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## Review Paper

# Herbosomal Gel Containing Manjistha (*Rubia Cordifolia*) And Turmeric (*Curcuma Longa*) For Psoriasis -A Detailed Review

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## ABSTRACT

Psoriasis is a chronic immune-mediated inflammatory skin disorder characterized by erythema, scaling, and keratinocyte hyperproliferation. The disease is mediated by cytokine pathways involving TNF- $\alpha$ , IL-17, and IL-23, and conventional therapies — including corticosteroids, methotrexate, and biologics — are associated with adverse effects and limited long-term safety. Herbal drugs such as *Rubia cordifolia* (Manjistha) and *Curcuma longa* (Turmeric) exhibit potent anti-inflammatory, antioxidant, and immunomodulatory activities; however, their poor bioavailability limits therapeutic efficacy in conventional formulations. Herbosomal drug delivery systems, which form phospholipid complexes with herbal phytoconstituents, enhance permeability, stability, and targeted skin delivery. This review comprehensively focuses on the role of herbosomal gel containing Manjistha and Turmeric in psoriasis treatment, encompassing formulation methodology, physicochemical evaluation, mechanisms of action, therapeutic advantages, and future prospects.

## INTRODUCTION

Psoriasis is a chronic, relapsing autoimmune skin disease that affects approximately 2–3% of the global population [1]. It is one of the most prevalent immune-mediated inflammatory disorders, resulting in significant morbidity and impaired quality of life. Clinically, psoriasis is characterized by well-demarcated erythematous

plaques covered by silvery-white scales, primarily affecting the elbows, knees, scalp, and lower back. The underlying pathophysiology involves complex interactions between the innate and adaptive immune systems, resulting in hyperproliferation of keratinocytes and dermal inflammation [2]. The immunological cascade in psoriasis is driven by dendritic cell activation, which promotes T-helper cell (Th1 and Th17)

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differentiation and subsequent release of pro-inflammatory cytokines including tumour necrosis factor-alpha (TNF- $\alpha$ ), interleukin-17 (IL-17), interleukin-23 (IL-23), and interleukin-22 (IL-22) [3]. These cytokines perpetuate the inflammatory cycle, stimulating keratinocyte proliferation and inhibiting normal differentiation.

Conventional therapeutic modalities include topical corticosteroids, vitamin D analogues, systemic immunosuppressants (methotrexate, cyclosporine), and biological agents (anti-TNF- $\alpha$ , anti-IL-17, anti-IL-23). While effective, these treatments are associated with significant adverse effects: prolonged corticosteroid use causes skin atrophy and tachyphylaxis; systemic agents carry risks of nephrotoxicity, hepatotoxicity, and immunosuppression; and biologics are costly and may lose efficacy over time [4].

This unmet clinical need has driven growing interest in herbal and complementary approaches to psoriasis management. Plants used in traditional Ayurvedic medicine — particularly *Rubia cordifolia* (Manjistha) and *Curcuma longa* (Turmeric) — possess well-documented anti-inflammatory, antioxidant, and immunomodulatory properties relevant to psoriasis pathophysiology. However, their clinical application has historically been limited by poor absorption and low bioavailability. Advanced herbosomal drug delivery, combining these phytoconstituents with phospholipid carriers, represents a promising strategy to overcome these limitations [5].

## 2. Role of Herbal Drugs in Psoriasis

### 2.1 Manjistha (*Rubia cordifolia*)

*Rubia cordifolia* L. (Family: Rubiaceae), commonly known as Manjistha or Indian Madder, is a perennial climbing herb extensively utilized in Ayurvedic, Unani, and traditional Chinese medicine. Its dried roots and stems are the primary therapeutic parts used, containing a rich array of

phytoconstituents including anthraquinones (purpurin, munjistin, xanthopurpurin, pseudopurpurin), triterpenoids, flavonoids, polysaccharides, and bicyclic hexapeptides [6].

In traditional Ayurveda, Manjistha is classified as a 'Rakta Shodhaka' (blood purifier) and 'Kaphapittashamaka' herb, used to treat a wide variety of skin disorders including psoriasis, eczema, acne, and chronic wounds. Modern pharmacological studies have corroborated many of these traditional claims [7].

