TREATMENT AND MANAGEMENT OF PSORIASIS –A COMPHREHENSIVE REVIEW

Dr A Geethalakshmi[1*], Dr G C Mamatha[2], Himadri Priya Gogoi[3], Aditi saha[4], Anil Kumar S[5], Likhitha N[6], Mohammed Owais Naqui Ahmed[7], Sahana BM[8]

[1*] Professor & HOD, Department of Pharmaceutics, Harsha College of Pharmacy, Nelamangala, Bangalore.
[2] Professor & Principal, Harsha college of Pharmacy, Nelamangala, Bangalore.
[3]Assistant Professor, Department of Pharmaceutics, Harsha college of Pharmacy, Nelamangala, Bangalore
[4,5,6,7,8]Department of Pharmaceutics, Harsha College of Pharmacy, Nelamangala, Bangalore.

Corresponding Author*
Dr A Geethalakshmi
Professor & HOD
Dept. of Pharmaceutics
Harsha College of Pharmacy
Nelamangala, Bangalore-562123
Mail ID: geelak74gmail.com
Mob No:9901418806

ABSTRACT:

Psoriasis is a chronic, immune-mediated inflammatory skin disorder with variable global prevalence. Affecting 2–3% of the global population, it manifests in various forms including plaque, guttate, pustular, and erythrodermic psoriasis, often with nail and joint involvement. A systematic review of population-based studies revealed that the disease's prevalence ranges widely by geography and age, being more common in regions farther from the equator. Prevalence among children ranged from 0% in Taiwan to 2.1% in Italy, 0.44% to 2.8% among adult population in India, while adult rates varied from 0.91% in the U.S to 8.5% in Norway. Treatment options depend on disease severity and patient preference. Topical therapies such as corticosteroids, vitamin D analogues, salicylic acid, and coal tar are first-line treatments for mild to moderate cases. Systemic therapies, including oral agents (methotrexate, cyclosporine) and biologics (e.g., TNF and IL inhibitors), are recommended for moderate to severe psoriasis or when topical treatments fail. Phototherapy remains an adjunctive approach in resistant cases. Additionally, herbal remedies such as Aloe vera, Milk Thistle (*Silybum marianum*), Dong Quai (*Angelica sinensis*), and Chamomile (*Matricaria recutita*) have shown potential anti-psoriatic effects through anti-inflammatory and immunomodulatory mechanisms. An integrated treatment approach, combining conventional and alternative therapies, may enhance patient outcomes and quality of life. The main objective of this review article is to compile the available information related to Psoriasis by using synthetic chemical drugs and herbs. It includes introduction prevalence, causes, signs and symptoms, types of psoriasis, conventional treatment, herbal plants used in psoriasis treatment and different dosage forms for psoriasis.

Key Words: Psoriasis, inflammation, chronic skin disease, pustular, plaque psoriasis, phototherapy.

1.1 INTRODUCTION:

Psoriasis is one of the life-threatening skin diseases. It is an immune-mediated disorder with hyperkeratosis and other inflammatory reactions. Psoriasis can be categorized as mild, moderate and severe conditions. Mild psoriasis leads to the formation of rashes, and when it becomes moderate, the skin turns into scaly[1].

Psoriasis is a chronic immune-mediated inflammatory dermatological condition, linked to comorbidities like psoriatic arthritis, psychiatric disorders, cardiovascular problems, and hepatic conditions. In 2014, the World Health Organisation acknowledged psoriasis as a significant non-communicable disease and emphasised the misery associated with misdiagnosis, insufficient treatment, and the stigmatisation of this condition[2].Psoriasis (from the Greek word psora, which means itching) is a common and highly stigmatising inflammatory skin disease. Behcet (1935) called it "the antidote to a dermatologist's ego," and while this is still true in some ways, significant advancements have been achieved in a number of crucial areas[3].

The immunological and genetic processes that underlie the pathophysiology of psoriasis are well understood. This better understanding has led to the development of some of the new biological treatments for psoriasis that have recently been licensed, while other treatments have contributed. For instance, the significant effectiveness of agents that block the cytokine tumour necrosis factor's actions highlighted the crucial role that tumour necrosis factor plays in the pathophysiology of the disease[3].

Additionally, there is growing evidence that psoriasis is not limited to skin conditions. According to epidemiological research, psoriasis sufferers have a higher standardised death rate, especially when it comes to heart disease and cancer. Additionally, prevalent and perhaps treatable is clinically meaningful psychological and mental comorbidity. An outline of these advancements and how they affect clinical practice is provided in this article[3].Even though psoriasis was identified as a separate illness in 1808, its pathogenic mechanisms were not understood for many years. In 1841, Ferdinand von Hebra gave it a formal definition as a separate clinical entity[4].

Psoriasis manifests clinically as elevated, distinct erythematous plaques with uneven borders and silvery scales. Both the upper and lower extremities are frequently impacted, although the elbows, knees, scalp, and trunk are more frequently affected. About 90% of cases are of psoriasis vulgaris, also known as plaque psoriasis. Guttate, inverted, erythrodermic, pustular, and palmoplantar psoriasis are some more types, and nail involvement is also commonly seen. Psoriasis can cause a variety of systemic symptoms in addition to skin conditions, such as metabolic syndrome, inflammatory joint disease, and problems of the eyes, heart, and gastrointestinal tract. The illness has a major negative impact on quality of life and frequently requires long-term care, which adds to the financial and social costs. Furthermore, psoriasis is linked to a lower life expectancy[5].

