



# Eczema and dermatitis

- Allergic contact dermatitis
- Irritant contact dermatitis
- Atopic dermatitis
- Microbial eczema
- Seborrheic dermatitis



# Allergic contact dermatitis

makes **5 – 15%** of all dermatoses

■ prevalence – **1,5-3%**

■ incidence – **5-10 / 1000** per year

■ **Hypersensitive reaction of the  
IVth type according Coombs & Gell**

# Allergic contact dermatitis

- **contact allergens** – molecules smaller than **500 D** – *penetration through the skin barrier*
- **binding of the molecule – hapten - to pt's own proteins in the skin forms an antigen** – with the molecular weight at least **5000 D**
- the conjugation of haptens with proteins takes place in LC (**antigen presenting cells**)

# Allergic contact dermatitis

**Induction phase** Penetration of allergen through the stratum corneum Interaction with APC

Phagocytosis of antigen  
subsequent expression of antigen on the surface of LC  
Migration to regional lymphatic nodes and presentation of the antigen to naive T-lymphocytes

# Allergic contact dermatitis

## Elicitation phase – in case of sensitization

proliferation of specific

Clone of effector T-lymphocytes migration  
to the site of allergen penetration

Cytotoxic effect of T-lymphocytes releasing  
cytokines leading to inflammation

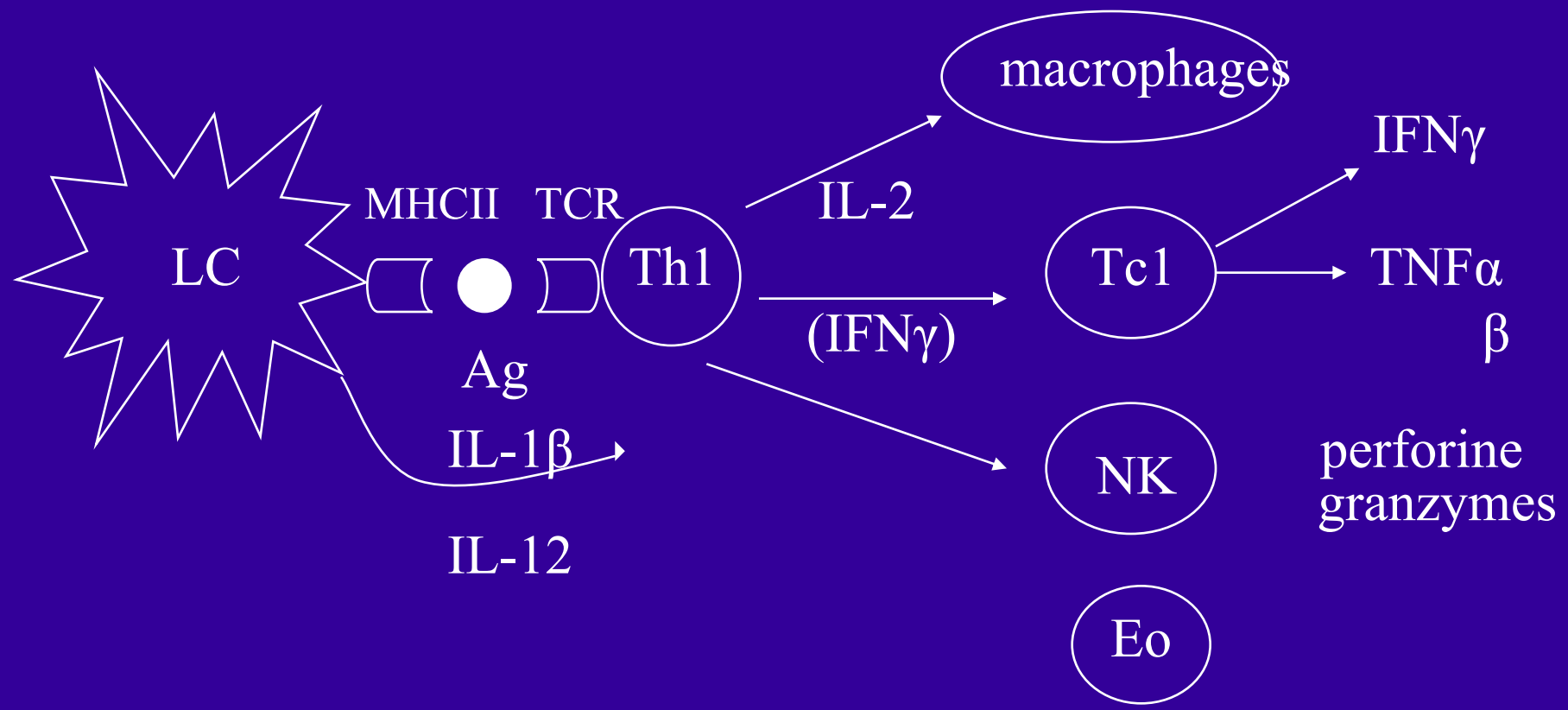
→ **allergic contact dermatitis**

**Shortest time to sensitization: 5-14 days**

migration of LC to regional LN takes about 5-24 hours

proliferation of T-lymphocytes – 5-10 days)

# Patophysiology of the late-type hypersensitivity



# Allergic contact dermatitis

## Factors influencing the ease of sensitisation:

- ◆ **Chemical structure of allergens**
- ◆ **Patient** – skin barrier status (fissures, maceration)  
localisation (eyelids x soles)  
age
- **Duration of hypersensitivity**
  - survival time of memory T-lymphocytes
  - character of allergens

# European Standard Series

- **Potassium dichromate 0,5 % pet.**
- **Neomycin sulphate 20 % pet.**
- **Thiuram mix 1% pet.**
- **Paraphenylenediamine 1% pet.**
- **Cobalt chloride 1% pet.**
- **Caine mix 10% pet**
- **Formaldehyde 1% aq.**
- **Colophony 20% pet.**
- **Hydroxyethyl metacrylate 2% pet.**
- **Balsam of Peru 25 % pet.**
- **N-isopropyl-N-phenyl-4-phenylenediamine 0,1% pet.**
- **Wool alcohols 20% pet.**
- **Mercapto mix 2% pet.**
- **Epoxy resin 1% pet.**
- **Paraben mix 16%**



# European Standard Series

- **pet.4-t- butylphenol formaldehyd resin 1% pet.**
- **Fragrance mix 8% pet.**
- **Quaternium 15 1% pet.**
- **Nickel sulphate 5% pet.**
- **Kathon CG 0,01% aq.**
- **Mercaptobenzothiazole %pet.**
- **Sesquiterpenlactone mix 0,1% pet.**
- **Propolis 10% pet.**
- **Tixocortol-21-pivalate 0,1% pet.**
- **Budesonide 0,01% pet.**
- **Methyldibromoglutaronitrile (1,2-dibromo-2,4-dicyanobutane)\***
- **Fragrance II 14% pet.**
- **Lyral 0,5 % pet.**
- **Methylisothiazolinone 0,02% aq**
- **Textile dye mix 6,6% pet.**



