

Atopic Dermatitis

Atopic dermatitis affects approximately 10% of the U.S. population and is more common in children than adults. Up to 99% of physician visits for atopic dermatitis are in primary care. Most cases can be managed successfully with topical treatments, including moisturizers and prescription anti-inflammatory treatments, such as corticosteroids, calcineurin inhibitors, phosphodiesterase-4 inhibitors, Janus kinase (JAK) inhibitors, and aryl hydrocarbon receptor agonists. For more refractory or severe atopic dermatitis, ultraviolet phototherapy and systemic treatments, usually prescribed by specialists, can be used. Systemic treatments include older off-label immunomodulators, such as methotrexate. Since 2017, multiple on-label injectable biologics and oral JAK inhibitors have been approved.

Epidemiology

Diagnosis

Treatment

Practice Improvement

CME/MOC activity available at [Annals.org](https://annals.org).

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Atopic dermatitis is a chronic inflammatory skin condition characterized by itch and erythematous skin lesions. Atopic dermatitis is burdensome at the individual and population levels (1) and is associated with the most disability among skin conditions globally (2). Itch associated with atopic dermatitis can be debilitating, leading to decreased quality of life and sleep loss, and the visible rash can lead to embarrassment and stigma. In addition, treatments, even when effective, can contribute to the burden of disease (3). The risk for developing atopic dermatitis is

driven by an interplay between genetic and environmental factors, the latter of which are poorly understood (4). Genetic and environmental factors lead to atopic dermatitis by 2 broad mechanisms: skin barrier disruption and type 2 helper cell inflammation (5). Other factors, such as aberrant skin microbiota and neuroimmune interaction, also contribute to pathogenesis (6, 7). Treatments of atopic dermatitis, including topical and systemic treatments, target these pathogenic actors to restore the skin barrier and reduce itch and inflammation.

Epidemiology

What is atopic dermatitis?

Atopic dermatitis is an itchy skin condition characterized by inflamed rashes. It has a fluctuating course, with unpredictable improvement and worsening and remission and relapse over time. The itch and rashes of atopic dermatitis can involve any part of the skin surface but are commonly flexural, particularly in children. The lesions are morphologically referred to as “eczematous,” with erythematous and edematous papules and plaques that are poorly circumscribed (Figure 1, top). This contrasts with psoriasis, which is typified by well-circumscribed scaly erythematous plaques (Figure 1, bottom) that most commonly affect the extensor surfaces (the elbows and knees).

Atopic dermatitis is commonly referred to as “eczema,” but this is a nonspecific term that is synonymous with “dermatitis.” “Eczema” or “dermatitis” can be used to refer to various clinical entities, including atopic dermatitis (atopic eczema), which is the focus of this article, and other conditions such as allergic contact dermatitis, irritant contact dermatitis, and nummular dermatitis.

Atopic dermatitis is the most common chronic skin disease worldwide. Prevalence estimates vary widely across countries and depend on the methods used for case ascertainment (8). The

Global Burden of Disease Study estimated the global prevalence to be 2% in 2021 (9), whereas national surveys in the United States have reported prevalence of 13% in children (10) and 7% in adults (11). Although often viewed as predominantly a disease of childhood, atopic dermatitis commonly persists into adulthood and can begin at any age.

Primary care data from 604 333 persons aged 0 to 99 years in the U.K. Health Improvement Network between 1994 and 2013 suggest there is a second peak in the incidence of atopic dermatitis after age 65 years, with a mean prevalence of 8.7% in those aged 75 to 99 years compared with 5.1% in those aged 18 to 74 years (12).

Although atopic dermatitis is not life-threatening, it can be debilitating. Chronic, persistent itch leads to decreased quality of life (1). Sleep disturbances are common, likely related to nocturnal itch (13). Atopic dermatitis has been associated with increased school absenteeism among children and decreased work productivity among adults (14, 15). The skin of most people with atopic dermatitis is colonized by *Staphylococcus aureus*, with colonization rates of 70% in lesional skin and 39% in nonlesional skin, with rates increasing with atopic dermatitis severity (16). Most

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Figure 1. Representative photos of eczematous (top) and psoriasiform (bottom) morphology.



instances of colonization do not represent clinical infections, but secondary *S aureus* skin infections are common in atopic dermatitis, and this can on rare occasions lead to bacteremia and sepsis (17).

What are triggers and risk factors for atopic dermatitis?

Atopic dermatitis has a strong genetic component. A study of twins from Denmark estimated that genetic factors contributed 82% of the individual susceptibility to atopic dermatitis (4). These genetic factors commonly relate to proteins that are important for either skin barrier or type 2 helper lymphocyte (type 2 or Th2) inflammation and immunity (18). A personal or family history of atopy also predicts more persistent

atopic dermatitis phenotypes from childhood (19).

Environmental risk factors for atopic dermatitis are less well understood. The “hygiene hypothesis” postulates that exposure to pathogens early in life reduces the risk for atopic dermatitis, but evidence for this is inconclusive. Studies have reported inverse associations with “dirty” conditions, such as early-life animal exposure, and positive associations with “clean” conditions, such as early-life antibiotic exposure (20). Pollution and climatic factors, including extreme heat and cold, are associated with increased atopic dermatitis prevalence and flares (21). Skin barrier disruption, either from genetic abnormalities or external assaults, and

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type 2 inflammation, either initiated by a genetic predisposition or in response to allergens or irritants, create a feedback loop. Epidermal barrier disruption leads to type 2 inflammation, and type 2 inflammation leads to impaired epidermal structure and function. Treatments of atopic dermatitis target these 2 components, and treatments targeting one also improve the other (22, 23).

What comorbid diseases are associated with atopic dermatitis?

Atopic dermatitis is strongly associated with other so-called atopic or allergic conditions whose pathogenesis also includes type 2 inflammation, including asthma, allergic rhinitis (hay fever), and food allergies (24).

A meta-analysis supporting recent American Academy of Dermatology (AAD) guidelines found that asthma was prevalent in 25% of adults with atopic dermatitis, with the association increasing as atopic dermatitis severity increased (24).