The innate and adaptive immune systems cells and molecules, as well as the aberrant reactions of normally healthy skin cells to immune-related stimuli, are the main causes of psoriasis vulgaris, an inflammatory skin condition. In many ways, the immune circuits that are constitutively present or inducible in healthy human skin are enlarged counterparts of the immunological pathways that are activated in psoriasis. Among these, epidermal keratinocytes are essential for innate immunity because they can activate and change different T cell subsets that are drawn to the skin. This review starts with a summary of the immunological characteristics of healthy human skin because the skin is an immune-competent organ with its own resident T cell populations[6].

The disease is frequently associated with comorbidities like rheumatoid arthritis, colitis, diabetes, metabolic syndrome, and hypertension. In contrast, atopic dermatitis and allergies are less commonly linked to psoriasis[7]. Psoriasis develops when the immune system tells the body to over-react and accelerate the growth of skin cells. Normally the skin cells mature and are shed from the skin’s surface every 28 to 30 days. When psoriasis develops, the skin cells mature in 3 to 6 days and move to skin surface. Instead of being shed, the skin cells pile up, causing the visible lesions. It is also found that genes that cause psoriasis can determine how a person’s

immune system reacts. These genes can cause psoriasis or other immune mediated conditions such as rheumatoid arthritis or Type-I Diabetes[8].

Typically, psoriatic lesions are red, well-defined, somewhat elevated, and covered in loosely connected, easily removable silver-white scales. Multiple layer peeling may result in cutaneous papillae bleeding in certain places. The scalp, sacral region, and limb extensor surfaces are common locations. Usually starting as little areas, lesions can eventually spread into regional patterns or, in extreme situations, encompass the entire body. More than 10% of individuals also develop psoriatic arthritis, and nail abnormalities are common[9].

1.1.1 PREVALANCE OF PSORIASIS:

It is still unclear how common psoriasis is throughout the world. We carried out a systematic assessment of population-based studies that reported on the incidence and prevalence of psoriasis in order to close this gap. Three electronic databases were thoroughly searched from the time of their creation to July 2011. 53 papers that examined the epidemiology of psoriasis in the general population out of the 385 critically evaluated articles satisfied the inclusion requirements[10].

The prevalence of psoriasis in India is estimated to be between 0.44% and 2.8% of the population. It's twice as common in males compared to female. The children's prevalence rates varied from 2.1% in Italy to 0% in Taiwan. The frequency among adults ranged from 8.5% in Norway to 0.91% in the US. 40.8 cases per 100,000 person-years was the only incidence estimate for children (from the United States) that was published. Incidence rates in adults varied from 230 per 100,000 person-years in Italy to 78.9 per 100,000 person-years in the united state. About 2% of people in the UK suffer from psoriasis, which is a global condition. Psoriasis is a chronic skin disease affecting approximately 1.5% of the Caucasian population, translating to over 7 million individuals in Europe. It is significantly less common in other ethnic groups, such as the Japanese [11,12].

According to the research, psoriasis is more common in areas further from the equator and varies in incidence by age and geography. Furthermore, studies only included adults had prevalence estimates that were generally greater than those that included people of all ages [13,14].

1.1.2 CAUSES OF PSORIASIS

1.1.2.1 Genetic factors

Psoriasis has a substantial hereditary component. Monozygotic and dizygotic twins have concordance rates of about 70% and 20%, respectively. Furthermore, a positive family history is reported by roughly 35% of patients. Individuals who have two parents with the condition are up to 50% more likely to get it themselves, according to family-based research. Psoriasis

has a complicated genetic background that incorporates several genes. Genes linked to the nuclear factor kappa B (NF-κB) signalling pathway, T-cell receptor formation and polarization, and antigen presentation have been identified.

HLA-Cw6: The major histocompatibility complex (MHC) class I allele encoded by the HLA-Cw6 gene is the one most thoroughly researched in psoriasis. This lends credence to the idea that psoriasis is a reaction to autoantigens mediated by T cells. There are two possible autoantigens linked to HLA-Cw6: LL-37: An antibacterial peptide associated with cathelicidin melanocytes and keratinocytes have ADAMTSL5, a disintgrin and metalloproteinase with thrombospondin motifs-like protein 5. Patient with psoriasis have 10.5% to 77.2% prevalence of HLA-Cw6, with white people having higher frequency then Asian people. HLA-Cw6 is linked particular clinic characteristics, such as: disease with an early onset (before age 21), Psoriasis of the guttate kind, increased engagement of the body's surface area, and the Koebner phenomenon has become more common[15].

1.1.2.2 Infections with streptococci

For more than a century, it has been known that streptococcal infections are linked to psoriasis, especially acute guttate psoriasis. Psoriasis can occur after perianal infections and streptococcal vulvovaginitis, but it is more frequently associated with streptococcal pharyngitis. Although guttate psoriasis usually resolves on its own, it can develop into chronic plaque psoriasis or reoccur with further streptococcal infections. Within ten years after their first bout of acute guttate psoriasis, about one-third of patients acquire chronic psoriasis. Streptococcal throat infections can worsen symptoms in people who already have plaque psoriasis. The underlying process most likely consists of hereditary immune response defects and molecular mimicry between human keratinocytes and streptococcal M protein. Patients whose psoriasis flares are closely associated with recurrent tonsillitis may benefit from tonsillectomy[16].

