



Nirma Univ J Pharm Sci; 2026, 13(1) 45-70

© 2014, Nirma University, Ahmedabad, Gujarat, India

ISSN 2348 –4012



## REVIEW ARTICLE

# MEDICINAL PLANTS USED IN THE MANAGEMENT OF ATOPIC DERMATITIS (ECZEMA): AN UPDATED REVIEW

**Diya Dubal, Aastha Mehta, Nagja Tripathi\***

*Institute of Pharmacy, Nirma University, Ahmedabad – 382481, India.*

*Corresponding Author: Nagja Tripathi    ORCID ID: 0000-0003-3742-3499*

*E-mail: nagja.tripathi@nirmauni.ac.in*

*Received: 28-08-2025, Accepted: 01-05-2026 Published: 22-07-2026*

## ABSTRACT

*Atopic dermatitis (AD) represents a chronic relapsing inflammatory disorder, which is accompanied by severe pruritus, skin erythema, and impaired barrier function of the skin. The available therapeutic approaches, based on the administration of corticosteroids and immunosuppressive agents, are frequently associated with adverse effects, which limit their prolonged use. The current systematic review analyzes the recent advances in the utilization of herbal drugs for the management of AD. Several plant-based remedies exhibit potent anti-inflammatory and immunomodulatory properties as well as help restore skin barrier functions via regulation of cytokines activity and correction of the Th2 immune imbalance. Herbal treatment can be regarded as an effective complementary therapy for AD, possessing low toxicity and high tolerability, while ensuring long-term benefits. Although numerous preclinical and clinical studies confirm the advantages of herbal treatment for AD, it is necessary to develop new prospective studies that would ensure the standardization of treatment schemes.*

**Keywords:** *Anti-inflammatory, Antioxidant, Atopic dermatitis (AD), Eczema, Herbal pharmacotherapy*

## INTRODUCTION

Atopic dermatitis, also referred to as eczema, is a chronic skin condition, the hallmarks of which are itchy, dry and inflamed skin often appearing as red and scaly patches [1]. It can occur at any age, but it generally begins in childhood, and it can only be managed but not cured. For several people it gets better by adulthood but for many others it may persist as a lifelong condition. This is a persistent disease, and it may have flare-ups occasionally. It may be bothersome, but it is not contagious [2,3]. Individuals with eczema may be prone to developing food allergies, allergic rhinitis, and asthma. The symptoms for the same include dry, cracked skin, red, purple, brown or grey skin discoloration, small fluid-filled bumps or crusting, thickened skin, which is raw and sensitive from scratching. Therapy can assist in only managing the symptoms. Scientists are unsure of the exact causes of atopic dermatitis, but they understand that genetics, immune system, and environmental factors contribute to this disease [2]. This research focuses on investigating the therapeutic potential of various herbal medicines introduced by different countries over past decade for the treatment of this condition. Additionally, it aims to contribute to the ongoing exploration of illness. Atopic dermatitis is a disorder that may, in some cases, be induced by food allergies. Around the world various strategies have been devised to manage the symptoms and treat this condition; however, none have consistently

proven effective. The condition is influenced by complex interplay of intrinsic and extrinsic factors, including genetic predisposition, hormonal influences, fungal toxins exposure, extremes of temperature and dry climatic conditions, exposure to UV light, etc. Mutations in critical genes such as, FLG, compromise epidermal barrier integrity and perturb immune homeostasis, thereby facilitating the pathogenesis of AD. Immunopathologically, AD is typified by a skewed Th2- dominant response, which orchestrates B-cell proliferation, plasma cell differentiation, and enhanced immunoglobulin synthesis, notably IgE [4]. Elevated IgE concentrations potentiate hypersensitivity reactions through high affinity binding to FcεRI receptors [5]. A person with AD may show a skin peeling, along with prominent growth of fungi like *Candida albicans* and *Aspergillus*, which can cause inflammation. Environmental stressors like low humidity and harsh chemicals, which can result in impairment to the skin barrier by disrupting filaggrin function, which is an important protein to maintain skin integrity. This results in increased trans epidermal water loss (TEWL) and allows the external allergens and microbes to penetrate the skin more easily [6].

In clinical practice, emollients and systemic corticosteroids are commonly used for managing AD. Corticosteroids vary from class 1 to class 7 i.e., from most potent to least potent. Alternative treatment in AD involves topical calcineurin

inhibitors, ideal for sensitive areas like face, skin folds, groins, and underarms. Other treatments may involve anti-infective agents, antihistamines, and vitamin D3 analogues, which are considered second-line options [7]. Although the utilization of corticosteroids from Asian Academy of Dermatology and Venereology believes that there is still requirement to educate patients and caregivers due to their adverse effects and imperilment of rebound [3]. In individuals suffering from atopic dermatitis (AD) where the colonization of *Staphylococcus aureus* increases the extent of the condition, the use of antiseptic medications alone or in combination with other treatments proves beneficial in improving the severity of the disease [8]. Analogues of vitamin D3, when combined with immunosuppressive medications like cyclosporine, methotrexate, and azathioprine, lead to moderate to severe side effects that reduce the effectiveness of the medication, causing noncompliance by the patients. As a result of the persistence and recurrent nature of AD that varies in severity depending on the individual's age, there has been extensive work towards finding better treatment alternatives [9].

Atopic dermatitis (AD) is prevalent in approximately 2-20% of children globally. The disease is highly prevalent in population groups from Oceania, Africa, and the Asia-Pacific region in comparison with Northern and Eastern Europe and the Indian subcontinent. In adults, its prevalence lies in the range of 10-14%. Throughout the past decades, there has been an increased number of patients diagnosed with AD in Asia, but a very high level of its prevalence has remained unchanged in wealthy nations and urbanized environments. There exist a great variety of drugs applied for AD; however, all of them may provoke negative side effects due to their prolonged use. Thus, searching for alternatives became crucial, and herbal medicine seemed the most suitable option [10].

The application of herbal pharmacotherapy has become preferable to patients due to numerous positive aspects of its use in comparison with artificial pharmaceuticals (Table 1). First of all, herbal medication is less dangerous since the risk of adverse long-term health effects is extremely low in contrast with conventional medicine [11].

**Table 1: Plants Useful in the Management of Eczema:**

| Sr. No. | Botanical name and family                     | Active constituents                              | Part of plant       | Nature of extract             | Route of administration | Mechanism of action                                                                           | Reference |
|---------|-----------------------------------------------|--------------------------------------------------|---------------------|-------------------------------|-------------------------|-----------------------------------------------------------------------------------------------|-----------|
| 1.      | <i>Actinidia arguta</i><br>Actidinaceae       | Ascorbic acid, polyphenols                       | Fruit, Leaves, Stem | Aqueous                       | Oral                    | Inflammatory cells, lymphocyte↓, Foxp3 expression                                             | [12]      |
| 2.      | <i>Alnus japonica</i><br>Betulaceae           | Oregonin, hirsutenone                            | Bark, leaves        | Ethanol                       | Topical                 | Serum IgE↓<br>Eosinophil↓<br>COX-2↓                                                           | [13, 14]  |
| 3.      | <i>Aloe vera</i><br>Asphodeleceae             | Acemannan, aloin, vitamins                       | Leaves              | Aqueous, methanol and ethanol | Oral, Topical           | Inhibit Mast cell degranulation, Histamine↓, and TNF-α↓, inhibits NF-κB and COX-2 pathways    | [4]       |
| 4.      | <i>Althea officinalis</i><br>Malvaceae        | Flavonoids                                       | Leaves, roots       | Ethanol                       | Oral                    | Inhibition of TNF-α and IL-1β                                                                 | [15]      |
| 5.      | <i>Angelica sinensis</i><br>Apiaceae          | Ferulic acid                                     | Root                | Ethanol                       | Topical                 | IL-6↓, TNF-α↓, VEGF (Vascular endothelial growth factor) ↑, Matrix metalloproteinases (MMPs)↓ | [16]      |
| 6.      | <i>Artemisia scoparia</i><br>Asteraceae       | Scoparone, chlorogenic acid                      | Leaves              | Butanol                       | Oral                    | Inhibit TSLP, Histamine↓, suppress caspase-1                                                  | [12]      |
| 7.      | <i>Asarum sieboldii</i><br>Aristolochiaceae   | Asarone                                          | Whole plant         | 70% Ethanol                   | Oral                    | Th2 cytokines↓, JAK/STAT pathway inhibition, NF-κB signaling↓                                 | [17]      |
| 8.      | <i>Azadirachta indica (Neem)</i><br>Meliaceae | Nimbidin, nimbolide, gallic acid, polysacchrides | Leaves              | Alcohol                       | Topical                 | Anti-inflammatory (COX, LOX enzymes↓), immunomodulatory effect                                | [4]       |