**Metal glasses**



**Metal ring**

**Allergic contact dermatitis – nickel**



**Metal watch**



**Metal button**

**ACD to chromium from leather boots**





**ACD- IPPD, antioxydant  
of black rubber**



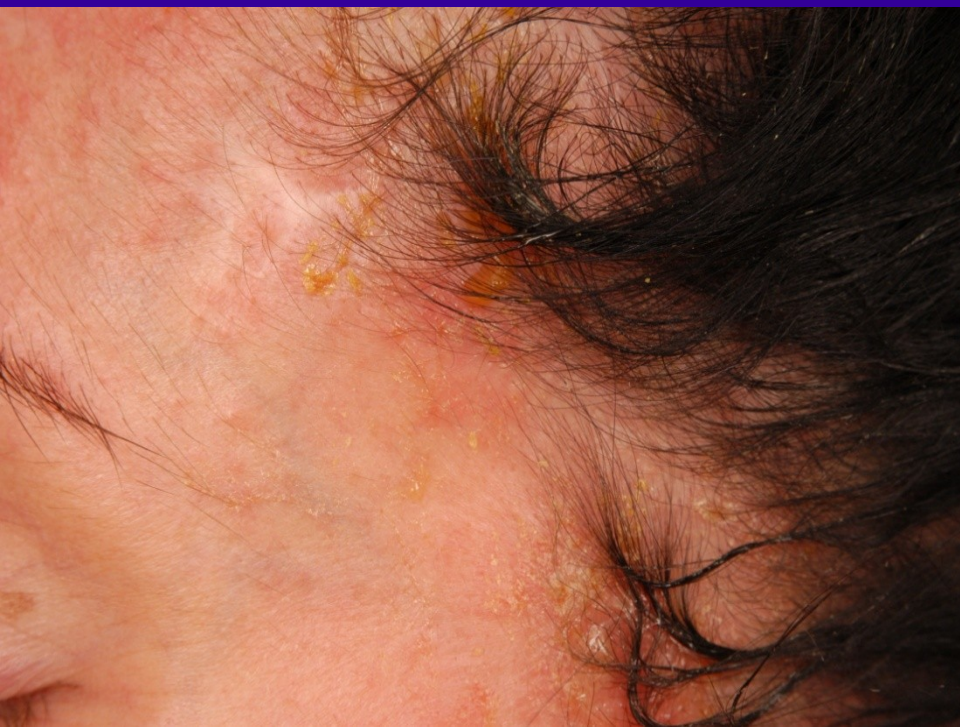
**Tonometer, stethoscope  
(nurse)**



**Rubber boot**



**ACD to PPD from hair dyes**





**ACD to fragrance (eau de toilette)**



# Corticosteroids

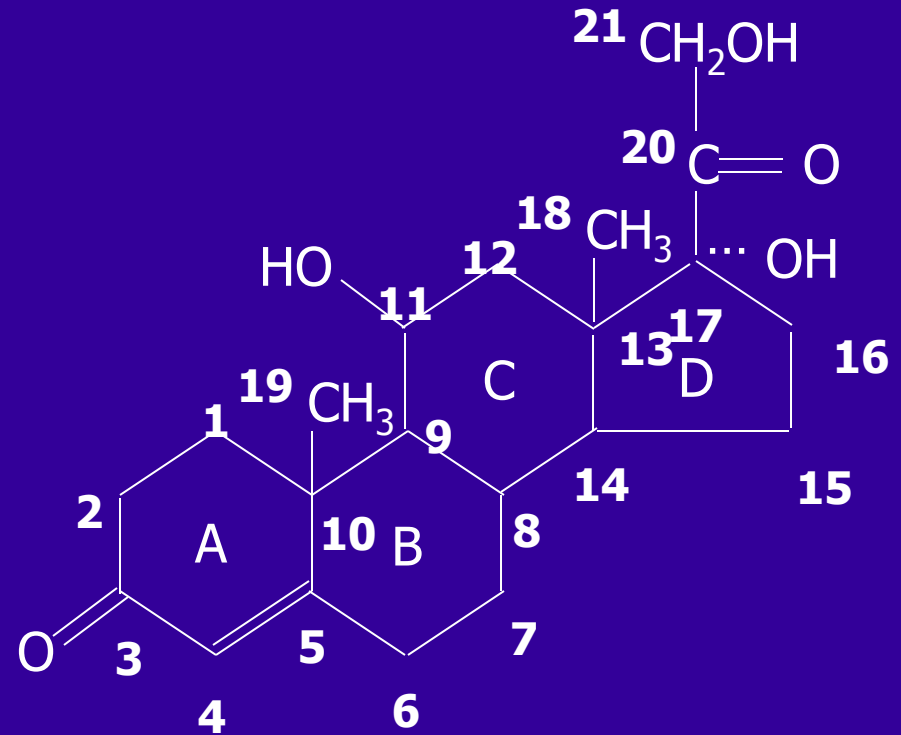
A - type **Hydrocortison**: D kruh nesubstituovaný, C 20, C 21 nesubstituovaný nebo C 17, C 21 krátký řetězec (acetáty nebo estery), event. C 21, thioester

B - type **Triamcinolon acetonid**: C 16, C 17 cis-ketal struktura nebo diol struktura

C - type **Betametason**: C 16 methyl substituce

D - type **Hydrocortison**

**butyrát**: C 17, a/nebo C 21 dlouhé esterové řetězce, event. C 16 methyl substituce





Patch test—  
contact  
allergy to  
**Budesonide**

**Budesonide** - Apulein ung, crm, liq, Pulmicort aer inh,  
Pulmicort, Turbuhaler plv inh, Rhinocort spr nas



Budesonid 1%

Betamethasoni dipropionat 0,05%

72h.

ec. 2%

Budesonid 0,025%

Rhinocort spray 72h.

ison ec. 1%

Hydrocortison



**Drug eruption in patient sensitized to topical CS after systemic exposure to - Prednisone tbl**



# Fragrances

## Fragrance mix I

- ❖ Cinnamic aldehyde
- ❖ Cinnamic alcohol
- ❖           myl-cinnamic aldehyde
- ❖ Eugenol
- ❖ Isoeugenol
- ❖ Geraniol
- ❖ Hydroxycitronellal
- ❖ Oak moss absolute (Akranorin)
- Sorbitan sesquioleate (emulgator)

## Frequency of sensitization:

worldwide

4,7-13,3%



**Allergic contact  
dermatitis—  
fragrance —  
cosmetic cream**



**Patch tests —  
contact allergy to  
fragrance and  
cinnamic alcohol**

# Fragrances

## Fragrance mix II

Lylal, Citral, Farnesol, Citronellol,  
hexyl cinnamic aldehyde, Coumarine

## Fragrance mix III

yasmine absolute 2,0 % vaz. Amylcinnamaldehyde 2,0 % vaz. Musk  
ketone 1,0 % vaz. Sandalwood oil 2,0 % vaz. Musk moskene 1,0 %  
vaz. Ylang-ylang 2,0 % vaz. Cananga oil 2,0 % vaz. Vanilin 10,0 %  
vaz. Jasmine synthetic 2,0 % vaz. Geranium oil Bourbon 2,0 % vaz. Musk  
xylene 1,0 % vaz. Lavaner absolute 2,0 % vaz. Rose oil 2,0 %  
vaz. Narcissus absolute 2,0 % vaz. Methyl anthranilate 5,0 % vaz. Benzyl  
salicylate 2,0 % vaz. Benzyl alcohol 1,0 % vaz.