Atopic dermatitis is strongly associated with mental health conditions, such as depression, anxiety, and attention deficit-hyperactivity disorder (24). Although it is unclear what proportion of

depressive disorders seen in people with atopic dermatitis are directly related to quality of life and other effects of atopic dermatitis itself, studies have shown that treatment of atopic dermatitis improves depressive symptoms along with the signs and symptoms of atopic dermatitis (25).

Many other medical conditions have been associated with atopic dermatitis, including autoimmune conditions, bone fractures, components of the metabolic syndrome, and cardiovascular disease (24). Clinicians should be aware of these associations as they may have relevance for treatment choices. For example, comorbid alopecia areata, an autoimmune condition, could be treated with oral Janus kinase (JAK) inhibitors prescribed for atopic dermatitis (26). Although the role of corticosteroids used to treat atopic dermatitis in fracture risk is unclear (27), data from the United Kingdom and Canada suggest that people prescribed oral corticosteroids in discontinuous bursts, as is often the case when oral corticosteroids are inappropriately prescribed for atopic dermatitis, have low rates of guideline-recommended fracture prevention (28).

Epidemiology... Atopic dermatitis is very common and burdensome in the general population. Although it is more common in children, it is still common in adults, either as persistent or relapsed disease from childhood or as new-onset disease later in life. The risk for atopic dermatitis is primarily genetic, with the influence of environmental factors being less clear. Genetic and environmental factors contribute to skin barrier defects and type 2 inflammation, which are the primary pathogenic factors of atopic dermatitis. Atopic dermatitis is commonly associated with other conditions associated with type 2 inflammation, including asthma.

CLINICAL BOTTOM LINE

Diagnosis

What symptoms and physical examination findings are important in the evaluation of patients with suspected atopic dermatitis?

Atopic dermatitis is diagnosed on the basis of clinical features. Multiple sets of formal diagnostic criteria are available but

are usually unnecessary for day-to-day clinical use. However, such criteria are helpful to understand the most important clinical features of atopic dermatitis and as a reference when the diagnosis is in doubt. The U.K. Working Party criteria require an itchy skin condition as a major

criterion for the diagnosis of atopic dermatitis (29). There are 5 minor criteria: 1) a history of flexural rashes, 2) visible flexural eczematous lesions, 3) a history of asthma or hay fever (or a family history in younger patients), 4) a history of generalized dry skin, and 5) onset before age 2 years. For a diagnosis to be made, 3 of 5 minor criteria must be present. Therefore, not all people with atopic dermatitis must have an early age at onset or a history of other atopic conditions, but if more than 2 of these key criteria are missing, other diagnoses should be considered (see the **Box: Important Clinical Features Used to Diagnose Atopic Dermatitis**).

In infants, lesions are typically focused on the face (cheeks) and extensors and spare the diaper area (30). After age 2 years, the distribution becomes more flexural, with involvement of the antecubital and popliteal fossa as well as the ankle and wrist flexures. Hand and periorcular dermatitis are more common in adult atopic dermatitis.

Although eczematous lesions are typically described as poorly demarcated erythematous plaques with or without scale, the lesions can be relatively well demarcated, particularly if they are chronic and lichenified. In darker skin types, erythema can be less prominent and appear either gray or violaceous (**Figure 2**). Clinicians should recognize this and avoid undertreatment. Eczematous lesions can also be follicular,

Important Clinical Features Used to Diagnose Atopic Dermatitis*

Major criterion (must be present): Itchy skin condition

Minor criteria (3 of 5 must be present):

1. History of flexural rashes
2. Visible flexural eczematous lesions (based on physical examination)
3. History of asthma or hay fever (or family history in younger patients)
4. Generalized dry, itchy skin
5. Early age at onset

* Simplified from the U.K. Working Party diagnostic criteria (29).

presenting as papules centered on hair follicles. Chronically rubbed, scratched, and picked areas can become lichenified plaques or discrete papulonodules called prurigo nodules.

Approximately 90% of children and adults with atopic dermatitis have either mild or moderate skin disease, with a minority having severe atopic dermatitis (31, 32). Severity is typically ascertained by a gestalt clinical assessment that includes the extent and severity of visible skin lesions and the severity of itch and other symptoms reported by the patient or their caregivers. There are multiple validated clinician- and patient-reported outcomes for atopic dermatitis (33), but these are not typically used in routine clinical practice. Actively inflamed dermatitis is sometimes referred to as “flaring,” but from a patient perspective, atopic dermatitis flares represent a worsening from baseline (34, 35). For many people with atopic dermatitis, their baseline skin condition is actively inflamed, requiring ongoing anti-inflammatory treatment.

What is the differential diagnosis for patients with a dry, itchy rash?

In most cases, the clinical diagnosis of atopic dermatitis will be obvious based on the presenting symptoms, signs, and clinical history. When clinical features are atypical or when atopic dermatitis does not respond as expected to standard treatments such as topical corticosteroids, other diagnoses should be considered (**Table 1**). The differential diagnosis changes somewhat with age, but at any age it includes seborrheic dermatitis (including cradle cap in infants), scabies, and psoriasis. Seborrheic dermatitis typically has a greasier scale and follows a seborrheic distribution in the scalp and “T-zone” of the face as well as the chest. Like atopic dermatitis, seborrheic dermatitis can often be managed with topical anti-inflammatory medications, such as corticosteroids, but it may also respond to topical antifungal treatments. Scabies often involves the groin and web spaces of the hands, and in

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Figure 2. Eczematous lesions in people with darker skin (68).



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Eczematous inflammation in dark skin often appears more purple (left) or gray (right) than red. (Reproduced from Gan C, Mahil S, Pink A, et al. Atopic dermatitis in skin of colour. Part 2: Considerations in clinical presentation and treatment options. *Clin Exp Dermatol.* 2023;48:1091-1101, with permission. Licensed under the Creative Commons Attribution 4.0 International License [<https://creativecommons.org/licenses/by/4.0/deed.en>]).

older children and adults who are not immunosuppressed, it does not affect the face or other parts of the head.

