

Atopic dermatitis

a special type of eczema

chronic disease, more frequent in childhood

Atopy (trias)

- bronchial asthma
- hay fever
- atopic dermatitis

Atopy (trias)

- bronchial asthma
- hay fever
- atopic dermatitis

Atopy= IgE responsiveness

- raised total serum IgE level
- one or more specific IgE response
- one or more + prick test

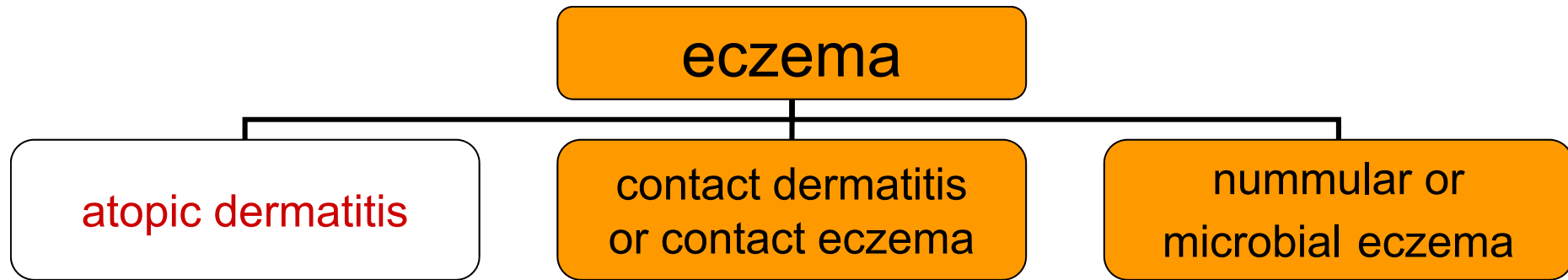
- In AD: 80% can be detected

- AD -- often the first manifestation of atopy
 - AD : can be the only manifestation of atopy
 - but: the physician should be aware of the possibility : future occurrence of hay fever and asthma

Atopic dermatitis

- Synonyms
 - = atopic eczema
 - = endogenous eczema
 - = disseminated neurodermatitis

Atopic dermatitis and other eczemas



other eczemas

Contact dermatitis

Late type IV.

other eczemas

Nickel contact dermatitis

PATCH testing



Patch testing



24, 48, 72, (96) hours

other eczemas

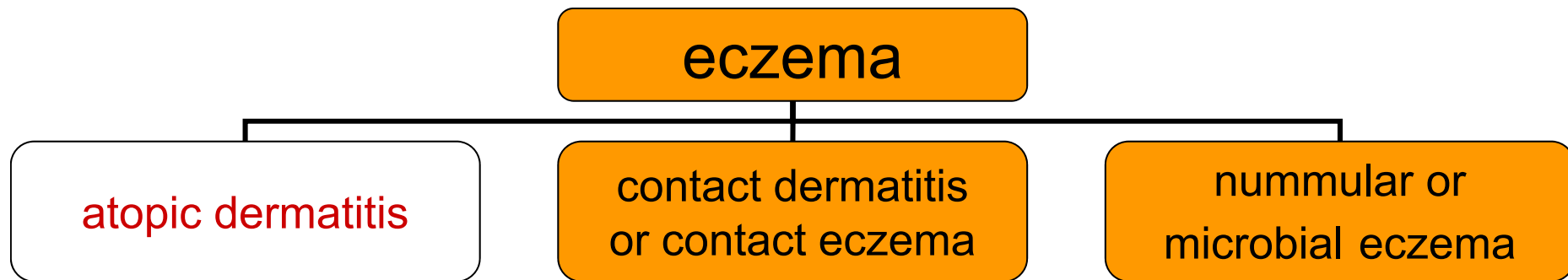
PPD contact dermatitis

other eczemas

Nummular (microbial) eczema

**Immunological skin
reactions to
bacterial or mycotic
antigens-
haematogenic spreading**

Atopic dermatitis and other eczemas



ATOPIC DERMATITIS

In the family common: type I. hypersensitivity (elevated serum IgE)

- allergic rhinitis
- bronchial asthma- croup
- atopic dermatitis
- migraine
- urticaria