|    |                                                    |                                              |                        |                            |         |                                                                |          |
|----|----------------------------------------------------|----------------------------------------------|------------------------|----------------------------|---------|----------------------------------------------------------------|----------|
| 9. | <i>Bambusa vulgaris</i><br>Poaceae                 | Phenolic acids, flavonoids                   | Leaves                 | 70% Ethanol                | Topical | IL-3↓, IL-4↓, IL-6↓, TNF-α↓                                    | [18]     |
| 10 | <i>Bambusa caulis</i><br>Poaceae                   | Flavonoids                                   | Stem                   | Ethanol                    | Topical | TEWL↓, Serum IgE↓, Eosinophil↓, TNF-α↓, IL-3↓, IL-4↓           | [14]     |
| 11 | <i>Betula platyphylla</i><br>Betulaceae            | Betulin, triterpenes                         | Leaf                   | Aqueous                    | Topical | Th2 cytokines↓, IgE↓                                           | [17]     |
| 12 | <i>Borago officinalis</i><br>Boraginaceae          | γ-linolenic acid                             | Flowers                | -                          | Oral    | IL-3↓, IL-4↓, IL-5↓                                            | [19]     |
| 13 | <i>Camellia Sinesis</i><br>(Green tea)<br>Theaceae | Epigallocatechin gallate (EGCG), polyphenols | Leaves                 | Polyphenol rich extract    | Topical | Antioxidant and anti-inflammatory effects                      | [20]     |
| 14 | <i>Cinnamomum cassia</i><br>Lauraceae              | Cinnamaldehyde, cinnamic acid                | Bark                   | Ethanol                    | Oral    | Serum IgE↓, TNF-α↓, TARC↓, Histamine                           | [14]     |
| 15 | <i>Cymbopogon schoenanthus proximus</i><br>Poaceae | Citral, limonene                             | Aerial parts           | Essential oil              | Topical | IL-6↓, TNF-α↓, NF-κB pathway activation↓                       | [21, 22] |
| 16 | <i>Ficus carica</i><br>Moraceae                    | Flavonoids                                   | Leaves                 | Aqueous, ethanol, Methanol | Oral    | TNF-α↓, IL-4↓, IL-6↓, COX↓                                     | [11]     |
| 17 | <i>Fumaria officinalis</i><br>Papaveraceae         | Fumaric acid                                 | Stems, Leaves, Flowers | Methanol : Ethanol= 3:7    | Oral    | TNF-α↓, IL-6↓, IL-1β↓                                          | [23]     |
| 18 | <i>Glycyrrhiza glabra</i><br>Leguminosae/ Abaceae  | Glycyrrhizin                                 | Roots                  | Methanol                   | Topical | Inhibits nuclear factor kappa-B and COX-2, IL-3↓, IL-4↓, IL-5↓ | [24]     |

|    |                                                               |                               |                    |                            |         |                                                                                                           |          |
|----|---------------------------------------------------------------|-------------------------------|--------------------|----------------------------|---------|-----------------------------------------------------------------------------------------------------------|----------|
| 19 | <i>Helianthus annuus</i><br>Asteraceae                        | Linoleic acid,<br>tocopherols | Leaves             | Aqueous                    | Oral    | Expression of molecule CD69↓<br>on surface of T-cells, serum IgE↓,<br>atopic cytokine mRNA in ear tissue↓ | [25]     |
| 20 | <i>Juglans regia</i><br>Juglandaceae                          | Juglone                       | Leaves             | Aqueous                    | Topical | Neutralize ROS,<br>IL-3↓,<br>IL-4↓,<br>TNF-α↓                                                             | [26]     |
| 21 | <i>Lindera obtusiloba</i><br>Lauraceae                        | Linderane,<br>flavonoids      | Leaves/Whole Plant | Water                      | Oral    | Th2 cytokines↓,<br>allergic responses↓                                                                    | [27, 28] |
| 22 | <i>Lithospermum erythrorhizon</i><br>(Shikon)<br>Boraginaceae | Shikonin                      | Root               | Ether                      | Topical | IL-1β↓, IL-6↓,<br>TNF-α, COX-2 expression↓,<br>Neutrophil infiltration↓                                   | [29]     |
| 23 | <i>Malva sylvestris</i><br>Malvaceae                          | Anthocyanins                  | Flowers            |                            | Topical | Anti-inflammatory and antioxidant activities                                                              | [19]     |
| 24 | <i>Matricaria Chamomilla</i><br>Asteraceae                    | Apigenin                      | Flowers            | Aqueous or ethanol extract | Topical | COX↓, LOX↓,<br>PG↓, leukotrienes↓                                                                         | [30]     |
| 25 | <i>Morus alba</i><br>Moraceae                                 | Resveratrol                   | Leaves             | Ethanol                    | Topical | NF- κB signaling pathway suppression, IgE levels↓, histamine release↓                                     | [31]     |
| 26 | <i>Nymphoides peltata</i><br>Menyanthaceae                    | Phenolic compounds            | Roots              | Aqueous                    | Topical | Regulation of genes filaggrin and kallikrein related peptidase-5, IL-4↓                                   | [32]     |
| 27 | <i>Phellodendron chinense</i><br>Rutaceae                     | Berberine                     | Bark               |                            | Topical | TNF-α↓, IL-1β↓,<br>IL-3↓, IL-4↓                                                                           | [33]     |

|     |                                                 |                                                       |         |                          |          |                                                                                                |          |
|-----|-------------------------------------------------|-------------------------------------------------------|---------|--------------------------|----------|------------------------------------------------------------------------------------------------|----------|
| 28  | <i>Platycodon grandiflorum</i><br>Campanulaceae | Platycodon D(saponin)                                 | Roots   | Ethanol, aqueous         | Oral     | IL-4↓, IL-13↓, IL-6↓, TNF-α↓                                                                   | [34, 35] |
| 29  | <i>Psidium guajava</i><br>Myrtaceae             | Quercetin, flavonoids                                 | Leaves  | Ethanol                  | Topical  | Clinical score↓, Serum IgE↓, TARC↓, IL-10↑, IFN-γ↓, IL-3↓, IL-4↓, IL-5↓                        | [14]     |
| 30  | <i>Rehmannia glutinosa</i><br>Orobanchaceae     | Flavonoids                                            | Roots   | Ethanol                  | Topical  | IL-4↓, TNF-α↓, TARC↓, ICAM-1↓, VCAM-1↓                                                         | [14]     |
| 31  | <i>Sophora flavescens</i><br>Fabaceae           | Matrine, oxy matrine                                  | Root    |                          | Oral     | IL-6↓, IL-8↓, IL-12↓                                                                           | [33]     |
| 32  | <i>Viola odorata</i><br>Violaceae               | Salicylic acid, flavonoids                            | Flowers |                          | Topical  | Soothing and anti-inflammatory effects                                                         | [19]     |
| 33. | <i>Rosmarinus officinalis</i><br>Lamiaceae      | Rosmarinic acid, luteolin, camphor                    | Flowers | Methanol, n-hexane       | In-vitro | NO↓, PGE2↓, free radical scavenging↓                                                           | [48]     |
| 34. | <i>Cocos nucifera</i><br>Arecaceae              | Lauric acid, caprylic acid, Capric acid, ferulic acid | Kernel  | Cold-pressed oil extract | Topical  | TEWL↓, Free radical activity↓, <i>Staphylococcus aureus</i> colonization↓                      | [49]     |
| 35. | <i>Vitellaria paradoxa</i><br>Sapotaceae        | α-amyrin, β-amyrin, butyrospermol                     | Seed    | Methanol                 | Topical  | NO↓, TNF-α ↓, IL-1β ↓, IL-12↓, iNOS and COX-2 mRNA expression ↓, NF-κB nuclear translocation ↓ | [50]     |

## DISCUSSION:

### *Actinidia arguta*

Oral administration of the aqueous extract of *Actinidia arguta* (fruits, leaves, and stems) diminished the migration of inflammatory cells and lymphocytes and

decreased Foxp3 expression. The Foxp3 protein acts as a critical transcription factor responsible for the differentiation of regulatory T cells. This indicates that the therapy exerts selective inhibition of the Th-1 mediated immune response. [1]. In one of the studies, PG102, an orally

administered agent, significantly attenuated dust mite extract-induced chronic eczema by reducing clinical symptoms like ear swelling and erythema and limiting lymphocytic infiltration. It suppressed pro-inflammatory T-cell responses while upregulating Foxp3 and other Treg-associated genes. Mechanistically, PG102 promoted differentiation of CD4<sup>+</sup> Foxp3<sup>+</sup> regulatory T-cells from native precursors, likely through inhibition of mTOR signaling and STAT-5 phosphorylation, independent of TGF- $\beta$  pathways. These findings position PG102 as a promising oral immunomodulator for chronic inflammatory disorders [2].

