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Eczema: A Comprehensive Review of Pathophysiology, Diagnosis, and Emerging Therapies

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ABSTRACT

Eczema, or dermatitis, encompasses a group of chronic inflammatory skin conditions marked by intense pruritus, xerosis, erythema, and episodic flares that significantly impair patient quality of life. Among its subtypes, atopic dermatitis (AD) is the most prevalent, driven by a multifactorial interplay of genetic susceptibility, epidermal barrier dysfunction, immune dysregulation, and environmental triggers. Mutations in the filaggrin gene (FLG) and alterations in ceramide composition disrupt skin barrier integrity, leading to increased trans epidermal water loss and heightened sensitivity to irritants and allergens. Recent advances highlight the critical role of the skin microbiome, particularly Staphylococcus aureus colonization, in promoting inflammation and exacerbating disease severity. Therapeutic strategies have progressed from conventional topical corticosteroids, calcineurin inhibitors, and emollients to advanced modalities including biologics (e.g., dupilumab, tralokinumab), Janus kinase (JAK) inhibitors, and innovations such as microbiome-based therapies and barrier-repair formulations. Despite these advancements, challenges persist in long-term disease control, prevention of flares, and addressing psychological and socioeconomic burdens. This review synthesizes current insights into the epidemiology, pathogenesis, diagnostic developments, and evolving treatment landscape of eczema, emphasizing the importance of integrated patient-centered care and ongoing research to bridge existing gaps in therapeutic outcomes. Further, growing evidence suggests that lifestyle factors and environmental exposures play an increasingly important role in eczema onset and severity. Urbanization, pollution, climate variability, reduced microbial exposure, and modern hygiene practices





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have been linked to rising global disease prevalence, particularly in children. Dietary influences, such as low intake of omega-3 fatty acids, vitamin D deficiency, and early-life food sensitization, have also been investigated for their association with eczema risk. Psychosocial stress is now recognized as a significant trigger capable of worsening inflammatory pathways and impairing treatment adherence.

KEY WORDS

Eczema, dermatitis, atopic dermatitis, epidermal barrier dysfunction, filagrin mutation, staphylococcus aureus, environmental triggers, patient education, holistic management, genetic environmental interactions, emollient therapy.

INTRODUCTION







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Fig 1: Eczema (atopic dermatitis)

Eczema, also referred to as dermatitis, represents a heterogeneous group of chronic, relapsing inflammatory skin disorders characterized by pruritus, xerosis, erythema, and epidermal barrier dysfunction. Among its various subtypes, atopic dermatitis (AD) is the most prevalent, affecting up to 20% of children and 10% of adults worldwide, with incidence steadily rising over recent decades (1). The pathogenesis of eczema is multifactorial, involving a complex interaction of genetic predisposition, immune dysregulation, environmental triggers, and alterations in skin barrier structure and function. Mutations in the filaggrin (FLG) gene have been identified as a major genetic risk factor, contributing to impaired epidermal barrier integrity and increased trans epidermal water loss(2).

In addition to genetic susceptibility, immune mechanisms—particularly the predominance of Th2-driven cytokines such as IL-4, IL-13, and IL-31—play a central role in driving inflammation and chronicity. Disruption of the skin microbiome, especially colonization by Staphylococcus aureus, further enhances inflammatory pathways and is strongly associated with disease flares and severity(3).. Environmental factors, including pollution, climate conditions, stress, diet, and modern hygiene practices, are increasingly recognized as significant contributors to disease onset and exacerbation.

Management of eczema traditionally relied on topical corticosteroids, emollients, and calcineurin inhibitors; however, recent advancements have transformed therapeutic approaches. Novel biologics, Janus kinase (JAK) inhibitors, microbiome-targeted therapies, and advanced barrier-repair formulations now offer improved outcomes for moderate to severe cases. Despite these advancements, eczema remains a substantial public health concern due to its chronic nature, impact on quality of life, and socioeconomic burden. Therefore, a comprehensive understanding of its epidemiology, pathophysiology, and emerging treatment modalities is essential for optimizing patient care(4).

TYPES

Atopic dermatitis is commonly used to describe eczema because it is the most common type.