### Pharmacological Activities of *Rubia cordifolia*:

- Anti-inflammatory: inhibits pro-inflammatory cytokines and mediators including TNF- $\alpha$  and IL-6
- Antioxidant: scavenges free radicals; reduces oxidative stress implicated in psoriatic plaques
- Antimicrobial: broad-spectrum antibacterial and antifungal activity
- Immunomodulatory: modulates both innate and adaptive immune responses
- Anti-proliferative: the ethyl acetate fraction of *R. cordifolia* root extract has demonstrated potent antiproliferative effects on HaCaT keratinocytes (IC<sub>50</sub> 0.9  $\mu$ g/mL) and promotes keratinocyte differentiation in vivo, confirming its antipsoriatic potential [8]
- Detoxifying and hepatoprotective: supports liver function and blood purification

Despite this promising pharmacological profile, the clinical translation of *Rubia cordifolia* extracts has been hindered by their hydrophilic nature, large molecular size, and poor lipid solubility, which collectively result in inadequate skin penetration and low systemic bioavailability when formulated conventionally [5].

### 2.2 Turmeric (*Curcuma longa*)

*Curcuma longa* L. (Family: Zingiberaceae), commonly known as Turmeric, is one of the most



extensively studied medicinal plants worldwide. The rhizome of *Curcuma longa* contains curcuminoids — primarily curcumin (diferuloylmethane, approximately 77%), demethoxycurcumin, and bisdemethoxycurcumin — which account for its characteristic yellow color and the majority of its therapeutic activity [9].

Curcumin acts on multiple molecular targets relevant to psoriasis pathogenesis:

- Inhibits cyclooxygenase-2 (COX-2) and lipoxygenase (LOX) enzymes, reducing prostaglandin and leukotriene synthesis
- Suppresses NF-κB activation, reducing transcription of pro-inflammatory genes
- Downregulates cytokines including TNF-α, IL-6, IL-17, IL-22, and IL-23
- Inhibits STAT3 phosphorylation and MAPK signalling pathways
- Inhibits phosphorylase kinase (PhK), an enzyme overexpressed in psoriatic skin
- Reduces keratinocyte hyperproliferation by arresting the cell cycle and promoting apoptosis

Clinical evidence supports the use of topical curcumin/turmeric formulations in psoriasis. A

randomized double-blind placebo-controlled trial demonstrated significant improvement in PASI scores with topical turmeric hydro-alcoholic gel applied twice daily for 9 weeks [10]. A separate double-blind clinical trial showed that oral Meriva (lecithin-based curcumin, 2 g/day) as adjuvant therapy with topical steroids produced greater PASI reduction and significantly decreased serum IL-22 levels compared to steroids alone [11]. A meta-analysis of 7 clinical RCTs and 19 preclinical studies confirmed that curcumin improved PASI scores as both monotherapy and combination therapy (SMD -0.83%; 95% CI -1.53 to -0.14; p = 0.02) [12].

However, curcumin's clinical utility is substantially limited by its poor aqueous solubility (~11 ng/mL at pH 7), low gastrointestinal absorption, rapid metabolism and elimination, and inadequate skin penetration. These pharmacokinetic challenges necessitate advanced delivery systems to realize its therapeutic potential [13].

### 3. Comparative Profile of Key Herbal Constituents

Parameter	<i>Rubia cordifolia</i> (Manjistha)	<i>Curcuma longa</i> (Turmeric)
Plant family	Rubiaceae	Zingiberaceae
Active constituents	Purpurin, munjistin, anthraquinones, flavonoids	Curcumin, demethoxycurcumin, bisdemethoxycurcumin
Key pharmacological actions	Anti-inflammatory, antioxidant, antimicrobial, immunomodulatory	Anti-inflammatory, antioxidant, anti-proliferative, immunomodulatory
Mechanism in psoriasis	Inhibits cytokines; reduces keratinocyte proliferation	Inhibits COX-2, PhK, NF-κB, STAT3, IL-17/IL-22
Bioavailability (conventional)	Low (poor lipid solubility)	Very low (~1% oral bioavailability)
Traditional use	Blood purifier, skin diseases, Ayurveda	Anti-inflammatory, wound healing, Ayurveda



#### 4. Need for Novel Drug Delivery in Psoriasis

The physicochemical properties of both *Rubia cordifolia* and curcumin present significant formulation challenges for effective topical delivery:

- Poor aqueous solubility limits drug concentration at the site of action
- Hydrophilic character impairs passive diffusion across the lipid-rich stratum corneum
- Large molecular size of polyphenolic compounds exceeds the 500 Da cutoff for passive skin permeation
- Rapid degradation in physiological conditions reduces drug stability
- Low skin residence time limits therapeutic exposure

The stratum corneum, composed of densely packed dead keratinocytes embedded in a lipid matrix, presents the primary barrier to topical drug delivery. Molecules with molecular weight >500 Da or inappropriate partition coefficients (log P outside 1–3) fail to penetrate adequately [14]. Curcumin, despite being lipophilic, forms aggregates in aqueous environments and is rapidly degraded by alkaline pH and light.

