Skin diseases in GP practice

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Agenda

- Atopic dermatitis
- Psoriasis
- Bacterial infections: erysipelas and cellulitis
- Furuncule

Atopic dermatitis

Atopic dermatitis

Atopic dermatitis (AD) is a chronic, highly pruritic, eczematous skin disease that follows patients from early childhood into puberty and sometimes adulthood

Also reffered to as eczematous dermatitits, the disease often has a remitting/flaring course, which may be exacerbated by social, enviromental and biological triggers

Atopic dermatitis - epidemiology

- 60 % of patients develop AD by 1 year of age
- 85 % of patients develop AD by age 5
- 10 % develop AD between 6 and 20 years of age
- Rarely AD has an adult onset,
- Earlier onset often indicates a more severe course
- **GENDER:** slightly more common in males than females

AD: aetiology

1. The inheritance
2. Exacerbating factors
3. Eliciting factors

The inheritance

The inheritance pattern has not been ascertained. However, in one series, 60 % of adults with AD had children with AD.

The prevalence in children was higher (81 %) when both parents had AD.

Eliciting factors

Inhalants: Specific aeroallergens, especially dust mites and pollens, have been shown to cause exacerbations of AD.

Microbial Agents: Exotoxins of *Staphylococcus aureus* may act as superantigens and stimulate activation of T cells and macrophages.

Autoallergens: IgE antibodies directed at human proteins

Foods: Subset of infants and children have flares of AD with eggs, milk, soybeans, fish and wheat.

Exacerbating factors

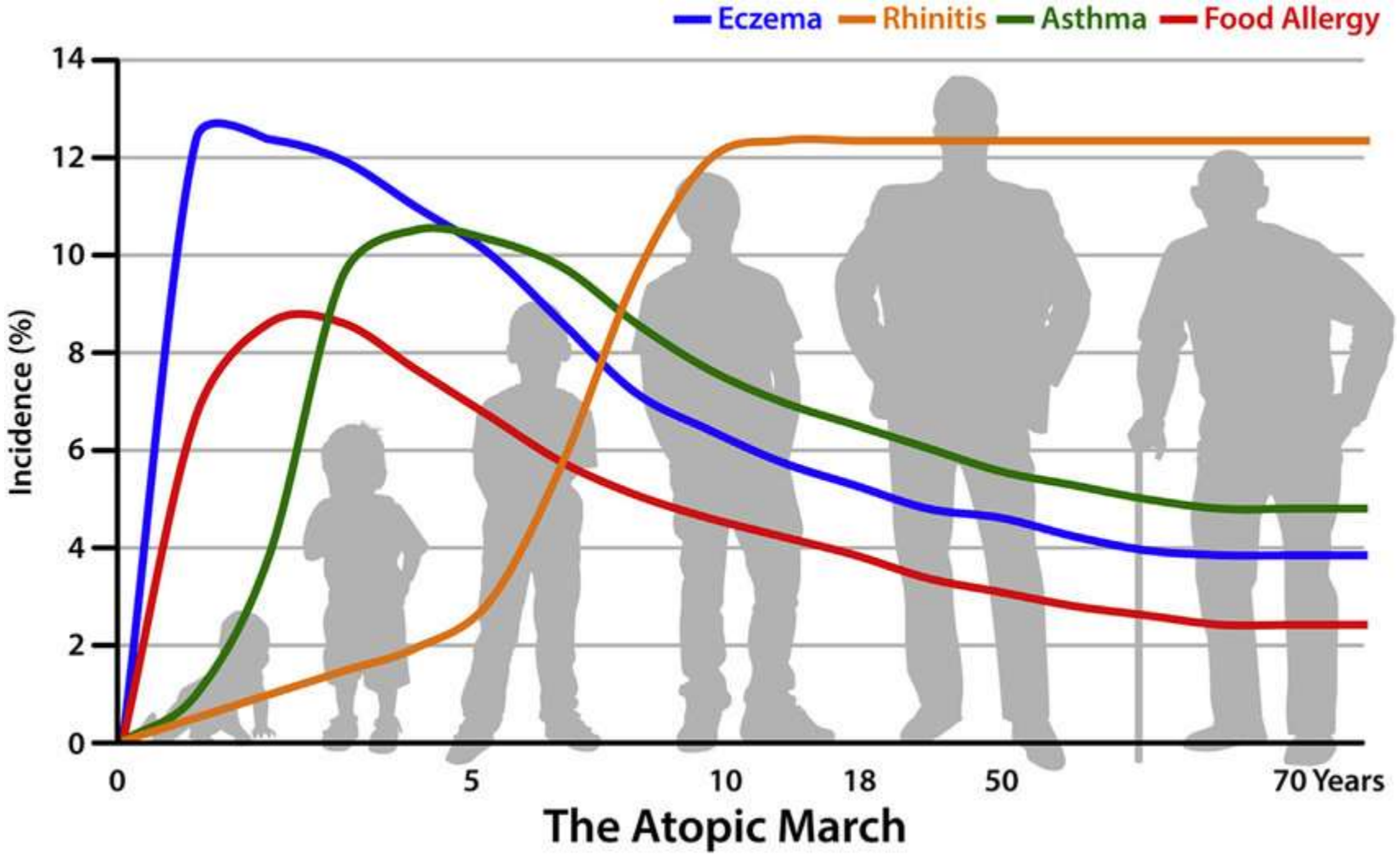
Skin barrier disruption: increase transepidermal water loss (TEWL) by frequent bathing and hand washing and dehydration

