MODERN MANAGEMENT OF ATOPIC DERMATITIS: KEY PHARMACOLOGIC UPDATES

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LEARNING OBJECTIVES

1. Identify key immunologic pathways involved in atopic dermatitis and the corresponding pharmacologic targets of available therapies.
2. Evaluate the efficacy, safety, and appropriate clinical use of topical and systemic treatments for atopic dermatitis.
3. Implement evidence based pharmacologic treatment strategies for patients with atopic dermatitis based on disease severity, prior treatment response, and patient specific risk factors.

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CONFLICT OF INTEREST

- No conflicts of interest to disclose.

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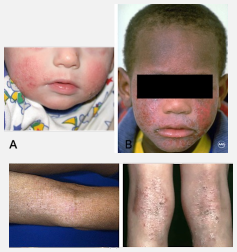
ATOPIC DERMATITIS: DEFINED

- Chronic, inflammatory skin condition
- Characterized by:

→Impaired skin barrier
→Immune dysregulation
→Intense pruritus
→Recurrent flares



(American Academy of Dermatology, 2026)

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ATOPIC DERMATITIS: CLINICAL HALLMARKS

- ***Pruritus
- Xerosis
- Acute

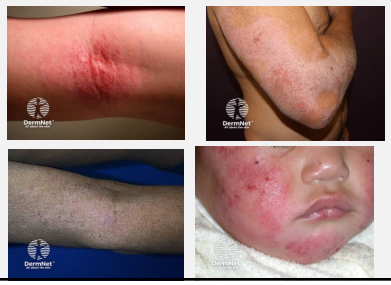
→Erythema, vesicles, ooze

- Chronic

→Lichenification, Excoriations, hyperpigmentation

- Diagnostic shortcut

→itch, flexural, chronic/relapsing history, personal/family history of atopy, widespread xerosis



(DermNet NZ, 2026)

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EPIDEMIOLOGY OF ATOPIC DERMATITIS

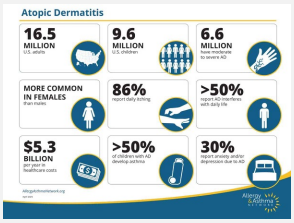
- Approximately 230 million sufferers world-wide

*the most common inflammatory skin disease

- Lifetime prevalence ~15%-

→especially in wealthier countries/urban areas

- All races can be affected; some races are more susceptible
- Atopic dermatitis often starts in infancy-but affects all ages.



(Allergy & Asthma Network, 2026)

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ATOPIC DERMATITIS: HEALTH IMPLICATIONS

- Psychological
→visible skin changes→stigma and low self esteem→social withdrawal
- Mental Health Risks
→Depression 20% prevalence, increased suicidal ideation, anxiety, OCD
- Biological Link "Brain-Skin Connection"
→proinflammatory cytokines→neurotransmitter imbalance, oxidative stress, reduced neurogenesis
- Sleep & Itch
→fatigue, poor concentration, mood disorders
- The "Atopic March"

(Courtney & Su, 2024)

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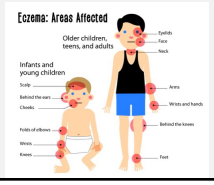
ATOPIC DERMATITIS: POPULATION IMPLICATIONS

CHILDREN AND ADOLESCENTS
- Bullying and stigma
- Poor self image
- Sleep problems→impaired school performance
- Links to ADHD and possibly autism

OLDER ADULTS
- Linked to cognitive impairment
→worse memory, attention, focus
- Chronic inflammation→increased risk of dementia and Alzheimer's



(Courtney & Su, 2024)

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ATOPIC DERMATITIS: ECONOMIC IMPLICATIONS

- High healthcare usage
- →71.7% sought healthcare in past 6 months; mean ~5.9 healthcare/acute care visits per month
- Work productivity loss
- →Absenteeism, reduced performance
- Out of pocket costs
- →~$200 per month USD per patient per month
- →comorbid disease burden increases costs even more
- Financial hardship
- →64.6% reported negative financial impact
- →24.5% reported significant or devastating impact

(Chovatiya et al., 2024)

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PATHOPHYSIOLOGY OF ATOPIC DERMATITIS

- Barrier Dysfunction
- Type 2 Inflammation
- Neuroimmune Itch
- Intracellular signaling

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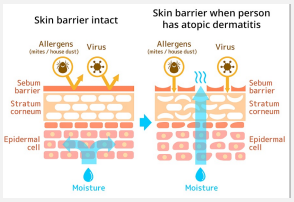
SKIN BARRIER DYSFUNCTION

- Genetic defects
→Filaggrin deficiency
- Structural defects
→Increased transepidermal water loss
→Reduced ceramide
→increased skin pH
- Barrier breakdown
→Enhanced allergen and microbial penetration
=immune activation



(Boothe et al., 2024)

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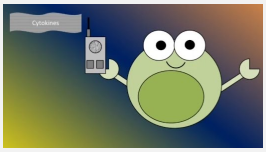
TYPE 2 INFLAMMATION

- Overactive allergy type immune system in the skin
- IL4 and IL13 increase inflammation, decrease barrier proteins, and increase IgE production
=Worsens eczema, keeps cycle going
- IL 13 directly stimulates nerves
=itch



(Boothe et al., 2024)