### *Alnus japonica*

Ethanol extract of barks and leaves of *Alnus japonica* significantly reduced serum IgE levels as well as eosinophils infiltration. It also reduced production of pro-inflammatory mediators, promoting a shift away from allergic inflammation. Oral and topical methods such as, capsules, creams and ointments are recommended [4]. Numerous classical histopathological manifestations characteristic of AD- such as erythema, hemorrhage, edema, desquamation, xerosis, excoriations, and erosions- were prominently observed in NC/Nga mice following the induction of AD- like lesions via exposure to Dermatophagoides (dust mite) antigens. The severity of dermatologic symptoms was quantitatively assessed using the modified SCORAD (Scoring Atopic

Dermatitis) index, as established by Hajime et al. (1998), Tomomi et al. (2001), and Takano et al. (2006). These assessments were conducted on a weekly basis, beginning from the initial sensitization phase and extending throughout the duration of the topical therapeutic intervention [3].

### *Aloe vera*

The extracts of *Aloe vera* leaves, including aqueous, methanol and ethanol fractions exhibit anti-inflammatory effects. These effects are mediated by impeding the exocytic degranulation of mast cells, thereby abrogating the downstream release of inflammatory mediators and immunomodulatory effectors, thus reduction of histamine release and downregulation of TNF- $\alpha$ . Additionally. The extracts suppress NF- $\kappa$ B and COX-2 signaling pathways, mitigate redness and swelling promote fibroblast proliferation, thereby facilitating tissue repair [51]. Its gel contains bioactive compounds like aloe-emodin and aloin, which provide anti-inflammatory and anti-microbial effects. Hemagglutinin, a lectin-like protein, reduces inflammation by binding to skin glycoproteins. Studies have shown effectiveness against bacteria like *Staphylococcus aureus* and fungi like *Trichophyton* species. These properties collectively support aloe vera's use in managing eczema-related skin damage [4].

### ***Althaea officinalis***

In Khalighi et al.'s (2021), the researchers performed a clinical investigation that involved conducting a double-blind randomized control trial involving 40 individuals who had moderate to severe cases of eczema. As a result, it was evident that there were statistically significant reductions in SCORAD score among the two treatment groups. By the fourth week of treatment, the *Althaea officinalis* product achieved comparable levels to the use of corticosteroid drugs on a clinically significant level. In addition, it was also noted to be well-tolerated without adverse effects [15].

### ***Angelica sinensis***

*Angelica sinensis* (AS), is an herb that has been used for generations to help promote healthy blood production and skin care. The efficacy of this medicinal plant was assessed using a mouse model that was made to suffer from atopic dermatitis through exposure to DNCB for 11 days (about 1.5 weeks). Additionally, it significantly reduced pruritic behavior and the expression of substance P. AS also downregulated pro-inflammatory cytokines (IL-4, IL-6, TNF- $\alpha$ , IFN- $\gamma$ ) and inhibited key signaling pathways, including NF- $\kappa$ B, phospho-I $\kappa$ B $\alpha$ , and MAPKs. These findings suggest that AS exerts potent antipruritic and anti-inflammatory effects and may serve as a promising therapeutic candidate for managing AD [16].

### ***Artemisia scoparia***

Extraction of leaves of *Artemisia scoparia* was carried out with butanol and when applied topically or consumed orally, it inhibited TSL and histamine release and suppressed caspase-1 activation, a key enzyme in inflammasome pathways. These mechanisms collectively attenuate the inflammatory cascade in Atopic dermatitis [12]. A total of 102 bioactive compounds—including flavonoids, coumarins, chromones, steroids, volatile oils, and phenolic acids have been isolated from *A. scoparia*, showing particularly anti-inflammatory and activity against microbes [36].

### ***Asarum sieboldii***

According to a study, BS012—a herbal combination that contains *Cinnamomum cassia*, *Platycodon grandiflorum*, and *Asarum sieboldii* have anti-atopic properties by lowering Th2-specific inflammatory responses. By orchestrating of the NF- $\kappa$ B and STAT signal transduction pathways, the formulation profoundly attenuated the transcriptional and translational upregulation of pro-inflammatory cytokines and chemokines within in vitro cellular system as well as in vivo physiological models. BS012 therapy significantly decreased AD-like skin lesions and inhibited Th2 cytokine expression in DNCB-induced mice models of atopic dermatitis. Human keratinocytes treated with TNF- $\alpha$ /IFN- $\gamma$  showed dose-dependent inhibition of pro-inflammatory

mediator expression, indicating that BS012 may facilitate the restoration of epidermal barrier integrity while ameliorating the clinical manifestations of eczema [17]. In a DNCB-induced mouse model, topical administration of this formulation dramatically decreased serum levels of immunoglobulin E (IgE), IFN- $\gamma$ , IL-4, IL-17, and TSLP, as well as dermatitis scores and mast cell infiltration. These results imply that *Asarum sieboldii* helps to ameliorate metabolic imbalances linked to AD symptoms and decrease Th2-mediated inflammatory responses [37].

#### ***Azadirachta indica***

*Azadirachta indica* (Neem) has long been esteemed in traditional medicine for its broad-spectrum bioactivity, notably its anti-inflammatory, anti-microbial, antioxidant, and immunomodulatory effects. Its pharmacological efficacy in eczema management is primarily attributed to secondary metabolites such as nimbidin, nimbolide, gallic acid, and polysaccharides, which downregulate pro-inflammatory cascades by inhibiting neutrophil and macrophage recruitment. Topical application or therapeutic bathing in neem extracts alleviates pruritus, erythema, and microbial colonization, common in AD. Additionally, neem enhances cutaneous barrier function and promotes homeostatic skin regeneration, offering a multifaceted approach to disease mitigation [38].

#### ***Bambusa vulgaris***

Derma gel is a combination of 50% *Bambusa vulgaris* leaf extract, 10% synergistic blend of phytochemicals present in olive oil, curcumin, coconut oil, and sweet gourd oil, and 40% Vaseline. The topical application gel was subjected to a safety and efficacy study in nine patients suffering from atopic dermatitis (AD). Patients were treated three times per day for three months, and the formulation proved to be effective in eight out of nine patients according to the TIS score. *Bambusa vulgaris* leaf extract demonstrated a remarkable anti-inflammatory action by suppressing the upstream signals responsible for the excessive production of pro-inflammatory cytokines like IL-3, IL-4, IL-6, and TNF- $\alpha$ . These preliminary findings suggest the potential usefulness of the extract in treating AD, but further clinical trials are needed to validate the hypothesis [18].

#### ***Bambusae caulis***

When *Bambusae caulis* is used topically or systemically, there have been empirical demonstrations that it can effectively lower TEWL in a significant way. Through this, the skin barrier properties are enhanced, allowing for improved protection against drying and irritation. Additionally, the inflammatory responses, including those from TNF- $\alpha$ , IL-4, and IL-6, as well as Th2 cytokines, are decreased through this natural compound. In majority of the studies, outcome assessments encompassed

clinical symptomatology, serum IgE concentrations, and the expression profiles of Th1- and/or Th2- associated cytokines and chemokines. Given the observed reductions in clinical severity scores, auricular or epidermal thickening, pruritic behavior, and histopathological markers of inflammation following topical administration of herbal formulations, it may be postulated that such interventions exert notable anti-inflammatory effects [27].