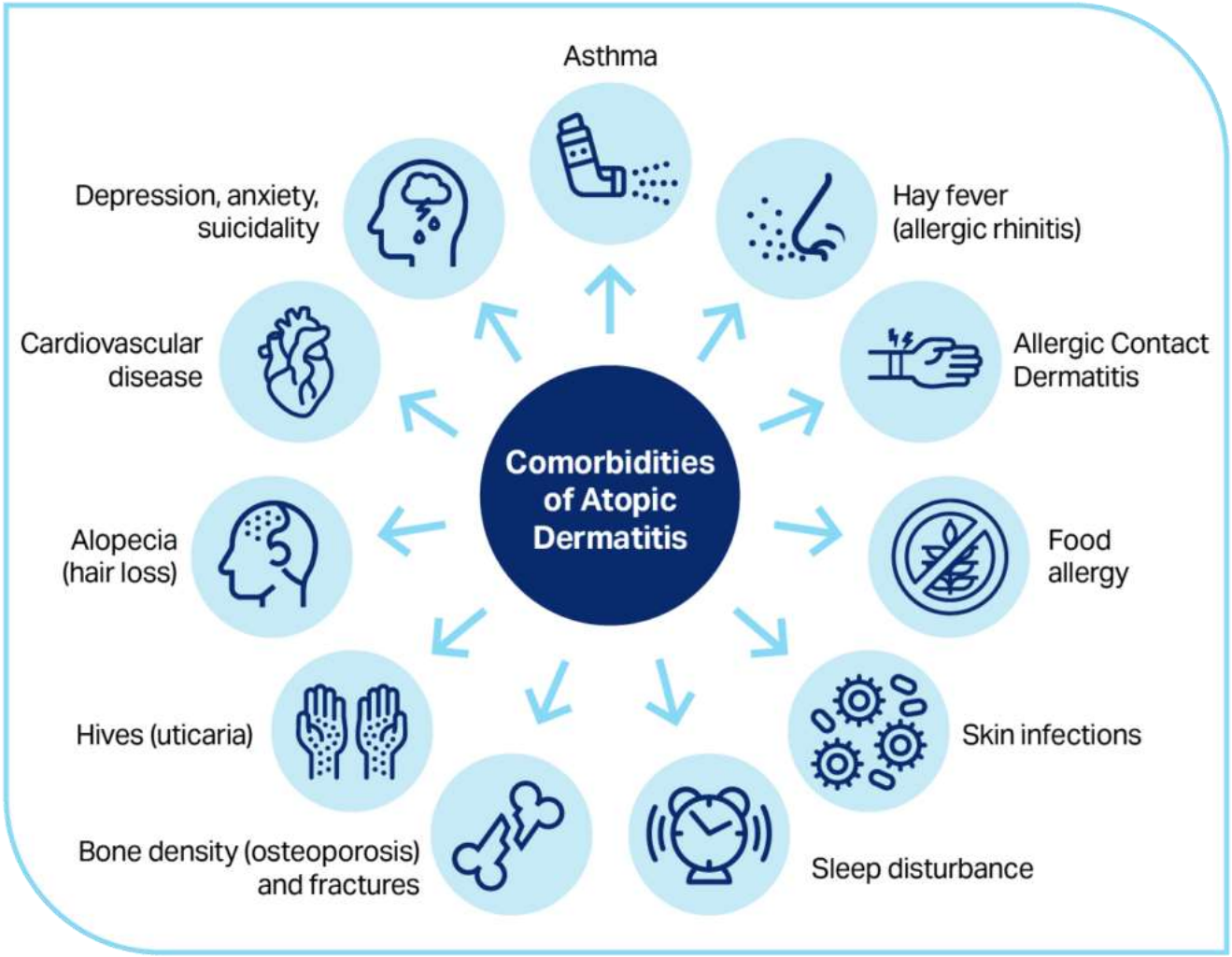
Infections: *S. aureus* present in severe cases; rarely fungus (dermatophytosis, candidiasis)

Season: AD improves in summer, flares in winter

Clothing: wool is important trigger; wool clothing or blankets (also wool clothing of parents)

Emotional stress


Eczema
Rhinitis
Asthma
Food Allergy
Incidence (%)
14
12
10
8
6
4
2
0
0
5
10
18
50
70 Years
The Atopic March

