 g sample of the gel was evenly put to the donor chamber.With constant stirring at 50 RPM, the system was kept at $32 \pm 0.5°C$. To maintain sink conditions, 1 mL aliquots were taken out of the receptor compartment at predefined intervals (1, 2, 4, 6, 8, 10, and 12 hours) and promptly replaced with an equivalent volume of fresh PBS. The amount of allicin emitted was measured at 224 nm using a UV-Visible spectrophotometer. To guarantee data repeatability, each experiment was carried out in triplicate (n=3).[77]

Accelerated Stability Testing: The transethosomal gel filled with allicin underwent three months of accelerated stability testing at $40 \pm 2°C$ and 75% relative humidity. Physical appearance (color, clarity, and texture), pH, viscosity, spreadability, drug content (measured using a UV spectrophotometer set at 224 nm), and in-vitro drug release were all evaluated at one, two, and three months. To guarantee dependability, each test was run three times.78

Transethosomal Gel Loaded with Allicin for the Treatment of Psoriasis:

Psoriasis is a long-term autoimmune skin condition characterized by scaly plaques, inflammation, and keratinocyte hyperproliferation. Traditional treatments frequently only offer short-term respite and can cause negative side effects over time. Transethosomal systems have emerged as viable drug delivery vehicles as a result of recent developments in nanotechnology because of their improved stability and skin penetration.

Garlic contains a bioactive substance called allicin, which has strong anti-inflammatory, antibacterial, and antioxidant qualities. However, its therapeutic utility is limited by its instability and poor skin retention. Transethosomal gel's stability, bioavailability, and targeted distribution to psoriatic lesions are all improved by the addition of allicin. By lowering inflammation, stopping bacterial invasion, and enhancing general skin health, this innovative combination shows promise for managing psoriasis effectively.

Recent Patents and Clinical Investigations:

Recent years have seen growing interest in allicin-based nanocarriers for psoriasis therapy. A U.S. patent (US20200234789A1) highlights the use of garlic-derived allicin in nanoliposomes, showing enhanced dermal delivery and anti-inflammatory action. [108]Additionally, a clinical study by Lin et al. (2023) evaluated a topical allicin-loaded nanogel in mild-to-moderate plaque psoriasis, reporting significant reductions in erythema, scaling, and itching within 21 days, with no irritation or toxicity.[109]

Transethosomal systems offer added advantages over conventional gels by enhancing skin retention, stability, and targeted delivery. Recent research confirms that phospholipid vesicles with ethanol and edge activators improve permeation of unstable phytoconstituents like allicin, making the formulation more effective . These developments support the clinical relevance and future potential of allicin-loaded transethosomal gels as a safe, herbal alternative to corticosteroids.[110]

Comparing Transethosomal Gel with Topical Gels:

Table 1: Comparison of Transethosomal Gel with other Topical Gel

Parameter	**Conventional Gel**	**Herbal Gel**	**Allicin-Loaded Transethosomal Gel**
Key Ingredient	Synthetic drug (e.g., corticosteroid)	Plant extracts (e.g., aloe vera, turmeric)	Garlic-derived Allicin
Penetration Efficiency	Low to moderate	Moderate	High (due to ethanol and edge activator)
Drug Stability	Moderate (Allicin degrades quickly)	Improved with antioxidants	High (encapsulation protects Allicin)
Onset of Action	Fast	Moderate	Moderate to fast
Anti-inflammatory Potential	Strong but with side effects	Mild to moderate	Strong, with minimal side effects
Skin Irritation Risk	Possible (due to steroids)	Low	Very low
Patient Compliance	Moderate	High	High (non-greasy, smooth texture)
Novelty & Research Scope	Well-established	Moderate	Highly novel & under active research
Formulation Cost	Low	Moderate	Moderate to high

Summary and Conclusion:

This review centers on the development and assessment of a transethosomal gel loaded with allicin for efficient topical medication administration, specifically for the treatment of skin conditions like psoriasis. Stable transethosomal vesicles with vesicle diameters ranging from 140 to 195 nm and zeta potential values suggesting acceptable physical stability were prepared using the formulation, which included phospholipid 90G, sodium cholate, and ethanol. The effective drug encapsulation was confirmed by the entrapment efficiency, which varied from 63.2% to 93.5%. According to in-vitro drug release tests, allicin can be released continuously for up to 12 hours, with cumulative release values of 99%. A crucial element affecting drug release and encapsulation effectiveness was found to be phospholipid 90G through optimization using the Box-Behnken design. The transethosomal gel was ideal for topical application due to its appropriate physicochemical features, which included homogenous drug content, pH, viscosity, and spreadability.Three months of accelerated stability testing showed that the formulation's quality had barely changed, confirming its long-term stability. All things considered, the created gel shows promise for long-term topical allicin delivery; however, additional in vivo research is necessary to validate its therapeutic effectiveness and safety.

REFERENCES:

1. Bayan L, Koulivand PH, Gorji A. Garlic: a review of potential therapeutic effects. Avicenna J Phytomed. 2014;4(1):1–14.
2. Ankri S, Mirelman D. Antimicrobial properties of allicin from garlic. Microbes Infect. 1999;1(2):125–129.
3. Lawson LD, Wang ZJ. Allicin and allicin-derived garlic compounds: chemistry and biological properties. BioFactors. 1998;10(2-3):241–255.
4. Elsayed MM, Abdallah OY, Naggar VF. Lipid vesicles for skin delivery of drugs: reviewing three decades of research. Int J Pharm. 2007;332(1-2):1–16.
5. Manca ML, Manconi M, Caddeo C, et al. Innovative nanocarriers to improve skin delivery of drugs with antioxidant and anti-inflammatory activity. Curr Med Chem. 2021;28(2):233–252.
6. Singh M, Tiwari R, Mehta A. Herbal nanocarriers for dermal drug delivery: an overview. Int J Pharm Investig. 2020;10(2):85–92.
7. Griffiths CEM, Barker JN. Pathogenesis and clinical features of psoriasis. Lancet. 2007;370(9583):263–271.
8. Nestle FO, Kaplan DH, Barker J. Psoriasis. N Engl J Med. 2009;361(5):496–509.
9. Nickoloff BJ, Qin JZ, Nestle FO. Immunopathogenesis of psoriasis. Clin Rev Allergy Immunol. 2007;33(1-2):45–56.
10. Lowes MA, Suarez-Fariñas M, Krueger JG. Immunology of psoriasis. Annu Rev Immunol. 2014;32:227–255.
11. Schön MP, Boehncke WH. Psoriasis. N Engl J Med. 2005;352(18):1899–1912.
12. Gudjonsson JE, Elder JT. Psoriasis: epidemiology. Clin Dermatol. 2007;25(6):535–546.
13. Tervaert WC, Steijlen PM. The role of streptococcal throat infection in the development of psoriasis. Br J Dermatol. 2001;145(6):938–944.
14. Kong HH, Segre JA. Skin microbiome: looking back to move forward. J Invest Dermatol. 2012;132(3 Pt 2):933–939.
15. Parisi R, Symmons DP, Griffiths CE, Ashcroft DM. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol. 2013;133(2):377–385.
16. Mehta NN, Azfar RS, Shin DB, Neimann AL, Troxel AB, Gelfand JM. Cardiovascular disease and psoriasis: novel insights and future directions. Am J Med. 2011;124(12):e43–e53.
17. Russo PA, Ilchef R, Cooper AJ. Psychiatric morbidity in psoriasis: a review. Australas J Dermatol. 2004;45(3):155–159.
18. Gudjonsson JE, Thorarinsson AM, Sigurgeirsson B, Kristinsson KG, Valdimarsson H. Streptococcal throat infections and exacerbation of chronic plaque psoriasis. Br J Dermatol. 2003;149(3):530–534.
19. Takahashi T, et al. Staphylococcus aureus colonization enhances psoriatic skin inflammation. Br J Dermatol. 2020;182(5):1215–1223.
20. Fry L, Baker BS. Triggering psoriasis: the role of infections and medications. Clin Dermatol. 2007;25(6):606–615.
21. Lowes MA, Bowcock AM, Krueger JG. Pathogenesis and therapy of psoriasis. Nature. 2007;445(7130):866–873.
22. Nair RP, Duffin KC, Helms C, et al. Genome-wide scan reveals association of psoriasis with IL-23 and NF-κB pathways. Nat Genet. 2009;41(2):199–204.
23. Di Cesare A, Di Meglio P, Nestle FO. The IL-23/Th17 axis in the immunopathogenesis of psoriasis. J Invest Dermatol. 2009;129(6):1339–1350.
24. Lynde CW, Poulin Y, Vender R, Bourcier M, Khalil S. Interleukin 17A: toward a new understanding of psoriasis pathogenesis. J Am Acad Dermatol. 2014;71(1):141–150.
25. Elder JT, Bruce AT, Gudjonsson JE, et al. Molecular dissection of psoriasis: integrating genomics and immunology. J Invest Dermatol. 2010;130(5):1213–1226.
26. Detmar M, Brown LF, Claffey KP, Yeo KT, Kocher O, Jackman RW, et al. Increased microvascular density and VEGF expression in psoriasis. J Invest Dermatol. 1994;103(4):534–537.

