BMJ Best Practice

Psoriasis

Straight to the point of care



Last updated: Sep 30, 2022

Table of Contents

Overview		3
	Summary	3
	Definition	3
Theory		4
	Epidemiology	4
	Etiology	4
	Pathophysiology	5
	Classification	5
	Case history	15
Diagnosis		19
	Approach	19
	History and exam	28
	Risk factors	37
	Investigations	38
	Differentials	39
	Criteria	40
Management		41
	Approach	41
	Treatment algorithm overview	46
	Treatment algorithm	47
	Emerging	57
	Patient discussions	58
Follow up		59
	Monitoring	59
	Complications	59
	Prognosis	59
Gui	delines	60
	Treatment guidelines	61
Online resources		63
Evi	Evidence tables	
References		67
Images		82
Disclaimer		92

Summary

Psoriatic lesions are red, inflamed, silvery-white scaly, and circumscribed papules and plaques; often affecting elbows, knees, extensor limbs, and scalp, and, less commonly, nails, ear, and umbilical region.

Psoriasis is a multifactorial disease with a genetic basis. Exacerbations of disease may be related to infection, alcohol, medications, stress, and intercurrent illness.

Diagnosis is usually clinical.

Mild or limited psoriasis is treated with topical corticosteroids and/or vitamin D analogs.

Moderate to severe and/or extensive psoriasis may require phototherapy, and systemic agents such as methotrexate, cyclosporine, acitretin, or biologic agents.

Definition

Psoriasis is a chronic inflammatory skin disease characterized by erythematous, circumscribed scaly papules and plaques. It can cause itching, irritation, burning, and stinging. Although approximately 30% of people with cutaneous psoriasis also have psoriatic arthritis, this topic only discusses cutaneous psoriasis.[1]

Epidemiology

More than 80% of countries in the world lack information on the epidemiology of psoriasis.[4] Published data report prevalence ranging from 0.09% to 11.43%.[5] [6]

A systematic analysis and modeling study reported psoriasis incidence of 30.3 per 100,000 person-years to 321.0 per 100,000 person-years, and prevalence of 0.14% to 1.99%.[4] Incidence and prevalence were relatively low in regions with young populations (e.g., south Asia and sub-Saharan Africa) and higher in regions with older populations (i.e., high-income regions).[4]

In the US, a population-based cross-sectional study (using National Health and Nutrition Examination Survey data) suggested that psoriasis affects 3% of the adult population (>7.5 million adults).[7] Psoriasis prevalence was similar between women and men.[7]

A UK population-based cohort study estimated psoriasis incidence to be 129 per 100,000 person-years.[8]

Peak incidence increases up to 39 years of age then decreases; there may be a second peak between 50 to 59 years or 60 to 69 years.[4]

Psoriasis is uncommon in children. Patients presenting at a younger age are more likely to have an affected parent and to demonstrate human leukocyte antigen association.[9]

Etiology

Factors including genetics, immunology, and infection may contribute.

Genetics

Psoriasis heritability is between 60% to 90%, which is higher than most other multifactorial diseases.[10] Studies of monozygotic twins, linkage studies, and genome-wide association studies provide evidence that psoriasis has a genetic predisposition.[11] [12] [13]

Most genes associated with psoriasis are involved in the immune response, and relatively few encode for skin-specific proteins. HLA-Cw6 encodes an antigen involved in T-cell activation. There is an increased prevalence of HLA-Cw6 in people with psoriasis compared with controls. Tumor necrosis factor (TNF)-alpha, another protein-coding gene associated with innate and adaptive immune response, is implicated in the etiology of psoriasis. Pathogenic involvement of genes related to Th17-cell activation have been demonstrated in people with psoriasis.[12] [13] [14] [15] [16]

Immunology

Psoriasis is believed to be triggered by external insults in genetically susceptible individuals. Recognized triggers include trauma, infection, and medications (e.g., beta-blockers, lithium). Following initiation by an insult, the host DNA forms complexes with antimicrobial peptides released from keratinocytes (skin cells), which results in inflammation and keratinocyte proliferation to cause disease presentation.[16] [17] [18]

Infection

Guttate psoriasis is often observed subsequent to upper respiratory infection, such as streptococcal pharyngitis. It may also be associated with HIV infection. Viral infection, immunization, and any intercurrent illness have been linked to flares of guttate and plaque psoriasis.[16] [19]

Pathophysiology

Psoriasis is a hyperproliferative disorder, involving a complex cascade of inflammatory mediators. Mitotic activity of basal and suprabasal cells is significantly increased, with cells migrating from the basal layer to the stratum corneum in just a few days. The silver scale on the surface of psoriasiform lesions is simply a layer of dead cells.[16] [20]

Early clinical studies of TNF inhibitors demonstrated the important role of these cytokines in psoriasis, prompting the condition to be regarded as primarily driven by T-helper-1 (Th-1) cells.[21] However, evidence supports the pivotal involvement of a different immunological axis underlying the pathogenesis of psoriasis; namely, T-helper cells producing interleukin (IL)-17 and IL-23.[22] [23] [24] IL-17 and IL-23 expression is increased in the serum, lesional skin, uninvolved skin, and even tear liquid of patients with psoriasis compared with patients without psoriasis. These cytokines are now considered to be central to the pathogenesis of psoriasis as demonstrated by the efficacy of therapeutics inhibiting IL-17 or IL-23 pathways.

The presence of T-cells reacting against autoantigens has been detected; three autoantigens have been identified: LL37, ADAMTS-like protein 5, and phospholipase-2-derived products.[25] [26] [27] [28]

Classification

International Psoriasis Council[2]

- 1. Plaque psoriasis
 - Raised inflamed plaque lesions with a superficial silvery-white scaly eruption. The scale may be scraped away to reveal inflamed and sometimes friable skin beneath.



Plaque psoriasis on legs

From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on back
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on knee
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on foot
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on scalp

2. Guttate psoriasis

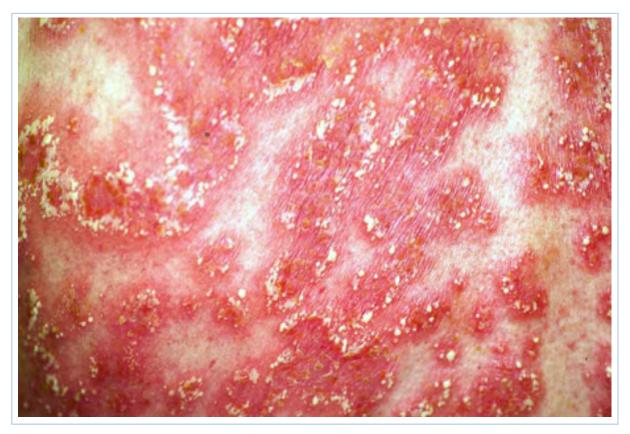
• Widespread, erythematous, fine, scaly papules (water drop appearance) on trunk, arms, and legs. The lesions often erupt after an upper respiratory infection.



Guttate psoriasis

3. Pustular psoriasis

- Acute generalized pustular psoriasis (von Zumbusch): rare, severe, urgent.
- Palmoplantar pustulosis: chronic involvement of hands and feet.



Pustular psoriasis

- 4. Erythroderma (erythrodermic psoriasis)
 - Generalized erythema with fine scaling. It is often associated with pain, irritation, and sometimes severe itching.



Erythroderma

From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

5. Psoriatic arthritis

- Unique in that, in addition to skin lesions, there is joint involvement that causes inflammatory damage and deformity. It affects approximately 20% of people with psoriasis, as reported by a 2019 epidemiologic study.[3]
- Most people with nail psoriasis have psoriatic arthritis. The arthritis most commonly involves fingers, hands, toes, and feet, and, less commonly, knees, elbows, and axial and sacroiliac joints.
- Cutaneous psoriatic lesions precede arthritis in 70% of cases.
- Psoriatic arthritis causes stiffness, inflammation, pain, and progressive and permanent joint damage. The arthritis is asymmetric in around 50% of cases.



Nail psoriasis - pitted nails
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Psoriatic arthritis
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

6. Keratoderma blennorrhagicum (reactive arthritis)

- Reactive immune disorder characterized by psoriasiform plaques, urethritis/cervicitis, conjunctivitis, and arthritis.
- Circular, scaly, scalloped-edged hyperkeratotic psoriasiform papules and plaques, which is sometimes
 painful and pustular (at the center of lesions), appears on soles and toes, and, less commonly, legs,
 palms, scalp, and penis.
- Occurs in genetically susceptible people with HLA-B27 following an infection (particularly *Yersinia enterocolitica* and *Yersinia pseudotuberculosis*).

Case history

Case history #1

A middle-aged man with a known history of psoriasis presents with white scaly papules and plaques on his elbows, extensor arms, knees, and shins. In the past 6 months, these lesions have become much worse and have started to appear on his waist and hip. Scaly and flaky eruptions are also present on his scalp, ears, and eyebrows. He describes the lesions as being itchy and irritating. He is a heavy smoker and has been unsuccessful in a previous attempt at smoking cessation.



Plaque psoriasis on legs
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on back
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on scalp
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

Case history #2

A young woman without a known history of psoriasis or skin disorder had a sudden onset of wide-spreading, white-scaly, oval- to round-shaped erythematous papules, which have been present for 2 weeks. Lesions are primarily on her trunk but also appear scattered on her arms and legs. She recalls a recent episode of sore throat and upper respiratory tract infection. A short course of antibiotics seemed to help, but did not clear the lesions.

Other presentations

Inverse psoriasis may present in the genital skin, the gluteal cleft and skin folds of the axillae, and under breasts. Palmar plantar psoriasis presents on palms and soles of feet. Pustular psoriasis presents as

sudden-onset disease with generalized pustulosis. However, the pustules carry no bacteria and patients are not febrile. Psoriatic arthritis is often insidious, with stiffness and inflammation around finger and toe joints only. The associated cutaneous lesions may be very minor, which makes it difficult to establish the diagnosis of psoriasis.



Pustular psoriasis

From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

Approach

Characteristic history and exam findings are often sufficient to diagnose the condition. Skin biopsy is reserved for atypical cases when lack of treatment response brings the diagnosis into question.

History

Most people with psoriasis have a positive family history.[11] [16]

Psoriasis usually begins as 1 or 2 limited lesions on elbows or scalp. In the majority of cases, psoriasis remains a limited disease. However, in some people, it may spread to involve other body sites over time.

Psoriasis has a fluctuating course of flares and remission but seldom completely subsides. Patients may describe the skin as highly sensitive, and itching can be severe. Bleeding may occur if the lesions are scratched. The skin can be painful, particularly if joints are involved. It may be aggravated by environmental, emotional, or infectious factors. As part of the diagnostic workup, determine what, if any, therapies have been used by the patient and how effective they have been. Abruptly stopping corticosteroid therapy for psoriasis or adding a known irritant medication might lead to sudden worsening.

Physical examination

The typical appearance of psoriasis is of erythematous, circumscribed, scaly papules and plaques on elbows, knees, extensor surfaces of limbs, and scalp. To help differentiate from eczema, examine the scalp, behind the ears, and the nails for pitting.



Nail psoriasis - pitted nails
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

Subtypes of psoriasis have a typical presentation:[2]

Plaque psoriasis

• Raised inflamed plaque lesions with a superficial silvery-white scaly eruption. The scale may be scraped away to reveal inflamed and sometimes friable skin beneath.



Plaque psoriasis on legs
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on back
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on knee
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on foot
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on scalp
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

Guttate psoriasis

• Widespread, erythematous, fine, scaly papules (water drop appearance) on trunk, arms, and legs. The lesions often erupt after an upper respiratory infection.



Guttate psoriasis

Pustular psoriasis

- Acute generalized pustular psoriasis (von Zumbusch): rare, severe, urgent.
- Palmoplantar pustulosis: chronic involvement of hands and feet.



Pustular psoriasis

From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

Erythroderma (erythrodermic psoriasis)

• Generalized erythema with fine scaling. It is often associated with pain, irritation, and sometimes severe itching.



Erythroderma

Psoriasis Area and Severity Index (PASI)

PASI is the most widely used tool to measure severity and extent of psoriasis.[47] is a tool that can be used to measure severity and extent of psoriasis. It is a composite score grading severity in four body regions according to erythema, scaling, thickness, and the total area of skin affected. Severity of each of erythema, scaling, and thickness is graded from 0 to 4, and the extent of body surface area involvement in each body region is graded categorically from 1 to 6. The final composite score ranges from 0 to 72, with a higher score indicating a greater severity of psoriasis. A PASI of 10 or above indicates severe disease.[48] Online PASI calculators are available.

Other useful tools

- Body surface area: evaluates the extension on the body surface area without considering lesion features like erythema and scaling.
- Physician Global Assessment: a qualitative evaluation of the overall disease severity that is not sensitive to modification of disease severity over time (a rough estimation).

• Dermatology Life Quality Index: useful in assessing the impact of the disease on patient quality of

History and exam

Key diagnostic factors skin lesions (common)

• Typically erythematous, circumscribed scaly papules and plaques on elbows, knees, extensor surfaces of limbs, scalp, and, less commonly, nails, ears, and umbilical region.



Nail psoriasis - pitted nails

From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission

• In plaque psoriasis, there are raised inflamed plaque lesions with a superficial silvery-white scaly eruption. The scale may be scraped away to reveal inflamed and sometimes friable skin beneath.[2] Pinpoint bleeding points are known as Auspitz sign.



