More than just skin deep: atopic dermatitis guidelines and update in 2019

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Saturday June 29th, 11:10-11:55am

Conflicts of Interest:

- Principal investigator and advisory board for *Regeneron/Sanofi Genzyme*

Learning objectives

- Describe the role of comorbid conditions like ichthyosis vulgaris and food allergies in patients with atopic dermatitis.
- Review the use and safety of prescription non-steroidal topical options for atopic dermatitis.
- Recognize the multifaceted approach to treatment and prevention of atopic dermatitis and related complications.
- Discuss new medications for pediatric AD.
- Translate and institute AD guidelines (2014) into your practice.

Who treats atopic dermatitis (AD)?

- Pediatricians 30%
- Dermatologists 25%
- Primary care physicians 20%

Atopic dermatitis (AD)

- Affects up to 20% of children in US
- Arises before age 5 y in 90% of individuals
- Marked increase in past decades
- Most common chronic inflammatory disease in early childhood
- Significant morbidity for patients/families
- Often first step in “atopic march”

Atopic march in children

- Asthma occurs in up to 50% with AD
- Allergic rhinitis develops in 50-80%
- Children with more severe AD
  - Higher risk of developing asthma
  - Sensitization to foods and environmental allergens
- Is it really a ”march”?

Timeline of AD

- Most common onset between 3-6 months
- 60% of patients in 1st year of life
- 90% of patients by 5 years
- 10-30% do not resolve by adulthood

Risk factors for AD

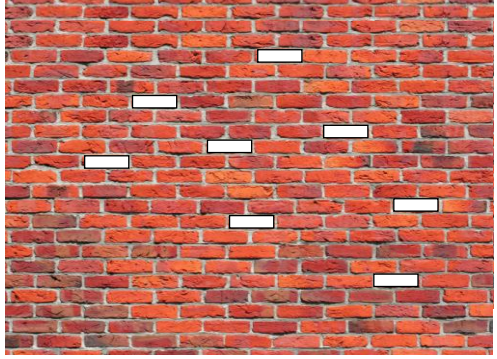
- ***Family history of atopy***
  - Odd 2-3X higher with 1 atopic parent
  - Odd 3-5X higher with 2 atopic parents
  - 70% of patients have (+) family history

- Loss of function mutation in *FLG* gene

Prevention of AD

- No clear evidence for:
  - Dietary antigen avoidance in pregnancy & breast-feeding, long-term breast feeding, hydrolyzed protein formulas, soy formulas, omega fatty acid supplementation, pre- or probiotics, delayed introduction of solid foods**
- Daily full body emollient therapy from birth may reduce the cumulative incidence of AD in high risk infants by 30-50%*

**Weidinger S and Novak N. Lancet 2016
*Horimukai K et al. J Allergy Clin Immunol. 2014
*Simpson EL et al. J Allergy Clin Immunol. 2014



AD & pathogenesis

- Epidermal barrier abnormality
- Cutaneous inflammation
  - *Immune dysregulation*--acquired and innate
    - Systemic “allergic” TH2 skewed cell response
    - Defect in innate immunity function
- Frequent *staphylococcus aureus* colonization and infections
  - Reduced expression of anti-microbial products
  - Impaired skin barrier

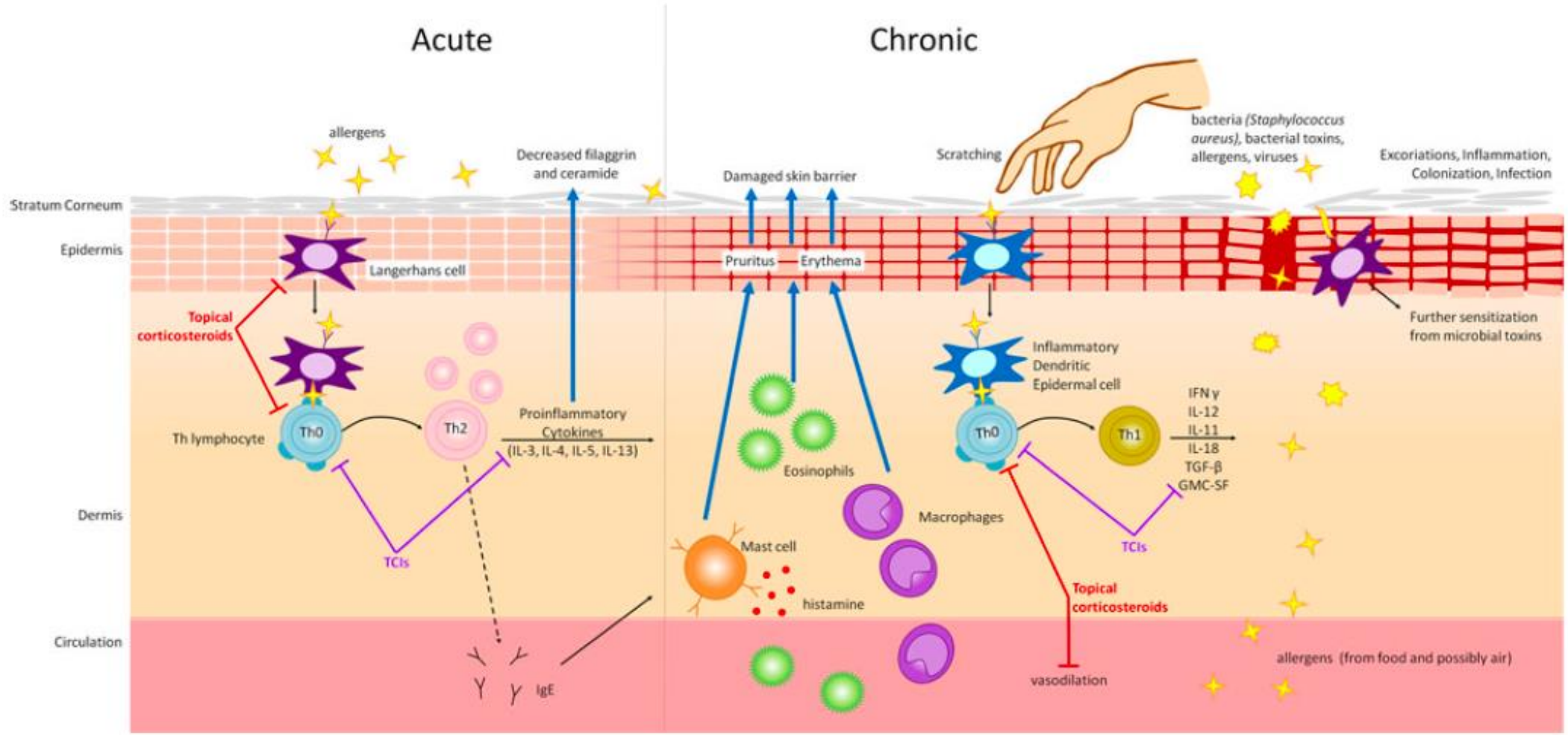


Fig. 1 The mechanism of atopic dermatitis and sites of action of topical calcineurin inhibitors and topical corticosteroids. *GMC-SF* granulocyte-macrophage colony-stimulating factor, *IFNγ* interferon γ, *IL* interleukin, *IgE* immunoglobulin E, *TCI* topical calcineurin inhibitor, *TGF-β* tumor growth factor- β, *Th* helper T lymphocyte