The papules and plaques of psoriasis are usually better demarcated than those of atopic dermatitis but can sometimes be difficult to distinguish when atopic dermatitis is more lichenified. Psoriasis and atopic dermatitis involving the scalp, palms, and soles can be particularly difficult to distinguish. Treatment of mild to moderate atopic dermatitis and psoriasis is often similar, with both responding to topical anti-inflammatory medications, such as corticosteroids. However, targeted systemic treatments, such as biologics, are different for the 2 conditions, so it is particularly important to distinguish between them when considering

advancing treatment beyond topical medications.

In infants and younger children, nutritional deficiencies, such as zinc deficiency, should be considered if the rash is more severe in the diaper area and does not improve with short courses of topical anti-inflammatory treatment. Immunodeficiencies, such as IgA deficiency and DOK8 deficiency, should be considered in refractory cases or when there are other concerning features, such as recurrent or atypical infections. Genodermatoses, such as ichthyoses and Netherton syndrome, are also in the differential diagnosis.

In older children and adults, cutaneous T-cell lymphoma (CTCL; mycosis fungoides) is in the differential diagnosis

Table 1. Important Entities in the Differential Diagnosis of Atopic Dermatitis

Entity	Features Distinguishing It From Atopic Dermatitis	Typical Response to Atopic Dermatitis Topical Treatments
Seborrheic dermatitis	Commonly distributed at the hair-line, eyebrows, and nasolabial folds Plaques have a “greasy” scale	Usually responds to anti-inflammatory treatments Can also respond to antifungal treatments
Scabies	Often affects the groin and web spaces of the hands and feet In immunocompetent adults, usually spares the head and neck Mites and burrows may not be readily apparent	Symptoms may respond to some extent, but itch and rashes will persist
Psoriasis	More well-demarcated plaques Typical extensor distribution	Treated with topical treatments similar to those for atopic dermatitis
Nutritional (e.g., zinc) deficiencies	More severe and refractory in the diaper area	Does not respond
Cutaneous T-cell lymphoma	Atrophic plaques Bathing trunk distribution	May respond to topical corticosteroids

of atypical dermatitis. Features that can help distinguish atopic dermatitis from CTCL include the fact that CTCL often preferentially affects sun-protected areas, such as the buttocks, and is less likely to affect the flexures. CTCL lesions are commonly atrophic plaques, which would be unusual for atopic dermatitis. Ruling out CTCL is one of the few indications for a skin biopsy in people presenting with possible atopic dermatitis with atypical features.

Other forms of dermatitis, such as allergic and irritant dermatitis, should be considered depending on the distribution of lesions (for example, predominantly hand dermatitis). Atopic dermatitis commonly coexists with contact dermatitis, as skin barrier disruption predisposes people to irritant dermatitis and allergic sensitization. Exposure to various topical products used to treat atopic dermatitis, such as preservatives in emollients and topical medications as well as specific ingredients such as antibiotics and even corticosteroids, can lead to contact sensitization to those ingredients.

What is the approach to diagnosing atopic dermatitis?

For most patients, atopic dermatitis is diagnosed using clinical features alone. The diagnosis can be readily made in primary care without the need for specialist assessment. When clinical features

are atypical or it does not respond to standard first-line therapy, referral to a dermatologist is warranted.

In infants, testing for specific nutritional deficiencies may be considered, particularly if they are refractory to topical therapy. Genetic testing for specific immunodeficiencies or genodermatoses may be warranted depending on the clinical history (for example, recurrent infections or family history), but this is rare.

Skin biopsies can help to distinguish atopic dermatitis from psoriasis and cutaneous lymphoma. However, biopsies are not useful in distinguishing among different forms of dermatitis; under a microscope, atopic dermatitis and allergic contact dermatitis are indistinguishable. Some biopsies may detect scabies if a mite or their byproducts are captured in cross-section, but this is often missed.

Allergy testing, usually by an allergy and immunology specialist, for IgE-mediated allergies using skin prick testing or blood assays for IgE to specific allergens is not recommended as part of routine management of atopic dermatitis. These tests can be useful as part of diagnosis and management of comorbid airway disorders (asthma, rhinitis) or food allergies, although this should be

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reserved for people who present with signs and symptoms of those disorders, not with atopic dermatitis alone.

Patch testing can be useful to distinguish atopic dermatitis from allergic contact dermatitis or to assess for concomitant allergic contact dermatitis in people with established atopic dermatitis. Among people with atopic dermatitis referred

for patch testing through the North American Contact Dermatitis Group, 56% of adults and 53% of children were diagnosed as having clinically relevant allergic contact dermatitis (36). However, the true rate of allergic contact dermatitis and the value of patch testing among unselected patients with atopic dermatitis are unknown.

Diagnosis... Atopic dermatitis is usually readily diagnosed in people of all ages based on clinical features, including the universal presence of itch and a combination of other minor features, including chronic or relapsing flexural eczema, early age at onset, dry skin, and a personal or family history of other atopic conditions. Laboratory testing or specialist assessment is usually unnecessary but can be considered when the diagnosis is in doubt or when first-line treatment is ineffective.

CLINICAL BOTTOM LINE

Treatment

What is the general approach to treating atopic dermatitis?

All management strategies for atopic dermatitis work by targeting either skin barrier disruption or aberrant type 2 inflammation. Treatments can be used for maintenance, to try to prevent flares, or as treatment of active chronic dermatitis or acute flares. Most of the topical and systemic treatments described in this section can be used as part of maintenance or active treatment, and selection of the treatment method depends on the severity and refractoriness of the patient's atopic dermatitis.

Treatment usually begins with topical therapy, including moisturizers and prescription topical anti-inflammatory medications, regardless of the severity at initial assessment. For people with moderate or severe atopic dermatitis that is refractory to topical therapy, advanced treatments, such as ultraviolet phototherapy or systemic immunomodulatory medications, including injectable biologics and oral medications, can be considered. All available topical therapies can be prescribed in

primary care, with specialist consultation and follow-up required when atopic dermatitis is refractory or advanced therapies are being considered. Phototherapy and systemic immunomodulators are generally prescribed by specialists, including dermatologists and allergists.

