Skin conditions in wrestling – how to prevent

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Disclosure

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- I have the Relationships with commercial interests:
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Objectives

- Normal and impaired skin barrier
- Atopic dermatitis – model for understanding barrier
- Skin diseases in wrestling
- Treatments
- Prevention techniques in skin infections

Skin barrier

Danger model:
”The basic function of immune system is not to distinct between self and non-self, but to recognize danger”

Polly Matzinger, PhD, Immunologist, NIH

In order to avoid or prevent a loss on the mat you need a good defense –

The same is true for skin (an active defense)

Skin barrier functions

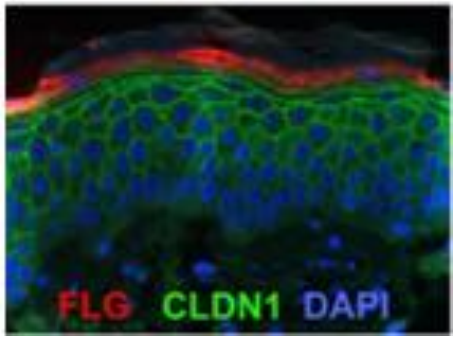
Physicochemical barrier and immunological barrier – in close morphological and functional connection

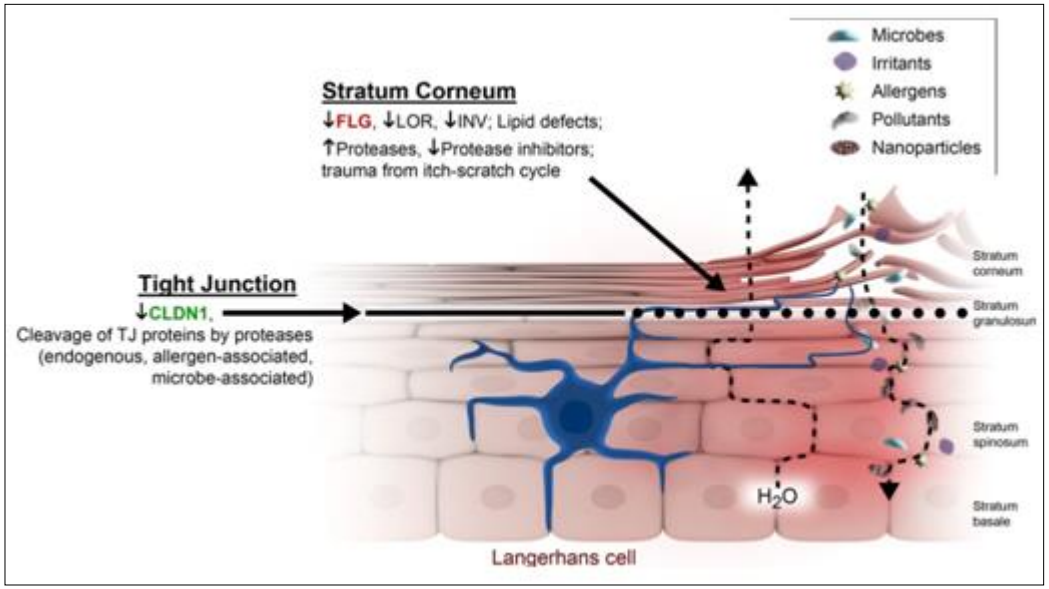
Physicochemical barrier

- Stratum corneum: corneocytes
- Stratum granulosum: keratinocytes
- Cornified envelop , structural proteins (filaggrin)
- Lipid layer, proteases, protease inhibitors, defensins
- Tight junctions, corneodesmosomes

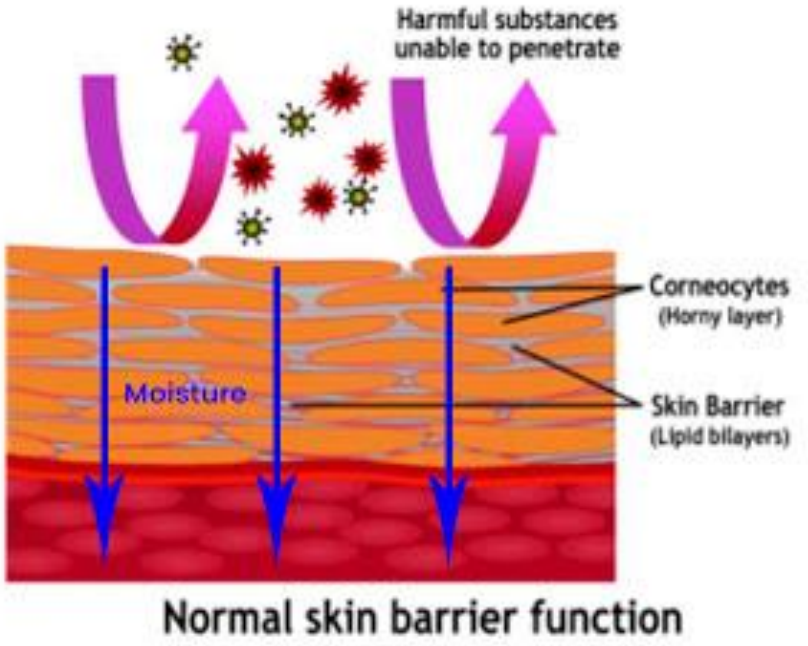
Immunological barrier (SIS)

- Epidermis, dermis
- Keratinocytes, dendritic cells, T cells
- Defensins, cytokines, chemokines





Physicochemical barrier



Normal skin barrier function

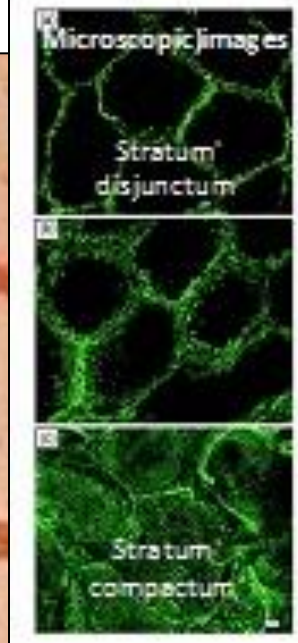
Genetics

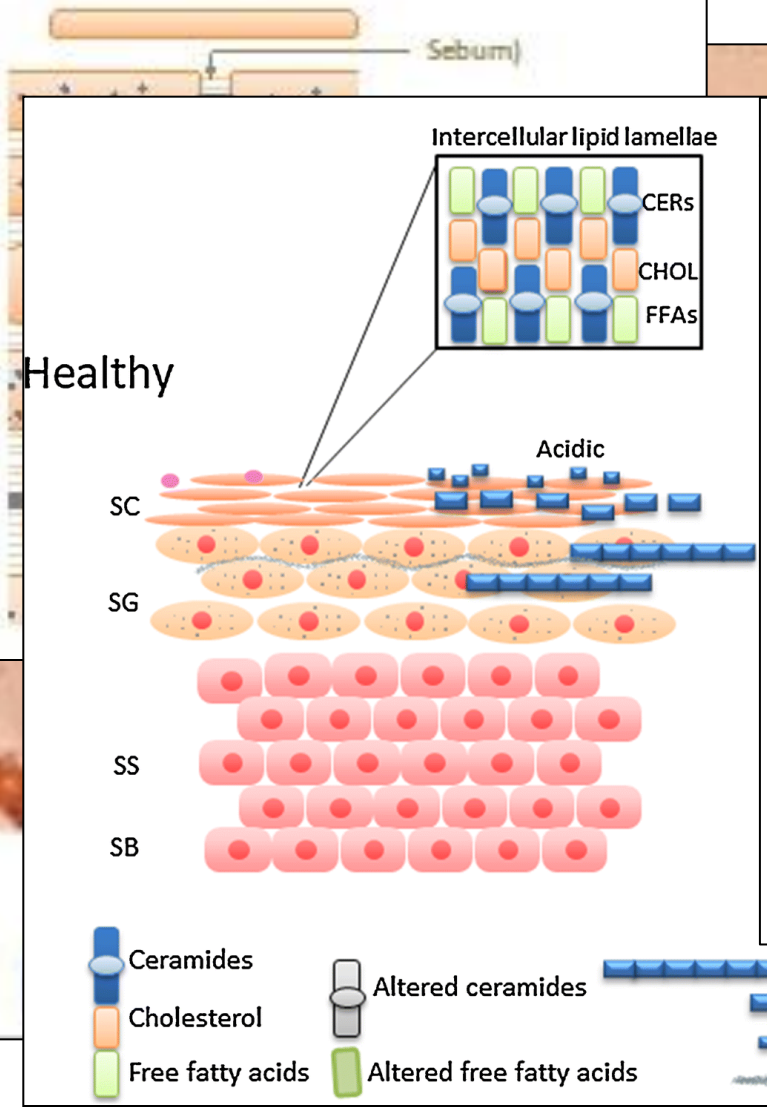
Environmental factors

- microbes (viruses, fungi, bacteria, parasites)
- physical factors (e.g. UV, humidity, scratching)
- chemicals (e.g. irritants)
- biological factors (allergens)

http://crmsnchrysnths.blogspot.com/2017/09/what-is-skin-barrier.html

Elements of intact physicochemical barrier





Barrier:

Cells: corneocytes

Intercellular junctions: corneodesmosomes

Intercellular matrix: lipids, enzymes

https://www.sheffield.ac.uk/iicd/research/rr/dermatology/skinbarrierfacility

Five major groups of skin barrier genes

Enzymes having role in desquamation and cross-linking

KLK7 KLK5 KLK14

TGM1 TGM3 TGM5

Tight junction components

CLDN1

CLDN16

CLDN23

OCLN

Barrier structure components

Keratins:

KRT1

KRT10

KRT6

KRT16

KRT17

KRT79

Other molecules:

FLG

LOR

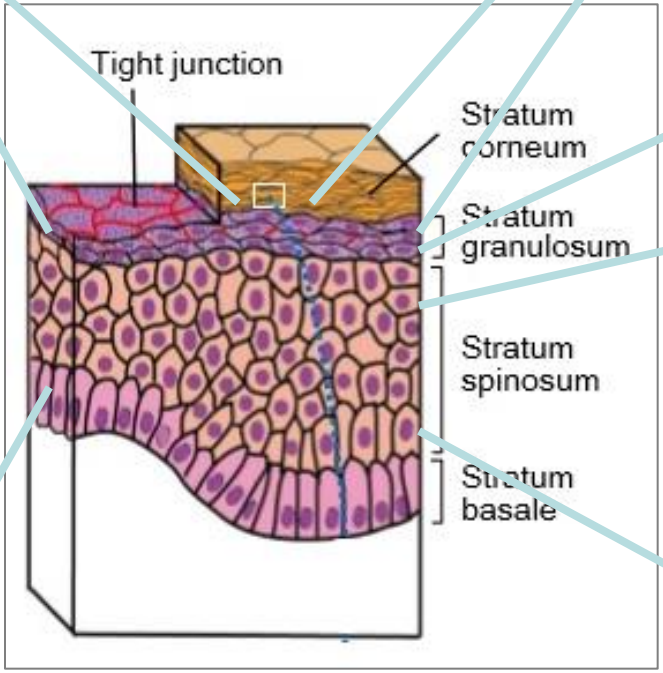
PPL

SPRR1A

SPRR2A

LCE1F

LCE1D



Corneodesmosome components

CDH1 DSG1

DSC1 PKP1

CDSN

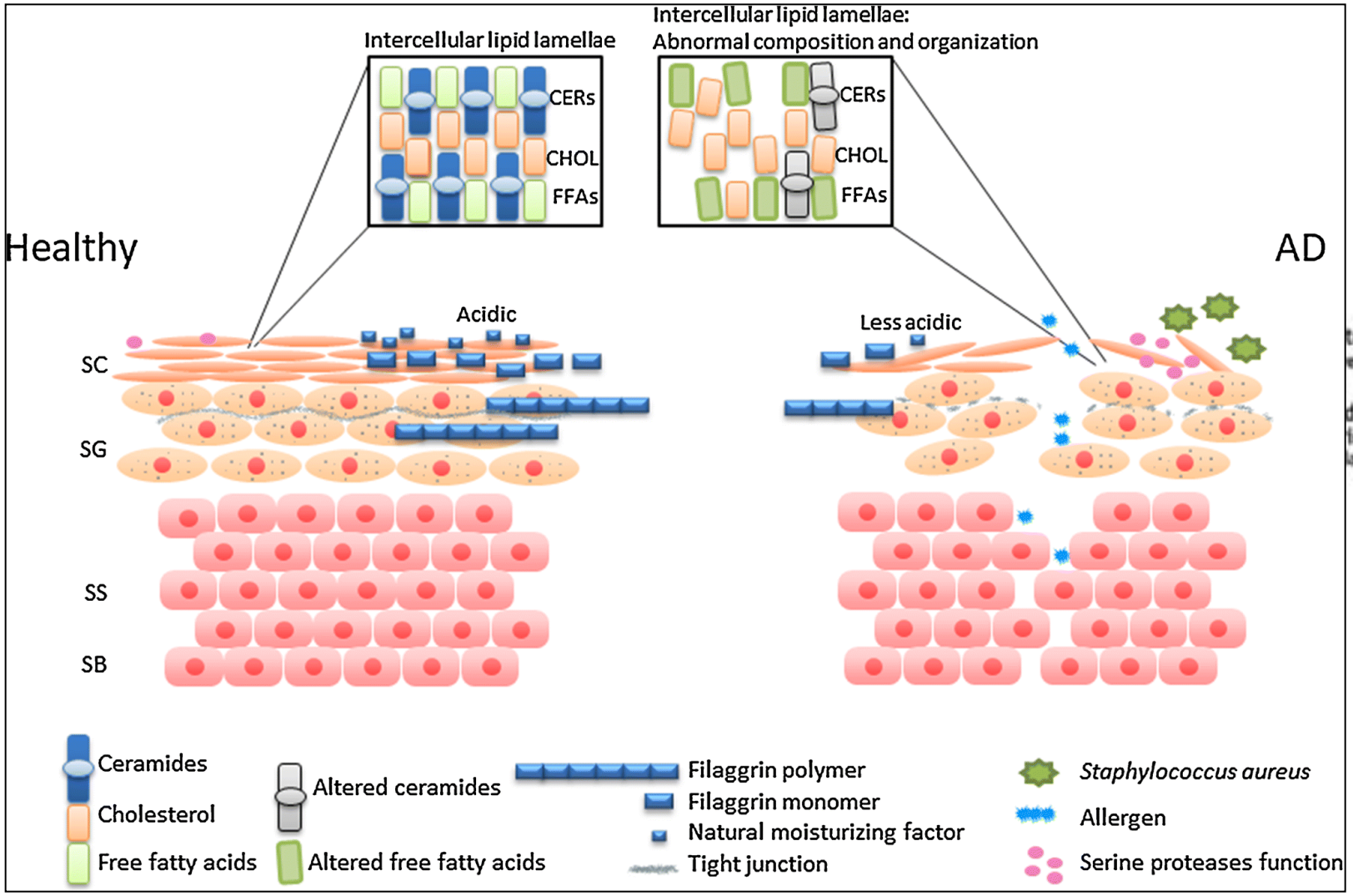
Antimicrobial peptides (AMPs)

S100A7 S100A8

S100A9 DEFB4B

LCN2 TSLP

Impaired physicochemical barrier

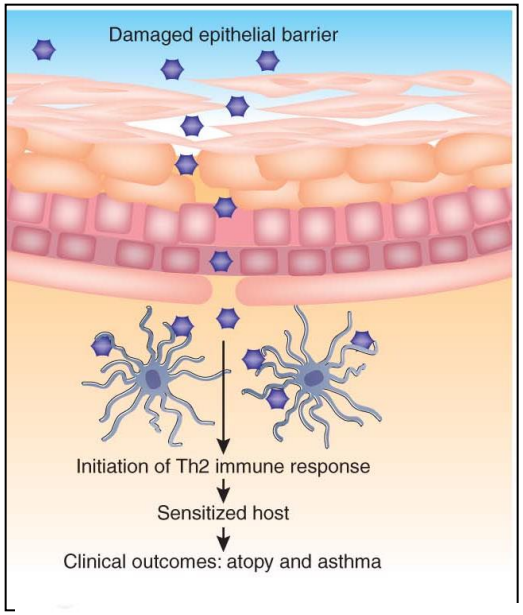


http://crmsnchrysnths.blogspot.com/2017/09/what-is-skin-barrier.html

Current Dermatology Reports December 2018, Volume 7, Issue 4, pp 209–220 Skin Barrier Function and Atopic Dermatitis

Consequences of impaired barrier

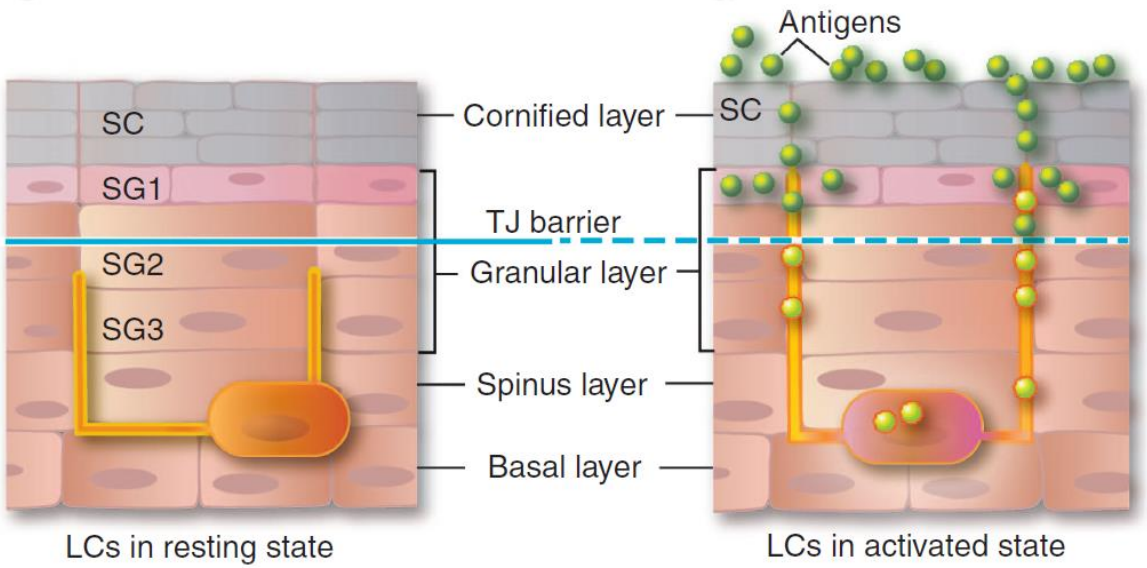
Increased penetration of allergens, irritants, pathogens



Increased transepidermal waterloss (TEWL)

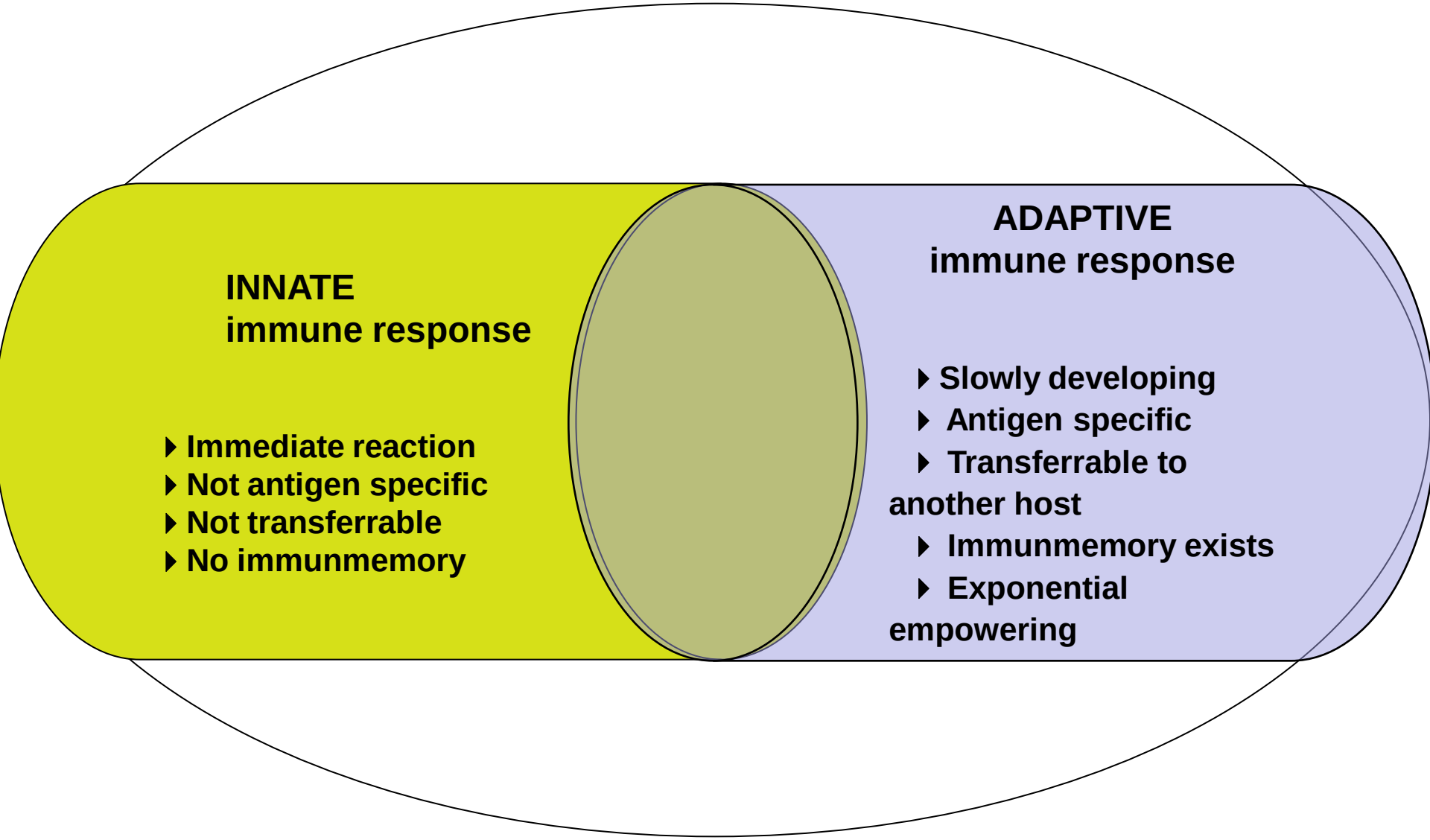
+ increased KC and LC activity that leads to increased immunbarrier activity