Plaque psoriasis on legs
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on back
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on knee
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



Plaque psoriasis on foot
From the collection of Professor Tsu-Yi Chuang, MD, MPH, FAAD; used with permission



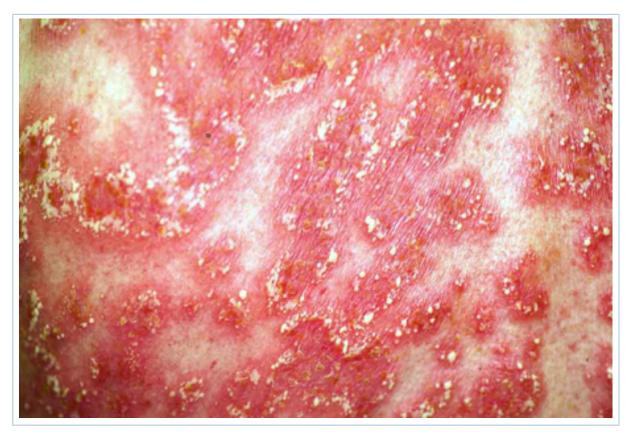
Plaque psoriasis on scalp

• In guttate psoriasis, there are widespread, erythematous, fine, scaly papules (water drop appearance) on trunk, arms, and legs. The lesions often erupt after an upper respiratory infection. [2]



Guttate psoriasis

• In pustular psoriasis, acute generalized pustular psoriasis (von Zumbusch) is rare, severe, and urgent; palmoplantar pustulosis affects palms and soles and is chronic.[2]



Pustular psoriasis

• In erythroderma (erythrodermic psoriasis), there is generalized erythema with fine scaling. It is often associated with pain, irritation, and sometimes severe itching.[2]



Erythroderma

Other diagnostic factors

family history (common)

- Most people with psoriasis have a positive family history.[11] [16]
- Studies of monozygotic twins, linkage studies, and genome-wide association studies provide evidence that psoriasis has a genetic predisposition.[11] [12] [13]

joint swelling or pain (common)

- Psoriatic arthritis occurs in 20% of people with psoriasis and can point towards a diagnosis of cutaneous psoriasis.[3]
- Psoriatic arthritis has several presentations including joint pain, tendinitis, enthesitis, or dactylitis.
 In most cases arthritis presents after the onset of cutaneous psoriasis, but it may be a presenting sign.[49]
- Risk factors for psoriatic arthritis include early age at first presentation, female sex, polyarticular involvement, and genetic predisposition.[50]

Risk factors

Strong

genetic

- Most people with psoriasis have a positive family history.[11] [16]
- Studies of monozygotic twins, linkage studies, and genome-wide association studies provide evidence that psoriasis has a genetic predisposition.[11] [12] [13]
- Psoriasis has been linked to a number of genes, with the strongest association to those involved in the immune response, particularly IL23R, IL12B, and tumor necrosis factor (TNF)-alpha.[14]

infection

Guttate psoriasis often is observed subsequent to upper respiratory infection, such as streptococcal
pharyngitis. It may also be associated with HIV infection. Viral infection, immunization, and any
intercurrent illness have been linked to flares of guttate and plaque psoriasis.[16] [19]

local trauma

• Trauma, such as surgical scars and injection sites, may result in the appearance of new psoriatic lesions at the site of injury.[29] This is known as the Koebner phenomenon.

medications

- Several medications may induce or exacerbate preexisting psoriasis (the incidence of psoriasis exacerbation is generally greater than that of psoriasis induction), including antihypertensives and lithium.[30]
- The latency period between drug ingestion and psoriasis flares varies, and can be considerable for certain medications.[31] [32]

Weak

stress

• Stress and sleep deprivation are recognized to exacerbate psoriasis. Stress reduction techniques may be useful in managing exacerbations.[33] [34]

smoking

 Systematic reviews and meta-analyses have found that that smoking is a risk factor for the development of psoriasis.[35] [36] • Risk increases the greater the number of cigarettes smoked per day and in longer durations of smoking.[35]

ethnicity

• Psoriasis is reported to be twice as common in white people than in black people.[7] [37]

alcohol

- Alcohol consumption may be associated with increased risk of psoriasis.[38] [39]
- Heavy alcohol intake exacerbates psoriasis and complicates treatment by increasing the inflammatory response, altering pharmacology, and potentially reducing adherence to medication.[40] [41]

greater body mass index (BMI)

- Obesity is more prevalent in people with psoriasis (30% to 40%) compared with the general population.[42]
- Obesity as measured by BMI, waist circumference, waist-to-hip ratio, and weight gain is associated with increased risk of psoriasis and exacerbation of preexisting psoriasis.[43] [44] [45] [46]

Investigations

1st test to order

Test	Result
clinical diagnosis	features of psoriasis
 Usually no tests are necessary. 	

Other tests to consider

Test	Result
 skin biopsy Order skin biopsy only when diagnosis is in doubt. Biopsy does not always show classic pathologic features. 	intraepidermal spongiform pustules and Munro neutrophilic microabscess within the stratum corneum; in addition to these classic features, others include focal parakeratosis and epidermal acanthosis with dilated capillaries within dermal papillae

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Eczema	Dry, scaly, red skin sometimes with excoriations visible (scratch marks). Exacerbations may be associated with skin infection causing weeping or oozing skin. The border of eczema is usually less well-defined than a plaque of psoriasis.	Skin biopsy shows changes consistent with atopic dermatitis.
Pityriasis rosea	 More common in children. Lesions may show features of guttate psoriasis but are in a characteristic Christmas tree-shaped distribution. Usually subsides within 8 weeks. 	Clinical diagnosis is usually sufficient.
Seborrheic dermatitis	 Scaly eruptions usually limited to scalp, eyebrows, paranasal region, ears, and chest, but can be widespread. Scales are fine, not lamellar. 	Skin biopsy shows changes consistent with seborrheic dermatitis.
Mycosis fungoides	 Usually presents with patches and plaques on the lower half of the body but can be widespread. Does not involve joints. 	Skin biopsy shows atypical lymphocytes and Pautrier abscess.
Tinea corporis	Annular scaly patches.	Skin scraping or biopsy confirms diagnosis.
Diaper dermatitis	Oozy, weepy.Only in diaper region.	Clinical diagnosis is usually sufficient.
Onychomycosis	Only involves nails.	Culture of nail shows fungus.
Squamous cell cancer/ actinic keratosis	 Actinic keratosis or actinic field change often affect the forehead and dorsal aspect of hands, which are less common sites for psoriasis. Usually presents at an older age. 	Skin biopsy shows proliferating atypical squamous cells.
Lichen planus	 Violaceous papules. Oral mucosa is more likely to be involved than in psoriasis. 	Skin biopsy shows lichenoid lymphocyte infiltrates under epidermis.

Condition	Differentiating signs / symptoms	Differentiating tests
Lichen simplex chronicus	 Usually limited to a few areas easily reached by hands. Lesions are thick and mostly without scaly or desquamated appearance 	Skin biopsy shows chronic dermatitis with epidermal acanthosis.
Subcorneal pustular dermatosis	 A differential for pustular psoriasis. Pustular lesions are subcorneal and in annular/ serpiginous forms, present on the abdomen, axillae, and groin. 	Culture of pustules shows no bacteria. Skin biopsy shows predominantly neutrophilic perivascular infiltrate; minimal spongiosis.
Keratoderma blennorrhagicum (reactive arthritis)	Lesions are circular, scaly, scalloped-edged hyperkeratotic psoriasiform papules and plaques, which are sometimes painful and pustular (at the center of lesions); appear on soles and toes, and, less commonly, legs, palms, scalp, and penis.	Skin biopsy may be done, but may show overlapping features with psoriasis.

Criteria

Psoriasis Area and Severity Index (PASI) score[47]

Composite score grading severity of psoriasis in four body regions according to erythema, scaling, thickness, and the total area of skin affected. Severity of each of erythema, scaling, and thickness is graded from 0 to 4, and the extent of body surface area involvement in each body region is graded categorically from 1 to 6. The final composite score ranges from 0 to 72, with a higher score indicating a greater severity of psoriasis. Online PASI calculators are available.

Physician Global Assessment (PGA)

The PGA was introduced in 1998 by a Food and Drug Administration panel as the preferred tool to assess and record the severity of disease in clinical studies.[47] Typically rates a patient's disease from "clear" to "severe" or "very severe".

Body surface area (BSA)

BSA percentage is a rapid and easy method to score psoriasis; BSA >10% is considered severe disease.[51]

Approach

The aim of treating psoriasis is to decrease the percentage of body surface involved (aiming for complete disease clearance) in as short a time as possible, and to maintain remission. Effectiveness of therapy is usually monitored by both a disease severity tool such as the Psoriasis Area and Severity Index (PASI) and a quality-of life index, usually the Dermatology Life Quality Index.[48]

Mild psoriasis

Topical treatments are the mainstay of therapy.[52] [53]

Choice of formulation depends on the area of cover (e.g., lotion for scalp; cream for moist weeping lesions; and ointment for dry, lichenified, or scaly lesions).

For patients with limited psoriasis involvement, start with topical corticosteroids and a topical vitamin D analog.[54] [55] [56] [Evidence A] Topical calcineurin inhibitors are second-line agents. Emollients may be considered by those averse to pharmacologic options.

Topical corticosteroids

- A topical corticosteroid in combination with a vitamin D analog is more effective in treating disease than either treatment alone.[55] [57]
- Combination therapy may help to reduce potential adverse effects associated with extensive use of topical corticosteroids.
- The potency of topical corticosteroid used is determined by the extent of disease and the responsiveness of the patient to medications. Low-potency treatments are appropriate for lesions on the face or intertriginous areas.[58]
- The combination product halobetasol/tazarotene has been approved for the treatment of plaque psoriasis in adults in some countries.

Topical vitamin D analogs

- Agents such as calcipotriene bind with vitamin D-selective receptors and inhibit the hyperproliferation and abnormal differentiation of keratinocytes characteristic of psoriatic lesions.[58]
- Calcipotriene has a relatively slow onset of action and its maximal effect is after 6 to 8 weeks.
 A two-compound formulation with betamethasone dipropionate appears to be superior to other topicals in scalp psoriasis and psoriasis vulgaris.[59] [60]
- Topical vitamin D analogs can be used alone for chronic therapy when psoriasis is under good control or when treatment needs to be applied long-term to the face or intertriginous areas.

Topical calcineurin inhibitors

 Tacrolimus or pimecrolimus are often used as second-line agents in the treatment of psoriasis, especially facial, flexural, and genital psoriasis; however, this use is off-label.[61] [62]

Moderate to severe psoriasis

Treatment options for moderate to severe psoriasis include phototherapy, conventional systemic therapy (including methotrexate, cyclosporine, or acitretin), apremilast, fumaric acid esters, and biologic therapy.[63]

Treatment should be supervised by a dermatologist.[64]

Phototherapy

- Phototherapy for moderate to severe psoriasis includes narrow-band UVB or PUVA.[65]
- Phototherapy is an effective treatment for psoriasis with skin clearance rates of 50% to 75% with narrow-band UVB, and up to 85% with PUVA.[66]
- Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.
- Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning
 if the dose is not adequately controlled. There is a small increased risk of skin cancer; the risk is
 higher in Fitzparick skin types I and II.

Conventional systemic therapy

Methotrexate

- A folic acid antagonist that works as an antiproliferative and anti-inflammatory agent that is considered a first-line systemic drug.
- Methotrexate may increase the incidence of liver fibrosis in people who are overweight or who have diabetes.[67]
- Folic acid is usually co-prescribed with methotrexate to minimize adverse effects (such as gastrointestinal symptoms and deranged liver function tests).[68]
- Subcutaneous methotrexate may be used in people who fail to respond to oral therapy or have nausea with oral treatment.

Cyclosporine

- Suppresses T cells and pro-inflammatory cytokines (such as interleukin 2), inhibits antigenpresenting capacity of Langerhans cells, and impedes mast cell function of degranulation and cytokine production.
- Cyclosporine is an effective treatment for psoriasis but has significant adverse effects, such as nephrotoxicity and hypertension.[69] It is, therefore, generally reserved for very extensive psoriasis requiring rescue to bring disease severity under relative control.
- Long-term use (i.e., >12 months) is not recommended.

· Acitretin

- An oral retinoid chemically related to vitamin A that helps to regulate epithelial cell growth.
- Moderately effective in many cases and often combined with other treatments.
- Do not use oral retinoids in women of childbearing age, as they are teratogenic.
- Monitor liver function and blood lipid concentration.

Apremilast

- An oral phosphodiesterase-4 inhibitor that works by modulating cyclic adenosine monophosphate levels, which in turn down-regulates inflammatory cytokines including tumor necrosis factor (TNF)-alpha and interleukins 23 and 17.
- Clinical trials have shown apremilast to have modest efficacy in patients with moderate to severe psoriasis.[70] [71] [72]

- Common adverse events included nausea, diarrhea, nasopharyngitis, and upper respiratory tract infection.[70] [71] [72]
- · Apremilast should be used with caution in patients with a history of depression.
- · Fumaric acid esters
 - Fumaric acid esters have immunosuppressive and anti-inflammatory properties.
 - Licensed for moderate to severe psoriasis in European countries. In the UK, dimethyl fumarate is licensed for the treatment of moderate to severe plaque psoriasis in adults.
 - Not approved in the US for cutaneous psoriasis but may be prescribed off-label in the US and other countries.[73] [74] [75] [76]

Biologic therapy

Biologics have been transformative in the management of psoriasis, clearing widespread severe disease and improving psoriatic arthritis. They act at a cellular level and target particular steps in the immunologic processes key to psoriasis activity.

A "living" (regularly updated) Cochrane network meta-analysis has demonstrated that all biologics are effective in improving psoriasis (90% or 90% improvement in PASI compared with baseline).[77] At class level, the biologic treatments that target interleukin (IL)-17, IL-12/23, IL-23, and TNF-alpha were significantly more effective than the small molecules and conventional systemic agents.[77]

The results from another network meta-analysis of randomized controlled trials suggest that brodalumab, guselkumab, ixekizumab, and risankizumab are associated with the highest PASI response rates for both short- and long-term therapy.[78]

Rare adverse effects include drug-induced lupus (associated with TNF-alpha inhibitors) and *Candida* infections (with IL-17 inhibitors, typically mucocutaneous).[79]

Tuberculosis screening (e.g., tuberculin skin test, interferon-gamma release assay, asking about exposure and travel history, and chest x-ray) is recommended prior to initiation of biologic therapy.[79] [80] Screening prior to initiation also includes an HIV and hepatitis B/C test.[79] [80]

All biologics are given as subcutaneous injections (patients administer themselves) except infliximab, which is given as an intravenous infusion.