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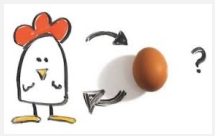
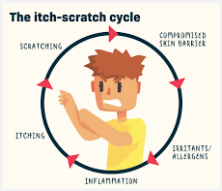
NEUROIMMUNE SIGNALING





(Boothe et al., 2024)

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INTRACELLULAR SIGNALING

- The response system inside the cell after the cytokine messenger binds

=IL4/IL13 binds to IL 4, IL31 binds to its receptor on nerves/skin cells

- Signal passed inside=JAK STAT pathway

=Receptor activates JAK enzymes→activates STAT proteins→STAT goes in nucleus→Genes change expression

- STAT in nucleus makes skin leakier and more allergic

(Boothe et al., 2024)

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DISEASE SEVERITY

- Body Surface Area (BSA)
- Eczema Area and Severity Index (EASI)
- Investigator Global Assessment (IGA)
- Body Surface Area (BSA)
- Patient Oriented Severity Score (POEM)
- Severity scoring of atopic dermatitis index (SCORAD)

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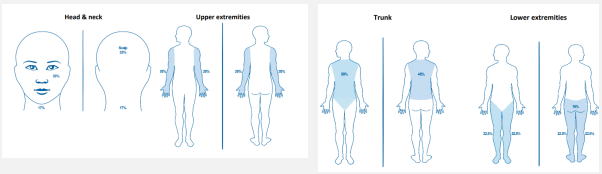
BODY SURFACE AREA



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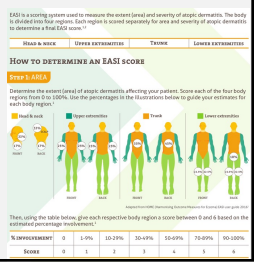
ECZEMA AREA AND SEVERITY INDEX (EASI)

- Clinician reported tool used to objectively manage severity and extent
- Focuses on physical exam findings (not symptoms)
- Gold standard in clinical trials

→Biologics use EASI-75; EASI-90 endpoints

- Gold standard in clinical trials



(American Academy of Family Physicians, 2026)

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INVESTIGATOR GLOBAL ASSESSMENT (IGA)

- Static ordinal scale
- Gestalt assessment of erythema, induration, papulation, lichenification, oozing/crusting
- Clinical trials

→primary endpoint often IGA 0 or 1 (>2 grade improvement)

Score	Morphological Description
0 – Clear	No inflammatory signs of atopic dermatitis (no erythema, no induration/papulation, no lichenification, no oozing/crusting). Post-inflammatory hyperpigmentation and/or hypopigmentation may be present.
1 – Almost clear	Barely perceptible erythema, barely perceptible induration/papulation, and/or minimal lichenification. No oozing or crusting.
2 – Mild	Slight but definite erythema (pink), slight but definite induration/papulation, and/or slight but definite lichenification. No oozing or crusting.
3 – Moderate	Clearly perceptible erythema (dull red), clearly perceptible induration/papulation, and/or clearly perceptible lichenification. Oozing and crusting may be present.
4 – Severe	Marked erythema (deep or bright red), marked induration/papulation, and/or marked lichenification. Disease is widespread in extent. Oozing or crusting may be present.

(Simpson et al., 2020)

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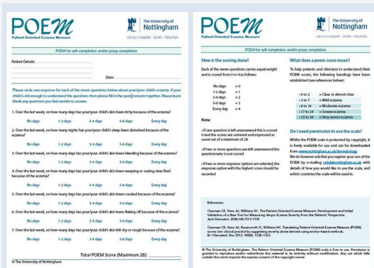
PATIENT ORIENTED ECZEMA MEASURE (POEM) SCORE



(Zhao & Murrell, 2019)

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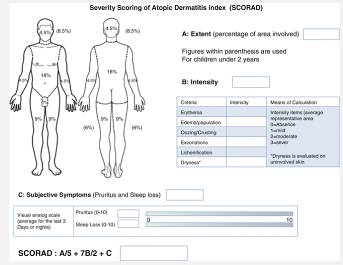
SEVERITY SCORING OF ATOPIC DERMATITIS INDEX (SCORAD)



Severity Scoring of Atopic Dermatitis index (SCORAD)

A: Extent (percentage of area involved)

Figures within parenthesis are used
For children under 2 years

B: Intensity

Criteria	Intensity	Means of Calculation
Erythema		Intensity items [average representative area 0=Absence 1=mild 2=moderate 3=sever *Dryness is evaluated on uninvolved skin
Edema/papulation		
Oozing/Crusting		
Excoriations		
Lichenification		
Dryness*		

C: Subjective Symptoms (Pruritus and Sleep loss)

Visual analog scale (average for the last 3 Days or nights)	Pruritus (0-10)	0 … 10
	Sleep Loss (0-10)	

SCORAD : A/5 + 7B/2 + C

(Humbert et al., 2017)

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STEPWISE THERAPY CONSIDERATIONS

- Affected areas of body/special sites
- Patient Age
- Disease severity

→Not everyone starts at step one, treat severity not the ladder.