Epidemiology

- **atopic dermatitis** prevalence: 2-4 %
raised up to 20 % lifetime prevalence in
the last decade mostly in western
lifestyle

Epidemiology

- 1946: AD prevalence: 5.7 %
 - 1958: 7.3%
 - 1970: 12.2%
 - 1994: 14.3%
 - 2003: 20.0%
-
- (England, Danemark)

ATOPIC DERMATITIS: dynamic of the disease

Most common in childhood

- 60% of patients develop skin symptoms within the first year of life
- 30% of them between ages 1-5

ATOPIC DERMATITIS: dynamic of the disease

Most common in childhood

- 60% of patients develop skin symptoms within the first year of life
- 30% of them between ages 1-5

Spontaneous remission

ATOPIC DERMATITIS: dynamic of the disease

Most common in childhood

- 60% of patients develop skin symptoms within the first year of life
- 30% of them between ages 1-5

Spontaneous remission

Relapse

- 25-40 %: relapse in adolescence
- 30-55%: hand eczema

Atopic dermatitis

- Barrier defect
- Filaggrin mutations

The skin forms an effective **physical** barrier

- between the organism and the environment
- preventing invasion of pathogens
- fending off chemical and physical assaults
- preventing unregulated loss of water and solutes

DAMAGE (dysfunction):



secondary damage of the immunological barrier

skin-barrier homeostasis

The skin forms an effective immunological barrier

- between the organism and the environment
- preventing invasion of pathogens
- fending off chemical and physical assaults

DAMAGE (dysfunction):



secondary damage of the physical barrier



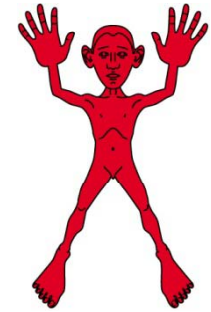
- unregulated loss of water and solutes
- skin-barrier homeostasis

Barrier defects present with uniform clinical symptoms

- the skin is

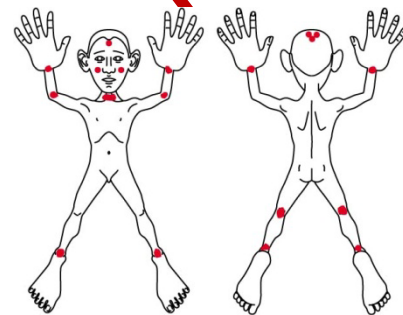
- red
- scaly, dry, itchy

generalized: erythroderma



more symptoms are **localized to surfaces exposed to**

- allergens
- bacteria
- UV
- mechanical irritation- rubbing



localized to mosaic fields of the skin

- Blaschko lines
- other mosaic patterns



Barrier defects present with uniform clinical symptoms + recurrent infections

- the skin is
 - red- erythematous
 - scaly, dry, itchy



- complicated bacterial and viral skin infections

Atopic dermatitis- clinical picture

- children
- **dry skin**
 - irritable - getting easily red
- itchy skin
 - lichenification mostly in skin folds
 - excoriations
- white dermographism

White dermographism

Atopic dermatitis

Atopic dermatitis

Atopic dermatitis

Impetigo

Why?