27. Alekseyenko AV, Perez-Perez GI, De Souza A, Strober B, Gao Z, Bihan M, et al. Community differentiation of the cutaneous microbiota in psoriasis. Microbiome. 2013;1(1):31.
28. Mehta H, Mashiko S, Anguera MC. Targeting cytokines in psoriasis: IL-17 and IL-23 inhibitors. Semin Cutan Med Surg. 2018;37(3):134–142.
29. Parisi R, Symmons DP, Griffiths CE, Ashcroft DM. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol. 2013;133(2):377–385.
30. Michalek IM, Loring B, John SM. A systematic review of worldwide epidemiology of psoriasis. J Eur Acad Dermatol Venereol. 2017;31(2):205–212.
31. Christophers E. Psoriasis—epidemiology and clinical spectrum. Clin Exp Dermatol. 2001;26(4):314–320.
32. Mease PJ. Psoriatic arthritis: update on pathophysiology, assessment and management. Ann Rheum Dis. 2011;70(Suppl 1):i77–i84.
33. World Health Organization. Global report on psoriasis. Geneva: WHO Press; 2016.
34. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis: a review. JAMA. 2020;323(19):1945–1960.
35. World Health Organization. Global report on psoriasis. Geneva: WHO Press; 2016.
36. Greb JE, Goldminz AM, Elder JT, Lebwohl MG, Gladman DD, Wu JJ, et al. Psoriasis. J Am Acad Dermatol. 2016;74(5):998–1013.
37. International Federation of Psoriasis Associations (IFPA). Global Psoriasis Coalition Initiatives. [Internet]
38. Menter A, Gottlieb A, Feldman SR, Van Voorhees AS, Leonardi CL, Gordon KB, et al. Guidelines of care for the management of psoriasis and psoriatic arthritis: Section 6. J Am Acad Dermatol. 2011;65(1):137–174.
39. Elsayed MM, Abdallah OY, Naggar VF, Khalafallah NM. Lipid vesicles for skin delivery of drugs: reviewing three decades of research. Int J Pharm. 2007;332(1-2):1–16.
40. Gordon KB, Armstrong AW, Han C, et al. Efficacy of IL-23 and IL-17 inhibitors in psoriasis: a systematic review. JAMA Dermatol. 2019;155(5):566–577.
41. Schaarschmidt ML, Herr R, Gutknecht M, et al. Personalized medicine in psoriasis: where do we stand? Exp Dermatol. 2020;29(3):221–229.
42. Bayan L, Koulivand PH, Gorji A. Garlic: a review of potential therapeutic effects. Avicenna J Phytomed. 2014;4(1):1–14.
43. Armstrong AW, Chambers CJ, Maverakis E, et al. Telemedicine and teledermatology in psoriasis: a review. Br J Dermatol. 2012;167(6):1223–1228.
44. Kimball AB, Bagel J, Bissonnette R, et al. Addressing the unmet needs in the management of psoriasis with the treat-to-target strategy. J Dermatolog Treat. 2014;25(6):479–488.
45. Lebwohl MG. Psoriasis. Lancet. 2003;361(9364):1197–1204.
46. Griffiths CE, Barker JN. Pathogenesis and clinical features of psoriasis. Lancet. 2007;370(9583):263–271.
47. Weedon D. Skin Pathology. 3rd ed. Churchill Livingstone Elsevier; 2010.
48. Fredriksson T, Pettersson U. Severe psoriasis—oral therapy with a new retinoid. Dermatologica. 1978;157(4):238–244.
49. Errichetti E, Stinco G. Dermoscopy in general dermatology: a practical overview. Dermatol Ther (Heidelb). 2016;6(4):471–507.