Carr W.W. Pediatr Drugs 2013

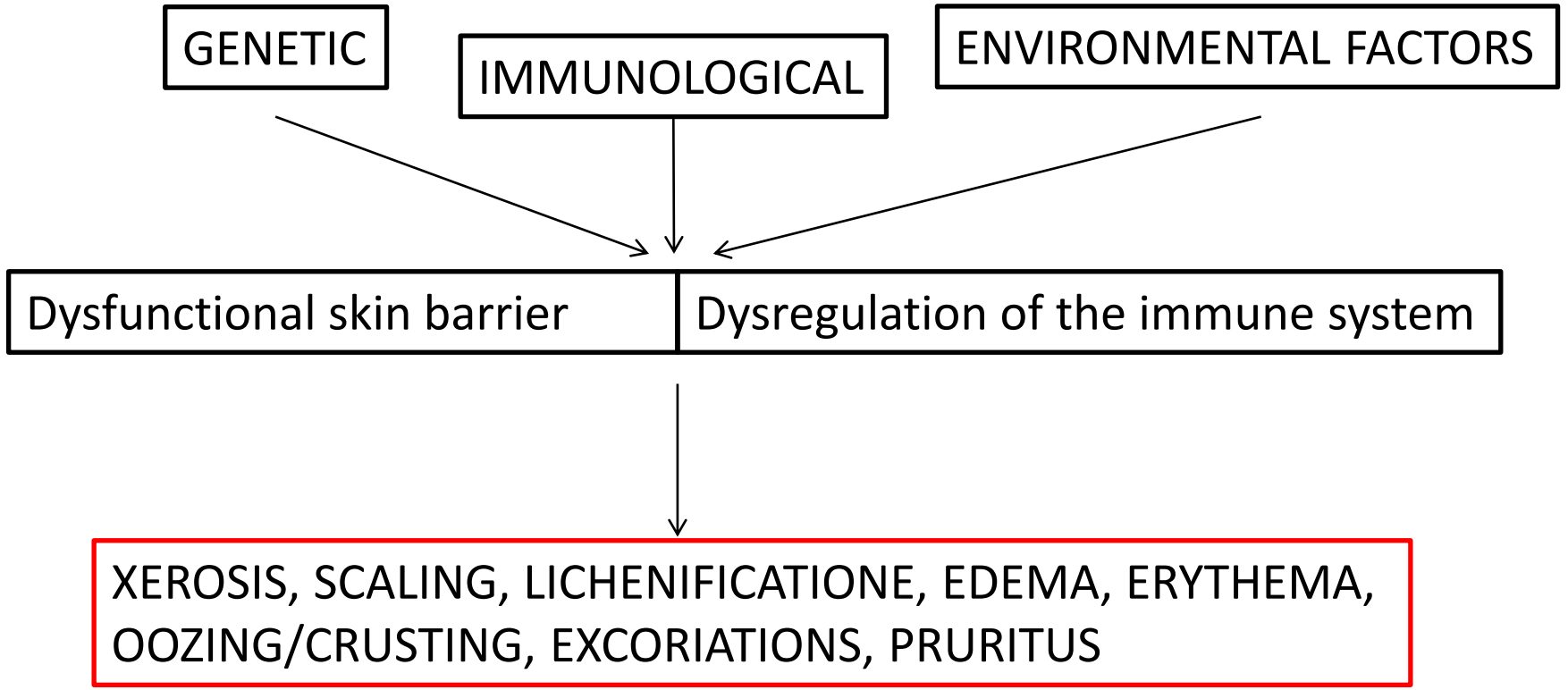
AD associations with environment

- Urban>rural areas
- Smaller families
- Higher socioeconomic classes

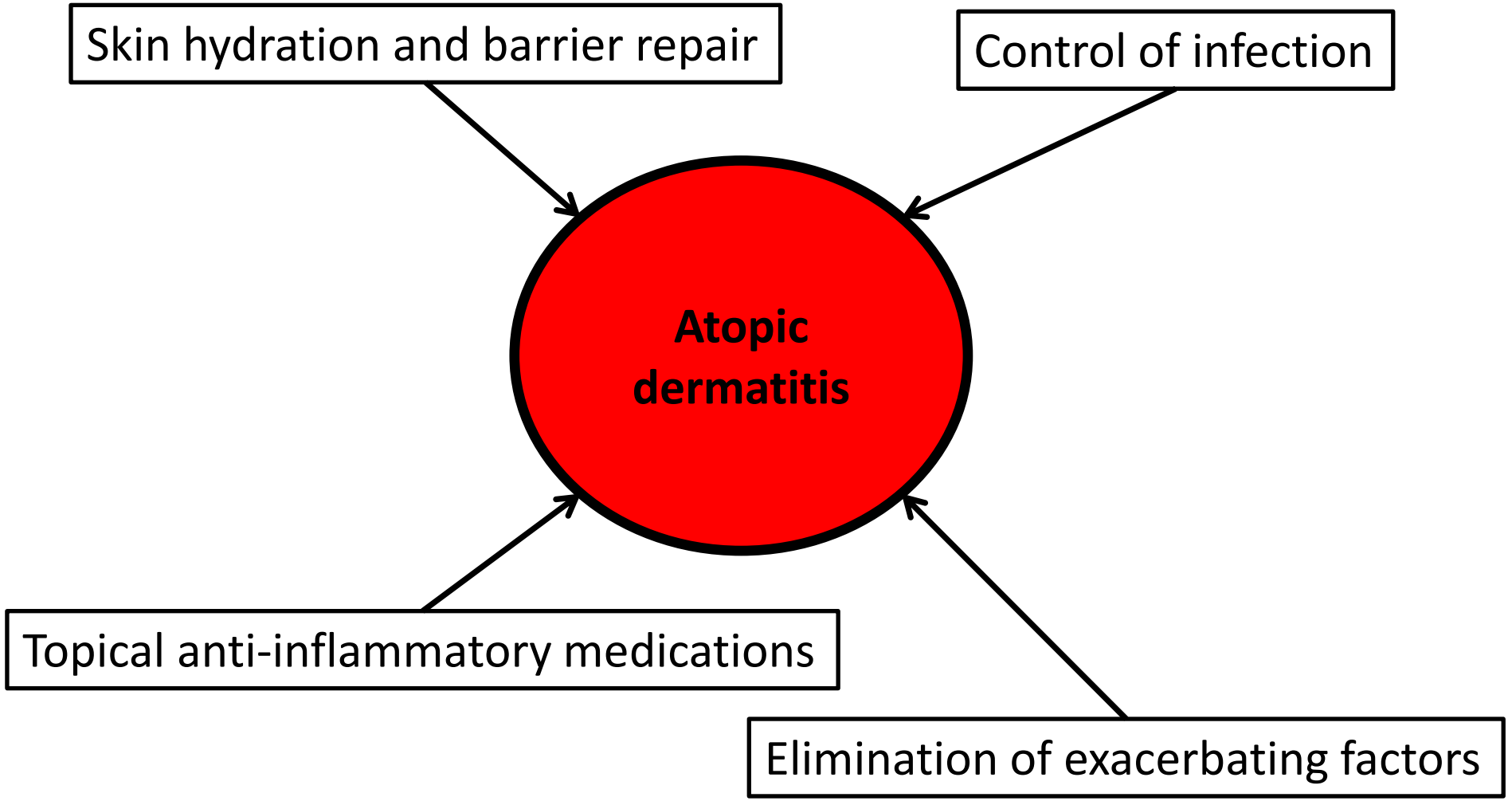
Other associations

- Xerosis
- Ichthyosis vulgaris
- Susceptibility to irritants and contact allergies
- Elevated IgE and peripheral eosinophilia (70-80%)
- Keratosis pilaris
- Cheilitis
- Prurigo nodularis