## Balsam of Peru

## Propolis

# Propolis

- natural product – is a resinous mixture that honey bees collect from tree buds, sap flows, or other botanical sources. The chemical composition of propolis varies depending on season, bee species and geographic location. Propolis has approximately **50 constituents**, primarily resins and vegetable balsams (50%), waxes (30%), essential oils (10%), and pollen (5%). Propolis has antibacterial, fungicidal, antipruritic and antiinflammatory effects and promotes epithelisation



**Allergic contact  
dermatitis–  
propolis (folk medicine  
preparations)**





Allergic contact  
dermatitis—  
**propolis** (folk medicine  
preparations)





# „new“ allergens

**Ketoprofene** – nonsteroidal antiinflammatory drug

**Derivative of propionic acid**

**Ketoprofene** – **topical**

Fastum

Profenid gel

Ketonal crm

and others

**systemic**

Ketoprofen tbl,sup

Ketonal cap,sup amp i.m.

Ketonal forte tbl

Ketonal ret tbl

Profenid cap,tob,sup amp

Profenid 100 mg pro inf

Toprec tbl

**Allergy potentiated by sun exposure – photocontact allergy**



**Photocontact allergy - ketoprofene  
(Fastum gel)**



**Patch test -  
alergická reakce  
na Fastum gel**



**Patch test -  
alergická reakce na  
ketoprofene**

**Photocontact allergy - ketoprofene -  
generalizace (Fastum gel)**

# Tea Tree Oil

**source:** leaves of the tea tree (*Melaleuca alternifolia*)

**occurrence:** Australia, Spain, Portugal

**use:** folk /traditional/ medicine

**effects:** antiseptic

antifungal

antibacterial

# Components of Tea Tree Oil

## Mixture of mono and sesquiterpens

- |                 |         |                |            |
|-----------------|---------|----------------|------------|
| ❖ Terpinen-4-ol | 30-45%  | ❖ 1,8 Cineol   | 0-15%      |
| ❖ Terpinen      | 10-28%  | ❖ Camphor      | stopa-8%   |
| ❖ γ-Terpinen    | 5-13%   | ❖ Aromadendren | stopa-7%   |
| ❖ Terpineol     | 1,5-8%  | ❖ Sabinen      | stopa-3,5% |
| ❖ Terpinolen    | 1,5-5%  | ❖ Globulol     | stopa-3%   |
| ❖ Pinen         | 1-6%    | ❖ Viridiflorol | stopa-1,5% |
| ❖ Cymene        | 0,5-12% | ❖ β-Caren      | stopa-0,2% |
| ❖ d-Limonen     | 0,5-4%  |                |            |



