Non-pharmacological therapies and their efficacy in atopic dermatitis: A narrative review

Mude Hemalatha[1], Anubha Dev[1], Rahul Mahajan[1], Dipankar De[1], Sanjeev Handa[1]

[1]Department of Dermatology, Venereology and Leprology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

Abstract

Atopic dermatitis (AD) is a complex immune-mediated disease characterised by recurrent eczematous lesions and pruritus, which adversely affects the quality of life (QoL). Genetic factors, environmental factors, immune dysregulation, and skin barrier dysfunction contribute to its pathophysiology. Non-pharmacological management strategies aim to preserve the skin barrier, address immune dysregulation, and minimise triggers. In this review, wediscuss various non-pharmacological interventions, including allergen (aeroallergens, food allergens, and contact allergens) avoidance, bathing-related measures, moisturisers, clothing choices, therapies targeting the skin microbiome, and allergen-specific immunotherapy, in addition to education and psychotherapy. Non-pharmacological therapies are essential for the holistic management of AD, but their effectiveness varies, highlighting the need for further research and tailored approaches to individual patient needs.

Keywords: Allergens, atopic dermatitis, moisturisers, non-pharmacological

Introduction

Atopic dermatitis (AD) is an immune-mediated disease influenced by multiple factors. It involves recurrent episodes of eczematous lesions, secondary skin infections, skin irritation, and pruritus that adversely impact patients' quality of life (QoL). It affects 15-20% of children and 2-5% of adults globally.[1,2] The pathophysiology of AD is intricate and multifaceted, involving components such as skin barrier disruption, dysregulated cell-mediated immune responses, environmental factors, genetics, IgE-mediated hypersensitivity, and microbial imbalance. Non-pharmacological management involves the use of approaches other than drugs as opposed to pharmacological management, which entails treatment using topical and oral medications to control exacerbations and maintain disease remission. Most non-pharmacological interventions focus on preserving the skin barrier, avoiding triggers, addressing immunological dysregulation, correcting skin-gut axis dysbiosis, and minimising bacterial colonisation. However, the efficacy of these measures varies across patients and studies. This review critically examines these approaches and their roles in treating AD. Though the role of non-pharmacological measures in the management of AD is well known, there is limited published literature on the role and efficacy of these methods. This review aims to highlight their efficacy, and limitations which will aid in more comprehensive counselling of patients and parents for control of AD.

Methods

A literature search was conducted in PubMed, EMBASE, and the Cochrane Library using the keywords 'allergens," 'allergy," 'atopic dermatitis," 'moisturisers," 'emollients," 'bath/bathing," 'cleansing," 'clothing," 'prebiotics," 'probiotics," 'symbiotics," 'skin microbiome," 'immunotherapy,"'psychotherapy,"'education," and 'non-pharmacological treatments" for articles published between 1964 and 2024. Boolean search strategy was used to combine the keywords in relevant combinations

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Corresponding author: Dr. Rahul Mahajan, Department of Dermatology, Venereology and Leprology, Postgraduate Institute of Medical Education and Research, Chandigarh, India. drrahulpgi@yahoo.com

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A total of 110 relevant studies were included, consisting of 35 randomised controlled trials and systematic reviews, 10 epidemiological studies, 15 retrospective studies, 5 guidelines, and 45 review articles. The studies which dealt with clinical outcomes were preferred for inclusion, studies that were entirely experimental, microbial, or immunological were preferably excluded with a few exceptions namely studies that dealt with the role of allergens, water hardness, pH of the skin, and role of the gut microbiome. There were 12 Indian studies, including 4 RCTs, systematic reviews, 4 review articles, and 7 pilot studies.

Aeroallergens

Figure 1 depicts the pathogenesis of AD and the various non-pharmacological strategies targeting each factor.