AD: complex pathogenesis



Management of AD: Multipronged approach



Skin hydration & barrier repair

Icthyosis vulgaris (IV)-model for defective barrier function

- Xerosis (dry skin)
- Hyperkeratosis (thick skin)
- Excess scaling
- Keratosis pilaris
- Palmar/plantar hyperlinearity
- Increased risk/severity of asthma, rhinitis, food allergies, and atopic dermatitis

Photo: R.C. Hansen

Icthyosis vulgaris (IV)

- Common in Europeans and Asians
- Loss of function mutations in filaggrin gene (*FLG*)
- Filaggrin protein responsible for:
  - Epidermal differentiation
  - Proper aggregation of keratin filaments
  - Skin’s natural moisturizing factor (NMF)

Effects of filaggrin deficiency

- Reduced level of NMF-reduction in hydration
- Elevated surface pH*
- Increased transepidermal water loss*
- Excessive scale (corneocytes cannot stay hydrated as they move up through SC)
- Epidermal hyperkeratosis (due to compensatory repair mechanisms)

*in double allele mutations

Effects on filaggrin deficient skin

- Increased permeation of
  - Chemicals
  - Allergens

Explains increased risk of sensitization
to aeroallergens and haptens in those with IV

Detection of IV

- Obtain a family history
- Inspect skin
  - Palms/soles
  - KP?
  - Xerosis
- Clinical diagnosis!
- (Skin biopsy, EM, and mutation testing)

FLG mutation & atopy

- AD patients without FLG mutation, show down-regulation in expression of gene
- 50% of cases of moderate-severe AD, 15% of mild-mod AD have some form of FLG mutation
- FLG mutations independently associated with food allergies, peanut allergies*
  - Sensitization through skin barrier?

*Asai Y et al. J Allergy Clin Immunol 2013
*Brown SI et al. J Invest Dermatol 2008

Barrier repair: emollients

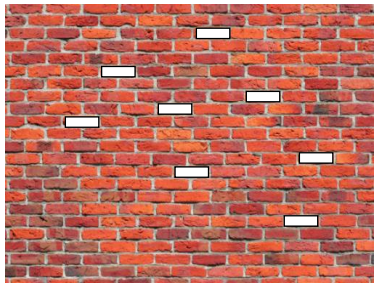
- Non-cosmetic moisturizers (lotions, creams, etc)
- Apply overtop of medications (variations exist)
- Apply to whole body, twice daily ideal
- After bathing when possible (3 minute rule)
- Avoid those with contact allergens (lanolin, fragrance, etc) whenever possible
  - Vanicream® products
  - Petrolatum jelly

Emollients & discerning vehicles

- Lotion: high water content, oil in water
  - Thin, light, easy to spread
  - Alcohol content, may sting
- Cream: oil in water (50/50)
  - Spread easily, wash off with water, soak in fast
  - May be better for “oozy” or “wet skin”
  - More preservatives, may sting
- Ointment: water in oil (oil base) (20/80)
  - “Greasy”, occlusive, do not soak in fast
  - Good on dry skin, help penetration of topical drugs

Ceramides in barrier repair

- Major lipid component of stratum corneum
- Make up the “mortar”
- Help maintain barrier and prevent TEWL
- Decreased levels associated with AD
- Now incorporated into skin care products
  - Cerave™ products
  - Cetaphil™ Restoraderm



Barrier repair: emollients

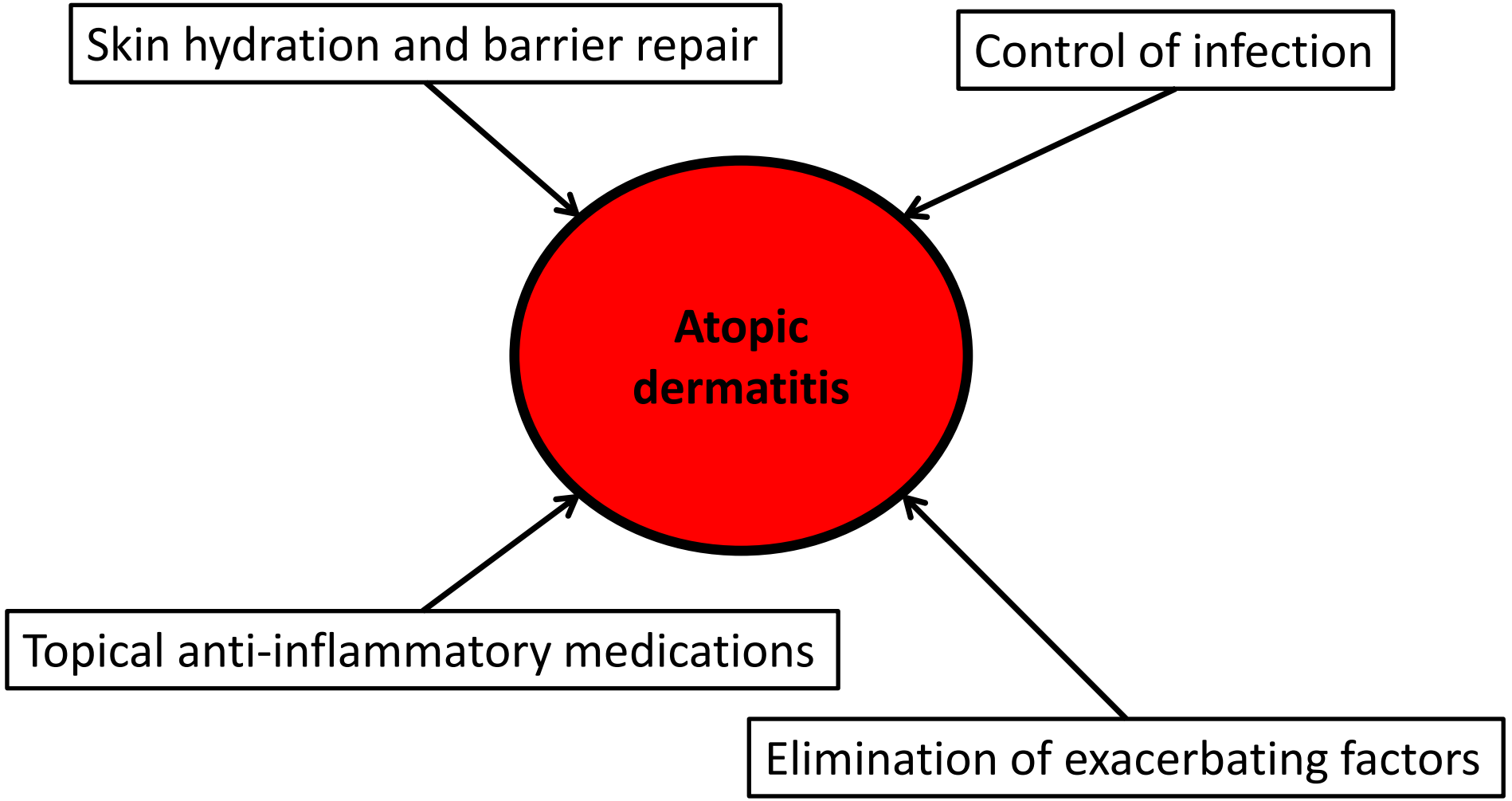
- Avoid salicyclic acid & other “stingy” ingredients
- Use ointments for infants and young children
  - Exception: hot/humid climate, occlusion problems
- Ointments at bedtime for older children
- Vehicle preference becomes important with age
- Consider ceramide-containing moisturizers

Water based lotions and creams are inadequately occlusive and can cause burning or stinging!