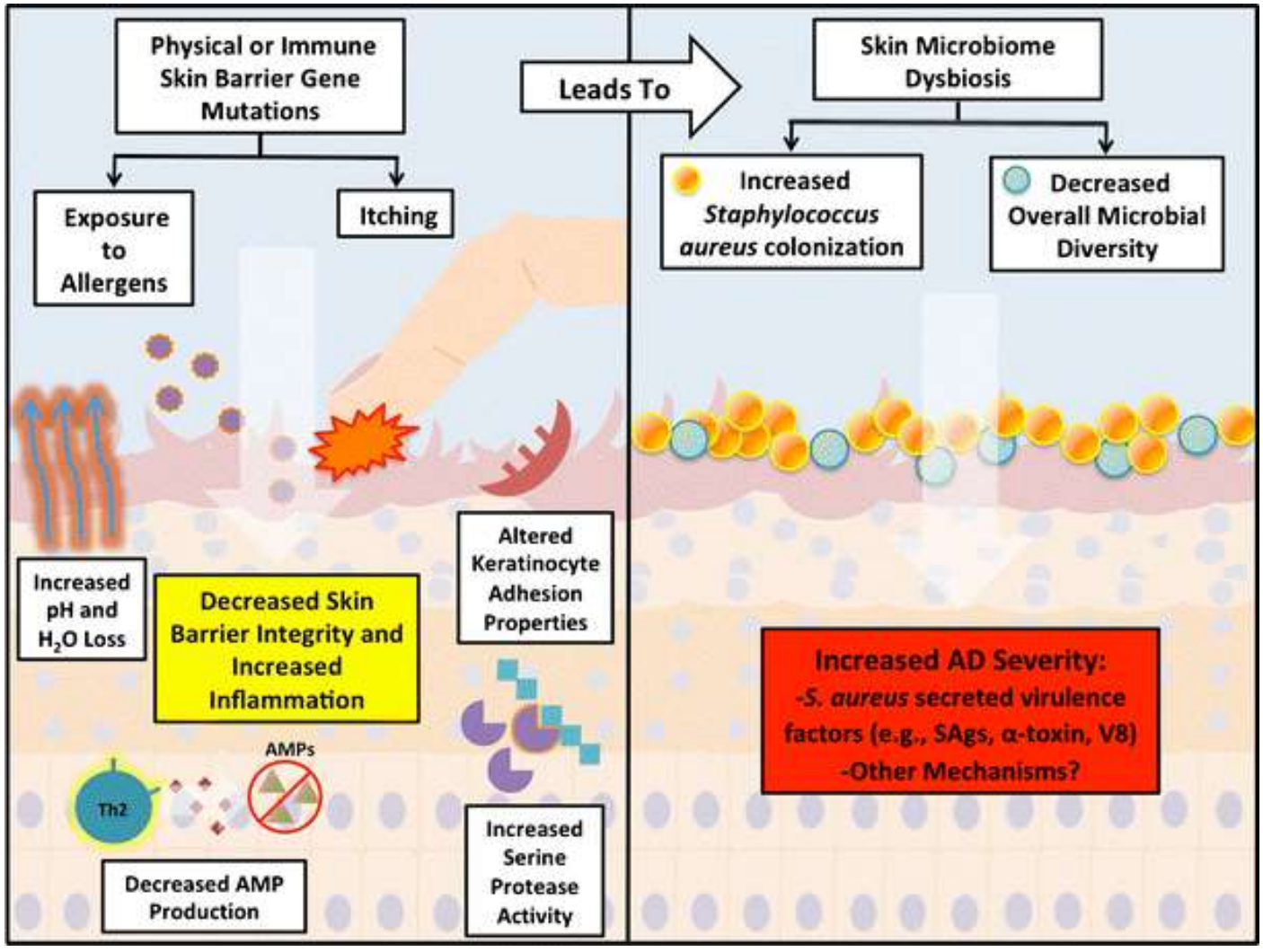

Comorbidities of Atopic Dermatitis
Asthma
Hay fever (allergic rhinitis)
Allergic Contact Dermatitis
Food allergy
Skin infections
Sleep disturbance
Bone density (osteoporosis) and fractures
Hives (uticaria)
Alopecia (hair loss)
Cardiovascular disease
Depression, anxiety, suicidality

