PSORIASIS

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Epidemiology

- 2-3% wide variations
- Equator, white
- 16-22,,57-62, 35%20y
- M=F
- Lifetime risks: 4%,25%,68%
- 2 types: type I, type II

Epidemiology

Triggers:

- Streptococcal infection
- HIV infection
- Stress
- Excessive alcohol
- Withdrawal steroids
- Trauma
- Cigarette
- sunlight
- Drugs(Lithium, B blockers, ACE inhibitors ,NSAIDs, Antimalarials , Interferons , TNF-a inhibitors,…)

Cont. Epidemiology

Associated inflammatory diseases:

- Psoriatic arthritis
- IBD
- Autoimmune thyroid dis.
- Diabetes(type I)
- AA
- Vitiligo

Associated diseases:

- Metabolic syndrome
- Chronic obstructive pulmonary dis.
- Chronic kidney dis.

Patients with severe psoriasis die at a younger age than unaffected people and cardiovascular disease accounts for the majority of this excess mortality. There is also an increased risk of peripheral vascular disease and atrial fibrillation.

Non-alcoholic fatty liver disease is the most frequently identified liver pathology, present in up to 50%. Alcoholic liver disease is common.

Pathophysiology

T cells: Th1, Th17, Th22

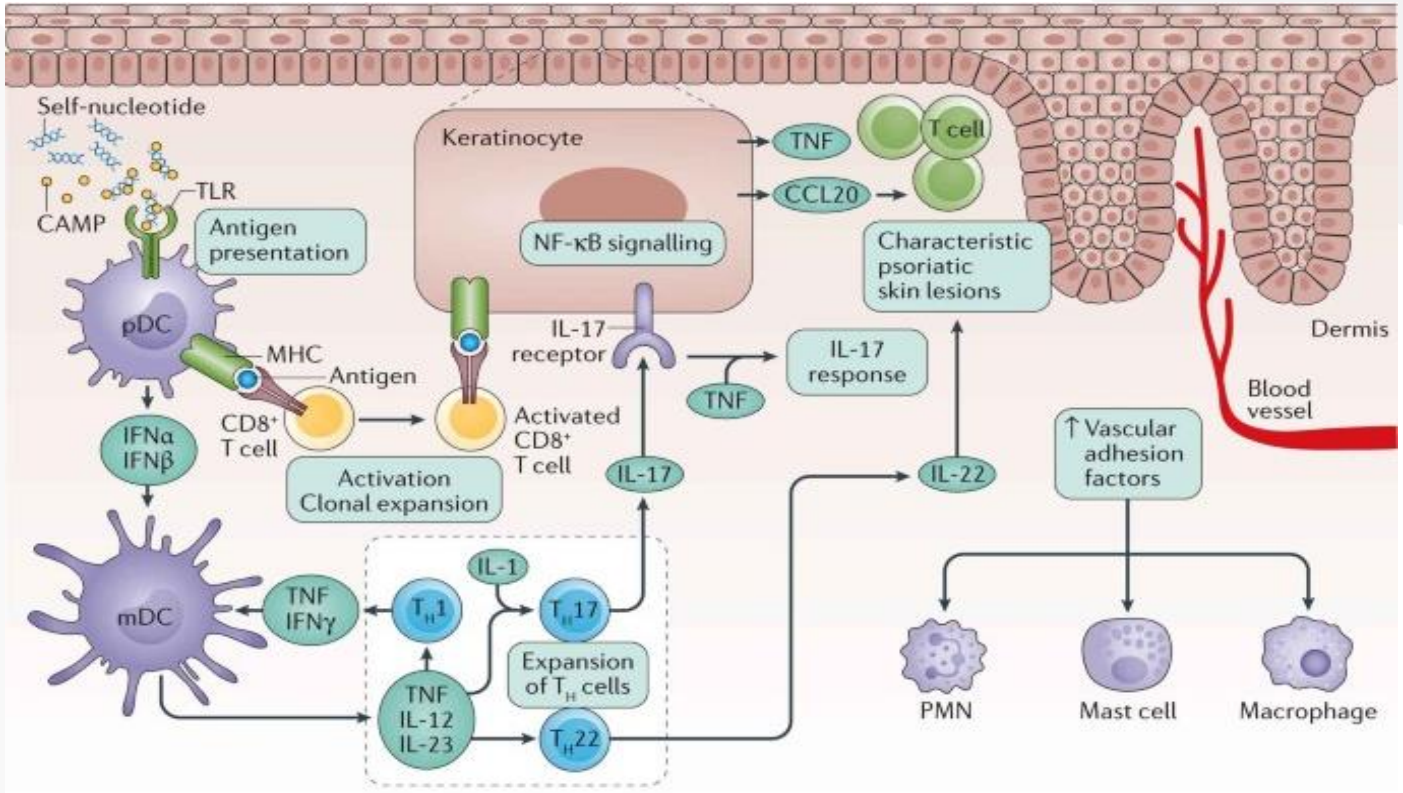
Cytokines:IL2, IL8, IL15,IL22,IL17,IL23,Interferon-Y ,
TNF-a

