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A COMPREHENSIVE REVIEW ON PSORIASIS

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ABSTRACT

Psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by excessive keratinocyte proliferation and the formation of red, scaly plaques. Affecting millions of people worldwide, the disease significantly impacts patients' physical health, emotional well-being, and quality of life. Current treatment options include topical therapies, phototherapy, systemic drugs, and biologic agents; however, these approaches often face challenges such as poor skin penetration, systemic side effects, and reduced patient compliance. Recent advances in pharmaceutical research have focused on the development of novel drug delivery systems to enhance treatment efficacy and safety. Nanotechnology-based carriers, including liposomes, solid lipid nanoparticles, nanocapsules, nanospheres, and pro-dispersion formulations, have shown considerable promise in improving drug solubility, stability, bioavailability, and controlled release. These systems facilitate targeted drug delivery to affected skin tissues while minimizing adverse effects. Drugs such as cyclosporin, tazarotene, and tretinoin have demonstrated improved therapeutic performance when incorporated into these advanced formulations. Additionally, biocompatible materials like chitosan offer supplementary benefits, including enhanced bioadhesion, wound healing, and antimicrobial activity. This review summarizes the pathogenesis of psoriasis, conventional treatment strategies, and recent advancements in nanotechnology-based drug delivery systems, highlighting their potential to provide more effective, safer, and patient-friendly therapeutic options for psoriasis management.

KEYWORDS: Psoriasis, inflammation, skin disorder, Nanotechnology, Drug Delivery Systems, Targeted Therapy.

INTRODUCTION[1]

Psoriasis is a lifelong immune-mediated inflammatory skin disease, associated with morbidities such as psoriatic arthropathy, psychological, cardiovascular and hepatic diseases. In 2014, the World Health Organization recognised psoriasis as a serious non-communicable disease and highlighted the distress related to misdiagnosis, inadequate treatment and stigmatisation of this disease.

Definition by WHO 2016 report

Psoriasis is a chronic, non-communicable, painful, disfiguring and disabling disease for which there is no cure and with great negative impact on patients' quality of life (QoL).

HISTORY[3,4,5,6]

Throughout history, the understanding of psoriasis has its roots in biblical references, associating it with skin disorders like tzaraath, often translated as leprosy. This translation led to misconceptions, compounded by Greek terminology—describing scaly skin diseases as lepra and itching skin conditions as psora. In the late 18th century, English physicians Robert Willan and Thomas Bateman differentiated between skin diseases, defining leprosy with uniform circular patches and psoriasis as consistently uneven.

Cornelius Celsus is believed to have been the first to describe psoriasis in ancient Rome. In 1813, British dermatologist Thomas Bateman suggested a connection between psoriasis and arthritic symptoms. Historical

accounts include Admiral William Halsey's inability to join the Battle of Midway in World War II due to developing psoriasis, necessitating treatment directed by Admiral Chester Nimitz.

Throughout history, various toxic and often ineffective treatments were used for psoriasis. Dermatologists employed arsenic-based Fowler's solution and mercury in the 18th and 19th centuries. Phenol, iodine, and sulphur were also used, mistakenly assuming psoriasis was infectious. In the early 1900s, coal tar topicals with UV light became common. Intravenous gold formulations were used for psoriatic arthritis, resembling treatments for rheumatoid arthritis during the same period.

EPIDEMIOLOGY[1,2,7]

The prevalence of psoriasis varies widely across regions, ranging from 0.09% in the United Republic of Tanzania to 11.4% in Norway. While there's limited evidence linking geographic latitude to psoriasis rates, it appears more common among northern European populations and less prevalent in eastern Asian groups.

Studies examining psoriasis prevalence among different ethnicities show varying rates. In a 2001 US poll, individuals of Caucasian, Black, and other ancestries had prevalence rates of 2.5%, 1.3%, and 1.0%, respectively. Another study from 2009-2010 in the US reported prevalence rates of 3.6% for Caucasians, 1.9% for Blacks, 1.6% for Hispanics, and 1.4% for other ethnic groups.

COMMON PSORIASIS SITES[8,9]

Common areas of distribution of psoriasis. The lesions are usually symmetrically distributed and are characteristically located on the ears, elbows, knees, umbilicus, gluteal cleft and genitalia. The joints (psoriatic arthritis), nails and scalp may also be affected.

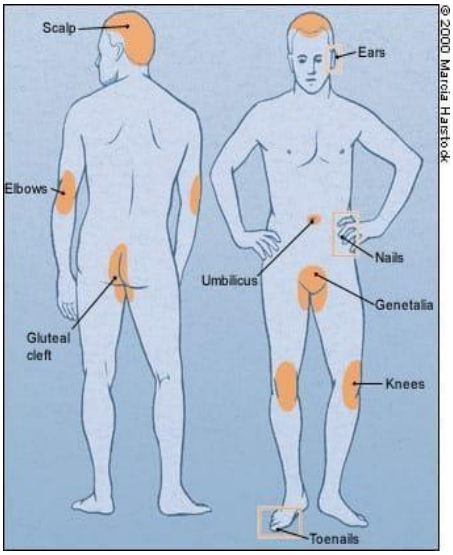

Figure 1: Common sites of Psoriasis.

TYPES OF PSORIASIS[2,3,4,5,9]

Type of Psoriasis	Description	Common Locations	Symptoms
Plaque Psoriasis	Most common type. Raised, inflamed, red skin with silvery, white scales.	Elbows, Knees, Scalp, Lower back	Itching, burning
Guttate Psoriasis	Often starts in children or young adults. Small, pink-red spots on the skin.	Trunk, Upper arms, Thighs, Scalp	May resolve without treatment
Inverse Psoriasis	Found in skin folds. Bright red, smooth, shiny patches that worsen with sweating and rubbing.	Armpits, Groin, Under breasts, Genitals	Aggravated by friction, sweating, infections
Pustular Psoriasis	Uncommon, causes pus-filled bumps surrounded by red or purplish skin. Can be serious.	Hands, Feet, or generalized	Fever, chills, nausea, fast heart rate, weakness
Erythrodermic Psoriasis	Very serious, affects most of the body, fiery skin resembling burns.	Widespread	Severe itching, burning, faster heart rate, temperature changes
Nail Psoriasis	Affects the nails, often associated with psoriatic arthritis. Various nail changes.	Nails	Pitting, tenderness, separation, color changes, thickening
Psoriatic Arthritis	Combines psoriasis and arthritis. Joint pain and swelling, often with nail changes.	Joints, Nails	Joint pain, swelling, sausage-like fingers/toes, warm joints