AD: Pathogenesis

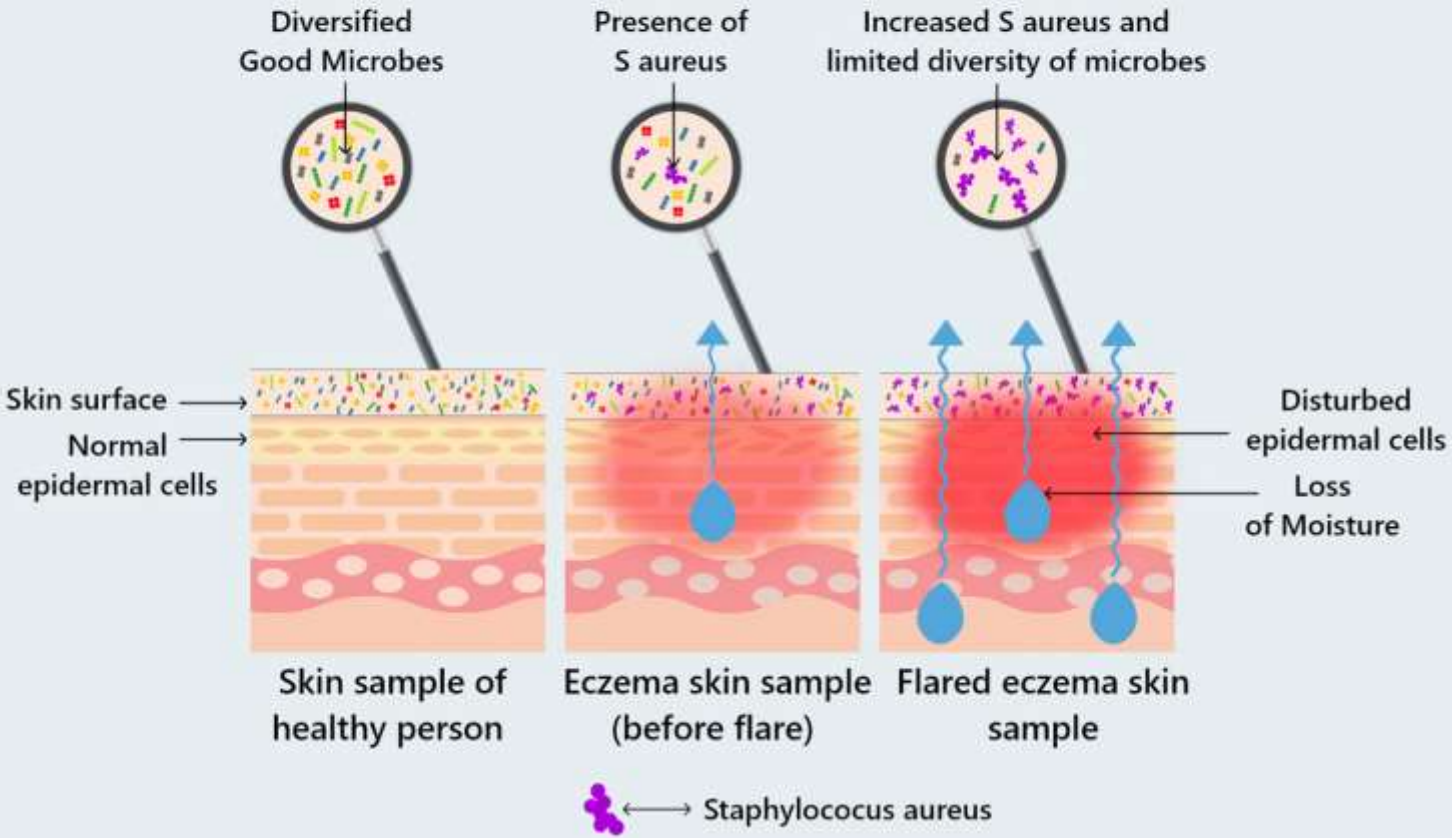
Skin barrier dysfunction

Immunologic reactions

Skin microbiome disruption


Physical or Immune Skin Barrier Gene Mutations
Exposure to Allergens
Itching
Leads To
Skin Microbiome Dysbiosis
Increased *Staphylococcus aureus* colonization
Decreased Overall Microbial Diversity
Increased pH and H_2O Loss
Decreased Skin Barrier Integrity and Increased Inflammation
Altered Keratinocyte Adhesion Properties
AMPs
Th2
Decreased AMP Production
Increased Serine Protease Activity
Increased AD Severity:
-*S. aureus* secreted virulence factors (e.g., SAgs, α-toxin, V8)
-Other Mechanisms?

Microbiome and Eczema



Treatment

Atopic Dermatitis

(most common type of eczema)

