



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

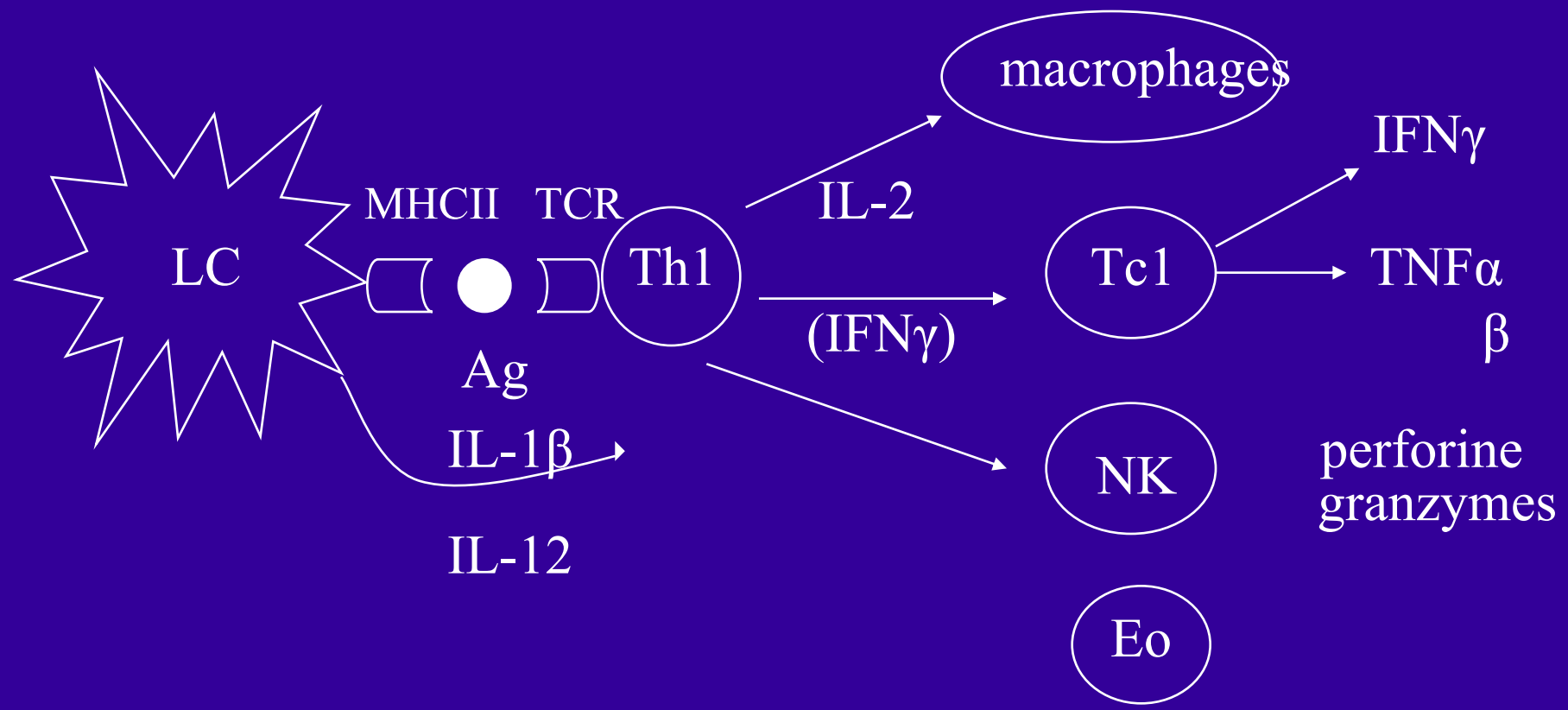
→ **allergic contact dermatitis**

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.**
- **Lyril 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**