- TNF-alpha inhibitors
 - Include adalimumab, etanercept, infliximab, certolizumab.[81] [82] [83] [84] [85] [86] [87]
 - If clinically needed, certolizumab may be used in pregnancy.
- Interleukin-12/23 inhibitors
 - Ustekinumab: a human monoclonal antibody that inhibits interleukins 12 and 23.[88] [89] [90]
 - Guselkumab: a monoclonal antibody that inhibits IL-23; believed to provide similar health benefits to ixekizumab and secukinumab.[92] [93]
 - Risankizumab: a human monoclonal antibody that targets IL-23; significantly improved symptoms of moderate to severe psoriasis in clinical trials.[94]

- Tildrakizumab: an IL-23 antagonist approved for the treatment of moderate to severe plaque psoriasis; efficacious when compared with placebo and etanercept in two phase 3 trials.[95]
- · Interleukin-17 inhibitors
 - Secukinumab: a human monoclonal antibody; efficacious in clearing psoriasis plaques. [96]
 - Ixekizumab: a monoclonal antibody; clinical trial data indicate that ixekizumab is highly effective in the treatment of moderate to severe psoriasis for up to 60 weeks of treatment.[99]
 - Brodalumab: a monoclonal antibody that targets the IL-17 receptor, blocking the signaling pathway of interleukins 17A, 17F, and 25. Appears to be well tolerated and efficacious over a 2-year period.[100] [101] [102]

Principles of biologic therapy management

When considering a biologic agent, factors to take into account include:[64]

- The goal of therapy (e.g., Physician Global Assessment, PASI, or body surface area)
- Disease phenotype and pattern of activity
- · Disease severity and impact
- Individual factors including age, comorbidities, conception plans, and body mass index.

Biologic therapy in patients with comorbid conditions

In patients with multiple sclerosis, TNF-alpha inhibitors are not recommended, while IL-17 inhibitors and ustekinumab are recommended first-line.

In patients with hepatitis B infection or latent tuberculosis, ustekinumab and IL-17 inhibitors are recommended, while TNF-alpha inhibitors should be used with caution.[103]

Biosimilars

Biosimilars are available for some biologic agents. The International Psoriasis Council has published a consensus statement to guide prescribing of biosimilars (generic agents highly similar to the originator biologic agent that can be prescribed at reduced cost).[104]

A 2021 systematic review in a small sample of psoriasis patients determined that switching between reference adalimumab and biosimilars has no impact on efficacy, safety, and immunogenicity.[105]

Erythrodermic psoriasis

Patients with erythrodermic psoriasis may need admission to hospital for intense topical treatment, fluid replacement, and electrolyte monitoring. Rapid and aggressive control is essential.

Initial treatment is often with cyclosporine for around 3 weeks to manage the flare. Patients who are more stable can be started with a biologic agent (e.g., a TNF-alpha inhibitor, ustekinumab).

Guttate psoriasis

The recommended treatment approach for guttate psoriasis largely mirrors the strategies employed for plaque psoriasis. Important differences include investigating for an infectious trigger, which may include a throat swab for streptococcal infection and a screen for HIV.

First-line treatment is phototherapy; oral systemic therapies (e.g., cyclosporine, methotrexate, acitretin) are second- and third-line options.[65] [68] [69] [106] [107] [108] Cyclosporine is often prescribed first if guttate psoriasis is widespread and has not responded to phototherapy.[64]

Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.

Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; risk is higher in Fitzparick skin types I and II.

Pustular psoriasis

Pustular psoriasis may require hospital admission if widespread. Fluid replacement, electrolyte monitoring, and supportive care is required for patients with extensive disease.

Pustular psoriasis may be treated with intestine topical therapy, acitretin, or a combination of acitretin and phototherapy. Other systemic agents such as methotrexate and cyclosporine may be prescribed. Cases are managed on a case-by-case basis under the supervision of a dermatologist.

Managing patients with comorbidities

Comorbidities in patients with psoriasis contribute to poorer health outcomes and have a significant health economic burden. Guidelines encourage physicians to address comorbidities when managing psoriasis.[109] [110]

Screen people with moderate to severe psoriasis for comorbidities annually. The most common comorbidities associated with psoriasis are hyperlipidemia, hypertension, obesity, type 2 diabetes, and depression.[111] [112]

People with psoriasis are also more likely to have non-alcoholic fatty liver disease and liver fibrosis, which may impact treatment with methotrexate.[113]

Management of psoriasis during the COVID-19 pandemic

- The International Psoriasis Council is recording data on psoriasis and the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic and will provide updates to the global dermatology community.[114]
- Data suggests that treatments for psoriasis, including biologics, do not alter the risk of acquiring COVID-19 or having worse outcomes. It is recommended that patients who are not infected continue their biologic or oral therapies in most cases.[115] [116]
- Established risk factors (being older, being male, being of nonwhite ethnicity, and having comorbidities) have been associated with higher hospitalization rates.[117]
- Infection with COVID-19 may cause a flare of psoriasis. Resumption of psoriasis treatments withheld during SARS-CoV-2 infection should be decided on a case-by-case basis.[115]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoin	g		(summary)
plaque			
	mild	1st	topical therapies
	moderate to severe	1st	phototherapy
		1st	methotrexate
		1st	cyclosporine
		1st	acitretin
		1st	apremilast
		1st	biologic agent
		2nd	fumaric acid esters
	erythrodermic	1st	cyclosporine or biologic agent
guttate			
		1st	phototherapy
		2nd	cyclosporine
		2nd	methotrexate
		3rd	acitretin
pustular			
		1st	supportive care, phototherapy, or systemic agents

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing

plaque

-- ■ mild

1st topical therapies

Primary options

» hydrocortisone topical: (2.5%) apply sparingly to the affected area(s) two to four times daily

-or-

» triamcinolone topical: (0.025 or 0.1%) apply sparingly to the affected area(s) two to four times daily

-or-

» betamethasone dipropionate topical: (0.05%) apply sparingly to the affected area(s) once or twice daily

-or-

» clobetasol topical: (0.05%) apply sparingly to the affected area(s) twice daily for a maximum of 2 weeks, maximum 50 g/week

--AND/OR--

» calcipotriene topical: (0.005%) apply sparingly to the affected area(s) once or twice daily

OR

» calcipotriene/betamethasone dipropionate topical: apply sparingly to the affected area(s) once daily for up to 4 weeks

OR

» halobetasol/tazarotene topical: (0.01%/0.045%) apply sparingly to the affected area(s) once daily, maximum 50 g/ week

Secondary options

» tacrolimus topical: (0.1%) apply sparingly to the affected area(s) twice daily

OR

» pimecrolimus topical: (1%) apply sparingly to the affected area(s) twice daily

- » Topical treatments are the mainstay of therapy.[52] [53]
- » Choice of formulation depends on the area of cover (e.g., lotion for scalp; cream for moist weeping lesions; and ointment for dry, lichenified, or scaly lesions).
- » For patients with limited psoriasis involvement, start with topical corticosteroids and a topical vitamin D analog.[54] [55] [56] [Evidence A] Topical calcineurin inhibitors are second-line agents. Emollients may be considered by those averse to pharmacologic options.
- » Topical corticosteroid: a topical corticosteroid in combination with a vitamin D analog is more effective in treating disease than either treatment alone.[55] [57] Combination therapy may help to reduce potential adverse effects associated with extensive use of topical corticosteroids. The potency of topical corticosteroid used is determined by the extent of disease and the responsiveness of the patient to medications. Low-potency treatments are appropriate for lesions on the face or intertriginous areas.[58] The combination product halobetasol/tazarotene has been approved for the treatment of plaque psoriasis in adults in some countries.
- "> Topical vitamin D analogs: agents such as calcipotriene bind with vitamin D-selective receptors and inhibit the hyperproliferation and abnormal differentiation of keratinocytes characteristic of psoriatic lesions. [58] Calcipotriene has a relatively slow onset of action and its maximal effect is after 6 to 8 weeks. A two-compound formulation with betamethasone dipropionate appears to be superior to other topicals in scalp psoriasis and psoriasis vulgaris. [59] [60] Topical vitamin D analogs can be used alone for chronic therapy when psoriasis is under good control or when treatment needs to be applied long-term to the face or intertriginous areas.
- » Topical calcineurin inhibitors: tacrolimus or pimecrolimus are often used as second-line agents in the treatment of psoriasis, especially facial, flexural, and genital psoriasis; however, this use is off-label.[61] [62]

moderate to severe

1st phototherapy

- » Phototherapy for moderate to severe psoriasis includes narrow-band UVB or PUVA.[65]
- » Phototherapy is an effective treatment for psoriasis with skin clearance rates of 50% to

75% with narrow-band UVB, and up to 85% with PUVA.[66]

- » Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.
- » Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; the risk is higher in Fitzparick skin types I and II.

1st methotrexate

Primary options

- » methotrexate: 10-25 mg orally/ subcutaneously once weekly on the same day of each week
- » A folic acid antagonist that works as an antiproliferative and anti-inflammatory agent that is considered a first-line systemic drug.
- » Methotrexate may increase the incidence of liver fibrosis in people who are overweight or who have diabetes.[67]
- » Folic acid is usually co-prescribed with methotrexate to minimize adverse effects (such as gastrointestinal symptoms and deranged liver function tests).[68]
- » Subcutaneous methotrexate may be used in people who fail to respond to oral therapy or have nausea with oral treatment.

1st cyclosporine

Primary options

- » cyclosporine modified: 2.5 to 4 mg/kg/day orally given in 2 divided doses
- » Cyclosporine suppresses T cells and proinflammatory cytokines (such as interleukin 2), inhibits antigen-presenting capacity of Langerhans cells, and impedes mast cell function of degranulation and cytokine production.
- » Cyclosporine is an effective treatment for psoriasis but has significant adverse effects, such as nephrotoxicity and hypertension.[69] It is, therefore, generally reserved for very extensive psoriasis requiring rescue to bring disease severity under relative control.
- » Long-term use (i.e., >12 months) is not recommended.

1st acitretin

Primary options

- » acitretin: 25-50 mg orally once daily
- » An oral retinoid chemically related to vitamin A that helps to regulate epithelial cell growth. Moderately effective in many cases and often combined with other treatments.
- » Do not use oral retinoids in women of childbearing age, as they are teratogenic.
- » Monitor liver function and blood lipid concentration.

1st apremilast

Primary options

- » apremilast: 10 mg orally once daily in the morning on day 1, followed by 10 mg in the morning and 10 mg in the evening on day 2, then 10 mg in the morning and 20 mg in the evening on day 3, then 20 mg in the morning and 20 mg in the evening on day 4, then 20 mg in the morning and 30 mg in the evening on day 5, then 30 mg twice daily thereafter
- » An oral phosphodiesterase-4 inhibitor that works by modulating cyclic adenosine monophosphate levels, which in turn downregulates inflammatory cytokines including tumor necrosis factor (TNF)-alpha and interleukins 23 and 17.
- » Clinical trials have shown apremilast to have modest efficacy in patients with moderate to severe psoriasis.[70] [71] [72]
- » Common adverse events included nausea, diarrhea, nasopharyngitis, and upper respiratory tract infection.[70] [71] [72]
- » Apremilast should be used with caution in patients with a history of depression.

1st biologic agent

Primary options

» adalimumab: 80 mg subcutaneously on day 1, followed by 40 mg every other week starting 1 week after initial dose

OR

» etanercept: 50 mg subcutaneously twice weekly for 3 months, followed by either 50

mg once weekly or 25 mg twice weekly, each dose should be 3-4 days apart

OR

» infliximab: 5 mg/kg intravenously at weeks 0, 2, 6, and then every 8 weeks thereafter

OR

» certolizumab pegol: 400 mg subcutaneously every 2 weeks
Can consider giving 400 mg subcutaneously at weeks 0, 2, and 4, then reducing dose to 200 mg every 2 weeks starting from week 6 in patients who weigh ≤90kg, as these patients may achieve an acceptable response with the lower dose.

OR

» ustekinumab: patient weight ≤100kg: 45 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter; patient weight >100kg: 90 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter

OR

» guselkumab: 100 mg subcutaneously at weeks 0 and 4, then every 8 weeks thereafter

OR

» risankizumab: 150 mg subcutaneously at weeks 0 and 4, and then every 12 weeks thereafter

OR

» tildrakizumab: 100 mg subcutaneously at weeks 0 and 4, then every 12 weeks thereafter

OR

» secukinumab: 300 mg subcutaneously at weeks 0, 1, 2, 3, and 4, followed by 300 mg every 4 weeks thereafter

OR

» ixekizumab: 160 mg subcutaneously at week 0, followed by 80 mg at weeks 2, 4, 6, 8, 10, and 12, then 80 mg every 4 weeks thereafter

OR

- » brodalumab: 210 mg subcutaneously at weeks 0, 1, and 2, followed by 210 mg every 2 weeks thereafter; consider discontinuing treatment if inadequate response within 12-16 weeks
- » Biologics have been transformative in the management of psoriasis, clearing widespread severe disease and improving psoriatic arthritis. They act at a cellular level and target particular steps in the immunologic processes key to psoriasis activity.
- » A "living" (regularly updated) Cochrane network meta-analysis has demonstrated that all biologics are effective in improving psoriasis (90% or 90% improvement in Psoriasis Area and Severity Index [PASI] compared with baseline).[77] At class level, the biologic treatments that target interleukin (IL)-17, IL-12/23, IL-23, and tumor necrosis factor (TNF)alpha were significantly more effective than the small molecules and conventional systemic agents.[77]
- » The results from another network metaanalysis of randomized controlled trials suggest that brodalumab, guselkumab, ixekizumab, and risankizumab are associated with the highest PASI response rates for both short- and longterm therapy.[78]
- » Rare adverse effects include drug-induced lupus (associated with TNF-alpha inhibitors) and *Candida* infections (with IL-17 inhibitors, typically mucocutaneous).[79]
- » Tuberculosis screening (e.g., tuberculin skin test, interferon-gamma release assay, asking about exposure and travel history, and chest x-ray) is recommended prior to initiation of biologic therapy.[79] [80] Screening prior to initiation also includes an HIV and hepatitis B/C test.[79] [80]
- » All biologics are given as subcutaneous injections (patients administer themselves) except infliximab, which is given as an intravenous infusion.
- » TNF-alpha inhibitors: these include adalimumab, etanercept, infliximab,

MANAGEMENT

Ongoing

certolizumab.[81] [82] [83] [84] [85] [86] [87] If clinically needed, certolizumab may be used in pregnancy.