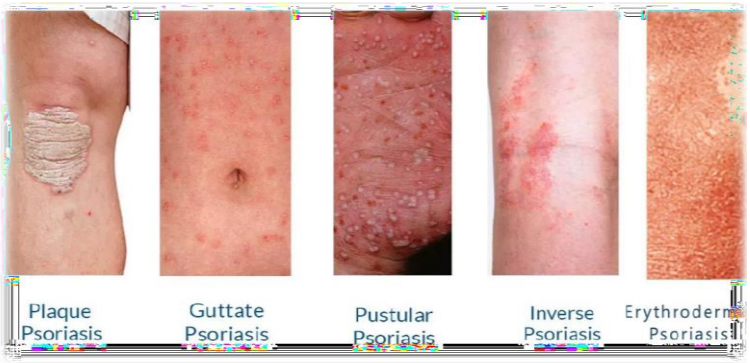

Figure 2: Types of Psoriasis.[11]

ETIOLOGY[9,12,13]

Psoriasis is a skin condition characterized by the excessive growth of keratinocytes in the epidermis, resulting in a faster turnover of skin cells. While the exact cause of this abnormal cell turnover is not fully understood, it is believed to involve a mix of genetic, environmental, and immunological factors. Let's explore these factors in more detail:

Environmental factors

Many factors besides stress have also been observed to trigger exacerbations, including cold, trauma, infections (eg, streptococcal, staphylococcal, human immunodeficiency virus), alcohol, and drugs (eg, iodides, steroid withdrawal, aspirin, lithium, beta-blockers, botulinum A, antimalarials). One study showed an increased incidence of psoriasis in patients with chronic gingivitis. Satisfactory treatment of the gingivitis led to improved control of the psoriasis but did not influence longterm incidence, highlighting the multifactorial and genetic influences of disease.Hot weather, sunlight, and pregnancy may be beneficial, although the latter is not universal. Perceived stress can exacerbate psoriasis. Some authors suggest that psoriasis is a stress-related disease and offer findings of increased concentrations of neurotransmitters in psoriatic plaques.

Genetic factors

Psoriasis is strongly influenced by genetics, with specific gene loci identified, although the exact triggering events are often unclear but are believed to be immunological. Many individuals first develop psoriasis lesions after upper respiratory tract infections. Certain human leukocyte antigen (HLA) alleles, including HLA-Cw6, are associated with psoriasis, with some families displaying autosomal dominant inheritance. Recent research has highlighted the significance of the deletion of two late cornified envelope (LCE) genes, LCE3C and LCE3B, in susceptibility to psoriasis across various populations. Additionally, obesity is linked to psoriasis, but whether it results from weight alone, genetic predisposition, or a combination of both remains uncertain. Some individuals experience psoriasis onset or improvement with changes in weight, underlining the intricate interplay of genetics.

Immunologic factors

Psoriasis is an autoimmune disease linked to excess T-cell activity and elevated TNF-α levels. TNF-α inhibitors are often effective treatments. Streptococcal superantigens can trigger psoriasis, and new drugs target lymphocyte function. Interestingly, HIV can worsen psoriasis despite T-cell reduction due to CD8 T-cell overactivity or direct HIV effects on keratinocytes. Opportunistic infections in HIV may increase superantigen exposure, amplifying psoriasis-related cascades. Additionally, guttate psoriasis can follow events like streptococcal infections or medication changes.

RISK FACTORS[15,17]

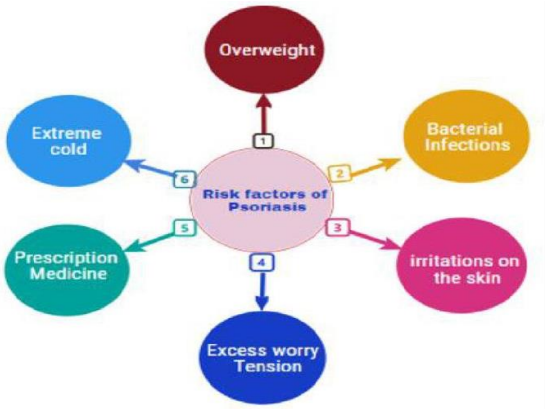

Figure 3: Risk factors of Psoriasis.

PSORIASIS TRIGGERED BY[16]

Many people who are predisposed to psoriasis (genetic) may be free of symptoms for years until the disease is triggered by some environmental factor. Common psoriasis triggers include.

1) Infections, such as strep throat or skin infections
2) Weather, especially cold, dry conditions
3) Injury to the skin, such as a cut or scrape, a bug bite, or a severe sunburn
4) Smoking and exposure to second hand smoke
5) Heavy alcohol consumption
6) Certain medications - including lithium, high blood pressure drugs, antimalarial drugs, interferons, imiquimod, angiotensin-converting enzyme inhibitors,terbinafine, Tetracycline, nonsteroidal anti-inflammatory drugs (NSAIDs), fibrate drugs.

7) Rapid withdrawal of oral or injected corticosteroids.

PATHOGENESIS OF PSORIASIS[17-24]

Pathogenesis of vascular changes in psoriasis

Vascular endothelial growth factor (VEGF) plays a critical role in the pathologic angiogenesis seen in psoriasis and other chronic inflammatory skin conditions. In psoriasis, there is enhanced expression of VEGF by epidermal keratinocytes and an increase in VEGF receptors on dermal microvessels. This abnormal angiogenesis process is associated with an increase in mast cells and elevated leukocyte rolling and adhesion in skin venules.

Early research by Braverman in the 1970s revealed that in psoriasis, the capillary loops in dermal papillae change from arterial to venous capillary structures. Treatment with Goeckerman therapy can reverse this change, transforming the capillary loops back to arterial structures typical of normal skin.

Psoriatic skin is characterized by a four-fold increase in the endothelial microvascular bed, indicating the significance of angiogenesis in the condition. Keratinocytes in psoriatic lesions are identified as a source of pro-angiogenic cytokines, including VEGF. Other cytokines, such as endothelial cell-stimulating angiogenesis factor (ESAF), tumor necrosis factor-alpha (TNF-α), and platelet-derived growth factors (PDGF), also play roles in angiogenesis.

Furthermore, psoriatic skin exhibits microvascular hyperpermeability and angioproliferation. VEGF, specifically, enhances microvascular permeability. Transforming growth factor alpha (TGF-alpha), which is overexpressed in psoriatic epidermis, induces VEGF gene expression in keratinocytes, leading to vascular hyperpermeability and angiogenesis. This complex interplay of factors contributes to the pathogenesis of psoriasis.