→Escalate if not improved

- Safety
- Patient preference
- Insurance constraints

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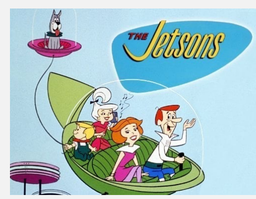
ATOPIC DERMATITIS PHARMACOLOGY: A JOURNEY THROUGH TIME



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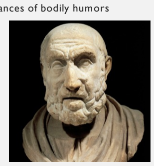
ANCIENT DESCRIPTIONS

HIPPOCRATES

- 460-370 BC
- Pruritic inflammatory skin disease
- Believed skin eruptions were related to imbalances of bodily humors



GALEN

- 129-216 AD
- Chronic, pruritic skin eruptions
- Recommended topical soothing preparations

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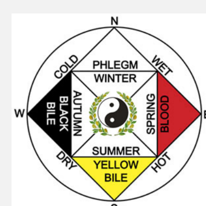
ANCIENT REMEDIES

- Theory of the four humors
- Therapy aimed to rebalance the body.
- Skin eruptions=body expelling excess humors



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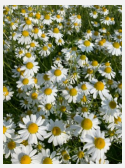
ANCIENT REMEDIES

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1800S

- "Eczema" becomes a medical diagnosis
- Ferdinand von Hebra (1816-1990)- one founder of modern dermatology
- Classified eczema as a distinct inflammatory skin disease

(Menter, 2000)

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1920S-1950S

- The concept of "Atopy" is coined-first time eczema linked to allergic predisposition by Dr. Fred Wise and Dr. Marion Sulzberger
- The concept of "Atopic Dermatitis" is coined-recognizing eczema occurring in allergic individuals as a distinct disease entity.
- Treatments helped symptoms but not underlying disease

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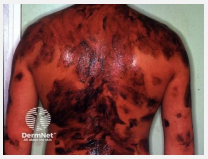
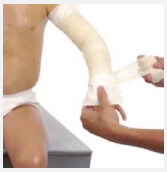
1920S-1950S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NON PHARMACOLOGIC THERAPY

- ***Moisturizers
- Wet wraps
- Bleach baths
- Phototherapy

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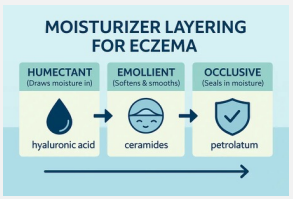
MOISTURIZER LAYERING FOR ECZEMA



- Moisturizers help replace lost lipids and water, improving barrier integrity
- Reducing Transepidermal Water Loss (TEWL)
- Form a protective occlusive layer on the skin surface
- Prevent excessive water evaporation → improves hydration and flexibility
- Help normalize the "brick-and-mortar" structure of the stratum corneum
- Decrease Inflammation & Itch
- Improved barrier reduces entry of irritants and allergens
- Leads to decreased immune activation and less itch-scratch cycles
- ***First line, enhances response to topical steroids and other anti-inflammatory treatments, reduces flares and prolongs remission

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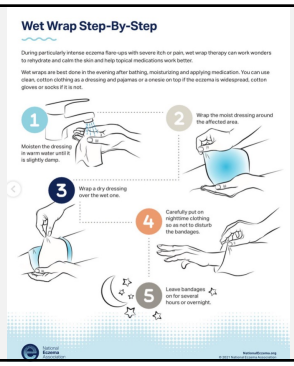

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PHOTOTHERAPY

Health care system factors
- Availability of phototherapy
- Ease of access
- Convenience and proximity to home and/or work
- Payer cost and insurance coverage
- Out-of-pocket costs, including copays, deductibles, transportation, lost work time, child care

Patient factors
- Skin type
- History of skin cancer
- History of photosensitive disorder and/or use of photosensitizing medications
- Fear of devices and treatment, particularly for children
- Location of AD lesions
- AD severity
- Baseline disease duration
- Persistence

(Boguniewicz et al, 2018)

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ATOPIC DERMATITIS PHARMACOLOGY: BACK TO THE FUTURE

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WHY DOES THIS MATTER?

- Therapies are now designed to target specific points along specific pathways.

→ restores skin barrier, blocks key cytokines, interrupting intracellular signaling through pathways (i.e. JAK signaling)

- Understanding where each drug works in the cascade = helping providers choose therapies that more precisely control inflammation, itch, and disease severity.
- Because we now understand these underlying pathways, modern treatments for atopic dermatitis are targeted to specific components of the disease process rather than broadly suppressing the immune system.

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TOPICAL TREATMENTS

- Topical corticosteroids
- Topical calcineurin inhibitors
- Topical PDE4 inhibitors
- Topical JAK inhibitors
- Topical AhR agonists

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1952-1959: TOPICAL CORTICOSTEROIDS

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Broad anti-inflammatory effect -reduces immune activation, vasoconstrictive, itch signaling	-binds intracellular glucocorticoid receptors -increases anti inflammatory proteins	-Mild to Moderate AD in all ages *with caution	-Local: skin atrophy, striae, telangiectasia, perioral dermatitis, steroid acne, (TSW?) -Systemic: HPA axis suppression, hyperglycemia	-Assess for skin thinning, discoloration, improper use	-BID -Itch Improved: 24-48 hours -Skin improved: 2-5 days -Max benefit: 1-2 wks

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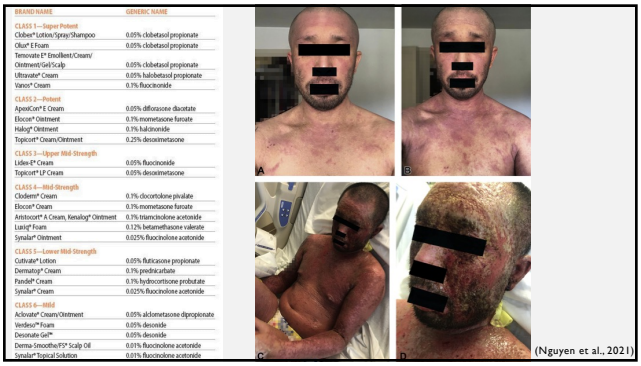