- » Interleukin-12/23 inhibitors: ustekinumab is a human monoclonal antibody that inhibits interleukins 12 and 23.[88] [89] [90] [91] Guselkumab is a monoclonal antibody that inhibits IL-23, and is believed to provide similar health benefits to ixekizumab and secukinumab.[92] [93] Risankizumab is a human monoclonal antibody that targets IL-23 and significantly improved symptoms of moderate to severe psoriasis in clinical trials.[94] Tildrakizumab is an IL-23 antagonist approved for the treatment of moderate to severe plaque psoriasis, and was efficacious when compared with placebo and etanercept in two phase 3 trials.[95]
- » Interleukin-17 inhibitors: secukinumab is a human monoclonal antibody that is efficacious in clearing psoriasis plaques.[96] [97] [98] Ixekizumab is a monoclonal antibody; clinical trial data indicate it is highly effective in the treatment of moderate to severe psoriasis for up to 60 weeks of treatment.[99] Brodalumab is a monoclonal antibody that targets the IL-17 receptor, blocking the signaling pathway of interleukins 17A, 17F, and 25; it appears to be well tolerated and efficacious over a 2-year period.[100] [101] [102]

2nd fumaric acid esters

Primary options

- » dimethyl fumarate: consult specialist for guidance on dose
- » Fumaric acid esters have immunosuppressive and anti-inflammatory properties.
- » Licensed for moderate to severe psoriasis in European countries. In the UK, dimethyl fumarate is licensed for the treatment of moderate to severe plaque psoriasis in adults.
- » Not approved in the US for cutaneous psoriasis but may be prescribed off-label in the US and other countries.[73] [74] [75] [76]

1st cyclosporine or biologic agent

Primary options

» cyclosporine modified: 2.5 to 4 mg/kg/day orally given in 2 divided doses

Secondary options

····

erythrodermic

» adalimumab: 80 mg subcutaneously on day 1, followed by 40 mg every other week starting 1 week after initial dose

OR

» etanercept: 50 mg subcutaneously twice weekly for 3 months, followed by either 50 mg once weekly or 25 mg twice weekly, each dose should be 3-4 days apart

OR

infliximab: 5 mg/kg intravenously at weeks0, 2, 6, and then every 8 weeks thereafter

OR

- » ustekinumab: patient weight ≤100kg: 45 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter; patient weight >100kg: 90 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter
- » Patients with erythrodermic psoriasis may need admission to hospital for intense topical treatment, fluid replacement, and electrolyte monitoring. Rapid and aggressive control is essential.
- » Initial treatment is often with cyclosporine for around 3 weeks to manage the flare. Patients who are more stable can be started with a biologic agent (e.g., a tumor necrosis factor [TNF]-alpha inhibitor, ustekinumab).

guttate

1st phototherapy

- » The recommended treatment approach for guttate psoriasis largely mirrors the strategies employed for plaque psoriasis. Important differences include investigating for an infectious trigger, which may include a throat swab for streptococcal infection and a screen for HIV.
- » First-line treatment is phototherapy. Phototherapy for moderate to severe psoriasis includes narrow-band UVB or PUVA.[65] Phototherapy is an effective treatment for psoriasis with skin clearance rates of 50% to 75% with narrow-band UVB, and up to 85% with PUVA.[66]

- » Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.
- » Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; risk is higher in Fitzparick skin types I and II.

2nd cyclosporine

Primary options

- » cyclosporine modified: 2.5 to 4 mg/kg/day orally given in 2 divided doses
- » Cyclosporine suppresses T cells and proinflammatory cytokines (such as interleukin 2), inhibits antigen-presenting capacity of Langerhans cells, and impedes mast cell function of degranulation and cytokine production.
- » Cyclosporine is an effective treatment for psoriasis but has significant adverse effects, such as nephrotoxicity and hypertension.[69] It is, therefore, generally reserved for very extensive psoriasis requiring rescue to bring disease severity under relative control.
- » Long-term use (i.e., >12 months) is not recommended.

2nd methotrexate

Primary options

- » methotrexate: 10-25 mg orally/ subcutaneously once weekly on the same day of each week
- » A folic acid antagonist that works as an antiproliferative and anti-inflammatory agent that is considered a first-line systemic drug.
- » Methotrexate may increase the incidence of liver fibrosis in people who are overweight or who have diabetes.[67]
- » Folic acid is usually co-prescribed with methotrexate to minimize adverse effects (such as gastrointestinal symptoms and deranged liver function tests).[68]

3rd acitretin

Primary options

» acitretin: 25-50 mg orally once daily

- » An oral retinoid chemically related to vitamin A that helps to regulate epithelial cell growth. Moderately effective in many cases and often combined with other treatments.
- » Do not use oral retinoids in women of childbearing age, as they are teratogenic.
- » Monitor liver function and blood lipid concentration.

pustular

1st supportive care, phototherapy, or systemic agents

Primary options

» acitretin: 25-50 mg orally once daily

Secondary options

» cyclosporine modified: 2.5 to 4 mg/kg/day orally given in 2 divided doses

OR

- » methotrexate: 10-25 mg orally/ subcutaneously once weekly on the same day of each week
- » Pustular psoriasis may require hospital admission if widespread. Fluid replacement, electrolyte monitoring, and supportive care is required for patients with extensive disease.
- » Pustular psoriasis may be treated with intestine topical therapy, acitretin, or a combination of acitretin and phototherapy.
- » Other systemic agents such as methotrexate and cyclosporine may be prescribed. Cases are managed on a case-by-case basis under the supervision of a dermatologist.

Emerging

Tapinarof

Tapinarof, a small-molecule topical aryl hydrocarbon receptor (AhR) agonist, is the first topical novel chemical entity (corticosteroid-free) treatment to be approved by the US Food and Drug Administration (FDA) in 25 years for adults with any severity of plaque psoriasis. Two identical phase 3 randomized controlled trials demonstrated tapinarof significantly reduced the severity of plaque psoriasis, compared with vehicle, in patients with mild to severe plaque psoriasis at 12 weeks.[118] Patients who completed the 12-week trial were eligible to be included in a 40-week phase 3 open-label trial with a 4-week follow up.[119] The trial reported that 41% of patients achieved complete disease clearance (physician global assessment [PGA] score 0) and that 58% of patients who entered the trial with PGA ≥2 achieved PGA of 0 or 1. The mean duration of remission for patients who achieved PGA 0 was 130 days. The most frequent adverse effects were folliculitis, contact dermatitis, and upper respiratory tract infection.[119]

Tofacitinib

Tofacitinib, an oral Janus kinase inhibitor, is approved for use in patients with psoriatic arthritis and has been evaluated in phase 3 randomized controlled trials of patients with moderate to severe chronic plaque psoriasis.[120] [121] [122] Systematic reviews conclude that tofacitinib is effective in reducing signs and symptoms of chronic plaque psoriasis.[123] [124] Tofacitinib appeared to be associated with an increased risk for infection (including serious infections and herpes zoster) in some studies.[120] [122] [123]

Deucravacitinib

Deucravacitinib, a first-in-class oral selective tyrosine kinase 2 (TYK2) inhibitor, has been approved by the FDA to treat moderate to severe plaque psoriasis in adults. Deucravacitinib improved clearing of psoriasis compared with placebo in two phase 3, double-blind, randomized controlled trials in patients with moderate to severe plaque psoriasis at 16 and 24 weeks.[125] [126] Efficacy continued to improve after 24 weeks, with 82% of patients who achieved Psoriasis Area and Severity Index (PASI) score of 75 with deucravacitinib at week 24 maintaining their response at week 52 in the first trial.[125] The second of the two phase 3 trials included a randomized withdrawal and retreatment after week 24, 80% of patients who continued with deucravacitinib maintained PASI 75 response compared with 31% of patients who were withdrawn.[126] The results of the trials were limited by a lack of cultural diversity, further phase 3 trials are underway.[127]

Bimekizumab

Bimekizumab, an immunoglobulin G1 monoclonal antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A and IL-17AF, is approved in Europe for the treatment of moderate to severe plaque psoriasis in adults who are candidates for systemic therapy. The FDA is currently reviewing an application for approval. In patients with moderate to severe psoriasis, treatment with bimekizumab resulted in greater skin clearance than treatment with secukinumab over 16 and 48 weeks; bimekizumab was associated with oral candidiasis.[128] A study on the 2-year safety profile of bimekizumab reported that the treatment was well tolerated with no increase in adverse effects with longer treatment duration for patients with plaque psoriasis, apart from an increased risk of mild to moderate oral candidiasis.[129]

Spesolimab

Spesolimab, a monoclonal antibody that inhibits the activation of the interleukin-36 receptor (IL-36R), is the first FDA-approved treatment specifically to treat generalized pustular psoriasis (GPP) flares in adults. The approval is based on the results of one phase 2 randomized controlled trial which demonstrated that spesolimab significantly increased lesion clearance at one week compared with placebo in patients with GPP.[130] Infections occurred in 47% of patients treated with spesolimab at 12 weeks, and anti-drug antibodies were detected in 46% of the patients treated with spesolimab. Longer and larger trials are needed to identify the efficacy and risks of spesolimab treatment.[130]

Roflumilast

Roflumilast, a topical phosphodiesterase type 4 (PDE-4) inhibitor, is approved by the FDA for the treatment of plaque psoriasis. A phase 2b, double-blind randomized controlled trial demonstrated that roflumilast significantly improved disease clearance at 6 weeks, compared with vehicle, in patients with plaque psoriasis.[131] Further trials are needed.

Patient discussions

Educate patients on diagnosis, step-wise therapeutic options, and the importance of adherence to both topical and systemic medications.

Patients should be weighed and have their blood pressure checked annually. If they are overweight or obese, they should be counseled on the potential impact on their psoriasis and overall health.[103]

Tell patients about support groups and services available including:

- [National Psoriasis Foundation] (https://www.psoriasis.org)
- [Psoriasis Association (UK)] (https://www.psoriasis-association.org.uk)

Monitoring

Monitoring

Frequency of monitoring depends on disease severity and the type of therapy that the patients are taking. In general, monitor patients with moderate to severe psoriasis at 3- to 6-month intervals. Clinicians need to ensure that treatment goals are met and continually monitor for drug safety.

Complications

Complications	Timeframe	Likelihood
cardiovascular complications	long term	high
Patients with psoriasis or psoriatic arthritis have an increased incidence of cardiovascular disease (e.g., myocardial infarction and stroke) and cardiovascular risk factors such as smoking, hypertension, and metabolic syndrome.[35] [141] [142] [143]		
There is also evidence to suggest that psoriasis is associated wi of diabetes and obesity, particularly in patients with severe psori known.[147] [148]		
psoriatic arthritis	long term	medium
Up to 30% of people with psoriasis have psoriatic arthritis.[1] The arthritis most commonly involves fingers, hands, toes, and feet, and, less commonly, knees, elbows, and axial and sacroiliac joints.		
depression	variable	medium
Patients with psoriasis have an increased risk of depression, anxiety, and suicidality.[139]		
lymphoma	variable	low
Patients with psoriasis have an increased risk of developing lymphoma. The cause of this association is unknown, but is thought to result from pathophysiology, treatment, or a combination of both.[140] Absolute risk is low.		
secondary infection	variable	low
Pruritus may lead to skin breaks from scratching		1

Prognosis

The exact natural history of psoriasis is poorly understood. However, psoriasis is generally considered a chronic disease with a fluctuating course. Long-term control with topical and/or systemic medications is necessary for many patients.

Treatment guidelines

International

Guideline on the management of vulval conditions (https://iusti.org/guidelines-resources/) [132]

Published by: International union against sexually transmitted infections Last published: 2021

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with topical therapy and alternative medicine modalities for psoriasis severity measures (https://www.aad.org/member/clinical-quality/guidelines/psoriasis) [133]

Published by: American Academy of Dermatology Last published: 2021

Joint AAD-NPF guidelines of care for the management of psoriasis with systemic non-biological therapies (https://www.aad.org/member/clinical-quality/guidelines/psoriasis) [75]

Published by: American Academy of Dermatology Last published: 2020

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis in pediatric patients (https://www.aad.org/member/clinical-quality/guidelines/psoriasis) [134]

Published by: American Academy of Dermatology Last published: 2020

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with phototherapy (https://www.aad.org/member/clinical-quality/quidelines/psoriasis) [66]

Published by: American Academy of Dermatology Last published: 2019

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with biologics (https://www.aad.org/member/clinical-quality/guidelines/psoriasis) [103]

Published by: American Academy of Dermatology Last published: 2019

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities (https://www.aad.org/member/clinical-quality/guidelines/psoriasis) [109]

Published by: American Academy of Dermatology Last published: 2019

Treatment targets for plaque psoriasis (https://www.jaad.org/article/S0190-9622(16)30909-4/fulltext) [135]

Published by: National Psoriasis Foundation Last published: 2017

EuroGuiDerm guideline on the systemic treatment of psoriasis vulgaris – part 2: specific clinical and comorbid situations (https://www.eadv.org/clinical-guidelines) [136]

Published by: European Dermatology Forum Last published: 2021

International

British Association of Dermatologists guidelines for biologic therapy for psoriasis (https://www.bad.org.uk/healthcare-professionals/clinical-standards/clinical-guidelines) [64]

Published by: British Association of Dermatologists (UK)

Last published: 2020

EuroGuiDerm guideline for the systemic treatment of psoriasis vulgaris – part 1: treatment and monitoring recommendations (https://www.eadv.org/clinical-guidelines) [137]

Published by: European Dermatology Forum

Last published: 2020

French guidelines on the use of systemic treatments for moderate-to-severe psoriasis in adults (https://www.sfdermato.org/page-24-recommandations) [138]

Published by: French Society of Dermatology Last published: 2019

Psoriasis: assessment and management of psoriasis (https://www.nice.org.uk/guidance/CG153) [110]

Published by: National Institute for Health and Care Excellence (UK) Last published: 2017

Online resources

- 1. National Psoriasis Foundation (https://www.psoriasis.org) (external link)
- 2. Psoriasis Association (UK) (https://www.psoriasis-association.org.uk) (external link)

Evidence tables

How do topical corticosteroids affect outcomes in people with scalp psoriasis?



This table is a summary of the analysis reported in a Cochrane Clinical Answer that focuses on the above important clinical question.