Role of keratinocytes in the pathogenesis of psoriasis

Epidermal Hyperplasia and Abnormal Keratinocyte Maturation: Psoriasis is associated with epidermal hyperplasia (thickening of the outer skin layer) and abnormal maturation of keratinocytes (skin cells). These factors contribute to the formation of characteristic scaly plaques.

Cytokines: The passage suggests that the interaction between keratinocytes and immune cells is mediated by cytokines. Cytokines are signaling molecules produced by both keratinocytes and leukocytes (white blood cells) and play a role in the inflammatory process seen in psoriasis.

Gamma Interferones and Interleukins (IL): The cytokine gamma interferon, as well as interleukin-6, have been found to be elevated in psoriatic plaques. These cytokines are involved in the immune response and may contribute to the abnormal proliferation of keratinocytes.

Transforming Growth Factor-Alpha (TGF-Alpha): Increased levels of TGF-alpha have been detected in active psoriatic plaques. This growth factor may play a role in the overexpression of IL-6 in psoriatic tissue, linking keratinocyte proliferation with immune activation and inflammation.

Roles of Keratinocytes: The passage emphasizes the importance of keratinocytes in the pathogenesis of psoriasis. They are not just passive cells but actively participate in the disease process.

Polymorphous nature of Psoriasis: Psoriasis is described as a polymorphous disease with complex interactions between susceptibility genes, immune mechanisms, and other factors. It's not solely an immune disorder or a primitive skin cell change but likely results from a combination of abnormalities in both compartments.

Innate Immunity: While T-cells are important in psoriasis development, innate immunity (the body's initial defense against pathogens) also plays a role. Many psoriasis susceptibility genes are linked to the immune system.

Biochemical Basis: Anomalies in protein expression are suggested as the basis for abnormal differentiation and hyper proliferation of keratinocytes in psoriatic lesions. Several markers of abnormal keratinocyte differentiation have been identified, and various biochemical factors, including growth factors, have been associated with the overproduction of keratinocytes in psoriatic skin.

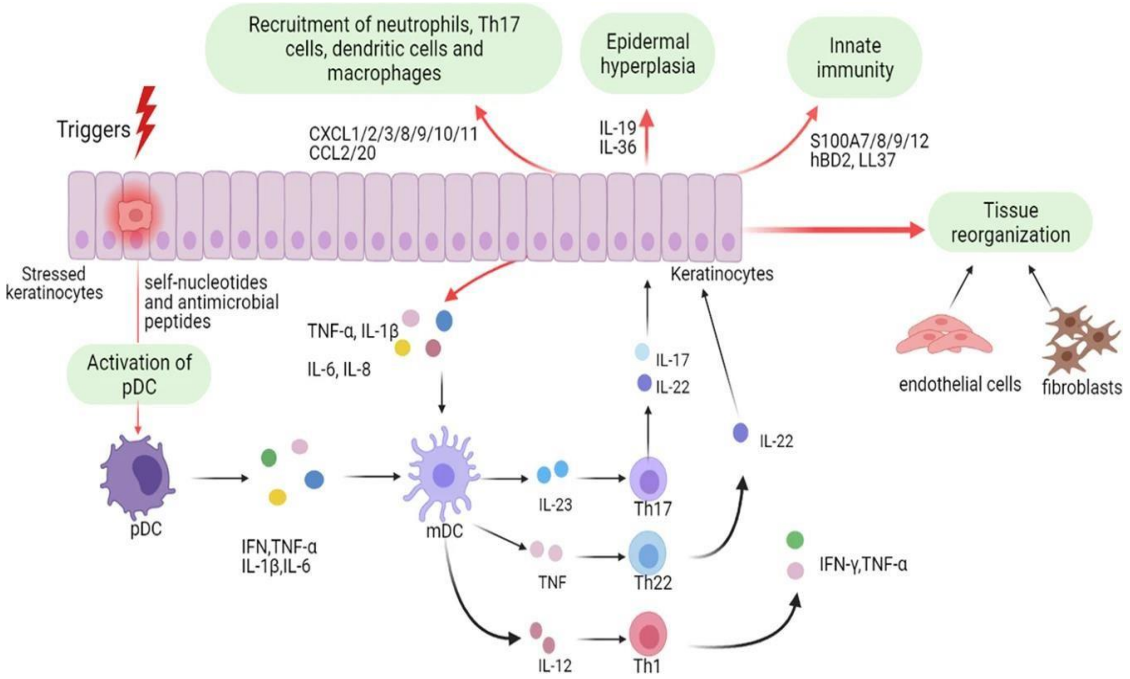

Figure 4: Roles of keratinocytes in pathogenesis of Psoriasis.

Cytokines and chemokines in the pathogenesis of psoriasis

The pathogenesis of psoriasis is closely linked to the role of cytokines and chemokines, which are signaling molecules involved in the immune response.

T cells, particularly T-helper type 1 (TH1) cells, are central to the development of psoriasis. Activated CD4+ and CD8+ T lymphocytes release TH1-type cytokines. Key TH1 cytokines implicated in psoriasis pathogenesis include:

Interferon Gamma (INF- y): IFN-y may inhibit the programmed cell death (apoptosis) of keratinocytes by stimulating the expression of anti-apoptotic proteins like Bcl-x. This contributes to the excessive keratinocyte proliferation seen in psoriatic lesions.

Interleukin-2 (IL-2):IL-2 stimulates the growth of T lymphocytes and is associated with psoriasis flares.

Tumour Necrosis Factor- alpha (TNF-α): TNF-α triggers the release of proinflammatory cytokines from T lymphocytes and macrophages, as well as chemokines from macrophages. It also induces the expression of adhesion molecules on vascular endothelial cells, promoting inflammation.

There is evidence of a TH1 cytokine profile in psoriatic lesions, including IFN-γ, TNF-α, and IL-12. These cytokines contribute to the production of adhesion molecules like ICAM-1 and chemotactic polypeptides like IL-8.

Chemokines and their receptors also play a role in psoriasis immunopathogenesis. Specific chemokines, such as CCL17, MIG, CXCL9, and RANTES (CCL5), are involved in the disease process.

Moreover, psoriasis is associated with increased levels of nitric oxide, which may contribute to angiogenesis, the formation of new blood vessels. Several growth factors are elevated in psoriatic plaques, including TGF-α, IGF-1, KGF, VEGF, NGF, amphiregulin, and IL-20. These factors further contribute to the complex pathogenesis of psoriasis, involving not only immune cells but also the regulation of growth and inflammation within the skin.