That said, there are other forms of the skin condition, including:





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- Allergic contact dermatitis: This is a skin reaction that occurs following contact with a substance or allergen that the immune system recognizes as foreign.
- **Dyshidrotic eczema:** This refers to irritation of the skin on the palms of the hands and soles of the feet. It is characterized by small blisters.
- **Neurodermatitis:** This leads to scaly patches of skin on the head, forearms, wrists, and lower legs. It occurs due to a localized itch, such as from an insect bite.
- **Discoid eczema:** Also known as nummular eczema, this type presents itself as circular patches of irritated skin that can be crusted, scaly, and itchy.







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Fig 2: Types of Eczema

SYMPTOMS

Symptoms of AD vary by individual and may depend on a few factors, such as a person's age, skin tone, and the condition's severity.

In most cases, AD symptoms are mild. However, symptoms may come and go and vary in severity.

Flare-ups refer to periods when symptoms worsen Trusted Source, while remissions are when symptoms improve or clear up(5).

The most common symptoms of AD include:

- · dry, scaly patches of skin
- thickened, discoloured skin
- open, crusted, or weeping sores
- skin flushing
- itching

The appearance of skin affected by AD will also depend on how much a person scratches their rashes.

For example, people with severe eczema may experience severe itching that could lead to continuous rubbing and scratching. This may cause skin infections like infected eczema.(6)

The location, type, and severity of rashes may also vary depending on a person's age.





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INFANTS

Babies under the age of 2 may experience:

- rashes on the scalp and cheeks
- · rashes that bubble up before leaking fluid
- · rashes that cause extreme itchiness, which may interfere with sleeping

CHILDHOOD

Children from the ages of <u>2 to puberty</u> may experience rashes that appear:

- behind the creases of elbows or knees
- on the neck, wrists, and ankles
- in the crease between the buttocks and legs They may also experience:
- bumpy rashes
- · rashes that can become lighter or darker
- skin thickening, also known as lichenification, which can then develop into a permanent itch

African American and Hispanic children may also have more severe eczema.

ADULTS

Adults may experience eczema <u>rashes</u> that could:

• be more scaly than those occurring in children





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- appear in the creases of the elbows, knees, or nape of the neck
- cover much of the body
- be permanently itchy

People over 18 years old may also experience skin infections or very dry skin on the affected area(7)(8).

Adults who developed AD as children but no longer experience the condition may still have:

- dry or easily irritated skin
- hand eczema
- eczema on the eyelids

Age	Location	Symptoms
Babies (under 2 years)	•scalp • cheeks	 rashes may bubble up before leaking fluid extreme itchiness, which may interfere with sleeping





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Children (2- puberty)	 behind the creases of elbows or knees neck wrists ankles crease between the buttocks and legs 	 bumpy rashes rashes may become lighter or darker skin thickening, also known as lichenification, which can then develop into a permanent itch
Adults (18+)	 creases of the elbows, knees, or nape of the neck cover much of the body 	 more scaly rashes very dry skin on the affected areas permanently itchy rashes skin infections

ETIOLOGY

The exact etiologic of eczema is not entirely understood, but it is believed to be a combination of genetic and environmental factors.

Genetic Factors





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There is a strong genetic component to eczema, with a family history of eczema, asthma, or allergies commonly found in affected individuals. Several genes associated with eczema have been identified, including those involved in the skin barrier function and the immune system(9).

Filaggrin Gene

One of the most well-known genes associated with eczema is the filaggrin gene (*FLG*). This gene provides instructions for making a protein called filaggrin, which is important in maintaining the skin barrier function. Mutations in this gene have been linked to eczema and other skin conditions and are thought to increase susceptibility to environmental irritants and allergens.

Other Skin Barrier Genes

In addition to the filaggrin gene, other genes involved in the skin barrier function have been implicated in the development of eczema. These include genes involved in lipid synthesis and transport, such as the ceramide synthase gene and the *ABCA12* gene.

Immune-related Genes

Several genes involved in the immune response have also been associated with eczema, including genes that regulate T-cells, cytokines, and immunoglobulins. These genes include interleukin (*IL*)-4, *IL*-13, *IL*-31, signal transducer and activator of transduction (*STAT*)3, and Fc fragment of immunoglobulin (Ig)E receptor Ig (*FCER1G*)(10).

Overall, the genetic factors involved in eczema are complex and likely involve multiple genes and genetic pathways. Although genetic testing is not routinely used to diagnose eczema, understanding the disease's genetic basis can help identify individuals at increased risk and guide treatment approaches.