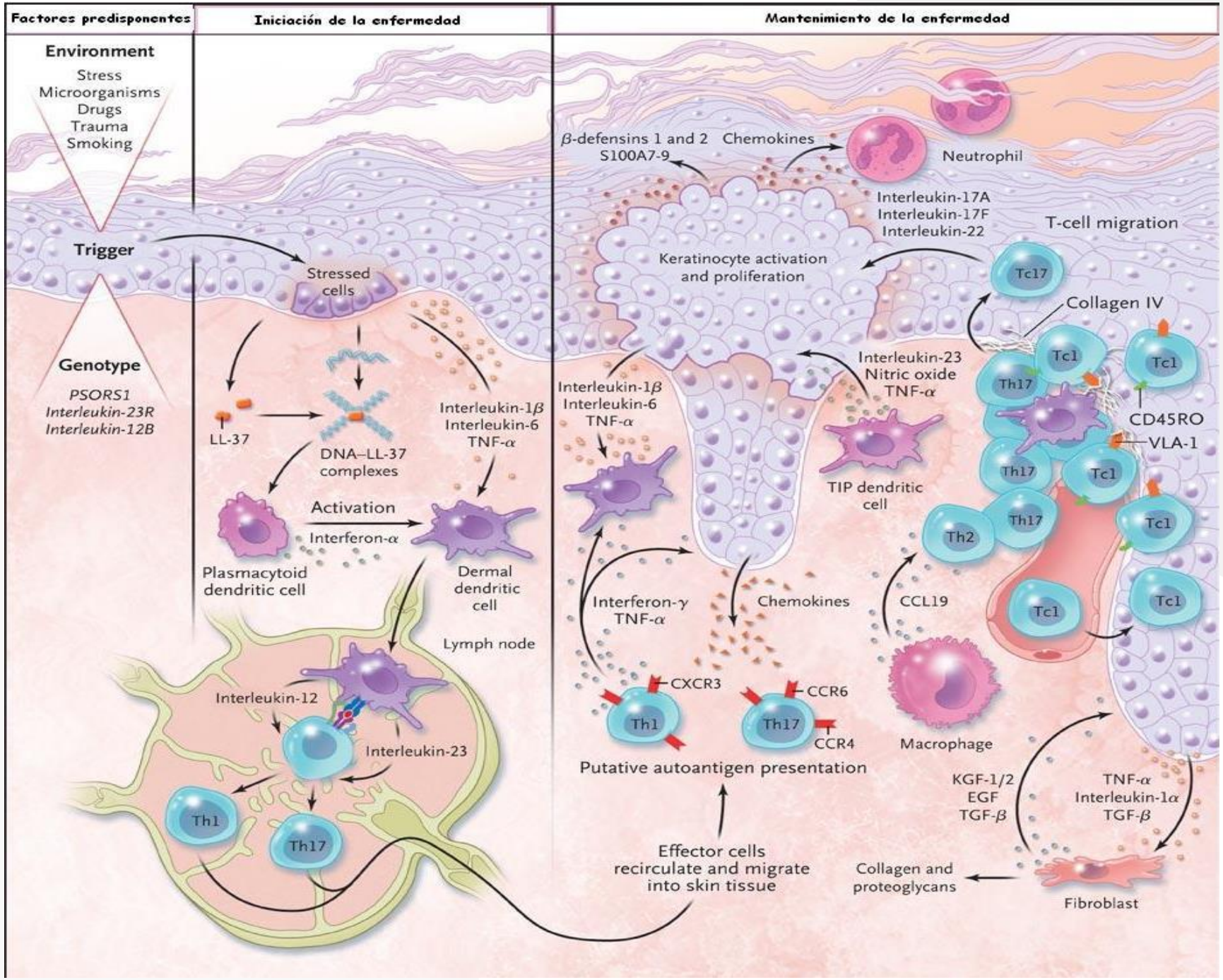
Absence IL4, IL10

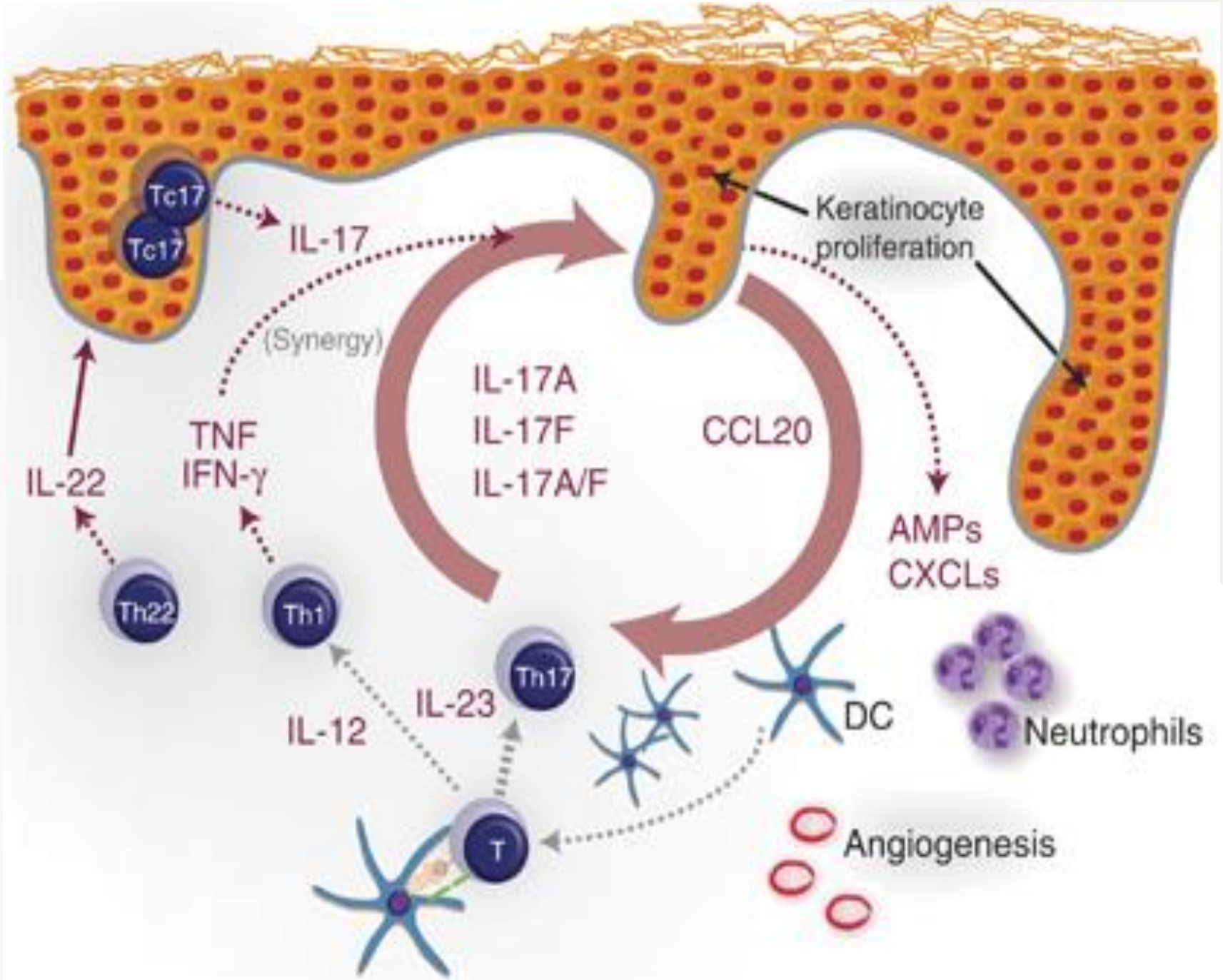
PATHOLOGY: 3 key features:

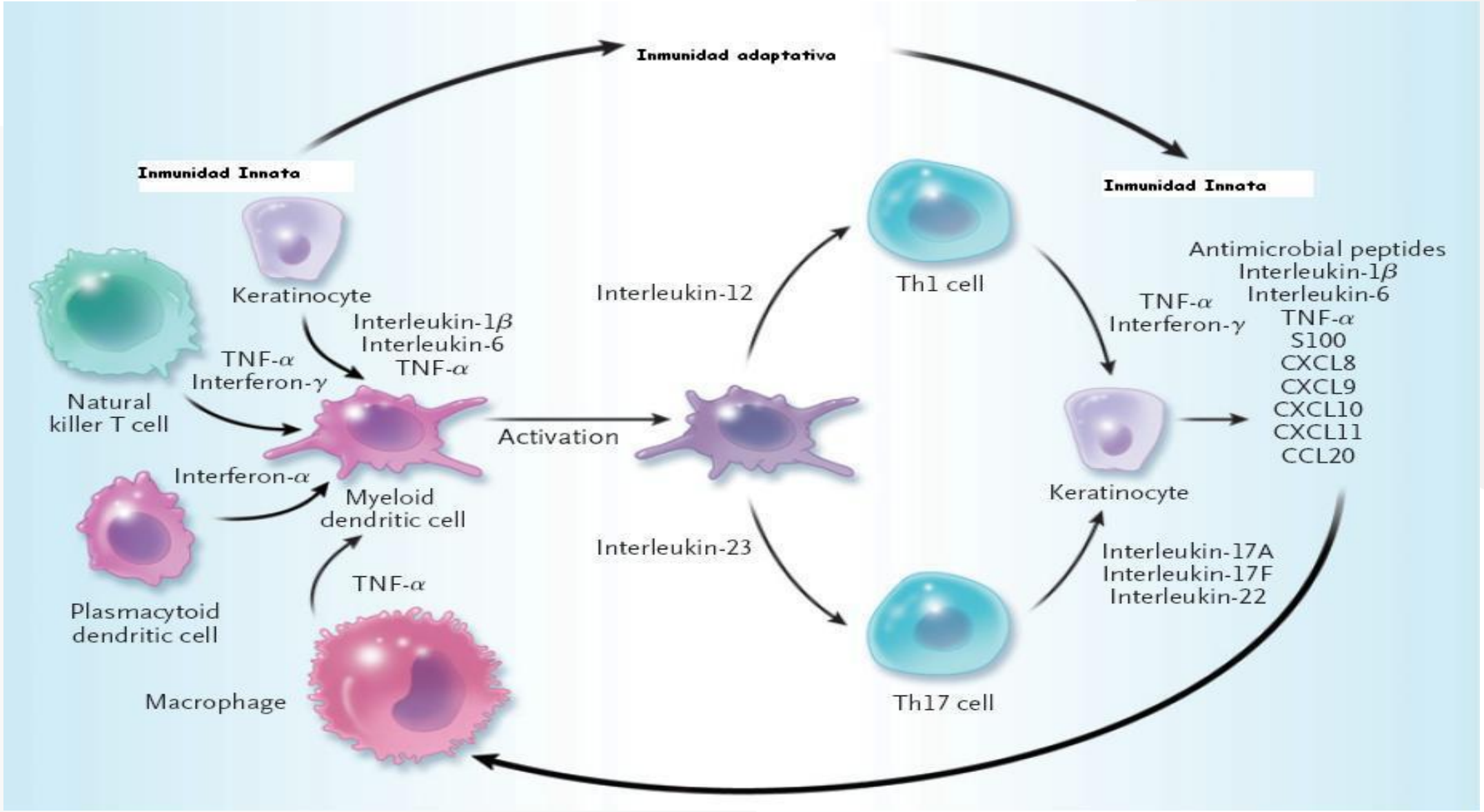
Hyperproliferation
Infiltration
Angiogenesis

No scar




Factores predisponentes
Iniciación de la enfermedad
Mantenimiento de la enfermedad
Environment
Stress
Microorganisms
Drugs
Trauma
Smoking
Trigger
Genotype
PSORS1
Interleukin-23R
Interleukin-12B
Stressed cells
LL-37
DNA–LL-37 complexes
Interleukin-1β
Interleukin-6
TNF-α
Activation
Interferon-α
Plasmacytoid dendritic cell
Dermal dendritic cell
Lymph node
Interleukin-12
Interleukin-23
Th1
Th17
β-defensins 1 and 2
S100A7-9
Chemokines
Neutrophil
Interleukin-17A
Interleukin-17F
Interleukin-22
T-cell migration
Keratinocyte activation and proliferation
Tc17
Collagen IV
Interleukin-23
Nitric oxide
TNF-α
Interleukin-1β
Interleukin-6
TNF-α
TIP dendritic cell
Tc1
Th17
Th2
CD45RO
VLA-1
Interferon-γ
TNF-α
Chemokines
CCL19
CXCR3
CCR6
CCR4
Th1
Th17
Macrophage
Putative autoantigen presentation
KGF-1/2
EGF
TGF-β
TNF-α
Interleukin-1α
TGF-β
Effector cells recirculate and migrate into skin tissue
Collagen and proteoglycans
Fibroblast


Tc17
Tc17
IL-17
Keratinocyte
proliferation
(Synergy)
IL-17A
IL-17F
IL-17A/F
CCL20
TNF
IFN-γ
IL-22
AMPs
CXCLs
Th22
Th1
Th17
IL-23
IL-12
DC
Neutrophils
T
Angiogenesis


Inmunidad adaptativa
Inmunidad Innata
Inmunidad Innata
Keratinocyte
Interleukin-1β
Interleukin-6
TNF-α
TNF-α
Interferon-γ
Natural killer T cell
Interferon-α
Plasmacytoid dendritic cell
Myeloid dendritic cell
TNF-α
Macrophage
Activation
Interleukin-12
Interleukin-23
Th1 cell
Th17 cell
TNF-α
Interferon-γ
Antimicrobial peptides
Interleukin-1β
Interleukin-6
TNF-α
S100
CXCL8
CXCL9
CXCL10
CXCL11
CCL20
Keratinocyte
Interleukin-17A
Interleukin-17F
Interleukin-22