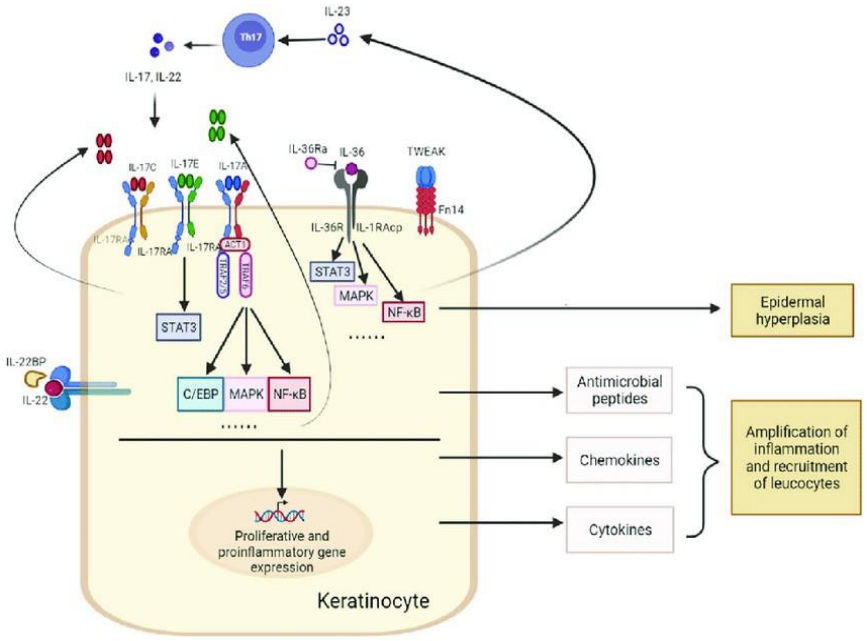

Figure 5: Roles of chemokines in pathogenesis of Psoriasis.

SIGNS AND SYMPTOMS[25]

Signs and symptoms of psoriasis may include the following:

- Worsening of a long-term erythematous scaly area
- Sudden onset of many small areas of scaly redness
- Pain (especially in erythrodermic psoriasis and in some cases of traumatized plaques or in the joints affected by psoriatic arthritis)
- Pruritus (especially in eruptive, guttate psoriasis)
- Afebrile (except in pustular or erythrodermic psoriasis, in which the patient may have high fever)
- Dystrophic nails, which may resemble onychomycosis
- Long-term, steroid-responsive rash with recent presentation of joint pain
- Joint pain (psoriatic arthritis) without any visible skin findings
- Conjunctivitis or blepharitis

COMPLICATIONS[26,27]

Psoriasis is a chronic autoimmune disease that extends beyond the skin and can lead to numerous physical, psychological, and systemic complications. Common skin-related problems include itching, pain, dryness, cracking, bleeding, and an increased risk of infections due to a compromised skin barrier. Approximately 30% of patients may develop psoriatic arthritis, which causes joint pain, stiffness, swelling, and, if untreated, permanent joint damage. Psoriasis is also associated with significant mental health challenges, including depression, anxiety, low self-esteem, social isolation, and reduced quality of life. In addition, patients face an increased risk of cardiovascular diseases, metabolic syndrome, obesity, hypertension, dyslipidemia, insulin resistance, type 2 diabetes, liver and kidney disorders, eye diseases such as conjunctivitis and uveitis, and certain malignancies. Severe psoriasis, particularly involving the genital area, can negatively affect sexual health and interpersonal relationships. The disease may also impair work productivity and daily activities, especially when joint involvement limits mobility. Furthermore, some systemic treatments can cause adverse effects, including increased susceptibility to infections and organ toxicity. Psoriasis may present additional challenges during pregnancy, requiring careful management to ensure maternal and fetal safety. Therefore, regular monitoring and a multidisciplinary approach involving dermatologists, rheumatologists, and mental health professionals are essential for effective disease management and prevention of complications.

DIAGNOSIS[13,19,26,27]

Psoriasis is a chronic inflammatory skin disorder that presents in several clinical forms. The most common type, chronic stationary or plaque psoriasis, typically affects the scalp, elbows, knees, trunk, and other extensor surfaces. Other variants include guttate psoriasis, which often develops suddenly after a streptococcal infection; inverse psoriasis, which affects skin folds and is frequently mistaken for fungal infections; pustular psoriasis, characterized by pustules on the palms, soles, or throughout the body; and erythrodermic psoriasis, a severe form involving widespread redness and scaling over most of the body. Additional manifestations include scalp, nail, oral, eruptive, napkin, and linear psoriasis, as well as psoriatic arthritis, which affects 10–30% of patients and commonly involves the joints of the hands and feet. Diagnosis is based on clinical examination, which typically reveals scaling, erythematous papules and plaques, along with possible ocular and musculoskeletal involvement. Laboratory findings are generally nonspecific, although elevated uric acid levels and inflammatory markers may occur in severe forms. Fungal studies, pustule analysis, and conjunctival

examinations may aid diagnosis and rule out other conditions. Differentiation from rheumatoid arthritis and gout is facilitated by the absence of their characteristic laboratory findings. The impact of psoriasis on a patient's well-being is often assessed using the Dermatology Life Quality Index (DLQI), which measures the disease's effect on quality of life.

Severity	BSA	PASI score
Mild	less than 3%	0–4
Moderate	3–10%	5–9
Severe	more than 10%	10+

Consider obtaining the following baseline laboratory studies in patients being initiated on systemic therapies (eg, immunologic inhibitors):

- Joint radiographs: Can facilitate the diagnosis of psoriatic arthritis
- Bone scans: Can identify joint involvement early
- Dermatologic biopsy: Can be used to make the diagnosis when some cases of psoriasis are difficult to recognize.
- Radiographs of affected joints: Can be helpful in differentiating types of arthritis

EXISTING TREATMENTS[28,29,30,31,32]

A range of topical and systemic therapies exist for psoriasis, tailored based on factors like concurrent health conditions, adverse effects, quality of life, self-care capability, and financial feasibility.

Treatment typically starts with cost-effective options and progresses to more advanced therapies. Modalities include topical treatments (e.g., corticosteroids, vitamin D analogs), systemic medications (e.g., methotrexate, biologics), and phototherapy (UVB, PUVA). The choice is personalized to achieve effective treatment while ensuring patient compliance.The treatment modalities are as follows:

TOPICAL TREATMENT

Corticosteroids

These medications are commonly prescribed for mild to moderate psoriasis, working by slowing down cell turnover and reducing inflammation and itching by suppressing the immune system. Low-potency corticosteroid ointments are often recommended for sensitive areas like the face or skin folds, as well as for treating extensive damaged skin patches. However, they can lead to adverse effects such as skin thinning, telangiectasia, and systemic side effects like diabetes, hypertension, and HPA suppression. Examples of corticosteroids used include Clobetasol propionate 0.05%, Amcinonide 0.1%, Betamethasone dipropionate, Betamethasone valerate in various concentrations, Halcinonide 0.1%, Desoximetasone 0.25%, and Mometasone furoate.