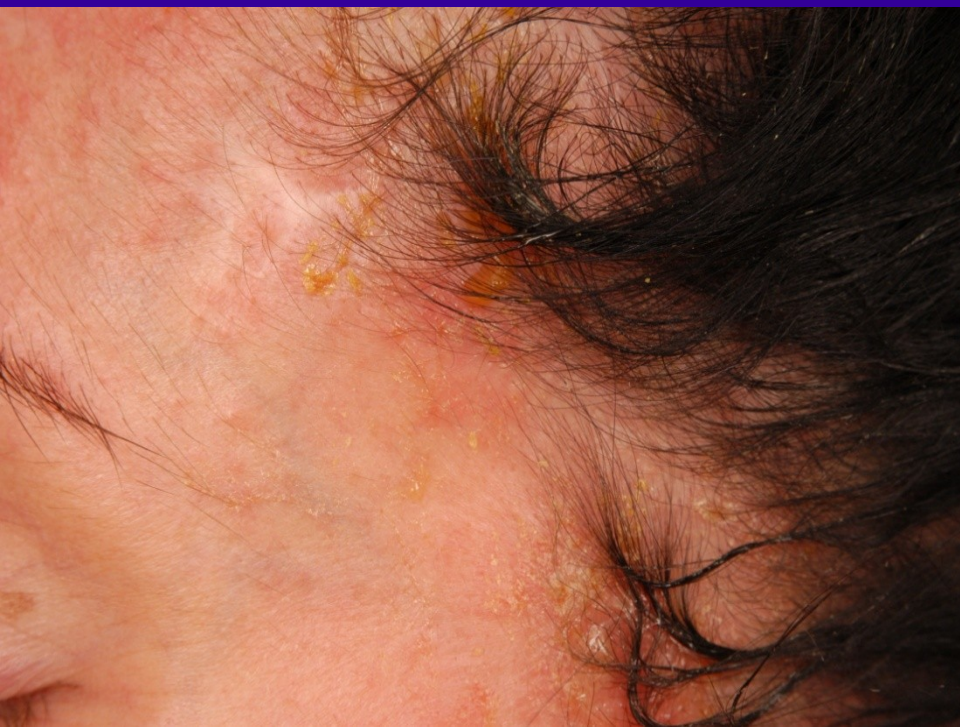
**Tonometer, stethoscope
(nurse)**



Rubber boot



ACD to PPD from hair dyes





ACD to fragrance (eau de toilette)



Corticosteroids

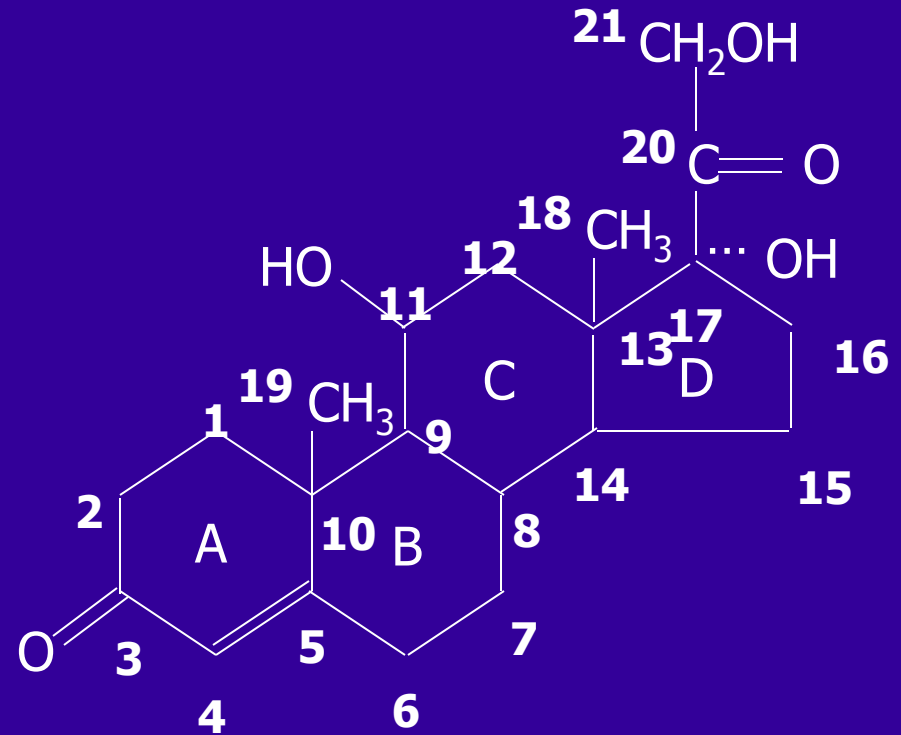
A - type **Hydrocortison**: D kruh nesubstituovaný, C 20, C 21 nesubstituovaný nebo C 17, C 21 krátký řetězec (acetáty nebo estery), event. C 21, thioester