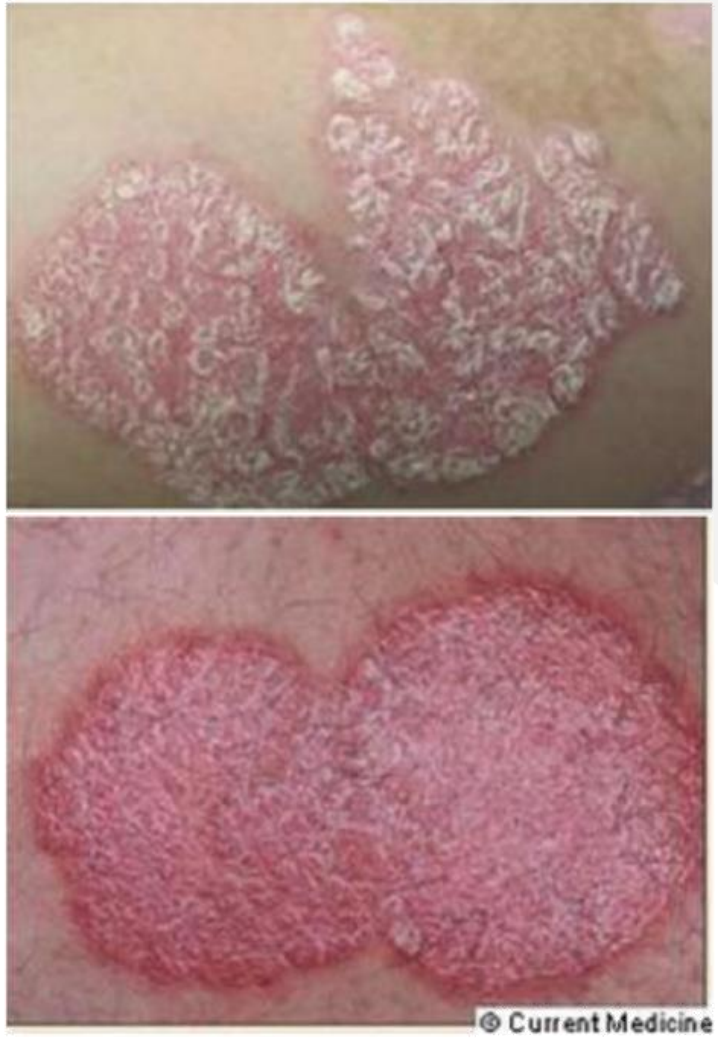
Classification of Psoriasis

Types and subtypes of disease	Features
1. Plaque psoriasis/vulgaris type	Sharply circumscribed, round–oval, or nummular (coin-sized) plaques
	Plaques occur on elbows, knee, trunk, back
	Most common type, occurring in 85%–90% of patients
2. Guttate psoriasis	Small, 2–10 mm diameter; acute onset, centripetal distribution
	Lesions occur shortly after an acute streptococcal infection of the pharynx or tonsils
	Common in children; occasionally in adults
3. Flexural (inverse) psoriasis	Affects the flexures (inframammary, perineal, and axillary)
	Lesions devoid of scale appear as red, shiny, well-demarcated plaques
	Differential diagnosis: candidal intertrigo, dermatophyte infections
4. Erythroderma	Total or subtotal involvement of the body skin
a. Chronic plaque psoriasis	May impair the thermoregulatory capacity of the skin, leading to hypothermia, high output cardiac failure, and metabolic changes
b. Unstable psoriasis	a. Lesions gradually progress as plaques and become confluent and extensive
5. Generalized pustular psoriasis (von Zumbusch)	b. Precipitated by infection, tar, drugs, withdrawal of corticosteroids; becomes rapidly extensive
	Monomorphic, sterile pustules, which may coalesce to form sheets
	Active, unstable disease; sometimes requires hospital admission
	Precipitated by withdrawal of systemic or potent topical corticosteroids and by infections
6. Palmoplantar pustulosis	Sterile, yellow pustules on erythemato-squamous background
	Frequently with nail involvement
	Prevalent in women, in the fourth to sixth decade, and in smokers
7. Psoriatic nail disease	Small pits, onycholysis, "oil spots" thickening, dystrophy, discoloration, subungual hyperkeratosis

a. Chronic Plaque Psoriasis

Chronic plaque psoriasis

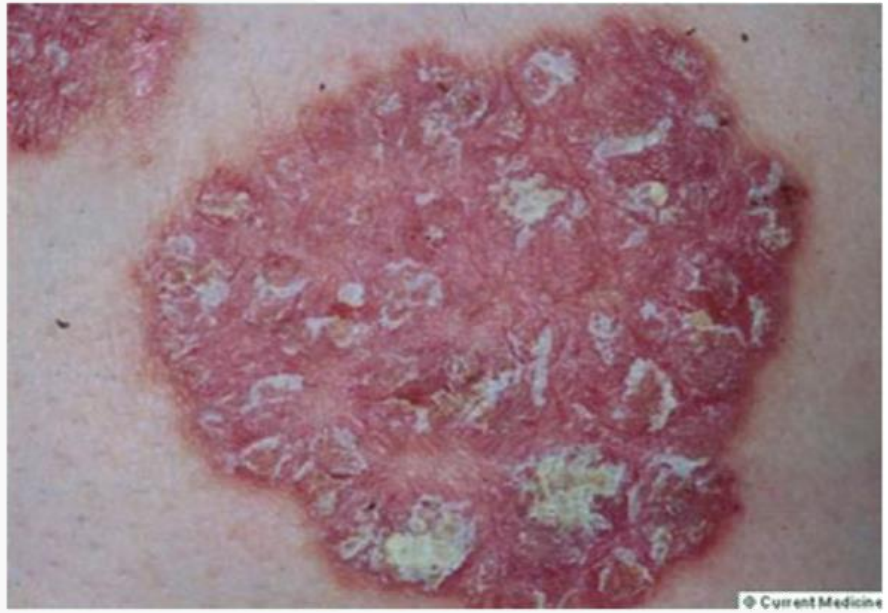
A. Most common variant
B. Chronic course, but remissions can occur
C. Symmetric distribution of sharply demarcated red plaques with thick silvery scale
D. Auspitz sign: pinpoint bleeding after scale removal
E. Most common on the scalp, extensor surfaces (elbows, knees), sacrum, umbilicus, hands, feet, genitalia (usually involves the glans penis in men)
F. Nail changes (also see Chap. 11, section 64l)
    1. Thickening and yellow discoloration of nail plate
    2. Distal onycholysis
    3. Pitting
    4. Red, spotted lunula
    5. Subungual hyperkeratosis
    6. Oil spots (pink-yellow)
G. Arthritis (sausage digits)



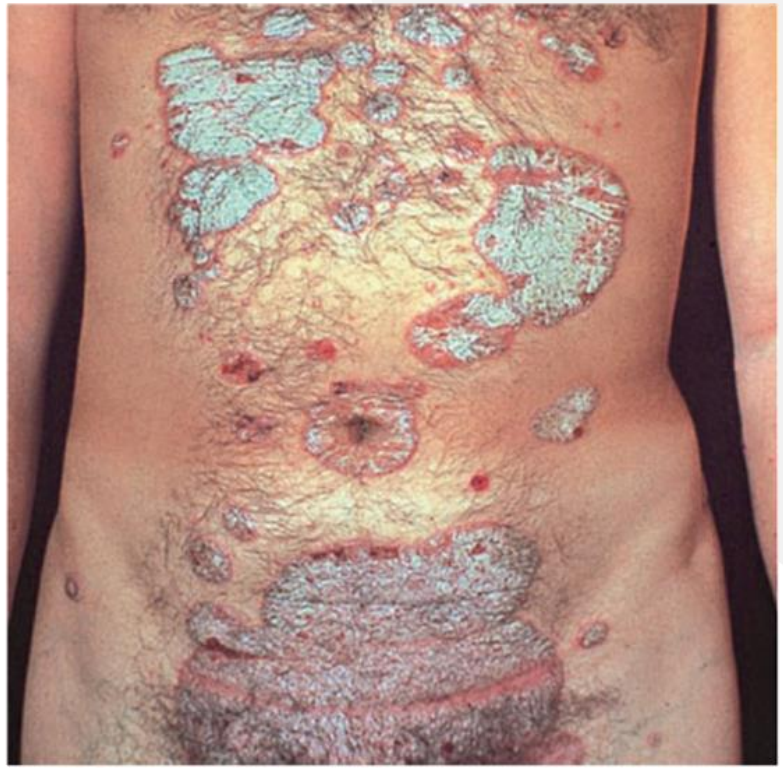
Chronic plaque psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

Chronic plaque psoriasis

A. Sharply demarcated, erythematous plaques with silvery-white, micaceous scales on lower chest, abdomen, and pubic area



Chronic plaque psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

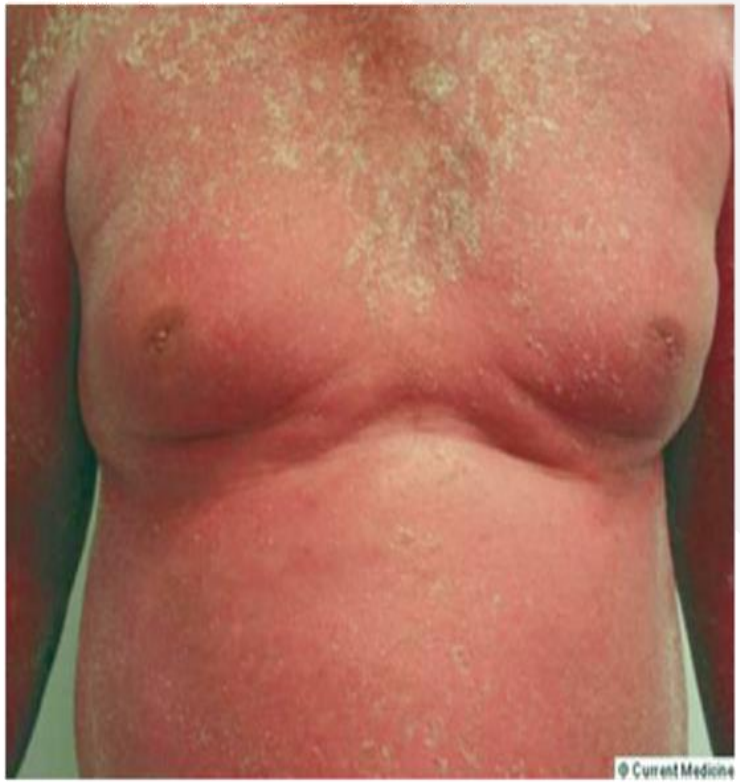


Chronic plaque psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

b. Erythrodermic Psoriasis

Erythrodermic psoriasis

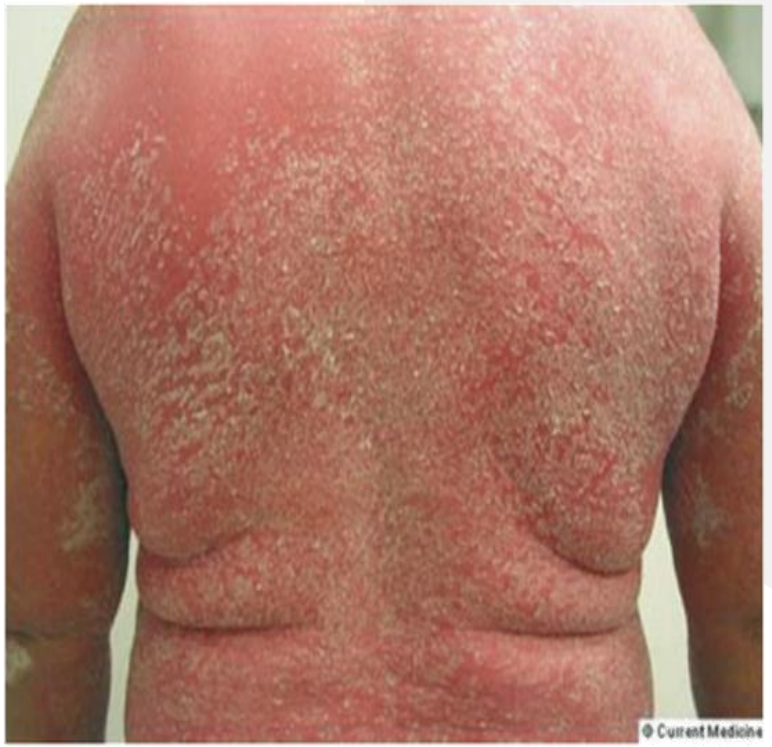
A. Severe form of psoriasis characterized by generalized erythema and scaling
B. Often associated with systemic illness including fever, hypotension, insensible fluid losses, and hypoalbuminemia
C. Can be spontaneous but usually is associated with a trigger factor such as abrupt discontinuation of corticosteroids or medications known to flare psoriasis such as lithium or beta-blockers, and infection