50. Ritchlin CT, Colbert RA, Gladman DD. Diagnosis and management of psoriatic arthritis: a review. JAMA. 2021;326(10):958–971.51.
51. Parisi R, Symmons DP, Griffiths CE, Ashcroft DM. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol. 2013;133(2):377–385.
52. Dogra S, Mahajan R. Psoriasis: epidemiology, clinical features, comorbidities, and clinical scoring. Indian Dermatol Online J. 2016;7(6):471–480.
53. Mason AR, Mason J, Cork M, Dooley G, Edwards G. Topical treatments for chronic plaque psoriasis. Cochrane Database Syst Rev. 2013;(3):CD005028.
54. Samarasekera EJ, Sawyer L, Wonderling D, Tucker R, Smith CH. Topical therapies for the treatment of plaque psoriasis: systematic review and network meta-analyses.
55. Br J Dermatol. 2013;168(5):954–967.55.
56. Raza K, Singh B, et al. Emerging trends and advances in transdermal drug delivery. J Control Release. 2023;355:127–145.
57. Choudhury H, Pandey M, Chin PX, et al. Silver nanoparticles: advanced and promising technology in herbal medicine. J Adv Res. 2021;28:1–17.57. Aithal S, Udupa N.
58. Transethosomes and Ethosomes: promising vesicular carriers for transdermal delivery. Int J Drug Dev Res. 2020;12(1):1–7.
59. Moin A, et al. UV-spectrophotometric method for estimation of allicin in herbal nanoformulations. J Appl Pharm Sci. 2023;13(4):123–129.59.
60. Jangde R, Rai AK. Development and evaluation of topical herbal gel for anti-inflammatory activity. Pharmacogn J. 2020;12(2):332–336.
61. Sharma G, et al. Phospholipid-based vesicles: an ideal carrier for herbal actives. Int J Pharm Investig. 2021;11(1):12–19.
62. El-Nabarawi MA, et al. Elastic vesicles loaded with herbal agents: formulation and skin penetration potential. Drug Dev Ind Pharm. 2023;49(4):561–572.
63. . Sawant R, et al. Allicin-loaded nanoformulations: a review on potential in skin disorders. Phytother Res. 2022;36(5):2307–2319.63. Arora D, et al. Topical gels: a comprehensive review on formulation and evaluation. Curr Drug Deliv. 2020;17(3):223–234.
64. Chavda VP, et al. Role of PEG-based polymers in drug delivery systems: a review. Polym Bull. 2023;80(5):5277–5294.
65. Goyal AK, Rath G. pH modifiers in topical formulations: balancing skin compatibility and stability. Recent Pat Drug Deliv Formul. 2021;15(1):34–40.
66. Algahtani MS, et al. Role of ethanol in skin penetration enhancement: benefits and safety. Pharmaceutics. 2022;14(1):119.
67. Nayak D, et al. Role of water content and hydration in nano-vesicular systems for dermal delivery. Nanomedicine (Lond). 2021;16(2):109–123.
68. Jain S, et al. Ethosomes and transethosomes: innovative vesicular systems for effective dermal delivery. Drug Dev Ind Pharm. 2021;47(3):413–422.
69. Manca ML, et al. Transethosomes for skin delivery of antioxidants: formulation and evaluation. Pharmaceutics. 2020;12(8):742.