Inflammation



Hudson TJ Nature Genetics 2006

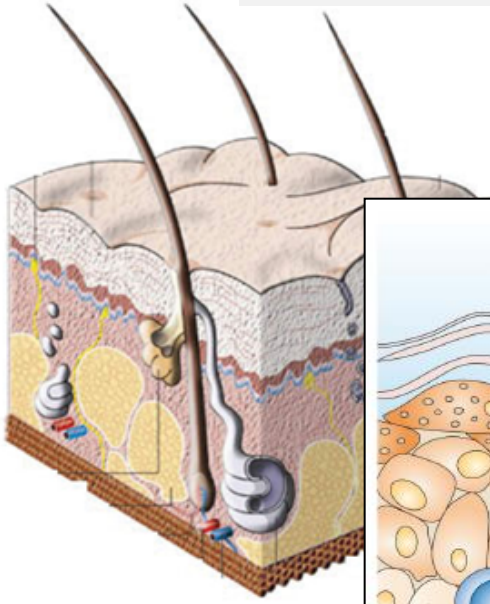
Immunological barrier



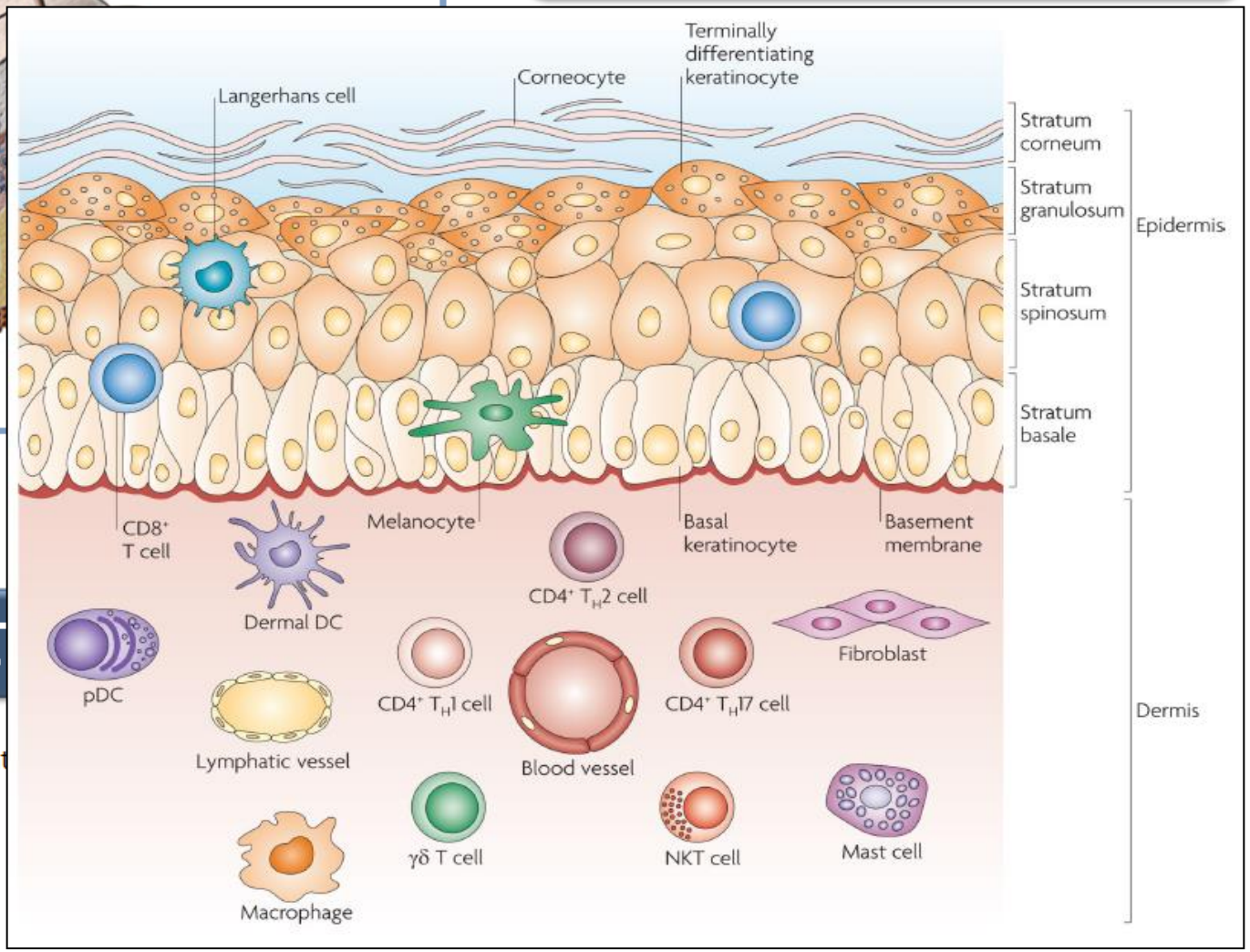
Major elements of cutaneous immune responses

	Innate immune response	Adaptive immune response
Cellular elements	Phagocytes (macrophages, granulocytes) Dendritic cells NK cells Keratinocytes Endothelial cells	T lymphocytes B lymphocytes
Humoral elements	Complement system Cytokines chemokines C-reactive protein Mannose-binding lectin Lipopolisacharide-binding protein Antimicrobial peptids etc.	Antibodies Interleukins (cytokines)