Erythrodermic psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

Erythrodermic psoriasis

A. Generalized erythema, scaling, and occasionally pustules

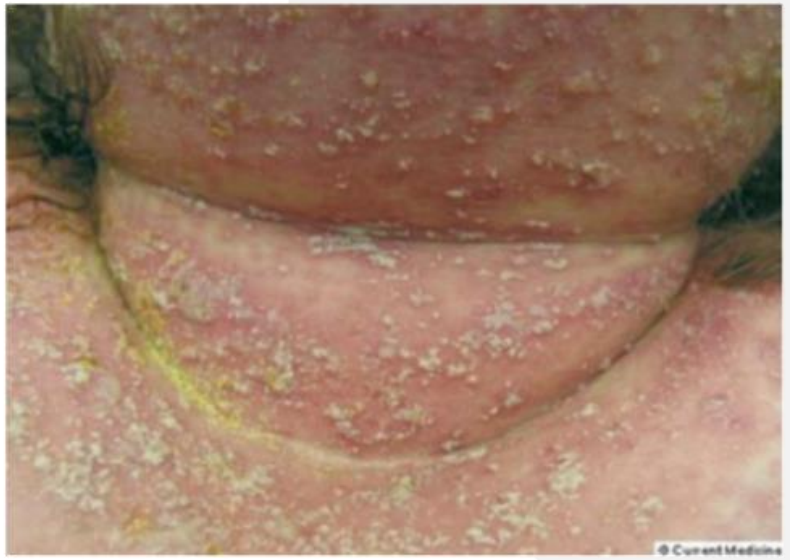


Erythrodermic psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

c. Generalized Pustular Psoriasis

Generalized pustular psoriasis

A. Large areas of erythema with numerous sterile pustules forming lakes of pus
B. Triggering factors include pregnancy, rapid tapering of steroids, hypocalcemia, infections
C. Four patterns of pustular psoriasis
    1. Von Zumbusch
        a. Generalized onset of erythema and pustules which resolves in several days with extensive scale
        b. Skin is painful
        c. Patients are ill with fever and chills
    2. Annular
        a. Annular inflammatory plaques with erythema and scale and pustules at the advancing edge
        b. Lesions expand centrifugally and heal with central clearing with scale
    3. Exanthematic
        a. Acute eruption of small pustules which disappear over a few days (looks like acute generalized exanthematous pustulosis)
    4. Localized
        a. Pustules appear within or at the edge of existing psoriatic plaque
        b. Can be seen after the application of irritants like tars

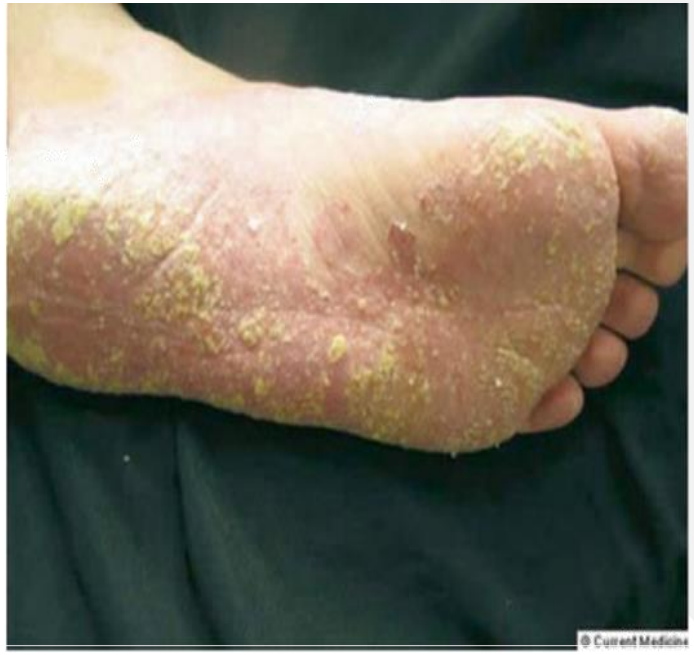


Generalized pustular psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

d. Palmoplantar Psoriasis

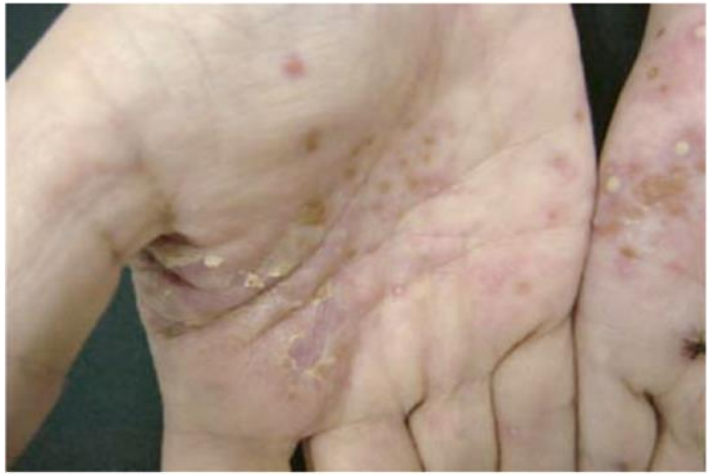
Palmoplantar psoriasis

A. Non-pustular variant
   1. Well-demarcated hyperkeratotic plaques with scaling and fissuring
   2. Can be difficult to differentiate from other diseases such as hand eczema and contact dermatitis



Palmoplantar psoriasis

A. Pustular variant
   1. Sterile pustules of the palmoplantar surfaces mixed with yellow-brown macules and scaly plaques
   2. Chronic course
   3. Associated with sterile inflammatory bone lesions

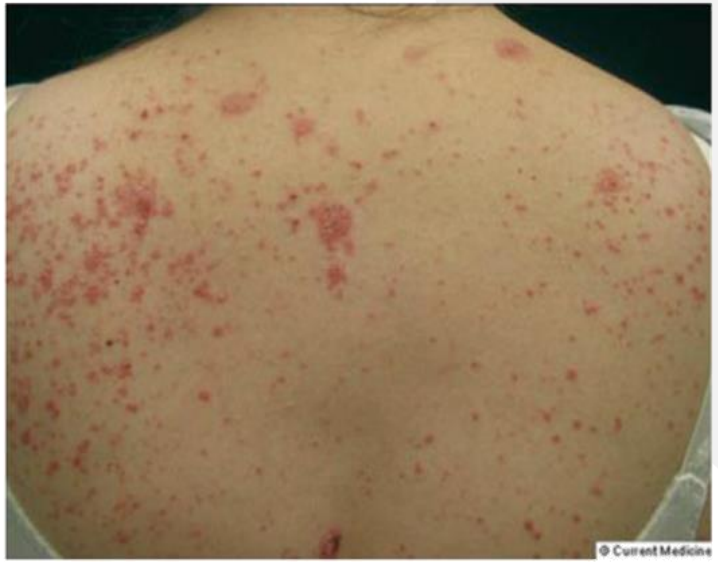


Palmoplantar psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

e. Guttate Psoriasis

Guttate psoriasis

A. Most common in children and adolescents
B. Frequently preceded by triggers including streptococcal pharyngitis, viral infections, medications, major stressors, or abrupt withdrawal of treatments (particularly corticosteroids or cyclosporine)
C. Numerous widely disseminated, bright red, discrete "drop shaped" small scaly papules and plaques
D. Most common on the trunk

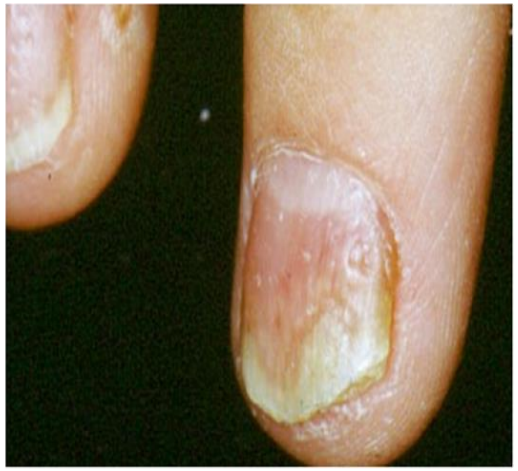


Guttate psoriasis
Photograph courtesy of SpringerImages Database, Springer Publishing Company

Psoriatic Nail Disease

Psoriatic nail disease

A. Nail pits, onycholysis, yellow discoloration of the nail plate, and subungual debris



Psoriatic nail disease
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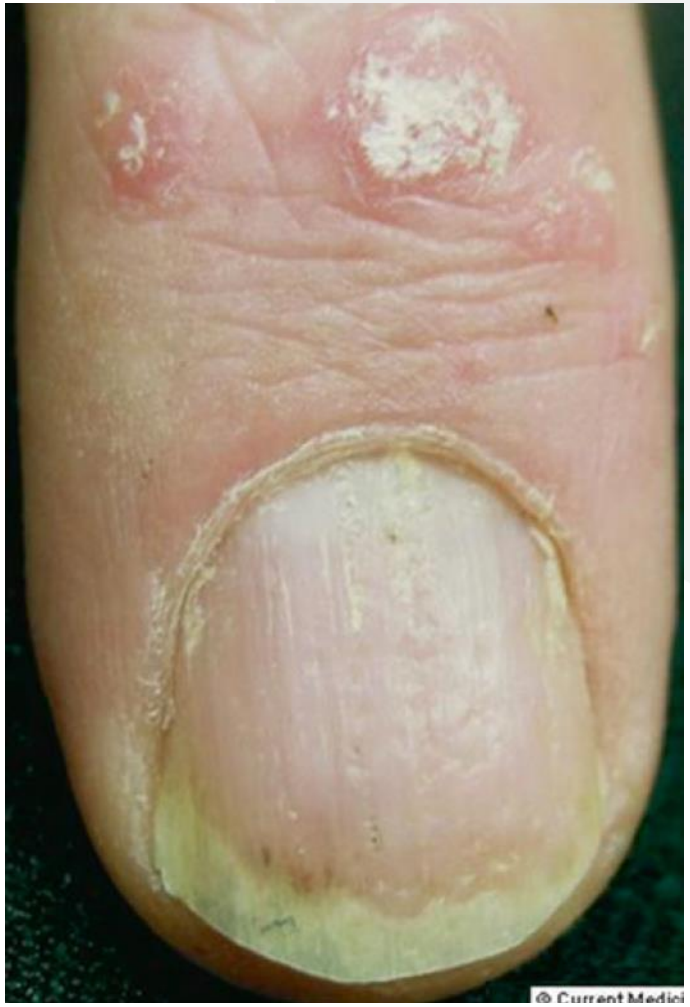


Psoriatic nail disease

A. Nail pits, salmon patches, and distal onycholysis

Disease Course

the majority of patients have chronic plaque
may develop other variants including guttate, pustular, inverse or erythrodermic variants.