(Nguyen et al., 2021)

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2000-2001: TOPICAL CALCINEURIN INHIBITORS (PIMECROLIMUS/ELIDEL + TACROLIMUS/PROTOPIC)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-topical immunomodulators/ non steroidal anti inflammatory	Inhibit calcineurin, which blocks T-cell activation and reduces cytokine release (especially Th2-driven inflammation)	-Mild to moderate AD in ages 2 and up -Steroid-sparing, for sensitive areas, maintenance therapy	-Common: burning, stinging No skin atrophy - Black box warning: theoretical risk of increased cancer risk *not strongly supported by data	-No routine labs required -Clinical monitoring for irritation -Minimal systemic absorption	-BID -Itch improved: 2-3 days -Skin improved: 1-2 wks -Max benefit: 3-6 wks consistent use

(Astellas Pharma US, Inc., 2026)

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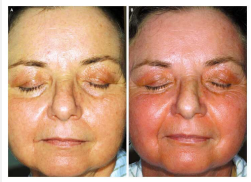

(Lubbe & Milingu, 2024)

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2016: TOPICAL PDE4 INHIBITOR CRISABAROLE (EUCRISA)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-PDE4 inhibition	PDE-4 inhibitor → increases cAMP in immune cells → reduces expression of IL-2, IL-4, IL-5, IL-13, TNF-α → reduces inflammation in skin	-Mild to AD in ages 3 months and up -Maintenance therapy to prevent flares in sensitive areas	-Common: mild burning/stinging -Non-steroidal, no skin atrophy -Minimal systemic absorption, well tolerated for long term use	-No routine labs required -Monitor for local irritation	-BID -Itch improved: 2-4 days -Skin improved: 1-2 wks -Max benefit: 3-4 wks

(Pfizer, 2026)

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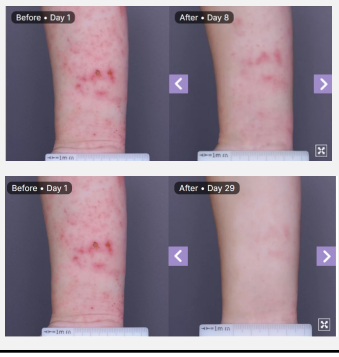

(Eucrisa, 2026)

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2021: RUXOLITINIB (OPZELURA)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Inhibition of the Janus kinase (JAK1/JAK2) signaling pathway	-Topical JAK1/JAK2 inhibitor → reduces transcription of pro-inflammatory cytokines (IL-4, IL-13, IL-31, IFN-γ) → decreases skin inflammation and itch	-Mild to moderate AD in patients ≥2 years -Suitable for sensitive areas -Non-steroidal, can be used short-term or intermittently for flares -used in vitiligo	-application site burning, stinging, folliculitis/acne - Minimal systemic absorption -Rare: mild headache, nasopharyngitis - Black box warning (malignancy, thrombosis, serious infections)	-monitoring for local irritation and response -Avoid excessive use over large BSA for prolonged periods (Max 60 g per wk)	-BID -Itch improved: 24-72 hr -Skin improved: 1-4 wks -Max benefit: 4-8 wk

(Incyte, 2026)

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(Incyte, 2026)

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2024: DELGOCITINIB (ZORYVE)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-PDE4 inhibitor	-PDE 4 inhibition→ increased cAMP→ Increased PKA signaling→ decreased NF-kB activity→ decreased inflammatory cytokines→ reduced inflammation	-Mild to moderate AD ≥2 years -Used for flares/ maintenance in sensitive areas -Seb Derm, Psoriasis	-application site reactions, nasopharyngitis -Rare: herpes zoster, local skin infections - Minimal systemic absorption	-no routine labs -monitoring for local irritation and response	-QD -Itch improved: 24-72 hr -Skin improved: 1-4 wks -Max benefit: 4-8 wk

(Arcutis, 2026)

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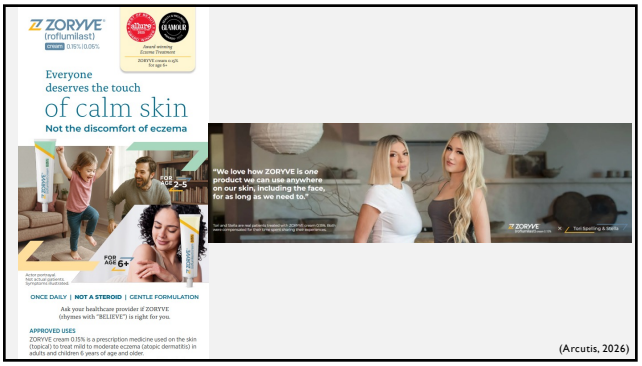

(Arcutis, 2026)

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2024: TAPINAROF (VTAMA)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Activates the aryl hydrocarbon receptor (AhR) pathway in skin cells (AhR Agonist)	-Topical AhR modulator → regulates gene expression involved in inflammation and skin barrier homeostasis → decreases pro-inflammatory cytokines and oxidative stress	-Mild to moderate AD in patients ≥2 years -used in psoriasis	-folliculitis, headache, application site reactions -Rare: hypersensitivity reactions – -Minimal systemic absorption	-no routine labs -monitoring for local irritation and response	-QD -Itch improved: 1 week -Skin improved: 4-8 wks -Max benefit: 3-6 mo