First Line: non-pharmacologic approaches

Topical Treatment Options

1st Line: Topical Corticosteroids

High Potency: Betamethasone dipropionate, clobetasol, halobetasol

Medium Potency: Triamcinolone, Fluticasone

Low Potency: Desonide, Hydrocortisone

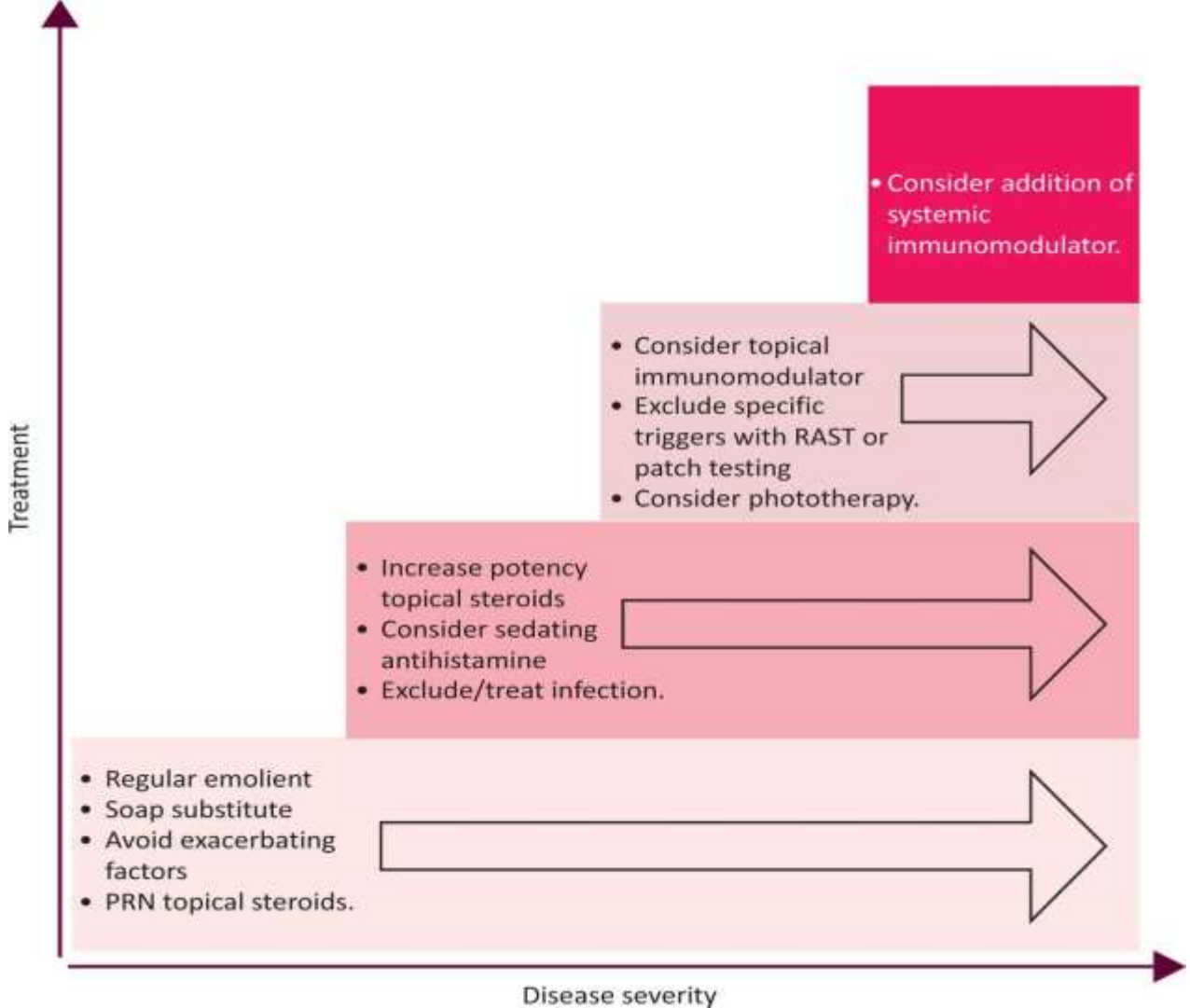
Topical Calcineurin Inhibitors

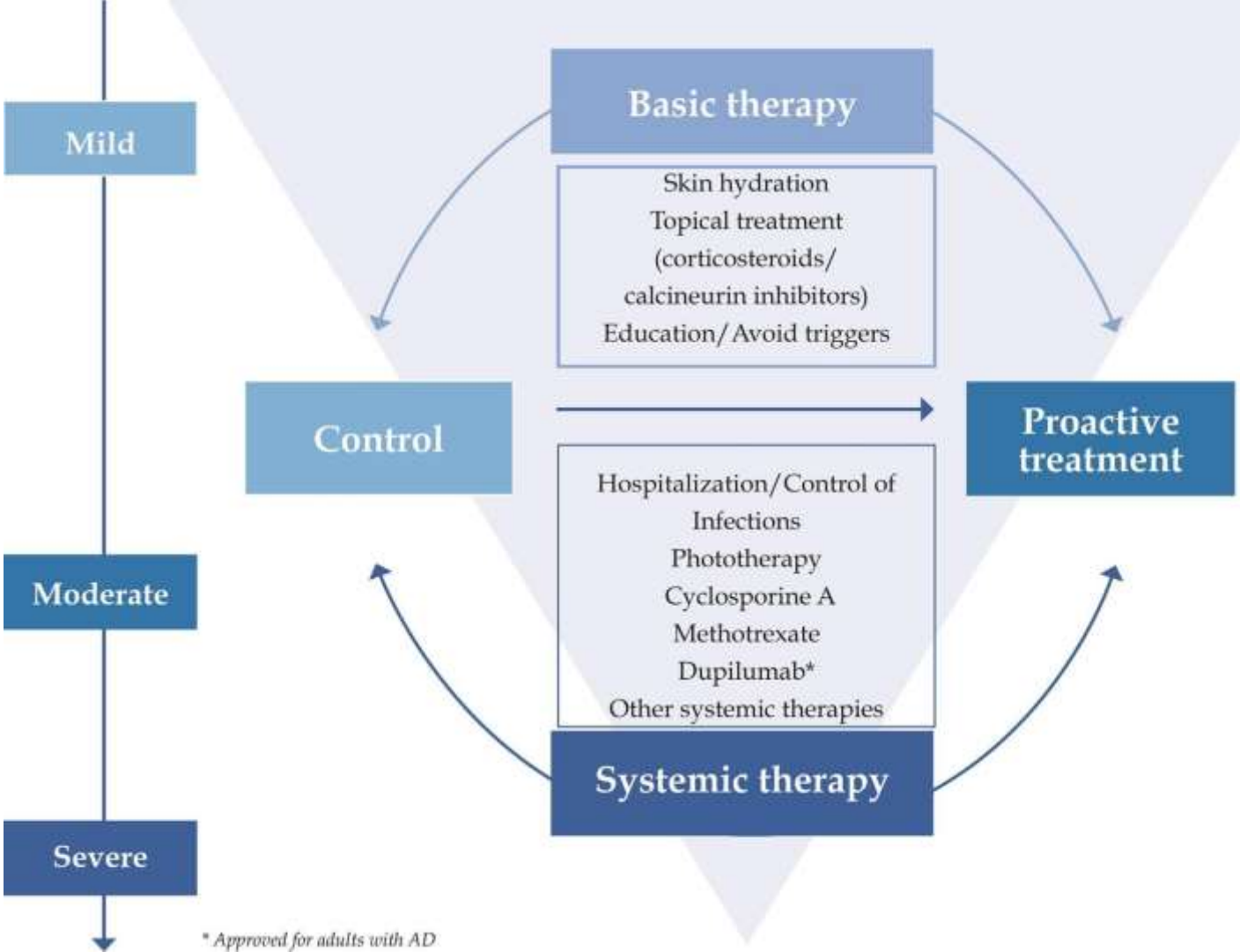
Elidel (pimecrolimus)

Protopic (tacrolimus)

Phosphodiesterase-4 (PDE4) Inhibitor

Eucrisa (crisaborol)


Treatment
• Consider addition of systemic immunomodulator.
• Consider topical immunomodulator
• Exclude specific triggers with RAST or patch testing
• Consider phototherapy.
• Increase potency topical steroids
• Consider sedating antihistamine
• Exclude/treat infection.
• Regular emolient
• Soap substitute
• Avoid exacerbating factors
• PRN topical steroids.
Disease severity


Mild
Moderate
Severe
Basic therapy
Skin hydration
Topical treatment
(corticosteroids/
calcineurin inhibitors)
Education/Avoid triggers