Plaque :

- chronic with intermittent remissions
- persist for months to years
- usually develop slowly
- active peripheral edge
- Resolution of a plaque typically begins at its center.
- post-inflammatory hypo- or hyper-pigmentation

internal involvement, including joints and extra-articular sites such as the eyes
Concomitant psoriatic arthritis occurs in up to 30%
In a minority ,symptoms of psoriatic arthritis appear before skin involvement.
The prevalence of ophthalmic involvement in 10% patients ,leading to blepharitis, peripheral keratopathy, acute anterior uveitis, posterior synechiae, conjunctivitis, and cataract formation.

Cont. Disease Course

Guttate psoriasis:

may clear spontaneously

elevated anti-streptolysin O (ASO) titer with involuting course

may become chronic and progress to plaque psoriasis, particularly in patients with a family history of psoriasis.

Palmoplantar pustulosis :

tends to remain localized

significantly aggravated by extrinsic factors, such as stress, smoking, and infections

It is less responsive to standard treatment and is commonly associated with sterile inflammatory bone lesions, such as the SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, and osteitis)

with or without evidence of classic plaque type disease

Generalized pustular psoriasis: (von Zumbusch)

small, monomorphic sterile pustules develop in painful inflamed skin which is triggered by pregnancy, rapid withdrawal of corticosteroids, infections, and hypocalcemia.

systemic symptoms of fever, chills, and fatigue, as well as electrolyte derangements and liver abnormalities

aggressive treatment with systemic immunosuppressive therapy

The mortality rate of generalized pustular psoriasis due to sepsis is high without appropriately aggressive treatment.

APPROACH

- significant effect on **quality of life**
- **addressing both psychosocial and physical aspects**
- Numerous **topical and systemic** therapies
- are chosen on the basis of **disease severity, relevant comorbidities, patient preference (including cost and convenience), efficacy, and evaluation of individual patient response**
- **medication safety** plays ,must be balanced by the risk of undertreatment of psoriasis, leading to inadequate clinical improvement and patient dissatisfaction.

The desired outcome of treatment differs
is dependent upon preferred amount of disease control and tolerance of specific treatments
A reasonable goal for patients who desire maximum resolution of skin disease is minimal to no skin involvement achieved with a well-tolerated treatment regimen
acceptable response for plaque psoriasis after three months of treatment as either less than 3 percent body surface area involvement or 75 percent improvement compared with baseline and the target response after six months as 1 percent body surface area

Psychosocial aspects

a frustrating disease for the patient and the provider

The clinician needs

to be empathetic and spend adequate time

touch ,disorder is neither repulsive nor contagious.

lay out reasonable aims , primary goal

is no cure for psoriasis

Educating

Psoriasis may affect patients' perceptions of themselves and this can potentially initiate or exacerbate psychologic disorders such as depression.

Patients with limited skin disease may still have significant psychosocial disability. Some patients with psoriasis may benefit from counseling and/or treatment with psychoactive medications.

Choice of therapy

the initial decision

between local (topical) and full body (phototherapy or systemic) therapy

even patients on systemic therapy will likely continue to need some topical agents

treatment planning

mild (or limited) disease and extensive (moderate to severe) disease categories

Mild managed with topical agents, while patients with moderate to severe need phototherapy or systemic therapy.

The location

the presence of psoriatic arthritis

Psoriasis of the hand, foot, or face can be debilitating functionally or socially and may deserve a more aggressive treatment approach.

Moderate to severe :

more than 5 to 10 percent of the body surface area (the entire palmar surface, including fingers, of one hand is approximately 1 percent of the body surface area) or

involvement of the face, palm or sole, or disease that is otherwise disabling.

Patients with more than 5 percent body surface area affected are generally candidates for phototherapy or systemic therapy, since application of topical agents to a large area is not usually practical or acceptable for most patients

Cont. Choice of therapy

. Attempts to treat extensive disease with topical agents alone

- are often met with failure,
- can add cost, and
- lead to frustration in the patient-clinician relationship.

However, topical agents are useful adjuncts for resistant, localized lesions in patients who are getting phototherapy or systemic agents for extensive involvement.

newer systemic therapies ("biologics")

- cost is a major consideration

Established therapies such as methotrexate and phototherapy continue to play a role

extensive or recalcitrant disease is a challenge even for experienced dermatologists

- the availability of biologic medications has reduced

Widespread pustular disease requires aggressive treatment, which may include hospitalization.

Limited disease

— Limited plaque

respond well to topical corticosteroids and emollients

Alternatives:

vitamin D analogs (eg, calcipotriene and calcitriol),
tar, and
topical retinoids (tazarotene)
For facial or intertriginous areas, topical tacrolimus or pimecrolimus may be used as alternatives or as corticosteroid-sparing agents, though improvement may not be as rapid as with potaent topical corticosteroids.

Localized phototherapy is another option for recalcitrant disease.

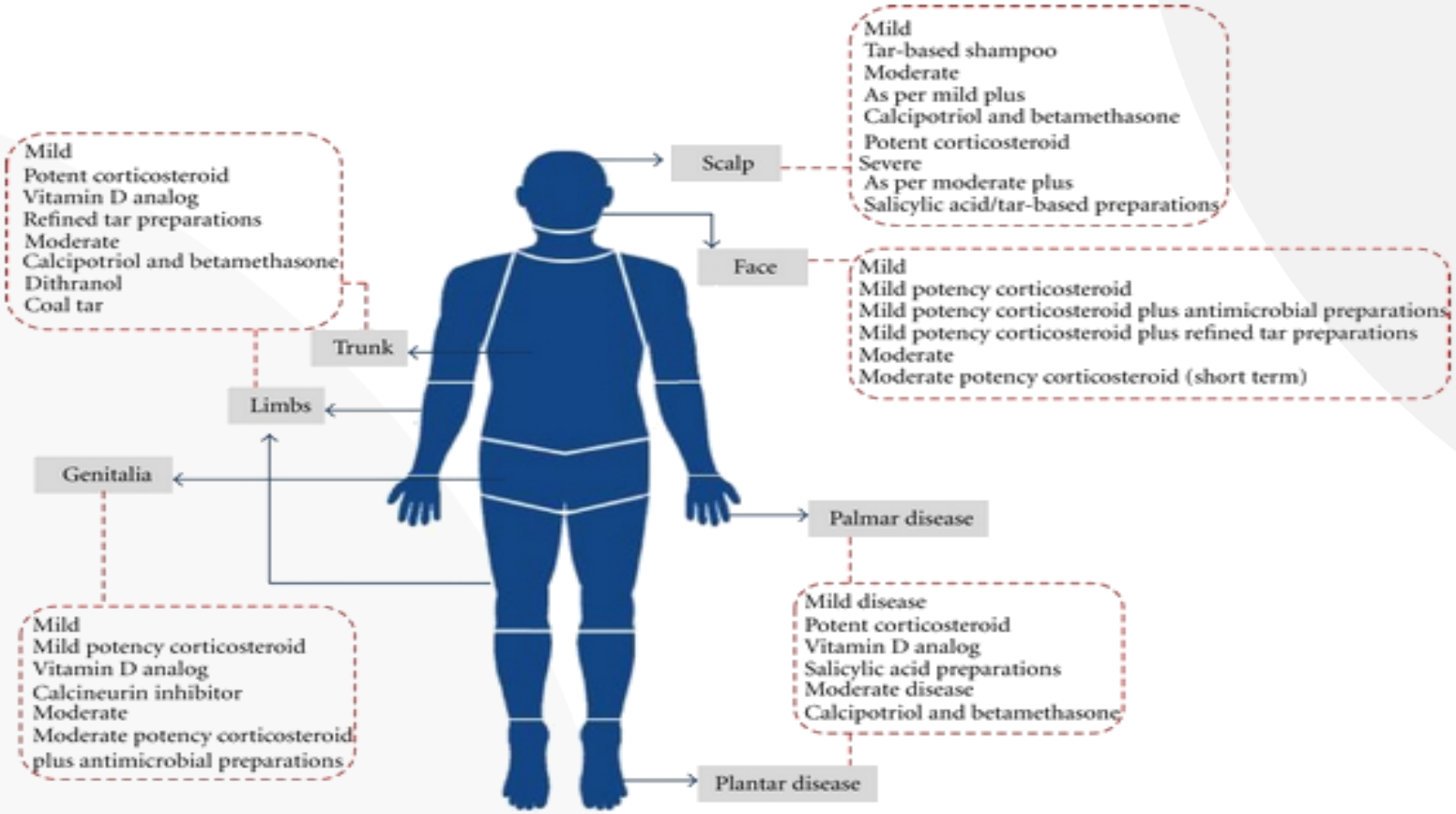
Combinations of potent topical corticosteroids and either calcipotriene, calcitriol, tazarotene, or UVB phototherapy