Bathing-do's and don'ts

- WE DO NOT LIMIT BATHING
- Mild non-alkaline (non-soap) cleansers (syndets) preferred when needed in soiled areas
- Do not soak in "soapy" water
- NO BUBBLE BATHES, scented salts or oils
- Watch for irritating contactants (such as those in shampoos/conditioners)
- Warm water soak for 10 minutes

Management of AD: Multipronged approach



Treatment updates in AD

Topical corticosteroid preparations (CS)

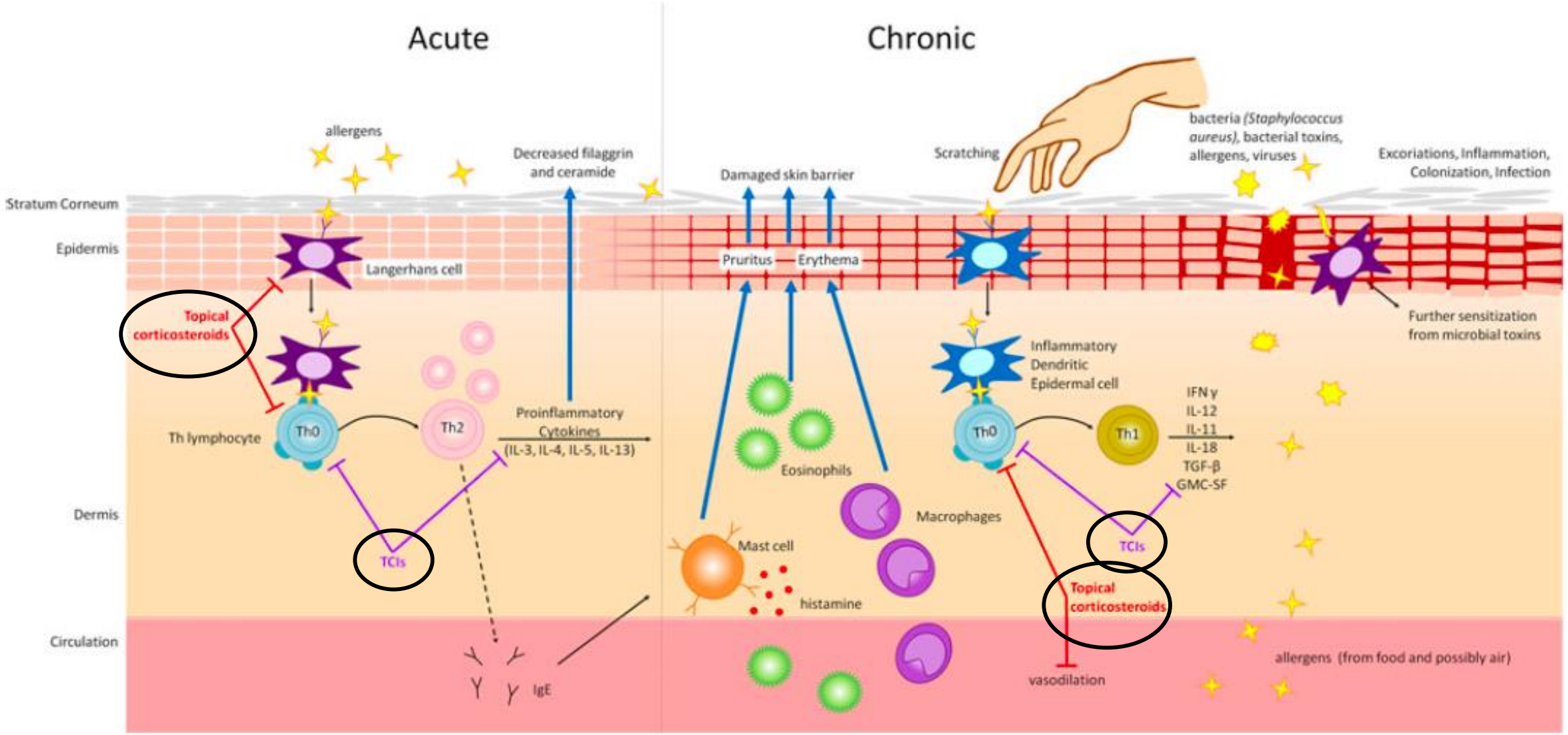
- *Topical CS still remain first line for AD treatment*
- Choose appropriate strength for location & severity
- Appropriate strength should allow for **clearance** of disease &“break” from CS or transition to maintenance routine
- Safe when used appropriately with low risk of side effects in children

Topical calcineurin inhibitors (TCI)

- Available since 2000-2001
- Pimecrolimus (1%)-mild/moderate dx, cream
- Tacrolimus (0.1/0.03%)-moderate/severe dx, oint
- FDA approved ages 2-15 y (pimecrolimus 1%, tacrolimus 0.03%)
- *Considered 2nd line for short term and chronic intermittent treatment*

TCIs--how do they work?

- Immunosuppressive
- Inhibiting the activation of T lymphocytes and therefore, proinflammatory cytokines
- DO NOT reduce the # of Th cells in healthy skin
- DO NOT have an effect on Langerhans cells


Acute
Chronic
allergens
Decreased filaggrin and ceramide
Damaged skin barrier
Scratching
bacteria (Staphylococcus aureus), bacterial toxins, allergens, viruses
Excoriations, Inflammation, Colonization, Infection
Stratum Corneum
Epidermis
Dermis
Circulation
Langerhans cell
Pruritus
Erythema
Topical corticosteroids
Th lymphocyte
Th0
Th2
Proinflammatory Cytokines (IL-3, IL-4, IL-5, IL-13)
TCIs
Further sensitization from microbial toxins
Inflammatory Dendritic Epidermal cell
IFN γ
IL-12
IL-11
IL-18
TGF-β
GMC-SF
Th1
Eosinophils
Macrophages
Mast cell
histamine
IgE
vasodilation
allergens (from food and possibly air)

TCIs and appropriate use

- Sensitive skin areas (face/eyelids/groin)
- Topical CSs have been proven ineffective
- Patients with contraindication to topical CS or side effects from topical CS

Topical calcineurin inhibitors (TCI)

- Side effect: burning/warmth sensation at site
- NO skin atrophy
- NO evidence of systemic immunosuppression or increased risk of malignancy
- Have to explain the “black box” to parents-theoretical increased risk of lymphoma & non-melanoma skin cancer
  - Issued in absence of safety data for long term use

TCIs and risk of cancer

- Long term safety not established at approval
  - Animal studies suggested possibility of immune- mediated malignancy with systemic exposure
  - Oral/IV forms in transplant patients associated with increased risk of immune-mediated malignancies
- “Theoretical risk” of lymphoma in Jan 2006
- Resulted in a BLACK BOX warning
- No casual risk had been demonstrated
- US FDA could not rule this out

TCIs and cancer: the facts

- Lymphoma incidence in pimecrolimus-exposed population up to 45 X less than general population*
- 5 epidemiological studies involving > 6.5 million AD patients have not provided evidence for increased lymphoma risk with pimecrolimus^
- No evidence of increase incidence of skin cancer with TCI use**
- Post-marketing data & prospective registries have not identified an increased risk of lymphoma with TCIs#