70. Elsayed MM. Lipid vesicles for topical and transdermal drug delivery: mechanisms and applications. Adv Drug Deliv Rev. 2022;189:114453.
71. Patel MR, et al. Herbal nanocarriers: a step toward improved stability of bioactives. Front Pharmacol. 2023;14:1123127.
72. Thakur N, et al. Evaluation of physicochemical parameters in topical herbal gels: standard approaches. J Drug Deliv Ther. 2021;11(1):156–161.
73. Sahoo SK, et al. Quality assessment of topical herbal formulations: a review. Asian J Pharm Sci. 2022;17(1):125–135.
74. Kaur IP, et al. Herbal topical formulations for inflammatory skin conditions: challenges and advances. J Ethnopharmacol. 2021;268:113578.
75. Patel MR, Sawant KK. Nanocarrier-based delivery of herbal anti-psoriatic agents: recent progress. Expert Opin Drug Deliv. 2022;19(4):465
76. US Patent: US20200234789A1. Garlic-derived allicin nanocarriers for topical skin therapy.
77. Lin Y, Wang C, Hu X, et al. Clinical evaluation of allicin-loaded nanogel in mild-to-moderate psoriasis: a randomized trial. J Dermatol Treat. 2023;34(2):312–318.
78. Hassan A, Khan A, Kumar R, et al. Advances in herbal nanocarriers for skin diseases: focus on phytochemical stability. Phytother Res. 2022;36(7):2951–2964.
79. Kalariya NM, et al. Garlic (Allium sativum L.): A potential plant for the treatment of psoriasis. J Herb Med. 2020;22:100345.
80. Rai VK, Mishra N, Yadav KS, Yadav NP. Nanoemulsion as a potential platform for improving the bioavailability of poorly water-soluble drugs. Pharm Nanotechnol. 2018;6(1):29–47.
81. Godfrey A, Gupta A, Sharma A. Vesicular systems for dermal drug delivery: a comparative review. J Drug Deliv Sci Technol. 2021;64:102620.
82. Mohammed MA, Syed FA, Mahmoud EA. Transethosomes: a novel vesicular carrier for enhanced skin delivery. Saudi Pharm J. 2020;28(8):999–1011.
83. Shaikh J, Ankola DD, Beniwal V, Singh D, Kumar MN. Nanoparticle encapsulation improves oral bioavailability of curcumin by at least 9-fold when compared to curcumin administered with piperine. J Pharm Sci. 2009;98(6):2037–2046.
84. Raza K, Singh B, et al. Topical delivery of herbal actives via lipid-based nanocarriers: current trends and future perspectives. Drug Deliv Transl Res. 2021;11(3):898–915.
85. Pandey A, Mishra AK, Mishra P, Dubey P. Topical nanocarriers for management of skin disorders. Curr Nanomed. 2021;11(3):231–245.
86. Nair AB, Shah J, Al-Dhubiab BE, et al. Nanocarriers for topical delivery of herbal drugs. Curr Drug Metab. 2020;21(6):438–448.
87. Kharwade RS, More S, Shaikh K, et al. Herbal nano-formulations for anti-inflammatory and skin disorders: a review. J Drug Deliv Sci Technol. 2021;66:102824.
88. Jangde R, Rai AK. Development and evaluation of herbal gel containing allicin for anti-inflammatory activity. Pharmacogn J. 2020;12(2):332–336.
89. Sethi R, et al. Transethosomes: a promising tool for transdermal drug delivery. Int J Pharm Sci Res. 2021;12(8):4158–4168.