View the full source Cochrane Clinical Answer (https://www.cochranelibrary.com/cca/doi/10.1002/cca.1344/full)

Evidence A *

Confidence in the evidence is high or moderate to high where GRADE has been performed and the intervention is more effective/beneficial than the comparison for key outcomes.

Population: Adolescents and adults with scalp psoriasis

Intervention: Topical corticosteroid a

Comparison: Topical vitamin D (calcipotriene) a

Outcome	Effectiveness (BMJ rating) [†]	Confidence in evidence (GRADE)
Severity score (total sign score [TSS])	See Notes ^b	GRADE assessment not performed for this outcome
Clearance of symptoms (investigator assessed)	Favors intervention	Moderate
Clearance of symptoms (patient assessed)	Favors intervention	GRADE assessment not performed for this outcome
Response to treatment (investigator assessed)	Favors intervention	High
Response to treatment (patient assessed)	Favours intervention	Moderate
At least one adverse event	Occurs more commonly with topical vitamin D compared with topical corticosteroids (favors intervention) ^c	GRADE assessment not performed for this outcome
At least one adverse event	No statistically significant difference d	GRADE assessment not performed for this outcome
Withdrawals due to adverse events	Occurs more commonly with topical vitamin D compared with	Moderate

Outcome	Effectiveness (BMJ rating)	Confidence in evidence (GRADE) [‡]
	topical corticosteroids (favors intervention)	
Disease-free period, duration of response, quality of life	-	None of the studies identified by the review assessed these outcomes

Note

- ^a This evidence table summarizes the findings for the comparison of topical corticosteroids versus topical vitamin D, which is the main comparison as stated in the Cochrane review Summary of Findings table. See the full Cochrane Clinical Answer (CCA) for information on other comparisons (topical corticosteroid versus placebo; topical corticosteroid plus vitamin D versus topical vitamin D).
- ^b Results reported narratively (five RCTs; all trials reported a greater reduction in TSS with topical corticosteroids compared with topical vitamin D).
- ^c At least one adverse event occurred more commonly with topical vitamin D when compared with the following three corticosteroids: 1 mg/mL betamethasone valerate solution; 0.5 mg/g betamethasone dipropionate gel; and 0.05% clobetasol propionate shampoo. Results reported separately as subgroup analyses.
- ^d No statistically significant difference was found when comparing 0.05% clobetasol propionate solution with topical vitamin D; result reported as a subgroup analysis.

* Evidence levels

The Evidence level is an internal rating applied by BMJ Best Practice. See the EBM Toolkit (https://bestpractice.bmj.com/info/evidence-tables/) for details.

Confidence in evidence

- A High or moderate to high
- **B** Moderate or low to moderate
- C Very low or low

† Effectiveness (BMJ rating)

Based on statistical significance, which demonstrates that the results are unlikely to be due to chance, but which does not necessarily translate to a clinical significance.

‡ Grade certainty ratings

High	The authors are very confident that the true effect is similar to the estimated effect.
Moderate	The authors are moderately confident that the true effect is likely to be close to the estimated effect.
Low	The authors have limited confidence in the effect estimate and the true effect may be substantially different.
Very Low	The authors have very little confidence in the effect estimate and the true effect is likely to be substantially different.

BMJ Best Practice EBM Toolkit: What is GRADE? (https://bestpractice.bmj.com/info/toolkit/learn-ebm/what-is-grade/)

Key articles

- Smith CH, Yiu ZZN, Bale T, et al. British Association of Dermatologists guidelines for biologic therapy for psoriasis 2020: a rapid update. Br J Dermatol. 2020 Oct;183(4):628-37. Full text (https://onlinelibrary.wiley.com/doi/10.1111/bjd.19039) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32189327?tool=bestpractice.bmj.com)
- Elmets CA, Lim HW, Stoff B, et al. Joint American Academy of Dermatology National Psoriasis Foundation guidelines of care for the management and treatment of psoriasis with phototherapy. J Am Acad Dermatol. 2019 Sep;81(3):775-804. Full text (https://www.jaad.org/ article/S0190-9622(19)30637-1/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31351884? tool=bestpractice.bmj.com)
- Menter A, Gelfand JM, Connor C, et al. Joint American Academy of Dermatology National Psoriasis Foundation guidelines of care for the management of psoriasis with systemic nonbiologic therapies. J Am Acad Dermatol. 2020 Jun;82(6):1445-86. Full text (https://www.jaad.org/article/ S0190-9622(20)30284-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32119894? tool=bestpractice.bmj.com)
- National Institute for Health and Care Excellence. Psoriasis: assessment and management.
 September 2017 [internet publication]. Full text (https://www.nice.org.uk/guidance/CG153) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/?tool=bestpractice.bmj.com)

References

- Mease PJ, Gladman DD, Papp KA, et al. Prevalence of rheumatologist-diagnosed psoriatic arthritis in patients with psoriasis in European/North American dermatology clinics. J Am Acad Dermatol. 2013 Nov;69(5):729-35. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23981683? tool=bestpractice.bmj.com)
- 2. Griffiths CE, Christophers E, Barker JN, et al. A classification of psoriasis vulgaris according to phenotype. Br J Dermatol. 2007 Feb;156(2):258-62. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17223864?tool=bestpractice.bmj.com)
- 3. Alinaghi F, Calov M, Kristensen LE, et al. Prevalence of psoriatic arthritis in patients with psoriasis: a systematic review and meta-analysis of observational and clinical studies. J Am Acad Dermatol. 2019 Jan;80(1):251-65. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29928910? tool=bestpractice.bmj.com)
- 4. Parisi R, Iskandar IYK, Kontopantelis E, et al. National, regional, and worldwide epidemiology of psoriasis: systematic analysis and modelling study. BMJ. 2020 May 28;369:m1590. Full text (https://www.bmj.com/content/369/bmj.m1590.long) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32467098?tool=bestpractice.bmj.com)

- 5. Gibbs S. Skin disease and socioeconomic conditions in rural Africa: Tanzania. Int J Dermatol. 1996 Sep;35(9):633-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/8876289?tool=bestpractice.bmj.com)
- 6. Danielsen K, Olsen AO, Wilsgaard T, et al. Is the prevalence of psoriasis increasing? A 30-year follow-up of a population-based cohort. Br J Dermatol. 2013 Jun;168(6):1303-10. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23374051?tool=bestpractice.bmj.com)
- 7. Armstrong AW, Mehta MD, Schupp CW, et al. Psoriasis prevalence in adults in the United States. JAMA Dermatol. 2021 Aug 1;157(8):940-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34190957? tool=bestpractice.bmj.com)
- 8. Springate DA, Parisi R, Kontopantelis E, et al. Incidence, prevalence and mortality of patients with psoriasis: a U.K. population-based cohort study. Br J Dermatol. 2017 Mar;176(3):650-8. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5363241) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27579733?tool=bestpractice.bmj.com)
- 9. Mercy K, Paller AS. Practice gaps. Prescribing patterns by dermatologists and primary care providers for pediatric psoriasis: comment on "Trends in pediatric psoriasis outpatient health care delivery in the United States". Arch Dermatol. 2012 Jan;148(1):71-2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22250234?tool=bestpractice.bmj.com)
- 10. Elder JT, Nair RP, Guo SW, et al. The genetics of psoriasis. Arch Dermatol. 1994 Feb;130(2):216-24. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/8304761?tool=bestpractice.bmj.com)
- Lønnberg AS, Skov L, Skytthe A, et al. Heritability of psoriasis in a large twin sample. Br J Dermatol. 2013 Aug;169(2):412-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23574549? tool=bestpractice.bmj.com)
- 12. Deng Y, Chang C, Lu Q. The inflammatory response in psoriasis: a comprehensive review. Clin Rev Allergy Immunol. 2016 Jun;50(3):377-89. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27025861? tool=bestpractice.bmj.com)
- Dand N, Mahil SK, Capon F, et al. Psoriasis and genetics. Acta Derm Venereol. 2020
 Jan 30;100(3):adv00030. Full text (https://www.medicaljournals.se/acta/content/
 html/10.2340/00015555-3384) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31971603?
 tool=bestpractice.bmj.com)
- Prieto-Pérez R, Cabaleiro T, Daudén E, et al. Genetics of psoriasis and pharmacogenetics of biological drugs. Autoimmune Dis. 2013;2013:613086. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/ PMC3771250) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24069534?tool=bestpractice.bmj.com)
- Bowcock AM, Barker JN. Genetics of psoriasis: the potential impact of new therapies. J Am Acad Dermatol. 2003 Aug;49(2 suppl):S51-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/12894126? tool=bestpractice.bmj.com)
- 16. Schon MP, Boehncke WH. Psoriasis. N Engl J Med. 2005 May 5;352(18):1899-912. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15872205?tool=bestpractice.bmj.com)

- 17. Philipp S, Wolk K, Kreutzer S, et al. The evaluation of psoriasis therapy with biologics leads to a revision of the current view of the pathogenesis of this disorder. Expert Opin Ther Targets. 2006 Dec;10(6):817-31. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17105370? tool=bestpractice.bmj.com)
- Mehlis SL, Gordon KB. The immunology of psoriasis and biologic immunotherapy. J Am Acad Dermatol. 2003 Aug;49(2 suppl):S44-50. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/12894125? tool=bestpractice.bmj.com)
- 19. Gudjonsson JE, Thorarinsson AM, Sigurgeirsson B, et al. Streptococcal throat infections and exacerbation of chronic plaque psoriasis: a prospective study. Br J Dermatol. 2003 Sep;149(3):530-4. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/14510985?tool=bestpractice.bmj.com)
- Weinstein GD, McCullough JL, Ross PA. Cell kinetic basis for pathophysiology of psoriasis. J Invest Dermatol. 1985 Dec;85(6):579-83. Full text (https://www.jidonline.org/article/S0022-202X(15)43859-X/pdf)
 Abstract (http://www.ncbi.nlm.nih.gov/pubmed/4067329?tool=bestpractice.bmj.com)
- 21. Nestle FO, Di Meglio P, Qin JZ, et al. Skin immune sentinels in health and disease. Nat Rev Immunol. 2009 Oct;9(10):679-91. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19763149? tool=bestpractice.bmj.com)
- 22. Di Cesare A, Di Meglio P, Nestle FO. The IL-23/Th17 axis in the immunopathogenesis of psoriasis. J Invest Dermatol. 2009 Jun;129(6):1339-50. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19322214? tool=bestpractice.bmj.com)
- 23. Lynde CW, Poulin Y, Vender R, et al. Interleukin 17A: toward a new understanding of psoriasis pathogenesis. J Am Acad Dermatol. 2014 Jul;71(1):141-50. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24655820?tool=bestpractice.bmj.com)
- 24. Martin DA, Towne JE, Kricorian G, et al. The emerging role of IL-17 in the pathogenesis of psoriasis: preclinical and clinical findings. J Invest Dermatol. 2013 Jan;133(1):17-26. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22673731?tool=bestpractice.bmj.com)
- 25. Cheung KL, Jarrett R, Subramaniam S, et al. Psoriatic T cells recognize neolipid antigens generated by mast cell phospholipase delivered by exosomes and presented by CD1a. J Exp Med. 2016 Oct 17;213(11):2399-412. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27670592? tool=bestpractice.bmj.com)
- 26. Lande R, Botti E, Jandus C, et al. The antimicrobial peptide LL37 is a T-cell autoantigen in psoriasis. Nat Commun. 2014 Dec 3;5:5621. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25470744? tool=bestpractice.bmj.com)
- 27. Krueger JG. An autoimmune "attack" on melanocytes triggers psoriasis and cellular hyperplasia. J Exp Med. 2015 Dec 14;212(13):2186. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26666753? tool=bestpractice.bmj.com)

- 28. Arakawa A, Siewert K, Stöhr J, et al. Melanocyte antigen triggers autoimmunity in human psoriasis. J Exp Med. 2015 Dec 14;212(13):2203-12. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26621454? tool=bestpractice.bmj.com)
- 29. Boyd AS, Neldner KH. The isomorphic response of Koebner. Int J Dermatol. 1990 Jul-Aug;29(6):401-10. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/2204607?tool=bestpractice.bmj.com)
- 30. Song G, Yoon HY, Yee J, et al. Antihypertensive drug use and psoriasis: a systematic review, meta-and network meta-analysis. Br J Clin Pharmacol. 2021 Oct 5 [Epub ahead of print]. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34611920?tool=bestpractice.bmj.com)
- 31. Armstrong AW. Psoriasis provoked or exacerbated by medications: identifying culprit drugs.