Atopic
dermatitis

Damaged skin
barriers

Reduced defensin
expression

Atopic dermatitis: juvenile plantar dermatitis

Atopic dermatitis- lichenification

Atopic dermatitis-lichenification

Atopic dermatitis

Predictors of persistency

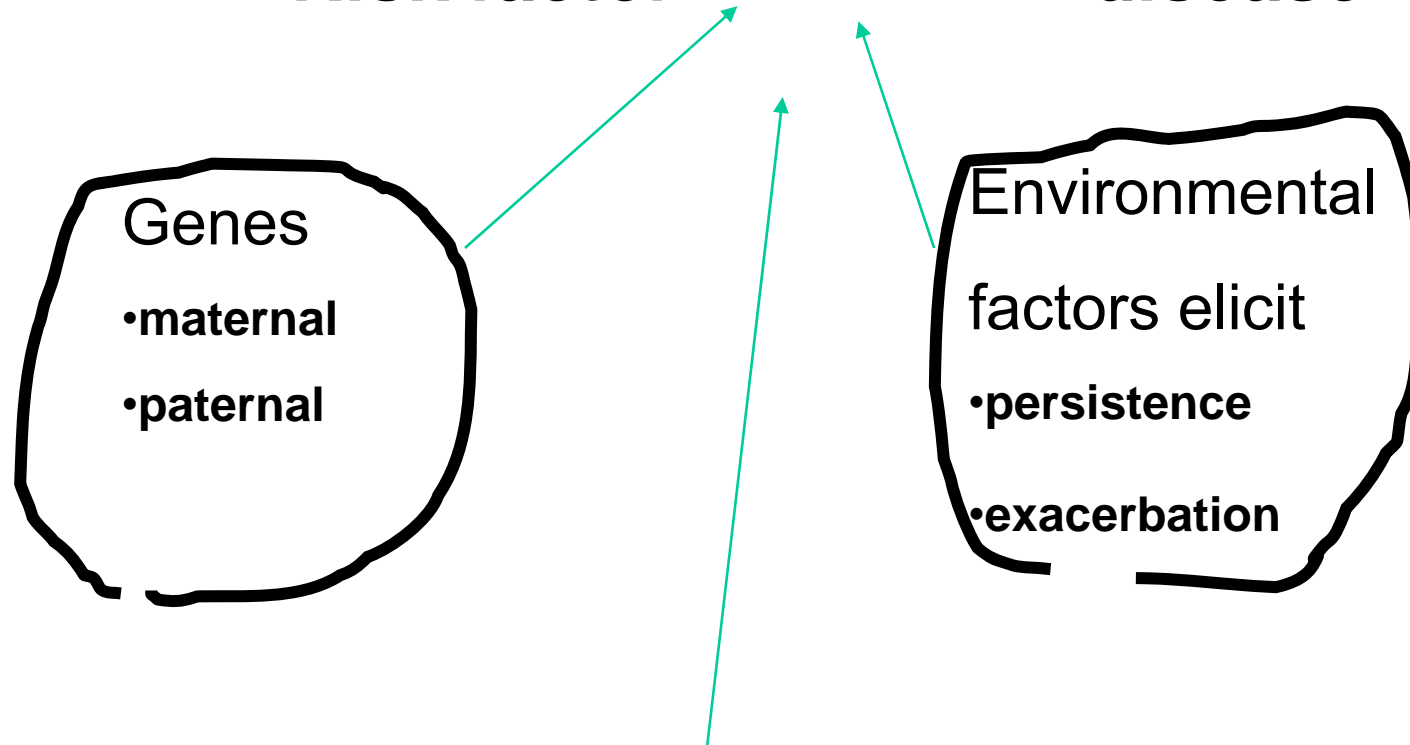
- Positive family
- Early onset
- Widespread disease
- Concomitant hay fever or asthma

Risk factors of atopic dermatitis

- Family history
- Concomitant atopic disease
- Allergens (too early introduction of dietary factors: cow-milk?)
- Small family size
- High social class
- Life-style factors

Atopic dermatitis: multifactorial disease

Risk factor \longrightarrow disease



effect modifier: gender and age

Atopic dermatitis- genetic background

- Dry skin: filaggrin mutations
- Dry and irritable skin: SPINK5 mutations

Sebum and sweat production

- dry skin-get easily irritated and erythematous
- skin is deficient in lipids derived from sebaceous glands
- transepidermal water loss is enhanced
- increased sweating to acetylcholin
- reduced or missing barrier function

Pruritus

- dry and irritable skin
- no primary elementary skin lesions only secondary ones due to pruritus
- lichenification (neuroderma), excoriations
- secondary skin infections