Vitamin D Analogues Vitamin D analogues

(Cacipotriol, Tacalcitol, Calcitriol, and Calcipotriene) have become significant alternatives to topical corticosteroids for long-term psoriasis therapy. They bind to the Vitamin D Receptor in the cytoplasm, then move into the nucleus to bind to the nuclear receptor, initiating the transcription of vitamin D responsive genes. These genes regulate cell differentiation and reduce cell proliferation and inflammation linked with psoriasis. Despite causing occasional peri-lesional irritation and erythema, they are generally considered safe. However, they might rarely raise serum and urine calcium levels, so the total weekly concentration should not exceed 100 grams. Calcitriol is more potent, while calcipotriene is the most established option, showing minimal impact on calcium balance. Clinical trials often demonstrate that combining vitamin D with corticosteroids is more effective than using either alone. Calcipotriene is available as a 0.005% cream, ointment, and scalp solution for twice-daily use, and is also combined with betamethasone dipropionate 0.064% in ointment and suspension formulations.

Anthralin (Dithranol)

Anthralin is a synthetic derivative of a natural substance present in goa powder from the araroba tree. Its action involves slowing down the growth of keratinocytes. While it can be cautiously used in both children and adults, prolonged exposure or contact with the eyes or genital area may result in contact dermatitis. Typically, Anthralin is left on the skin for 5 to 20 minutes before washing it off to prevent clothing from developing brown discoloration. Additionally, it can be combined with salicylic acid for improved stability when compounded.

Coal tar

Coal tar is one of the oldest topical therapies utilized either alone or in combination with other treatments for psoriasis. Its polycyclic aromatic hydrocarbons heighten skin sensitivity to UV light, though the exact mechanism of action remains unclear. Coal tar exhibits anti-inflammatory, anti-proliferative, and potent anti-itch properties. However, its unpleasant odor, tendency to stain, and potential mutagenicity have reduced patient compliance. To enhance compliance, non-staining and washable formulations like lotions and shampoos are available, either as standalone treatments or combined with other active agents.

Topical Calcineurin Inhibitor

Topical Calcineurin inhibitors like Tacrolimus 0.1% ointment and Pimecrolimus 1.0% cream are applied twice daily, especially for sensitive areas like the face and folds in psoriasis treatment. They're also used as steroid-sparing options or for long-term management. Although generally safe with minimal systemic absorption and skin atrophy risk, concerns exist due to rare reported cases globally linking them to lymphoma and skin neoplasms. Despite this, they're often recommended for children over 2 years old. Healthcare providers should be aware of ongoing debates regarding their safety.

Intralesional Corticosteroid Triamcinolone Acetonide
Intralesional injections effectively deliver a small amount of corticosteroid to targeted areas in psoriasis treatment, such as the proximal nail fold for nail psoriasis. A fine-gauge insulin needle is recommended for precision. Anesthesia may be optional, based on patient preference, especially for deeper injections targeting subungual hyperkeratosis or onycholysis. In mild psoriasis cases, where a few plaques resist topical treatments, intralesional steroids can be beneficial. To minimize skin atrophy risk, the lowest effective dose of triamcinolone acetonide (3–5 mg/mL) is advised. These injections, especially into the nail bed, can be painful, so they should be spaced 6 to 8 weeks apart and used only when necessary.

Salicylic acid
Salicylic acid, a potent keratolytic agent, is frequently combined with corticosteroids and calcineurin inhibitors in psoriasis treatment. Its combination with calcineurin inhibitors enhances drug absorption into psoriasis plaques. However, using salicylic acid alone for scalp psoriasis might cause temporary telogen hair shedding, which typically resolves upon discontinuation. Concentrations exceeding 10% on large body areas should be avoided due to potential side effects like oral mucosal burn, headaches, and nausea. Research suggests that salicylic acid, when combined with calcineurin inhibitors, vitamin D analogues, or corticosteroids, acts as a penetration enhancer, aiding better skin permeation and sustained release of medications.

SYSTEMIC TREATMENT
Biologics
These are the molecules, which are developed for target-based therapy. They have a more precise action and side effects are thought to be less as compared to the broad traditional therapies. These agents act on the varied steps of the pathogenesis of the psoriasis and are divided into various groups. The listed medications—Adalimumab (Humira), Etanercept (Enbrel), Infliximab (Remicade), Ustekinumab (Stelara), Secukinumab (Cosentyx), Ixekizumab (Taltz), Brodalumab (Siliq), Guselkumab (Tremfya), Tildrakizumab (Ilumya), and Risankizumab (Skyrizi)—are biologics used to treat autoimmune conditions like arthritis and psoriasis by targeting specific molecules in the immune system. They are prescribed when conventional treatments are ineffective or unsuitable for the patient's condition

Phototherapy
Ultraviolet (UV) light application is considered suitable for inducing remission in cases of moderately severe psoriasis or when psoriasis hasn't responded adequately to topical treatments alone. However, it is not recommended for long-term maintenance therapy due to the increased risk of skin cancer associated with prolonged use. The most frequently utilized type of UV therapy currently is narrow-spectrum UVB therapy, specifically at 311 nanometers.

An alternative effective method involves UVA irradiation after the intake of a photosensitizer, such as psoralen, which can be administered orally, added to bathwater, or applied as a cream. This combination therapy, known as PUVA (psoralen + UVA), has shown high effectiveness in managing psoriasis.

A) Narrowband ultraviolet B (NB-UVB) and excimer laser
NB-UVB light (311 nm) and excimer lamp/laser (308 nm) are two highly effective phototherapies considered primary treatments for stable plaque psoriasis. Both therapies demonstrate comparable efficacy in clearing psoriatic skin lesions. NB-UVB is often chosen after topical agents have proven ineffective. Phototherapy involves repeated exposure of the skin to UV light, with the treatment's UV radiation inducing apoptosis in T lymphocytes and keratinocytes in the epidermis to manage psoriatic lesions.

NB-UVB treatment is a cost-effective option and is particularly preferred for specific patient groups like those with HIV, internal malignancies, or during pregnancy, where systemic immune reactions could be contraindicated when using other psoriasis treatments.

The excimer laser, more effective than the excimer lamp, is recommended by the AAD for PASI < 10. While NB-UVB phototherapy can effectively treat various parts of the body, challenging areas such as the nose, ear, and eyelids pose difficulties in psoriasis treatment. Therefore, the excimer laser stands out as a better option for targeting these specific areas. Beyond its potency and effectiveness, psoriasis patients prefer the excimer laser due to its potential for long-term remission of psoriasis.