 JAMA Dermatol. 2014 Sep;150(9):963. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24989499?

 tool=bestpractice.bmj.com)
- 32. Wu S, Han J, Li WQ, et al. Hypertension, antihypertensive medication use, and risk of psoriasis. JAMA Dermatol. 2014 Sep;150(9):957-63. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4184206) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24990147?tool=bestpractice.bmj.com)
- 33. Russo PA, Ilchef R, Cooper AJ. Psychiatric morbidity in psoriasis: a review. Australas J Dermatol. 2004 Aug;45(3):155-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15250891? tool=bestpractice.bmj.com)
- 34. Naldi L, Chatenoud L, Linder D, et al. Cigarette smoking, body mass index, and stressful life events as risk factors for psoriasis: results from an Italian case-control study. J Invest Dermatol.2005

 Jul;125(1):61-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15982303?tool=bestpractice.bmj.com)
- 35. Armstrong AW, Harskamp CT, Dhillon JS, et al. Psoriasis and smoking: a systematic review and meta-analysis. Br J Dermatol. 2014 Feb;170(2):304-14. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24117435?tool=bestpractice.bmj.com)
- 36. Richer V, Roubille C, Fleming P, et al. Psoriasis and smoking: a systematic literature review and metaanalysis with qualitative analysis of effect of smoking on psoriasis severity. J Cutan Med Surg. 2016 May;20(3):221-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26553732?tool=bestpractice.bmj.com)
- 37. Rachakonda TD, Schupp CW, Armstrong AW. Psoriasis prevalence among adults in the United States. J Am Acad Dermatol. 2014 Mar;70(3):512-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24388724?tool=bestpractice.bmj.com)
- 38. Zhu KJ, Zhu CY, Fan YM. Alcohol consumption and psoriatic risk: a meta-analysis of case-control studies. J Dermatol. 2012 Sep;39(9):770-3. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22568495? tool=bestpractice.bmj.com)
- 39. Brenaut E, Horreau C, Pouplard C, et al. Alcohol consumption and psoriasis: a systematic literature review. J Eur Acad Dermatol Venereol. 2013 Aug;27 (Suppl 3):30-5. Full text (https://onlinelibrary.wiley.com/doi/10.1111/jdv.12164) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23845150?tool=bestpractice.bmj.com)

- 40. Cassano N, Vestita M, Apruzzi D, et al. Alcohol, psoriasis, liver disease, and anti-psoriasis drugs. Int J Dermatol. 2011 Nov;50(11):1323-31. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22004481? tool=bestpractice.bmj.com)
- 41. Parisi R, Webb RT, Carr MJ, et al. Alcohol-related mortality in patients with psoriasis: a population-based cohort study. JAMA Dermatol. 2017 Dec 1;153(12):1256-62. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5817445) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28914955?tool=bestpractice.bmj.com)
- 42. Mahil SK, McSweeney SM, Kloczko E, et al. Does weight loss reduce the severity and incidence of psoriasis or psoriatic arthritis? A critically appraised topic. Br J Dermatol. 2019 Nov;181(5):946-53. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30729517?tool=bestpractice.bmj.com)
- 43. Budu-Aggrey A, Brumpton B, Tyrrell J, et al. Evidence of a causal relationship between body mass index and psoriasis: A mendelian randomization study. PLoS Med. 2019 Jan;16(1):e1002739. Full text (https://www.doi.org/10.1371/journal.pmed.1002739) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30703100?tool=bestpractice.bmj.com)
- 44. Aune D, Snekvik I, Schlesinger S, et al. Body mass index, abdominal fatness, weight gain and the risk of psoriasis: a systematic review and dose-response meta-analysis of prospective studies. Eur J Epidemiol. 2018 Dec;33(12):1163-78. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6290660) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29680995?tool=bestpractice.bmj.com)
- 45. Snekvik I, Nilsen TIL, Romundstad PR, et al. Metabolic syndrome and risk of incident psoriasis: prospective data from the HUNT Study, Norway. Br J Dermatol. 2019 Jan;180(1):94-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29904911?tool=bestpractice.bmj.com)
- 46. Davidovici BB, Sattar N, Prinz J, et al. Psoriasis and systemic inflammatory diseases: potential mechanistic links between skin disease and co-morbid conditions. J Invest Dermatol. 2010 Jul;130(7):1785-96. Full text (https://www.jidonline.org/article/S0022-202X(15)34901-0/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20445552?tool=bestpractice.bmj.com)
- 47. Feldman SR, Krueger GG. Psoriasis assessment tools in clinical trials. Ann Rheum Dis. 2005

 Mar;64(suppl 2):ii65-8. Full text (https://ard.bmj.com/content/64/suppl_2/ii65.long) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15708941?tool=bestpractice.bmj.com)
- 48. Cabrera S, Chinniah N, Lock N, et al. Inter-observer reliability of the PASI in a clinical setting. Australas J Dermatol. 2015 May;56(2):100-2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25753553?tool=bestpractice.bmj.com)
- 49. Tillett W, Orbai AM, Ogdie A, et al. GRAPPA-OMERACT initiative to standardise outcomes in psoriatic arthritis clinical trials and longitudinal observational studies. Ann Rheum Dis. 2018 May;77(5):e23. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28747326?tool=bestpractice.bmj.com)
- 50. Reich K, Krüger K, Mössner R, et al. Epidemiology and clinical pattern of psoriatic arthritis in Germany: a prospective interdisciplinary epidemiological study of 1511 patients with plaque-type psoriasis.

 Br J Dermatol. 2009 May;160(5):1040-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19210498? tool=bestpractice.bmj.com)

- 51. Strober B, Ryan C, van de Kerkhof P, et al. Recategorization of psoriasis severity: Delphi consensus from the International Psoriasis Council. J Am Acad Dermatol. 2020 Jan;82(1):117-22. Full text (https://www.jaad.org/article/S0190-9622(19)32573-3/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31425723?tool=bestpractice.bmj.com)
- 52. Bailey JW. Topical treatments for chronic plaque psoriasis. Am Family Physician. 2010 Mar 1;81(5):596. Full text (https://www.aafp.org/afp/2010/0301/p596.html) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20187595?tool=bestpractice.bmj.com)
- 53. Chiricozzi A, Pimpinelli N, Ricceri F, et al. Treatment of psoriasis with topical agents: recommendations from a Tuscany consensus. Dermatol Ther. 2017 Nov;30(6). Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28940579?tool=bestpractice.bmj.com)
- 54. Schlager JG, Rosumeck S, Werner RN, et al. Topical treatments for scalp psoriasis. Cochrane Database Syst Rev. 2016 Feb 26;(2):CD009687. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD009687.pub2/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26915340?tool=bestpractice.bmj.com)
- 55. Samarasekera EJ, Sawyer L, Wonderling D, et al. Topical therapies for the treatment of plaque psoriasis: systematic review and network meta-analyses. Br J Dermatol. 2013 May;168(5):954-67. Full text (https://onlinelibrary.wiley.com/doi/10.1111/bjd.12276) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23413913?tool=bestpractice.bmj.com)
- 56. Mason AR, Mason J, Cork M, et al. Topical treatments for chronic plaque psoriasis. Cochrane Database Syst Rev. 2013 Mar 28;(3):CD005028. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD005028.pub3/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23543539?tool=bestpractice.bmj.com)
- 57. Devaux S, Castela A, Archier E, et al. Topical vitamin D analogues alone or in association with topical steroids for psoriasis: a systematic review. J Eur Acad Dermatol Venereol. 2012 May;26 (Suppl 3):52-60. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22512681?tool=bestpractice.bmj.com)
- 58. Lebwohl M. A clinician's paradigm in the treatment of psoriasis. J Am Acad Dermatol.2005 Jul;53(1 suppl 1):S59-69. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15968265?tool=bestpractice.bmj.com)
- 59. van de Kerkhof P, de Peuter R, Ryttov J, et al. Mixed treatment comparison of a two-compound formulation (TCF) product containing calcipotriol and betamethasone dipropionate with other topical treatments in psoriasis vulgaris. Curr Med Res Opin. 2011 Jan;27(1):225-38. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21142833?tool=bestpractice.bmj.com)
- 60. Bottomley JM, Taylor RS, Ryttov J, et al. The effectiveness of two-compound formulation calcipotriol and betamethasone dipropionate gel in the treatment of moderately severe scalp psoriasis: a systematic review of direct and indirect evidence. Curr Med Res Opin. 2011 Jan;27(1):251-68.

 Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21142838?tool=bestpractice.bmj.com)
- 61. Guenther L, Lynde C, Poulin Y. Off-label use of topical calcineurin inhibitors in dermatologic disorders. J Cutan Med Surg. 2019 Sep/Oct;23(4 Suppl):27S-34S. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31476936?tool=bestpractice.bmj.com)

- 62. Lebwohl M, Freeman AK, Chapman MS, et al. Tacrolimus ointment is effective for facial and intertriginous psoriasis. J Am Acad Dermatol. 2004 Nov;51(5):723-30. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15523350?tool=bestpractice.bmj.com)
- 63. Gisondi P, Altomare G, Ayala F, et al. Italian guidelines on the systemic treatments of moderate-to-severe plaque psoriasis. J Eur Acad Dermatol Venereol. 2017 May;31(5):774-90. Full text (https://onlinelibrary.wiley.com/doi/full/10.1111/jdv.14114) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28244153?tool=bestpractice.bmj.com)
- 64. Smith CH, Yiu ZZN, Bale T, et al. British Association of Dermatologists guidelines for biologic therapy for psoriasis 2020: a rapid update. Br J Dermatol. 2020 Oct;183(4):628-37. Full text (https://onlinelibrary.wiley.com/doi/10.1111/bjd.19039) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32189327?tool=bestpractice.bmj.com)
- 65. Lapolla W, Yentzer BA, Bagel J, et al. A review of phototherapy protocols for psoriasis treatment. J Am Acad Dermatol. 2011 May;64(5):936-49. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21429620? tool=bestpractice.bmj.com)
- 66. Elmets CA, Lim HW, Stoff B, et al. Joint American Academy of Dermatology National Psoriasis Foundation guidelines of care for the management and treatment of psoriasis with phototherapy. J Am Acad Dermatol. 2019 Sep;81(3):775-804. Full text (https://www.jaad.org/article/S0190-9622(19)30637-1/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31351884?tool=bestpractice.bmj.com)
- 67. van der Kraaij GE, Balak DMW, Busard CI, et al. Highlights of the updated Dutch evidence- and consensus-based guideline on psoriasis 2017. Br J Dermatol. 2019 Jan;180(1):31-42. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6849803) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30604536?tool=bestpractice.bmj.com)
- 68. Prey S, Paul C. Effect of folic or folinic acid supplementation on methotrexate-associated safety and efficacy in inflammatory disease: a systematic review. Br J Dermatol. 2009 Mar;160(3):622-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18945303?tool=bestpractice.bmj.com)
- 69. Maza A, Montaudie H, Sbidian E, et al. Oral cyclosporin in psoriasis: a systematic review on treatment modalities, risk of kidney toxicity and evidence for use in non-plaque psoriasis. J Euro Acad Dermatol Venereol. 2011;25(suppl 2):19-27. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21388455? tool=bestpractice.bmj.com)
- 70. Papp K, Reich K, Leonardi CL, et al. Apremilast, an oral phosphodiesterase 4 (PDE4) inhibitor, in patients with moderate to severe plaque psoriasis: results of a phase III, randomized, controlled trial (Efficacy and Safety Trial Evaluating the Effects of Apremilast in Psoriasis [ESTEEM] 1). J Am Acad Dermatol. 2015 Jul;73(1):37-49. Full text (https://www.jaad.org/article/S0190-9622(15)01494-2/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26089047?tool=bestpractice.bmj.com)
- 71. Van Voorhees AS, Stein Gold L, Lebwohl M, et al. Efficacy and safety of apremilast in patients with moderate to severe plaque psoriasis of the scalp: results of a phase 3b, multicenter, randomized, placebo-controlled, double-blind study. J Am Acad Dermatol. 2020 Jul;83(1):96-103. Full text (https://

- www.jaad.org/article/S0190-9622(20)30158-4/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32032692?tool=bestpractice.bmj.com)
- 72. Paul C, Cather J, Gooderham M, et al. Efficacy and safety of apremilast, an oral phosphodiesterase 4 inhibitor, in patients with moderate-to-severe plaque psoriasis over 52 weeks: a phase III, randomized controlled trial (ESTEEM 2). Br J Dermatol. 2015 Dec;173(6):1387-99. Full text (https://onlinelibrary.wiley.com/doi/10.1111/bjd.14164) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26357944?tool=bestpractice.bmj.com)
- 73. Atwan A, Ingram JR, Abbott R, et al. Oral fumaric acid esters for psoriasis. Cochrane Database Syst Rev. 2015 Aug 10;2015(8):CD010497. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD010497.pub2/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26258748?tool=bestpractice.bmj.com)
- 74. Nast A, Amelunxen L, Augustin M, et al. S3 guideline for the treatment of psoriasis vulgaris, update: short version part 1 systemic treatment. J Dtsch Dermatol Ges. 2018 May;16(5):645-69. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29750443?tool=bestpractice.bmj.com)
- 75. Menter A, Gelfand JM, Connor C, et al. Joint American Academy of Dermatology National Psoriasis Foundation guidelines of care for the management of psoriasis with systemic nonbiologic therapies. J Am Acad Dermatol. 2020 Jun;82(6):1445-86. Full text (https://www.jaad.org/article/S0190-9622(20)30284-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32119894? tool=bestpractice.bmj.com)
- 76. National Institute for Health and Care Excellence. Dimethyl fumarate for treating relapsing-remitting multiple sclerosis. August 2014 [internet publication]. Full text (https://www.nice.org.uk/guidance/ta320)
- 77. Griffiths CE, Strober BE, van de Kerkhof P, et al. Comparison of ustekinumab and etanercept for moderate-to-severe psoriasis. N Engl J Med. 2010 Jan 14;362(2):118-28. Full text (https://www.nejm.org/doi/10.1056/NEJMoa0810652) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20071701?tool=bestpractice.bmj.com)
- 78. Thaçi D, Blauvelt A, Reich K, et al. Secukinumab is superior to ustekinumab in clearing skin of subjects with moderate to severe plaque psoriasis: CLEAR, a randomized controlled trial. J Am Acad Dermatol. 2015 Sep;73(3):400-9. Full text (https://www.jaad.org/article/S0190-9622(15)01683-7/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26092291?tool=bestpractice.bmj.com)
- 79. Kamata M, Tada Y. Safety of biologics in psoriasis. J Dermatol. 2018 Mar;45(3):279-86. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29226369?tool=bestpractice.bmj.com)
- 80. Deodhar A, Mease PJ, McInnes IB, et al. Long-term safety of secukinumab in patients with moderate-to-severe plaque psoriasis, psoriatic arthritis, and ankylosing spondylitis: integrated pooled clinical trial and post-marketing surveillance data. Arthritis Res Ther. 2019 May 2;21(1):111. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6498580) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31046809?tool=bestpractice.bmj.com)