*Carr W.W. Pediatr Drugs 2013

^Luger T. et al. Pediatr Allergy and Immun 2015

**Tennis P et al. Br J Dermatol 2011

TCIs and infection

- No compelling evidence to indicate association with overall increased infections
- May be slight increased risk for viral skin infections, like eczema herpeticum

Summary: TCI use and risks

- Risk of malignancy based on animal studies not translatable to humans and transplant patients
- Incidence of lymphoma no greater in TCI-treated patients than that in general population
- Post-marketing surveillance data show #s of lymphoma cases lower among patients exposed to TCIs as compared to general populations
- No increased risk of infection or skin cancer, possible increase in viral skin infections

TCIs (other) barriers to use

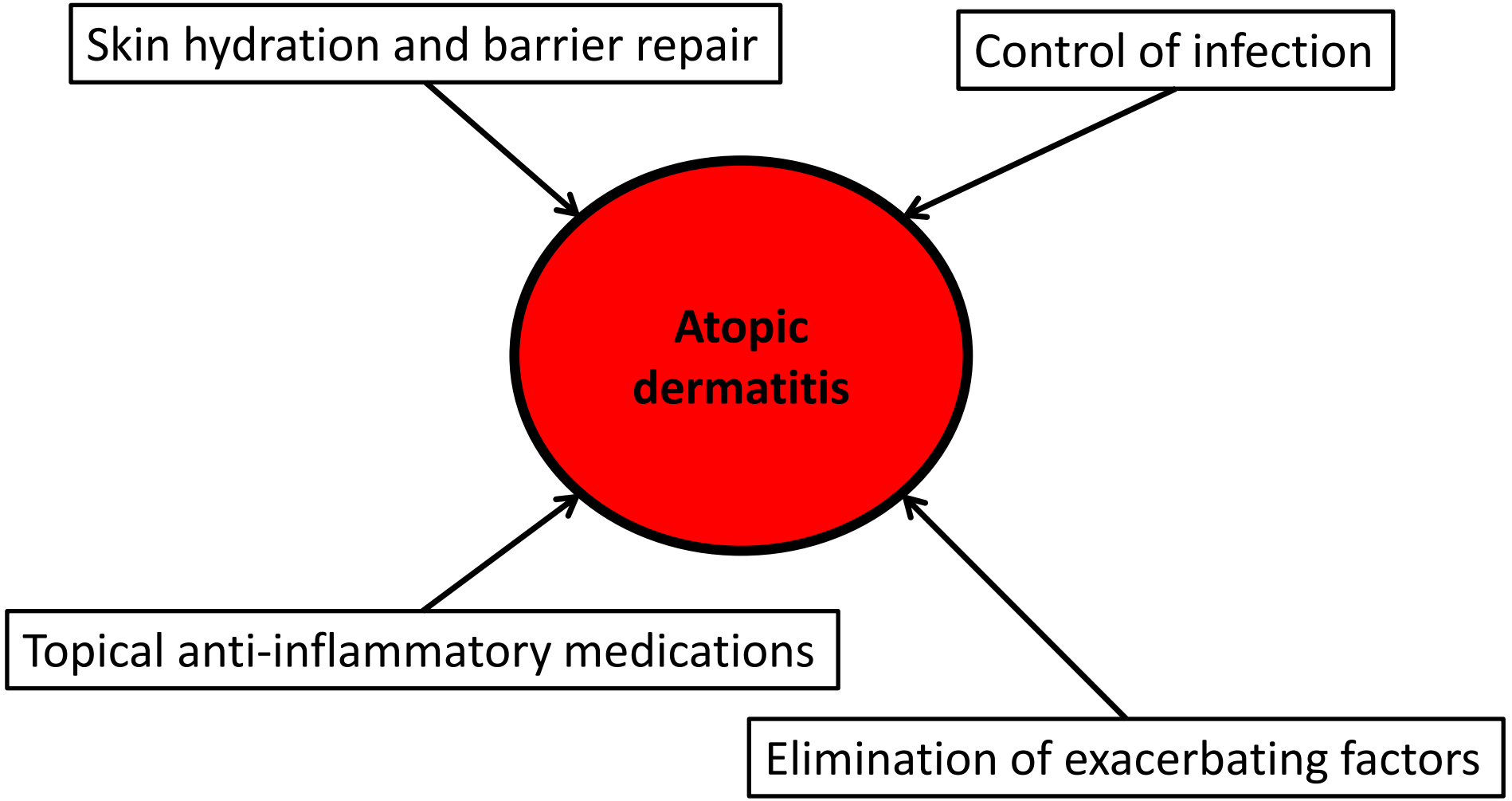
- Fears in parents and health care providers
- Lack of insurance coverage despite FDA approval in appropriate age groups
- No approval under 2 years despite safety and efficacy data down to 3 months of age
- High cost

New medications on the horizon

- ***Topical phosphodiesterase-4 inhibitors***
  - Inhibition of PDE4 blocks hydrolysis of cAMP
  - Prolong/enhance cAMP resulting in suppression of Th1 and Th2 immune response

- ***Dupilumab for severe atopic dermatitis***
  - Fully human monoclonal antibody against IL-4Ra
  - Blocks IL-4 and IL-13 (drivers of Th2 inflammation)

Management of AD: Multipronged approach



Treat and prevent infection in AD

Photos: R.C. Hansen

Bacterial infections in AD

- Suspect when sudden worsening of eczema
- Skin pain, malaise, other symptoms/signs
- Pustules, increased drainage, spreading redness, furuncles/carbuncles
- Most common organism is *staph aureus*
  - Know local resistance patterns for MRSA

Bacterial infections in AD

- Often a sign of severe uncontrolled AD
- Differentiate colonization vs. infection
  - Up to 95% of our patients are colonized with *staph*
- Perform a bacterial culture!
- Base treatment on cx & susceptibility patterns

Treatment of active infection

- Topical antibacterial for localized infection
  - Mupirocin ointment, retapamulin
  - Avoid neomycin/bacitracin containing agents
- Oral antibacterial agents for more severe, generalized infections
  - Base on culture and sensitivities
- Use antibacterial prophylaxis measures for colonization and repeated infections

Dilute sodium hypochlorite bathes

- Decrease *staph* colonization and need for Abx
- Decrease flares and overall eczema severity
- NO known resistance to bleach
- SAFE at recommended dilution
- Rare complaints of burning, increased itching
- Do not use detergent bleaches or bleach alternatives

Dilute bleach bathes-how to do it*

- ¼ cup in half tub, ½ cup in full tub
- Perform twice weekly
- Soak for 10 minutes
- +/- Rinse after bath
- Increase for flares of AD, can decrease frequency over time if stable

*Concentration for 6% sodium hypochlorite
Use of 8.25% requires use 1/3 more water

Bacterial decolonization

- Bleach bathes 2-3X week, soak for 10 min
- Dilute bleach body wash, 2-3X week, leave on for 90 sec while in shower (older children)
- Chlorhexidine, other anti-bacterial cleansers
- Intranasal mupirocin (also perianal, umbilical, axillary), 5-7 days/month, BID
- If family h/o *staph*, may encourage other household members to participate

Eliminate exacerbating factors (irritants, allergens, contributors)

Elimination of exacerbating factors

- Identify triggers and eliminate when possible
  - Foods
  - Aeroallergens
  - Irritant, allergic contact allergens