Impetigo

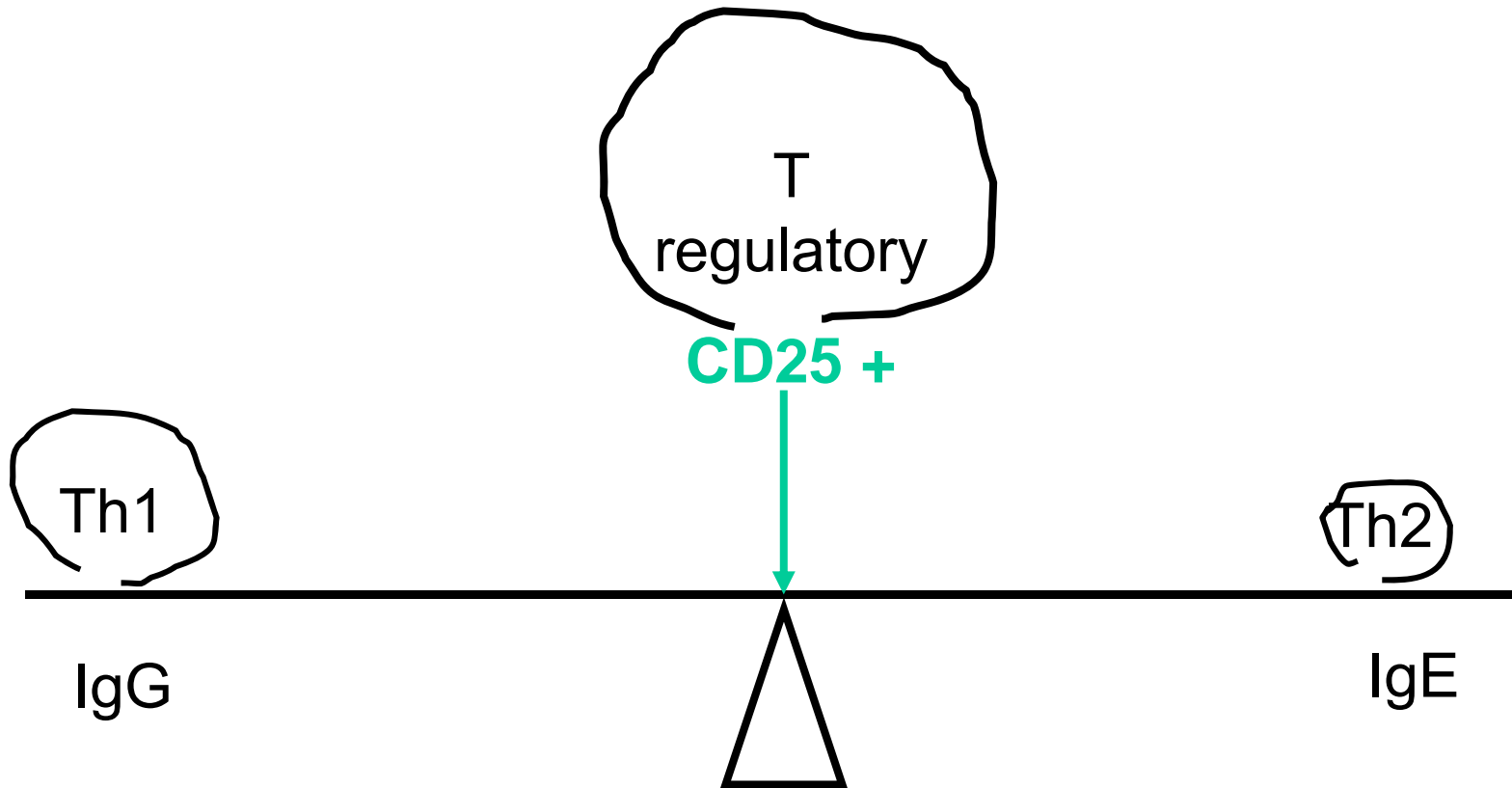
► Atopic dermatitis?

- medical history for 3 atopies
- family history for 3 atopies
- Clinical examination of skin:
▼