Skin as an immune organ



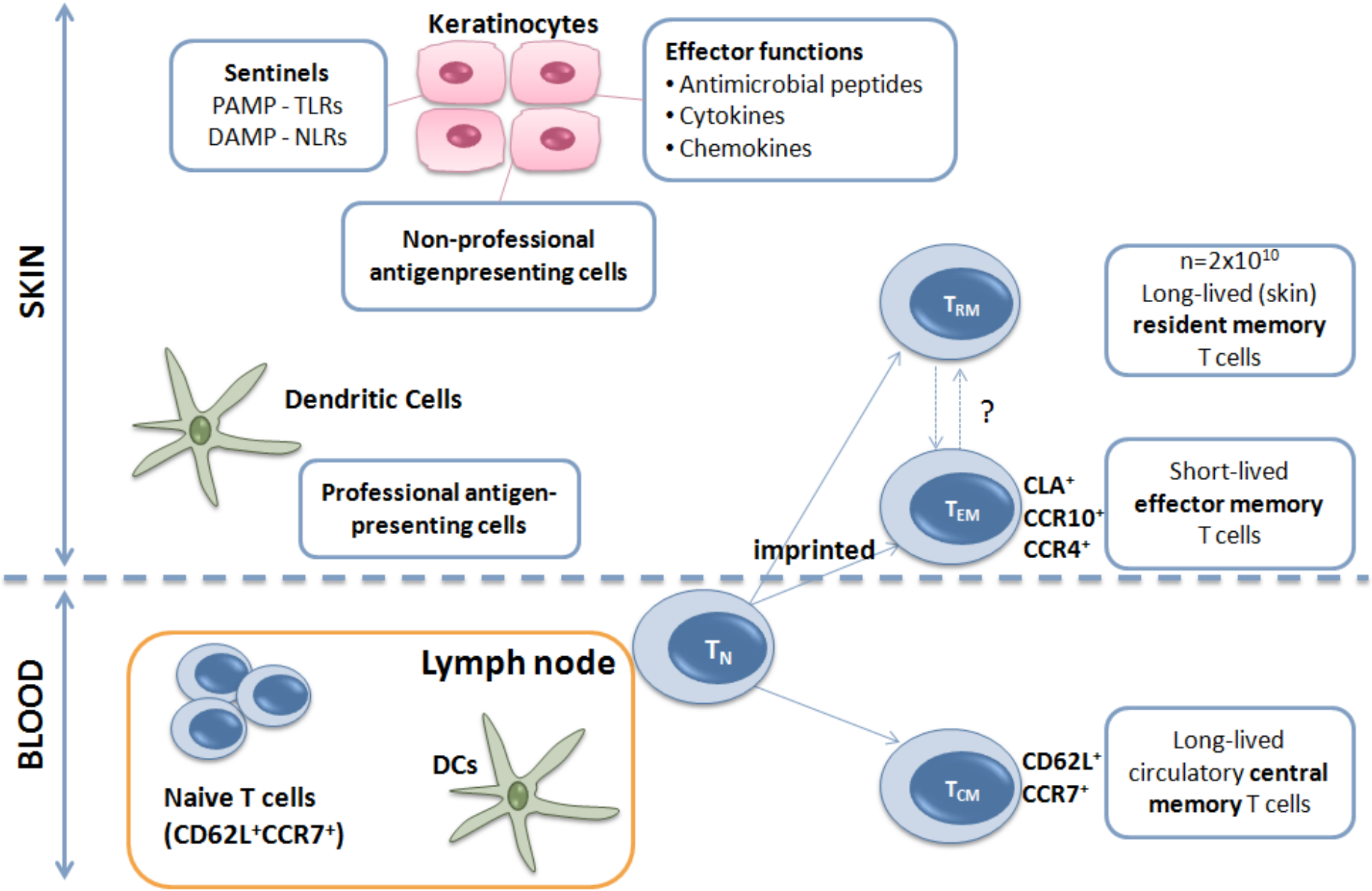
Integrated role of skin resident cells



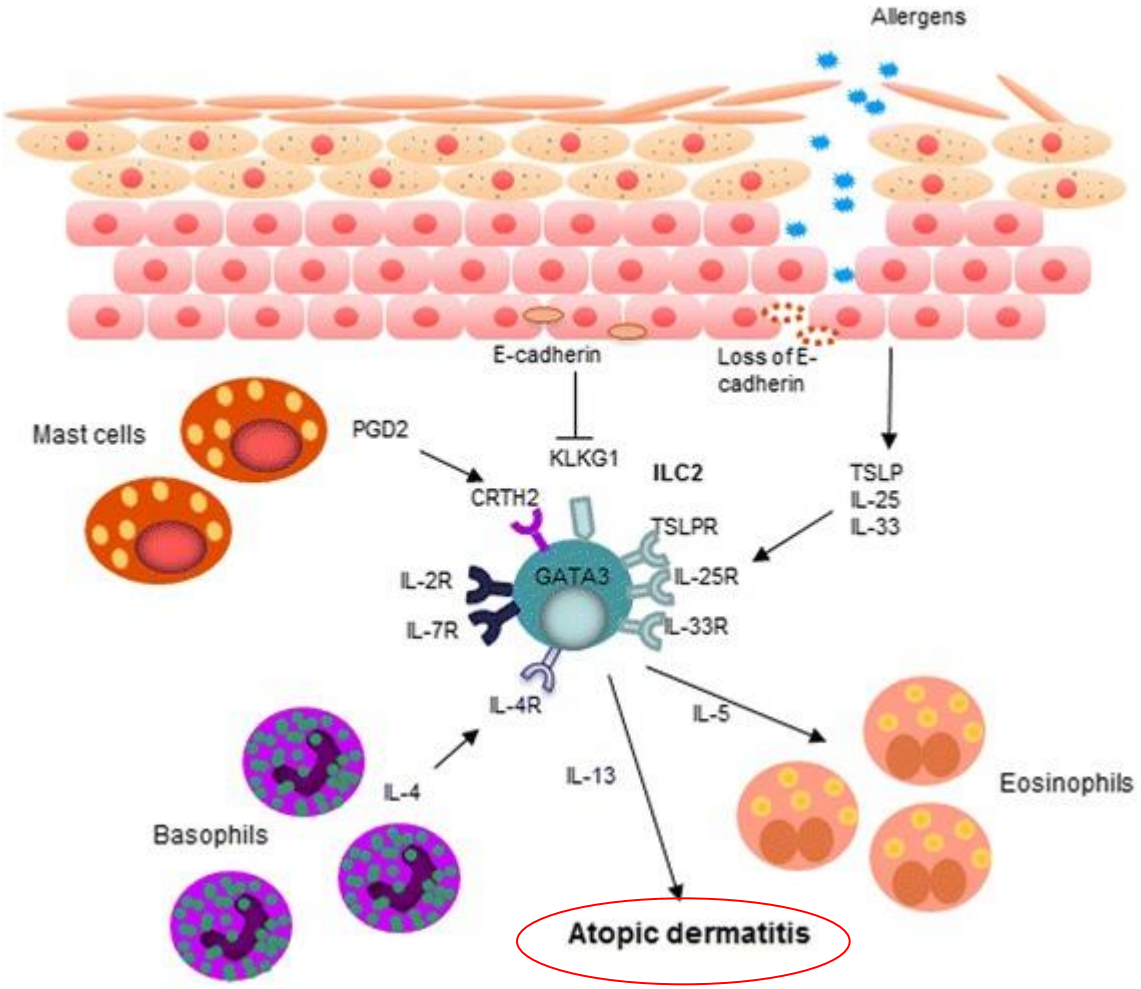
SAL

Streilein JW.
J Invest Dermat

Resident skin immune cells



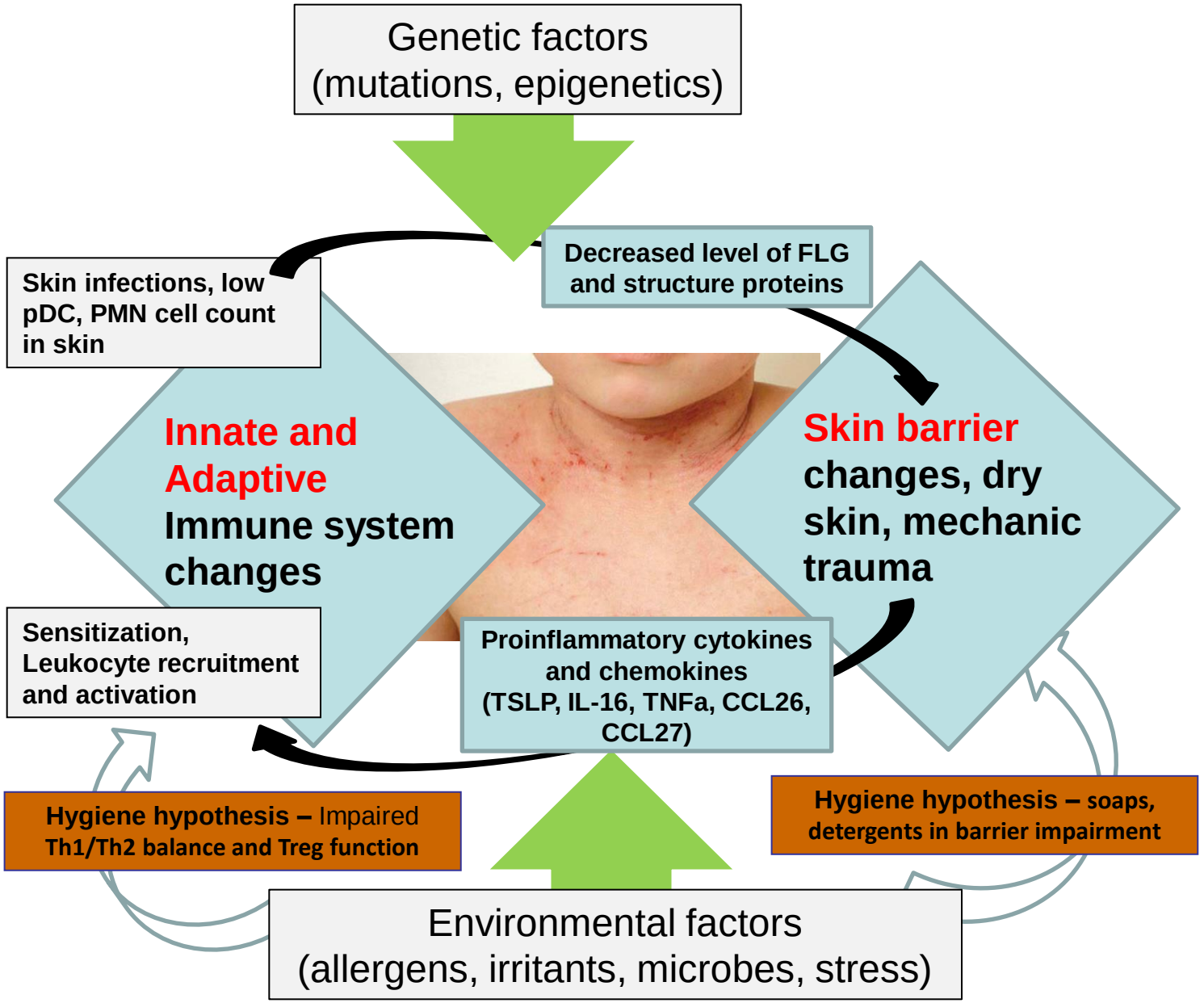
Impaired immunological barrier



Good model of impaired physicochemical and immunological barrier

Inflamm Regen. 2017 Jun 5;37:14. doi: 10.1186/s41232-017-0044-7.

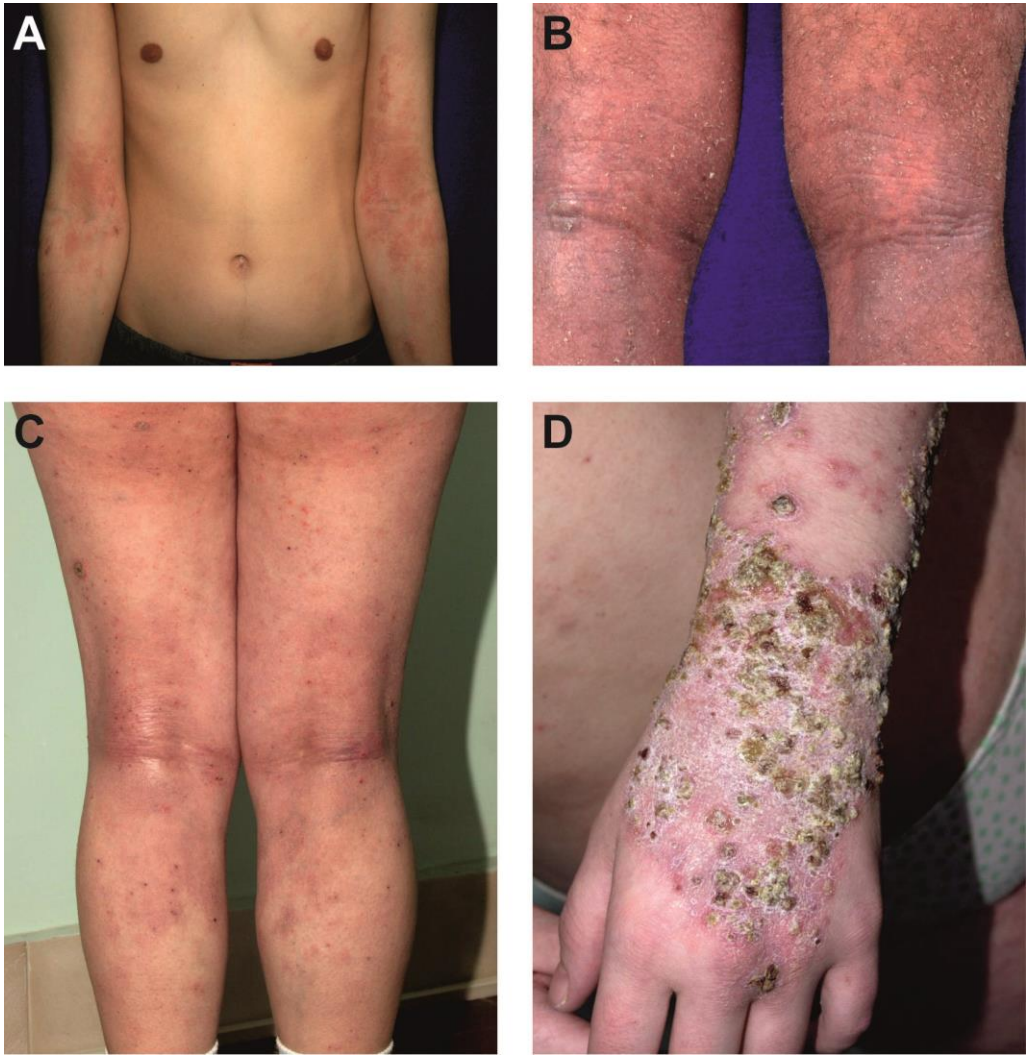
Pathogenesis of AD



Close and **not independent connection** between the abnormalities of epidermal barrier function and immunological mechanisms, with genetic and environmental changes in the background

Atopic Dermatitis

- Chronic, non-contagious inflammatory skin disease.
- Dry skin, pruritus, possible superinfections (>90% S. aureus colonization).
- Prevalence in Europe in children 15-25%, in adults 2-10%, continuously increasing.