Avoidance of allergy triggers

- Foods in infants
- Aeroallergens in children/teens
- *Negative skin prick/RAST tests are highly predictive*
- Positive skin test often shows no correlation with clinical signs of eczema

IgE mediated food allergy

- Often children with moderate-severe AD
- Milk, hens eggs, soy, wheat, peanuts (90%)
- *Most foods cause immediate urticarial or maculopapular eruptions not dermatitis*
- Many foods act as irritants (citrus) on skin

Food allergies in AD

- 2010 clinical guidelines
  - “an adverse health effect arising from a specific immune response that occurs reproducibly on exposure to a given food”
- Testing recommended in those who who have experienced an immediate reaction following ingestion of a certain food
- Routine screening with AD not warranted

Food allergies as triggers

- (+) Tests serve only as a potential guideline
- Tests have low accuracy for predicting triggers
- 10-15% children overall have allergies that may be relevant to skin disease
  - Almost always those with more severe disease
- Consider in children: WHEN
  - Not responsive to optimized eczema therapies
  - With suspected allergic conditions
  - Temporal relationship with food and skin

Food allergies as triggers

- Most foods hard to avoid
- Restrictions should not worsen QOL more than atopic disease
- Restrictive diets can lead to weight loss, malnutrition, worse disease
- After few years, risk of reactivity diminishes

Aeroallergens in AD

- Reactivity increases with age (unlike foods)
- House dust mite, grass pollens, animal dander, molds, plant pollens (ragweed)
- Exposed areas of face, neck, arms, legs, chest
- Dust mite allergen avoidance may lead to improvement
- Immunotherapy not useful for AD treatment

Allergic contact dermatitis (ACD)

- Delayed type hypersensitivity response (IV)
- Skin contacts with haptens that activate antigen-specific T-cells in sensitized patients
- 13-25% of pediatric patients
- Results from repeated, prolonged contact
- As prevalent in AD patients as healthy children
  - Recent studies state higher (65%)

Allergic contact dermatitis (ACD)

- Well-circumscribed
- Itchy, red, papular, vesicular eruption
- Resolves with removal of cause
- Diagnosis (REFER)
  - Requires detailed history
  - Prolonged observation
  - Can confirm with **patch testing**—gold standard

Allergic contact dermatitis (ACD) in pediatric population

- Prevalence of (+) patch test in those referred for suspected ACD is 27-95%
- Consider in those not improving with traditional therapies, new areas
- Do barrier defects in AD enhance allergen penetration and thus sensitization?

ACD in children

- Top offenders
  - Nickel
  - Neomycin
  - Cobalt
  - Fragrance
  - Gold
  - Formaldehyde
  - Lanolin/wool alcohol
  - Thimerosal
  - Potassium dichromate
  - *Myroxylon pereirae*

Photo: R.C. Hansen

Consequences of pediatric AD

- Decreased QOL
- Disfigurement
- Less sleep for parents and patient
- Financial burden (loss of time from work, medications, emollients)
- Missed time from school/activities for child
- Behavioral disorders
  - *Higher rates of ADHD in moderate-severe AD*
  - *Depression, anxiety*
- Increased stress

Itch and AD

- “Itch that rashes”
- Cardinal symptom of AD
  - Sleep disturbance in 60%
  - Sleep disturbance 80% in acute flare
- Adversely impacts quality of life
- Treating AD properly can help control itch
- Consider sedating anti-histamine before bedtime
  - Hydroxyzine, doxepin (off-label)

New medications for AD: crisaborole 2% ointment

- Mild to moderate AD indication
- Steroid free alternative
- First topical treatment in over a decade
- Phosphodiesterase 4 inhibitor
- Age 2 and up
- Safe for face and groin

New medications for AD: crisaborole 2% ointment

- Down-sides:
  - Costly, access
  - Stings and burns
  - Takes a long time to see results, not a rescue
- Upsides:
  - Steroid free
  - No location restriction
  - Prolonged use OK
  - Pruritus relief

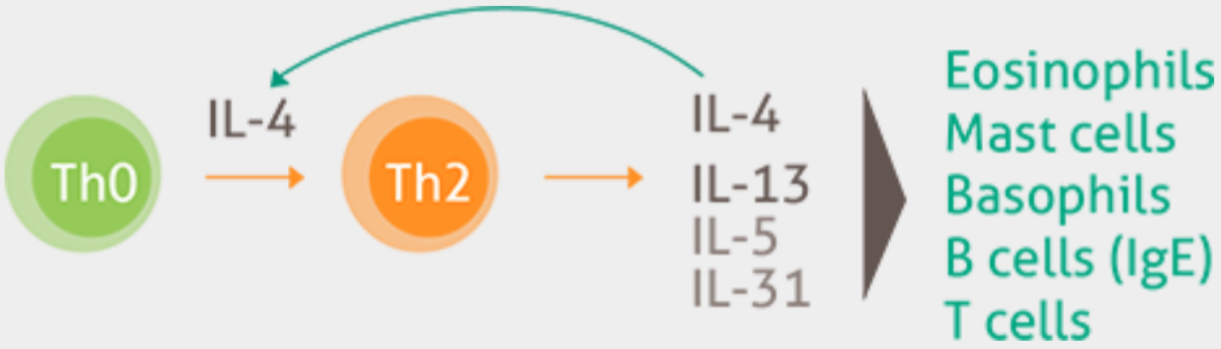
New medications for AD: dupilumab

- First biologic for AD, and for children with AD
- Ages 12 and up
- Uncontrolled moderate to severe AD
  - Candidates for systemic therapy
  - Patients that failed stronger TCSs
- NOT considered immunosuppressive, can be used with TCSs
- NO screening or routine bloodwork needed*
- Weight-based dosing, SQ injection

*per PI; screening labs should be ordered on discretion of provider

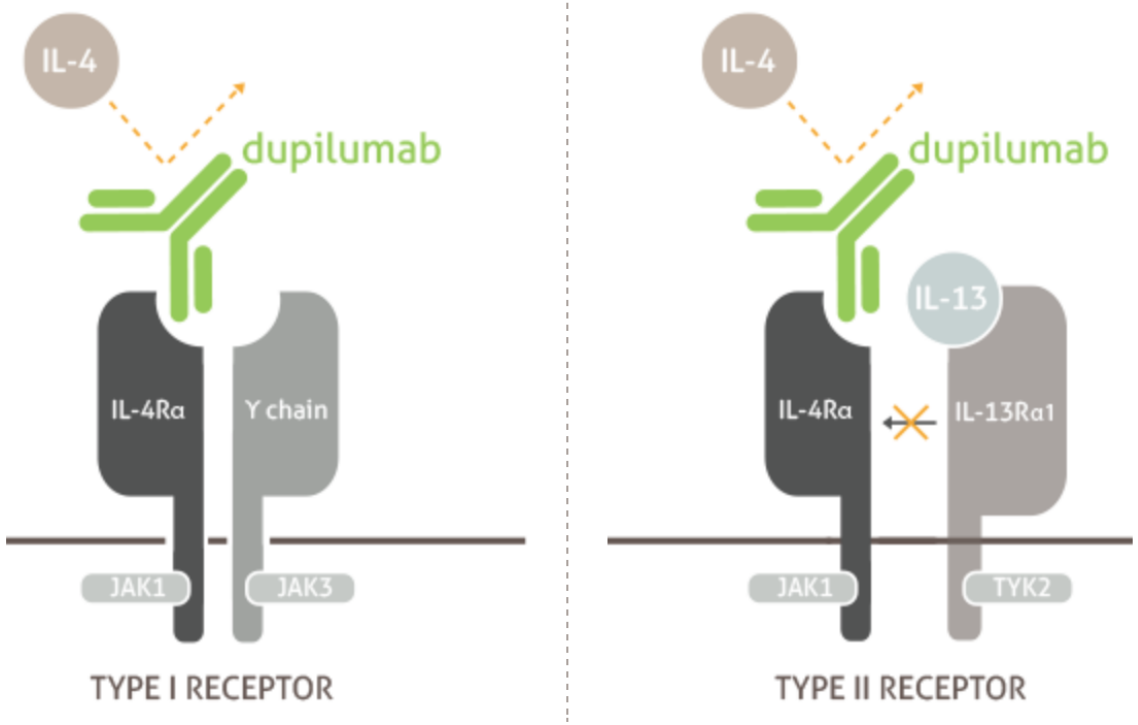
Specifically Targets a Source of Underlying Inflammation in Atopic Dermatitis