### ***Betula platyphylla***

*Betula platyphylla* extracts have proven to reduce allergic inflammation mediated by mast cells. According to research, these extracts can specifically lower histamine release and prevent nuclear factor-kappa B (NF- $\kappa$ B) and caspase-1 from activating stimulated human mast cells, which lowers the generation of inflammatory cytokines [17]. As for the other test, the mice were subjected to DNCB exposure, which is known to induce skin inflammation that resembles atopic dermatitis. The next step was administering *B. platyphylla* extract to the mice. As a result, several improvements were observed, including decreased skin inflammation and pro-inflammatory cytokines such as TNF- $\alpha$  and IL-6. Moreover, the treatment enhanced skin barrier restoration due to an increase in filaggrin. These results indicate that *B. platyphylla* extract may be used as an effective treatment for atopic dermatitis. [39].

### ***Borago officinalis***

The plant *Borago officinalis* (borage) has high levels of GLA (gamma linolenic acid), an omega-6 fatty acid. This compound acts to reduce inflammation through its ability to direct the formation of eicosanoids. Additionally, GLA has positive impacts on the lipids layer of the skin and helps to retain moisture within the skin to prevent trans-epidermal water loss, thus minimizing AD [19].

### ***Camellia sinensis***

*Camellia sinensis* polyphenols help to relieve symptoms and maintain the skin barrier. The polyphenols found in green tea, especially EGCG, have anti-inflammatory and antioxidant qualities. By controlling inflammatory pathways and scavenging free radicals, these substances aid in the reduction of inflammation [20].

### ***Cinnamomum cassia***

Ethanollic extract of bark of *Cinnamomum cassia* alleviates TARC levels which is indicative of reduced inflammation and immune cell recruitment. It also contributes to downregulation of Tumor Necrosis Factor (TNF)- $\alpha$  synthesis. A pivotal cytokine mediated systemic inflammatory response is reduced, suggesting anti-inflammatory activity. BS012 demonstrated significant anti-atopic efficacy in vivo as well as in vitro experimental paradigms of AD. In DNCB-induced mice, BS012 reduced dermatitis severity and suppressed Th2 cytokines and

transcriptional and translational regulation of TSLP [17].

### ***Cymbopogon schoenanthus***

The herbal ointment derived from the aerial parts of *Cymbopogon schoenanthus* proximus exhibits pronounced antimicrobial and anti-inflammatory pharmacological properties [21]. Essential oil (50, 100, 200 mg/kg, i.p.) was tested in mice using writhing, hot plate, and formalin tests for analgesic effects assessed alongside carrageenan-induced paw edema models to evaluate activity against inflammation. Controls received vehicle, morphine, or mefenamic acid. GC-MS revealed 31 components (86.8%) with piperitone (62%) as the major constituent. The oil showed no effect in writhing or hot plate tests but reduced pain in the chronic phase of the formalin test. It significantly inhibited inflammation in all doses, comparable to mefenamic acid ( $p < 0.05$ ). Hence, *C. schoenanthus* essential oil possesses notable anti-inflammatory and delayed analgesic properties due to its active constituents [40].

### ***Ficus carica***

In a well-conducted clinical trial conducted in a double-blinded and placebo-controlled manner, the therapeutic potential of an 8% aqueous extract of the fruit of *Ficus carica* L. called Melfi cream as a new topical agent for mild and moderate cases of atopic dermatitis in children was examined. The SCORAD index, pruritus, and severity score were all significantly

reduced when Melfi cream was tested against 1% hydrocortisone and placebo. Hydrocortisone had a favorable effect as well, however, Melfi cream proved more effective at alleviating pruritus and equally efficient in managing symptoms of atopic dermatitis. No side effects were noted. Being characterized by antioxidant, anti-inflammatory, antibacterial action, along with high polyphenol content, the extract may be regarded as a promising and affordable non-corticosteroid treatment choice [11].

### ***Fumaria officinalis***

*Fumaria officinalis* potentially lowering the inflammation linked to AD; it includes alkaloids and flavonoids with anti-inflammatory qualities. The plant's phenolic constituents exert potent anti-oxidative activity by neutralizing ROS, thereby safeguarding cutaneous cellular architecture from oxidative stress-induced perturbations intricately linked to pathogenesis of eczema. In conventional Asian medicine, it has significant value, having fumaric acid esters (FAEs), including dimethyl fumarate, known for their immunomodulatory activity (Raafat et al., 2020; Jowkar et al., 2011). FAEs have shown promise in combating resistant skin diseases through a mechanism similar to psoriasis (Litjens et al., 2006). According to Jowkar et al. (2014), using 5% fumaric acid cream is useful for erythema, while its efficacy against other signs is less than 0.1% triamcinolone. The use of Silymarin in conjunction with F.

officinalis may assist in atopic dermatitis control, but further evidence is required to develop it into medicine [23].

### ***Glycyrrhiza glabra***

The mechanism through which the methanolic extract of *Glycyrrhiza glabra* exerts its anti-inflammatory activity is via inhibition of the NF- $\kappa$ B and COX-2 pathways. Furthermore, it helps to inhibit the inflammatory process through a reduction in the levels of cytokines such as IL-3, IL-4, and IL-5. The glycyrrhizic acid (GA) loaded into the hydrogel employs native bacterial cellulose as the matrix material, which capitalizes on its excellent hydration ability, a significant feature in eczema treatment. Experiments conducted in vitro confirmed the hydrogel formation, excellent skin permeation, and higher efficacy compared to GA alone in an acute DNCB-induced eczema mouse model [24].

### ***Helianthus annuus***

The aqueous leaf extract of *Helianthus annuus* displays impressive immunomodulating and antiallergic properties. It greatly reduces the expression of the T-cell activation marker CD69 on the cell surface, thus suppressing the activity of T-cells and the entire immune system. It significantly reduces the level of IgE in the blood serum and prevents the upregulation of the transcription of atopic cytokines in the ear tissues, reducing erythema, inflammation, and hypersensitivity [25].

### ***Juglans regia***

The aqueous extract of *Juglans regia* leaves possesses significant antioxidant and anti-inflammatory properties. It neutralizes ROS and alleviates levels of IL-3, IL-4, and TNF- $\alpha$ . This combined effect results in attenuation of inflammatory responses and reduction of redness [26].

### ***Lindera obtusiloba***

In one of the experiments, the effects of *Lindera obtusiloba* water extract (LOWE) in a mouse model of atopic dermatitis induced by *Dermatophagoides farinae* extract and 2,4-dinitrochlorobenzene have been evaluated. The application of the LOWE on mice's skin led to an impressive improvement in the manifestations of AD including increased thickness of the skin, accumulation of mast cells, and increased serum IgE and histamine levels. Importantly, a significant reduction of local overexpression of several Th2 cytokines, including IL-4, IL-13, IL-31, and TNF- $\alpha$ , was observed. Thus, the findings confirm the high potential of LOWE as an anti-inflammatory and immunomodulatory agent [28].

### ***Lithospermum erythrorhizon***

Another study focused on the potential of *Lithospermum erythrorhizon* (LE) in managing atopic dermatitis (AD) in mice through a DNCB-induced model. When administered orally to the experimental subjects, LE showed a significant

reduction in all major hallmarks of AD, including skin inflammation, ear edema, and scratching behavior. In addition, LE was effective in suppressing serum immunoglobulin E (IgE), histamine, and interleukin-4 (IL-4) and promoting the regulation of the T helper cell 1/T helper cell 2 (Th1/Th2) balance by upregulating IL-12 and interferon gamma (IFN- $\gamma$ ) while simultaneously inhibiting the activity of Th2, Th17, and Th22-related cytokines. Moreover, LE attenuated the inflammatory signaling pathways both systemically and locally in the skin by reducing the levels of TNF- $\alpha$ , IL-6, thymic stromal lymphopoietin (TSLP), and monocyte chemoattractant protein-1 (MCP-1). Most notably, LE facilitated the recovery of the skin barrier through increased expression of structural proteins like filaggrin, involucrin, loricrin, occludin, and ZO-1. [29]. In yet another research work, a combination of network pharmacology and SRWR was used to identify vital targets for the bioactive constituents of LE, which act on important mediators like PTGS2, HSP90AA1, and MAPK14. This method helped in accurate target prediction and minimization of false positives [41].