1.1.2.3 HIV or human immunodeficiency virus:

The frequency of psoriasis in people with HIV is on par with or higher than in the general population. HIV can aggravate pre-existing psoriasis and cause new cases, and the severity of the condition is frequently correlated with deteriorating immune function. Although all types can occur, guttate, inverse, and erythrodermic psoriasis are common clinical subtypes in HIV. Even though psoriasis is largely a TH1-driven disease and HIV is a TH2-mediated disorder, CD8+ memory T cell proliferation during immunosuppression may cause aggravation. Careful treatment selection is necessary to prevent further immunological impairment.

1.1.2.4 Additional infections:

Helicobacter pylori: H. pylori infection triggers systemic inflammation by increasing cytokines like TNF-α and IL-17, which are also involved in psoriasis pathogenesis.

Candida species: Candida can trigger an immune response, leading to increased production of inflammatory cytokines like IL-17 and TNF-α, which are also involved in psoriasis pathogenesis[17].

1.1.2.5 Living styles and psoriasis

Although the degree of these correlations varies, lifestyle factors like obesity, smoking, and alcohol use have been linked to the development and progression of psoriasise[16].

- **Being overweight**: It is known that obesity is a low-grade, chronic inflammatory state that can contribute to psoriasis development exacerbate an already-existing illness. The pathophysiology and severity of psoriasis may be influenced by the pro-inflammatory cytokines and adipokines generated in adipose tisuue[16].

- **Smoking**: One important modifiable risk factor for psoriasis is smoking. Evidence points to smoking raises the chance of getting psoriasis. There is a dose-response association between the incidence of psoriasis and the quantity and duration of smoking. Cigarette smoking has been identified as a significant modifiable risk factor for both the onset and severity of psoriasis. The pathophysiological mechanisms linking smoking to psoriasis involve complex interactions between oxidative stress, immune activation, and vascular dysfunction. Smoking promotes the release of pro-inflammatory cytokines such as TNF-α and IL-17, which are central to the immunopathogenesis of psoriasis. Additionally, nicotine exposure has been shown to enhance keratinocyte proliferation and alter T-cell function, favoring a Th1/Th17-dominant immune response[16].

- **Drinking alcohol**: Alcohol intake has been increasingly recognized as a potential aggravating factor in the development and progression of psoriasis. It exerts multiple effects that can exacerbate the disease. One of the primary mechanisms involves the modulation of immune function. Alcohol can impair immune regulation, promoting a pro-inflammatory state that facilitates the overproduction of cytokines such as TNF-α, which play a central role in psoriasis pathophysiology[16].