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 unguentum, cream, liquid, Pulmicort inhaler, Pulmicort, Turbuhaler inhaler, Rhinocort nasal spray

Budesonid 1%

Betamethasoni dipropionat 0,025%

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**

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% | ❖ Cadinen | stopa-8% |
| ❖ 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

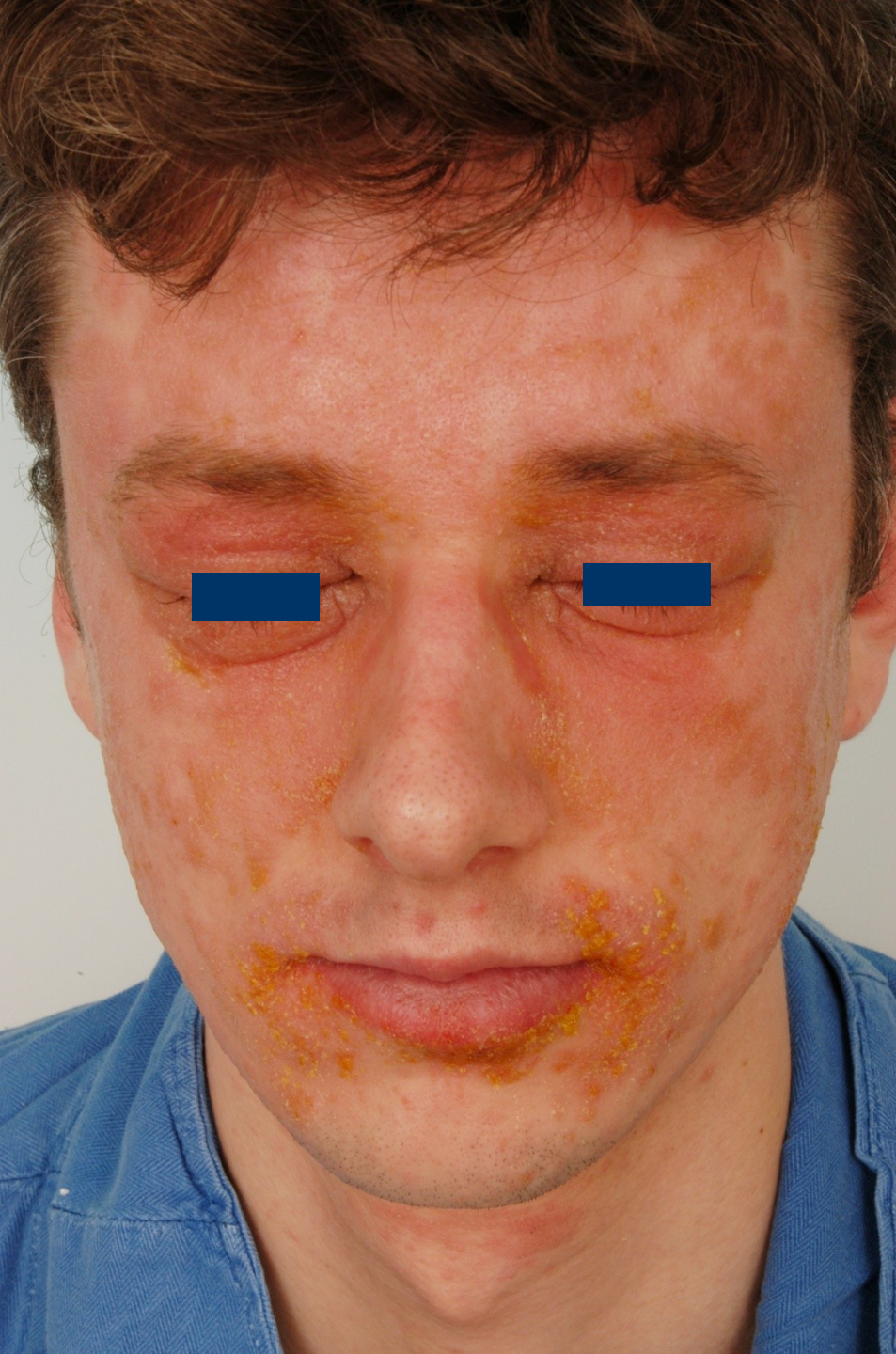
Extr. Calendulae - marigold

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



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

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)

infants	16%
children under 2 y	14%
children under 14 y	12%
adults	2%

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:

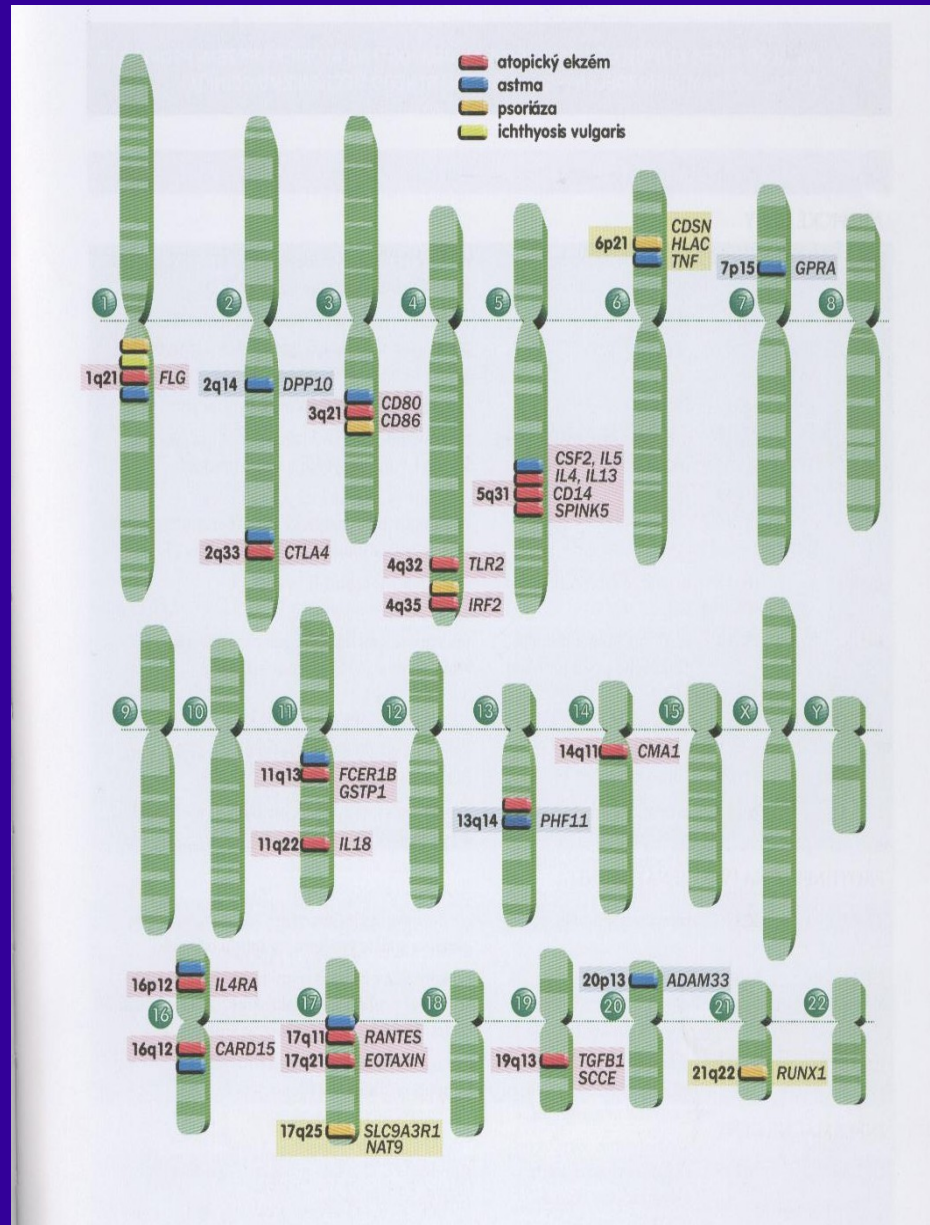
Filaggrin: null mutation of FLG R501X and 2282del4 alleles lead to increased permeability of skin barrier and they are associated with AD (in about 50% cases), as well as with ichthyosis vulgaris