**Allergic contact dermatitis – tea tree oil  
(cosmetic preparations)**



**Patch tests –  
contact allergy to  
tea tree oil and  
other etheric oils**

# **Plant extracts**

## **family of Compositae**

**main allergens - sesquiterpenolaktone**

**Extr. Chamomillae - chamomile**

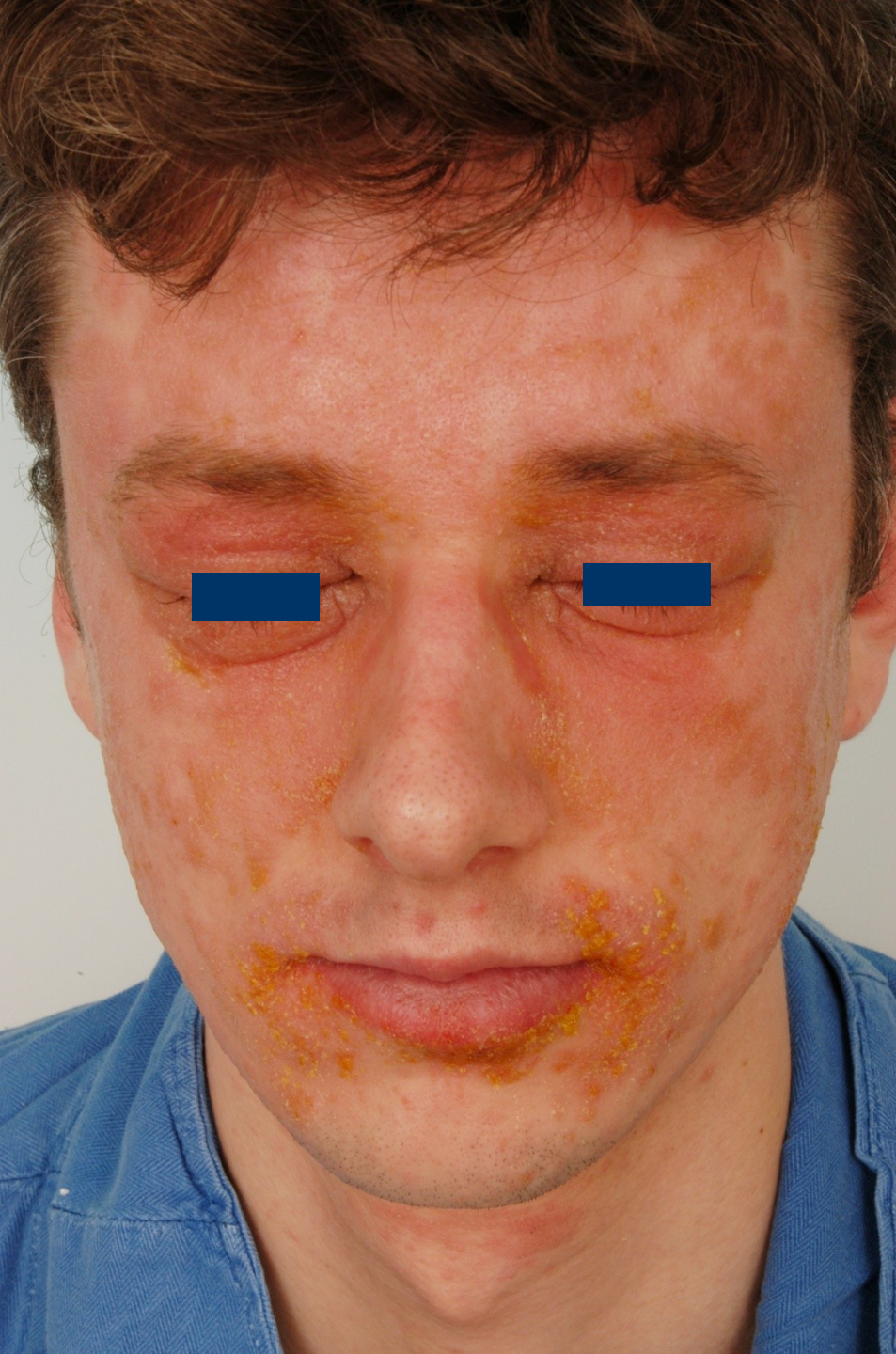
**Extr. Calendulae - marigold**

**Extr. Arnicae - arnica**

**others:**

**Sunflower - Helianthus annuus,**

**Chrysanthemum, Cynia, Astra etc.**



**ACD to marigold (extr. Calendulae)**

**ACD to marigold in the terrain of atopic dermatitis**





**Eczema contactum -**  
**chloramphenicol, extr.**  
**Chamomillae**



**Eczema atopicum et**  
**contactum - extr.**  
**Chamomillae**



**Eczema contactum -**  
**Neomycin, extr.**  
**Chamomillae**



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# Irritant contact dermatitis

- Nonallergic reaction
- Dose dependent
- Exposition to exogenous more or less toxic agent
- More common than allergic contact dermatitis

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# Irritant contact dermatitis

## Causes:

- **chemical agents:**
  - alkaline & acid solutions
  - Organic solvents (toluene...)
  - Detergents
  - Disinfectants
  - Food stuffs (fruit acids, mustard...)
  - Even water
- **physical agents:** UV radiation, heat, cold, mechanical factors

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# Clinical picture

- Lesion sharply bordered
- Intensity depends on the toxicity of the substance (more toxic.. more acute reaction)
- Toxic agents:

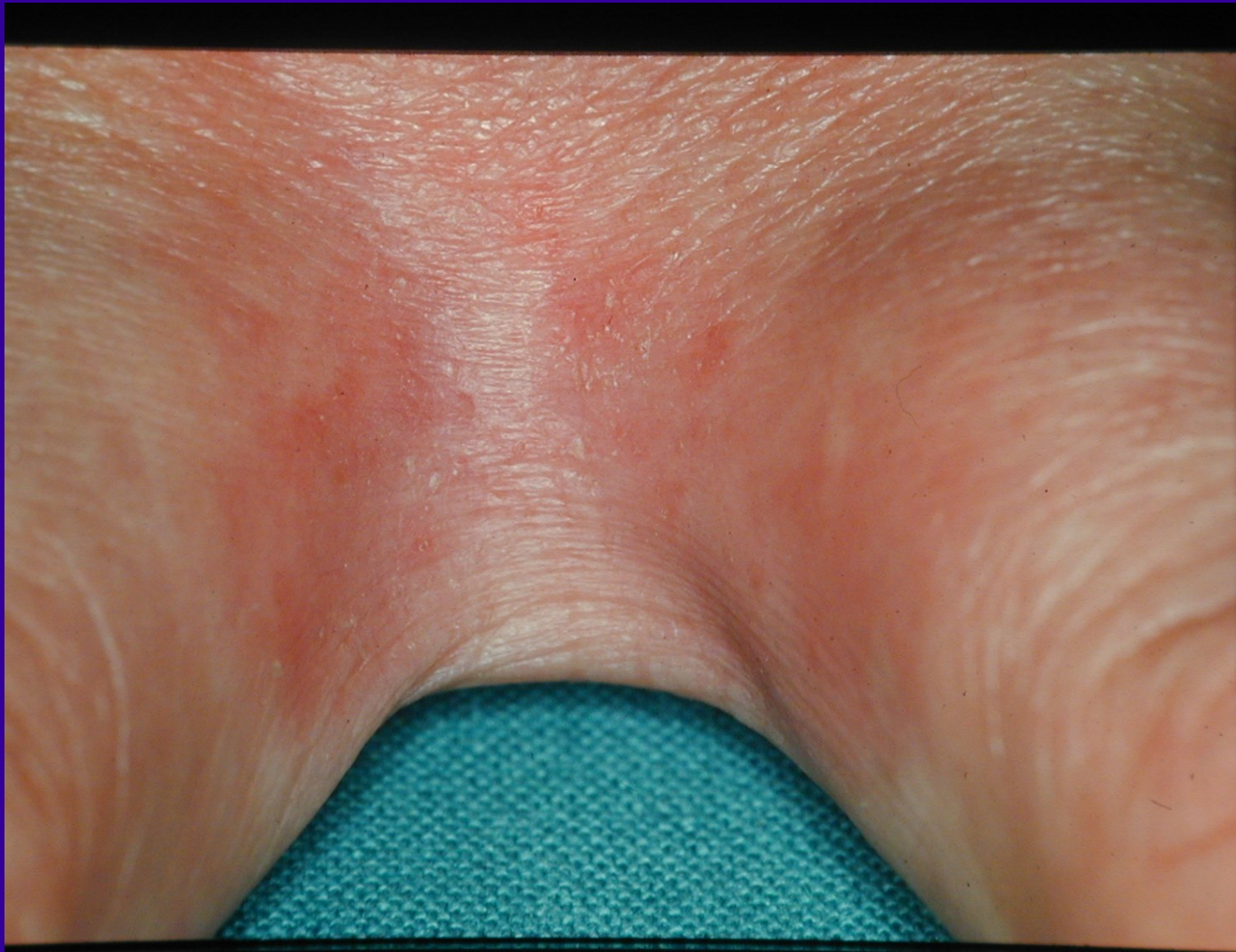
redness – swelling - blisters - necrosis

- Less toxic agents – chronic ICD

Redness, scales, lichenification, hyperkeratosis

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# Acute ICD



# Chronic ICD



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# Treatment of ACD & ICD

## Topical corticosteroids

### Class I - low potency CS

HCT acetate (HCT ung.), DXM acetate (DXM crm.)

### Class II mid-potent CS

HCT butyrate (Locoid crm., lotio), TMC acetonid (TMC crm.), alclomethason (Afloderm crm, ung.)

prednikarbate (Dermatop crm., ung.)

methylprednisolon aceponate (Advantan crm.)