Environmental Factors

Environmental factors also play a role in the development of eczema. Patients with eczema have a defect in their skin barrier function, leading to increased water loss and susceptibility to environmental





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irritants and allergens. Common triggers for eczema flares include exposure to irritants such as detergents, soaps, solvents, and allergens such as dust mites, pet dander, and certain foods. Other factors that can exacerbate eczema symptoms include stress, changes in temperature and humidity, and infections(11).

Immune System Activation

In addition to genetic and environmental factors, the immune system is also thought to play a role in the development of eczema. Patients with eczema have an overactive immune response to environmental triggers, leading to inflammation and skin damage.

EPIDERMOLOGY

The lifetime prevalence of eczema is about 15% to 30% in children and 2% to 10% in adults. About 60% of cases will develop the disease within the first year of life. The prevalence of eczema is more common in rural areas than in urban ones. This incidence emphasizes the link between lifestyle and environmental factors in the mechanisms of AD.

Eczema is part of the triad known as "the atopic march." This relates to the association between patients with atopic dermatitis, asthma, and allergic rhinitis. About 50% of patients with severe eczema will develop asthma, and 75% will develop allergic rhinitis(12).

PATHOPHYSIOLOGY

Research shows there is a genetic component to eczema. One common mutation has been observed in *FLG*, a vital gene for skin cell maturity. This gene is responsible for creating the tough, flat corneocytes that form the outermost protective layer of skin. In a patient with normal skin cells, the corneocytes are tightly packed in an organized manner. A patient with an *FLG* mutation will have a dysfunctional skin barrier due to the haphazard organization of the skin cells. This dysfunction causes a "leaky" skin barrier, allowing water loss and decreased protection from harmful substances (13).





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People with eczema also have reduced numbers of β -defensins in the skin. β -defensins are host defense peptides vital for fighting off certain bacteria, viruses, and fungi. A decrease in these peptides leads to increased colonization and infection, especially with *Staphylococcus aureus* (*S. aureus*)(14).

DIAGNOSIS AND HISTORY

It is important that the diagnosis is correct, so that the right treatment and lifestyle advice can be given and potential wider implications can be discussed(15).

All types of eczema are generally diagnosed by history and clinical examination, observing symptoms. If there is diagnostic doubt, further investigation may be necessary: skin scraping, for example, may be done to detect fungal infection; skin swabs may be taken to diagnose bacterial infection (this is not routine, as skin infection is usually diagnosed on clinical appearance); a skin biopsy may be required to detect other skin conditions or to make a firm eczema diagnosis. If itch is a major symptom in adults, and an eczema diagnosis is uncertain, a pruritic screen can be taken to exclude other causes of underlying itch (16)(17).

There are no specific tests for trigger factors, unless allergy is suspected. Trigger factors are diagnosed on patient history.

Contact allergic dermatitis is diagnosed by patch tests . If allergy is suspected to food or other triggers, specific IgE blood tests will be taken .

The history-taking should include the following questions and identify any trigger factors:

- Onset: When did the eczema first start?
- Duration: How long has the condition been present?
- Site: What areas are affected?
- Pruritus (itch): How great is the degree of pain, itching and soreness associated with the eczema? What measures are used to cope with this, and how does it impact on the patient's life? For older patients complaining of pruritus, consider further screening.(18).
- Family history: Is there, or has there been, anyone else in the family with skin disease, eczema, asthma or hay fever?





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- Occupation: What types of products does the patient use? What kinds of protection are used at work (e.g.clothing,gloves and barrier creams)? Does the skin improve when the patient is not at work?
- Hobbies and leisure time: What types of hobbies does the patient have? Do they have contact with particular materials and substances, animals and plants or exposure to sunlight?
- Clothing: What types of clothing fabrics are usually worn and which fabrics flare/irritate the eczema?
- Jewellery: What types of watches and jewelry are worn?
- Impact on quality of life: How are school, work, family and relationships affected by the patient's eczema?
- Skin care: What everyday products (e.g. shampoo, soaps, wipes) are used? What skin-care products have been and are being used? What types of make-up, perfumes and after- shave are used?
- Medication: What medicines are taken regularly? What topical medicaments are used? (Ask about prescribed and over-the-counter products.
- Allergies: Does the patient have any known allergies to medicines or products that come into contact or are applied to the skin?
- Diet: Is there anything in the environment that makes the skin worse? Is the patient affected by seasonal changes? In particular for children under 2 years, does the parent think their child is affected by diet?(19).