Various allergens, including aeroallergens, food allergens, chemicals, hard water, and textiles like wool, have been known to exacerbate AD .[3] Amongst aeroallergens, the most commonly implicated ones are house dust mites (, cat epithelia, and pollen.[4] Aeroallergens are implicated when a patient has asthma, allergic rhinoconjunctivitis along with AD, a history of seasonal worsening of the dermatitis in an airborne distribution pattern, and relief in lesions with a change of environment.[5-9]

1. Role of house dust mite

Dermatophagoides pterynosinnus and *Dermatophagoides farinae* are the species of house dust mite most frequently linked to the exacerbation of AD. The major clinically relevant allergens are Der p 1, Der p 2, Der p 23, Der p 5, Der p 7, and Der p 21.[10] These are proteases that trigger the disruption of tight junctions in the skin and bronchial mucosa.[11] Proteases disrupt the epidermal barrier, acting as haptens and IgE antigens on impaired skin. Patients with elevated IgE levels and a history of asthma are more susceptible to atopic dermatitis exacerbations triggered by dust mites.[12] The possible methods that can be employed to reduce exposure to aeroallergens include mattress encasings, cleaning with high-efficiency particulate air filter-equipped (HEPA) vacuum cleaners, and using acaricides such as tannic acid, benzyl benzoate, neem oil, and eucalyptus oil.[13-15] Other easy methods include clearing the bedroom of fluffy toys, upholstery, curtains, sheepskins, and pillows, as well as frequently washing the bedding, flooring, and drapes.[16] Table 1 elaborates on the personal experiences of the authors along with anecdotal evidence in the role of control of aeroallergens and other non-pharmacological methods in AD.

Der p 1 and Der p 1 plus Der f 1 allergen concentrations significantly decreased in a study using house dust mite -impermeable encasings, but neither the non-house dust mite mattress encasing group nor the house dust mite -mattress encasing group experienced significant changes in clinical parameters as a result of this decrease in allergen load.[17] Two studies have demonstrated a definite advantage of house dust mite avoidance measures in the amelioration of AD.[18,19] Seven randomised controlled studies were included

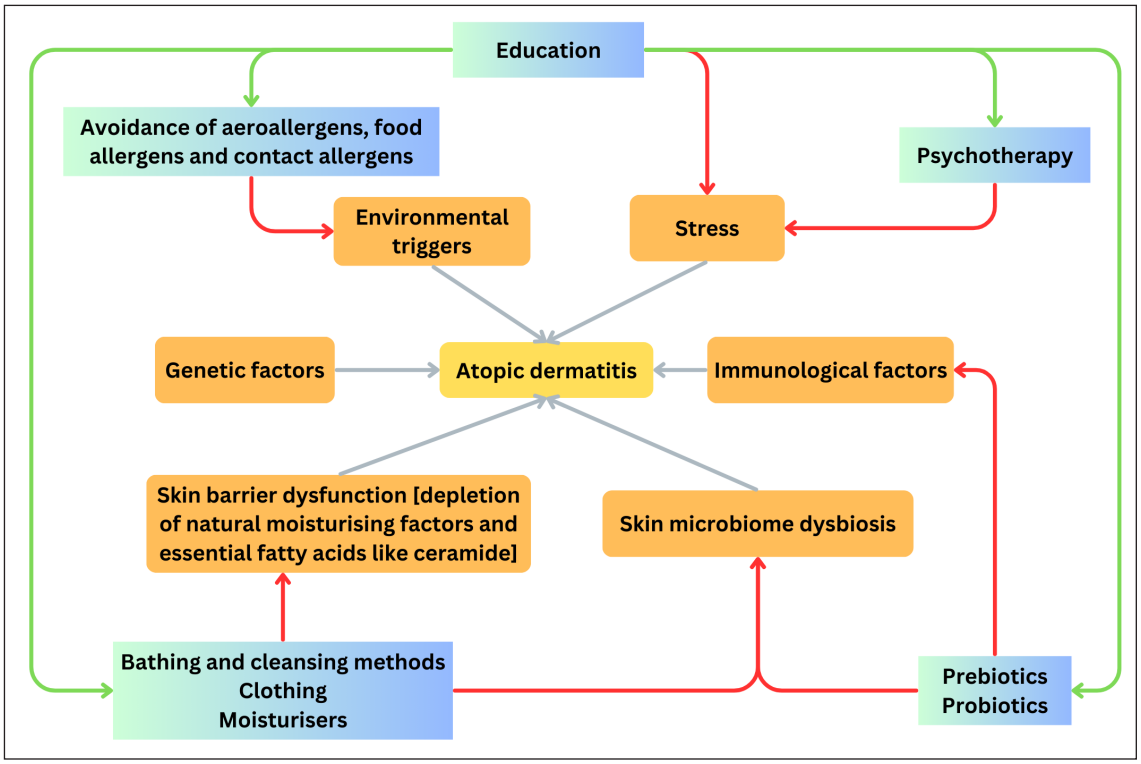

Figure 1: Flowchart depicting the pathogenetic mechanisms of AD and the various non-pharmacological strategies targeting each factor.