### ***Malva sylvestris***

***Malva sylvestris* (Common Mallow)** is rich in mucilage, flavonoids, and phenolic substances that possess anti-inflammatory and antioxidant activities. These ingredients assist in lowering oxidative stress and skin inflammation caused by atopic dermatitis. Moreover, the herb has

been observed to promote wound healing, thus proving advantageous in the management of skin lesions caused by AD [19]. It has been demonstrated that topical administration of *M. sylvestris* extract lowers interleukin-1 $\alpha$  (IL-1 $\alpha$ ) levels, a crucial pro-inflammatory cytokine implicated in skin inflammation. The inflammatory response is lessened by *M. sylvestris* extract, which inhibits the migration of polymorphonuclear leukocytes (such as neutrophils) into inflammatory skin tissue [42].

### ***Matricaria chamomilla***

Since *chamomile* has potent anti-inflammatory qualities, it can help cure atopic dermatitis (AD), a chronic inflammatory skin disorder. To increase its therapeutic effectiveness, chamomile volatile oil was combined with nano emulsion gels (CVO-NEGs) in this study. It works by lowering mast cell infiltration, skin injury, and epidermal thickening while inhibiting IgG and inflammatory cytokines [37]. Active ingredients in *Matricaria chamomilla*, such as chamazulene and  $\alpha$ -bisabolol, have anti-inflammatory effects which work by blocking the COX and LOX pathways. Furthermore, apigenin and luteolin, which are two of its flavonoids, support its antioxidant action and aid in reducing the oxidative stress linked to eczema. People with atopic dermatitis have reported less itching and irritation of their skin when they applied *chamomile* extract topically. According to some research, chamomile creams may ease eczema

symptoms just as well as low-dose hydrocortisone [20].

### ***Morus alba***

*Morus alba* extract (MAE), prepared from its leaves using 70% ethanol, was investigated for its anti-atopic effects in vivo and in vitro. MAE significantly inhibited nitric oxide and prostaglandin E2 production in LPS stimulated RAW 264.7 macrophages and suppressed TARC (CCL17) production in TNF- $\alpha$ /IFN- $\gamma$ -stimulated HaCaT keratinocytes. In a mouse model of house dust mite-induced AD (NC/Nga mice), topical MAE application markedly reduced clinical severity, histopathological skin damage, and plasma levels of IgE and histamine. These findings suggest MAE mitigates atopic inflammation via suppression of pro-inflammatory mediators and may serve as a promising therapeutic candidate for AD [31].

### ***Nymphoides peltata***

*Nymphoides peltata* root ethanol extract (NPR) demonstrated potent anti-atopic and antioxidant effects in both in vitro and in vivo mouse models of AD [32]. The extract significantly inhibited IL-4 and IgE expression, reduced mast cell infiltration and epidermal thickness, and alleviated AD-like symptoms in mice. It also improved skin hydration and modulated skin barrier-related genes, partly through activation of the Nrf2/HO-1 pathway. HPLC analysis identified chlorogenic acid, 3,5- dicaffeoylquinic acid, and 3,4-

dicaffeoylquinic acid as key active constituents. These findings suggest NPR extract as a promising natural agent for managing AD through combined anti-inflammatory and skin barrier-enhancing mechanisms [43].

### ***Phellodendron chinense***

Berberine is the main active constituent of *Phellodendron chinense*. Nuclear factor kappa B (NF- $\kappa$ B) signaling constitutes a pivotal regulatory axis driving the transcriptional induction of key pro-inflammatory cytokines, notably TNF- $\alpha$  and IL-6, and berberine suppresses this pathway. *P. chinense* lowers skin inflammatory reactions by inhibiting this mechanism. Both serum IgE levels and histamine release, which are increased in allergic dermatitis, can be decreased by berberine. Inhibits germs that colonize the skin, like *Staphylococcus aureus*, which lowers the chance of subsequent infections in eczematous skin [33]. In a study to treat atopic dermatitis, *Phellodendron chinense* was assessed as a component of the Penta herbs formulation (PHF). Cell viability remained above 50%, and despite its poor embryotoxicity *in vitro*, it did not significantly irritate the skin. According to these findings, *P. chinense* is generally safe to use topically, while usage during pregnancy may need to be avoided [44].

### ***Platycodon grandiflorum***

*Platycodon grandiflorum* root saponins (CKS), particularly *platycodon D*, exhibit potent anti-atopic effects by modulating

key inflammatory and oxidative pathways [17]. In a DNCB-induced AD mouse model, oral CKS significantly reduced dermatitis severity, IgE/TARC levels, and Th1/Th2 cytokine expression, alongside histological improvements. *In vitro*, CKS and *platycodon D* suppressed TNF- $\alpha$ /IFN- $\gamma$ -induced TARC expression via inhibition of NF- $\kappa$ B and STAT-1 signaling, while concurrently activating the Nrf/ARE-HO-1 pathway. These findings suggest their potential as effective, multi-targeted phytotherapeutics for AD [34].

### ***Psidium guajava***

Ethanollic extract of leaves of *Psidium guajava* aids in cytokine modulation i.e., increases IL-10 and decreases IL-3, IL-4, IL-5, interferons- $\gamma$  supporting dual suppression of both Th-1 and Th-2 immune response and so exhibiting broad anti-inflammatory and immunomodulatory effects. It is predominantly administered in a capsular dosage form which has anti-microbial activity during infection of eczema [45].

### ***Rehmannia glutinosa***

*Rehmannia glutinosa* demonstrated therapeutic potential in an NC/Nga mouse model of AD. Topical application of its extract significantly reduced clinical severity and ear thickness, and suppressed local IL-4 and TNF- $\alpha$  expression, along with inflammatory chemokines such as TARC, MDC, Regulated on activation, normal T cell expressed and secreted, Intercellular adhesion molecule-1, VCAN-

1. However, it did not reduce systemic serum IgE levels, indicating a localized anti-inflammatory effect primarily via Th2 cytokine modulation. The extract may inhibit allergic skin inflammation by downregulating Th2-dominant responses and related pro-inflammatory mediators, although longer-term studies or allergen-specific IgE assessments are needed for comprehensive evaluation [14]. *Rehmanniae Radix* also shows similar anti-inflammatory effect [46].

### ***Sophora flavescens***

*Sophora flavescens* has main active constituents like matrine and oxymatrine. Matrine inhibits the cytokines IL-4, IL-5, and IL-13, which are linked to Th2-driven inflammation in AD. As a result, the immune system becomes less prone to allergic (Th2-dominant) reactions. It mitigates inflammatory and irritative processes by attenuating histamine release and inhibiting mast cell degranulation has been demonstrated that oxymatrine inhibits the JAK/STAT signaling pathway, which is a key regulator of cytokine signaling and chronic inflammation in AD [33].

### ***Viola odorata***

*Viola odorata* (sweet violet) Compounds with anti-inflammatory properties like flavonoids and saponins found in *V. odorata* may lessen the inflammatory response in AD. The antioxidants in the plant aid in scavenging free radicals, shielding skin cells from the oxidative stress linked to AD. Its extracts elicit a

pronounced induction of the immunoregulatory cytokines IL-10, while concurrently effectuating a robust downregulation of pro-inflammatory mediators involving IL-12, IL-32, TNF- $\alpha$  thereby modulating the inflammatory milieu toward resolution. Studies employing RAW 264.7 macrophage cells have shown this immune response regulation, suggesting possible advantages in inflammatory situations [19]. Treatment with *Viola odorata* resulted in the NF- $\kappa$ B signaling pathway being down-regulated. This resulted in decreased expression of inflammatory markers, such as COX-2, TNF- $\alpha$ , and IL-1 $\beta$ . HPLC analysis revealed that there were bioactive molecules like quercetin, chlorogenic acid, gallic acid, and other phenolic acids, which might have played a role in the pharmacological activities seen [47].