1.1.3 TYPES OF PSORIASIS

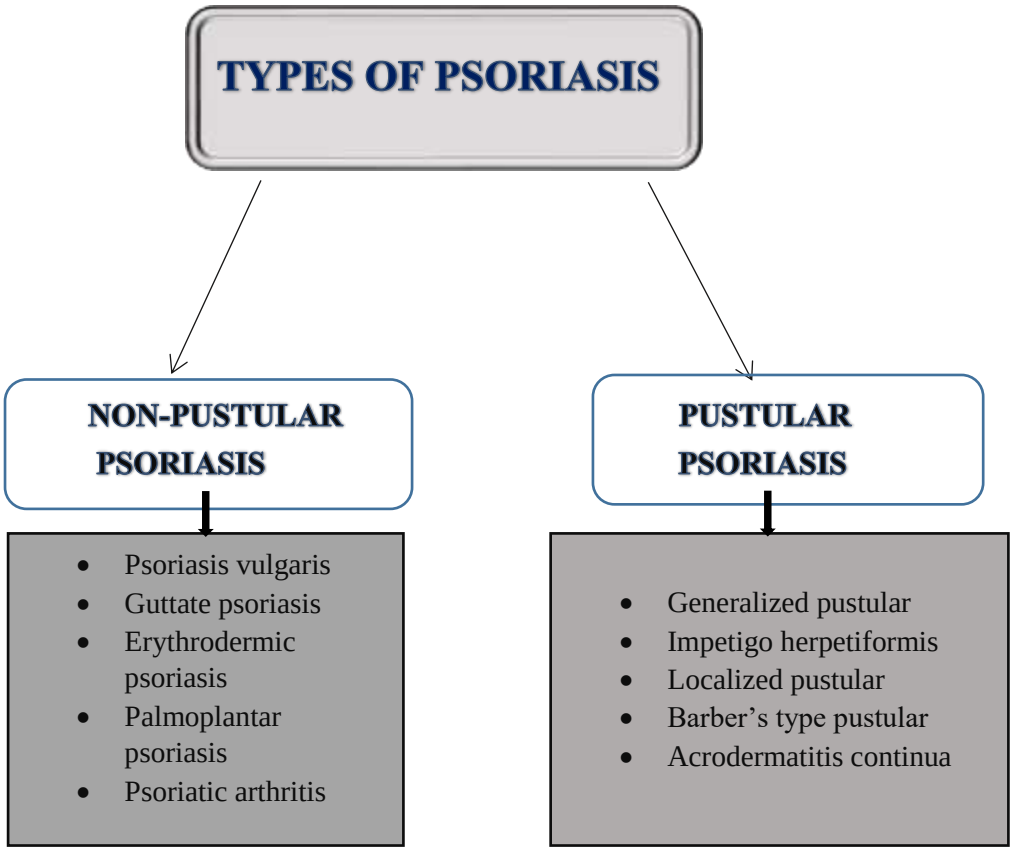

Figure 1: Types of Psoriasis

1.1.3.1 NON-PUSTULAR PSORIASIS

a. Psoriasis vulgaris

Psoriasis vulgaris is the most common clinical form of psoriasis, accounting for about 90% of all cases. Lesions exhibit symmetric distribution and are most commonly found on the knees, elbows, scalp, and sacral area. Predilection for these lesions may be the result of traumatic incision [17,18].

b. Guttate psoriasis

This type of psoriasis commonly affects children and young adults. It typically begins suddenly, presenting as small, drop-like lesions and occasionally as scaly psoriatic papules. It often appears following a streptococcal infection and is strongly linked to the HLA-Cw6 gene. Elevated antistreptolysin levels are frequently observed. In many cases, the skin lesions resolve

on their own once the infection subsides. These lesions usually occur on the trunk, upper limbs, face, and scalp, and tend to improve within 3 to 4 months. However, in some cases, they may increase in size and develop into plaque-type psoriasis[18].

c. Erythrodermic psoriasis

In this widespread form of psoriasis, skin lesions can cover up to 80% of the body surface. The lesions are mainly red (erythematous), and the usual appearance of papules and plaques becomes less defined. Peeling or scaling of the skin is generally less noticeable. Individuals with erythrodermic psoriasis may experience hypothermia as a result of extensive blood vessel dilation throughout the skin [18,19].

d. Palmoplantar psoriasis

This form of psoriasis typically affects both palms and soles in a symmetrical pattern, with the thenar areas being more commonly involved than the hypothenar regions. Redness (erythema) may not always be present, but when it is, it often appears as pinkish-yellow discoloration. The most noticeable feature is scaling, and in some cases, the thickened scales can resemble keratoderma[20].

e. Psoriatic arthritis (PsA)

Psoriatic arthritis (PsA) has a general population prevalence of approximately 0.02% to 0.1%, but it occurs in about 5.4% to 7% of individuals with psoriasis. The likelihood of developing PsA increases significantly—up to 30–40%—in those with severe skin involvement, especially in cases of pustular psoriasis. Typically, uncomplicated psoriasis begins in the second or third decade of life, whereas PsA becomes more common starting in the third decade. The condition affects males and females equally, with a male-to-female ratio of 1:1. In about 75% of PsA patients, skin symptoms appear before joint involvement. Around 15% experience skin and joint symptoms simultaneously, while in roughly 10%, joint issues precede the skin manifestations. Additionally, nail changes are observed in about 80% of those with psoriatic arthritis[21].

f. Inverse Psoriasis

Psoriasis that appears in skin folds is referred to as flexural or inverse psoriasis. Due to the constant moisture and friction in these areas, typical scaling is usually absent. Instead, the condition presents as well-defined, bright red plaques that are symmetrical, inflamed, and often have surface cracks. These sharply bordered, fissured plaques are characteristic of this type. It is more commonly observed in individuals with obesity and often shows features similar to seborrheic dermatitis. This form of psoriasis tends to be more resistant to standard treatments[22].

1.1.3.2 POSTULAR PSORIASIS

a. Generalized pustular psoriasis

This uncommon type of psoriasis is marked by the development of pustules and is most often observed in younger individuals. It may arise independently or as a secondary reaction to psoriasis vulgaris, particularly following abrupt cessation of systemic corticosteroids, exposure to irritants, hypocalcemia, or other triggering factors[22].

The condition usually begins suddenly on a red, inflamed base and is accompanied by systemic symptoms such as high fever, fatigue, and joint pain involving multiple joints. Laboratory findings may include elevated erythrocyte sedimentation rate (ESR), increased white blood cells, low lymphocyte count, and a negative nitrogen balance. The pustules typically dry out within a few days, but new ones tend to reappear. Redness around the pustules can spread, potentially leading to erythroderma. Early medical intervention is crucial, as the acute phase of the widespread form can become life-threatening if left untreated[23].

b. Impetigo herpetiformis

This rare form of psoriasis, also referred to as generalized pustular psoriasis of pregnancy, typically presents with red, inflamed skin covered in pustules. These lesions often begin in the body's flexural areas and tend to spread outward, sometimes merging together. In skin folds, they may take on a vegetative appearance. Mucosal surfaces can become involved, and nail changes such as onycholysis may occur due to pustules forming beneath the nails. The lesions may cause itching or a burning sensation and often emit an unpleasant odor. Along with a general decline in health, symptoms such as fatigue, fever, chills, nausea, and vomiting are common. This condition is frequently linked to low calcium levels and usually arises in the final trimester of pregnancy or during the postpartum period. It often recurs in future pregnancies[24].

c. Localized pustular psoriasis

Palmoplantar pustulosis is classified into two main types: Barber's pustular psoriasis and acrodermatitis continua of Hallopeau.