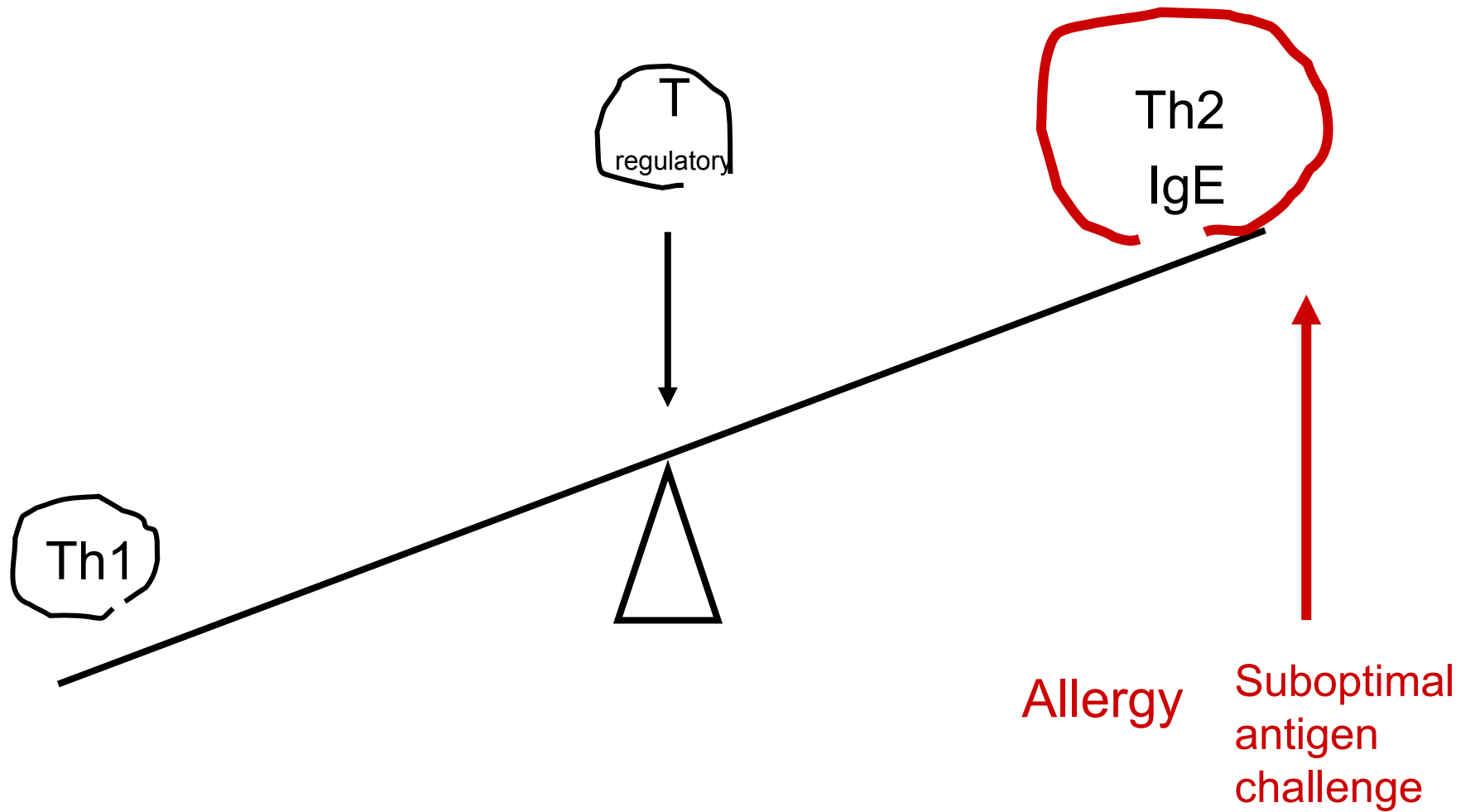
Atopic dermatitis - white dermographism

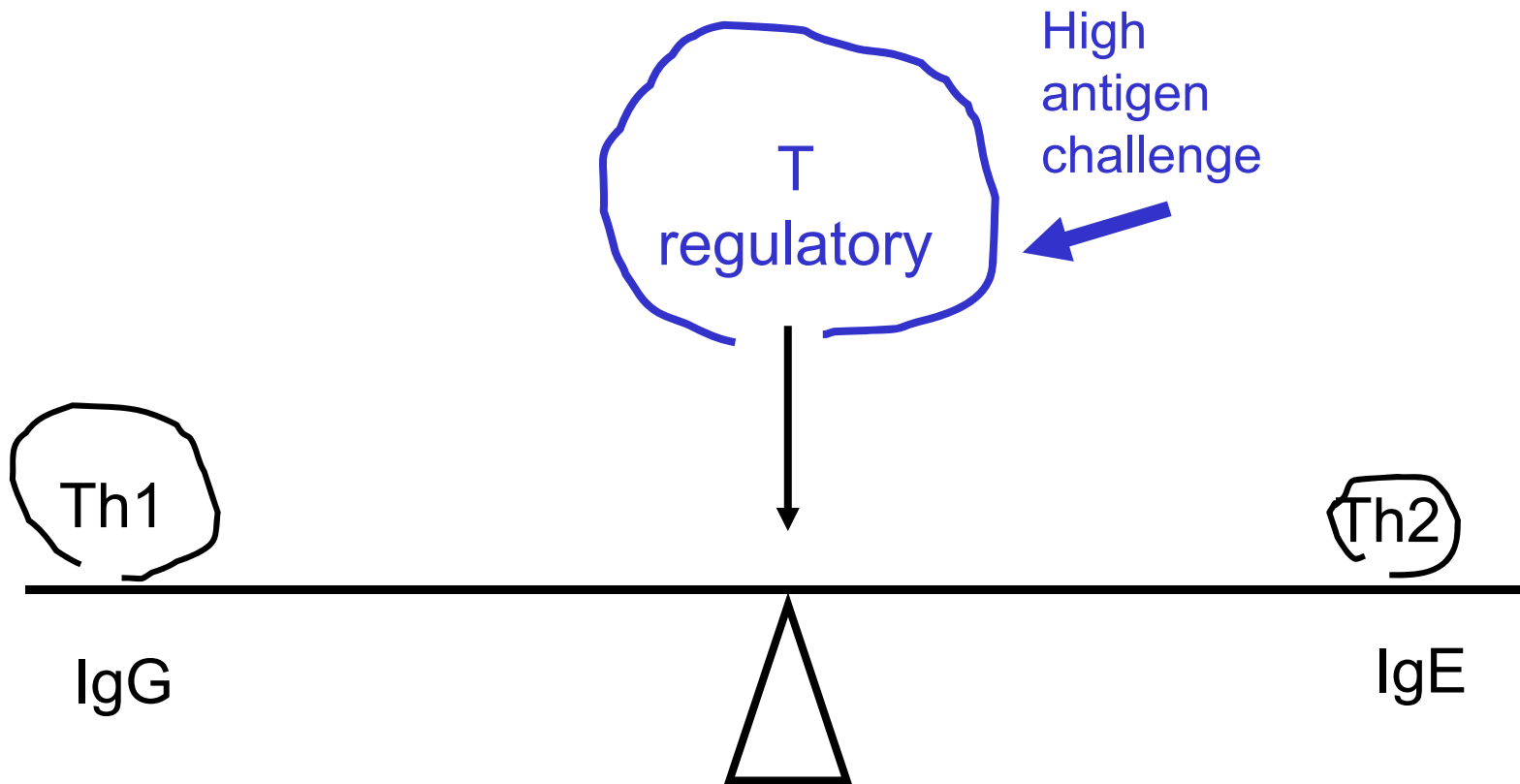
Pathology of AD

- Th1 / Th2 ratio changed ↓
- reduced Th1
- relative higher Th2



Healthy





Tolerance

Etiology

Role of **IgE and exogenous allergens**

- elevated serum IgE level
- sometimes parallel skin and airway symptoms

Etiology

Role of **IgE** and **exogenous allergens**

- elevated serum IgE level
- sometimes parallel skin and airway symptoms
- atopy patch test

Atopy - PATCH test with pollens and external allergens



Etiology

IgE sensitivity: tests: often no clinical relevance

- RAST (radio-allergo-sorbent-test) IgE against common allergens: pollens, molds, foods, bacteria
- positive skin test to one or more antigens

Atopic dermatitis

- Genetics (family)
- Food allergy may be responsible for a AD flare in up to a third of the cases, but the presence of an allergy should be demonstrated before the prescription of an elimination diet
- Extrinsic factors- air pollution

Atopic dermatitis and skin infections

- Staphylococcus aureus colonisation
- 75-90% of the patients have Staphylococcus aureus on lichenified skin areas

Atopic dermatitis and skin infections: the role of bacterial superantigens

- 5%- of the *Staphylococcus aureus* present in normal population produces superantigens
- 37-57 % of the *Staphylococcus aureus* in atopic dermatitis patients produces superantigen

AD and bacterial superantigens

- Staphylococcus enterotoxin A (SEA)
- Staphylococcus enterotoxin B (SEB)
- Toxic shock syndrome toxin 1 (TSST 1)