TREATMENTS

There is currently no cure for AD. However, treatments aim to heal the affected skin and prevent symptom flare-ups(20).

A doctor will develop a treatment plan based on a person's:

- age
- symptoms
- current health condition





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For some people, eczema may go away over time. For others, it is a lifelong condition.



Fig 3: Treatment of eczema

MEDICATIONS

Several types of medications may help treat eczema symptoms.

- **Topical corticosteroid creams and ointments:** These anti-inflammatory medications are applied directly to the skin to help relieve the main symptoms of eczema, such as inflammation and itchiness. They are available as over-the-counter (OTC) or prescription medications, depending on the strength needed. **Eg:** Hydrocortisone cream, desonide cream (21).
- **Oral medications:** A doctor may prescribe systemic corticosteroids or immunosuppressants, which are available as injections or oral tablets. People should only use them for short periods of time. Also, it is important to note that the symptoms may worsen upon stopping these drugs if the person is not already taking another medication for the condition. **Eg:** prednisolone, cyclosporine.





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- Antibiotics: Doctors prescribe antibiotics if eczema occurs alongside a bacterial skin infection. Eg: Flucloxacillin, cephalexin.
- **Antihistamines:** These can reduce the risk of nighttime scratching, as they tend to cause drowsiness. **Eg:** cetrizene, loratadine.
- **Topical calcineurin inhibitors:** These medications suppress the immune system, which helps decrease inflammation and prevent flares. **Eg:** Tarcolimus ointment.
- Barrier repair moisturizers: These reduce water loss and work to repair the skin.
- **Phototherapy:** This involves exposure to ultraviolet (UV) waves and may help treat moderate AD(22).

HOME CARE

Some home remedies may help people with eczema support their skin health and alleviate symptoms, including:

- taking lukewarm baths
- applying moisturizer within 3 minutes of bathing to "lock in" moisture
- moisturizing every day
- · wearing cotton and soft fabrics
- avoiding rough, scratchy fibres and tight-fitting clothing
- using a humidifier in dry or cold weather (23).
- using a mild soap or a nonsoap cleanser when washing
- taking extra precautions to prevent eczema flares in winter
- air drying or gently patting the skin dry with a towel, rather than rubbing the skin after bathing or taking a shower.
- avoiding rapid changes in temperature and activities that cause sweating
- learning and avoiding individual eczema triggers (24).
- keeping fingernails short to prevent scratching from breaking the skin
 People can also try various natural remedies for eczema, including aloe vera, coconut oil, and apple cider vinegar.





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CONCLUSION

Eczema is a multifaceted chronic inflammatory skin disorder that significantly affects patient comfort and quality of life. Reviewing its introduction, clinical types, etiology, mechanisms, diagnosis, and treatment highlights the complexity of the condition and the importance of a comprehensive understanding. The diverse forms of eczema share common features such as pruritus and barrier dysfunction, yet each type presents distinct clinical patterns that aid in accurate identification. Its causes are multifactorial, involving genetic predisposition, immune dysregulation, environmental influences, and impaired epidermal barrier function. The underlying pathogenic mechanisms, particularly cytokine-driven inflammation and hypersensitivity responses, explain the relapsing and persistent nature of the disease. Diagnosis relies on careful clinical evaluation supported by targeted investigations to identify triggers and classify the specific subtype. Effective management requires an individualized approach that focuses on skin-barrier repair, inflammation control, trigger avoidance, and patient education. With continued advancements in topical therapies, systemic agents, and biologics, outcomes for patients with eczema are steadily improving. Overall, understanding the interconnected aspects of types, causes, mechanisms, diagnosis, and treatment is essential for optimizing care and reducing the long-term burden of eczema.

Eczema is a common yet often misunderstood skin condition that can significantly impact daily life. While its causes are complex and symptoms vary from person to person, effective management is possible through proper skincare, awareness of triggers, and timely medical support. Advances in treatment options—ranging from moisturizers and topical medications to newer biologic therapies—offer hope for long-term relief and improved quality of life. By staying informed, practicing consistent care, and seeking professional guidance when needed, individuals with eczema can take meaningful steps toward healthier skin and greater comfort.





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