IL-4 and IL-13 are important in the development of Type 2 inflammation and its downstream effects.[1-3]



Multiple cell types that express IL-4Rα (eg, mast cells, eosinophils, macrophages, lymphocytes, epithelial cells) and inflammatory mediators (eg, histamine, eicosanoids, leukotrienes, cytokines, chemokines) are involved in Type 2 inflammation.[1]

DUPIXENT inhibits signaling of IL-4 and IL-13, Type 2 cytokines[1,2]



Inhibits IL-4 and IL-13 cytokine-induced responses, including the release of[1]:

- Proinflammatory cytokines and chemokines
- IgE

The first treatment of its kind to target IL-4 and IL-13 receptor signaling[1]

New medications for AD: dupilumab

- Side effects:
  - Conjunctivitis/keratitis
  - Injection site reaction
  - Hypersensitivity reactions (<1%)
- No increase in skin infections
- Treat pre-existing helminth infections
- Do not stop CSs for asthma
- No live vaccines (per PI)

AD Guidelines (2014)

- In 2013, round table to review evidence and discuss challenges
  - 4 part series in J Am Acad Dermatol
- Participants across a variety of disciplines, patient advocates
- Should be concise, severity-based, treatment algorithms
- Include: initial and ongoing evaluation, multidisciplinary input
- Provide guidance as to when to refer

Translating AD Guidelines into Practice

Making the diagnosis

TABLE 2 Diagnostic Criteria

Essential Features	Important Features	Associated Features
Both must be present	Add support to the diagnosis, observed in most cases of AD	Suggestive of AD, but too nonspecific to be used for defining or detecting AD in research or epidemiologic studies
1. Pruritus	1. Early age of onset	1. Atypical vascular responses (eg, facial pallor, white dermographism, delayed blanch response)
2. Eczema (acute, subacute, chronic)	2. Atopy	2. Keratosis pilaris/pityriasis alba/hyperlinear palms/ichthyosis
a. Typical morphology and age-specific patterns	a. Personal and/or family history	3. Ocular/periorbital changes
•Infants/children: facial, neck, and extensor involvement	b. IgE reactivity	4. Other regional findings (eg, perioral changes/periauricular lesions)
•Any age group: current or previous flexural lesions	3. Xerosis	5. Perifollicular accentuation/lichenification/prurigo lesions
•Sparing of the groin and axillary regions		
b. Chronic or relapsing history		
Exclusionary Conditions		
Diagnosis of AD depends on excluding conditions		
•Scabies	•Seborrheic dermatitis	•Photosensitivity dermatoses
•Psoriasis	•Contact dermatitis (irritant or allergic)	•Immune deficiency diseases
•Ichthyoses	•Cutaneous T-cell lymphoma	•Erythroderma of other causes

AD, atopic dermatitis; IgE, immunoglobulin E. Adapted from Eichenfield et al.[14]

IF diagnosis is unclear→REFER

Eichenfield L et al. Pediatrics 2015

Basic management

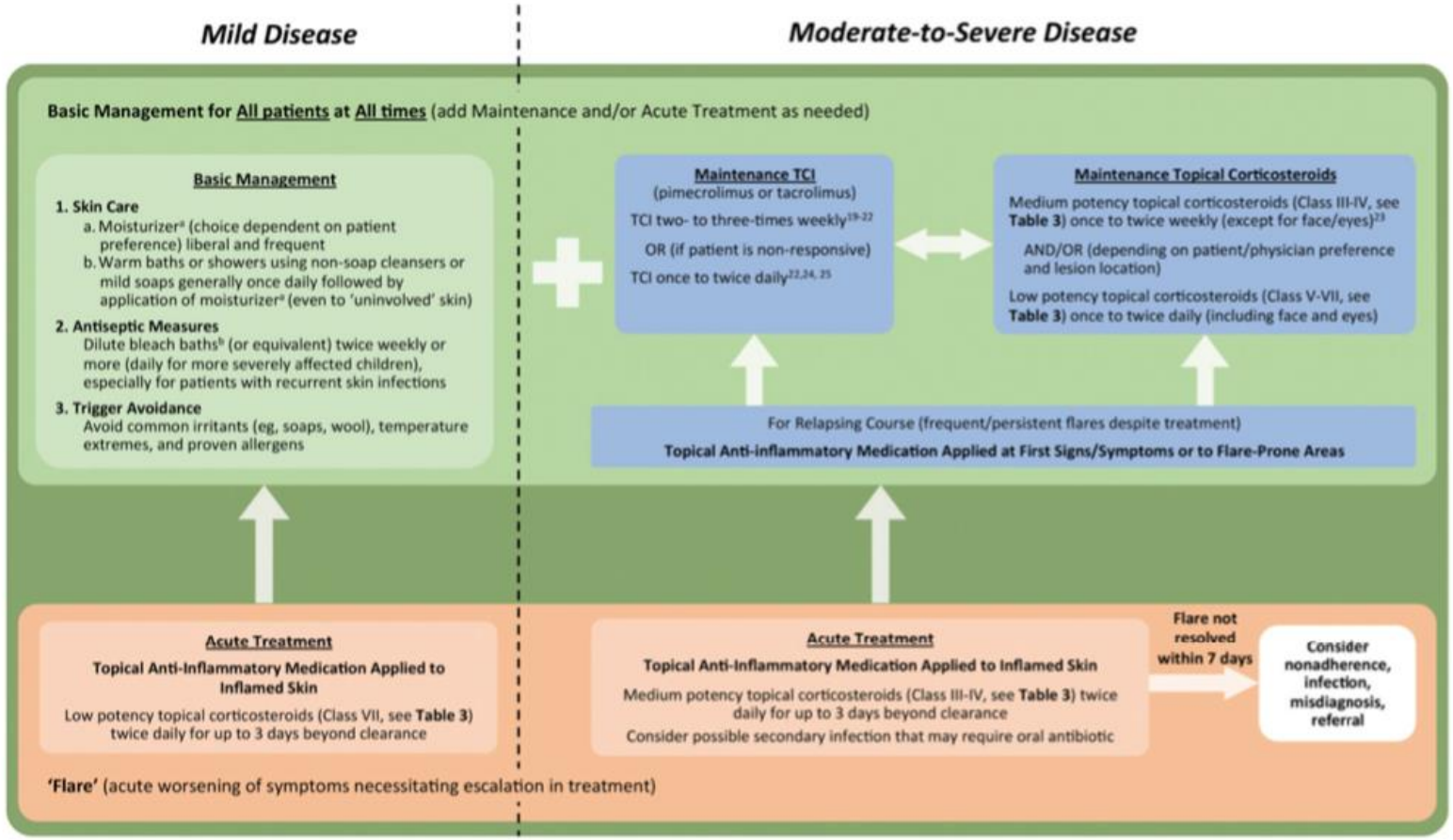


FIGURE 1

Proposed treatment model/eczema action plan for pediatricians and other primary care providers. [a]As tolerated during flare; direct use of moisturizers on inflamed skin may be poorly tolerated; however, bland petrolatum is often tolerated when skin is inflamed. [b]Approximately 0.5 cups sodium hypochlorite per 40 gallons of water/full bathtub or 1 mL/L. TCI, topical calcineurin inhibitor