- 81. Menter A, Tyring SK, Gordon K, et al. Adalimumab therapy for moderate to severe psoriasis: a randomized, controlled phase III trial. J Am Acad Dermatol. 2008 Jan;58(1):106-15. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17936411?tool=bestpractice.bmj.com)
- 82. Paller AS, Siegfried EC, Langley RG, et al; Etanercept Pediatric Psoriasis Study Group. Etanercept treatment for children and adolescents with plaque psoriasis. N Engl J Med.2008 Jan 17;358(3):241-51. Full text (https://www.nejm.org/doi/10.1056/NEJMoa066886) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18199863?tool=bestpractice.bmj.com)
- 83. Carrascosa JM, Rebollo F, Gómez S, et al. Effects of etanercept on the patient-perceived results (PROs) in patients with moderate-to-severe plaque psoriasis: systematic review of the literature and meta-analysis. J Dermatolog Treat. 2018 Dec;29(8):806-11. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29671665?tool=bestpractice.bmj.com)
- 84. Feldman SR, Gottlieb AB, Bala M, et al. Infliximab improves health-related quality of life in the presence of comorbidities among patients with moderate-to-severe psoriasis. Br J Dermatol. 2008 Sep;159(3):704-10. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18627375? tool=bestpractice.bmj.com)
- 85. Saurat JH, Stingl G, Dubertret L, et al. Efficacy and safety results from the randomized controlled comparative study of adalimumab vs. methotrexate vs. placebo in patients with psoriasis (CHAMPION). Br J Dermatol. 2008 Mar;158(3):558-66. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18047523?tool=bestpractice.bmj.com)
- 86. Lebwohl M, Blauvelt A, Paul C, et al. Certolizumab pegol for the treatment of chronic plaque psoriasis: Results through 48 weeks of a phase 3, multicenter, randomized, double-blind, etanercept- and placebo-controlled study (CIMPACT). J Am Acad Dermatol. 2018 Aug;79(2):266-76. Full text (https://www.jaad.org/article/S0190-9622(18)30526-7/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29660425?tool=bestpractice.bmj.com)
- 87. Gordon KB, Warren RB, Gottlieb AB, et al. Long-term efficacy of certolizumab pegol for the treatment of plaque psoriasis: 3-year results from two randomized phase III trials (CIMPASI-1 and CIMPASI-2). Br J Dermatol. 2021 Apr;184(4):652-62. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8247431) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32652544?tool=bestpractice.bmj.com)
- 88. Leonardi CL, Kimball AB, Papp KA, et al. Efficacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 76-week results from a randomised, double-blind, placebo-controlled trial (PHOENIX 1). Lancet 2008 May 17;371(9625):1665-74. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18486739?tool=bestpractice.bmj.com)
- 89. Papp KA, Langley RG, Lebwohl M, et al. Efficacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 52-week results from a randomised, double-blind, placebo-controlled trial (PHOENIX 2). Lancet. 2008 May 17;371(9625):1675-84. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18486740?tool=bestpractice.bmj.com)
- 90. Zhu X, Zheng M, Song M, et al. Efficacy and safety of ustekinumab in Chinese patients with moderate to severe plaque-type psoriasis: results from a phase 3 clinical trial (LOTUS). J Drugs

Dermatol. 2013 Feb;12(2):166-74. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23377389? tool=bestpractice.bmj.com)

- 91. Kimball AB, Papp KA, Wasfi Y, et al. Long-term efficacy of ustekinumab in patients with moderate-to-severe psoriasis treated for up to 5 years in the PHOENIX 1 study. J Eur Acad Dermatol Venereol. 2013 Dec;27(12):1535-45. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23279003? tool=bestpractice.bmj.com)
- 92. National Institute for Health and Care Excellence. Guselkumab for treating moderate to severe plaque psoriasis. June 2018 [internet publication]. Full text (https://www.nice.org.uk/guidance/ta521)
- 93. Reich K, Armstrong AW, Foley P, et al. Maintenance of response through up to 4 years of continuous guselkumab treatment of psoriasis in the VOYAGE 2 phase 3 study. Am J Clin Dermatol. 2020 Dec;21(6):881-90. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32910434? tool=bestpractice.bmj.com)
- 94. Augustin M, Lambert J, Zema C, et al. Effect of risankizumab on patient-reported outcomes in moderate to severe psoriasis: the UltIMMa-1 and UltIMMa-2 randomized clinical trials. JAMA Dermatol. 2020 Dec 1;156(12):1344-53. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7557488) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33052382?tool=bestpractice.bmj.com)
- 95. Reich K, Papp KA, Blauvelt A, et al. Tildrakizumab versus placebo or etanercept for chronic plaque psoriasis (reSURFACE 1 and reSURFACE 2): results from two randomised controlled, phase 3 trials. Lancet. 2017 Jul 15;390(10091):276-88. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28596043? tool=bestpractice.bmj.com)
- 96. Langley RG, Elewski BE, Lebwohl MN, et al. Secukinumab in plaque psoriasis results of two phase 3 trials. Engl J Med. 2014 Jul 24;371(4):326-38. Full text (https://www.nejm.org/doi/10.1056/NEJMoa1314258) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25007392?tool=bestpractice.bmj.com)
- 97. Ordenes-Cavieres G, Andino-Navarrete R. Secukinumab for plaque psoriasis. Medwave. 2018 Nov 30;18(7):e7364. Full text (https://www.medwave.cl/link.cgi/Medwave/PuestaDia/ResEpis/7364)
 Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30507896?tool=bestpractice.bmj.com)
- 98. Torres T, Balato A, Conrad C, et al. Secukinumab drug survival in patients with psoriasis: a multicenter, real-world, retrospective study. J Am Acad Dermatol. 2019 Jul;81(1):273-5. Full text (https://www.jaad.org/article/S0190-9622(19)30290-7/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30790602?tool=bestpractice.bmj.com)
- 99. Gordon KB, Blauvelt A, Papp KA, et al; UNCOVER-1 Study Group; UNCOVER-2 Study Group; UNCOVER-3 Study Group. Phase 3 trials of ixekizumab in moderate-to-severe plaque psoriasis. N Engl J Med. 2016 Jul 28;375(4):345-56. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27299809? tool=bestpractice.bmj.com)
- 100. Papp KA, Reich K, Paul C, et al. A prospective phase III, randomized, double-blind, placebocontrolled study of brodalumab in patients with moderate-to-severe plaque psoriasis. Br J

- Dermatol. 2016 Aug;175(2):273-86. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26914406? tool=bestpractice.bmj.com)
- 101. Lebwohl M, Strober B, Menter A, et al. Phase 3 studies comparing brodalumab with ustekinumab in psoriasis. N Engl J Med. 2015 Oct;373(14):1318-28. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26422722?tool=bestpractice.bmj.com)
- 102. Puig L, Lebwohl M, Bachelez H, et al. Long-term efficacy and safety of brodalumab in the treatment of psoriasis: 120-week results from the randomized, double-blind, placebo- and active comparator-controlled phase 3 AMAGINE-2 trial. J Am Acad Dermatol. 2020 Feb;82(2):352-9. Full text (https://www.jaad.org/article/S0190-9622(19)30899-0/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31175909?tool=bestpractice.bmj.com)
- 103. Menter A, Strober BE, Kaplan DH, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with biologics. J Am Acad Dermatol. 2019 Apr;80(4):1029-72. Full text (https://www.jaad.org/article/S0190-9622(18)33001-9/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30772098?tool=bestpractice.bmj.com)
- 104. Cohen AD, Vender R, Naldi L, et al. Biosimilars for the treatment of patients with psoriasis: a consensus statement from the Biosimilar Working Group of the International Psoriasis Council. JAAD Int. 2020 Nov 23;1(2):224-30. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8361899)
 Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34409344?tool=bestpractice.bmj.com)
- 105. García-Beloso N, Altabás-González I, Samartín-Ucha M, et al. Switching between reference adalimumab and biosimilars in chronic immune-mediated inflammatory diseases: a systematic literature review. Br J Clin Pharmacol. 2021 Oct 8 [Epub ahead of print]. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34622969?tool=bestpractice.bmj.com)
- 106. Chen X, Yang M, Cheng Y, et al. Narrow-band ultraviolet B phototherapy versus broad-band ultraviolet B or psoralen-ultraviolet A photochemotherapy for psoriasis. Cochrane Database Syst Rev. 2013 Oct 23;(10):CD009481. Full text (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD009481.pub2/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24151011?tool=bestpractice.bmj.com)
- 107. Montaudie H, Sbidian E, Paul C, et al. Methotrexate in psoriasis: a systematic review of treatment modalities, incidence, risk factors and monitoring of liver toxicity. J Europ Acad Dermatol Venereol. 2011 May;25(suppl 2):12-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21388454? tool=bestpractice.bmj.com)
- 108. Paul C, Gallini A, Maza A, et al. Evidence-based recommendations on conventional systemic treatments in psoriasis: systematic review and expert opinion of a panel of dermatologists. J Eur Acad Dermatol Venereol. 2011 May;25 (Suppl 2):2-11. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21388453?tool=bestpractice.bmj.com)
- 109. Elmets CA, Leonardi CL, Davis DMR, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities. J Am Acad Dermatol. 2019 Apr;80(4):1073-113. Full text (https://www.jaad.org/article/S0190-9622(18)33002-0/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30772097?tool=bestpractice.bmj.com)

- 110. National Institute for Health and Care Excellence. Psoriasis: assessment and management. September 2017 [internet publication]. Full text (https://www.nice.org.uk/guidance/CG153) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/?tool=bestpractice.bmj.com)
- 111. Shah K, Mellars L, Changolkar A, et al. Real-world burden of comorbidities in US patients with psoriasis. J Am Acad Dermatol. 2017 Aug;77(2):287-92. Full text (https://www.jaad.org/article/S0190-9622(17)30426-7/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28623046? tool=bestpractice.bmj.com)
- 112. Dowlatshahi EA, Wakkee M, Arends LR, et al. The prevalence and odds of depressive symptoms and clinical depression in psoriasis patients: a systematic review and meta-analysis. J Invest Dermatol. 2014 Jun;134(6):1542-51. Full text (https://www.jidonline.org/article/S0022-202X(15)36822-6/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24284419?tool=bestpractice.bmj.com)
- 113. Phan K, Onggo J, Charlton O, et al. Relationship between psoriasis and non-alcoholic fatty liver disease: updated systematic review and adjusted meta-analysis. Australas J Dermatol. 2019 Nov;60(4):e352-5. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30906989? tool=bestpractice.bmj.com)
- 114. International Psoriasis Council. IPC statement on COVID-19 and psoriasis. September 2020 [internet publication]. Full text (https://www.psoriasiscouncil.org/blog/COVID-19-Statement.htm)
- 115. Gelfand JM, Armstrong AW, Bell S, et al. National Psoriasis Foundation COVID-19 task force guidance for management of psoriatic disease during the pandemic: version 1. J Am Acad Dermatol. 2020 Dec;83(6):1704-16. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7471802) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32891785?tool=bestpractice.bmj.com)
- 116. National Institute for Health and Care Excellence. COVID-19 rapid guideline: dermatological conditions treated with drugs affecting the immune response. April 2021 [internet publication]. Full text (https://www.nice.org.uk/guidance/ng169)
- 117. Mahil SK, Dand N, Mason KJ, et al. Factors associated with adverse COVID-19 outcomes in patients with psoriasis-insights from a global registry-based study. J Allergy Clin Immunol. 2021 Jan;147(1):60-71. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7566694) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33075408?tool=bestpractice.bmj.com)
- 118. Lebwohl MG, Stein Gold L, Strober B, et al. Phase 3 trials of tapinarof cream for plaque psoriasis. N Engl J Med. 2021 Dec 9;385(24):2219-29. Full text (https://www.doi.org/10.1056/NEJMoa2103629) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34879448?tool=bestpractice.bmj.com)
- 119. Strober B, Stein Gold L, Bissonnette R, et al. One-year safety and efficacy of tapinarof cream for the treatment of plaque psoriasis: results from the PSOARING 3 trial. J Am Acad Dermatol. 2022 Oct;87(4):800-6. Full text (https://www.doi.org/10.1016/j.jaad.2022.06.1171) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35772599?tool=bestpractice.bmj.com)
- 120. Papp KA, Menter MA, Abe M, et al. Tofacitinib, an oral Janus kinase inhibitor, for the treatment of chronic plaque psoriasis: results from two randomized, placebo-controlled, phase III trials. Br J

- Dermatol. 2015 Oct;173(4):949-61. Full text (https://onlinelibrary.wiley.com/doi/10.1111/bjd.14018) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26149717?tool=bestpractice.bmj.com)
- 121. Bachelez H, van de Kerkhof PC, Strohal R, et al. Tofacitinib versus etanercept or placebo in moderate-to-severe chronic plaque psoriasis: a phase 3 randomised non-inferiority trial. Lancet. 2015 Aug 8;386(9993):552-61. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26051365? tool=bestpractice.bmj.com)
- 122. Asahina A, Etoh T, Igarashi A, et al. Oral tofacitinib efficacy, safety and tolerability in Japanese patients with moderate to severe plaque psoriasis and psoriatic arthritis: a randomized, double-blind, phase 3 study. J Dermatol. 2016 Aug;43(8):869-80. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5067558) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26875540?tool=bestpractice.bmj.com)
- 123. Kerschbaumer A, Smolen JS, Nash P, et al. Points to consider for the treatment of immune-mediated inflammatory diseases with Janus kinase inhibitors: a systematic literature research. RMD Open. 2020 Nov;6(3):e001374. Full text (https://rmdopen.bmj.com/content/6/3/e001374.long) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33188136?tool=bestpractice.bmj.com)
- 124. Tian F, Chen Z, Xu T. Efficacy and safety of tofacitinib for the treatment of chronic plaque psoriasis: a systematic review and meta-analysis. J Int Med Res. 2019 Jun;47(6):2342-50. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6567701) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31096817?tool=bestpractice.bmj.com)
- 125. Armstrong AW, Gooderham M, Warren RB, et al. Deucravacitinib versus placebo and apremilast in moderate to severe plaque psoriasis: efficacy and safety results from the 52-week, randomized, double-blinded, placebo-controlled phase 3 POETYK PSO-1 trial. J Am Acad Dermatol. 2022 Jul 9. Full text (https://www.doi.org/10.1016/j.jaad.2022.07.002) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35820547?tool=bestpractice.bmj.com)
- 126. Clinicaltrials.gov. An investigational study to evaluate experimental medication BMS-986165 compared to placebo and a currently available treatment in participants with moderate-to-severe plaque psoriasis. ClinicalTrials.gov Identifier: NCT03611751. 3 Dec 2021 [internet publication]. Full text (https://clinicaltrials.gov/ct2/show/NCT03611751)
- 127. Clinicaltrials.gov. An investigational study to evaluate experimental medication BMS-986165 compared to placebo in participants with plaque psoriasis in mainland China, Taiwan, and South Korea. Clinicaltrials.gov Identifier: NCT04167462. 8 Mar 2022 [internet publication]. Full text (https://clinicaltrials.gov/ct2/show/NCT04167462)
- 128. Reich K, Warren RB, Lebwohl M, et al. Bimekizumab versus secukinumab in plaque psoriasis. N Engl J Med. 2021 Jul 8;385(2):142-52. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33891380? tool=bestpractice.bmj.com)
- 129. Gordon KB, Langley RG, Warren RB, et al. Bimekizumab safety in patients with moderate to severe plaque psoriasis: pooled results from phase 2 and phase 3 randomized clinical Trials. JAMA Dermatol. 2022 Jul 1;158(7):735-44. Full text (https://www.doi.org/10.1001/jamadermatol.2022.1185) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35544084?tool=bestpractice.bmj.com)