What is the role of behavioral and nonpharmacologic approaches to managing atopic dermatitis?

There is abundant evidence from randomized controlled trials that moisturizers, most of which are available over the counter without the need for a prescription, are effective for both flare prevention and treatment (37). Therefore, they are recommended for people of all ages with atopic dermatitis of all severity levels (38, 39).

Guidelines do not recommend a specific moisturizer or group of moisturizers over others (38, 39). Therefore, people with atopic dermatitis should be encouraged to select a moisturizer based on personal preference.

A trial randomly assigned 550 children in the United Kingdom to a lotion,

57. Bieber T, Simpson EL, Silverberg JI, et al; JADE COMPARE Investigators. Abrocitinib versus placebo or dupilumab for atopic dermatitis. *N Engl J Med*. 2021;384:1101-1112. [PMID: 33761207]
58. Drucker AM, Walwyn C, Chu C, et al. Living network meta-analysis to compare nemolizumab against other available targeted systemic treatments for atopic dermatitis. *Br J Dermatol*. 2025;193:548-552. [PMID: 40334148]
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60. Drucker AM. Treatment of atopic dermatitis. *JAMA*. 2025;334:1103-1104. [PMID: 40833742]
61. Waljee AK, Rogers MAM, Lin P, et al. Short term use of oral corticosteroids and related harms among adults in the United States: population based cohort study. *BMJ*. 2017;357:j1415. [PMID: 28404617]
62. Drucker AM, Eyerich K, de Bruin-Weller MS, et al. Use of systemic corticosteroids for atopic dermatitis: International Eczema Council consensus statement. *Br J Dermatol*. 2018;178:768-775. [PMID: 28865094]
63. Apfelbacher CJ, van Zuuren EJ, Fedorowicz Z, et al. Oral H1 antihistamines as monotherapy for eczema. *Cochrane Database Syst Rev*. 2013;2013:CD007770. [PMID: 23450580]
64. Chi CC, Wang SH, Wojnarowska F, et al. Safety of topical corticosteroids in pregnancy. *Cochrane Database Syst Rev*. 2015;2015:CD007346. [PMID: 26497573]
65. Gregoriou S, Routsis E, Koumprentziotis IA, et al. Dupilumab use for atopic dermatitis during pregnancy: a systematic review. *J Eur Acad Dermatol Venereol*. 2025;39:e750-e755. [PMID: 39902922]
66. Deleuran M, Dézfoulian B, Elberling J, et al. Systemic anti-inflammatory treatment of atopic dermatitis during conception, pregnancy and breastfeeding: interdisciplinary expert consensus in Northern Europe. *J Eur Acad Dermatol Venereol*. 2024;38:31-41. [PMID: 37818828]

cream, gel, or ointment moisturizer to be used twice daily and as required. After 16 weeks, there was no difference in atopic dermatitis symptoms among groups (global $P = 0.77$) (40).

“Trigger avoidance” is often described as a cornerstone of atopic dermatitis management. Guidelines from the American Academy of Allergy, Asthma & Immunology (AAAAI) and the American College of Allergy, Asthma & Immunology (ACAAI) include a best practice statement that before initiating any new therapy, clinicians should address trigger avoidance with their patients (38). Guidelines from the AAD recommend first identifying and avoiding triggers (41). In reality, trigger avoidance can be very difficult to implement. Because of the naturally fluctuating, relapsing, and remitting nature of atopic dermatitis, it is often not possible to pinpoint triggers of a flare, such as environmental exposures, contact irritants, allergens, or foods. A search for possible triggers often does not lead to any actionable measures to improve control of atopic dermatitis.

What topical anti-inflammatory treatment options for active atopic dermatitis are available?

Topical anti-inflammatory treatment options for atopic dermatitis include corticosteroids, calcineurin inhibitors, phosphodiesterase-4 (PDE-4) inhibitors, JAK inhibitors, and an aryl hydrocarbon receptor agonist (Table 2).

Topical corticosteroids have been used for decades and are highly effective and safe for treatment of active inflammatory lesions of dermatitis (42, 43). They are available in multiple formulations of varying potency according to the individual corticosteroid molecule, its concentration, and its vehicle (for example, cream or ointment) (Table 3) (44). Several potency classification systems are available; in the United States, a 7-item system is most commonly used, ranging from class VII (least potent) to class I (super potent) (44).

When selecting an appropriate corticosteroid for an individual patient,

clinicians should consider the severity of the eczematous lesions being treated, their location on the body, and whether other topical medications have already been tried. Moderate- or higher-potency steroid formulations should be avoided on the face and genitals. Higher-potency formulations may be required for thicker lesions and lesions on the hands and feet. Creams and ointments are preferred for the face, trunk, and extremities, whereas lotions, foams, oils, and shampoos are preferred for the scalp. Topical corticosteroids are often prescribed for twice-daily use, but a meta-analysis of clinical trials found that once-daily use is as effective as twice-daily use, which may be easier for patient adherence (42). Adverse effects, including skin atrophy, growth abnormalities, and adrenal suppression, are rare (43). Recently, increasing attention has focused on topical corticosteroid withdrawal, including the implementation of medication safety warnings in the United Kingdom and Canada (45, 46). Topical corticosteroid withdrawal remains poorly defined and understood, and rigorous studies to estimate its prevalence are lacking.

Nonsteroidal topical anti-inflammatory medications can be used as alternatives and in a similar fashion to topical corticosteroids for active inflammatory lesions of atopic dermatitis. They are particularly useful for chronic dermatitis on the face and genitals, where the risk for adverse effects from corticosteroids is higher.

Topical calcineurin inhibitors for atopic dermatitis include tacrolimus 0.1% and 0.03% ointment and pimecrolimus 1% cream. Although theoretical concerns about possible cancer risk with these medications led the U.S. Food and Drug Administration (FDA) to apply a black box warning to these medications, multiple safety studies have shown that long-term use is unlikely to be associated with clinically important risk (47). Their use is often limited by application site reactions, such as stinging or burning.