### Class III - potent CS

betamethasone dipropionate (Beloderm, Diprosone crm.)

fluocinolone acetonide ( Gelargin gel, ung.)

momethason furoate ( Elocom crm., ung., lotio)


### Class IV – very potent CS

clobethasol propionate (Dermovate crm., ung)

Antihistamines, systemic corticosteroids – short courses

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# Atopic dermatitis

- strongly pruritic chronic or chronically relapsing non-infectious dermatitis with variable morphology and clinical course, usually starting during early childhood
- often associated with positive personal or family history in terms of allergic rhinitis, conjunctivitis and bronchial asthma.
- genetic predisposition
- In about 80% associated with  IgE levels

# Atopic dermatitis - epidemiology

**Incidence in population: 0,5 - 5%**  
(higher incidence – scandinavian countries)

<b>infants</b>	<b>16%</b>
<b>children under 2 y</b>	<b>14%</b>
<b>children under 14 y</b>	<b>12%</b>
<b>adults</b>	<b>2%</b>



# Atopic dermatitis

two forms, same clinical picture

**extrinsic 80%**

**elevated IgE**

sensitization to airborne  
and/or food allergens (sIgE)

- **association with allergic  
rhinoconjunctivitis and/or  
allergic asthma**

**intrinsic 20%**

**normal levels of IgE**

**skin barrier disturbance**

# **Etiology of AD: unknown**

**basis = genetic predisposition**

- 1) skin barrier disturbance**
- 2) hyperreactivity of the skin**

**environmental triggers:**

- 1) irritant substances, allergens**
- 2) stress**
- 3) many others ....**

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## I. skin barrier disturbance

Genetically conditioned:

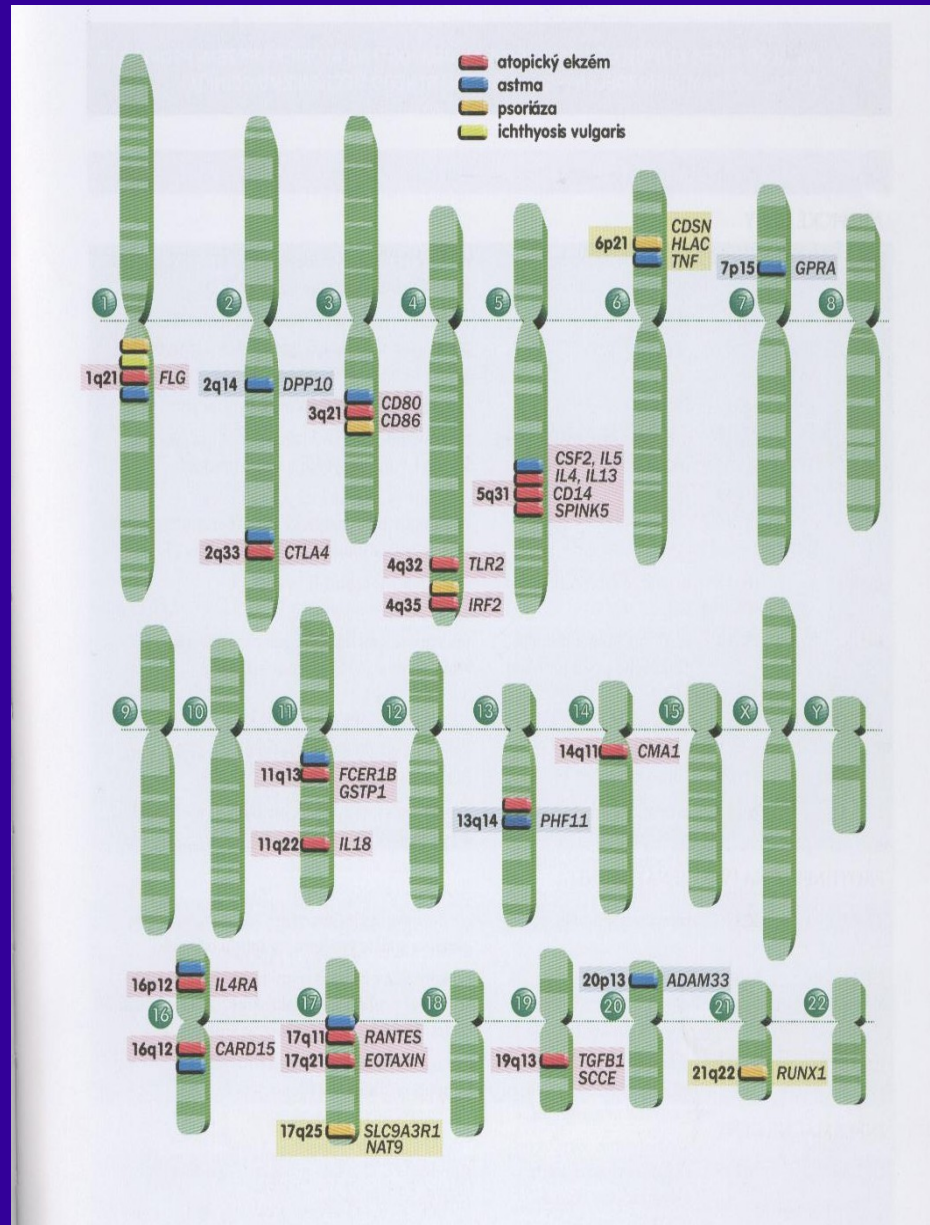
**Filaggrin:** null mutation of FLG R501X and 2282del4 alleles lead to increased permeability of skin barrier and they are associated with AD (in about 50% cases), as well as with ichthyosis vulgaris