Table 1: Non-pharmacological approaches in the management of atopic dermatitis: An authors' perspective

Target area		Prevention strategies
Aeroallergens	Prevention methods for house dust mite removal	• Wet dusting is recommended over dry dusting to minimise the spread of dust and allergens, particularly HDM. • To reduce dust mite exposure, prioritise using protective covers for bedding, usage of polyurethane bedding, maintaining washable items, removing carpets, and minimising upholstered furniture, drapes, and curtains in the bedroom • Appropriate use of a mask or should leave the room after cleaning due to disturbance of dust
	Handling of soft furnishings	• Avoid heavy, thick drapes and curtains, remove carpets and soft, fluffy toys as well as velvety and thick bed linens, and prefer thin, cotton bed linens
	Strategies of washing	• Mattress pads, sheets, and blankets should be washed every 1-2 weeks. • Prefer easily cleanable surfaces like area rugs, which can be vacuumed regularly
	Role of sun-drying	• Helps eliminate allergens such as dust mites, bacteria, and fungi that can exacerbate the condition. • Additionally, sun drying reduces moisture retention in fabrics, preventing the growth of mold.
	Role of ventilation and vacuum cleaning	• Maintain humidity to below 45% • Ventilate during low outdoor humidity • Vacuum cleaning may not capture microscopic house dust mite allergens and can stir up dust, causing them to resettle or become airborne. • Additionally, traditional vacuums without HEPA filters may not fully remove allergens, and they are less effective on soft furnishings like mattresses and upholstery, requiring frequent maintenance for optimal performance. • In the Indian context, affordability and adaptability issues related to HEPA filters and vacuum cleaners should be considered.
	Role of seasonal variation	• During spring, an increase in pollen levels can lead to the exacerbation of AD and allergic rhinoconjunctivitis. • During winter, the dryness and intolerance to wool can worsen AD. • During summer, patients who experience flare-ups due to sweating may have an exacerbation of atopic dermatitis.
	Role of incense cones or sticks (dhoop or agarbattis)	• The fragrance in these agents can trigger exacerbation in patients with contact sensitisation
	Role of fabric dyes	• Contact sensitisation to fabric dyes can lead to exacerbation in patients with allergic contact dermatitis.
	Prevention strategies for pollen	• Patients should minimise outdoor activities and keep windows and doors closed during these seasons, especially in the early morning or late afternoon • Opening windows only around midnight or early morning for ventilation. • It's also advisable to wear masks outdoors, and wash and change clothes upon returning indoors. • Vacuuming once a week and the use of air filtration devices has also been recommended.
Food allergens	Identification and confirmation of food allergens	• It is advisable to avoid only the foods patients reacted to rather than broadly eliminating many items. • Children who are prescribed elimination diets should be taught the signs and symptoms of acute-type reactions, including anaphylaxis, and should have access to self-injectable epinephrine. • Intermittent reintroduction of restricted foods items should be undertaken every 6 to 12 months to prevent an anaphylaxis reaction when ingested accidentally.
Skin microbiome		• Early use of antibiotics and a predisposition to cutaneous infections were found to be associated with an increased risk of developing AD, and the consumption of curd during the first two years of life appeared to have a protective effect according to the author's personal experience
	Role of sodium bicarbonate bath	• Sodium bicarbonate has antimicrobial and antipruritic properties; hence it may be beneficial in AD
Skin barrier maintenance	Role of oils	• Oils rich in oleic acid, such as olive oil, should be avoided as they can impair skin barrier function and increase TEWL. • Coconut oil can cause irritation and allergic reaction in a few patients due to cocamide diethanolamine. • Mustard oil can lead to pigmentation due to isothiocyanates • Sunflower oil could be a better option. • Due to the lack of robust evidence supporting the superiority of one moisturizer over another, opting for a cost-effective and accessible moisturiser for long-term use is recommended by the authors.
Complementary and alternative medicine	Acupuncture, acupressure, hypnotherapy, relaxation and massage, balneotherapy, herbal therapies, oral oils and fatty acids	• As evidence is limited for these therapies, the authors do not recommend these therapies in the management of AD

contd.,

Table 1: (*Contd...*)