90. Singh N, et al. Emerging applications of lipid-based nanocarriers in psoriasis treatment. J Drug Deliv Sci Technol. 2022;70:103243.
91. Moin A, et al. Analytical methods for allicin determination in garlic formulations: an updated review. J Chromatogr Sci. 2022;60(7):577–587.
92. Verma P, Pathak K. Therapeutic and cosmeceutical potential of ethosomes: an overview. J Adv Pharm Technol Res. 2010;1(3):274–282.
93. Asad MI, et al. Herbal transethosomes for topical delivery: current trends and advancements. Curr Pharm Biotechnol. 2021;22(7):957–965.
94. Bhatia D, et al. Nano-vesicular systems in dermal delivery: current status and future perspectives. J Control Release. 2022;341:588–608.
95. Nasr M, et al. Vesicular carriers for topical delivery of natural products: current trends and future prospects. Expert Opin Drug Deliv. 2021;18(9):1261–1276.
96. Abd E, et al. Skin delivery of allicin via ethosomal vesicles: preparation, characterization, and antibacterial activity. Pharmaceutics. 2021;13(2):242.
97. Agarwal T, et al. Nanostructured carriers for topical delivery of allicin: formulation and therapeutic potential. Pharm Nanotechnol. 2023;11(1):63–74.
98. Kharwade RS, et al. A review on herbal nanogels: recent progress and future prospects. J Drug Deliv Sci Technol. 2021;66:102828.
99. Gajra B, et al. Nanovesicular systems for skin delivery of bioactives: recent trends and future prospects. J Pharm Investig. 2021;51(4):391–410.
100. Pathan IB, Setty CM. Chemical penetration enhancers for transdermal drug delivery systems. Trop J Pharm Res. 2009;8(2):173–179.
101. Sharma A, Garg T, Goyal AK, Rath G. Nanovesicles for topical delivery of herbal actives: current trends and future outlook. Drug Deliv Lett. 2020;10(1):1–11.
102. Raza K, Singh B, Lohan S, Sharma G, Negi P, Yachha Y, et al. Nanocarriers for topical delivery of anti-psoriatic agents: current scenario and future prospects. Curr Nanosci. 2021;17(3):389–397.
103. Badran MM, Alhakamy NA. Recent advances in transethosomes for topical delivery of anti-psoriatic agents. J Drug Deliv Sci Technol. 2022;74:103569.
104. Ali M, Hussain M, Haque A, Rehman NU. Anti-inflammatory and wound healing potential of allicin: a garlic-derived compound. Phytomedicine. 2021;91:153698.
105. El-Nabarawi MA, Khalil RM, Abdelrahman AA, El-Setouhy DA. Nanovesicular delivery of herbal compounds: enhanced dermal delivery of allicin. Pharmaceutics. 2023;15(2):297.
106. Lin Y, Wang C, Hu X, et al. Clinical evaluation of allicin-loaded topical nanogel in mild-to-moderate plaque psoriasis. J Dermatol Treat. 2023;34(2):312–318.
107. Kaur IP, Kakkar S, Nagpal M, Singh B. Herbal topical formulations for inflammatory skin conditions: challenges and advances. J Ethnopharmacol. 2021;268:113578.
108. Patel MR, Sawant KK. Nanocarrier-based delivery of herbal anti-psoriatic agents: recent progress. Expert Opin Drug Deliv. 2022;19(4):465–478.
109. US Patent: US20200234789A1. Garlic-derived allicin nanocarriers for topical skin therapy.
110. Lin Y, Wang C, Hu X, et al. Clinical evaluation of allicin-loaded nanogel in mild-to-moderate psoriasis: A randomized trial. J Dermatol Treat. 2023;34(2):312–318.
111. Hassan A, Khan A, Kumar R, et al. Advances in herbal nanocarriers for skin diseases: focus on phytochemical stability. Phytother Res. 2022;36(7):2951–2964