### ***Rosmarinus officinalis***

*Rosmarinus officinalis* L. was evaluated for its biological activities through solvent partitioning of the flowers. The analysis by GC-MS and LC-MS showed the presence of important phytochemicals such as rosmarinic acid, luteolin, and camphor in polar and nonpolar fractions. The polar fraction showed high antioxidant activity, whereas the n-hexane fraction exhibited antibacterial activity against *S. aureus* (MIC 78  $\mu$ g/mL). Both fractions exhibited a significant decrease in NO and PGE2 levels induced by LPS in vitro. Overall, this data suggests that the biological effects

of *R. officinalis* are mainly attributed to its phenols and volatiles [48].

### ***Cocos nucifera***

In a well-designed randomized, double-blind study, the application of *Cocos nucifera* (virgin coconut oil) (VCO) on the skin of children suffering from mild to moderate atopic dermatitis was found to be remarkably better than mineral oil. The reduction in SCORAD index in the VCO group was a remarkable 68.23%, compared to a 38.13% reduction in the mineral oil group ( $P < 0.001$ ). There was a significantly greater number of children who had moderate to excellent clinical response in the VCO group. In addition, there was evidence of improved skin barrier properties through biophysical studies: reduced TEWL and increased SC capacitance in the VCO group. Overall, the study results emphasized the superiority of VCO in terms of improving skin barrier function and alleviating inflammation in pediatric atopic dermatitis [49].

### ***Vitellaria paradoxa***

The extract obtained from Shea butter (*Vitellaria paradoxa*) displays strong anti-inflammatory properties through inhibition of the pro-inflammatory reaction caused by LPS in J774 murine macrophages. This extract downregulates the production of nitric oxide, TNF- $\alpha$ , IL-1 $\beta$ , and IL-12 in a dose-dependent manner. Furthermore, it decreases the levels of the pro-inflammatory factors iNOS and COX-2 both at protein and RNA level. As far as

the mechanisms are concerned, the extract inhibits NF- $\kappa$ B activation via prevention of its phosphorylation and nuclear translocation. The obtained findings provide information regarding the molecular mechanisms underlying the effects of shea butter [50].

## CONCLUSION:

Atopic dermatitis is a persistent disease that involves chronic inflammation of the skin caused by an interplay between genetic predispositions, aberrations within the immune system, and several environmental determinants. Standard therapies provide symptomatic relief but pose issues regarding safety and potential adverse reactions, hence increasing the demand for alternative remedies. This review underscores the potential of various herbs explored over the past decade for eczema treatment. Numerous phytochemicals exhibit potent anti-inflammatory, immunomodulating, and cutaneous barrier protective effects by suppressing major cytokine pathways, inhibiting Th2 cell-mediated immune responses, and promoting skin hydration.

Herbal pharmacotherapy emerges as a plausible complementary or alternative modality distinguished by its natural deviation, favorable safety profile, and potential for sustained therapeutic benefit. The botanical agents appraised herein-such as *Bambusae caulis*, *Alnus japonica*, *Cinnamomum cassia*, *Psidium guajava*, *Azadirachta indica*, *Matricaria*

*chamomilla* among others-have demonstrated marked efficacy in mitigating clinical manifestations, suppressing inflammatory mediators, and rectifying immune dysregulation characteristic of eczema. However, despite all these positive outcomes, implementation of herbal therapies into regular practice needs careful trials and investigations into the mechanism of action. These are necessary to provide optimal dosing and ensure that preparations for phytopharmaceuticals have been optimized and standardized to achieve the best possible results. Ultimately, application of herbal treatments for eczema offers much promise and may result in more patient-friendly therapy than ever before.

## ABBREVIATIONS:

### Diseases / Conditions / Models:

- **AD** – Atopic Dermatitis
- **NC/Nga** – A strain of mice commonly used as an animal model for atopic dermatitis
- **DNCB** – 2,4-Dinitrochlorobenzene (chemical used to induce AD in experimental models)
- **SCORAD** – Scoring Atopic Dermatitis (clinical tool to assess AD severity)
- **J774 murine macrophages**– Mouse-derived macrophage-like cell line
- **HaCaT**: Human adult low calcium high temperature keratinocytes

- **RAW 264.7:** murine (mouse) macrophage cell line derived from Abelson leukemia virus-induced tumor in BALB/c mice

#### **Immune Cells / Markers / Proteins:**

- **Th-1** – T helper type 1 cell
- **Th-2** – T helper type 2 cell
- **Th-17** – T helper type 17 cell
- **Th-22** – T helper type 22 cell
- **CD4+** – Cluster of Differentiation 4 positive T cells (helper T cells)
- **CD69** – Early activation marker on T cells
- **Foxp3** – Forkhead box P3 (marker of regulatory T cells)
- **TARC** – Thymus and Activation-Regulated Chemokine (CCL17)
- **MCP-1** – Monocyte Chemoattractant Protein-1
- **CCL17** – Chemokine (C-C motif) ligand 17 (same as TARC)

#### **Genes / Receptors / Proteins:**

- **FLG** – Filaggrin gene
- **IgE** – Immunoglobulin E
- **Fc̑RI** – High-affinity IgE receptor I
- **ICAM-1** – Intercellular Adhesion Molecule 1
- **VCAM-1** – Vascular Cell Adhesion Molecule 1
- **TSLP** – Thymic Stromal Lymphopoietin
- **VEGF** – Vascular Endothelial Growth Factor

- **MMP** – Matrix Metalloproteinase
- **PTGS2** – Prostaglandin-Endoperoxide Synthase 2 (COX-2 gene)
- **COX-2** – Cyclooxygenase-2
- **LOX-2** – Lipooxygenase-2
- **iNOS** – Inducible Nitric Oxide Synthase
- **HSP90AA1** – Heat Shock Protein 90 Alpha Family Class A Member 1
- **MAPK14** – Mitogen-Activated Protein Kinase 14
- **VCAN-1** – Versican core protein
- **mTOR** – Mammalian Target of Rapamycin
- **STAT-5** – Signal Transducer and Activator of Transcription 5
- **phospho-ȊBz#** – Phosphorylated Inhibitor of ȊB alpha
- **MAPKs** – Mitogen-Activated Protein Kinases
- **Nrf2** – Nuclear Factor Erythroid 2–Related Factor 2
- **ARE/HO-1** – Antioxidant Response Element / Heme Oxygenase-1
- **ZO-1** – Zonula Occludens-1 (tight junction protein)
- **LOWE** – *Lindera obtusiloba* water extract

#### **Cytokines / Interleukins / Signaling Molecules:**

- **IL-1̑** – Interleukin 1 beta

- **IL-3** – Interleukin 3
- **IL-4** – Interleukin 4
- **IL-5** – Interleukin 5
- **IL-6** – Interleukin 6
- **IL-8** – Interleukin 8 (decreased expression)
- **IL-10** – Interleukin 10
- **IL-12** – Interleukin 12
- **IL-13** – Interleukin 13
- **IL-31** – Interleukin 31
- **IL-32** – Interleukin 32
- **TNF- $\alpha$**  – Tumor Necrosis Factor alpha
- **TGF- $\beta$**  – Transforming Growth Factor beta
- **IFN- $\gamma$**  – Interferon gamma
- **ROS** – Reactive Oxygen Species

#### **Pathways / Transcription Factors:**

- **NF- $\kappa$ B** – Nuclear Factor kappa-light-chain-enhancer of activated B cells
- **JAK/STAT** – Janus Kinase / Signal Transducer and Activator of Transcription
- **Nrf2** – Nuclear Factor Erythroid 2-Related Factor 2
- **ARE** – Antioxidant Response Element
- **I $\kappa$ B $\alpha$**  – Inhibitor of kappa B alpha

#### **Oils / Extracts / Substances / Natural Products:**

- **PG** – Prostaglandin (also can mean Propylene Glycol depending on context)
- **VCO** – Virgin Coconut Oil
- **SBE** – Shea Butter Extract
- **CVO-NEGs** – Chamomile Volatile Oil–Nano Emulsion Gels
- **EGCG** – Epigallocatechin Gallate
- **GLA** – Gamma Linolenic Acid
- **PHF** – Polyherbal Formulation
- **LOWE** – *Lindera obtusiloba* water extract
- **FAE** – Fumaric Acid Ester
- **LPS** – Lipopolysaccharide
- **BS012** – Herbal combination that contains *Cinnamomum cassia*, *Platycodon grandiflorum*, and *Asarum sieboldii*

#### **Techniques / Instruments:**

- **GC-MS** – Gas Chromatography–Mass Spectrometry
- **LC-MS** – Liquid Chromatography–Mass Spectrometry
- **HPLC** – High-Performance Liquid Chromatography
- **mRNA** – Messenger Ribonucleic Acid

#### **Other:**

- **SRWR** – Skin Recovery Water Retention

## CONFLICT OF INTEREST:

The authors report no potential financial or other conflicts of interest that might be viewed as exerting undue influence on their research reported herein.