These challenges collectively necessitate novel drug delivery approaches that can: (1) improve solubilization and stability, (2) enhance skin

permeation, (3) achieve sustained drug release, and (4) target drug delivery to psoriatic lesions.

#### 5. Herbosome Technology

##### 5.1 Definition and Concept

Herbosomes (also termed phytosomes or phyto-phospholipid complexes) are phospholipid-based drug delivery systems in which standardized herbal extracts or isolated phytoconstituents are complexed with phospholipids in defined molar ratios to form stable amphiphilic complexes. The term derives from 'herbo' (plant) and 'some' (body/structure). Herbosomes bridge conventional and novel drug delivery systems, combining the therapeutic advantages of herbal medicine with the pharmacokinetic superiority of lipid-based carriers [15].

Unlike simple encapsulation (as in liposomes), in herbosome formation the phytoconstituent chemically interacts with the phospholipid head group through hydrogen bonding between the polar functional groups of the phytochemical and the phosphate moiety of phosphatidylcholine. This results in a new molecular complex with distinct physicochemical properties superior to the individual components [16].

##### 5.2 Composition of Herbosomes

Component	Examples	Role
Herbal extract	<i>Rubia cordifolia</i> extract, Curcumin extract	Therapeutic active ingredient
Phospholipid	Phosphatidylcholine (soy lecithin), Phosphatidylserine	Complexation agent; enhances lipophilicity
Organic solvent	Dichloromethane, Ethyl acetate	Medium for complex formation
Aqueous phase	Distilled water, Buffer	Hydration medium for vesicle formation
Gelling agent	Carbopol 940, HPMC	Provides gel matrix for topical application
Penetration enhancer	Propylene glycol, Ethanol	Improves skin permeation



### 5.3 Advantages of Herbosomal Delivery

- Enhanced bioavailability: phospholipid complexation converts hydrophilic phytoconstituents into lipid-compatible molecules, dramatically improving absorption
- Improved skin permeation: phospholipid bilayer structure facilitates partitioning into the stratum corneum
- Increased stability: protection from hydrolysis, oxidation, and enzymatic degradation (including cytochrome P-450 and P-glycoprotein)
- Controlled and sustained drug release: reduces dosing frequency
- Reduced systemic toxicity: targeted topical delivery minimizes systemic exposure
- Better patient compliance: topical gel formulation is non-invasive and cosmetically acceptable
- Synergistic activity: combination of two herbal extracts may produce additive or synergistic anti-inflammatory effects

## 6. Formulation of Herbosomal Gel

### 6.1 Preparation of Herbosomes

The solvent evaporation method is most commonly employed for herbosome preparation. The following steps outline the process:

1. Accurately weighed quantities of herbal extract (*Rubia cordifolia* or *Curcuma longa*) and phospholipid (phosphatidylcholine/soy lecithin) are dissolved together in a suitable

organic solvent (dichloromethane or ethyl acetate) in a molar ratio of 1:1 or 1:2

2. The mixture is transferred to a rotary evaporator flask and the solvent is evaporated under reduced pressure at 40°C, forming a thin, uniform film on the flask walls
3. The thin film is hydrated with a small volume of n-hexane or distilled water with vigorous shaking to form a herbosomal suspension
4. The suspension is stirred on a magnetic stirrer for 30 minutes, then centrifuged at 3000 rpm for 15 minutes to separate untrapped extract
5. The resultant herbosomal concentrate is collected and characterized prior to gel incorporation

### 6.2 Preparation of Herbosomal Gel

The gel base is prepared using Carbopol 940 as the primary gelling agent:

6. Carbopol 940 (0.5–2.0% w/w) is slowly dispersed in distilled water and allowed to hydrate with gentle stirring for 2 hours
7. Triethanolamine (TEA) is added dropwise with continuous stirring to neutralize and activate the gel (target pH 6–7)
8. Propylene glycol (as penetration enhancer and humectant) and preservatives are incorporated
9. The prepared herbosomal suspension is gradually incorporated into the gel base under gentle stirring
10. Homogeneity is confirmed and the gel is transferred to appropriate containers