67. Santer M, Muller I, Becque T, et al. Eczema Care Online behavioural interventions to support self-care for children and young people: two independent, pragmatic, randomised controlled trials. *BMJ*. 2022;379:e072007. [PMID: 36740888]
68. Gan C, Mahil S, Pink A, et al. Atopic dermatitis in skin of colour. Part 2: Considerations in clinical presentation and treatment options. *Clin Exp Dermatol*. 2023;48:1091-1101. [PMID: 37119261]

Table 2. Topical Anti-inflammatory Treatments of Atopic Dermatitis

Treatment	Mechanism	Formulations	Cost Considerations	Notes
Corticosteroids	Corticosteroid	See Table 3	Variable, but many generic formulations are available	Use mild formulations (e.g., hydrocortisone 1%) for the face and folds, moderate (e.g., triamcinolone 0.1%) for the trunk and extremities, and potent (e.g., fluocinonide 0.05%) or very potent (e.g., clobetasol 0.05%) for areas that are refractory to moderate-potency formulations. Adverse effects are uncommon but include local skin atrophy.
Tacrolimus	Calcineurin inhibitor	0.1% ointment 0.03% ointment	Generic formulations available	0.1% formulation is more effective and preferred in most instances. Has a class-wide safety warning regarding cancer risk.
Pimecrolimus	Calcineurin inhibitor	1% cream	Generic formulations available	Probably less effective than tacrolimus 0.1% ointment. Has a class-wide safety warning regarding cancer risk.
Crisaborole	PDE-4 inhibitor	2% ointment	Expensive; no generic formulations available	Among the least effective topical anti-inflammatory treatments in a network meta-analysis.
Roflumilast	PDE-4 inhibitor	0.15% cream	Expensive; no generic formulations available	Among the newest topical anti-inflammatory treatments. Also available in 0.3% cream and foam, which can be used off-label for atopic dermatitis.
Ruxolitinib	JAK inhibitor	1.5% cream	Expensive; no generic formulations available	Among the newest topical anti-inflammatory treatments. Has a class-wide safety warning regarding infection and cardiovascular and thrombosis risk.
Tapinarof	Aryl hydrocarbon receptor agonist	1% cream	Expensive; no generic formulations available	Among the newest topical anti-inflammatory treatments.

JAK = Janus kinase; PDE-4 = phosphodiesterase-4.

Topical PDE-4 inhibitors approved for atopic dermatitis are crisaborole 2% ointment and roflumilast 0.15% cream. Roflumilast is also available as a 0.3% cream approved for psoriasis and a 0.3% foam approved for psoriasis and seborrheic dermatitis; the foam formulation can be a useful off-label treatment of atopic dermatitis affecting the scalp.

Ruxolitinib 1.5% cream is a topical JAK inhibitor that is approved to treat atopic dermatitis and may also be among the most effective topical treatments.

A Cochrane systematic review and network meta-analysis found that tacrolimus 0.1% (odds ratio [OR], 6.27 [95% CI, 1.19 to 32.98]),

potent topical corticosteroids (OR, 5.99 [CI, 2.83 to 12.69]), and ruxolitinib 1.5% (OR, 5.64 [CI, 1.26 to 25.25]) are most effective for symptoms, although confidence is low (48).

Delgocitinib 2% cream is another topical JAK inhibitor that is approved by the FDA to treat chronic hand eczema but is sometimes used off-label for atopic dermatitis. Based on data on oral JAK inhibitors used in other disease states, the FDA has issued safety warnings about all JAK inhibitors, including ruxolitinib and delgocitinib creams, regarding the risk for cancer, cardiovascular events, blood clots, and death. Although use over large body surface areas can result in meaningful systemic

absorption, it is unclear whether users of topical JAK inhibitors are at increased risk for serious adverse events (49).

Tapinarof 1% cream is an aryl hydrocarbon receptor agonist that is approved for both atopic dermatitis and psoriasis.

Can topical anti-inflammatory treatments be used as maintenance treatment of atopic dermatitis?

Moisturizers are recommended as maintenance treatment for all people with atopic dermatitis, even when there is little or no disease activity. For people whose atopic dermatitis flares regularly in the same locations on the body, guidelines recommend

Table 3. Relative Potencies of Topical Corticosteroids*

Class	Drug	Dosage Form(s)	Strength, %
I. Very high potency	Augmented betamethasone dipropionate	Ointment	0.05
	Clobetasol propionate	Cream, foam, ointment	0.05
	Diflorasone diacetate	Ointment	0.05
	Halobetasol propionate	Cream, ointment	0.05
II. High potency	Amcinonide	Cream, lotion, ointment	0.1
	Augmented betamethasone dipropionate	Cream	0.05
	Betamethasone dipropionate	Cream, foam, ointment, solution	0.05
	Desoximetasone	Cream, ointment	0.25
	Desoximetasone	Gel	0.05
	Diflorasone diacetate	Cream	0.05
	Fluocinonide	Cream, gel, ointment, solution	0.05
	Halcinonide	Cream, ointment	0.1
	Mometasone furoate	Ointment	0.1
	Triamcinolone acetonide	Cream, ointment	0.5
III-IV. Medium potency	Betamethasone valerate	Cream, foam, lotion, ointment	0.1
	Clocortolone pivalate	Cream	0.1
	Desoximetasone	Cream	0.05
	Fluocinolone acetonide	Cream, ointment	0.025
	Flurandrenolide	Cream, ointment	0.05
	Fluticasone propionate	Cream	0.05
	Fluticasone propionate	Ointment	0.005
	Mometasone furoate	Cream	0.1
	Triamcinolone acetonide	Cream, ointment	0.1
V. Lower-medium potency	Hydrocortisone butyrate	Cream, ointment, solution	0.1
	Hydrocortisone probutate	Cream	0.1
	Hydrocortisone valerate	Cream, ointment	0.2
	Prednicarbate	Cream	0.1
VI. Low potency	Alclometasone dipropionate	Cream, ointment	0.05
	Desonide	Cream, gel, foam, ointment	0.05
	Fluocinolone acetonide	Cream, solution	0.01
VII. Lowest potency	Dexamethasone	Cream	0.1
	Hydrocortisone	Cream, lotion, ointment, solution	0.25, 0.5, 1
	Hydrocortisone acetate	Cream, ointment	0.5-1

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 * Includes representative examples and not all available agents.

maintenance treatment with mid-potency topical corticosteroids or topical calcineurin inhibitors 2 to 3 times per week to areas that frequently flare (38, 39). Although there is limited or no randomized trial evidence for maintenance therapy with other classes of topical medications, they could theoretically provide similar benefits.