Control
Proactive treatment
Hospitalization/Control of
Infections
Phototherapy
Cyclosporine A
Methotrexate
Dupilumab*
Other systemic therapies
Systemic therapy
* Approved for adults with AD

Psoriasis

Psoriasis

Psoriasis is a chronic, immune-mediated dermatosis that results from a polygenic predisposition combined with environmental triggers.

The natural history is chronic with intermittent remissions.

The characteristic lesion is a sharply demarcated erythematous plaque with scale; the plaques may be localized or widespread in distribution.

Two peaks in age of onset have been reported:

one at 20–30 years of age and a second peak at 50–60 years.

In approximately 75% of patients, the onset is before the age of 40 years, and in 35–50%, it is before the age of 20 years.

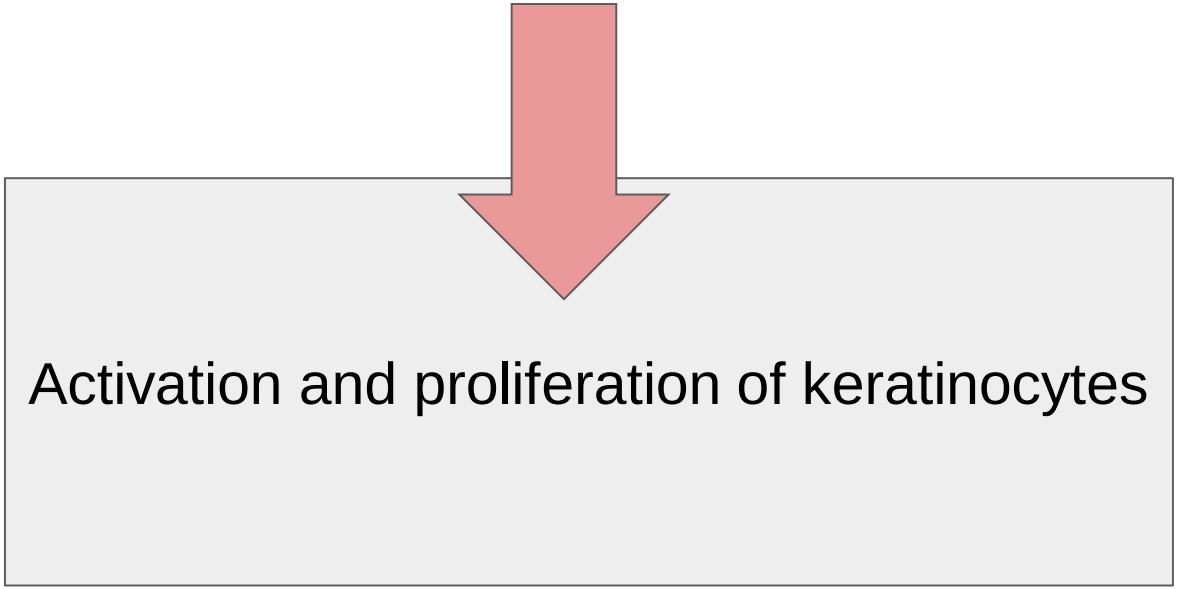
Type I disease (HLA-Cw6+) have an earlier onset, more widespread disease and frequent recurrences, compared to those with type II psoriasis.