Skin barrier prevention – levels of prevention

- Primary prevention: healthy person
- Secondary prevention: atopic, symptom free (+atopy march)
- Tertiary prevention: atopic, chronic patient

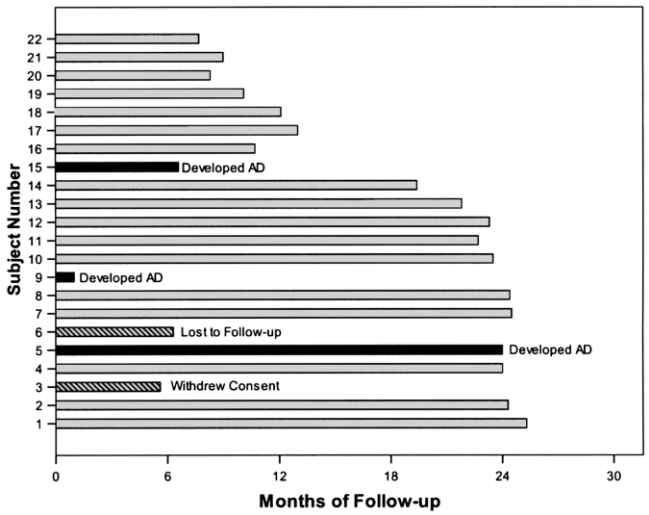
Prevention techniques – possibilities

- Avoidance mechanisms (specific and non-specific triggers, provocation factors) (?)
- Diet, probiotics
- Continuous **emollient use**

Simpson et al JACI 2014

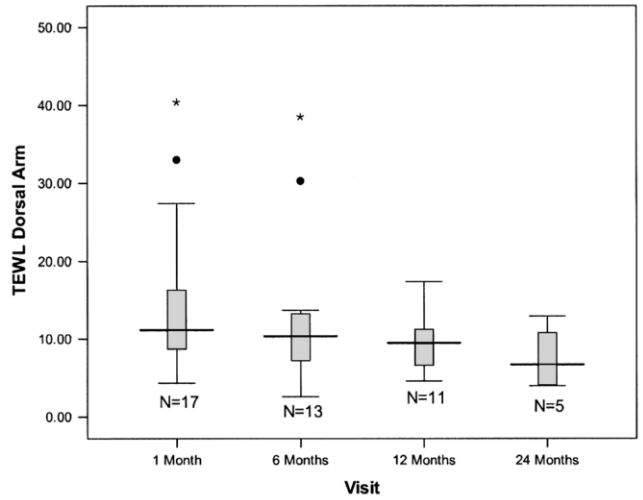
The First: open-label, prospective study

High AD-risk children: 30-50% development of AD in first 2 years



22 high AD-risk newborns
Emollient therapy
From week 1 for 2 years (2006-2008)
Daily at least 1x (QD)

15 % developed disease



Transepidermal waterloss (TEWL) decreased

Simpson E et al. JAAD 2010

The First: Randomized, controlled study

124 high AD-risk newborns (108 finished study)
Emollient therapy (oil, cream, ointment) from M1 for 6 months (2010-2011)
Daily at least 1x
In control group no emollient

Emollient use **well tolerated** by patients

AD cumulative **incidence (50% drop):** emollient group 22%, control group 44%

Therapy **adherence** high

TABLE III. Additional sensitivity analyses

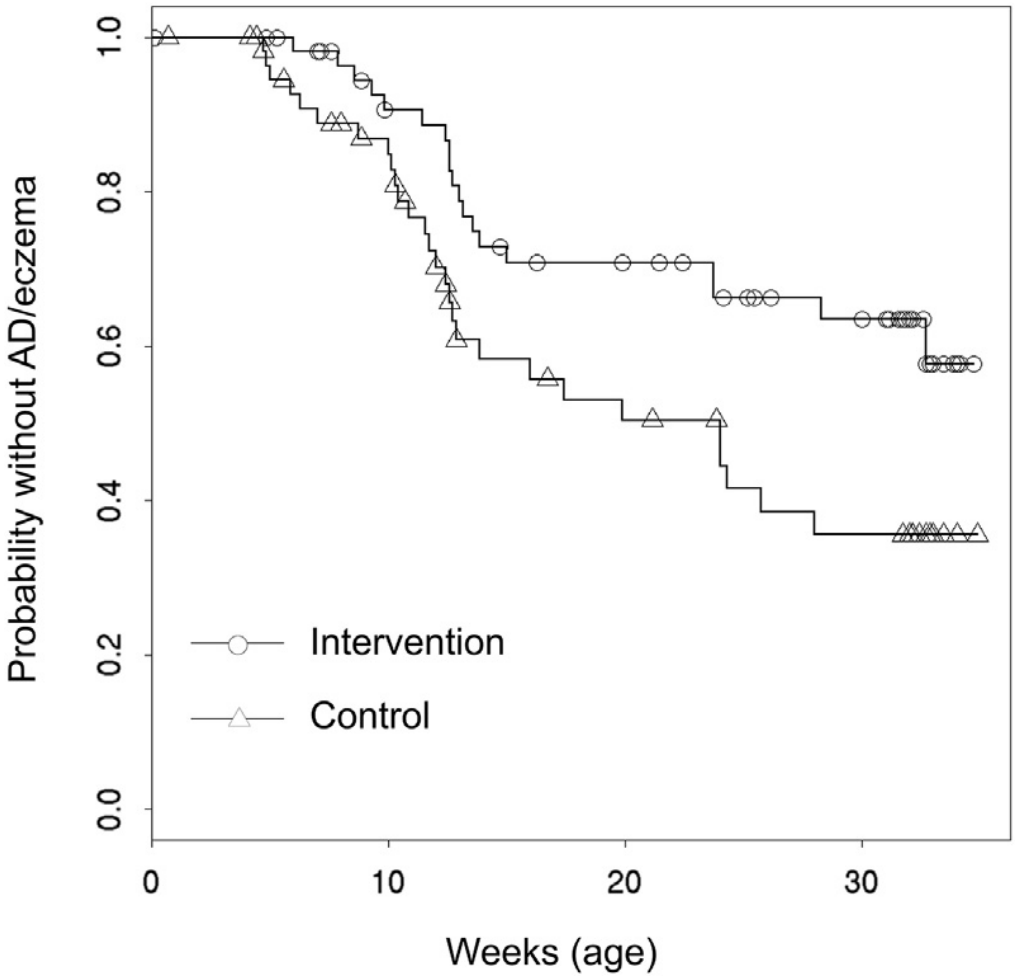
Case	Total no.	Had AD (control group)	Had AD (emollient group)	RR (95% CI)	*P* value
Complete (no imputation)	108	23/53 (43.4%)	12/55 (21.8%)	0.50 (0.28-0.90)	.017
Missing (assumed to develop AD)	124	30/60 (50.0%)	21/64 (32.8%)	0.66 (0.43-1.01)	.03
Missing (assumed not to develop AD)	124	23/60 (38.3%)	12/64 (18.8%)	0.49 (0.27-0.89)	.02
Worst-case scenario*	124	23/60 (38.3%)	21/64 (32.8%)	1.0 (0.61-1.62)	.99
Best-case scenario†	124	30/60 (50.0%)	12/64 (18.8%)	0.38 (0.2-0.66)	<.001

Simpson E et al. JACI 2014

Prospective, randomized, controlled trial

118 high AD-risk newborns
Emollient therapy from M1 for 32 weeks (2010-2013)
Daily at least 1x
In control group no emollient

AD cumulative incidence dropped by 33%



Horimukai et al. JACI 2014

New clinical studies

Higher patient number
More frequent use
AD incidence? Presence of sensitization?
Longer trials

Studies underway				
BEEP[59] (ISRCTN21528841)	1,400, England, family history of allergic disease	Daily Double base Gel or Dipro base Cream for the first year of life	Food allergy diagnosis at 24 mo (combination of parent report, allergic sensitization, and food challenge); wheeze and allergic rhinitis at 12 to 24 mo	Results available in 2019
PEBBLES–phase III, (ACTRN12617001380381p)	760, Australia, family history of allergic disease	Twice-daily EpiCeram from <2 wk to 6 mo	Skin prick test to 3 food allergens at 12 mo followed by food challenge if wheal is ≥1 mm	Yet to commence recruitment; intended completion data in 2021
PREVENTADALL, (NCT02449850)	2,500, Norway, general population	Bath oil and Ceridal face cream ≥5 times/wk from 0.5 to 9 mo of age; factorial design with early food introduction as co-intervention	Skin prick test at 6 mo and then annually form 12 mo; food allergy at 12, 36, 48 mo and annually thereafter; asthma and allergic rhinitis	Commenced December 2014
Shimojo et al (UMIN000010838)	800, Japan, general population	Skin care and Synbiotics factorial design	Sensitization to food allergens at 9 mo and food allergy at 12 mo	Trial closed and analyses underway
PACI (UMIN000028043)	650, Japan, 7- to 13-wk-old infants with AD	Aggressive management of AD with topical anti-inflammatory medications	Challenge-proved IgE-mediated egg allergy at 28 wk	Commenced July 2017

Why important to restore the barrier?

Basic treatment

- Complex treatment in AD is **unavoidable and inevitable** in both acute and chronic form of disease.
- Helps in **rehydration of skin**, decreasing skin dryness. Replaces essential fatty acids locally.
- Increases skin elasticity. **Decreases** tension and itch of skin. **Cleaning** effect.
- **Daily min. 2x** in case of symptoms and **1x daily for preventive** reasons
- **Whole body** use

Emollient therapy in AD

- Emollients right after shower (~5 min; ~27 Celsius), after gentle drying (skin still moist).
- Long effect even without shower.
- Allergens in emollients must be avoided to prevent sensitization via skin.

- By its water-binding elements (e.g. urea, glycerol, hyaluronic acid) it hydrates stratum corneum
- Occlusive characters (lipid, fat, oil contain) TEWL decreases
- Further effects: changes microbiome and pH; increases AMP level; FLG and loricrin expression increases; T cell and DC infiltration decreases; antifungal, anti-pruritic, anti-inflammatory effects

FLG: filaggrin; TEWL: transepidermal waterloss; AMP: antimicrobial peptide

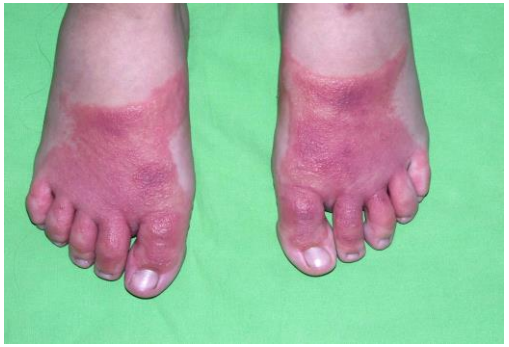
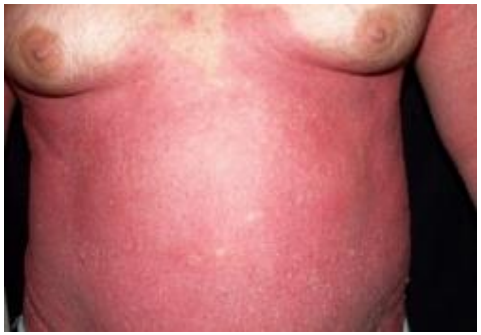
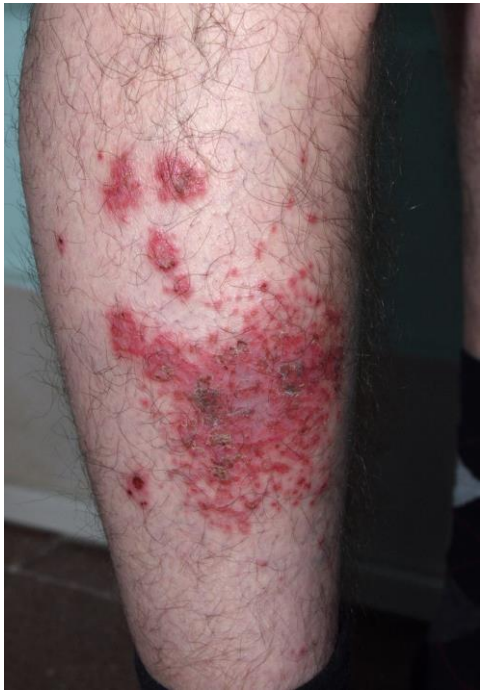
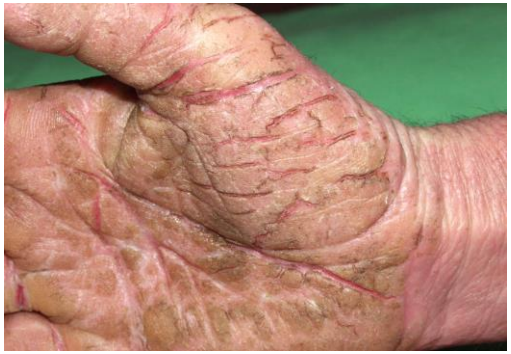
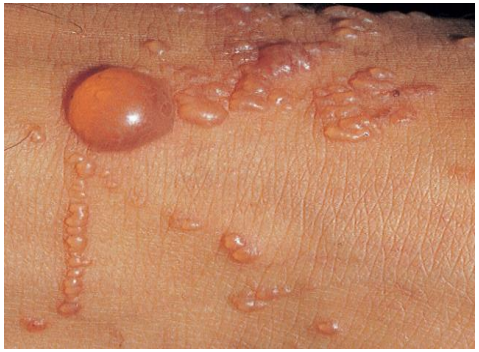
Wollenberg és mtsai 2003; Boussault és mtsai 2007; Chiang & Eichenfield, 2009