Claudin- 1, corneodesmosin

Increased activity of serin proteases

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Genes involved in AD





skin barrier disturbance

- Defective synthesis of ceramides

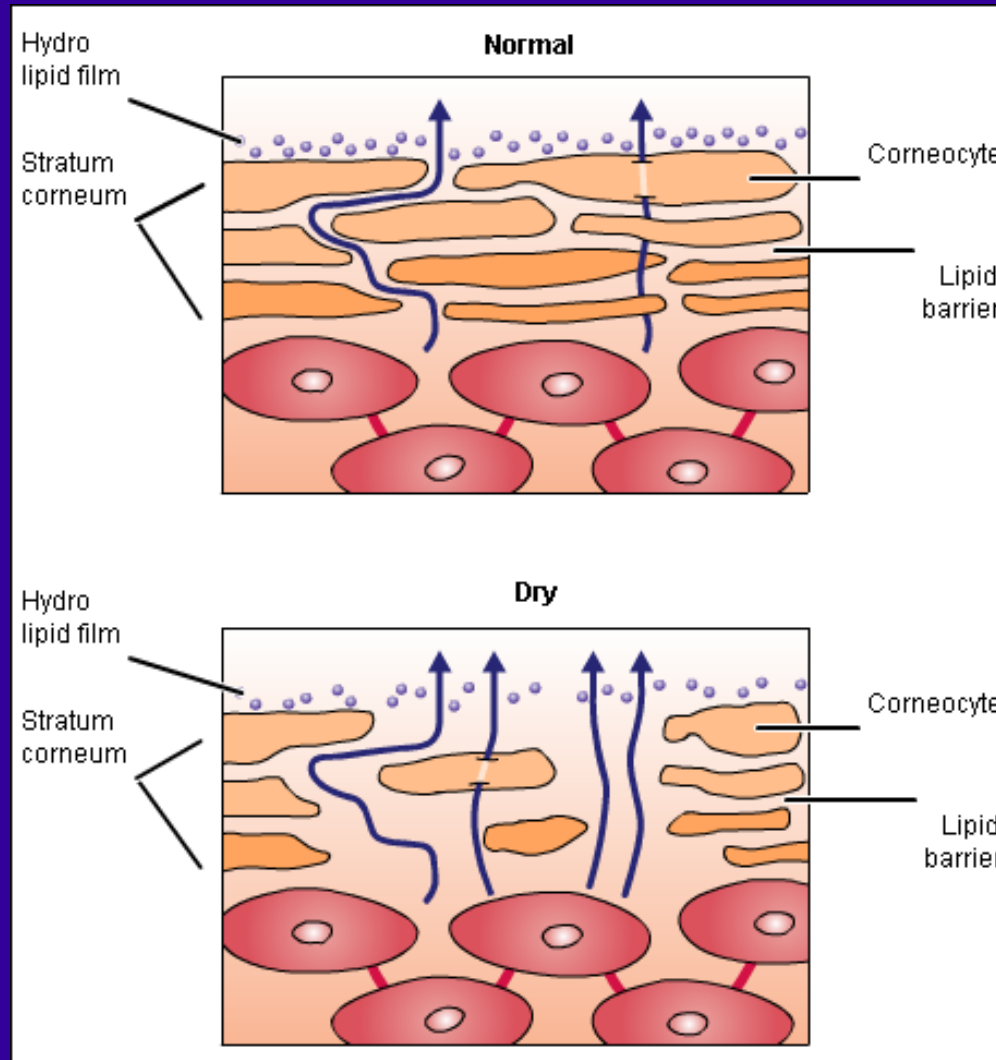
(takes place in lamellar bodies in granular layer of epidermis)