Allergen-specific immunotherapy (AIT)	Role of AIT along with oral immunosuppressants	• As evidence of the efficacy of AIT is scarce, we do not recommend its usage in routine practice
Holistic management		• Management of AD should involve a multifaceted approach, including skin manifestations, asthma, allergic rhinoconjunctivitis, and emotional and social well-being. • Early evaluation and identification of associated psychological disorders, and early management through counselling, education, and support groups, play a key role in improving disease outcomes and overall QoL.
HDM: House dust mite, TEWL: Trans epidermal water loss		

in a Cochrane review to assess the efficacy of various house dust mite prevention techniques. It had a high risk of bias as there was heterogeneity in the interventions performed as well as outcome measures. Of the three studies that evaluated the reduction in severity score after implementing multiple avoidance strategies, only one study showed a statistically significant effect, while the other two did not.[13] Based on the currently available evidence, we cannot conclude the benefit of various house dust mite reduction strategies in clinical scenarios. Although frequent cleaning with a high-efficiency particulate air filter-equipped filter-equipped vacuum cleaner reduces mite load and allergens, no studies compared the clinical improvement with this strategy.[20,21]

2. Role of pollen allergy

Studies have shown that exposure to grass pollen intensifies itching and severity of AD.[22,23] Pollen-induced allergy is suspected when symptoms deteriorate during pollen seasons, like summer and spring, and improve with reduced exposure.

3. Role of pet allergens

A pet allergy is suspected when a patient's symptoms aggravate following contact with pets, and the patient has rhinoconjunctivitis and/or asthma.[9,24] The uteroglobin-like protein Fel d 1 antigen is the most common and well-studied cat allergen.[24] Allergic sensitisation can be detected using serum-specific IgE or skin prick testing (SPT) with allergen extracts.[25] If definite evidence of pet allergy is present, contact with animals must be avoided. Although cats are the most usually implicated in pet allergies, it is best to avoid dogs as well to avoid skin infections in patients with AD.[3,26]

Pollutants

Indoor and outdoor pollutants, including tobacco smoke and traffic exhaust, have also been reported to contribute to the development, progression, and persistence of AD.[3] A meta-analysis of observational studies indicates that both active and passive smoking exposure increases the prevalence of AD in children and adults, with an inconsistent association with maternal smoking.[27]

Food allergens

Food allergy is suspected in cases of severe AD that quickly worsen when medication is ceased, in cases with a reliable history of immediate allergic reactions to specific foods, and in children under 5 years with recalcitrant AD.[28] An IgE-specific food allergy is present in 40% of newborns and young children with moderate to severe AD and 8% of children overall. Cow's milk, hen's egg, peanut, soya, almonds, seafood, nightshades, food-colouring agents, food additives, and preservatives are the most common food allergens responsible for AD aggravation in young children.[28] Older children, adolescents, and adults may experience pollen-related food allergies due to cross-reactivity with plant proteins. Symptoms typically involve the oropharyngeal mucosa and occur within 5-10 minutes after eating certain fresh fruits, vegetables, nuts, legumes, and seeds.[29-31]

Maintaining a food diary can help identify potential food triggers for AD flares, particularly in children with suspected food allergies. We can perform *in-vivo* tests skin prick test (SPT), *in-vitro* tests (serum-specific IgE), and atopic patch testing (APT) when a patient is suspected to have a food allergy. SPT is excellent for detecting immediate anaphylactic IgE-mediated reactions. Wheal diameters ≥8mm for milk and peanut and ≥7mm for egg are used as cut-offs since they have 100% positive predictive value.[32] Atopic patch testing detects non-anaphylactic delayed reactions, but it is not standardised for commonly consumed foods and is performed using fresh food items diluted in saline.[3,33] Skin prick test has a lower sensitivity and can be challenging for young children due to limited cooperation, fear of needles, and heightened anxiety.[33] Atopic patch testing has limitations like lack of standardisation, skin irritation, and angry back syndrome; it is also time-consuming.[34]