**Claudin- 1, corneodesmosin**

**Increased activity of serin proteases**

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# Genes involved in AD



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## skin barrier disturbance

- Defective synthesis of ceramides

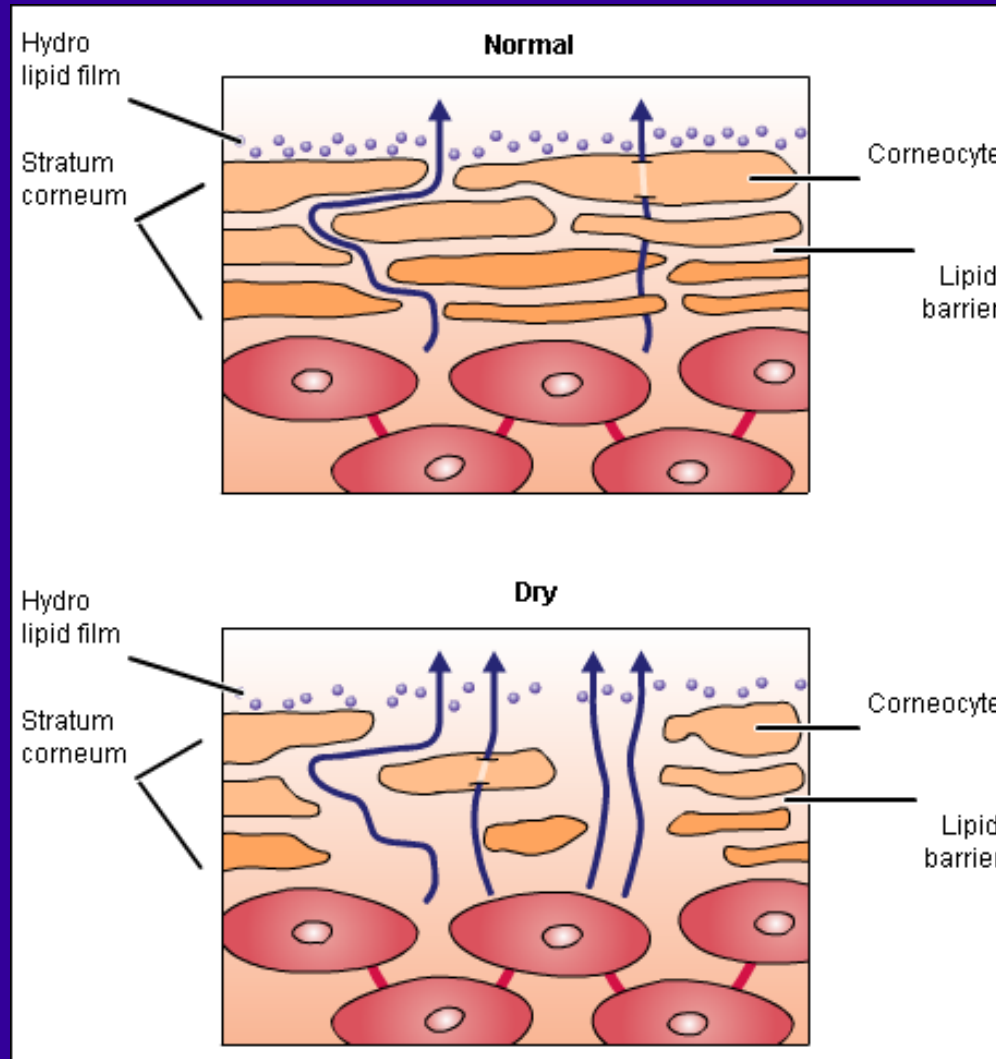
(takes place in lamellar bodies in granular layer of epidermis)



decreased ability to bind water in the skin

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# skin barrier disturbance



# AD and skin barrier

## Defective structure and function of skin barrier

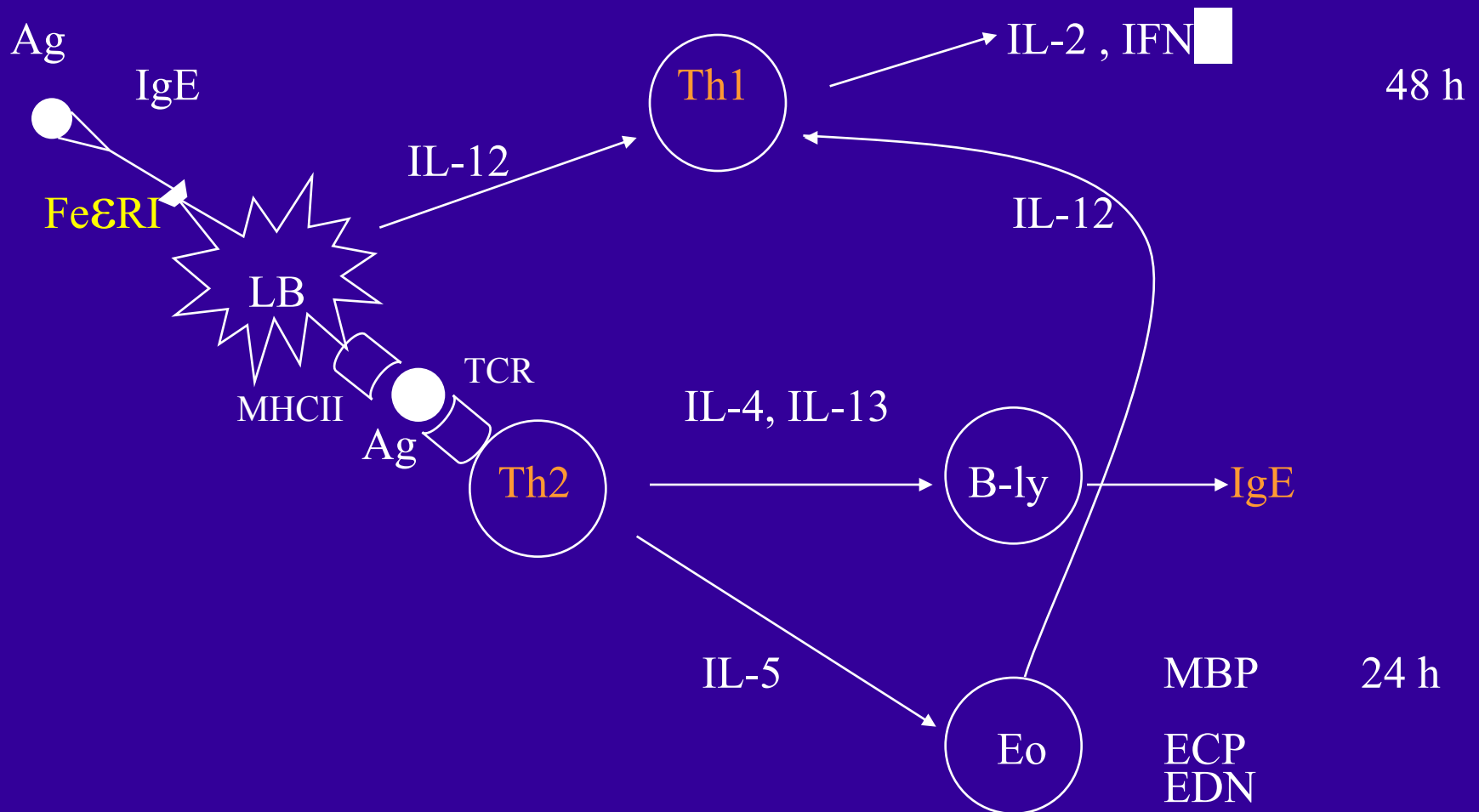
insufficient hydration (TEWL ↑)

dryness - xerosis

increased irritability of the skin  
possibility of contact sensitization

# II: Immunological abnormalities in AD

biphasic model of AD (Th2 → Th1 shift)





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## III. Staphylococcus aureus and AD