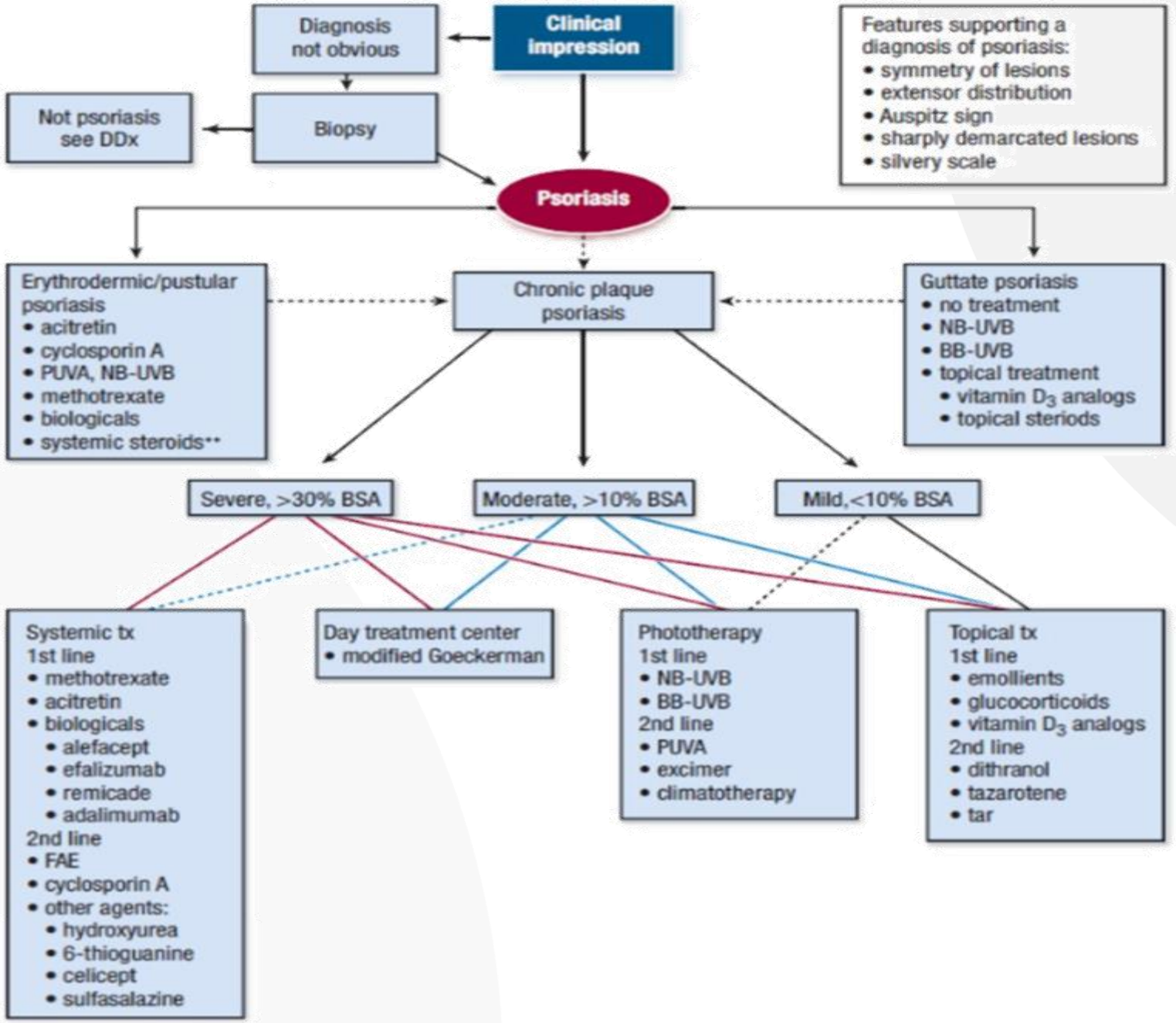
Calcipotriene in combination with class I topical corticosteroids is highly effective for short-term control. Calcipotriene alone can then be used continuously and the combination with potent corticosteroids used intermittently (on weekends) for maintenance

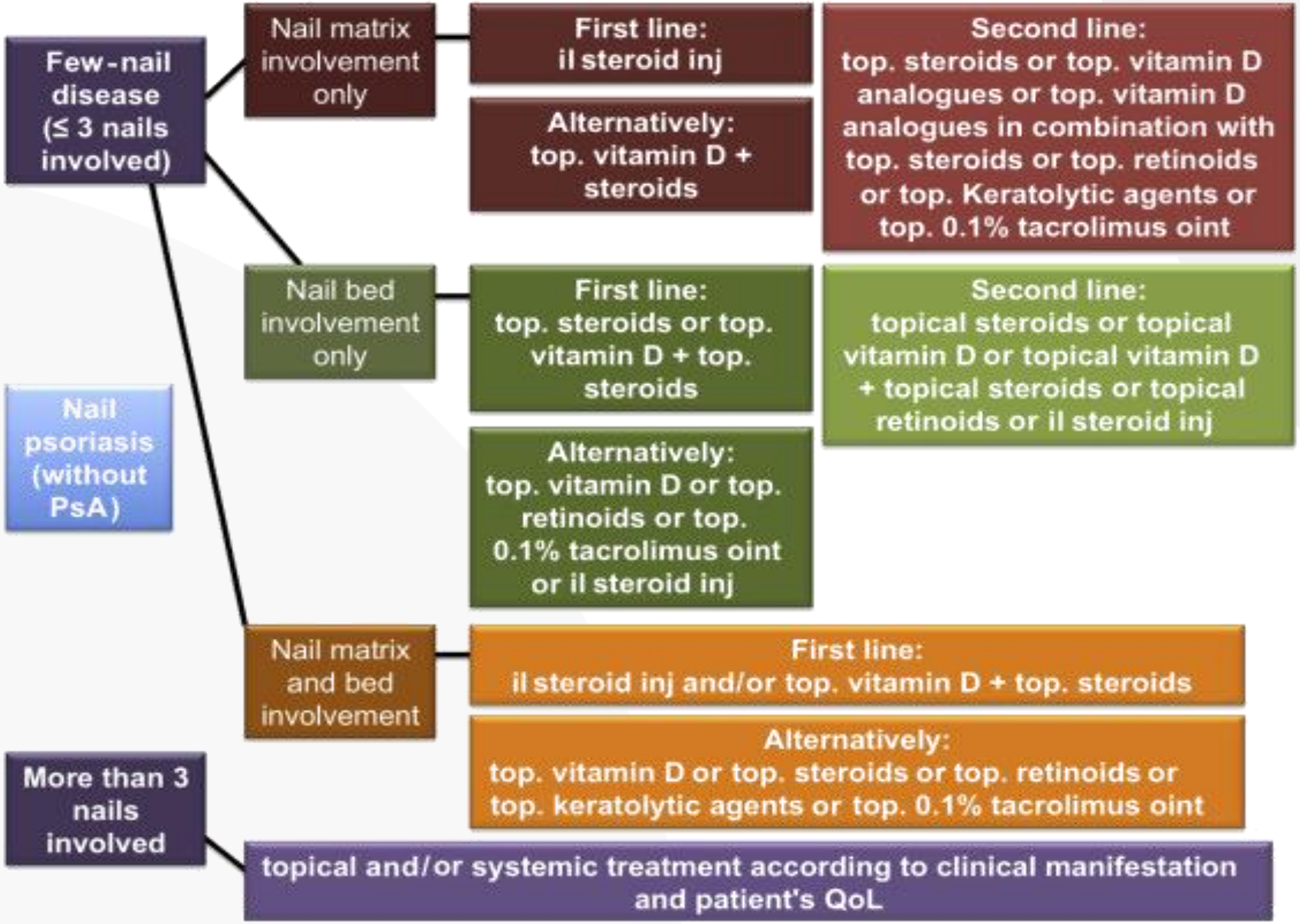
With proper adherence, considerable improvement with topical therapies may be seen in as little as one week, though several weeks may be required for full benefits.