In vitro tests are effective when skin prick test cannot be employed (e.g. dermographism or UV- and drug-induced skin hyporeactivity, dermatitis at the test site, or lack of cooperation in young children).[3] They provide quantitative data on the degree of sensitisation, which aids in estimating the likelihood of a clinical reaction. An IgE cut-off ≥0.35 kUA/L is widely accepted to determine a test as positive, but it should be clinically correlated.[35] However, the gold standard for detection of food allergy is a double-blind placebo-controlled food challenge.[36] It should be conducted after the patient has abstained from the suspected food for four to six weeks. Avoiding the trigger food item lessens disease severity in patients with a confirmed food allergy. Current research suggests that for children with known food allergies and documented true IgE-mediated allergies, it is advisable to avoid only the foods they reacted to rather than broadly eliminating many items.[37] Recent studies like the Enquiring About Tolerance (EAT) study, Learning Early

About Peanut Allergy (LEAP) study, and Preventing Atopic dermatitis and ALLergies in Children (PreventADALL) studies indicate that introducing a variety of complementary foods to high-risk infants at 4-6 months of age can help in averting the occurrence of food allergies.[38-40]

Contact allergens

Patients with moderate-to-severe AD that is resistant to treatment and has an atypical pattern or distribution affecting the hands, feet, lips, and eyelids with an onset in adolescence or adulthood should be evaluated for contact allergy. Nickel, perfumes, rubber compounds, formaldehyde, neomycin, and lanolin are the commonly implicated allergens. According to an Indian study, patients with severe AD demonstrated positive patch test results with fragrance mix, nickel, cobalt chloride hexahydrate, and potassium dichromate, which were the most commonly detected allergens, with the fragrance mix being the most prevalent allergen.[41]

Bathing and cleansing

Skin cleansing is essential; however, it must be done gently to preserve the epidermal barrier. Mechanical cleansing is more essential. It is recommended to take a 5 to 10-minute bath in lukewarm water (27 to 30°C).[3,16] A systematic review concluded that daily baths or showers did not correlate with the exacerbation of AD and therefore, should be encouraged in AD patients.[17] Commercially available soaps include prototype anionic surfactants, glycerine bars, superfatted soaps, antibacterial soaps, syndets/cleansing bars, and novel cleansers that contain emollients. Soaps consist of anionic surfactants with a pH of 9 to 10, whereas bar and liquid syndets have a slightly acidic or neutral pH.[42-43] The alkalinity of commonly available cleansing products disturbs the skin's acid mantle layer, leading to increased permeability, decreased antimicrobial activity, and modified enzymatic activity.[44,45] Hence, cleansing agents with a neutral pH are preferred.[46]

Antiseptic agents that can be added to the bath are bleach and potassium permanganate ($KMnO_4$).

1. Bleach bath:

The ideal bleach concentration is 2.5 µL/mL, or 0.005% NaOCl, which can be achieved by dissolving half a cup of 6% normal household bleach in a standard 150 L bathtub of water or one tablespoon for 20 litres bucket of water.[47] Two systematic reviews demonstrate that bleach baths improve AD severity scores, but there is no conclusive evidence for their superiority over water baths.[48,49] However, it is not well accepted by Indian patients, though it is appropriate for resource-poor situations.[50] Modifications like bleach suits can also be employed, which involves soaking a cotton pyjama suit in a dilute bleach solution and having the child wear it for 10 minutes, 2–3 times a week.[51]

2. KMnO4 bath:

The optimal dilution of $KMnO_4$ for medicinal usage is 1:10,000, which can be obtained by mixing 400 mg $KMnO_4$ in 4L of water.[52] For ease, dilute the solution to reach a pink colour that matches with nail bed is achieved. $KMnO_4$ possesses an oxidising property that aids in the prevention of secondary infections, as well as anti-pruritic and anti-inflammatory qualities that aid the drying up of denuded areas.[53] Though the use and benefits of $KMnO_4$ are known, evidence in the literature is limited.[54]

Role of water hardness:

Hard water may contribute to epidermal barrier defects.[43] One study found no correlation between hardness of water and an increased risk of AD,[55] despite high levels of calcium carbonate (hardness of water) being positively associated with an increased risk of AD in younger children.[56-59] On the other hand, in a systematic review of 16 studies, 7 showed a relationship between higher $CaCO_3$ levels and increased development of AD in children.[60] However, no discernible positive effect has been observed with the use of water softeners.[56-59,61,62]