- **Barber's Type Pustular Psoriasis**
  This is a chronic and recurring condition that tends to affect women more often, particularly those with a family history of palmoplantar pustulosis. Clinically, it presents as 2-4 mm pustules localized on the palms and soles, especially in the thenar an hypothenar areas, which are typically red and inflamed. Although the exact cause is unclear, contact sensitivity appears to play a significant role. Factors such as smoking, tonsillitis, heat, humidity, and high temperatures can trigger flare-ups [25].

- **Acrodrmatitis Continua (Hallopeau's Disease):**
  This is a slowly spreading skin disorder marked by the appearance of sterile pustules on the fingers and toes. In more severe cases, it can lead to nail loss and even destruction of

the distal phalanges. The pustules may merge to form small, purulent, fluid-filled vesicles with a polycyclic pattern. Whether this condition is a distinct variant of psoriasis remains under discussion[26].

1.1.4 SIGNS AND SYMPTOMS OF PSORIASIS:

Psoriasis is a chronic immune system disease that makes the skin cells multiply too fast. The signs and symptoms of psoriasis can vary common depending on the type of psoriasis. like,

1. Skin rashes or patches
2. Itchy
3. Painful skin
4. Problems with your finger nails and toe nails
5. Small red dots
6. Pus-filled bumps
7. Smooth red patches in skin folds
8. Psoriasis plaques, sleep disturbance
9. Dry, cracked skin
10. Eye inflammation, skin pain[27].

1.1.5 PREVENTION OF PSORIASIS:

1.Avoid triggers
Like: Stress, infection, skin injuies, medication.
2.Life style modification
Like: Healthy diet, limit alcohols, no smoking.
3.Skin care
Like: Moisturize regularly, gentle skin products.
4.Monitor and manage other conditions
Like: Obesity, diabetes, hypertension.
5.Regular medical check up
6. Regular exercise
7. Avoid harsh treatments or over-crossing[26].

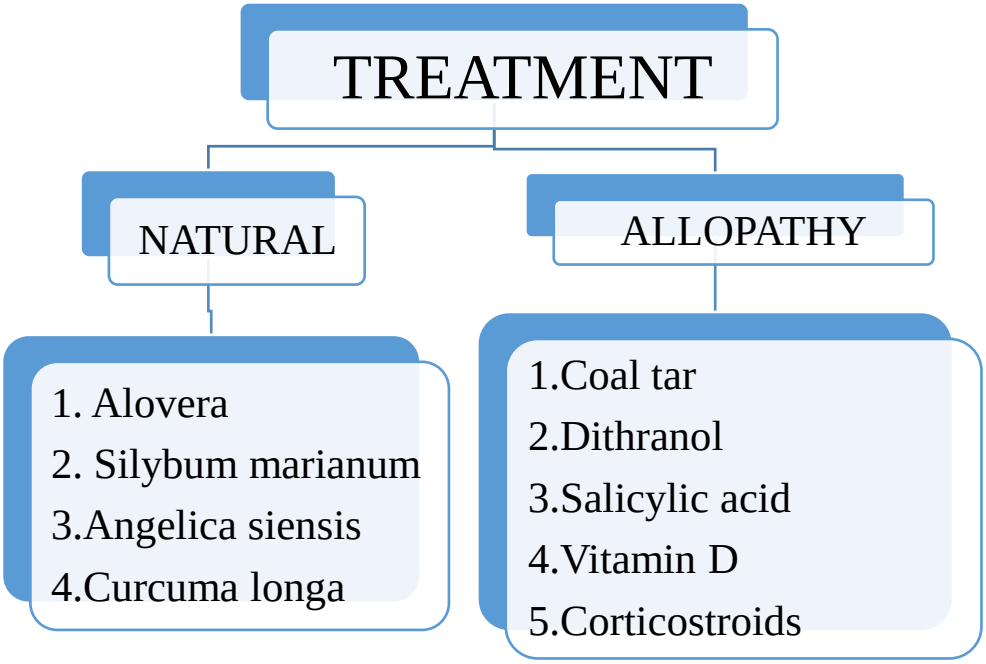

Figure 2: Types of Treatment

1.1.6 DOSAGE FORMS FOR PSORIASIS TREATMENT[30].

The choice of formulation depends on the type**,** severity, and location of psoriasis, as well as patient preference and response to treatment.

Table 2: Dosage forms for Psoriasis Treatment.

SLNO.	Treatment	Drug	Dosage forms
1.	Topical	a. Corticosteroids b. Vitamin D anlogues c. Combination drugs d. Coal tar composition	Gel, ointment, creams Creams, ointment, solution Gel, foam, ointment Shampoo, gel, cream
2.	Biological therapies	a.TNF-Inhibitors b.Oral small molecules	Vials, autoinjectors, syringes. Tablets
3.	Photographs	a.UVA + Psoralen (Moderate Psoriasis)	Creams, ointments

1.1.7 CONVENTIONAL TREATMENT OF PSORIASIS

1.Coal Tar

Coal tar has been used to treat psoriasis since ancient times, particularly for scalp psoriasis, but its use has declined in recent decades in comparison to other tropical. This is most likely due

to its unsightly characteristics, which include odorous odour and fabric discolouration, particularly in people with non-black hair. Polycyclic aryl hydrocarbon is the most studied of the thousand molecules that make up the tar since it is thought to have a therapeutic effect on psoriasis[31].