Formulation Component	Quantity/Concentration	Function
Rubia cordifolia herbosome	Optimized batch	Therapeutic active
Curcuma longa herbosome	Optimized batch	Therapeutic active
Phosphatidylcholine (soy lecithin)	1:1 to 1:2 molar ratio	Complexation agent
Carbopol 940	0.5–2.0% w/w	Primary gelling agent
Triethanolamine (TEA)	q.s. to pH 6–7	Neutralizing/pH adjusting agent



Formulation Component	Quantity/Concentration	Function
Propylene glycol	5–15% v/v	Penetration enhancer, humectant
Methyl paraben / Propyl paraben	0.1% / 0.02%	Antimicrobial preservatives
Distilled water	q.s. to 100%	Aqueous phase base

## 7. Evaluation of Herbosomal Gel

Comprehensive evaluation of the herbosomal gel encompasses characterization at three levels: vesicle characterization, gel physicochemical properties, and drug release behaviour.

### 7.1 Physicochemical Parameters

Parameter	Method	Optimized Result
Vesicle particle size	Dynamic Light Scattering (DLS)	~98.7 nm
Polydispersity index (PDI)	DLS	<0.3 (monodisperse)
Zeta potential	Electrophoretic mobility	Negative (stability indicator)
Entrapment efficiency	Centrifugation / UV spectroscopy	98.12%
Drug loading	UV-Vis spectrophotometry	98.62%
pH of gel	Digital pH meter	6.39 ± 0.03
Viscosity	Brookfield viscometer	Optimized for spreadability
Spreadability	Parallel plate method	Good (>30 mm spread)
Cumulative drug release (8h)	Franz diffusion cell, pH 7.4 PBS	78.23 ± 0.045%
Drug-excipient interaction	FTIR spectroscopy	No interaction observed
Stability (25°C)	ICH guidelines	Stable; no phase separation

### 7.2 Drug Release Profile

In vitro drug release studies using Franz diffusion cells with dialysis membrane (MWCO 12,000–14,000 Da) and pH 7.4 phosphate buffer saline (PBS) as receptor medium demonstrate sustained release kinetics. The optimized herbosomal gel formulation showed cumulative release of 78.23 ± 0.045% over 8 hours, compared to approximately 45–50% for conventional non-herbosomal gel formulations, indicating significantly enhanced and prolonged release [17].

Drug release follows a combination of diffusion and erosion mechanisms, best fitted to the Higuchi or Korsmeyer-Peppas kinetic model, confirming anomalous (non-Fickian) transport consistent with swelling-controlled release from the carbopol gel matrix.

### 7.3 Stability Studies

Stability studies conducted at 25°C ± 2°C/60% ± 5% RH (long-term) and 40°C ± 2°C/75% ± 5% RH (accelerated) per ICH Q1A guidelines confirm the formulation remains stable over the study period



with no significant changes in particle size, pH, viscosity, drug content, or appearance. No phase separation, syneresis, or microbial contamination was observed [17].

## 8. Mechanism of Action in Psoriasis

The herbosomal gel containing Manjistha and Turmeric targets multiple pathogenic pathways in psoriasis simultaneously, conferring a multi-mechanistic therapeutic approach:

Mechanism	Molecular Target	Effect in Psoriasis
Anti-inflammatory	TNF- $\alpha$ , IL-17, IL-22, IL-23, NF- $\kappa$ B, COX-2, LOX	Reduces dermal and epidermal inflammation; breaks inflammatory cycle
Anti-proliferative	PhK, STAT3, MAPK, cell cycle regulators	Normalizes keratinocyte hyperproliferation
Antioxidant	ROS scavenging, Nrf2 pathway	Reduces oxidative stress that amplifies inflammation in psoriatic skin
Immunomodulatory	Th1/Th17 cytokine balance, dendritic cell activity	Corrects aberrant immune activation underlying psoriasis
Enhanced skin permeation	Stratum corneum lipid bilayers	Phospholipid structure facilitates deeper drug penetration
Sustained release	Gel matrix + vesicle wall	Maintains therapeutic drug concentrations at target site

The phospholipid bilayer of the herbosome closely mimics biological membranes, enabling it to fuse with skin lipids and facilitate transcutaneous delivery to the viable epidermis and dermis — the primary sites of psoriatic inflammation. The vesicular structure protects curcumin from photodegradation and alkaline hydrolysis, maintaining drug stability during the permeation process [18].

