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Therapeutic Moisturizers in Eczema and Xerosis Management

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Introduction

Eczema is a chronic relapsing dermatitis and, as such, it is imperative to maintain the hydration and barrier function of the skin in these patients with daily moisturizer use. Emollients have long been used to maintain the skin barrier function in patients with eczema (atopic dermatitis). Ceramide and urea-based moisturizers have been shown to be beneficial in reducing transepidermal water loss (TEWL), improving barrier function, and maintaining hydration of the stratum corneum layer of the epidermis; thus, they should be considered a mainstay of treatment in patients with xerosis (dry skin) and eczema.

Overview of Eczema

Eczema is a chronic, pruritic, inflammatory skin disease with wide ranging severity; it is usually the first manifestation of atopic disease. Eczema is a major public health problem worldwide that commonly presents during early infancy and childhood, but can persist or start in adulthood (prevalence in children is 10-20% and 1-3% in adults).[1] Prevalence has increased by two to threefold during the past 30 years in urban areas and industrialized countries, but it remains much lower in rural and less industrialized regions.[2]

- The causes of eczema are not completely understood, but dysfunction of the skin barrier, likely the result of both genetic and environmental factors, and immune dysregulation are important in its pathophysiology.[3]
- Acute eczema presents as erythematous patches, papules, plaques, and excoriations secondary to scratching; there may also be weeping of serous exudate. Chronic lesions have the same characteristics, with the addition of lichenification, fissures, and occasional alopecia.[4]
- Partly due to ease of accessibility for scratching, infantile eczema predominantly involves extensor surfaces of the arms and legs, face, and trunk. Scaling, exudate, and fissures are also common findings in infants.
- In adults, flexural areas, face and neck, wrists, and the dorsal areas of the hands and feet are the most commonly affected regions.

Treatment Rationale

The major goal of disease management is to reduce the frequency and severity of flares, and prolong periods of remission. Comprehensive long-term management addresses both skin barrier dysfunction and immune dysregulation, but also includes patient and caregiver education, flare prevention through trigger avoidance and hydration, as well as pharmacologic and non-pharmacologic therapies.[3]

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Treatment Rationale (continued)

- Non-pharmacologic patient-specific strategies include removal of allergens (e.g., foods, pet dander, pollen), identification of trigger factors (e.g., stress, low humidity), and a balanced intake of dietary nutrients.[5]
- Particularly during infancy, a higher intake of vitamin A may reduce the incidence of eczema seen in children with a positive family history of atopy. The use of *Lactobacillus* during pregnancy and while nursing may postpone the onset of eczema in infants and children.[5]
- Pharmacologic therapy includes the use of emollients, topical corticosteroids, and topical calcineurin inhibitors.
- For mild eczema, over-the-counter (OTC) emollients and topical corticosteroids, e.g., hydrocortisone 0.5% (low potency) and clobetasone 0.05% (mid potency) are available for self-treatment.
- Pharmacists are increasingly educating patients and caregivers on safely self-treating and recommending when it is appropriate to seek help from a physician.
- The goals of self-treatment are to stop the itch-scratch cycle, maintain skin hydration, and avoid or minimize factors that can trigger or aggravate eczema.
- An ideal moisturizer is one that performs four functions:[6]
  1. repair the skin barrier,
  2. maintain skin integrity and appearance,
  3. reduce transepidermal water loss (TEWL),
  4. restore the lipid barrier's ability to attract, hold, and redistribute water.
- It is appropriate for patients or caregivers to consult a physician if OTC treatments are not providing adequate relief, eczematous lesions appear to be infected, or the patient's sleep is frequently disturbed by pruritus.[5]

Available Formulations of Therapeutic Moisturizers

Ceramide-based Moisturizers

- Recent biochemical findings indicate that disturbances of epidermal lipid compartment structures (particularly of ceramides) account for the defects in barrier function of atopic dry skin.[7]
- Optimal barrier function requires the presence of sufficient extracellular lipids to form a competent lamellar bilayer system of the stratum corneum.[7]
- Ceramides, which consist of different sub-fractions of lipids, represent one of the major lipid constituents of the extracellular lipids and are functionally important for the stability of the multilamellar bilayer system.
- Studies have revealed that ceramides are reduced in the whole atopic population, but particularly in those individuals in an active phase of the disease.[8]
- A reduction of ceramides has been inversely correlated with TEWL, which can result in chronically dry skin.
- Topical ceramide supplementation controls ceramide deficiency and improves the overall skin condition.[6]
- Their benefits are derived from prophylactic and regular use, which may reduce the need for topical corticosteroids and calcineurin inhibitors, and possibly mitigate the side-effects from these medications.
- OTC ceramide-based moisturizers include Impruv® cream and Cetaphil Restoraderm™ lotion. CeraVe™ and TriCeram® are available in the U.S. only.

Prescription Ceramide-based Moisturizers

- These consist of a higher percentage (compared to OTC brands) combination of ceramides, cholesterol, and fatty acids that mimic those naturally found in the skin.[9]
- EpiCeram® was approved by Health Canada in September 2009 as a Class 2 medical device for use as a non-steroidal lipid barrier emulsion to manage burning and itching symptoms associated with dry skin conditions, such as eczema.
  - In a study involving 113 children with moderate to severe atopic dermatitis, similar efficacy to a mid-strength topical corticosteroid was demonstrated.[9]
  - This multi-lipid emulsion has a favourable safety profile and does not appear to have substantial restrictions for use, such as treatment duration or patient age.
- Prescription ceramide-dominant formulations include EpiCeram® cream (available in Canada and the U.S.) and Atopiclair® and MimyX® (available in the U.S. only).