Acute flare treatments

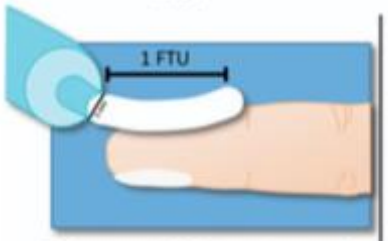
TABLE 3 Topical Corticosteroid Potencies, Strengths, and Formulations

Class	Strength, %	Available Formulations					
Drug		Ointment	Cream	Lotion	Foam	Solution	Gel
I. Very high potency							
Augmented betamethasone dipropionate	0.05	✓					
Clobetasol propionate	0.05	✓	✓		✓	✓	
Diflorasone diacetate	0.05	✓					
Halobetasol propionate	0.05	✓	✓				
II. High potency							
Amcinonide	0.1	✓	✓	✓			
Augmented betamethasone dipropionate	0.05		✓				
Betamethasone dipropionate	0.05	✓	✓		✓	✓	
Desoximetasone		0.25	0.25				0.05
Diflorasone diacetate	0.05		✓				
Fluocinonide	0.05	✓	✓			✓	✓
Halcinonide	0.1	✓	✓				
Mometasone furoate	0.1	✓					
Triamcinolone acetonide	0.5	✓	✓				
III–IV. Medium potency							
Betamethasone valerate	0.1	✓	✓	✓	✓		
Clocortolone pivalate	0.1		✓				
Desoximetasone	0.05		✓				
Fluocinolone acetonide	0.025	✓	✓				
Flurandrenolide	0.05	✓	✓				
Fluticasone propionate		0.005	0.05				
Mometasone furoate	0.1		✓				
Triamcinolone acetonide	0.1	✓	✓				
V. Lower-medium potency							
Hydrocortisone butyrate	0.1	✓	✓			✓	
Hydrocortisone probutate	0.1		✓				
Hydrocortisone valerate	0.2	✓	✓				
Prednicarbate	0.1		✓				
VI. Low potency							
Alclometasone dipropionate	0.05	✓	✓				
Desonide	0.05	✓	✓		✓		✓
Fluocinolone acetonide	0.01		✓			✓	
VII. Lowest potency							
Dexamethasone	0.1		✓				
Hydrocortisone	0.25, 0.5, 1	✓	✓	✓		✓	
Hydrocortisone acetate	0.5–1	✓	✓				

Includes representative examples and not all available agents. Adapted from Paller and Mancini.[18]

Patient/caregiver education

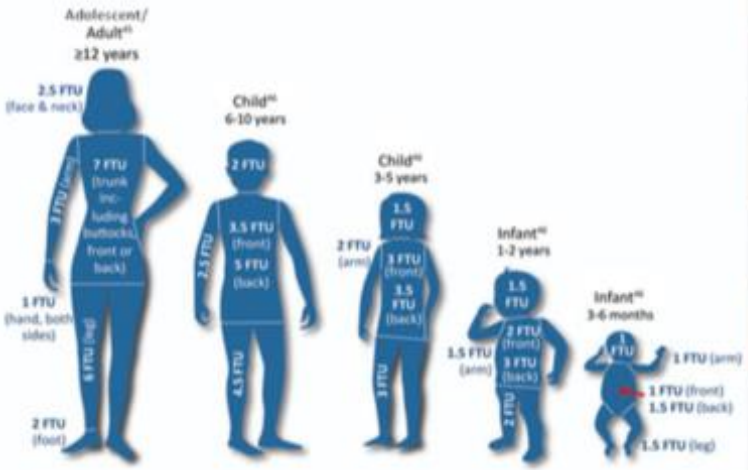
FTUs[45]



FTU = amount of ointment expressed from a tube with a 5-mm diameter nozzle measured from the distal skin crease to the tip of the palmar surface of an adult's index finger[a] (~0.5 g)

1 FTU = adequate amount of ointment for "thin and even" application to an area of skin equal to ~2 adult hands[a] (fingers together)

Quantity of Ointment per Application per Body Area by Age Group[a,b,c]



Weekly/Monthly Quantities by Age Group (Whole Body Application)[a]

Moisturizer[c]	Basic Management
Infant	100 g/week
Child	150–200 g/week[a]
Adolescent/Adult	500 g/week[a]

Ointment	Acute Treatment[45,46] 2-times daily	Maintenance Treatment 1-2–times weekly[d]	2-3–times weekly[d]	1-2–times daily[d]
Infant	60–100 g/week	10 g/month	15 g/month	75 g/month
Child	125–250 g/week	20 g/month	30 g/month	150 g/month
Adolescent/Adult	260–300 g/week	40–60 g/month	60–90 g/month	300–450 g/month

Cream[b]	Acute Treatment 2-times daily	Maintenance Treatment 1-2–times weekly	2-3–times weekly	1-2–times daily
Infant	66–110 g/week	15 g /month	20 g/month	100 g/month
Child	140–275 g/week	25 g/month	35 g/month	175 g/month
Adolescent/Adult	290–330 g/week	45–70 g/month	70–100 g/month	350–500 g/month

FIGURE 2

Topical application amounts. [a]Measurements/quantities are relative to adult hand/finger sizes, regardless of age group. [b]Quantity for creams should be increased by ~10% over ointment.[30] [c]Quantity for moisturizers may exceed the suggested values. [d]Estimated based on monthly amounts for 2 to 3 times weekly application per Ring et al.[7] FTU, fingertip unit.

Helpful tool box items:

- Acronyms for EHR for treatment plans
- Basic management queries in EHR
- Eczema “action plan”
- Handouts on bleach bathes, skin care items
- Recommended websites (instead of Dr. Google)

Support for patients &families

- National Eczema Association: nationaleczema.org
- All about bleach bathes: bleachbath.org
- Local support groups
- AAD/Camp Discovery: aad.org
- Society for Pediatric Dermatology: pedsderm.net
  - Handouts (free)
  - Find a pediatric dermatologist

- NEA: http://nationaleczema.org
- AD information from the Asthma and Allergy Foundation of America: http://www.aafa.org/display.cfm?id=9&sub=23&cont=325
- The Eczema Center at Rady Children's Hospital San Diego: http://eczemacenter.org
- Northwestern Multidisciplinary Eczema Center: http://eczema.nm.org
- The Pediatric Atopic Dermatitis Program at National Jewish Health: http://www.nationaljewish.org/programs/pediatric/atopic-dermatitis
- AD information from the AAD: http://www.aad.org/dermatology-a-to-z/diseases-and-treatments/a---d/atopic-dermatitis
- AD information from the National Institute of Arthritis and Musculoskeletal and Skin Diseases: http://www.niams.nih.gov/Health_Info/Atopic_Dermatitis

Thank you!

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