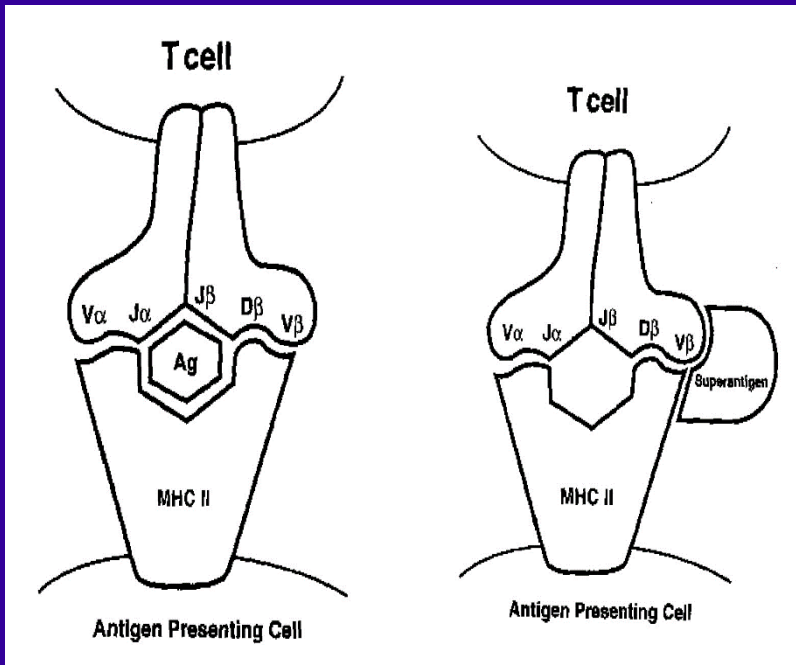
- colonization of AD lesions in 74 - 96% atopic patients, 30 - 56% even on „healthy“ skin

### Mechanisms:

- Defective skin barrier with „naked“ laminin and fibronectin enables SA binding the skin
- Decreased defensive mechanisms: defective signalling via TLR 2
  - defensins and cathelicidins
  - production of IFN  $\gamma$

# Staphylococcus aureus and AD

- 1) Toxic effect: staphylococcal exfoliatine
- 2) Stimulation of sIgE production (sIgE → stimulation of basophils → histamine)
- 3) **superantigens**: SEA- SEE a TSST-1



- without previous processing by LC
- able to bridge V chain of TC Receptor,
- not necessary exact conformity of all 5 subunits of the receptor
- 1000x stimulation
- non-specific but huge stimulation of Tly (1 SA even 20% of circulating lymph.)

# Triggering and mainaining factors of AD

**Allergy** ( house dust mites, pollen, pets, molds, foods — milk, eggs, wheat, soya, nutts, fish )

**Microbes** — Staphylococcus aureus

**Irritant substances** (water,detergents etc.)

- climatic (temperature, wind, low humidity ..)

**Psychological stress**

# Clinical picture of AD

## AD in infants

**Exudative form – acute eczema  
(oozing, crusting)**

**location**

- periorally

- periorbitally

**possibility of spreading - erythroderma**



**Atopic dermatitis –  
Infant AD**



**Infant AD**

# Clinical picture of AD

## AD in children and adolescents

### Decrease of exudation - lichenification

■ most often – flexural eczema

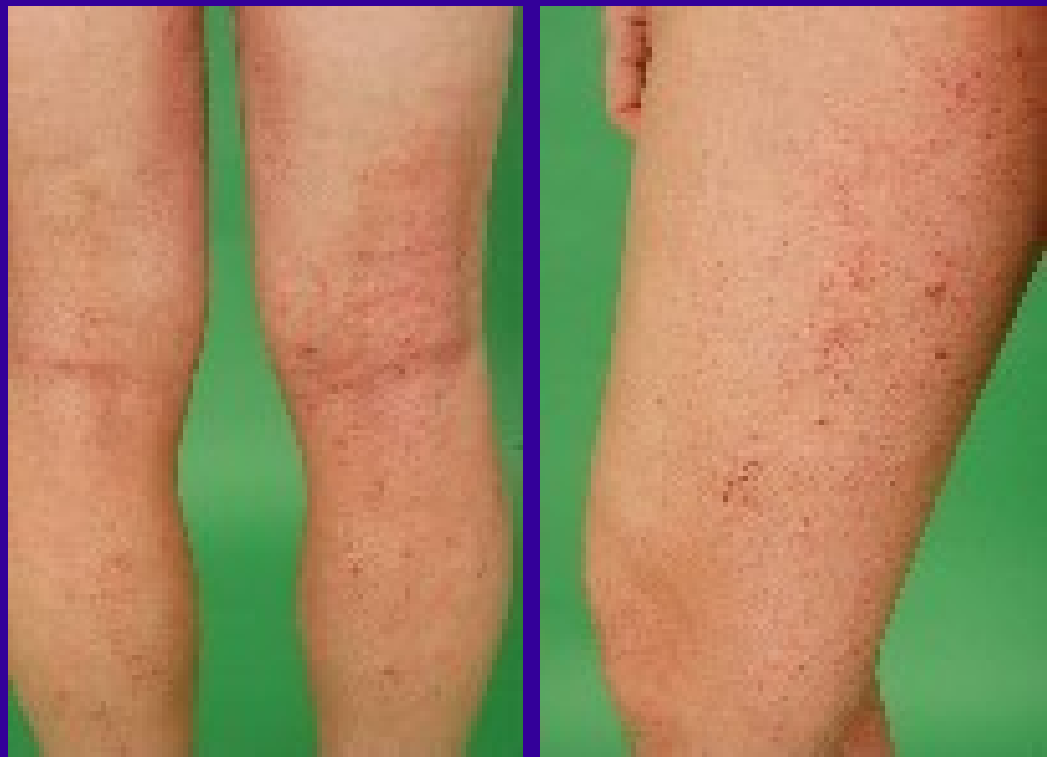
- facial eczema

■ less often - erythroderma



**Atopic dermatitis – flexural  
eczema**





**Atopic dermatitis –  
erythrodermic form**

# Clinical picture of AD

## AD in adults

(about 15% of cases appear after puberty)

flexural

prurigininous

neurodermitic

erythrodermic

chronic  
course

acute  
flares  
possible



**Adult AD – pruriginous form**



**Adult AD – neurodermitic form**



**Adult AD – erythrodermic form**

# AD in adults

**atypical forms** - nummular, dyshidrotic,  
hyperkeratotic forms

**minimal forms** - cheilitis sicca, stomatitis  
angularis, pulpitis sicca,  
intertrigo retroauricularis, aj.



**Adult AD - dyshidrotic form**



Eczema atopicum hyperkeratoticum





**AD eyelid dermatitis, lip dermatitis**





**AD retroauricular dermatitis**

# Complications of AD

**bacterial** - impetiginization (St. aureus)

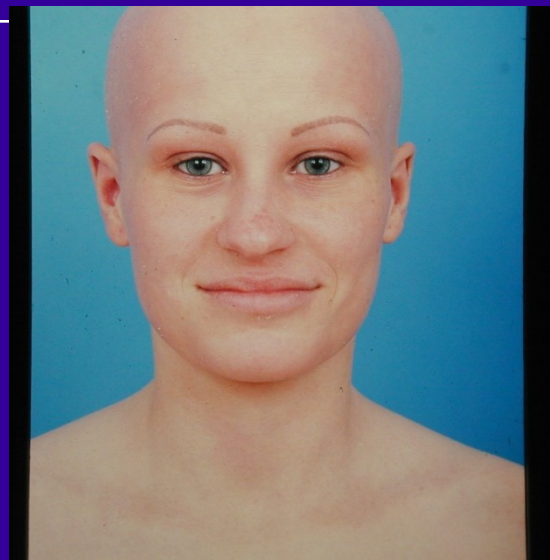
**viral** – herpetication-HSV, warts, mollusca