B) Psoralen plus ultraviolet A (PUVA)
PUVA (Psoralen + UVA) phototherapy is an effective treatment for chronic plaque psoriasis when other options like topical medications and NB-UVB (Narrowband UVB) phototherapy prove insufficient. Psoralens, derived from plants like Psoralea corylifolia (Babchi), are light-sensitive compounds used in PUVA therapy to sensitize the skin to UVA light.

Combining psoralens with UVA light significantly reduces psoriasis symptoms by approximately 75% to 80% in many cases. However, due to its side effects and potential long-term risks, PUVA therapy is usually considered a secondary treatment, with concerns about skin damage and an increased risk of skin cancer with prolonged use.

When NB-UVB treatment fails or leads to relapses, particularly in conditions like pustular psoriasis or pityriasis rubra pilaris, introducing PUVA therapy might be considered. Psoralen sensitization helps boost the therapeutic effects of UVA light in these situations.

Application of psoralens can lead to various side effects,

including skin reactions like burning, itching, and changes in pigmentation. Nausea is also reported as a possible side effect in individuals undergoing PUVA therapy.

Healthcare professionals should carefully weigh the risks and benefits of PUVA therapy for each patient, including the choice and administration of psoralens. Regular monitoring and follow-up during and after PUVA treatment are crucial to manage potential side effects and evaluate treatment effectiveness.

Natural treatments of psoriasis[33]

Herbal medicines are often considered to have fewer side effects compared to synthetic drugs, offering easily accessible and user-friendly treatment options. Presently, herbal resources are playing a crucial role in managing skin and inflammatory diseases.

Table 1: Summary of Herbal Plants Used in Psoriasis Management.

Plant (Scientific Name)	Common Name	Active Constituents	Key Findings
Capsicum annuum / Capsicum frutescens	Cayenne	Capsaicin	May reduce inflammation, angiogenesis, and keratinocyte proliferation
Aloe vera	Aloe Vera	Anthraquinones, Acemannan, Salicylic acid	Clinical trial showed significant plaque clearance
Silybum marianum	Milk Thistle	Silymarin	May improve psoriasis through liver and inflammatory pathway modulation
Angelica sinensis	Dong Quai	Psoralens	Two-thirds of patients reportedly achieved complete relief
Heracleum sphondylium	Hogweed	Psoralens	Insufficient efficacy and safety data
Matricaria recutita	Chamomile	Chamazulene, Quercetin, Apigenin	Potential improvement in psoriatic inflammation
Melaleuca alternifolia	Tea Tree Oil	Terpinen-4-ol, α-terpineol, α-pinene	No direct psoriasis studies; potential benefit requires investigation
Gaultheria procumbens	Wintergreen	Methyl salicylate	Potential benefit; risk of systemic salicylate toxicity
Ulmus rubra	Slippery Elm	Mucilage	Small study showed improvement in PASI scores
Curcuma longa / Curcuma domestica	Turmeric	Curcuminoids, Volatile oils	Improved psoriasis markers without major adverse effects
Mahonia aquifolium	Oregon Grape	Berberine, Berbamine, Oxyacanthine	Commonly used for psoriatic plaques
Alpinia galanga	Galangal	Various phytochemicals	Demonstrated anti-psoriatic molecular effects
Thespesia populnea	Portia Tree	Flavonoids, Triterpenoids, Tannins	Animal study showed 25% increase in orthokeratosis
Annona squamosa	Custard Apple	Various phytochemicals	Experimental anti-psoriatic activity reported
Smilax china	China Root	Quercetin	Significant anti-psoriatic activity in cell-line studies

THERAPY FOR DIFFERENT TYPES OF PSORIASIS[35,36]

Type of Psoriasis	Characteristics	Treatment
Plaque Psoriasis	Most common form; raised, scaly plaques	Reduce inflammation, slow skin cell growth, control plaques
Guttate Psoriasis	Small, red, teardrop-shaped lesions	Relieve itching and inflammation, clear lesions
Inverse Psoriasis	Smooth, inflamed lesions in skin folds	Reduce inflammation and discomfort
Pustular Psoriasis	Pus-filled blisters with inflamed skin	Eliminate pustules, reduce inflammation and discomfort
Erythrodermic Psoriasis	Severe, widespread redness and scaling	Control severe symptoms and prevent complications
Nail Psoriasis	Nail pitting, discoloration, thickening	Improve nail appearance and function
Psoriatic Arthritis	Psoriasis with joint inflammation and pain	Reduce pain, inflammation, and joint damage

PREVALENCE IN INDIA[35, 36]

Studies conducted in India focusing on psoriasis prevalence have predominantly relied on hospital-based data. These studies, spanning various regions, have provided comparative insights into the incidence and demographic characteristics of individuals affected by psoriasis.

A comprehensive study by Okhandiar et al. encompassed multiple medical colleges across cities such as Dibrugarh, Calcutta, Patna, Darbhanga, Lucknow, New Delhi, and Amritsar. Their findings revealed 3varying incidence rates of psoriasis among skin patients, ranging from 0.44% to 2.2%, with an overall incidence of 1.02%. Notably, Amritsar exhibited a higher incidence at 2.2%, speculated to be influenced by environmental factors like temperature extremes, dietary habits, and possible genetic variations unique to that region compared to Eastern India. They observed a significantly higher male-to-female ratio (2.46:1) across these areas, without a definitive explanation. The age group most affected was between 20 and 39 years, with a similar average age of onset between males and females. In North India, Bedi conducted a study reporting a psoriasis prevalence of 0.8% among skin patients, although the sample size was limited. The male-to-female sex ratio remained high at 2.5:1. Interestingly, this study noted that females tended to experience psoriasis at a younger age compared to males. In a subsequent study by Bedi with a larger sample size of 530 subjects, the prevalence among dermatology outpatients increased to 2.8%, while the male-to-female ratio remained consistent. From a tertiary healthcare center in North India, it was observed that psoriasis patients accounted for 2.3% of total dermatology outpatients. Among these patients, 67% were men and 33% were women, maintaining a male-to-female ratio of 2.03:1. Patients ranged in age from infancy to the eighth decade, with an average age of 33.6 years. Notably, children constituted 4.4% of total psoriasis patients. Women tended to experience onset at a slightly younger age (27.6 years) compared to men (30.9 years).

In summary, psoriasis prevalence in India varies between 0.44% and 2.8%, with a higher occurrence among males. Most patients tend to present with the condition during their third or fourth decade of life. However, these studies lack standardized diagnostic criteria, and there is insufficient data regarding the longitudinal trends of the disease.