Moisturisers

Moisturisers are a class of products that increase skin hydration by increasing water retention in the stratum corneum (humectant), preventing water loss (occlusive), and smoothing the skin's surface by forming a protective coating (emollient).[63] They play a prime role in the management of AD by restoring epidermal barrier function. Emollients commonly include fatty acids, fatty alcohols, cholesterol, squalene, natural plant oils, ceramides, and pseudo-ceramides. Humectants include urea, sorbitol, panthenol, glycerol, propylene glycol, hyaluronic acid, and α-hydroxy acids. Mineral oil, petroleum jelly, beeswax, silicones, zinc oxide, shea/ mango/cocoa butter, and paraffin act as occlusives. Protein rejuvenators are a newer class of moisturisers that include collagen, elastin, and keratin. Natural oils like coconut oil, olive oil, sunflower oil, safflower oil, mustard oil, sweet almond oil, jojoba oil, and evening primrose oil are widely utilised. The fatty acid content in the oil determines its effects. Large amounts of linoleic acid support barrier function by activating Peroxisome proliferator-activated receptor alpha (PPAR-α), promoting keratinocyte growth, lipid synthesis, and modulating inflammation.[64,65] In contrast, oleic acid impairs barrier function by increasing transepidermal water loss, permeability, and inflammation, and has limited antibacterial properties.[66,67] Coconut, sunflower, and safflower oils are preferred due to their higher levels of beneficial fatty acids, while olive oil is not recommended due to its high oleic acid levels.[64,68]

Two systematic reviews reported that moisturisers extended the flare-free period, reduced topical corticosteroid use, and alleviated pruritus and xerosis, but did not affect objective

scores[69,70] It is difficult to choose an appropriate moisturiser due to the heterogeneity of the individual components in commercial formulations. There is a dearth of evidence on the relative efficacies and superiority among various moisturisers. However, ceramide-based moisturisers have been meticulously investigated.[71-74] A systematic review which compared the efficacy of various moisturisers with ceramide-based formulations revealed that ceramide-based moisturisers were superior in terms of improvement in SCORAD.[75]

Although there is a suggestion that emollient therapy starting at birth is a practical, safe, and successful strategy for preventing the future development of AD, larger studies such as the Preventing Atopic dermatitis and ALLergies in Children (PreventADALL) and Barrier Enhancement for Eczema Prevention (BEEP) trials failed to find any advantage in using emollients early in life.[39,76] Emollients therefore help in maintaining remission, but not in disease prevention.

Wet wrap therapy

Wet-wrap therapy involves applying wet bandages over areas of atopic eczema after using emollients or topical corticosteroids to enhance skin hydration, reduce pruritus, and improve medication penetration. Though it increases the risk of skin infections,[77] this risk is not significant in short-term paediatric use and is outweighed by its efficacy in refractory and acute cases.

Clothing

Conventionally, people with AD are advised to avoid wool and to wear cotton and silk garments, because of the softness, breathability, and comfort of the latter.[78,79] Nevertheless, even cotton short interwoven fibres and damp fabrics irritate the skin, while superfine or ultrafine merino wool is well tolerated.[80] Promising novel materials are being developed for AD patients, including cellulose-based, chitosan-coated, and silver-coated materials, which help decrease the severity of disease or the *S. aureus* burden.[81] In a study, patients of AD preferred lyocell, a synthetic cellulose fibre, over cotton, but there are limited data and varied results with other materials, making it difficult to choose the ideal apparel.[82]

Skin and gut microbiome

Disruption of the skin microbiome in patients with AD results in decreased normal flora and colonisation by *Staphylococcus aureus*. Through the stimulation of various T-cell clones and cytokines (IL-3, histamine, and IL-13), virulence factors secreted by *S. aureus* colonies, including superantigens, α-toxin, δ-toxin, protein A, and phenol-soluble modulins (PSM), exacerbate inflammation and enhance the inflammatory response.[83-85] Topical probiotics containing organisms such as Lactobacillus johnsonii, Streptococcus thermophilus, and lysates of Vitreoscilla filiformis have been used in creams to help restore microbial balance and improve skin microbiome.

Gut microbes play a crucial role in regulating systemic inflammation, and an imbalanced gut microbiome, and less diverse gut microbiome has been linked to the development of AD. Correction of gut dysbiosis has been undertaken by multiple authors in the form of probiotics, prebiotics, postbiotics, and synbiotics.