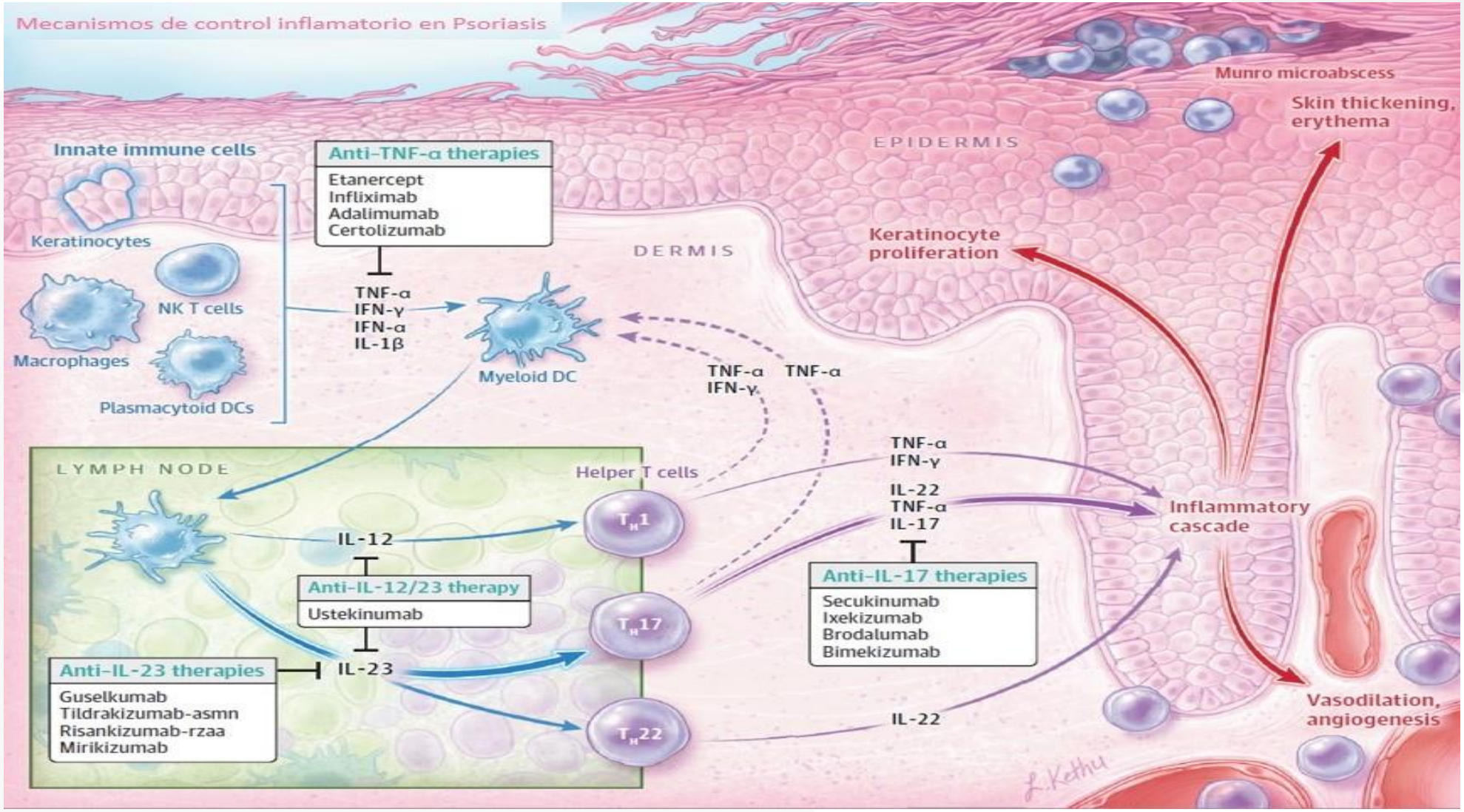
adherence to topical

a major hurdle,
keeping the treatment regimen simple and
using treatment vehicles that the patient finds acceptable is often beneficial


Mild
Potent corticosteroid
Vitamin D analog
Refined tar preparations
Moderate
Calcipotriol and betamethasone
Dithranol
Coal tar
Trunk
Limbs
Genitalia
Mild
Mild potency corticosteroid
Vitamin D analog
Calcineurin inhibitor
Moderate
Moderate potency corticosteroid plus antimicrobial preparations
Scalp
Mild
Tar-based shampoo
Moderate
As per mild plus
Calcipotriol and betamethasone
Potent corticosteroid
Severe
As per moderate plus
Salicylic acid/tar-based preparations
Face
Mild
Mild potency corticosteroid
Mild potency corticosteroid plus antimicrobial preparations
Mild potency corticosteroid plus refined tar preparations
Moderate
Moderate potency corticosteroid (short term)
Palmar disease
Mild disease
Potent corticosteroid
Vitamin D analog
Salicylic acid preparations
Moderate disease
Calcipotriol and betamethasone
Plantar disease


Diagnosis not obvious
Clinical impression
Features supporting a diagnosis of psoriasis:
• symmetry of lesions
• extensor distribution
• Auspitz sign
• sharply demarcated lesions
• silvery scale
Not psoriasis see DDx
Biopsy
Psoriasis
Erythrodermic/pustular psoriasis
• acitretin
• cyclosporin A
• PUVA, NB-UVB
• methotrexate
• biologicals
• systemic steroids**
Chronic plaque psoriasis
Guttate psoriasis
• no treatment
• NB-UVB
• BB-UVB
• topical treatment
• vitamin D_3 analogs
• topical steriods
Severe, >30% BSA
Moderate, >10% BSA
Mild,<10% BSA
Systemic tx
1st line
• methotrexate
• acitretin
• biologicals
• alefacept
• efalizumab
• remicade
• adalimumab
2nd line
• FAE
• cyclosporin A
• other agents:
• hydroxyurea
• 6-thioguanine
• celicept
• sulfasalazine
Day treatment center
• modified Goeckerman
Phototherapy
1st line
• NB-UVB
• BB-UVB
2nd line
• PUVA
• excimer
• climatotherapy
Topical tx
1st line
• emollients
• glucocorticoids
• vitamin D_3 analogs
2nd line
• dithranol
• tazarotene
• tar


Nail psoriasis (without PsA)
Few-nail disease (≤ 3 nails involved)
Nail matrix involvement only
First line: il steroid inj
Alternatively: top. vitamin D + steroids
Second line: top. steroids or top. vitamin D analogues or top. vitamin D analogues in combination with top. steroids or top. retinoids or top. Keratolytic agents or top. 0.1% tacrolimus oint
Nail bed involvement only
First line: top. steroids or top. vitamin D + top. steroids
Alternatively: top. vitamin D or top. retinoids or top. 0.1% tacrolimus oint or il steroid inj
Second line: topical steroids or topical vitamin D or topical vitamin D + topical steroids or topical retinoids or il steroid inj
Nail matrix and bed involvement
First line: il steroid inj and/or top. vitamin D + top. steroids
Alternatively: top. vitamin D or top. steroids or top. retinoids or top. keratolytic agents or top. 0.1% tacrolimus oint
More than 3 nails involved
topical and/or systemic treatment according to clinical manifestation and patient's QoL


Mecanismos de control inflamatorio en Psoriasis
Munro microabscess
Skin thickening, erythema
EPIDERMIS
Innate immune cells
Anti-TNF-α therapies
Etanercept
Infliximab
Adalimumab
Certolizumab
Keratinocytes
DERMIS
Keratinocyte proliferation
NK T cells
TNF-α
IFN-γ
IFN-α
IL-1β
Macrophages
Myeloid DC
TNF-α
IFN-γ
TNF-α
Plasmacytoid DCs
LYMPH NODE
Helper T cells
TNF-α
IFN-γ
IL-22
TNF-α
IL-17
Inflammatory cascade
IL-12
Anti-IL-12/23 therapy
Ustekinumab
Anti-IL-17 therapies
Secukinumab
Ixekizumab
Brodalumab
Bimekizumab
Anti-IL-23 therapies
Guselkumab
Tildrakizumab-asmn
Risankizumab-rzaa
Mirikizumab
IL-23
IL-22
Vasodilation, angiogenesis

Topical Corticosteroids

Topical corticosteroids (TCS) are a mainstay in treatment of a wide range of inflammatory dermatoses. There are seven classes of topical steroids ranging from superpotent (class 1) to very low-potency topical steroids (class 7). These classes have been developed based on vasoconstrictor assays .

Corticosteroid classification system, adapted from

Table 1

Corticosteroid classification system, adapted from [12].

Class	Name	Vehicle		
		Oinmtent	Cream	Lotion
Superpotent Class I USA; class I UK; class IV Germany	Betamethasone dipropionate glycol 0.05% Clobetasol 17-propionate 0.05% Halobetasol propionate 0.05%			
High potency Class II/III USA; class II UK; class III Germany	Amcinonide 0.1% Betamethasone dipropionate 0.05% Desoximetasone 0.25% Diflucortolone valerate 0.1% Fluocinonide 0.05% Halcinonide 0.1% Mometasone furoate 0.1% Triamcinolone acetonide 0.5%			

Cont. Corticosteroid classification system, adapted from

Moderate potency Class IV/V USA; class III UK; class II Germany	Betamethasone dipropionate 0.05% Betamethasone valerate 0.1% Clobetasone 17-butyrate 0.05% Desonide 0.05% Desoximetasone 0.05% Fluocinonide 0.025% Hydrocortisone 17-valerate 0.2% Prednicarbate 0.1% Triamcinolone acetonide 0.1%
Low potency Class VI/VII USA; class IV UK; class I Germany	Betamethasone valerate 0.05% Desonide 0.05% Fluocinonide 0.01% Hydrocortisone 1.0%, 2.5% Hydrocortisone acetate 0.5%, 1.0% Prednicarbate 0.05% Triamcinolone acetonide 0.025%

Cont. Topical Corticosteroids

Pharmacokinetics/Mechanism of Action

Three factors determine the pharmacokinetics and potency :

the structure

the vehicle, and

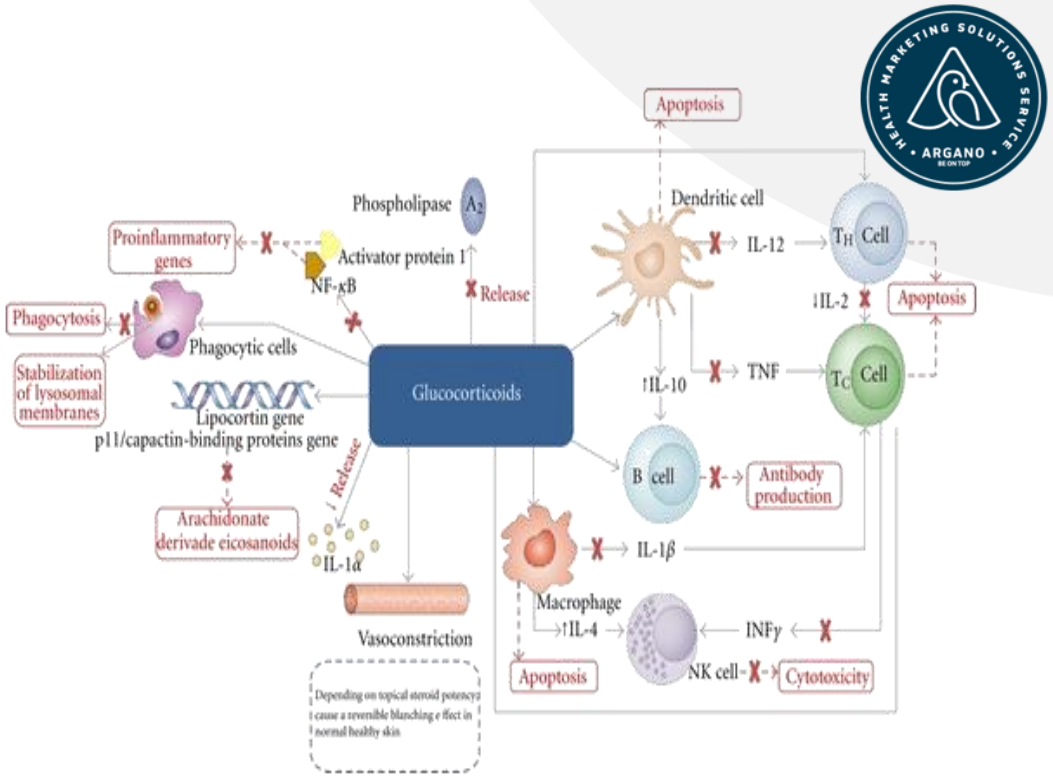
the skin

Hydrocortisone is the central structure of most topical corticosteroids

Variations are formed by placing hydroxyl groups onto the 11-β, 17-α, and 21 positions

modifies the molecule's lipophilicity, solubility, percutaneous absorption and glucocorticoid receptor binding Ability, potency, glucocorticoid receptor binding activity, mineralocorticoid activity.