(Organon, 2026)

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(Organon, 2026)

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ORAL TREATMENTS

- Calcineurin Inhibitor (Cyclosporine)
- Antimetabolite/Folate Agonist (Methotrexate)
- Purine synthesis inhibitor (Azathioprine)
- Oral JAK inhibitors (Upatacitinib, Abrocitinib)

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CYCLOSPORINE

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-calcineurin inhibitor, immunosuppressant	-Rapid suppression of T-cell–driven inflammation, decreased cytokines (IL-2, IL-4, IL-13 indirectly), strong anti-flare effect	-severe AD (ages 2 and up) -severe acute flares/ rapid rescue therapy -bridge therapy before biologic -short term therapy	-hypertension, nephrotoxicity, tremors, hypertrichosis, electrolyte imbalances	-prior: blood pressure, CBC/CMP, lipid panel -creatinine + blood pressure every 1-2 wks, then monthly labs for 2-3 months, if using longer every 4-8 weeks	-PO 2.5-5 mg/kg/day divided twice daily -adjust based on BP/kidney function -onset of action 1-2 wks (sometimes days)

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METHOTREXATE

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Antimetabolite folate antagonist; immunomodulator	-Inhibits DHFR → decreases DNA synthesis in rapidly dividing cells; increases extracellular adenosine -decreased T-cell activation, cytokines (IL-4, IL-13, IL-31 indirectly), eosinophilic inflammation, skin immune hyperreactivity	-moderate to severe AD (ages 2 and up) -refractory cases -long term control if cost or access limits use	-nausea, fatigue, mouth ulcers, hair thinning, transaminitis/ hepatotoxicity, bone marrow suppression, lung toxicity, opportunistic infections -NO pregnancy, severe liver disease, immunodeficiency, active serious infection	-prior: CBC/CMP, Hepatitis panel, TB screening, pregnancy test -CBC + LFTs q 2-4 weeks, then every 1-3 months	-PO 7.5-15 mg once a week -onset of action 4-8 weeks -folic acid 1 mg daily reduces toxicity

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AZATHIOPRINE

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-purine synthesis inhibitor	Broad suppression of T-cell and B-cell proliferation, decreases Th2-driven inflammation, decreases IgE-associated immune activity	-moderate to severe AD -chronic relapsing disease -alternative to cyclosporine intolerance	-nausea, GI upset, fatigue, transaminitis, bone marrow suppression, pancreatitis, increased infection risk, increased malignancy risk long term	-prior: CBC/CMP, TPMT/NUDT15 enzyme, pregnancy test -first 4-8 weeks CBC/LFTs every 1-2 wks, months 2-3 every 2-4 wks, long term stable every 1-3 months	-PO 1-3 mg/kg/day, adjust based on response -slow onset of action (4-12 wks) =long term maintenance possible with monitoring

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2022: UPATACITINIB (RINVOQ)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Selective JAK 1 inhibitor	-Blocks JAK-STAT signaling → decreases cytokine signaling (IL-4, IL-13, IL-31, IFN, etc.)	-moderate to severe AD in patients ≥12 years -alternative to biologic -fast relief	-acne, URIs, headache, nausea -increased cholesterol, decreased neutrophil, transaminitis -black box warning- infectious, thrombosis, CV events, malignancy risk	-Baseline CBC/CMP,TB Screening, hepatitis screening Then again 4-12 wks after start -Monitor for infections, thrombotic events, CV risk factors	-PO 15-30 mg daily based on response -Itch improved: 24-72 hrs -Skin improved: 2-4 wks -Max benefit: 12 wks

(Abbvie, 2026)

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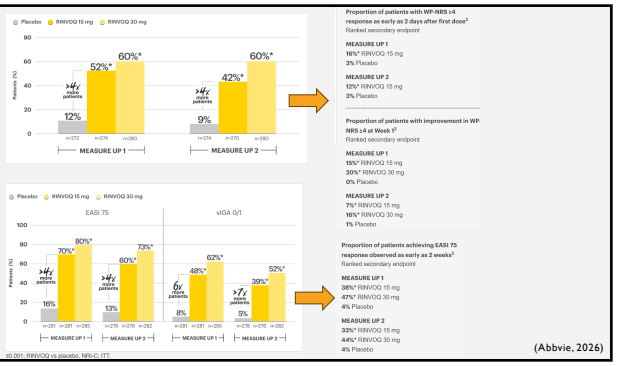

(Abbvie, 2026)

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2022: ABROCITINIB (CIBINQO)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Selective JAK 1 Inhibitor	Inhibits JAK1 → blocks JAK-STAT signaling → decreases cytokine signaling (IL-4, IL-13, IL-31, IFN)	-moderate to severe AD in patients ≥12 years -alterative to biologic -patients with high itch burden	-nausea, headache, acne, nasopharyngitis -infection risk (herpes zoster, bacterial infections) -Lab abnormalities -Black box warning	-Baseline and periodic CBC, liver function tests, lipids – -Monitor for infections, thrombotic events, CV risk factors	-PO 100-200 mg daily based on response -Itch improved: 24-48 hrs -Skin improved: 2-4 wks -Max benefit: 12 wks