What is the role of phototherapy in treating atopic dermatitis?

Ultraviolet phototherapy, most commonly with ultraviolet B (UVB) wavelengths, can be used to treat active atopic dermatitis (51). It is recommended for people with moderate or severe atopic dermatitis that is refractory to topical therapy (38, 52). The biggest drawback of UVB

phototherapy is that it is usually delivered in specialized clinics with 2 to 3 treatments per week and approximately 30 treatments total and is therefore not available or feasible for all people for whom it might be medically appropriate.

What systemic pharmacologic treatment options for atopic dermatitis are available?

Systemic treatments of atopic dermatitis include older oral immunomodulators, subcutaneous biologics, and oral JAK inhibitors (Table 4). These medications are usually prescribed by specialists for people with moderate to severe atopic dermatitis that is inadequately treated by topical medications.

Biologics are considered first-line systemic treatment because of their efficacy and favorable safety profile (38, 52). Unlike biologics in some other areas of medicine, the biologics approved to date for atopic dermatitis are not considered significantly immunosuppressive with regard to infection risk.

Dupilumab was the first biologic approved for atopic dermatitis and is now approved in adults and children as young as 6 months. Dupilumab binds the interleukin-4 (IL-4) receptor, which is involved in IL-4 and IL-13 signaling. Lebrikizumab and tralokinumab are biologics that target IL-13 directly and are approved for people aged 12 years or older. All 3 biologics that target the IL-4 and

Table 4. Systemic Immunomodulatory Treatments of Atopic Dermatitis

<i>Treatment</i>	<i>Mechanism of Action</i>	<i>Route and Adult Dosing*</i>	<i>Cost Considerations</i>	<i>Notes</i>
Older immunomodulatory medications				
Methotrexate	Immunomodulatory (broad)	PO, SC, IM Typical dose is 15-25 mg once weekly	Inexpensive; generic formulations available	Used off-label for atopic dermatitis Baseline immunosuppressive work-up required Monitoring required for liver effects and reduced blood counts
Cyclosporine	Immunomodulatory (broad)	PO 3-5 mg/kg daily	Variable; generic formulations available	Used off-label for atopic dermatitis; approved on-label for atopic dermatitis in Europe Baseline immunosuppressive work-up required Monitoring required for hypertension, renal effects, dyslipidemia, and reduced blood counts
Azathioprine	Immunomodulatory (broad)	PO 1-2.5 mg/kg daily	Variable; generic formulations available	Used off-label for atopic dermatitis Baseline immunosuppressive work-up required Monitoring required for liver effects and reduced blood counts Not safe for long-term use
Mycophenolate	Immunomodulatory (broad)	PO Mycophenolate mofetil: 500-1500 mg BID Mycophenolic acid: 360-1080 mg BID	Variable; generic formulations available	Used off-label for atopic dermatitis Baseline immunosuppressive work-up required Monitoring required for liver effects and reduced blood counts
Biologics				
Dupilumab	Blocks IL-4 receptor α	600 mg, then 300 mg SC every 2 wk	Expensive; no generic formulations available	No baseline work-up or monitoring required Conjunctivitis is a common adverse effect
Tralokinumab	Blocks IL-13	600 mg, then 300 mg SC every 2 wk	Expensive; no generic formulations available	No baseline work-up or monitoring required Conjunctivitis is a common adverse effect
Lebrikizumab	Blocks IL-13	500 mg, then 250 mg SC every 2 wk until week 16, then 250 mg every 4 wk	Expensive; no generic formulations available	No baseline work-up or monitoring required Conjunctivitis is a common adverse effect
Nemolizumab	Blocks IL-31	60 mg, then 30 mg SC every 4 wk	Expensive; no generic formulations available	No baseline work-up or monitoring required
JAK inhibitors				
Upadacitinib	JAK inhibitor	15 or 30 mg PO daily	Expensive; no generic formulations available	Baseline immunosuppressive work-up required Monitoring required for dyslipidemia, liver effects, and reduced blood counts There is a class-wide safety warning for infection and cardiovascular and thrombosis risk

Continued on following page

Table 4—Continued

Treatment	Mechanism of Action	Route and Adult Dosing*	Cost Considerations	Notes
Abrocitinib	JAK inhibitor	100 or 200 mg PO daily	Expensive; no generic formulations available	Baseline immunosuppressive work-up required Monitoring required for dyslipidemia, liver effects, and reduced blood counts There is a class-wide safety warning for infection and cardiovascular and thrombosis risk
Baricitinib	JAK inhibitor	2 or 4 mg daily	Expensive; no generic formulations available	Approved for atopic dermatitis in Europe but not in North America Baseline immunosuppressive work-up required Monitoring required for dyslipidemia, liver effects, and reduced blood counts There is a class-wide safety warning for infection and cardiovascular and thrombosis risk

Adapted from Drucker AM. Treatment of atopic dermatitis. *JAMA*. 2025;334:1103-1104 (60).

BID = twice daily; IL = interleukin; IM = intramuscular; JAK = Janus kinase; PO = oral; SC = subcutaneous.

* Pediatric dosing is not shown in this table.

IL-13 pathways are associated with ocular surface inflammation (for example, conjunctivitis) as a common adverse effect.

Nemolizumab is the biologic most recently approved by the FDA. It targets IL-31, which is an important cytokine in itch pathogenesis.