Finally, epidermal enzymes cause de-esterification of topical corticosteroids into inactive metabolites. Increased potency can be accomplished by inhibiting de-esterification through halogenation at the 21 position.



Cont. Topical Corticosteroids

Vehicle

influence percutaneous absorption and therapeutic efficacy.

desired potency based on the severity and the location .Then, one must decide on the vehicle based on
the type of lesion to be treated, need for hydration or drying effect,
location and
potential for irritation by components of the vehicle,
and patient preference. Lotions are often preferred for the face, ointments work well for dry lesions, and gels are more useful in hairy areas or for a drying effect for wet lesions.

Patients prefer vehicles :
quickly absorbed,
non-greasy, and
easy to apply, such as lotions and foams,but patient preferences may vary greatly.

Potent and superpotent :avoided on the thin skin of the face and intertriginous :risk of skin atrophy

The vehicle may alter the pharmacokinetics and potency

Bioavailability and penetration increase with inflamed or diseased skin, as well as with increased hydration of the stratum corneum.

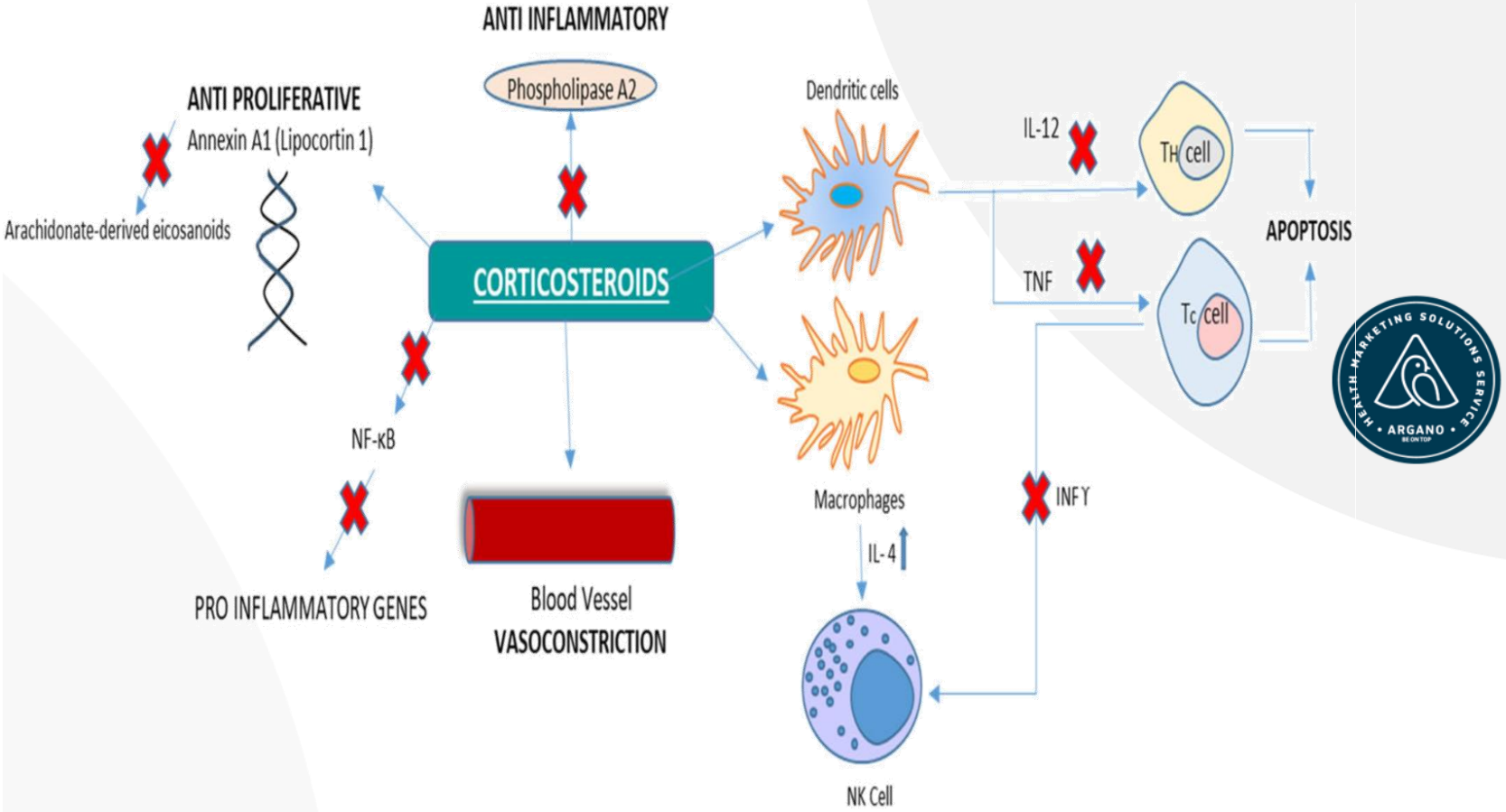
The thickness of the stratum corneum is inversely proportional to the degree of penetration of the topical corticosteroid.

Very occlusive agents, such as ointments, increase the absorption of topical corticosteroids through increased hydration of the stratum corneum .

Cont. Topical Corticosteroids

Immunologic Mechanisms

- all aspects of inflammation, affecting both adaptive and innate mmunity
  - decrease the number and function of Langerhans' cells, important in initiating immune responses.
  - Neutrophils are decreased, less adherent to vascular endothelium and have decreased phagocytic function
  - leukocytes show decreased antibody-dependent cellular toxicity and natural killer cell function
  - the production of many cytokines is decreased including interleukin (IL)-1, IL-2, interferon (IFN)-γ, tumor necrosis factor and granulocyte-monocyte-stimulating factor
- decrease the mitotic rate of the epidermis, thinning of the stratum corneum and granulosum and flattening
- atrophy of the dermis through inhibition of fibroblast proliferation, migration, chemotaxis and protein synthesis
- inhibition of fibroblast synthesis of both glycosaminoglycans and collagen .



Cont. Topical Corticosteroids

Use in Psoriasis

The antiproliferative and atrophogenic characteristics of TCS are useful in treating psoriasis. Topical corticosteroids are the mainstay of treatment and often first-line for the management of mild to moderate psoriasis, as well as for intertriginous areas and genitalia, as these areas can become irritated with the use of other topical agents . In general, for the treatment of localized plaque-type psoriasis, high potency or superpotent TCS are prescribed twice daily. Optimal improvement with high potency TCS is often achieved after 2 weeks. Superpotent TCS are recommended for the treatment of nail psoriasis ,whereas low-to-mid-potency TCS are typically used for intertriginous and genital psoriasis.

Cont. Topical Corticosteroids

Combination with Other Therapies

In patients with psoriasis, vitamin D analogues are frequently added at the onset, as there is a synergistic effect with TCS. Topical corticosteroids work synergistically with light therapy as well as many systemic agents. Psoriasis clears faster when using psoralen plus ultraviolet A(PUVA) with TCS versus PUVA alone. The addition of topical corticosteroids to cyclosporine therapy also leads to more rapid clearance of psoriasis . Topical steroids may also be combined with other topical agents, such as tazarotene , salicylic acid, or anthralin , providing increased efficacy due to increased penetration.

Cont. Topical Corticosteroids

Adverse Effects

Systemic adverse effects from topical corticosteroids are uncommon and are increased with young age, liver disease, renal disease, the potency of the drug, amount of skin surface involvement, the use of occlusion, frequency of application and the duration of treatment . The liver metabolizes corticosteroids and the kidneys excrete both metabolized and unmetabolized corticosteroid . Infants and young children are particularly predisposed to systemic adverse effects from TCS due to a higher skin surface-to-body ratio, increased cutaneous permeability, and immature renal function . Catch-up growth is expected when topical corticosteroids are discontinued in this population. Cushing's syndrome and hypothalamic- pituitary-adrenal (HPA) axis suppression has been noted in patients applying high quantities of topical corticosteroids for prolonged periods of time . Screening for HPA axis suppression is done using the 8 AM plasma cortisol level and definitive diagnosis requires the cosyntropin test. Local adverse effects are also rare but occur more frequently than systemic adverse effects.

Cont. Adverse Effects

Cutaneous atrophy is the most commonly observed side effect, and is characterized by telangiectasias, striae, hypopigmented, wrinkled or shiny skin . Striae are typically seen after many weeks to months of topical steroid use; risk factors include the potency of corticosteroid, location of application, occlusion, and use in infancy/childhood. “Corticosteroid phobia” is an exaggerated and often irrational fear of using topical steroids, and is common amongst patients, often resulting in treatment nonadherence . Patients’ primary concerns often stem from TCS “thinning the skin,” but a 2011 study by Hong et al. demonstrated that appropriate long-term use of topical corticosteroids in children with dermatitis does not cause skin atrophy . Application of TCS may also result in perioral dermatitis, characterized by erythematous papules in a periorificial distribution. Treatment for perioral dermatitis consists of an oral tetracycline in addition to a long taper with a non-fluorinated topical corticosteroid, such as hydrocortisone acetate cream.

Cont. Adverse Effects

Prolonged use of topical glucocorticoids on the eyelids can lead to glaucoma and cataracts, and thus is not recommended . These complications may also occur in patients who apply TCS peripherally on their body, if inadvertent eye contact occurs. Allergic contact dermatitis to topical steroids may occur and can be suspected when a patient fails to respond to topical steroid therapy or flares with topical steroid therapy . This allergy may be to the vehicle or the actual corticosteroid molecule, which can be confirmed with patch testing. Topical corticosteroids often have a delayed reaction and persist for at least 96 h, thus requiring a delayed check . Loss of clinical effect may occur with repeated application of topical corticosteroids—known as tachyphylaxis. This phenomenon occurs more commonly with higher strength topical corticosteroids, but often subsides after a rest period of a few days. There is no established regimen to prevent tachyphylaxis. A commonly recommended regimen is twice daily application of TCS for 2 weeks followed by a 1 week rest period or weekend-only application .

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