2.Dithranol

One of the earliest methods for treating plaque psoriasis is Dithranol, commonly referred to as Anthralin, which works by preventing keratinocyte development and triggering apoptosis. illustrated using keratinocyte cells from humans. Essentially, despite being the subject of numerous investigations, the direct or indirect effects of Dithranol on the inflammatory response of psoriasis remain poorly understood. Despite this claim, a recent study demonstrated that topical Dithranol causes the PASI score to drop quickly, which in turn causes a decrease in epidermal hyperproliferation Vitamin A's vitamers or compounds are called retinoid. Tazrotene and Acitretin are the only retinoid that the FDA has approved for use in treating psoriasis. 92 Whereas acitretin is an oral medication, Tacarotene is a topical medication. The retinoid suppress keratinocyte growth by acting on retinoic acid receptors and retinoid-X-receptors, which start the modification of inflammatory cytokine gene expression. and a delayed reduction in inflammatory penetration in psoriatic skin. The study also revealed that keratinocytes, which are found in the epidermis, rather than immune cells, can be targeted to reduce psoriatic activity[31,32].

3.Salicylic acid.

A strong keratolytic, Salicylic acid is frequently used in conjunction with other topical medications such Calcineurin inhibitors and Corticosteroids. It is thought that when used with calcineurin inhibitors, it improves the absorption of medications into psoriasis plaques. Telogen hair may temporarily shed while using Salicylic acid alone to treat scalp psoriasis, but this problem may be resolved if the medication stopped. Furthermore, using Salicylic acid to broad body surface areas at concentrations higher than 10% is not advised. This is primarily to prevent potential adverse effects like nausea, frontal headaches, and burns to the oral mucosa[33].

4.Vitamin D

Two forms of Vitamin D mimics that are produced from sunshine are calcitriol and calcipotriol. Psoriasis is thought to be linked to vitamin D deficiency as Its presence in sufficient amounts aids in maintaining the body's health by inhibiting the synthesis of powerful cytokine mediators of inflammation, such as IL-2, IL-6 and IFN-g. Additionally, Th17 cells—which support a robust immune system—and Vitamin D are correlated in psoriasis sufferers. 108 Vitamin D is a natural suppressor of cell growth because it clearly activates suppressor T cells, which control other immune system cells and prevent cytotoxicity[32].

5.Corticosteroids.

The Cornerstone of topical treatments for psoriasis is Corticosteroids. They function by preventing phospholipase A2 from being released, and they have a direct effect on inflammatory cytokines and deoxyribonucleic acid (DNA). By decreasing inflammation and

slowing down cell hyper proliferation, corticosteroids alone function as first-line topical therapy, particularly in genital psoriasis. As was previously mentioned, several studies have shown that the benefits of Corticosteroids are enhanced when combined with other topical medications, specifically Retinoid, Salicylic acid, and Vitamin D analogues[34].

6.Systemic therapies

A medication that circulates throughout the body is used in systemic treatment. It can be separated into two categories: oral agents and biological agents that enter the body through intravenous (IV) infusion or injection. According to international guidelines for the treatment of psoriasis, systemic nonbiologic (oral agent) and biologic therapies may be sought to achieve skin clearing if topical and phototherapy treatments are shown to be ineffective[35].

7.Oral medications.

Oral ingestion of systemic non biologic treatments suppresses inflammatory reactions. The two well-known oral, systemic treatments for psoriasis that have been used for decades are Methotrexate and Cyclosporine; they are frequently advised as monotherapy or in combination with other systemic treatments. Systemic non biologic medications provide several benefits, even though biologics have received a lot of attention lately for treating psoriasis[35].

1.1.8 HERBAL PLANTS IN PSORIASIS TREATMENT.

There are many herbal plants that are used for the treatment of psoriasis some of them are described below:

1. *Aloe vera*

Aloe vera is a popular plant used in cosmetic care and first aid products in case of thermal injuries. Aloe contains Anthroquinones, Steroids, Saponins, Mucopolysaccharides and Salicylic acid. Syed and colleagues (1996) conducted a double - blind, placebo – controlled study on 60 patients with psoriasis with slight to moderate plaque type psoriasis and an average 8.5year duration of their disease. Patients self - administered topical Aloe vera extract cream or vehicle placebo three times a day without occlusion for 4 weeks to their psoriatic plaques. The aloe group showed significantly higher rates of clearing the psoriatic plaques in almost all patients. The primary active ingredients in Aloe vera, Anthraquinone and Acemannan, have antibacterial properties against *Streptococcus* and Staphylococcus species, which may explain why they are effective treatments for psoriasis. Furthermore, Aloe vera contains Salicylic acid, a Keratolytic that may help explain its claimed effectiveness in psoriatic plaque desquamation[36].