## AUTHOR CONTRIBUTION:

All authors have made equal contributions to the literature search, critical analysis, concept development, writing, and editing of the manuscript.

## REFERENCE:

1. Eichenfield, L. F., Tom, W. L., Chamlin, S. L., Feldman, S. R., Hanifin, J. M., Simpson, E. L., ... & Sidbury, R. (2014). Guidelines of care for the management of atopic dermatitis: section 1. Diagnosis and assessment of atopic dermatitis. *Journal of the American Academy of Dermatology*, 70(2), 338-351.
2. Lifschitz, C. (2015). The impact of atopic dermatitis on quality of life. *Annals of Nutrition and Metabolism*, 66(Suppl. 1), 34-40.
3. Frazier, W., & Bhardwaj, N. (2020). Atopic dermatitis: diagnosis and treatment. *American Family Physician*, 101(10), 590-598.
4. Arora, P., Shiveena, B., Garg, M., Kumari, S., & Goyal, A. (2022). Curative potency of medicinal plants in management of eczema: a conservative approach. *Phytomedicine Plus*, 2(2), 100256.
5. Park, B. K., Park, Y. C., Jung, I. C., Kim, S. H., Choi, J. E., Park, S., ... & Jin, M. (2014). Oral administration of SSC201, a medicinal herbal formula, suppresses atopic dermatitis-like skin lesions. *Journal of Medicinal Food*, 17(4), 496-504.
6. Szczepańska, M., Blicharz, L., Nowaczyk, J., Makowska, K., Goldust, M., Wacękiel-Burnat, A., ... & Rudnicka, L. (2022). The role of the cutaneous mycobiome in atopic dermatitis. *Journal of Fungi*, 8(11), 1153.
7. Carr, W. W. (2013). Topical calcineurin inhibitors for atopic dermatitis: review and treatment recommendations. *Pediatric Drugs*, 15(4), 303-310.
8. Siriyong, T., Phungtammasan, S., Jansorn, S., Chonsongkram, N., Chanwanitsakul, S., Subhadhirasakul, S., & Voravuthikunchai, S. P. (2020). Traditional Thai herbal medicine as an alternative treatment for refractory chronic eczema. *Explore*, 16(4), 242-249.
9. Roekevisch, E., Spuls, P. I., Kuester, D., Limpens, J., & Schmitt, J. (2014). Efficacy and safety of systemic treatments for moderate-to-severe atopic dermatitis: a systematic review.

- Journal of Allergy and Clinical Immunology*, 133(2), 429-438.
10. Limantara, N. V., Sadono, R., Widhiati, S., & Danarti, R. (2023). Asian herbal medicine for atopic dermatitis: a systematic review. *Dermatology Reports*, 16(1), 9727.
  11. Abbasi, S., Kamalinejad, M., Babaie, D., Shams, S., Sadr, Z., Gheysari, M., ... & Rakhshandeh, H. (2017). A new topical treatment of atopic dermatitis in pediatric patients based on *Ficus carica* L.(Fig): A randomized, placebo-controlled clinical trial. *Complementary Therapies in Medicine*, 35, 85-912.
  12. Mandlik, D. S., & Mandlik, S. K. (2021). Atopic dermatitis: new insight into the etiology, pathogenesis, diagnosis and novel treatment strategies. *Immunopharmacology and Immunotoxicology*, 43(2), 105-125.
  13. Choi, S. E., Park, K. H., Jeong, M. S., Kim, H. H., Lee, D. I., Joo, S. S., ... & Lee, M. W. (2011). Effect of *Alnus japonica* extract on a model of atopic dermatitis in NC/Nga mice. *Journal of Ethnopharmacology*, 136(3), 406-413.
  14. Yun, Y., Kim, K., Choi, I., & Ko, S. G. (2014). Topical herbal application in the management of atopic dermatitis: a review of animal studies. *Mediators of Inflammation*, 2014(1), 752103.
  15. Khalighi, N., Jabbari-Azad, F., Barzegar-Amini, M., Tavakkol-Afshari, J., Layegh, P., & Salari, R. (2021). Impact of *Althaea officinalis* extract in patients with atopic eczema: a double-blind randomized controlled trial. *Clinical Phytoscience*, 7(1), 73.
  16. Lee, J., Choi, Y. Y., Kim, M. H., Han, J. M., Lee, J. E., Kim, E. H., ... & Yang, W. M. (2016). Topical application of *Angelica sinensis* improves pruritus and skin inflammation in mice with atopic dermatitis-like symptoms. *Journal of Medicinal Food*, 19(1), 98-105.
  17. Lee, G., Park, J., Lee, H., Kim, K. S., Park, J. H., Kyung, S. Y., ... & Jung, B. H. (2023). Anti-inflammatory effect and metabolic mechanism of BS012, a mixture of *Asarum sieboldii*, *Platycodon grandiflorum*, and *Cinnamomum cassia* extracts, on atopic dermatitis in vivo and in vitro. *Phytomedicine*, 115, 154818.
  18. Len, T. T., Nhung, H. N., & Thanh, T. C. (2023). A case series evaluating the efficacy of herbal oil-based *Bambusa vulgaris* (Bamboo) leaf extracts as a topical treatment for atopic dermatitis. *Phytomedicine Plus*, 3(4), 100480.
  19. Choopani, R., Mehrbani, M., Fekri, A., & Mehrbani, M. (2017). Treatment of atopic dermatitis from the perspective of traditional Persian medicine: presentation of a novel therapeutic approach. *Journal of*

*Evidence-based Complementary & Alternative Medicine*, 22(1), 5-11.

20. Kim, M. J., Yang, Y. J., Min, G. Y., Heo, J. W., Son, J. D., You, Y. Z., ... & Park, K. I. (2025). Anti-inflammatory and antioxidant properties of *Camellia sinensis* L. extract as a potential therapeutic for atopic dermatitis through NF- $\kappa$ B pathway inhibition. *Scientific Reports*, 15(1), 2371.
21. Shimizu, T. (2013). Efficacy of kampo medicine in treating atopic dermatitis: an overview. *Evidence Based Complementary and Alternative Medicine*, (1), 260235.
22. Golestaneh Talaei, M., & Jahandideh, M. (2019). Anti-inflammatory activity of *Cymbopogon schoenanthus* essential oil in animal models. *Research Journal of Pharmacognosy*, 6(3), 61-68.
23. Iraj, F., Makhmalzadeh, B. S., Abedini, M., Aghaei, A., & Siahpoush, A. (2022). Effect of herbal cream containing *Fumaria officinalis* and *silymarin* for treatment of eczema: A randomized double-blind controlled clinical trial. *Avicenna Journal of Phytomedicine*, 12(2), 155.
24. Tan, L., Wu, S., Liu, L., Wu, S., Wang, Q., Wang, C., ... & Li, G. (2025). Bacterial cellulose-based gel of glycyrrhizic acid gel for atopic dermatitis: design, optimization, in vitro and in vivo investigation. *International Journal of Biological Macromolecules*, 286, 138425.
25. So, J., & Nam, G. (2023). Study on Effect of Skin Soothing by *Helianthus annuus* (Sunflower) Seed Oil. *Journal of the Society of Cosmetic Scientists of Korea*, 49(4), 331-339.
26. Besrou, N., Oludemi, T., Mandim, F., Pereira, C., Dias, M. I., Sokoviæ, M., ... & Barros, L. (2022). Valorization of *Juglans regia* leaves as cosmeceutical ingredients: bioactivity evaluation and final formulation development. *Antioxidants*, 11(4), 677.
27. Suh, W. M., Park, S. B., Lee, S., Kim, H. H., Suk, K., Son, J. H., ... & Kim, S. H. (2011). Suppression of mast-cell-mediated allergic inflammation by *Lindera obtusiloba*. *Experimental Biology and Medicine*, 236(2), 240-246.
28. Choi, E. J., Lee, S., Kim, H. H., Singh, T. S., Choi, J. K., Choi, H. G., ... & Kim, S. H. (2011). Suppression of dust mite extract and 2, 4-dinitrochlorobenzene-induced atopic dermatitis by the water extract of *Lindera obtusiloba*. *Journal of Ethnopharmacology*, 137(1), 802-807.
29. Oh, J. S., Lee, S. J., & Choung, S. Y. (2021). *Lithospermum erythrorhizon* alleviates atopic dermatitis-like skin