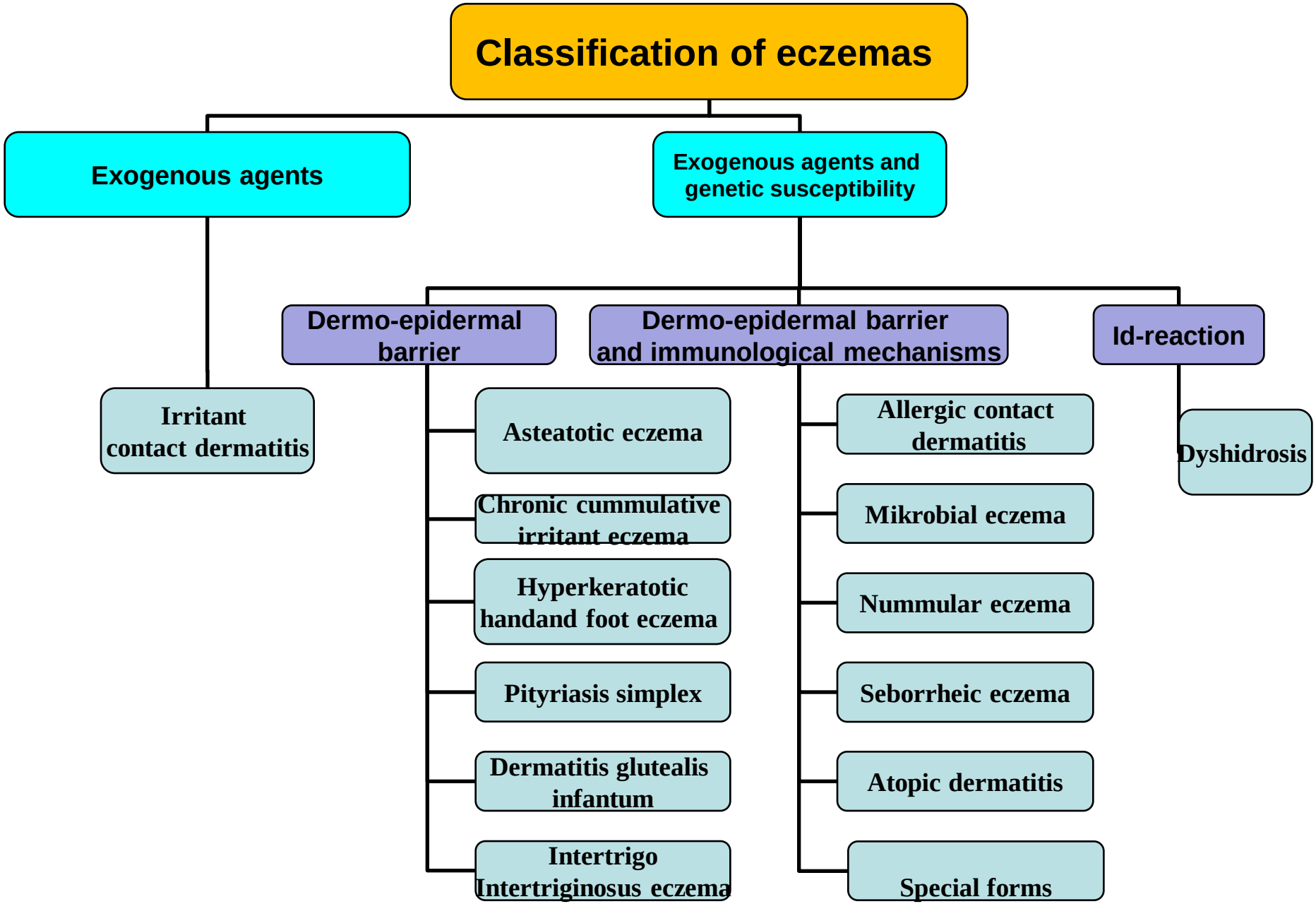
Skin diseases in wrestling

- **Trauma**
- **Eczema (contact dermatitis)**
- **Infection (fungal, bacterial, viral, parasitic)**

Contact dermatitis

- **Heterogeneous** group
- **Noninfectious** inflammatory dermatoses in which the the pathological changes in the epidermis and the upper dermis produce distinctive clinical pictures
- Extremely **common**, 15-25% of patients with skin diseases
- Occupational dermatosis (No1)


Classification of eczemas
Exogenous agents
Exogenous agents and genetic susceptibility
Irritant contact dermatitis
Dermo-epidermal barrier
Dermo-epidermal barrier and immunological mechanisms
Id-reaction
Asteatotic eczema
Chronic cummulative irritant eczema
Hyperkeratotic handand foot eczema
Pityriasis simplex
Dermatitis glutealis infantum
Intertrigo Intertriginosus eczema
Allergic contact dermatitis
Mikrobial eczema
Nummular eczema
Seborrheic eczema
Atopic dermatitis
Special forms
Dyshidrosis

Treatments in general

- **Conventional medicine** must be recommended and prescribed by physicians or health care professionals.

- Wrestlers not reporting a skin condition/infection and using **unusual and unhealthy treatments** (e.g. nail polish remover, bleach, salt, vinegar solutions) are causing unwanted adverse events (e.g. suffocate or burn an infection, leaving extensive scars).

- **”Home remedies”** – may be successful, but **do not guarantee to** kill the infection (only eliminating visible symptoms temporarily). Thus infections may not be symptomatic, but still remain transmittable.

Contact dermatitis – treatment

- **D**efine etiology, classification
- **E**liminate provoking factors
- **R**estore epidermal barrier function
- **M**oisturize
- **A**nti-inflammatories, immunosuppression (topical corticosteroids, topical calcineurin inhibitors)

Acronym: **DERMA** (skin)

Skin infections in wrestling

10% of time-loss injuries in wrestling are due to skin infections

- **Fungal**
- **Bacterial**
- **Viral**
- **Parasitic**

By National Collegiate Athletic Association's (NCAA) Injury Surveillance System

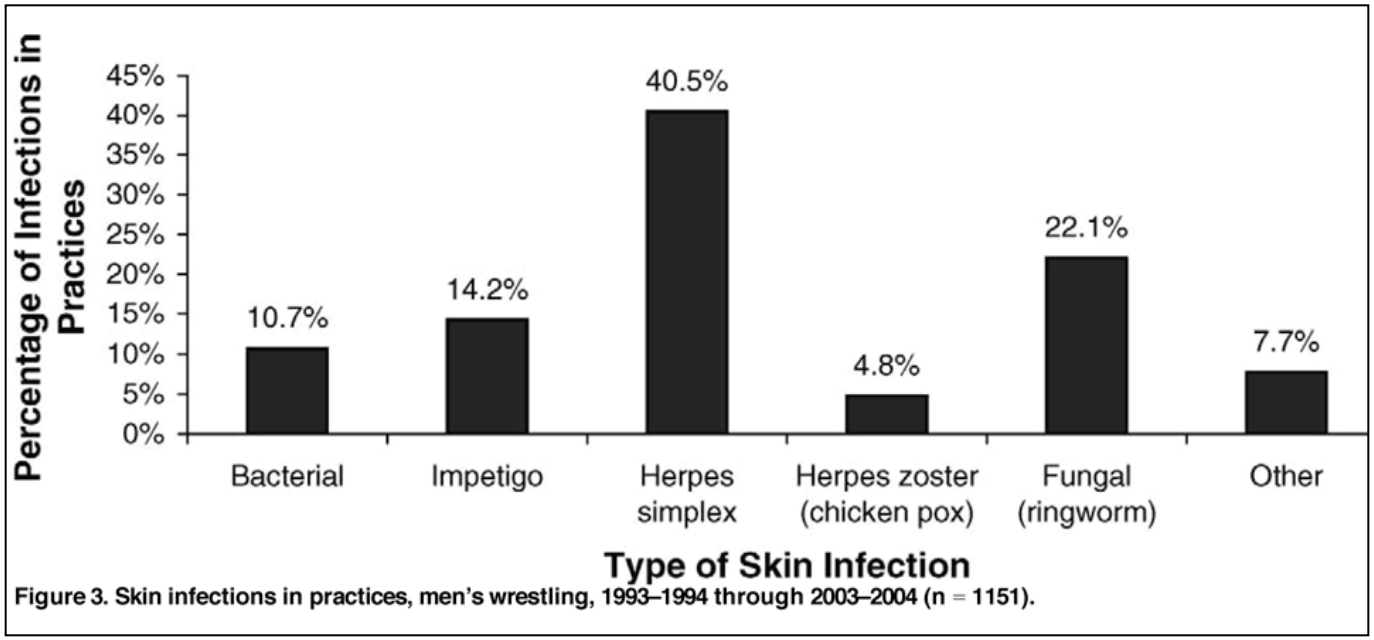


Figure 3. Skin infections in practices, men's wrestling, 1993–1994 through 2003–2004 (n = 1151).