(Pfizer, 2026)

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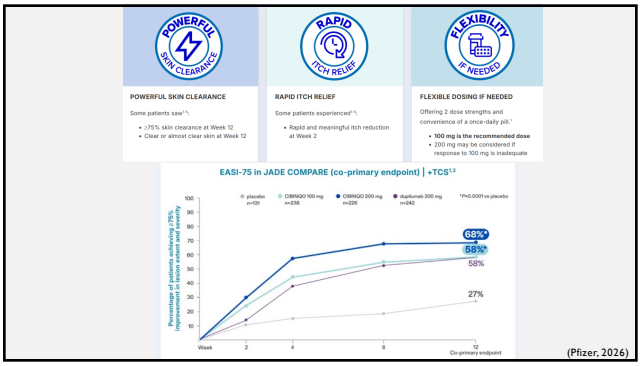

(Pfizer, 2026)

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INJECTABLE TREATMENTS/ BIOLOGICS

- IL4 receptor alpha antagonist (Dupilumab)
- IL13 antagonist (Tralokinumab)
- IL 13 antagonist (Lebrikizumab)
- IL 31 receptor antagonist (Nemolizumab)

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2017: DUPILUMAB (DUPIXENT)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-IL 4 receptor alpha antagonist/ monoclonal	-inhibits IL-4, IL 13 receptor signaling -reduces Th2 cytokine mediated inflammation + IgE production	-moderate to severe AD in children 6 months & above, adults -other uses in prurigo nodularis, asthma, chronic rhinosinusitis, nasal polyps	-injection site reactions -conjunctivitis, blepharitis, keratitis -rare hypersensitivity reaction, urticaria, -eosinophilia (transient), flu, headache	-no routine lab monitoring required -no known drug interactions -ophthal referral if persistent eye symptoms	-Subq inj loading then q 2 wks -Itch improved: 3-7 days -Skin improved: 2-8 wks -Max benefit: 16 wks

(Sanofi and Regeneron Pharmaceuticals, Inc., 2026)

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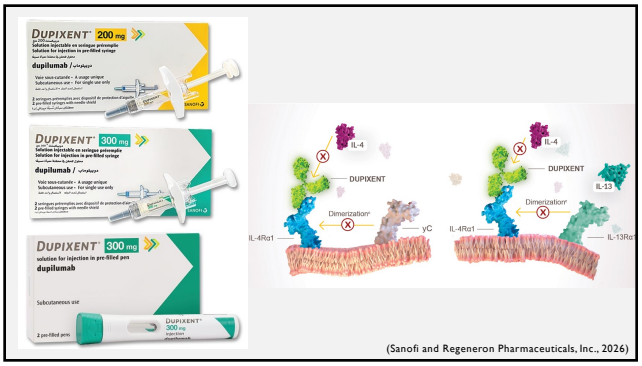

(Sanofi and Regeneron Pharmaceuticals, Inc., 2026)

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2021: TRALOKINUMAB (ADBRY)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-IL 13 antagonist -monoclonal antibody	-blocks IL13 signaling pathway → reduces downstream pro inflammatory cytokine signaling (ie Th2) → decreases downstream inflammatory signaling and skin inflammation	-Mild to moderate AD in patients ≥12 years	-injection site reactions, URI, headache -conjunctivitis/ keratitis -fatigue, arthralgia, hypersensitivity, eosinophilia, infections ie herpes simplex	-no lab monitoring -monitor for efficacy -monitor for eye symptoms	-subq inj -Itch improved: 2-4 wks -Skin improved: 4-8 wks -Max benefit: 4 months

(LEO Pharma Inc, 2026)

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(LEO Pharma Inc, 2026)

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2024: LEBROKIZUMAB (EBGLYSS)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-Selective inhibition of IL 13→ reduces Th2-mediated inflammation -monoclonal antibody	-binds IL-13 → prevents IL-13 from interacting with its receptor → decreases downstream STAT6 phosphorylation	-Mild to moderate AD in patients ≥12 years	-injection site reactions, URI, headache, viral infections -Rare: hypersensitivity reactions -Minimal systemic monitoring required	-no lab monitoring -monitor for efficacy -monitor for eye symptoms	-subq inj -Itch improved: 2-4 wks -Skin improved: 4-8 wks -Max benefit: 4 months

(Eli Lilly and Company, 2026)

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(Eli Lilly and Company, 2026)

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2024: NEMOLIZUMAB (NEMLUVIO)

Pathway	Mechanism	Indications	Safety	Monitoring	Application + Use
-IL 31 receptor antagonist -monoclonal antibody	-binds IL-31 receptor alpha → prevents IL-31 from activating sensory neurons and inflammatory pathways, reducing Th2 inflammation	-moderate to severe AD in patients ≥12 years -used for prurigo nodularis	-headache, nasopharyngitis, joint pain, hives, nummular eczema, muscle aches -Rare: hypersensitivity reactions	-Observe for injection site reactions -Monitor for signs of infection -Assess clinical response and improvement	-subq inj -Itch improved: 2-4 wks -Skin improved: 4-8 wks -Max benefit: 16 wks

(Galderma, 2026)

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(Galderma, 2026)

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CASE-BASED TREATMENT APPROACH

- Mild disease: topical therapies
- Moderate disease: advanced topicals or biologics
- Severe disease: systemic agents