Additionally, curcumin has been shown to inhibit the NLRP3 inflammasome pathway, reducing IL-18 and IL-22 production, and to downregulate STAT3 phosphorylation — a key transcription factor in psoriatic keratinocyte hyperproliferation [3]. The anthraquinones from *Rubia cordifolia* complement these actions through independent anti-inflammatory and anti-proliferative mechanisms.

## 9. Therapeutic Advantages over Conventional Formulations

- Superior bioavailability: herbosomal technology increases skin permeation several-fold compared to conventional topical formulations of the same herbal extracts
- Improved therapeutic efficacy: particle size of ~98 nm enables deep tissue penetration; entrapment efficiency >98% ensures maximum drug delivery
- Sustained release profile: controlled release (78% over 8 hours) reduces application frequency and improves patient compliance
- Reduced adverse effects: targeted topical delivery minimizes systemic exposure compared to oral or parenteral conventional drugs
- Synergistic herbal combination: the complementary anti-inflammatory and anti-



proliferative mechanisms of *Rubia cordifolia* and *Curcuma longa* produce enhanced therapeutic outcomes

- Natural and safe: derived from Ayurvedic medicinal plants with centuries of traditional use and established safety profiles
- Cost-effective: herbal raw materials are widely available and economical compared to biological therapies
- No immunosuppression: unlike systemic methotrexate or biologics, does not cause broad immunosuppression or organ toxicity
- Cosmetically acceptable: gel base with appropriate pH, viscosity, and spreadability ensures patient acceptability

## 10. Future Perspectives

Herbosomal gel technology for psoriasis represents an evolving field with several promising directions for future research and development:

- Standardization of extracts: development of validated analytical methods (HPLC, LC-MS) for quantification of marker compounds in *Rubia cordifolia* and *Curcuma longa* extracts to ensure batch-to-batch consistency
- Nanoherbosomal systems: integration of herbosomal technology with nanoparticle platforms (nanostructured lipid carriers, transfersomes, ethosomes) to further enhance skin penetration and target deeper lesional skin layers
- Combination therapy protocols: investigation of herbosomal gel as adjuvant to phototherapy (narrowband UVB) or low-dose systemic therapy for moderate-to-severe psoriasis
- Robust clinical trials: well-designed, multi-centre randomized controlled trials with adequate sample sizes to establish clinical efficacy and safety endpoints (PASI score reduction, DLQI improvement)

- Pharmacokinetic studies: *in vivo* skin bioavailability and pharmacokinetic studies using tape stripping, confocal microscopy, and microdialysis to characterize penetration depth and drug residence time
- Biomarker studies: assessment of cytokine profiles (IL-17, IL-23, TNF- $\alpha$ ) in psoriatic skin biopsies following herbosomal treatment to confirm mechanistic activity *in vivo*
- Regulatory pathway development: establishment of quality standards and regulatory frameworks for novel herbosomal formulations under applicable pharmacopeial guidelines
- Theranostic applications: exploration of dual-purpose herbosomal carriers that can simultaneously deliver therapy and provide imaging contrast for treatment monitoring

## CONCLUSION

Psoriasis remains a challenging chronic condition demanding safe, effective, and well-tolerated long-term treatments. The dual herbal combination of *Rubia cordifolia* (Manjistha) and *Curcuma longa* (Turmeric), formulated as a herbosomal gel, represents a rational and scientifically substantiated approach to psoriasis management. By addressing the key pharmacokinetic limitations of these phytoconstituents — poor solubility, inadequate skin penetration, and rapid degradation — through herbosomal technology, this formulation achieves superior drug delivery characteristics: nanoscale vesicle size (~98 nm), high entrapment efficiency (~98%), and sustained drug release (~78% over 8 hours) with excellent physicochemical stability.

The multi-mechanistic action — encompassing anti-inflammatory, antioxidant, immunomodulatory, and anti-proliferative effects operating through TNF- $\alpha$ , IL-17/IL-23 axis modulation, NF- $\kappa$ B suppression, STAT3 inhibition, and keratinocyte cycle arrest — directly



targets the pathogenic pathways of psoriasis. This approach harnesses the synergistic potential of Ayurvedic herbal wisdom combined with modern nanotechnology to produce an effective, safe, and patient-friendly alternative to conventional therapies.

Future clinical investigation, supported by robust pharmacokinetic evaluation and regulatory standardization, has the potential to establish herbosomal gel as a viable, evidence-based therapeutic option in the psoriasis treatment armamentarium.

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