2. *Silybum marianum*

It is commonly known as Milk Thistle. This plant is very well known for its hepatoprotective activity. Numerous changes have been detected in the liver of patients with psoriasis, including steatosis, periportal inflammation, fibrosis, necrosis and cirrhosis. A multifactorial etiology of liver disease in patients with psoriasis includes changes due to alcohol use, nutritional factors,

anti – psoriatic medications and a direct effect of the psoriasis itself. Abnormally high levels of cAMP and leukotrienes have been observed in psoriatic patients and normalization of these levels may improve the condition. The importance of silymarin in the treatment of psoriasis may be due to its ability to improve endotoxin removal by the liver, inhibit cAMP phosphodiesterase and inhibit leukotriene synthesis[36].

3. *Angelica sinensis*

It is often referred to as Dong Quay. Psoralen, a strong furocoumarin, is present in the extracts of this Chinese herbal remedy. Psoralens are strong photosensitisers when UVA rays are present. After psoralen use, exposure to UVA results in cross-linking of epidermal DNA, which lowers the rate of epidermal DNA synthesis. By ingesting dong quay and then undergoing ultraviolet light therapy or natural sunlight, patients are self-administering a type of psoralen–UVA (PUVA) therapy. Two-thirds of the patients in Koo & Arain's 1998 study of psoriasis patients saw total remission from their condition following oral administration of this plant extract. Although hogweed (*Heracleum sphondylium*), another herb used to cure psoriasis, also contains psoralen, its effectiveness and adverse effects are unknown[36].

4. *Matricaria recutita*

It is frequently referred to as chamomile. There is a long history of using chamomile flowers as a remedy for gastrointestinal conditions. The oil's usage in psoriasis is justified by the fact that chamazulene, a by product of the non-volatile extract matricin, is known to have anti-inflammatory properties through the inhibition of lipoxygenase and the subsequent generation of leukotriene B4 (LTB4). Psoriatic plaques have been linked to elevated LTB4 production, according to data; as a result, inhibition improves the condition *Staphylococcus* and *Candida* are two skin infections that chamomile oil exhibits antibacterial efficacy against. The flower's active ingredients are Flavonoids including Quercetin and Apigenin. According to reports, Quercetin is a strong inhibitor of lipoxygenase and, to a lesser extent, cyclooxygenase. Additionally, Quercetin exhibits good skin penetrating properties[36].

5. ***Gaultheria procumbens***

It is also referred to as Wintergreen. Native Americans have traditionally utilised wintergreen, a plant indigenous to the Eastern United States, as an analgesic. It has anti-inflammatory qualities because it contains Methyl Salicylate. Wintergreen is used topically to treat psoriasis, but it can also have systemic symptoms like tinnitus, vomiting, tachypnea, and acid-base imbalances. Systemic toxicities are more likely to occur in patients using aspirin or a prescription salicylic acid compound along with a salicylate herbal supplement. Furthermore, wintergreen oil can raise the international normalised ratio (INR) of clotting and prothrombin time, which can cause issues for people using warfarin. Although its effectiveness in treating psoriasis has not been studied, it may have an anti-inflammatory impact and requires more scientific research before being used[36].

6. *Ulmus rubra*

Because of its mucilage component, which comes from the elm's inner bark, it is usually referred to as slippery elm. Native Americans have traditionally applied this extract as a

poultice to wounds and boils. At the moment, it is sold as a remedy for cystitis, reflux, and irritable bowel syndrome. A study group of five patients with persistent plaque-type psoriasis who were following a six-month medical nutritional therapy regimen at home was assessed by Brown and colleagues (2004). Throughout the trial time, they evaluated intestinal permeability and psoriasis symptoms, and they noted improvements in every parameter under investigation. Averaged post-test results for psoriasis area and severity index demonstrated a notable improvement[37].

7. ***Curcuma longa/ Curcuma domestica***

The use of turmeric to treat kidney stones and infections dates back many years. It is a relatively recent adjuvant for psoriasis. Curcuminoids and volatile oils are believed to include the anti-inflammatory components, which work by selectively inhibiting phosphorylase kinase (PhK). The epidermis contains the enzyme PhK. Clinical activity of psoriasis has been observed to correspond with significantly higher levels. Decreases in keratinocyte transferrin receptor expression, density of epidermal CD8 + T cells, and the degree of parakeratosis are also shown to correlate with lower PhK activity in the curcumin and calcipotriol-treated groups. Although contact dermatitis is a documented side consequence, the study did not report any other negative outcomes[38].

8. *Mahonia aquifolium*

This plant is often used to treat skin conditions, particularly psoriatic plaques. Muller et al. (Muller et al., 1994) examined the impact of *Mahonia aquifolium* bark extract and its primary components (oxyacanthine, berberine, and berbamine) on lipid peroxidation and 5-lipoxygenase. Additionally, he observed that the bark extract of Mahonia aquifolium inhibits the formation of keratinocytes. Oxyacanthine and berbamine, two benzylisoquinoline alkaloids, were more powerful inhibitors[39].

9. *Alpinia galangal* and *Annoa squamosal*

The anti-psoriatic properties of the plants *Alpinia galanga*, *Curcuma longa*, and *Annona squamosa* were reported by Chanachai et al. (2009). They described how the extracts suppress psoriasis at the molecular level by controlling NF-kB signalling biomarkers. They reported gene assays in 10 distinct genes of the NF-kB signalling network in HaCaT cells using semi-quantitative RT-PCR[39].