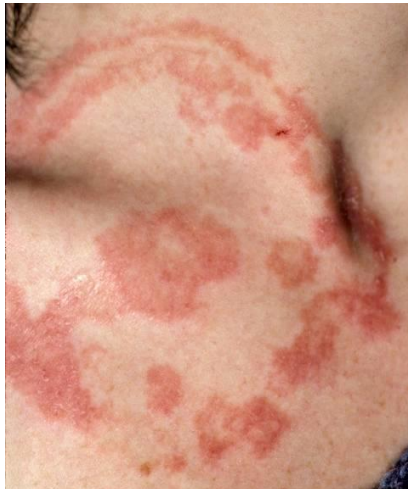
- Overall skin infection rate 14/10000
- 22% recurrence rate
- Rate for viral infections was 1.7x the rate of bacterial and 2.1x the rate of fungal infections

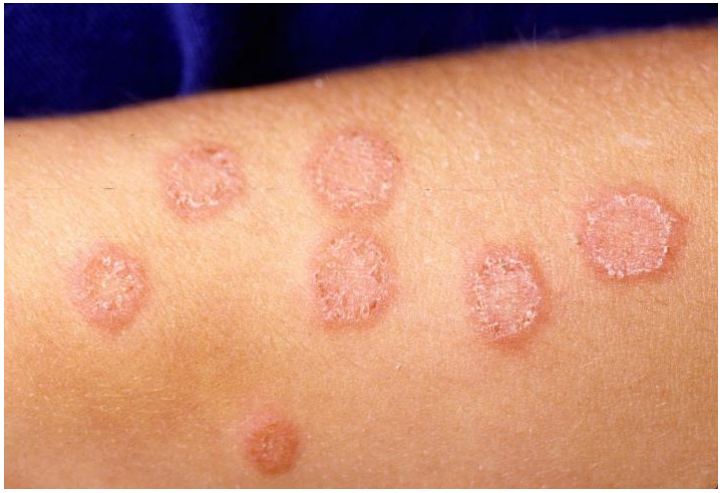
NCAA GUIDELINE 2j Skin Infections in Athletics 2008

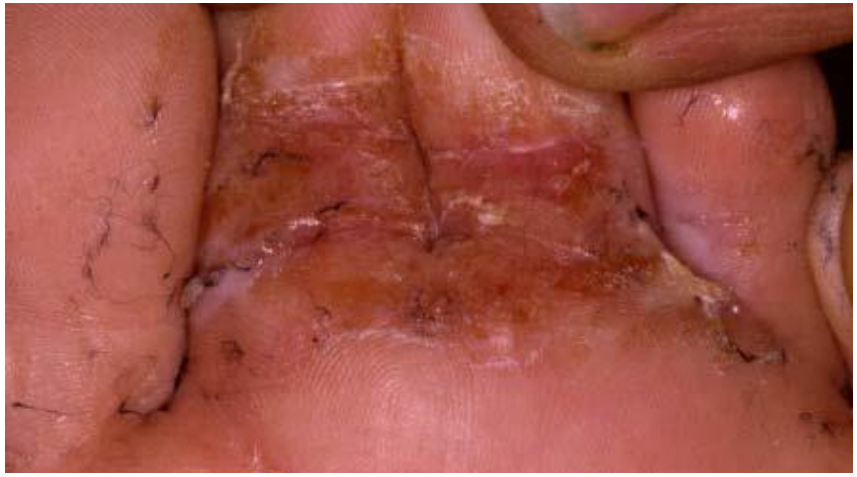
Herzog MM et al *J Athletic Training* 2017; 52:457-63.

Skin infections in wrestling

- **Fungal**
- Bacterial
- Viral
- Parasitic







- Direct contact
- Indirect sources (mats, headgear, towel, uniform)
- In scalp may get deeper lesions
- Tinea corporis gladiatorum;
- Athletes foot; jock itch; ringworm

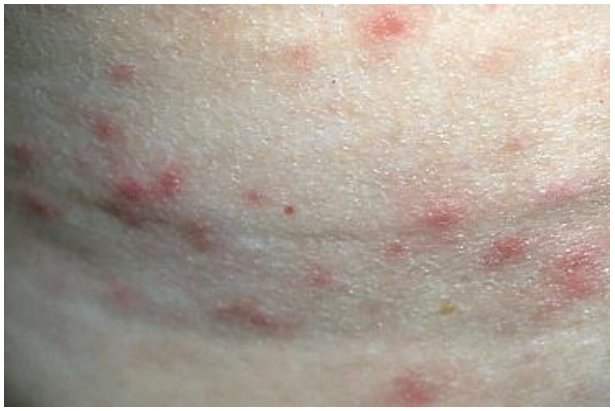
Fungal skin infections treatment

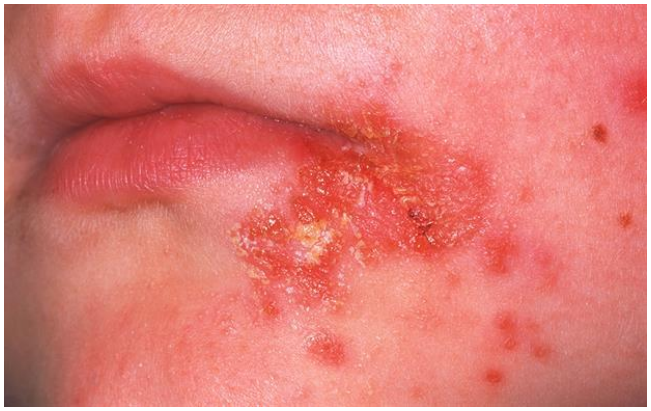
- **Topical – cream, ointment**
  - Once a day;
  - Do not cover;
  - 7-10 days
- **Systemic – tablets, capsules**
  - Extent forms of disease
  - Scalp involvement
  - Until total clearance (weeks)
- **Combination**
- **In recurrent cases antifungal prevention may be possible**

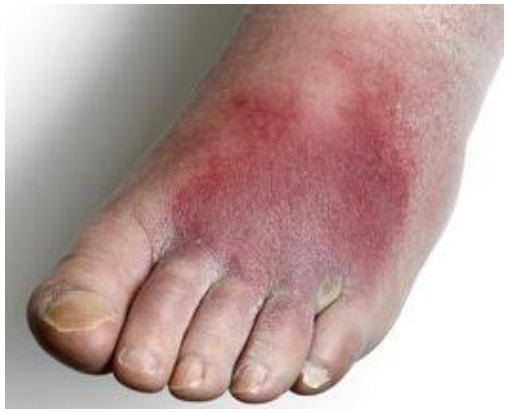
Skin infections in wrestling

- Fungal
- **Bacterial**
- Viral
- Parasitic

- Folliculitis, impetigo, erysipelas, cellulitis
- Streptococcus pyogenes; Staphylococcus aureus; Pseudomonas
- Contagious (crust covered erosions)
- Itch
- Direct contact
- Predisposing factors (shaving, haircut, eczema), primary sites (head, extremities)







Bacterial skin infections treatment

- **Topical – cream, ointment**
  - Topical antibiotics, disinfectants
  - Once a day;
  - Remove crust;
  - Cover;
  - 7-10 days

- **Systemic – tablets, capsules**
  - Antibiotics (in prevention not possible – resistance)
  - Extent forms or systemic symptoms
  - 7-10 days

- **Combination**

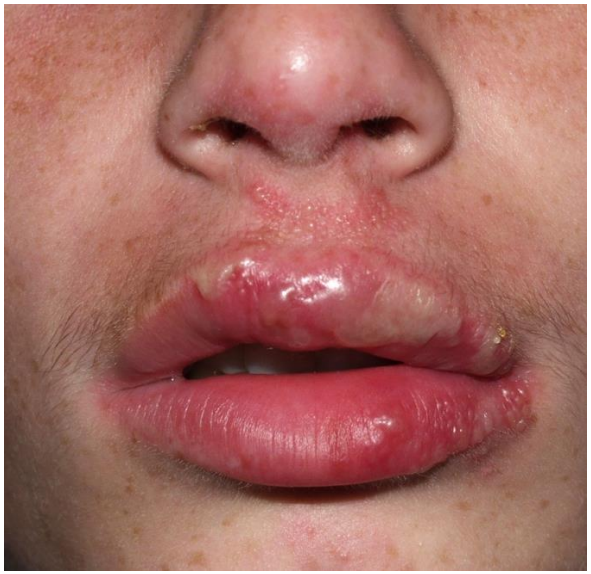
Community Associated Methicillin Resistant Staph. Aureus (CA-MRSA)

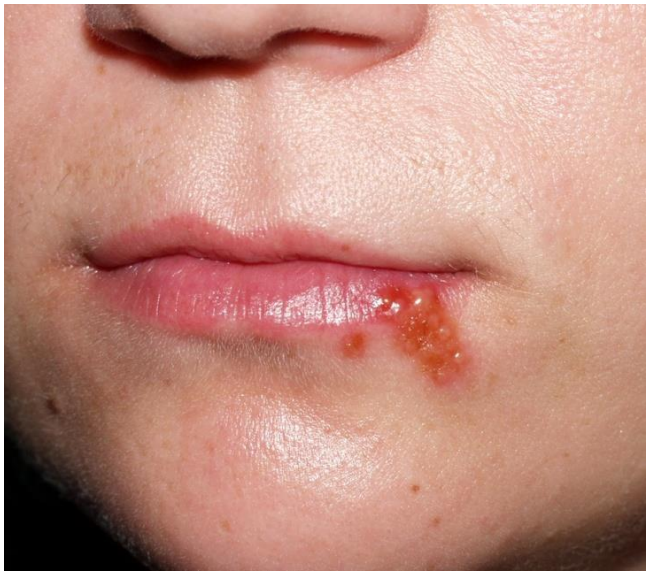
- Looks **identical** to other forms of *S. aureus*, but different strains. **Irresponsive** to regular antibiotics (e.g. Penicillin), but not multidrug-resistant
- Seen **in community**; believed **due to** antibiotic abuse/overuse for ear infection or viral infections
- Very **invasive and destructive** to skin and soft tissue
- Can **spread** to the lungs causing serious pneumonia
- Can only be diagnosed by **culturing** an infection
- When it occurs, usually **seen as** an abscess or boil (59%) vs cellulitis (42%) or folliculitis (7%)
- Primarily seen in **contact sports** (wrestling, rugby)
- Clindamycin (4x300mg) for 10 days; incision and drainage

Skin infections in wrestling

- Fungal
- Bacterial
- **Viral – herpes**
- Parasitic

- Latent virus (cluster)
- Contagious (30% chance to contract) (vesicles, open sores, early crusts)
- HSV-1, HSV-2
- Painful
- Lips, body, genitals
- Recurrence (stress, immuncompromised)
- Secondary bacterial superinfection





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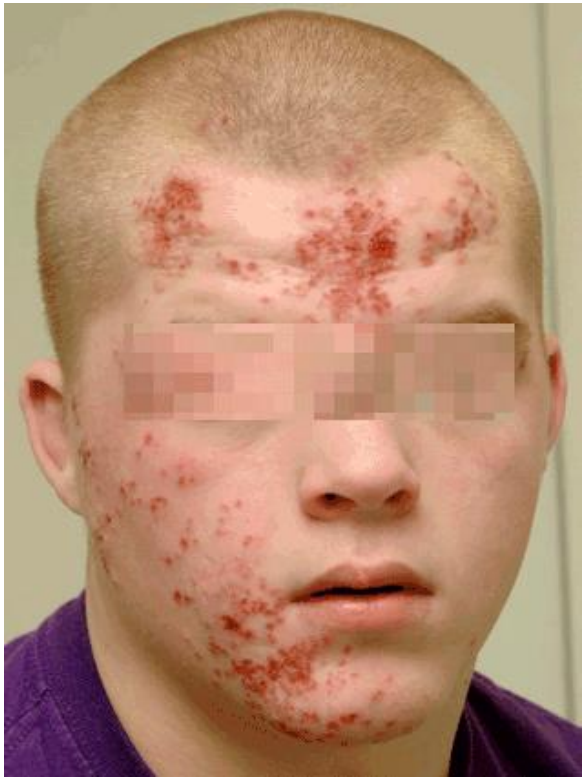