decreased ability to bind water in the skin



skin barrier disturbance



AD and skin barrier

Defective structure and function of skin barrier

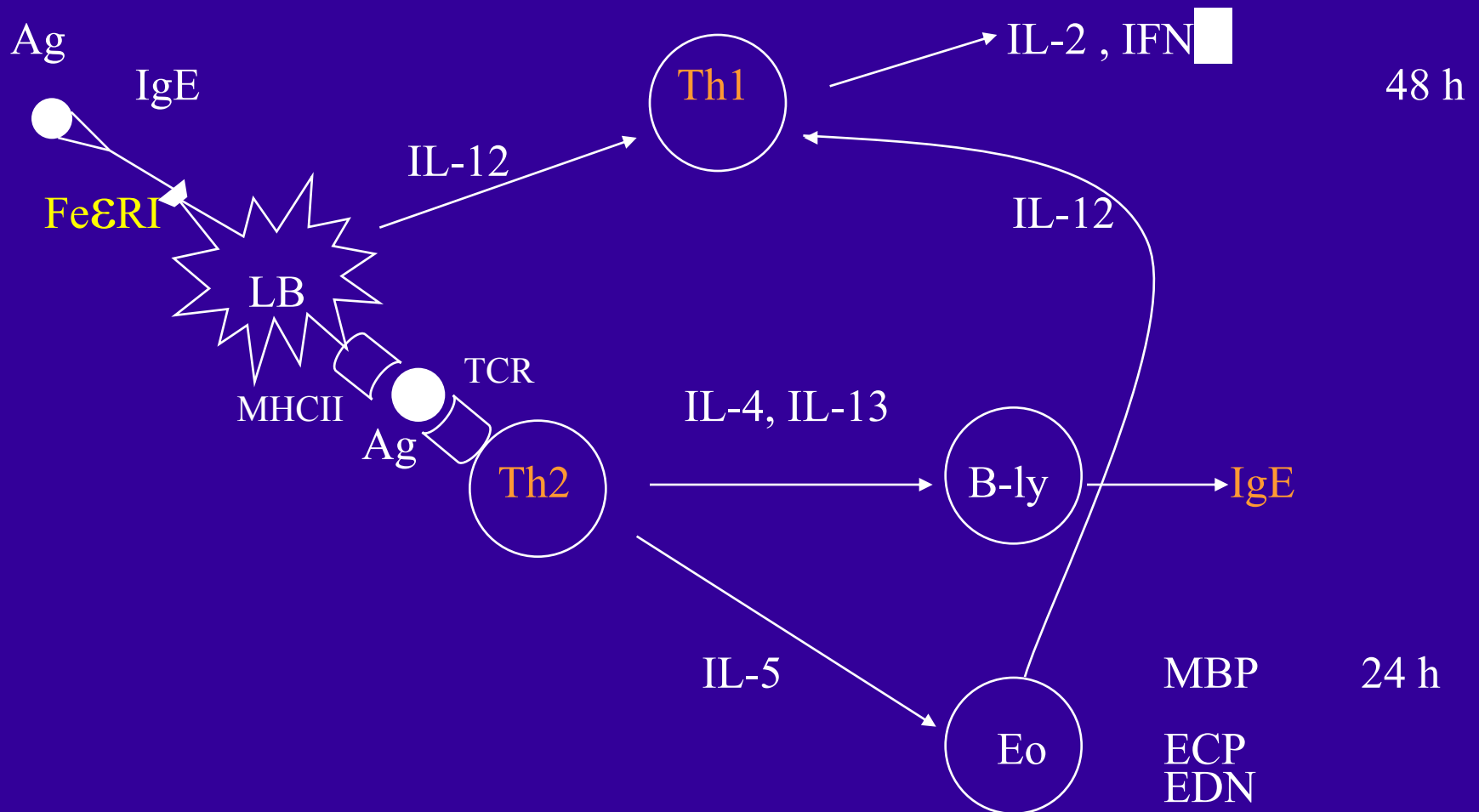
insufficient hydration (TEWL ↑)