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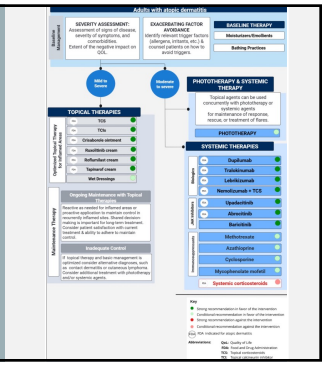
JAAD Journal of the American Academy of Dermatology

FOCUSED UPDATE: GUIDELINES OF CARE FOR THE MANAGEMENT OF ATOPIC DERMATITIS IN ADULTS

(Dawn et al., 2025)



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ADULT AD GUIDELINES: CORE CLINICAL THEMES

- AD is a targetable immune disease, treatment depends on itch severity, immune phenotype, disease burden

→emphasizes type 2 inflammation (IL 4, IL 13, IL 31 pathways)

- Itch is a primary therapeutic endpoint
- Stepwise but earlier escalation to targeted therapies

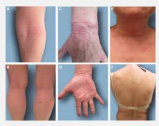
→emollients→ topical steroids→ nonsteroidal topicals→ systemic therapy

- Steroid sparing emphasis
- Evidence limitations acknowledged

→trials are short duration or limited head to head comparisons; long term safety and durability remain uncertain

- Strong Recommendations

→Tapinarof, Roflumilast, Lebrikizumab, Nemolizumab



(Dawn et al., 2025)

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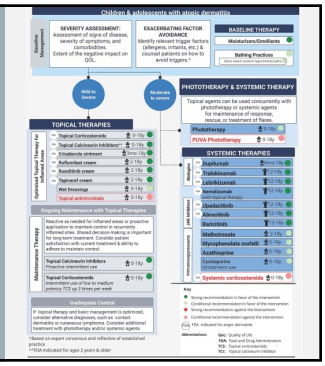
JAAD Journal of the American Academy of Dermatology

GUIDELINES OF CARE FOR THE MANAGEMENT OF ATOPIC DERMATITIS IN PEDIATRIC PATIENTS

(Davis et al., 2026)



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PEDIATRIC AD GUIDELINES: CORE CLINICAL THEMES

- AD is a distinct disease population; emphasizes age-appropriate considerations

→skin barrier immaturity, growth/development concerns, caregiver adherence

- Moisturizers are first line and essential core therapy (not adjunctive)

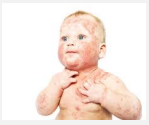
→dietary changes, probiotics, vitamins not recommended

- Stepwise severity based treatments

→emollients→ topical steroids→ nonsteroidal topicals→ systemic therapy

- Shift towards targeted immunologic therapy, recognition of type 2 inflammation
- Steroid stewardship

→Appropriate potency, avoid overuse, avoid systemic corticosteroids



(Dawn et al., 2025)

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PEDIATRIC AD GUIDELINES: CORE CLINICAL THEMES

- Adjunctive therapies

→Bathing routines, bleach baths, wet wrap therapy, phototherapy, topical antimicrobials

- Comorbidity awareness and holistic care

→Atopic diseases, psychosocial burden, screening awareness, multidisciplinary care when needed

- Patient-caregiver-clinician partnership

→adherence strategies, shared decision making

(Dawn et al., 2025)

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SPECIAL POPULATIONS

- Pediatric patients

→Skin barrier is more sensitive and permeable , Higher risk of systemic absorption, growth, neurodevelopment, long-term safety matter ,Adherence depends on caregiver burden, some treatments have age restrictions

- Pregnant patients

→balance maternal control vs fetal safety, prefer topical and lowest systemic exposure, limited high quality RCT data-guideline based conservative approach

- →steroids, TCIs, PDE4 (w/ caution)→Dupixent or cyclosporine if severe; NbUVB
- Elderly patients

→thin fragile skin higher steroid atrophy, polypharmacy, higher infection risk, CV disease, malignancy history, xerosis is dominant feature

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KEY TAKEAWAYS

- • AD is driven by barrier dysfunction, Type 2 inflammation/chronic immune dysregulation, neuroimmune itch, and intracellular signaling
- Treatment is always stepwise and individualized
- Targeted therapies have improved outcomes. Mechanism matters in clinical outcomes!
- →IL4/IL13 blockage=stronger skin clearance, IL31 inhibition=rapid itch relief, JAK inhibition=broad, fast anti inflammatory effects

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THANK YOU! QUESTIONS?