Probiotics

Probiotics are live, beneficial microbes that have an immunomodulatory effect by lowering proinflammatory cytokines (IL-4, IL-6, tumour necrosis factor-α, and INF-γ), suppressing Th2 response, and increasing the Th1/Th2 ratio.[86,87] They include non-pathogenic yeast like *Saccharomyces boulardii* and bacteria from *Bifidobacterium, Streptococcus*, and *Lactobacillus* families.[88] The dosage of *Lactobacillus* ranges from 5×10^9 to $1x10^{10}$ CFU, administered once or twice daily for 4 to 12 weeks.[89] The dosage of *Bifidobacterium* ranges from 3×10^9 to 1×10^{10} CFU, given once or twice daily.[89] Probiotics have been shown to have beneficial effects in systematic reviews; however, 2 studies failed to show beneficial effects.[90-93]

Prebiotics

Prebiotics are nondigestible compounds (fructo-oligosaccharides, galacto-oligosaccharides, and long-chain inulin) that promote the growth of beneficial micro-organisms.[94,95] They increase the formation of short-chain fatty acids (acetate, propionate, and butyrate), which have anti-inflammatory properties and enhance the Th1/Th2 ratio.[96,97]

Postbiotics

Postbiotics are composed of inanimate microbes and/or their components that offer immunomodulatory, anti-inflammatory, and antimicrobial benefits without the ability to colonise the host.[98] Postbiotics offer several advantages, including a well-defined chemical composition, lack of antibiotic resistance transfer, greater stability, and extended shelf life.[99] A systematic review reported symptom improvement with oral *Lactobacillus postbiotics* for AD in adults, but findings were inconsistent in paediatric patients, due to varying dosages.[100]

Synbiotics

A synbiotic has a mixture of prebiotics and probiotics, thus utilising the synergistic effects of these components. While some studies suggest that synbiotics may enhance treatment outcomes, their superiority over prebiotics or probiotics alone remains debatable.[101] A recent study found no significant difference in SCORAD scores between patients treated with synbiotics versus those receiving prebiotics alone.[102]

Educational intervention

This involves providing sufficient time to explain the condition, discussing the importance of compliance with proper skin care, implementing behavioural modifications through patient and parental education, and addressing

patient concerns.[103] It can be facilitated by dermatologists, paediatricians, psychiatrists, psychologists, and nurses.[104] Group discussions with parents of similar-age children are also essential to address shared concerns, reduce isolation, and provide support for families with AD, fostering a sense of community and validation.[103]

Psychotherapy

Psychological and emotional aspects are recognised factors that modify the clinical course of AD.[105] Stress can aggravate the disease and alter the itch-scratch cycle.[106] Itching in AD can impair sleep quality, leading to mental health issues such as depression, anxiety, and there can be social isolation due to visible skin lesions. A meta-analysis found the odds of depression, anxiety, sleep disorders, and conduct disorder to be 1.42, 1.33, 2.10, and 1.49, respectively.[107] Psychoeducation and psychotherapeutic interventions like cognitive behavioural therapy and interpersonal therapy can help in improving the QoL of patients.

Allergen-specific immunotherapy (AIT)

Patients with respiratory allergies and stinging insect hypersensitivity may benefit from allergen-specific immunotherapy.[108] This involves administering increasing dosages of the allergen gradually to modify the response and encourage peripheral immunological tolerance mechanisms, which leads to a shift from a Th2 response to a Th1 response, a decrease in mediator release from mast cells, and the generation of blocking antibodies IgG4.[106] This desensitisation is performed via sublingual or subcutaneous routes. In one systematic review with eight randomised controlled trials, there was a statistically significant benefit in patients receiving allergen-specific immunotherapy when compared to placebo, but there was a high level of heterogeneity.[109] In another systematic review with 23 RCTs, immunotherapy to house dust mites demonstrated improvement in AD severity, but the impact on long-term AD control and flares was less certain.[110]

Conclusion

Non-pharmacological therapies have an important role in maintaining the skin barrier function, preventing exacerbation due to allergen exposure, preventing skin infections, preserving the skin-gut microbiome, and overall improving the overall QoL of patients with AD. Evidence in the literature is heterogeneous, and clinical experience varies across different settings, especially for these therapies as standalone modalities. Nevertheless, dermatologists and physicians need to be aware of the various components of non-pharmacological therapies in order to provide holistic care for patients with AD.

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