**fungal** (Tr. rubrum, Pityrosporum ovale)

**contact sensitization** (nickel, fragrances, KS...)

**association:**

- alopecia areata
- ichthyosis vulgaris
- vitiligo





Eczema atopicum impetiginisatum



**Eczema atopicum herpeticatum**



# Treatment of AD

*mild form of AD (30-40% of patients):*

education of patient ( or parents)

identification of triggering factor

and their elimination

emollients and baths

topical corticosteroids

pimecrolimus

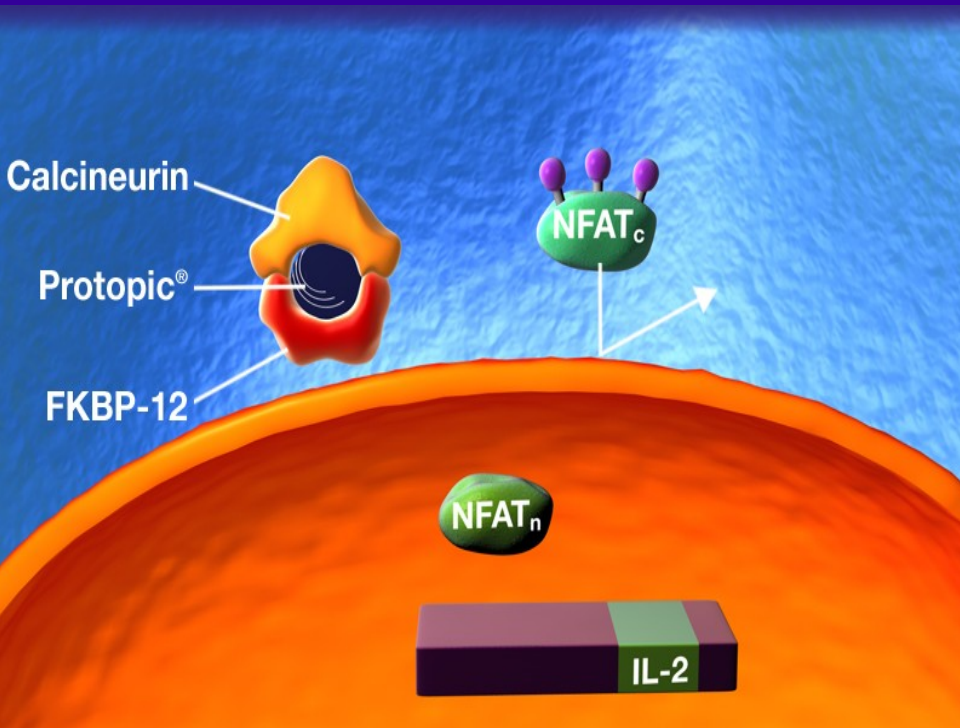
antihistamines during flares



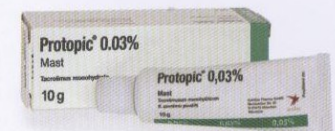
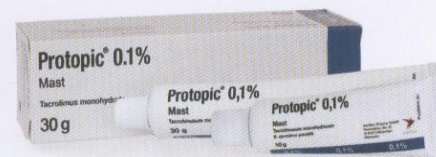
# Treatment of AD

- mid-severe form of AD (40-50% of patients):
  - treatment similar as in mild form
    - + tacrolimus
    - or
    - hospitalization – lab. and clinical tests (triggers)
      - traditional topical treatment /tar/
      - or
      - phototherapy (UVB 311nm, UVA-1)

# Tacrolimus (PROTOPIC oinment)



- Topical Immunomodulator
- Blocks calcineurin
- antiinflammatory
- antipruritic
- Long - term treatment
- No skin atrophy





# Treatment of AD

- severe form of AD (5-10% patients)
  - phototherapy (PUVA, UVA-1)
  - systemic corticosteroids (short courses)
  - immunosuppressives: cyclosporine A, MMF, AZT, MTX
  - immunomodulants: IFN  $\alpha$  (?)
  - experimental therapies: i.v. Ig
    - JAK, PDE inhibitors
    - biologicals (dupilumab...)

# Microbial eczema

Allergy of IVth type to bacterial allergens –  
mostly to Staph. aureus

appears mostly secondary:

in pyodermas, scabies, atopic dermatitis, ICD  
around fistulas, stomias, in varicous terrain on legs  
around sites of inflamamtion (chronic rhinitis, otitis)

variant: **nummular dermatitis** (coin shaped  
patches and/or plaques) usually in patients with  
focal bacterial infection (tooth gfranuloma, chronic  
tonsillitis, chronic urogenital infections etc.



**Microbial eczema**



**Microbial eczema**



**Microbial eczema in patients with CVI  
= varicous eczema**



**Microbial eczema in a patient with chronic otitis**



**Microbial eczema in a patient  
with scabies**

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# Treatment of microbial eczema

## Acute phase:

- Drying compresses
- Topical zinc preparations
- Topical corticosteroids in lotion base

## Subacute and chronic phase:

- ATB paste, endiaron paste, tar preparations
- Combination with topical CS (TMC-E, Belogent, Fucicort)

## Systemic ATBs

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# Seborrheic dermatitis

- **localisation:** seborrheic predilection sites
- **etiology:** genetic predisposition
  - dysseborrhea – altered composition of sebum
  - Malassezia furfur** = pityrosporon ovale
  - immunodeficiency - AIDS
  - depletion of zinc

Clinical picture: erythematous scaly lesions

In typical sites: scalp, eyebrows, nasolabial folds, midchest region, around umbilicus, groins & axillae

- Subjective complaints: itching, burning







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# Treatment of seborrheic dermatitis

- Topical imidazole antifungals + topical corticosteroids
- Topical imidazole antifungals
- Topical preparation with zinc
- zinc supplementation
- (Systemic antifungals)

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