- **Herpes gladiatorum**
- Prevalence: 3-20% (varies in age groups)
- Primary outbreak: malaise, pharyngitis, fever, lymphadenopathy
- Primarily at ”lock-up” position: 70% head and face; 40% extremities; 30% trunk
- Skin-to-skin contact
- 3-8 days after contact, lasts for about 10 days
- All wrestlers in contact with it, should be isolated and monitored for 8 days. If no lesions develop, return to competition

www.medscape.com

B.J. Anderson, M.D.; Boynton Health Service University of Minnesota; Team Physician for Augsburg College Wrestling Team

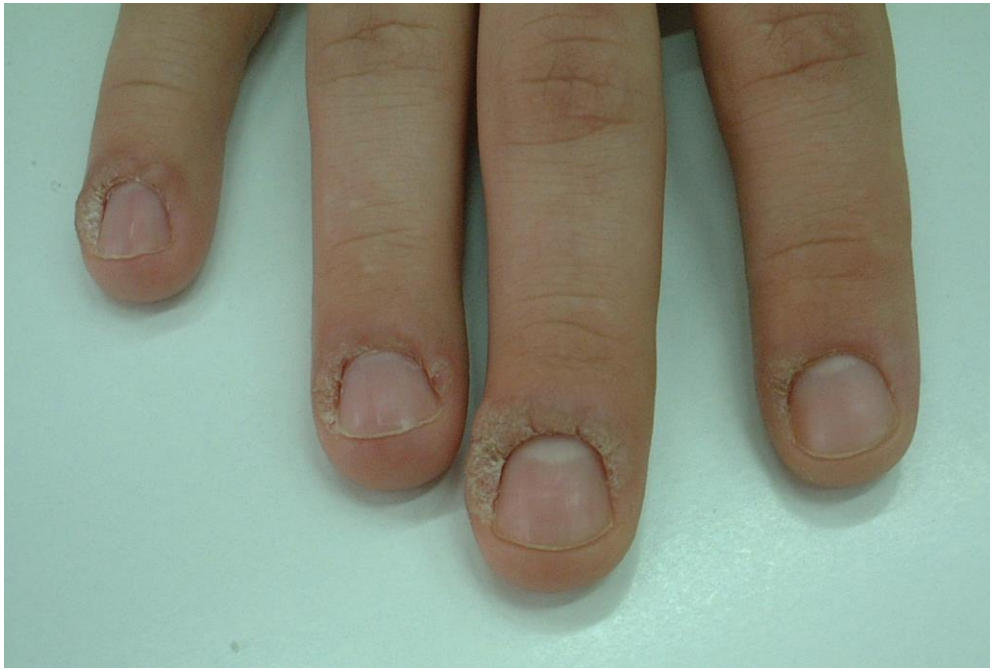
Herpes infection – treatment

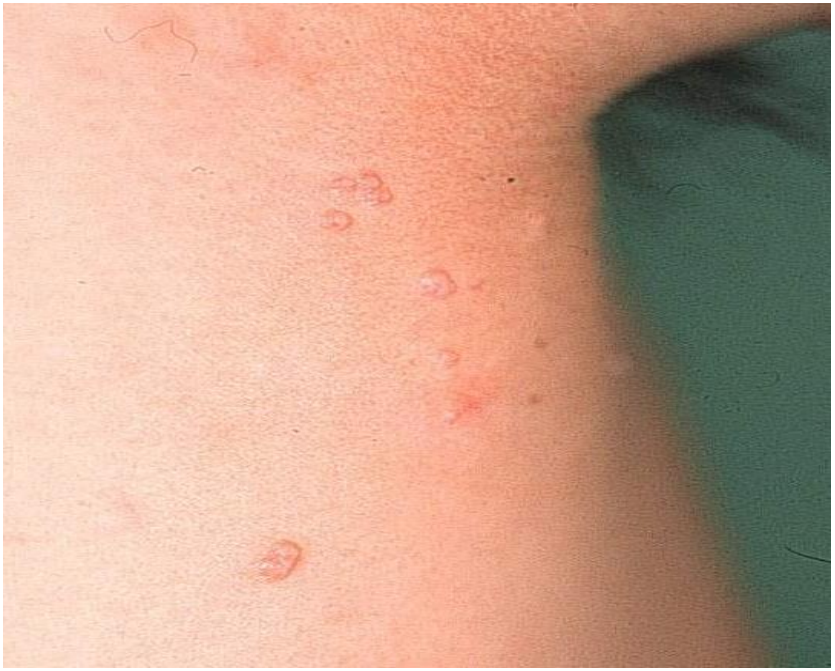
- **Topical – cream, ointment**
  - Not so effective
- **Systemic – Acute**
  - Start within 3 days
  - Acyclovir 5x200mg for 7-10 days (even longer)
- **Systemic – Recurrence**
  - Min 3x in 6 months
  - Acyclovir 2-3x200mg or 2x400mg for 6 months
- **Preventative antiviral** medication may be possible starting five days before the season and continuing throughout the season. Prophylactic valacyclovir (QD 1g) for 1M in wrestlers resulted in 85% decrease in the probability and 90% decrease in the incidence of outbreak.

Skin infections in wrestling

- Fungal
- Bacterial
- **Viral – wart, molluscum**
- Parasitic

- HPV; Pox virus
- Direct contact
- Contagious until removed





Wart/molluscum infection – treatment

- **Topical – exfoliative**
  - Salicylic acid
- **Topical – cytostatic**
  - Podophyllum
- **Topical – immunomodulatory**
  - Imiquimod

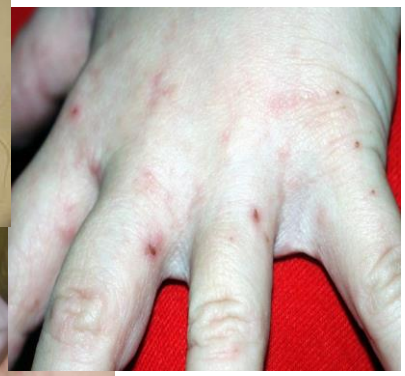
- **Surgical**
  - Liquid nitrogen
  - Electrosurgery
  - Curettage
  - Laser (ablative)

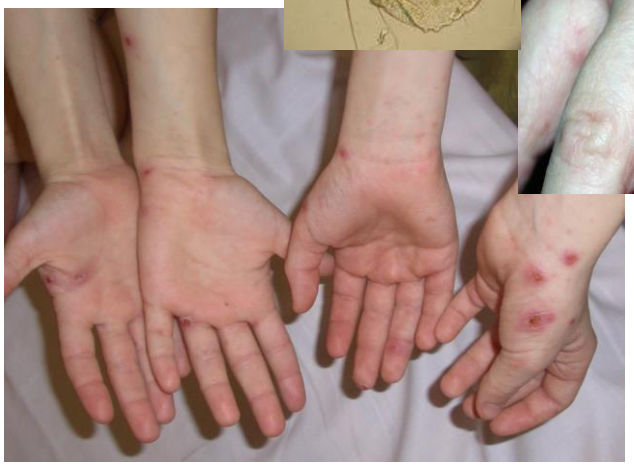
Skin infections in wrestling

- Fungal
- Bacterial
- Viral
- **Parasitic – scabies, lice**

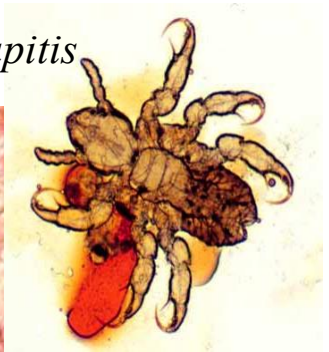
- Direct contact
- Intense pruritus
- Predilection areas (neck, hands, genitals)
- Contagious until treated (STI!)
- Eczema remains
- Possible superinfection
- Benzyl benzoate; Permethrin

Sarcoptes scabiei (mite)





Pediculus humanus capitis



Guidelines for Infected Athletes

Condition	NCAA[54]	NFHS[53]	NATA[55]	AAP Notes[51]
GABHS and *S aureus*, (abscesses, cellulitis, folliculitis, impetigo, carbuncles)				
T capitis	• Minimum of 2 wk of systemic antifungal therapy • Presence of extensive and	• Minimum of 2 wk of systemic antifungal therapy	• Minimum of 2 wk of systemic antifungal therapy	—
Verruca vulgaris (caused by subtypes of HPV)	• If multiple digitate lesions present on face, will be disqualified if cannot be covered by a mask	• No treatment or restrictions	—	—
S scabiei (scabies)[35]				—
P capitis[36] (head lice)	• Appropriate pharmacologic treatment and reexamination for completeness of response before return	—	—	—
T cruris	—	—	—	Exclude from swimming pools until treatment has been initiated[28,37]

- **Appropriate treatment (3-5-14 days depending on the disease)**
- **No new lesions present (for 2-3 days)**
- **Proper bandage (cover)**

NFHS: National Federation of State High School Associations; NCAA: National Collegiate Athletic Association; NATA: National Athletic Trainers Association; AAP: American Academy of Pediatrics

Davies HD et al. *Pediatrics* 2017;140(4):e20172477

Preventions in skin infections

- **Utilize recommended procedures** for cleaning and disinfection of surfaces. **Clean** workout gear, clothes, towels for each practice. **Mats must be cleaned** before each practice with appropriate disinfectant.
- Regular **skin check** (performed every day before practices) (visual enough no palpation necessary)
- Improve wrestlers' **hygiene practices**
- Shave your **face only** (otherwise opportunity for infection)
- Wrestlers must **shower** immediately after practice (nearly 10% do not do!)
- Coaches and trainers must be **educated** on skin infections

Prevention Key to Reducing Skin Infections in High School Wrestling by B.J. Anderson, M.D., and Larry Cooper, MS, LAT, ATC on December 21, 2015

Skin examination and rules

- At wrestling meets, **skin must be checked** by medical experts or trained referees**.**
- Any **skin condition must be** stated **non-infectious**, adequately **medicated and covered** with bandage.
- Wrestlers must have developed **no new lesions 72 hours** prior to examination.
- Open wounds and infectious **skin conditions** that **cannot be adequately protected** are considered grounds for **disqualification** (from both practice and competition).
- Wrestlers **undergoing treatment must provide written** documentation from a physician (diagnosis; culture results - if possible; date therapy began; names of medications.

Conclusion – Take home message

- **Skin infections** may be **significant problems** in wrestling
- Look after your **environment (clean equipment)**! Protect yourself of harm/danger (**danger model**)!
- Keep the **barrier intact**! Prevention by **moisturizing**
- Prompt and **proper diagnosis by specialist dermatologist** inevitable in case of dermatological disorders
- **Isolation and observation** of individual
- **Targeted treatment** if necessary (avoid ”home remedies”)
- Importance of **withdrawal of wrestler’s permission** from competition