Table: Marketed Formulations Available for the Management of Psoriasis[37-40]

Drug/Active Ingredient	Brand Name (Example)	Dosage Form	Route of Administration	Indication in Psoriasis
Calcipotriol (Calcipotriene)	Dovonex®	Cream, Ointment, Scalp Solution	Topical	Plaque psoriasis
Calcipotriol + Betamethasone Dipropionate	Daivobet® / Taclonex®	Ointment, Gel, Foam	Topical	Mild to moderate plaque psoriasis
Tazarotene	Tazorac®	Cream, Gel	Topical	Plaque psoriasis and nail psoriasis
Coal Tar	Psorent® (various brands)	Shampoo, Ointment, Lotion	Topical	Scalp and chronic plaque psoriasis
Salicylic Acid	Various brands	Ointment, Gel, Shampoo	Topical	Removal of scales in psoriasis
Anthralin (Dithranol)	Drithocreme®	Cream	Topical	Chronic plaque psoriasis
Clobetasol Propionate	Clobex®	Shampoo, Spray, Lotion, Cream	Topical	Moderate to severe plaque psoriasis
Hydrocortisone	Various brands	Cream, Ointment	Topical	Mild psoriasis and sensitive areas
Methotrexate	Trexall®	Tablet, Injection	Oral/Parenteral	Moderate to severe psoriasis
Cyclosporine	Neoral® / Sandimmune®	Capsule, Oral Solution	Oral	Severe plaque psoriasis
Acitretin	Soriatane®	Capsule	Oral	Severe psoriasis and pustular psoriasis
Apremilast	Otezla®	Tablet	Oral	Moderate to severe plaque psoriasis and psoriatic arthritis
Etanercept	Enbrel®	Prefilled Syringe, Injection	Subcutaneous	Moderate to severe psoriasis
Adalimumab	Humira®	Injection	Subcutaneous	Moderate to severe plaque psoriasis
Infliximab	Remicade®	Intravenous Infusion	Intravenous	Severe plaque psoriasis

Ustekinumab	Stelara®	Injection	Subcutaneous	Moderate to severe psoriasis
Secukinumab	Cosentyx®	Injection	Subcutaneous	Moderate to severe plaque psoriasis
Ixekizumab	Taltz®	Injection	Subcutaneous	Moderate to severe plaque psoriasis
Guselkumab	Tremfya®	Injection	Subcutaneous	Moderate to severe plaque psoriasis
Risankizumab	Skyrizi®	Injection	Subcutaneous	Moderate to severe plaque psoriasis
Bimekizumab	Bimzelx®	Injection	Subcutaneous	Moderate to severe plaque psoriasis
Tapinarof	Vtama®	Cream	Topical	Plaque psoriasis in adults
Roflumilast	Zoryve®	Cream, Foam	Topical	Plaque and scalp psoriasis

Future Scope[41]

The future of psoriasis management is expected to be driven by advances in precision medicine, biotechnology, and nanotechnology, leading to more effective, safer, and patient-centered therapies. Emerging research is focused on developing targeted drug delivery systems, such as liposomes, solid lipid nanoparticles, nanocapsules, microneedles, and other nanocarriers, to enhance drug penetration, improve bioavailability, and reduce systemic adverse effects. The continued evolution of biologic therapies targeting specific inflammatory pathways, including interleukins and cytokines involved in psoriasis pathogenesis, is likely to provide improved long-term disease control with fewer side effects. Personalized medicine based on genetic, immunological, and biomarker profiling may enable clinicians to tailor treatments according to individual patient characteristics, maximizing therapeutic efficacy while minimizing treatment failure. Additionally, novel therapeutic approaches such as gene therapy, RNA-based therapeutics, stem cell therapy, microbiome modulation, and artificial intelligence-assisted treatment selection are being actively explored. Digital health technologies and teledermatology are also expected to improve disease monitoring, treatment adherence, and access to specialized care. Although these innovations show considerable promise, further preclinical studies, large-scale clinical trials, and regulatory evaluations are required to establish their safety, efficacy, and cost-effectiveness. Overall, future advancements are anticipated to transform psoriasis management by providing more targeted, durable, and personalized treatment options that significantly improve patient outcomes and quality of life.

CONCLUSION

Psoriasis remains a challenging chronic inflammatory skin disease that significantly affects patients' quality of life and requires long-term management. While conventional therapies such as topical agents, phototherapy, systemic drugs, and biologics have demonstrated therapeutic benefits, their effectiveness is often limited by issues including poor skin penetration, systemic toxicity, adverse effects, and reduced patient adherence. Recent advances in nanotechnology-based drug delivery systems have opened new avenues for improving psoriasis treatment. Innovative carriers such as liposomes, solid lipid nanoparticles, nanocapsules, nanospheres, and pro-dispersion formulations have shown the potential to enhance drug stability, bioavailability, targeted delivery, and controlled release while minimizing side effects. Furthermore, the incorporation of therapeutic agents such as cyclosporin, tazarotene, and tretinoin into these advanced delivery platforms has demonstrated improved efficacy and safety profiles. Ongoing research continues to explore novel nanomedicine approaches and personalized treatment strategies tailored to individual patient needs. Although these technologies show great promise, further preclinical and clinical studies are necessary to establish their long-term safety, effectiveness, and regulatory acceptance. Overall, the integration of nanotechnology into psoriasis therapy represents a significant advancement that may lead to more efficient, safer, and patient-friendly treatment options in the future.

REFERENCES

1. World Health Organization. “Global report on psoriasis”. World Health Organization, 2016, 5-6. [Google Scholar]
2. Hay SI, Abajobir AA, Abate KH. "A systematic analysis for the Global Burden of Disease Study." 2016; 390: 1260–344.
3. Antony Raharja, Satveer K Mahil, Jonathan N Barker. “Psoriasis a: Brief overview”. Clinical Medicine. 2021; 21(3): 170–173.
4. Meenan FO, "A note on the history of psoriasis". Irish Journal of Medical Science, 1995; 30(351): 141–2.
5. Benedek TG, "Psoriasis and psoriatic arthropathy: historical aspects: part II". Journal of Medical Rheumatology. 2013, 19 (5): 267–71.
6. Gruber F, Kastelan M, Brajac I. "Psoriasis treatment-yesterday, today, and tomorrow". Acta Dermatoven -erologica Croatica, 2004; 12(1): 30-4.
7. Benedek TG. "Psoriasis and psoriasis arthropathy: historical aspects."Journal of clinical Rheumatology, 2013; 19(5): 267-71.
8. Gibbs S. "Skin dsease & socio -economic conditions in rura Afrca: Tanzania." International Journal of