- 130. Bachelez H, Choon SE, Marrakchi S, et al. Trial of spesolimab for generalized pustular psoriasis. N Engl J Med. 2021 Dec 23;385(26):2431-40. Full text (https://www.doi.org/10.1056/NEJMoa2111563) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34936739?tool=bestpractice.bmj.com)
- 131. Lebwohl MG, Papp KA, Stein Gold L, et al. Trial of roflumilast cream for chronic plaque psoriasis. N Engl J Med. 2020 Jul 16;383(3):229-39. Full text (https://www.doi.org/10.1056/NEJMoa2000073)

 Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32668113?tool=bestpractice.bmj.com)
- 132. van der Meijden WI, Boffa MJ, Ter Harmsel B, et al. 2021 European guideline for the management of vulval conditions. J Eur Acad Dermatol Venereol. 2022 Jul;36(7):952-72. Full text (https://www.doi.org/10.1111/jdv.18102) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35411963? tool=bestpractice.bmj.com)
- 133. Elmets CA, Korman NJ, Prater EF, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with topical therapy and alternative medicine modalities for psoriasis severity measures. J Am Acad Dermatol. 2021 Feb;84(2):432-70. Full text (https://www.jaad.org/article/S0190-9622(20)32288-X/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32738429?tool=bestpractice.bmj.com)
- 134. Menter A, Cordoro KM, Davis DMR, et al. Joint American Academy of Dermatology-National Psoriasis Foundation guidelines of care for the management and treatment of psoriasis in pediatric patients. J Am Acad Dermatol. 2020 Jan;82(1):161-201. Full text (https://www.doi.org/10.1016/j.jaad.2019.08.049) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31703821?tool=bestpractice.bmj.com)
- 135. Armstrong AW, Siegel MP, Bagel J, et al. From the Medical Board of the National Psoriasis Foundation: treatment targets for plaque psoriasis. J Am Acad Dermatol. 2017 Feb;76(2):290-8. Full text (https://www.jaad.org/article/S0190-9622(16)30909-4/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27908543?tool=bestpractice.bmj.com)
- 136. Nast A, Smith C, Spuls PI, et al. EuroGuiDerm Guideline on the systemic treatment of psoriasis vulgaris part 2: specific clinical and comorbid situations. J Eur Acad Dermatol Venereol. 2021 Feb;35(2):281-317. Full text (https://onlinelibrary.wiley.com/doi/10.1111/jdv.16926) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33547728?tool=bestpractice.bmj.com)
- 137. Nast A, Smith C, Spuls PI, et al. EuroGuiDerm Guideline on the systemic treatment of psoriasis vulgaris part 1: treatment and monitoring recommendations. J Eur Acad Dermatol Venereol. 2020 Nov;34(11):2461-98. Full text (https://onlinelibrary.wiley.com/doi/10.1111/jdv.16915) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33349983?tool=bestpractice.bmj.com)
- 138. Amatore F, Villani AP, Tauber M, et al. French guidelines on the use of systemic treatments for moderate-to-severe psoriasis in adults. J Eur Acad Dermatol Venereol. 2019 Mar;33(3):464-83. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6593704) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30793796?tool=bestpractice.bmj.com)
- 139. Kurd SK, Troxel AB, Crits-Christoph P, et al. The risk of depression, anxiety, and suicidality in patients with psoriasis: a population-based cohort study. Arch Dermatol. 2010 Aug;146(8):891-5. Full text

- (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2928071) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20713823?tool=bestpractice.bmj.com)
- 140. Gelfand JM, Shin DB, Neimann AL, et al. The risk of lymphoma in patients with psoriasis. J Invest Dermatol. 2006 Oct;126(10):2194-201. Full text (https://www.jidonline.org/article/S0022-202X(15)32634-8/fulltext) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/16741509?tool=bestpractice.bmj.com)
- 141. Armstrong EJ, Harskamp CT, Armstrong AW. Psoriasis and major adverse cardiovascular events: a systematic review and meta-analysis of observational studies. J Am Heart Assoc. 2013 Apr 4;2(2):e000062. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3647278) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23557749?tool=bestpractice.bmj.com)
- 142. Tobin AM, Veale DJ, Fitzgerald O, et al. Cardiovascular disease and risk factors in patients with psoriasis and psoriatic arthritis. J Rheumatol. 2010 Jul;37(7):1386-94. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20472927?tool=bestpractice.bmj.com)
- 143. Armstrong AW, Harskamp CT, Armstrong EJ. Psoriasis and metabolic syndrome: a systematic review and meta-analysis of observational studies. J Am Acad Dermatol. 2013 Apr;68(4):654-62. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23360868?tool=bestpractice.bmj.com)
- 144. Armstrong AW, Harskamp CT, Armstrong EJ. Psoriasis and the risk of diabetes mellitus: a systematic review and meta-analysis. JAMA Dermatol. 2013 Jan;149(1):84-91. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23407990?tool=bestpractice.bmj.com)
- 145. Armstrong AW, Harskamp CT, Armstrong EJ. The association between psoriasis and obesity: a systematic review and meta-analysis of observational studies. Nutr Diabetes. 2012 Dec 3;2(12):e54. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3542430) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23208415?tool=bestpractice.bmj.com)
- 146. Khalid U, Hansen PR, Gislason GH, et al. Psoriasis and new-onset diabetes: a Danish nationwide cohort study. Diabetes Care. 2013 Aug;36(8):2402-7. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3714512) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23491525? tool=bestpractice.bmj.com)
- 147. Neimann AL, Shin DB, Wang X, et al. Prevalence of cardiovascular risk factors in patients with psoriasis. J Am Acad Dermatol. 2006 Nov;55(5):829-35. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17052489?tool=bestpractice.bmj.com)
- 148. Gelfand JM, Neimann AL, Shin DB, et al. Risk of myocardial infarction in patients with psoriasis. JAMA. 2006 Oct 11;296(14):1735-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17032986? tool=bestpractice.bmj.com)

Images



Figure 1: Plaque psoriasis on legs



Figure 2: Plaque psoriasis on back



Figure 3: Plaque psoriasis on knee



Figure 4: Plaque psoriasis on foot



Figure 5: Plaque psoriasis on scalp



Figure 6: Guttate psoriasis



Figure 7: Pustular psoriasis

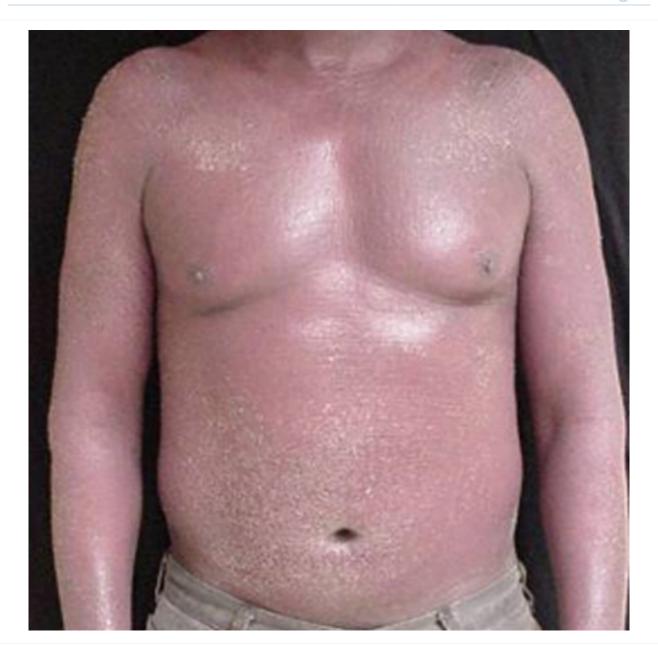


Figure 8: Erythroderma

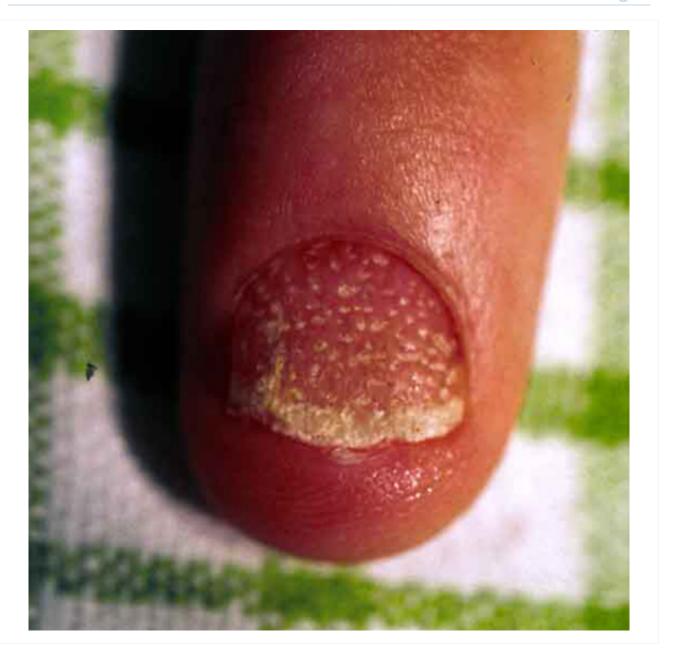


Figure 9: Nail psoriasis - pitted nails



Figure 10: Psoriatic arthritis

Disclaimer

BMJ Best Practice is intended for licensed medical professionals. BMJ Publishing Group Ltd (BMJ) does not advocate or endorse the use of any drug or therapy contained within this publication nor does it diagnose patients. As a medical professional you retain full responsibility for the care and treatment of your patients and you should use your own clinical judgement and expertise when using this product.

This content is not intended to cover all possible diagnosis methods, treatments, follow up, drugs and any contraindications or side effects. In addition, since such standards and practices in medicine change as new data become available, you should consult a variety of sources. We strongly recommend that you independently verify specified diagnosis, treatments and follow-up and ensure it is appropriate for your patient within your region. In addition, with respect to prescription medication, you are advised to check the product information sheet accompanying each drug to verify conditions of use and identify any changes in dosage schedule or contraindications, particularly if the drug to be administered is new, infrequently used, or has a narrow therapeutic range. You must always check that drugs referenced are licensed for the specified use and at the specified doses in your region.

Information included in BMJ Best Practice is provided on an "as is" basis without any representations, conditions or warranties that it is accurate and up to date. BMJ and its licensors and licensees assume no responsibility for any aspect of treatment administered to any patients with the aid of this information. To the fullest extent permitted by law, BMJ and its licensors and licensees shall not incur any liability, including without limitation, liability for damages, arising from the content. All conditions, warranties and other terms which might otherwise be implied by the law including, without limitation, the warranties of satisfactory quality, fitness for a particular purpose, use of reasonable care and skill and non-infringement of proprietary rights are excluded.

Where BMJ Best Practice has been translated into a language other than English, BMJ does not warrant the accuracy and reliability of the translations or the content provided by third parties (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages). BMJ is not responsible for any errors and omissions arising from translation and adaptation or otherwise. Where BMJ Best Practice lists drug names, it does so by recommended International Nonproprietary Names (rINNs) only. It is possible that certain drug formularies might refer to the same drugs using different names.

Please note that recommended formulations and doses may differ between drug databases drug names and brands, drug formularies, or locations. A local drug formulary should always be consulted for full prescribing information.

Treatment recommendations in BMJ Best Practice are specific to patient groups. Care is advised when selecting the integrated drug formulary as some treatment recommendations are for adults only, and external links to a paediatric formulary do not necessarily advocate use in children (and vice-versa). Always check that you have selected the correct drug formulary for your patient.

Where your version of BMJ Best Practice does not integrate with a local drug formulary, you should consult a local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing before prescribing.

Interpretation of numbers

Regardless of the language in which the content is displayed, numerals are displayed according to the original English-language numerical separator standard. For example 4 digit numbers shall not include a comma nor a decimal point; numbers of 5 or more digits shall include commas; and numbers stated to be less than 1 shall be depicted using decimal points. See Figure 1 below for an explanatory table.

BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

Our full website and application terms and conditions can be found here: Website Terms and Conditions.

Contact us

+ 44 (0) 207 111 1105 support@bmj.com

BMJ BMA House Tavistock Square London WC1H 9JR UK

BMJ Best Practice

Contributors:

// Authors:

Andrea Chiricozzi, MD

Assistant Professor

University of Pisa, Consultant Dermatologist, University Hospital of Pisa, Pisa, Italy DISCLOSURES: AC has served as an advisory board member and consultant and has received fees and speaker's honoraria or has participated in clinical trials for AbbVie, Almirall, Biogen, Fresenius Kabi, Leo Pharma, Lilly, Janssen, Novartis, Sanofi Genzyme, and UCB Pharma; AC is also an author of a reference cited in this topic.

// Acknowledgements:

Dr Andrea Chiricozzi would like to gratefully acknowledge Dr April W. Armstrong and Dr Tsu-Yi Chuang, previous contributors to this topic.

DISCLOSURES: TC declares that he has no competing interests. AWA is an investigator and consultant for Abbott, Amgen, and Janssen; AWA is also an author of references cited in this topic.

// Peer Reviewers:

David Burden, MD, FRCP

Western Infirmary, Glasgow

Consultant Dermatologist Dermatology, Glasgow, UK

DISCLOSURES: DB has been reimbursed as a consultant, researcher, and lecturer for Abbott, Leo, Pfizer, Merck, Janssen-Cilag, and Novartis.

Paradi Mirmirani, MD

Physician

Department of Dermatology, Kaiser Permanente Vallejo Medical Center, Vallejo, CA DISCLOSURES: PM declares that she has no competing interests.