AD and viral infections

- Herpes simplex – eczema herpeticum
- Molluscum contagiosum
- HPV

Etiology: multifactorial inheritance- interactions

- **Genes** implicating a defect in the outer **barrier** (e.g. filaggrin mutations)
- Clusters of **genes** with general effects on dermal **inflammation** and **immunity**
- The atopic component (**IgE**) of AD **may be secondary**

Therapy

- regular treatment of the skin by ointments and emollients
- topical steroids against inflammation
- topical anti-infectious therapy but not antibiotics for superinfections and colonization of the skin by staphylococcus aureus

information to the parents on the chronic course with recurrent flares

Treatment

Topical steroid ointments

- use only to the inflammatory skin
- face: mild local steroid only with
- reduce steroids as much as possible
 - side effects: local atrophy, teleangiectasias, superinfection

Therapy

- immuno-modulatory macrolides (tacrolimus and pimecrolimus) represent a new alternative to topical steroids
- their effects on the long-term are unknown

Antihistamines

H1 –receptor antagonist

- **Aminoalkyl ethers**
 - Clemastin - **Tavegyl**
- **Substituted alkylamins**
 - Dimethinden – **Fenistil**
- **Phenothiasin-derivates**
 - Prometasin – **Pipolphen**
 - Thiethylperasin – **Torecan**
 - Mequitazin – **Primalan**
- **Piperazin derivates**
 - Oxatomid – **Tinset** (*deliberation and receptor blocking of H₁, H₂, SRSA*)

not for acute events alone, for prevention

- **Cetirizin** – **Zyrtec**- not under pregnancy
slightly anticholinergic, quick

Antihistamines

H1 –receptor antagonist

- Aminoalkyl ethers
 - Clemastin - *Tavegyl*
- Substituted alkylamines
 - Dimethinden – *Fenistil*

- **Cetirizin** – *Zyrtec*- not under pregnancy
slightly anticholinergic, quick

Antihistamines

Other H1 –receptor antagonist

- Loratidine – **Claritine** no sedative effects
- Ketotifen – **Zaditen** (*slow, long lasting, asthma*)
- Acrivastin – **Semprex** (*short, quick*)
- Cyproheptadine – **Peritol** (+ serotonin antagonist, anticholinerg)(*weight, appetite*↑)
- Terfenadin - *Teldane* **CAVE: macrolid AB, ketakonazol, intrakonasol**

Prolongation of the Q-T interval

- Hidroksizinum hydrochloricum – **Atarax** + anticholinerg
- Chlorphenamine- chlorpheniramin - + anticholinerg
- Diphenhydramine
- Astemisol- long acting - chronic urticaria

Antihistamines

Other H1 –receptor antagonist

- Loratidine – **Claritine** no sedative effects
- Ketotifen – **Zaditen** (*slow, long lasting, asthma*)

Antihistamins

- H2 receptor antagonist
 - Cimetidine

Please remember 3 H1 receptor antagonists!!

Treatment

Topical emollients = fatty ointments as basis therapy

- It allows to reduce topical steroids in quantity and potency
- use oil or emollients both in and after bath (avoid soap, use liquid paraffin-or like oils)
- It should be free of preservatives and fragrances - **avoid sensibilisation**
- addition of urea can increase the efficiency

Treatment

Antibiotics/ skin infections

- do not use locally- induce resistance and allergy
- fucidin, mupirocin,

Antihistamines

- reduce itch, prevent excoriations
- change

Treatment

- **Bandages and wraps: prevent scratching**
- Wet or dry wraps
- Weak antiseptics, mild steroid
- Fatty ointments

Treatment

- Sun or UV
 - UVB irradiation (290-320 nm)
 - Narrow band: UVB 311 nm
 - Salt UVB therapy
 - Balneo-phototherapy
 - UVA monotherapy (340-400)
- Climatotherapy

Treatment

- Dietary restriction – exclusion of specific foods
 - rarely effective
 - no clinical correlation with specific IgE level
 - trials are worth of consideration for 4-6 weeks
- Dietary restriction – exclusion of histamin liberators from the food
 - useful in severe cases

Adult form of AD

- from childhood on AD
- de novo AD: suspect for lymphomas or other malignancies