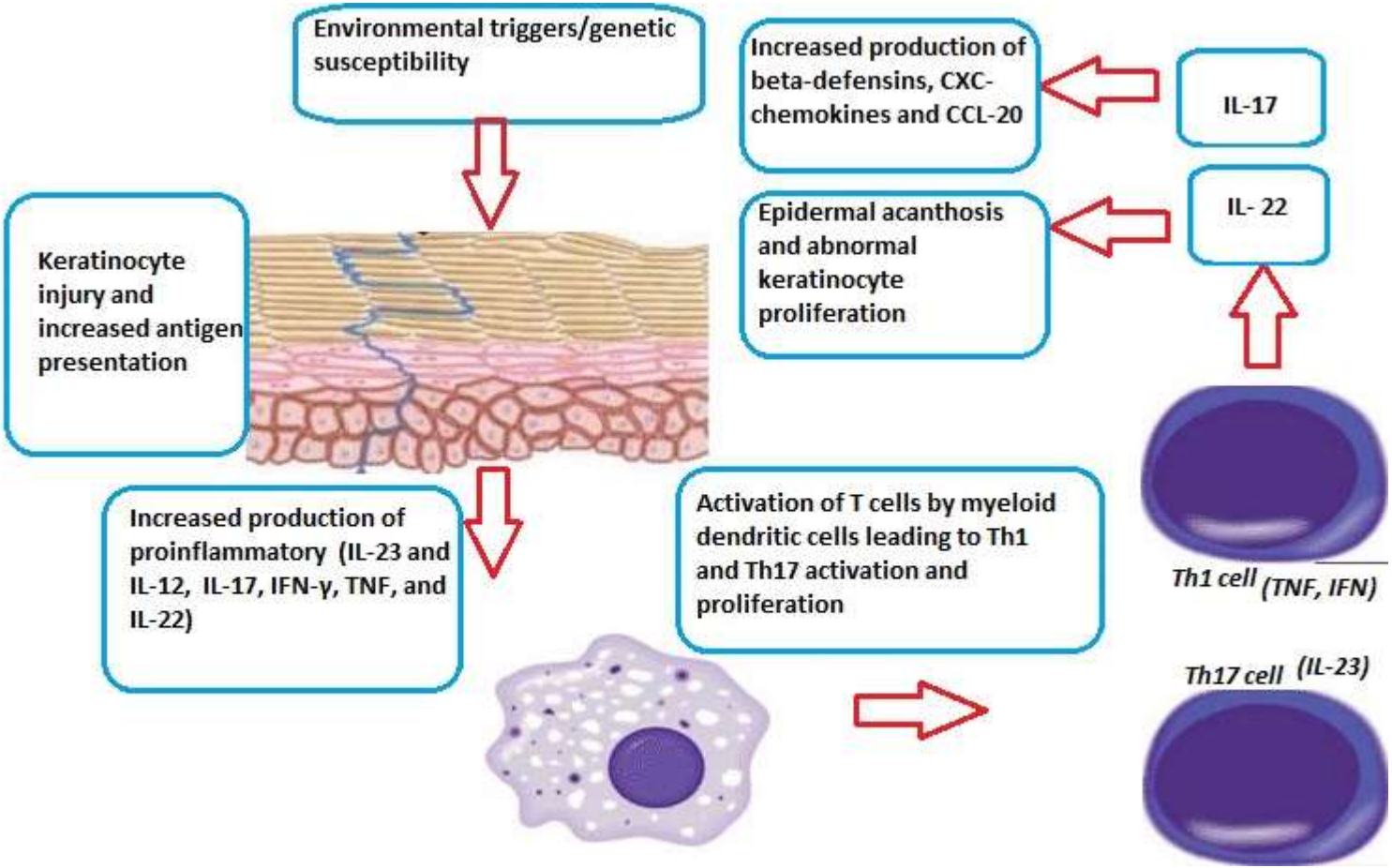
Genetic factors

- a positive family history has been reported by 30%of patients with psoriasis
- If one parent has psoriasis, the risk of their childdeveloping psoriasis is 10%
- if both parents has psoriasis, the risk of their childdeveloping psoriasis is 50%
- concordance rates among monozygotic tweens is 70-90%