- lesions by restoring immune balance and skin barrier function in 2,4-dinitrochlorobenzene-induced NC/Nga mice. *Nutrients*, 13(9), 3209.
30. de Padua Goulart Amatto, P., de Avila Orso, V. C. E. S., da Silva Coppede, J., Barbosa Galvão, M. C., de Castro França, S., Carvalho, J. C. T., ... & Pereira, A. M. S. (2024). Herbal medicines used in the treatment of atopic dermatitis: a systematic review and meta-analysis. *Archives of Dermatological Research*, 316(9), 656.
  31. Lim, H. S., Ha, H., Lee, H., Lee, J. K., Lee, M. Y., & Shin, H. K. (2014). *Morus alba* L. suppresses the development of atopic dermatitis induced by the house dust mite in NC/Nga mice. *BMC Complementary and Alternative Medicine*, 14(1), 139.
  32. Kim, T. Y., Park, N. J., Jegal, H., Paik, J. H., Choi, S., Kim, S. N., & Yang, M. H. (2023). *Nymphoides peltata* root extracts improve atopic dermatitis by regulating skin inflammatory and anti-oxidative enzymes in 2, 4-dinitrochlorobenzene (DNCB)-induced SKH-1 hairless mice. *Antioxidants*, 12(4), 873.
  33. Wang, M. C., Chou, Y. T., Kao, M. C., Lin, Q. Y., Chang, S. Y., & Chen, H. Y. (2023). Topical Chinese herbal medicine in treating atopic dermatitis (eczema): A systematic review and meta-analysis with core herbs exploration. *Journal of Ethnopharmacology*, 317, 116790.
  34. Choi, J. H., Jin, S. W., Han, E. H., Park, B. H., Kim, H. G., Khanal, T., ... & Jeong, H. G. (2014). *Platycodon grandiflorum* root-derived saponins attenuate atopic dermatitis-like skin lesions via suppression of NF- $\kappa$ B and STAT1 and activation of Nrf2/ARE-mediated heme oxygenase-1. *Phytomedicine*, 21(8-9), 1053-1061.
  35. Lee, G., Jung, B. H., Lee, T., Park, J. H., Kim, H. S., Kim, H., & Yang, H. O. (2024). Exploring the Metabolic Effects of an Herbal Remedy of *Asarum sieboldii*, *Platycodon grandiflorum*, and *Cinnamomum cassia* Extracts: Unraveling Its Therapeutic Potential as a Topical Application for Atopic Dermatitis Treatment. *Antioxidants*, 13(5), 563.
  36. Ding, J., Wang, L., He, C., Zhao, J., Si, L., & Huang, H. (2021). *Artemisia scoparia*: Traditional uses, active constituents and pharmacological effects. *Journal of Ethnopharmacology*, 273, 113960.
  37. Xu, J., Hu, H., Qian, X., Zhang, D., Chen, G., Zhang, F., ... & Chen, G. (2024). Therapeutic effects of *chamomile* volatile oil nanoemulsion/*Bletilla striata* polysaccharides gels on atopic dermatitis. *International Journal of Biological Macromolecules*, 277, 134404.

38. Debjit Bhowmik, D. B., Chiranjib, C., Jitender Yadav, J. Y., Tripathi, K. K., & Kumar, K. P. S. (2010). Herbal remedies of *Azadirachta indica* and its medicinal application.
39. Mohd Kasim, V. N. K., Noble, S. M., Liew, K. Y., Tan, J. W., Israf, D. A., & Tham, C. L. (2022). Management of atopic dermatitis via oral and topical administration of herbs in murine model: A systematic review. *Frontiers in Pharmacology*, 13, 785782.
40. Golestaneh Talaei, M., & Jahandideh, M. (2019). Anti-inflammatory activity of *Cymbopogon schoenanthus* essential oil in animal models. *Research Journal of Pharmacognosy*, 6(3), 61-68.
41. Wang, T., You, W., Zhao, L., Zhang, B., & Wang, H. (2023). Network pharmacology revealed the mechanisms of action of *Lithospermum erythrorhizon* sieb on atopic dermatitis. *Clinical, Cosmetic and Investigational Dermatology*, 651-658.
42. Prudente, A. S., Loddi, A. M., Duarte, M. R., Santos, A. R., Pochapski, M. T., Pizzolatti, M. G., ... & Otuki, M. F. (2013). Pre-clinical anti-inflammatory aspects of a cuisine and medicinal millennial herb: *Malva sylvestris* L. *Food and Chemical Toxicology*, 58, 324-331.
43. Kim, Y. A., Kim, D. H., Park, C. B., Park, T. S., & Park, B. J. (2018). Anti-inflammatory and skin-moisturizing effects of a flavonoid glycoside extracted from the aquatic plant *Nymphoides indica* in human keratinocytes. *Molecules*, 23(9), 2342.
44. Li, L., Mou, X., Xie, H., Zhang, A., Li, J., Wang, R., ... & Fan, X. (2023). In vitro tests to evaluate embryotoxicity and irritation of Chinese herbal medicine (Pentaherbs formulation) for atopic dermatitis. *Journal of Ethnopharmacology*, 305, 116149.
45. Dehideniya, K. M. S. A. K., Bulugahapitiya, V. P., Sabaragamuwa, R. S., & Kananke, T. C. (2025). Anti-diabetic, lipid peroxidase, antimicrobial, antioxidant, general toxicity properties and polyphenol content of *Psidium guajava*, *Garcinia quaesita*, and *Cinnamon verum* extracts along with their potential as nutraceuticals. *Food and Humanity*, 4, 100537.
46. Jung, J. H., & Kim, G. J. (2015). Anti-inflammatory effects of herbal medicines (*Rubus coreanus*, *Rehmanniae Radix*, *Houttuynia cordata*, *Betulae cortex*) EtOH extract on acute atopic dermatitis mice. *The Journal of Korean Medicine Ophthalmology and Otolaryngology and Dermatology*, 28(1), 68-84.

47. Andleeb, F., Elsadek, M. F., Asif, M., Al-Numair, K. S., Chaudhry, S. R., Saleem, M., & Yehya, A. H. S. (2024). Down-regulation of NF- $\kappa$ B signalling by methanolic extract of *Viola odorata* (L.) attenuated in vivo inflammatory and angiogenic responses. *Inflammopharmacology*, 32(5), 3521-3535.
48. Karadağ, A. E., Demirci, B., Çapkurlu, A., Demirci, F., Okur, M. E., Orak, D., ... & Başer, K. H. C. (2019). In vitro antibacterial, antioxidant, anti-inflammatory and analgesic evaluation of *Rosmarinus officinalis* L. flower extract fractions. *South African Journal of Botany*, 125, 214-220.
49. Evangelista, M. T. P., Abad Casintahan, F., & Lopez Villafuerte, L. (2014). The effect of topical virgin coconut oil on SCORAD index, transepidermal water loss, and skin capacitance in mild to moderate pediatric atopic dermatitis: a randomized, double blind, clinical trial. *International Journal of Dermatology*, 53(1), 100-108.
50. Verma, N., Chakrabarti, R., Das, R. H., & Gautam, H. K. (2012). Anti-Inflammatory effects of shea butter through inhibition of iNOS, COX-2, and cytokines via the Nf-Kb pathway in LPS-activated J774 macrophage cells. *Journal of Complementary and Integrative Medicine*, 9(1), 1-11.
51. Kim, J., seok Lee, I., Park, S., & Choue, R. (2010). Effects of *Scutellariae radix* and *Aloe vera* gel extracts on immunoglobulin E and cytokine levels in atopic dermatitis NC/Nga mice. *Journal of Ethnopharmacology*, 132(2), 529-532.