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

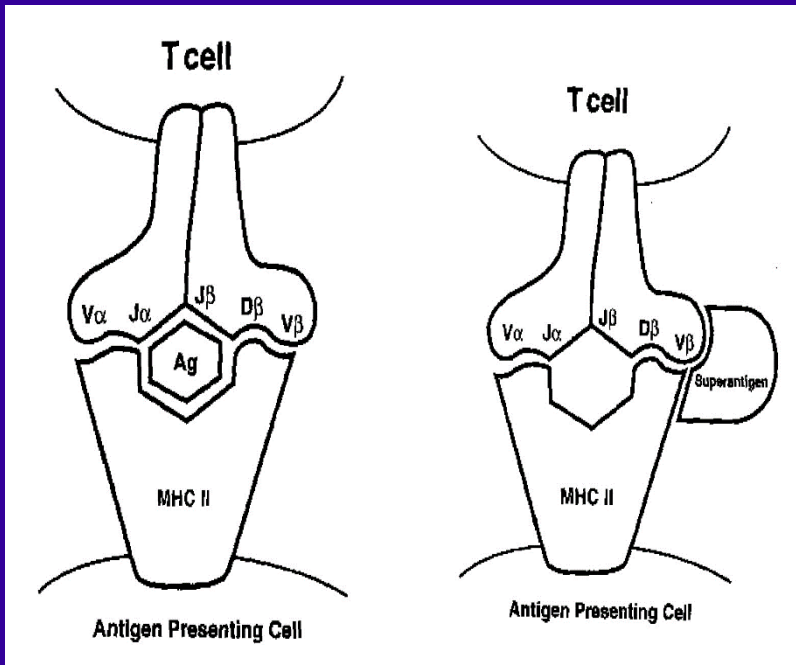
- colonization of AD lesions in 74 - 96% atopic patients, 30 - 56% even on „healthy“ skin

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 γ

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

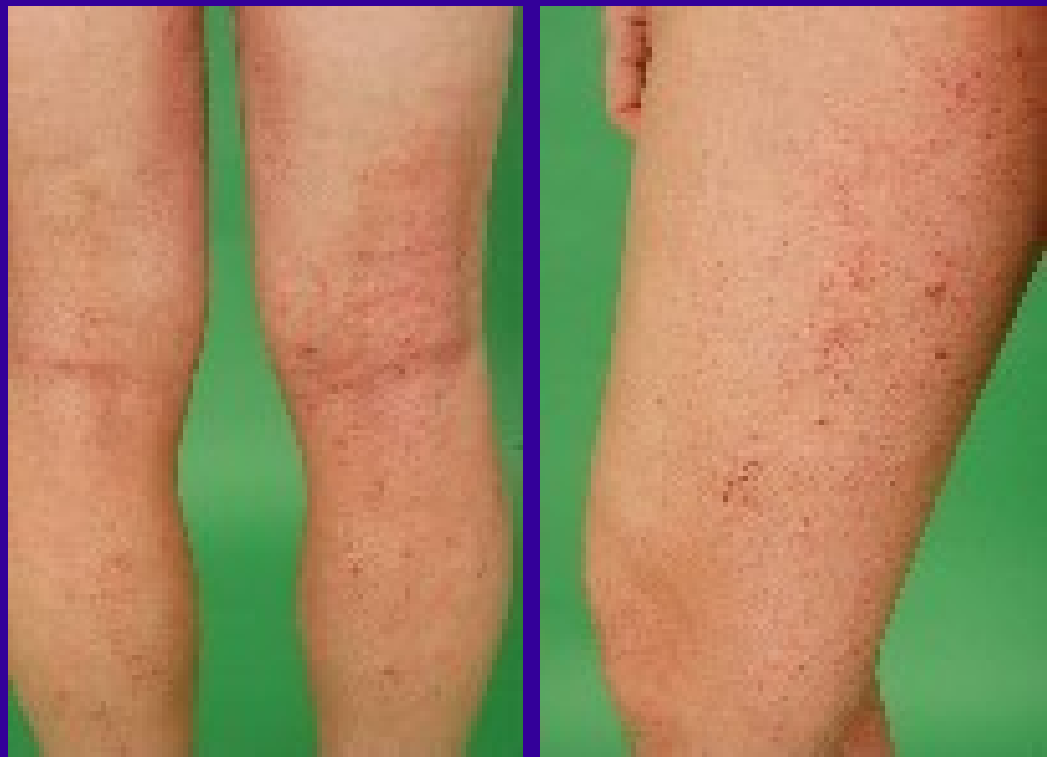
■ most often – flexural eczema

- 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)