Dermatology, 1996; 35(9): 633–9.
9. Rajeev Patrick Das, Arun Kumar Jain, V. Ramesh, "Current concepts in the pathogenesis of psoriasis", Indian Journal of Dermatology, 2009; 54(1): 7-12.
10. Asha G. Pardasani, Steven R. Feldman, Adele R. Clark. "Treatment of Psoriasis: An Algorithm-Based Approach for Primary Care Physicians". American Family Physician, 2000; 61(3): 725-733.
11. https: //ik.imagekit.io/4wkn9yhcxgvwt/OfTLLtdDyG9JyTNwzFbvXL8iBi5hhEvYcRnF3g94.jpeg accessed on 04 November, 2023.
12. Stern RS, Nijsten T, Feldman SR, Margos DJ, Rostad T. "Psoriasis is common, carries a substantial burden even when not extensive, & associated with widespread treatment dissatisfaction." J Investg Dermatol Symp Proc., 2004; 9(2): 136–9.
13. Jacquiline Habashy, William D James. "Psoriasis". https: //emedicine.medscape.com/article/1943419-overview?form=fpf . Sept 14, 2022.
14. Keller JJ. "The Effect of Chronic Periodontitis & its Treatment."Br J Dermatol 2012.
15. Sarfaraz Ahmed. https: //www.researchgate.net/figure/Risk-factors-of-15Psoriasis_fig1_336079978 Accessed on 04 November, 2023
16. Woodrow JC, llchysyn A. "HLA antigen in psoriasis and psoriasis arthritis." J Med Genet, 1985; 22(6): 492-5
17. Riveira-Munoz E, He SM, Escaramìs G. "Meta-analysis confirms the LCE3C- LCE3B Deletion as a Risk Factor for Psoriasis in Several Ethnic Group & Find Interaction with HLA-Cw6." J Invest Dermatol., 2011; 131(5): 1105-9.
18. Braverman IM, Yen A. "Ultrastructure of the capillary loops in the dermal papillae of psoriasis." J Invest Dermatol. 1977; 68(1): 53-60
19. Liew SC, Das-Gupta E, Chakravarthi S, Wong SF, Lee N, Safdar N, Jamil A. "Differential expression of the angiogenesis growth factors in psoriasis vulgaris." BMC Res Notes, 2012; 201.
20. Detmar M, Brown LF, Claffey KP, Yeo KT, Kocher O, Jackman RW, Berse B, Dvorak HF. "Overexpression of vascular permeability factor/vascular endothelial growth factor and its receptors in psoriasis." J Exp Med 1994; 180(3): 1141-6.
21. Gottlieb AB. "Immunologic mechanisms in psoriasis." J Invest Dermatol., 1990; 95(5): 18-19.
22. Julien D. "Pathogenesis of psoriasis. Ann Dermatol Venereol." 2012; 139 (2): 68-72.
23. Grove T. "The Pathogenesis of Psoriasis: Biochemical Aspects. Biological & Biomedical Sciences."2001.
24. Krueger G, Ellis CN. "Psoriasis—recent advances in understanding its pathogenesis and treatment."J Am Acad Dermatol., 2005; 53: 94-100
25. Nickoloff BJ, Nestle FO. "Recent insights into the immunopathogenesis of psoriasis provide new therapeutic opportunities". J Clin Invest, 2004; 113(12): 1664–75.
26. Krueger JG. "The immunologic basis for the treatment of psoriasis with new biologic agents." J Am Acad Dermatol., 2002; 46(1): 1-23.
27. Hani A. Al-Shobaili and Muhammad Ghaus Qureshi. "Psoriasis: Types, Causes and Medication".1st Edn; Intech open publishing, 2013, 428.
28. Siddhartha Dutta, Shalini Chawla, Sahil Kumar."Psoriasis: A Review of Existing Therapies and Recent Advances in Treatment". J Rational Pharmacother Res., 2018; 4(1): 13-17.
29. Adam Felman,"Tracing the path Psoriasis" Medical News Today.2023.
30. Erine A. Kupetsky, Matthew Keller."Psoriasis Vulgaris: An Evidence-Based Guide for Primary Care". Jouranal of the American Board of Family Medicine, 2013. 26 (6): 787-801.
31. Mrowietz U, Reich K." Psoriasis – New Insights into Pathogenesis and Treatment "Dtsch Arztebl Int., 2009; 106(1-2): 11-9.
32. E. Rácz and E. P. Prens, Ultraviolet Light in Human Health, Diseases and Environment. Advances in Experimental Medicine and Biology, S. AhmadSpringer, Cham., 2017; 287–294.
33. Singh, K.K. and Tripathy, S." Natural Treatment Alternative for Psoriasis: A Review on Herbal Resources. Journal of Applied Pharmaceutical Science." 2014; 4: 114-121.
34. Hervé Bachelez, Siew-Eng Choon, Slaheddine Marrakchi, Tsen-Fang Tsai, Akimichi Morita; "Trial of Spesolimab for Generalized Pustular Psoriasis ". The New England Journal of Medicine, 2021; 385: 2431-2440.
35. Rajagopalan Murlidhar, Chatterjee Manas De, Abhishek Dogra. "Systemic Management of Psoriasis Patients in Indian Scenario".Indian Dermatology Online Journal, 2021; 12(5): 674-682.
36. Manas Chatterjee. "Comorbidities in Paediatric Psoriasis: Experience from a Tertiary Care Centre". Indian Journal of Dermatology, Sep-Oct 2023; 68(5): 491.
37. Ummu Umaimah Mohd Nordin, Noraini Ahmad, Norazlinaliza Salim, Nor Saadah Mohd Yusof . "Lipid-based nanoparticles for psoriasis treatment: a review on conventional treatments, recent works, and future prospects". RSC Advances, 2021; 46.
38. Bing-Xi Yan, Xue-Yan Chen, Li-Ran Ye, Jia-Qi Chen, Min Zheng, Xiao-Yong Man. "Cutaneous and Systemic Psoriasis: Classifications and Classification for the Distinction". Frontiers in medicine, 2021; 8.
39. Sunil Dogra, Savita Yadav. "Psoriasis in India: Prevalence and pattern". Indian Journal of Dermatology, Venereology and Leprology. 76. 2010. 595-601.
40. Nitha Vincent, Devi D Ramya, Hari BN Vedha. "Progress in Psoriasis Therapy via Novel Drug Delivery Systems". Dermatol Reports, 2014; 6(1):

5451.
41. R. Feldman, "Psoriasis causes as much disability as other major medical diseases". Journal of the American Academy of Dermatology, 2020; 82(1): 256-257.