Abrocitinib and upadacitinib are oral JAK inhibitors that are approved for treatment of atopic dermatitis in people aged 12 years or older. Because oral JAK inhibitors are immunosuppressive and because of data showing that other oral JAK inhibitors in other disease states increase risk for cancer, blood clots, and cardiovascular outcomes (53), they are considered second-line systemic therapy. Both abrocitinib and upadacitinib are available in low- and high-dose formulations (100 and 200 mg daily for abrocitinib, and 15 and 30 mg daily for upadacitinib). This enables treatment strategies of starting at the lower dose and escalating if there is an inadequate response (54) or

starting at the higher dose to achieve disease control and then reducing the dose for maintenance. Screening and follow-up laboratory testing are recommended for oral JAK inhibitors to rule out tuberculosis and viral hepatitis and to monitor for uncommon adverse effects, such as triglyceride elevation. Baricitinib is another oral JAK inhibitor that is approved in Europe for atopic dermatitis and in the United States for other inflammatory conditions.

Although all of these novel therapies for atopic dermatitis are effective, network meta-analysis and some head-to-head clinical trials have shown that higher doses of abrocitinib and upadacitinib are the most effective treatments, with their lower doses having efficacy similar to that of dupilumab and lebrikizumab for improving signs and symptoms of atopic dermatitis (55-57). Nemolizumab and tralokinumab have lower efficacy than other biologics for most clinical outcomes (58).

Methotrexate, cyclosporine, azathioprine, and mycophenolate are older immunomodulatory medications that are effective for atopic dermatitis. The evidence base underpinning their use is of lower certainty than for newer treatments (59), so the AAD guidelines make only conditional recommendations for their use and the AAAAI/ACAAI guidelines make conditional recommendations against their use (38, 52).

What is the role of oral corticosteroids and antihistamines for atopic dermatitis?

Oral corticosteroids are not recommended to treat atopic dermatitis (38, 52). Although they can be highly effective in the short term, they are associated with rebound flares, and even short-term use of oral corticosteroids is associated with substantial harms (61). As such, oral corticosteroids should be reserved for very limited circumstances (62).

Oral antihistamines are also not recommended to treat atopic dermatitis (38). Itch and inflammation in atopic dermatitis are not histamine-mediated, so mechanistically they are not sensible treatments, and a Cochrane review suggests they are not useful (63).

How should clinicians manage acute atopic dermatitis flares?

Most acute flares respond to topical anti-inflammatory treatments. For people who are already using appropriate topical therapy, escalating to a potent or very potent topical corticosteroid can be con-

sidered, as can switching to a different topical medication.

Unfortunately, biologics and JAK inhibitors are expensive, and insurance prior authorization and other coverage issues can make accessing them in a timely fashion for acute flares challenging. Of the older systemic medications, cyclosporine acts most quickly.

How is atopic dermatitis treated in pregnancy?

Long-standing atopic dermatitis often flares during pregnancy, including atopic dermatitis that was previously in remission.

Topical corticosteroids and topical calcineurin inhibitors are considered safe in pregnancy (64). The safety of other classes of topical medications in pregnancy is uncertain, and JAK inhibitors should be avoided in pregnancy. Phototherapy is considered safe. There is some evidence that dupilumab can be used safely in pregnancy, but the body of evidence is small (65). Cyclosporine can be used carefully in pregnancy, but other older immunomodulators and oral JAK inhibitors cannot (66).

Treatment... Moisturizers are recommended for people with atopic dermatitis of all severity levels as both maintenance and active treatment. Topical anti-inflammatory treatments can be used once or twice daily to treat actively inflamed areas of dermatitis. Factors affecting the choice of topical treatment include the severity of the dermatitis and the body site involved. For people with more severe or refractory atopic dermatitis, advanced treatments, such as phototherapy, biologics, oral JAK inhibitors, and older immunomodulators, can be used.

CLINICAL BOTTOM LINE

Practice Improvement

What do professional organizations recommend?

Recent guidelines from major U.S. dermatology and allergy groups have thoroughly assessed the evidence and made evidence-based treatment recommendations on topical and systemic therapy for atopic dermatitis (24, 38, 39, 41, 52).

The AAD guidelines, which were updated in 2025, make strong recommendations in favor of topical corticosteroids, calcineurin inhibitors, PDE-4 inhibitors, ruxolitinib, and tapinarof cream (41). The AAD guidelines make strong recommendations for all of the approved biologics

and oral JAK inhibitors, conditional recommendations for each of the older broad immunomodulators, and a conditional recommendation against systemic corticosteroids. Recommendations in the AAAAI/ACAAI guidelines from 2024 are similar, but the recommendation for crisaborole ointment is only conditional (due to limited efficacy) and the recommendation for ruxolitinib cream is conditional against its use (due to concerns related to safety warnings). Another difference between the dermatology and allergy guidelines is that the allergy guidelines conditionally recommend against (rather than for) methotrexate, mycophenolate, and azathioprine.

What resources are available for patients?

Patient groups such as the National Eczema Association in the United States (<https://nationaleczema.org>), the Eczema Society of Canada (<https://eczemahelp.ca>), and the National Eczema Society in the United Kingdom (<https://eczema.org>) have excellent patient-facing resources. Eczema Care Online (<https://eczemacareonline.org.uk>) is an online resource for people with atopic dermatitis and their caregivers that has been shown in randomized controlled trials to reduce atopic dermatitis symptoms (12, 38, 40, 41, 48, 55, 67).

In the Clinic Tool Kit

Atopic Dermatitis

Patient Information

<https://medlineplus.gov/eczema.html>

<https://medlineplus.gov/languages/eczema.html>
Information on eczema in English and other languages from the National Institutes of Health's MedlinePlus.

[https://www.aaaai.org/tools-for-the-public/conditions-library/allergies/eczema-\(atopic-dermatitis\)-overview](https://www.aaaai.org/tools-for-the-public/conditions-library/allergies/eczema-(atopic-dermatitis)-overview)

Overview of eczema from the American Academy of Allergy, Asthma & Immunology.