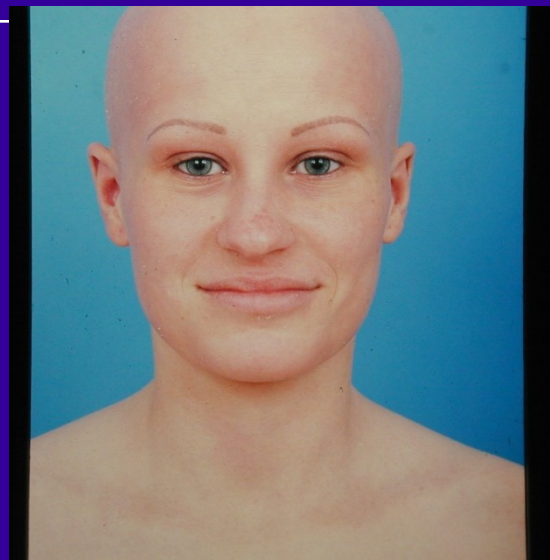
viral – herpetication-HSV, warts, mollusca

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

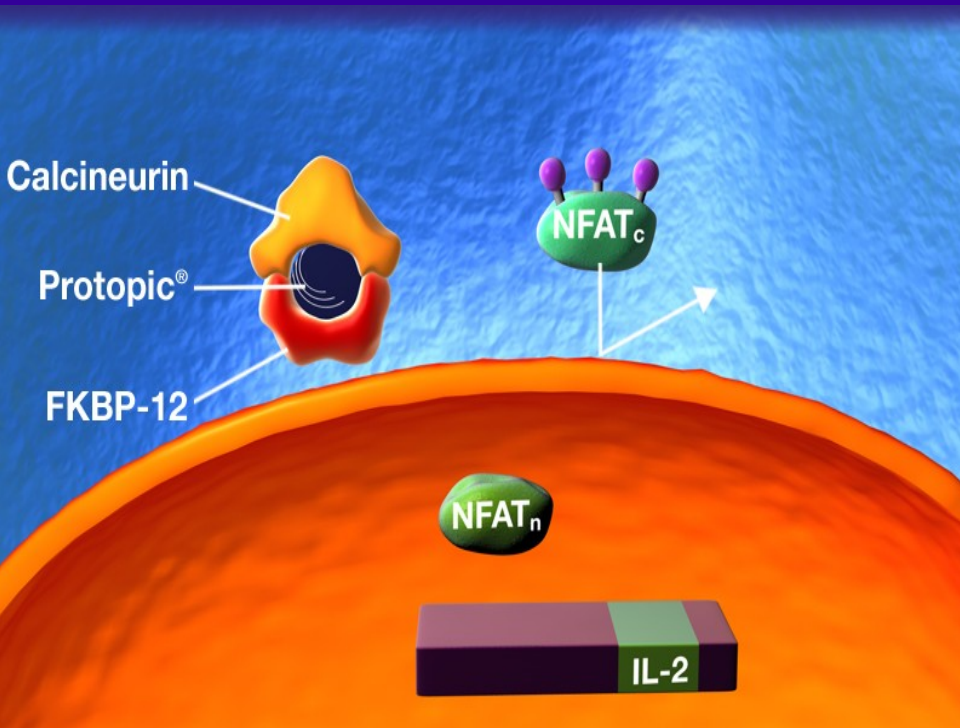
antihistamines during flares



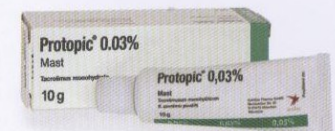
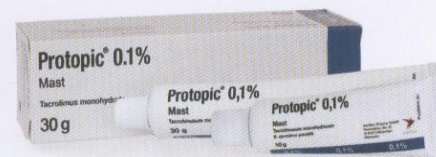
Treatment of AD

- 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 α (?)
 - 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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