10. ***Thespesia populnea***

Thespesia populnea bark extract was shown to have an anti-psoriatic action on Perry's Scientific Mouse Tail model by Shrivastava et al. (2009). A 25% rise in orthokeratosis was reported. According to reports, plants include proteins, lipid/fixed oil, flavonoids, triterpenoids, carbohydrates, glycosides, tannins, and phytosterols[40].

1.1.9 CURRENT RESEARCH ON PSORIASIS TREATMENT:

Table 2: Current research on psoriasis treatment

SLNO	DRUG	FORMULATION	STUDY OUTCOME
1	Methotrexate	Topical Formulation	Polysorbate 60/80-based methotrexate-loaded Nanostructured lipid carriers(NLCs) demonstrated spherical morphology, approximately 65% drug entrapment, and stable nanoscale charactistics. Topical psoriasis With improved skin penetration, particularly with P60, they allowed for about 70% medication release in 2 hours, suggesting potential for topical psoriasis treatment[41,42].
2	Barbaloin gel	Hydrogel Formulation	This study used a variety of gelling agents to create barbaloin-loaded hydrogels (F1–F10) for the topical treatment of psoriasis. To find the best candidate for in vitro drug release investigations, formulations were assessed for their physicochemical and microscopical characteristics[43].
3	Tacrolimus	Hydrogel formulation	To improve the topical distribution of tacrolimus (TAC) in the treatment of psoriasis, a novel composite hydrogel including nanocarriers based on mPEGhexPLA was created. It improved skin histology in vivo and delivered double the TAC of ProtopicTM, demonstrating greater skin delivery and therapeutic effects[44].
4	Resveratrol Based Polymeric Micelles	Carbomer gel	A QbD technique was used to create resveratrol-loaded polymeric micelles (PM), which were then combined to create a gel (PMG) for improved psoriasis treatment. In a mouse model, PMG outperformed standard gel in terms of skin penetration, antioxidant activity, and therapeutic efficacy. Babchi oil was also added to a methoxsalen formulation to

			improve epidermal localization and skin penetration[45].
5	Methoxsalen And Babchi oil	Nanoemul gel	High-pressure homogenization was used to create nanoemulgel formulations that combined methoxsalen with Babchi oil. These formulations demonstrated stable droplet sizes (51.3–146.7 nm) and high entrapment efficiency (92.76–98.10%). In vivo, the optimized nanoemulgel (NG2) greatly reduced psoriasis symptoms and improved skin penetration[51].
6	Clobetasol	Solid lipid nanoparticles	Solid lipid nanoparticles (SLNs) loaded with clobetasol propionate were made by emulsification–homogenization and optimized using a 3^3 factorial design. The SLNs' 78.1% entrapment efficiency, high stability, and smaller particle size (133.3 nm) suggested that they might find application in medicine[46].
7	Betamethasone-dipropionate and salicylic acid	Nanocarrier based hydrogel	In order to improve skin penetration and maintain betamethasone dipropionate (BD) administration, this study investigated microemulsion-based gel formulations that combine BD with salicylic acid. The goal of the strategy was to increase BD's anti-psoriatic effectiveness and overcome its low permeability[47].
8	Karanjin-loadgel and soya lecithin	Ethosomal-nanogel	A 3^2 factorial design was used to create a karanjin-loaded ethosomalangle, which had an entrapment effectiveness of 94.88% and a vesicle size of 334 nm. Its promise as an effective topical treatment was highlighted by in vivo studies that demonstrated a considerable improvement in psoriasis symptoms[48].
9	Montesano-furoate-loaded	Aspasomal gel	Using film hydration and a 3^2 factorial design, a novel mometasone furoate-loaded aspasomal gel was created. It demonstrated sustained 24-hour drug

			release, 282.9 nm vesicle size, and 74.72% entrapment. Effective epidermal depot development without irritation was validated by ex vivo and in vivo experiments, suggesting the possibility of better psoriasis treatment[49].
10	Dithranol	Liposomal Formulation	In a six-week experiment for stable plaque psoriasis, a novel liposomal Dithranol gel had encouraging effects, with the majority of patients reporting notable improvement. It was aesthetically pleasing, well-tolerated, and might be better than conventional formulations[50].

1.1.10 CONCLUSION:

Psoriasis is a complex, chronic, immune-mediated inflammatory skin disorder with significant physical, psychological, and systemic impacts. Affecting 2–3% of the global population, it manifests in diverse clinical forms and often coexists with comorbidities such as psoriatic arthritis, cardiovascular disease, and mental health disorders. Advances in understanding its immunological and genetic basis have led to improved targeted therapies, significantly enhancing disease management. However, psoriasis remains a highly stigmatizing condition that severely affects patients' quality of life and longevity. Comprehensive care that addresses both skin symptoms and associated comorbidities is essential for improving patient outcomes and reducing the overall burden of the disease.

A widespread, long-lasting inflammatory skin condition that is mostly genetically mediated is psoriasis. Numerous medical and psychological comorbidities, including as metabolic syndrome, anxiety, depression, and cardiovascular disease, are linked to it. The quality of life for those who suffer with psoriasis could be greatly enhanced by these treatments. An integrated treatment approach, combining conventional and alternative therapies, may enhance patient outcomes and quality of life.

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