<https://www.niams.nih.gov/health-topics/atopic-dermatitis>

Information on atopic dermatitis from the National Institute of Arthritis and Musculoskeletal and Skin Diseases.

<https://www.mayoclinic.org/diseases-conditions/atopic-dermatitis-eczema/symptoms-causes/syc-20353273>

Patient information on atopic dermatitis from the Mayo Clinic.

<https://nationaleczema.org>

Patient information from the National Eczema Association.

<https://eczemahelp.ca>

Patient information from the Eczema Society of Canada.

<https://eczema.org>

Patient information from the National Eczema Society.

Information for Health Professionals

[https://www.jaad.org/article/S0190-9622\(25\)02125-5/fulltext](https://www.jaad.org/article/S0190-9622(25)02125-5/fulltext)

Focused update of the guidelines of care for the management of atopic dermatitis in adults from the American Academy of Dermatology.

[https://www.jaad.org/article/S0190-9622\(23\)00004-X/fulltext](https://www.jaad.org/article/S0190-9622(23)00004-X/fulltext)

Guidelines of care for the management of atopic dermatitis with topical therapies in adults from the American Academy of Dermatology.

[https://www.jaad.org/article/S0190-9622\(23\)02878-5/fulltext](https://www.jaad.org/article/S0190-9622(23)02878-5/fulltext)

Guidelines of care for the management of atopic dermatitis with phototherapy and systemic therapies in adults from the American Academy of Dermatology.

[https://www.jaad.org/article/S0190-9622\(22\)00080-9/fulltext](https://www.jaad.org/article/S0190-9622(22)00080-9/fulltext)

Guidelines on awareness of comorbidities associated with atopic dermatitis in adults from the American Academy of Dermatology.

[https://www.annallergy.org/article/S1081-1206\(23\)01455-2/fulltext](https://www.annallergy.org/article/S1081-1206(23)01455-2/fulltext)

2023 recommendations on atopic dermatitis (eczema) from the American Academy of Allergy, Asthma & Immunology/American College of Allergy, Asthma & Immunology Joint Task Force on Practice Parameters.

In the Clinic

WHAT YOU SHOULD KNOW ABOUT ATOPIC DERMATITIS

In the Clinic
Annals of Internal Medicine

What Is Atopic Dermatitis?

Atopic dermatitis, also known as eczema, is a common, chronic, inflammatory skin condition that causes itchy, inflamed rashes. These rashes can appear anywhere on the body but often affect the flexural areas like the inner creases of the elbows, knees, wrists, and ankles. The itch can be intense, affecting sleep, quality of life, and even school or work. Although atopic dermatitis is more common in children, it can persist into adulthood or start later in life.

What Are the Symptoms?

- Intense itching
- Red, inflamed skin (can appear gray or violet on darker skin)
- Dry, cracked skin
- Rashes, often in the creases of the elbows, knees, wrists, and ankles
- Thickened, leathery skin from chronic scratching

How Is It Diagnosed?

Your doctor will diagnose atopic dermatitis based on your symptoms, a physical examination of your skin, and your medical history. No specific laboratory tests are needed in most cases.

Diagnosis requires an itchy skin condition and 3 of the following 5 criteria:

- History of rashes in the flexural areas (such as the elbows or knees)
- Visible eczematous lesions in the flexural areas
- History of asthma or hay fever, or family history of these conditions
- Generalized dry, itchy skin
- Early age at onset (for example, before age 2 years)

What Are the Triggers?

- Genetic factors: A strong genetic component contributes to atopic dermatitis.
- Environmental factors: Exposure to pathogens early in life, pollution, extreme heat and cold, allergens, and irritants can trigger atopic dermatitis. However, it is often difficult to identify avoidable triggers to reduce flares.

What Other Illnesses Are Associated With Atopic Dermatitis?

- Asthma
- Allergic rhinitis (hay fever)
- Food allergies



- Mental health conditions (depression, anxiety, attention deficit-hyperactivity disorder)
- Autoimmune conditions
- Bone fractures

How Is It Treated?

The goal of treatment is to reduce itch and inflammation, repair the skin barrier, and prevent flares.

- Use moisturizers frequently throughout the day and immediately after bathing to hydrate the skin and repair the skin barrier. Choose fragrance-free and hypoallergenic options.
- Topical corticosteroids are the most common first choice for therapy. Milder strengths can be used on the face, and higher strengths can be used on thicker skin or areas that are less responsive to milder treatments.
- There are also topical nonsteroidal options, including calcineurin inhibitors (tacrolimus, pimecrolimus), PDE-4 inhibitors (crisaborole, roflumilast), JAK inhibitors (ruxolitinib), and aryl hydrocarbon receptor agonists (tapinarof).
- Moderate to severe disease may require ultraviolet light therapy at a specialized clinic or systemic medications like injectable biologics (for example, dupilumab, lebrikizumab, tralokinumab, or nemozumab) or oral JAK inhibitors (for example, abrocitinib or upadacitinib) that reduce inflammation.

Questions for My Doctor

- What if I am pregnant or thinking of becoming pregnant?
- Do I need to use the topical creams or ointments even if I have no symptoms?
- Are there any long-term problems I should worry about?
- What should I do if my symptoms get worse (flare)?

For More Information



American College of Physicians
Leading Internal Medicine, Improving Lives

National Eczema Association

<https://nationaleczema.org>

Eczema Society of Canada

<https://eczemahelp.ca>

National Eczema Society

<https://eczema.org>

American Academy of Allergy, Asthma & Immunology

[https://www.aaaai.org/tools-for-the-public/conditions-library/allergies/eczema-\(atopic-dermatitis\)-overview](https://www.aaaai.org/tools-for-the-public/conditions-library/allergies/eczema-(atopic-dermatitis)-overview)

National Institute of Arthritis and Musculoskeletal and Skin Diseases

<https://www.niams.nih.gov/health-topics/atopic-dermatitis>

Mayo Clinic

<https://www.mayoclinic.org/diseases-conditions/atopic-dermatitis-eczema/symptoms-causes/syc-20353273>