- Email: joannehurtsky@email.com

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REFERENCES

- Allergy & Asthma Network. (2026). *Eczema statistics*. https://allergyasthmanetwork.org/what-is-eczema/eczema-statistics/
- AbbVie Inc. (2025). *Rinvoq (upadacitinib) prescribing information*. Rinvoq prescribing information.
- American Academy of Dermatology Association. (2026). *Atopic dermatitis: Overview*. https://www.aad.org/public/diseases/eczema/types/atopic-dermatitis
- American Academy of Family Physicians. (2026). *Eczema area and severity index (EASI)*. World EMR. https://worldemr.org/download/eczema-area-and-severity-index/
- Astellas Pharma US, Inc. (2026). *Protopic (tacrolimus) ointment prescribing information*. https://mc-df05ef79-e68e-4c65-8ea2-953494-cdn-endpoint.azureedge.net/-/media/corporatecommunications/us/therapeutic-expertise/our-product/protopicpi.pdf
- Arcutis Biotherapeutics, Inc. (2026). *ZORYVE (roflumilast) cream: Prescribing information*. https://www.arcutis.com/wp-content/uploads/USPI-roflumilast-cream.pdf
- Boguniewicz, M., Fonacier, L., Guttman-Yassky, E., Ong, P.Y., Silverberg, J., Farrar, J. R., & Simpson, E. L. (2018). Atopic dermatitis yardstick: Practical recommendations for an evolving therapeutic landscape. *Annals of Allergy, Asthma & Immunology*, 120(1), 10–22.e2.
- Boothe, W. D., Tarbox, J. A., & Tarbox, M. B. (2024). *Atopic dermatitis: Pathophysiology*. In *Advances in Experimental Medicine and Biology*. https://doi.org/10.1007/978-3-031-54513-9_3
- Chovatiya, R., Begolka, W. S., Thibau, I. J., & Silverberg, J. I. (2022). *Impact and associations of atopic dermatitis out-of-pocket health care expenses in the United States*. Dermatitis, 33(6 Suppl), S7. https://doi.org/10.1097/DER.0000000000000795
- Courtney, A., & Su, J. C. (2024). *The psychology of atopic dermatitis*. *Journal of Clinical Medicine, 13*(6), 1602. https://doi.org/10.3390/jcm13061602

75

REFERENCES

- Davis, D. M. R., Alikhan, A., Bercovitch, L., Singh, A. M., Wu, P. A., Sidbury, R., *et al.* (2026). *Guidelines of care for the management of atopic dermatitis in pediatric patients*. *Journal of the American Academy of Dermatology*. Advance online publication.
- DermNet. (2026). *Atopic dermatitis*. https://dermnetnz.org/topics/atopic-dermatitis
- Eli Lilly and Company. (2026). *EBGLYSS® (lebrikizumab-lbkz) clinical data*. Retrieved from https://ebglyss.lilly.com/hcp/clinical-data
- Galderma Laboratories, L.P. (2026). *NEMLUVIO® (nemolizumab-ilto) clinical trial results for eczema (atopic dermatitis)*. Retrieved from https://www.nemluvio.com/eczema/about-nemluvio/nemluvio-results
- Humbert, P., Fanian, F., Maibach, H. I., & Agache, P. (Eds.). (2017). *Agache's measuring the skin: Non-invasive investigations, physiology, normal constants* (2nd ed.). Springer. https://doi.org/10.1007/978-3-319-32383-1
- Incyte Corporation. (2026). *Opzelura (ruxolitinib) prescribing information*. https://www.opzelura.com/opzelura-prescribing-information
- LEO Pharma Inc. (2026). *ADBRY® (tralokinumab-ldrm) autoinjector: Dosage and administration for adults*. AdbryHCP. Retrieved from https://www.adbryhcp.com/dosage-and-administration/adult/autoinjector
- Lübbe, J., & Milingou, M. (2004). *Tacrolimus ointment, alcohol, and facial flushing*. *The New England Journal of Medicine, 351*(26), 2740. https://doi.org/10.1056/NEJMicm040139
- Nguyen, A. P., Kong, J. S., & Teuber, S. S. (2021). *Severe topical corticosteroid withdrawal syndrome from over-the-counter steroids*. *The Journal of Allergy and Clinical Immunology: In Practice*, 9(11), 4147–4148. https://doi.org/10.1016/j.jaip.2021.08.015
- Organon & Co. (2026). *Atopic dermatitis resources*. VTAMA® (tapinarof) cream, 1%. https://vtamahcp.com/atopic-dermatitis/resources/

76

REFERENCES

- Pfizer Inc. (2026). *CIBINQO® (abrocitinib) efficacy: Skin clearance pivotal trial data (EASI-75)*. PfizerPro. Retrieved from https://cibinqo.pfizerpro.com/efficacy/skin-clearance-pivotal-trial-data#tabpanel-easi75
- Pfizer Inc. (2016). *EUCRISA® (crisaborole) ointment: Professional information*. https://eucrisa.pfizerpro.com/
- Sanofi and Regeneron Pharmaceuticals, Inc. (2026). *DUPIXENT® (dupilumab) mechanism of action in atopic dermatitis*. DupixentHCP. Retrieved from https://www.dupixenthcp.com/atopicdermatitis/about/mechanism-of-action
- Simpson, E. L., Bissonnette, R., Eichenfield, L. F., Guttman-Yassky, E., King, B., Silverberg, J. I., Beck, L. A., Bieber, T., Reich, K., Kabashima, K., Seyger, M., Siegfried, E., Stingl, G., Feldman, S. R., Menter, A., van de Kerkhof, P., Yosipovitch, G., Paul, C., Martel, P., Dubost-Brama, A., & Paller, A. S. (2020). *The validated Investigator Global Assessment for atopic dermatitis (vIGA-AD): The development and reliability testing of a novel clinical outcome measurement instrument for the severity of atopic dermatitis*. *Journal of the American Academy of Dermatology*. https://doi.org/10.1016/j.jaad.2020.04.104
- Zhao, C. Y., Murrell, D.F. (2019). Atopic Dermatitis Outcome Measures. In: Maibach, H., Osman, N. (eds) Cutaneous Biometrics. Springer, Cham. https://doi.org/10.1007/978-3-319-56591-0_49-2

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