Immunopathogenesis

Psoriasis is considered to be a disease with prominent involvement of helper T-cell subsets (Th1 and Th17) and their secreted cytokines: TNF – alfa, interleukin 12, interleukin 23, interleukin 17




Environmental triggers/genetic susceptibility
Keratinocyte injury and increased antigen presentation
Increased production of proinflammatory (IL-23 and IL-12, IL-17, IFN-γ, TNF, and IL-22)
Activation of T cells by myeloid dendritic cells leading to Th1 and Th17 activation and proliferation
Th1 cell (TNF, IFN)
Th17 cell (IL-23)
Epidermal acanthosis and abnormal keratinocyte proliferation
IL- 22
Increased production of beta-defensins, CXC-chemokines and CCL-20
IL-17

External triggering factors:

- UV radiation, rentgen radiation, surgery, iniections, burns, expositions for toxic substances.

- morbilliform drug eruption, viral exanthem, rosacea, contact dermatitis.

WAX CANDLE SIGN: If the superficial silvery white scales are removed via curettage(grattage method), a characteristic coherenceis observed, as if one has scratched on a wax candle (“signe de la tache de bougie”).

AUSPITZ SIGN: If the latter is removed, then awet surface is seen with characteristic pinpoint bleeding. This finding, is the clinical reflection of elongated vessels in the dermal papillae together with thinning of the suprapapillary epidermis.

KOEBNER PHENOMENON: after skin injury typical psoriatic leasions are seen in affecte skin.

Psoriatic arthritis

Psoriatic arthritis is more prevalent among patients with relatively severe psoriasis.

Risk factors for a more severe course of the arthritis include:

initial presentation at an early age,

female

gender,

polyarticular involvement,

genetic

predisposition,

radiographic signs of the disease early on.

Psoriasis treatment

TOPICAL:

Corticosteroids, vitamin d3 analogues, retinoids (tazaroten), anthralin acetylic acid

SYSTEMIC:

PUVA/UVB311nm

Retinoids (acitretin)

Metotrexat

Cyclosporine A

Biologics agents (infliximab, etanercept, adalimumab, guselkumab, ustekinumab, ixekizumab)

Topical glicocorticosteroids

They are often first-line therapy in mild to moderate psoriasis and in sites such as the flexures and genitalia, where other topical treatments can induce irritation.

• Once-daily application has been shown to be as effective as twice-daily application, and long-term remissions may be maintained by applications on alternate days

Erysipelas and cellulitis

Erysipelas and cellulitis

Erysipelas is a bacterial (usually streptococcal) infection of the dermis and upper subcutaneous tissue, its hallmark is a well-defined, raised edge.

• Cellulitis is an acute, subacute or chronic inflammation of loose connective tissue, mainly applied to inflammation of subcutaneous tissue in which an infective, generally bacterial, cause is proven or assumed.

- the commonest site is leg
- site of entry: superficial wound, ulcer or inflammatory lesion (e.g. tinea)
- complications: fasciitis, myositis, subcutaneous abscesses, septicaemia, nephritis, in more severe cases can be fatal
  - constant features are erythema, heat, swelling, pain or tenderness
  - edge in erysipelas is well-demarcated and raised, in cellulitis it is diffuse
  - severe cellulitis may also show bullae and can progress
  - to dermal necrosis and uncommonly to fasciitis and myositis
  - lymphangitis and lymphadenopathy are frequent

Treatment

- Penicillinase-resistant antibiotics (i.v. or p.o. In early stages)

- Management of the underlying condition

Furuncule

Furuncule (boil)

Definition: acute, necrotic infection of a hair follicle with S. aureus

• more common in adolescence and in early adulthood, uncommon in early childhood

(except in atopic subjects)

• the infecting strain of Staphylococcus is usually also present in the nares or the perineum

• Predisposing factors: mechanical damage, malnutrition, diabetes, HIV (furunculosis)

Clinical features

• small, follicular, inflammatory nodule, soon becoming pustular then necrotic

• healing after discharge of a necrotic core leaving a violaceous macule, and ultimately, a permanent scar

• rate of development is variable: days-weeks pain, occasionally fever, rarely pyaemia, septicaemia

Common sites

- face
- neck
- arms
- wrists
- fingers
- buttocks
- anogenital region

Complications

Cavernous sinus thrombosis
is a rare and dangerous complication of the furuncle on the upper lip and cheek.

Treatment

Systemic:

• penicillinase-resistant antibiotic

Topical:

• topical antibacterial agent (reduces contamination of the surrounding skin)