Urea-containing Moisturizers without Hydrocortisone

- Urea-based moisturizers are OTC formulations that are indicated for xerotic skin with or without pruritus.
- Urea works by enhancing the water-binding capacity of the stratum corneum and long-term treatment with urea has been demonstrated to decrease TEWL.[10]
- Application of these moisturizers is recommended shortly after bathing, while the skin is still wet.
- The short-term therapeutic effects of urea-based moisturizers are apparent in patients even after 1 week of daily application in those with dry skin and eczema.[11]
- It has also been shown that long-term urea application reduces the susceptibility to skin irritation from sodium lauryl sulfate, a surfactant commonly used in many soaps, shampoos, detergents, and toothpastes.
- The protective effect (after prolonged application) of urea-containing moisturizers has promising clinical ramifications, such as reduction of contact dermatitis from irritating stimuli.[10]
- Higher concentration urea-based formulations induce more prominent keratolytic (softening/shedding) activity that can increase skin irritation. A lower concentration is generally used on the face and body, whereas a higher concentration may be applied to thickened skin areas (e.g., feet).

Available Formulations of Therapeutic Moisturizers (continued)

- OTC urea-based moisturizers include various strengths of urea: 5% (e.g., Eucerin® cream); 10% (e.g., Uremol® 10 cream or lotion, Eucerin® lotion or cream, Urisec™ cream); 12% (e.g., Uresec™ lotion); 20% (e.g., Uremol® 20 cream); 22% and 40% urea creams.
- Urea 40% cream is a potent keratolytic that is not suitable for use as a regular moisturizer.

Urea-containing Moisturizers with Hydrocortisone

- Urea-based moisturizers with hydrocortisone are prescription strength formulations and are effective for xerotic skin with inflammation and mild eczema.[4]
- Topical corticosteroids are effectively used for controlling active skin inflammation in eczema. The lowest effective potency of topical corticosteroids is always preferred for the local treatment of lesions.
- Combining an emollient with a corticosteroid has been shown to be effective. A cohort study found that the addition of 10% urea to a commercially prepared steroid cream gave better results in treating subacute atopic eczema than the steroid cream alone.[12]
- Side-effects from topical steroids are directly related to the potency of the compound and the length of use.
- Potential risks from long-term topical steroid use include fungal infections, impetigo, viral warts, and herpes simplex. As well, discontinuation of topical corticosteroids may lead to a flare of symptoms.
- Low-potency hydrocortisone 1% cream has been found to be quite safe for cutaneous use.
- Prescription-based urea moisturizers containing 10% urea with 1% hydrocortisone are available in lotion or cream preparations (e.g., Uremol® HC).

Diabetic Skin Care Management

- Xerosis of the feet is a common skin condition; incidence increases with age, exposure to dry winter conditions, and physiological changes that alter circulatory supply to the lower extremities (e.g., diabetes).
- People with diabetes have a high incidence of xerosis of the feet, especially on the heels.
- While assessing for predictors of foot lesions in diabetic patients, one study found that 82.1% of this cohort had skin with dryness, cracks, or fissures.[11] An unpublished survey of 105 consecutive patients with diabetes conducted by one of the authors revealed that 75% had clinical manifestations of dry skin.
- Dry skin often leads to cracks and fissures that can act as portals of entry for bacteria. These cracks and fissures are associated with an increased risk of cellulitis and foot ulceration that, if left unchecked, can eventually lead to amputation.
- Xerosis of the feet in diabetic individuals can be controlled with the regimented use of moisturizers.[11]
- Healthcare providers should routinely inspect the feet of diabetic patients and encourage daily moisturization.
- Urea has been found to be a potent skin humidifier (by decreasing TEWL) and descaling agent.
- Studies of diabetic patients revealed that urea is safe and effective in controlling xerosis of the feet and showed longer-lasting effect than other emollient creams.[11]
- Urea cream (especially at 40% strength) works as a keratinolytic and helps in the treatment of corns and calluses of the feet.[13] This can be functionally important as these hyperkeratotic papules can be uncomfortable, and even painful, thereby restricting physical activity in affected individuals.

Conclusion

Eczema is a chronic relapsing dermatitis and, as such, it is imperative to maintain the hydration and barrier function of the skin in these patients with daily moisturizer use. Ceramide and urea-based moisturizers have been shown to be beneficial in reducing TEWL, improving barrier function, and maintaining hydration of the stratum corneum layer of the epidermis, and thus, should be a mainstay of treatment in patients with dry skin and eczema. Failure to adequately moisturize the skin can lead to a flare of symptoms or an increased incidence of infections. However, adherence to a schedule of regular moisturizer use is associated with improved patient quality of life outcomes (e.g., reduced pruritus, improved sleep patterns, less depression) and a reduction in the severity and frequency of eczematous flares.[14]

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Head Lice: A Review of Topical Therapies and Rising Pediculicidal Resistance

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Introduction

Head lice infestations (*Pediculosis capitis*) are a worldwide problem with prevalence estimates typically ranging between 1-3% in elementary school aged children.[1,2] Although this obligate parasite is a nuisance, infestation does not pose a health risk. Infestations tend to occur more frequently in females,[3] and less frequently in black children,[4] as it may be more difficult for lice to grasp their oval-shaped hair shafts. It is not associated with poor hygiene. Infestation occurs across all levels of society, but occurs more frequently under conditions of overcrowding. Recent evidence suggests increasing frequency of topical treatment failure may be related to a growing resistance to the neurotoxic pediculicides that have been the first-line treatment for the last 40 years.[5] Herein, we will review the current topical treatment options (Table 1), including newer non-pediculicidal options.

Overview of Facts on Lice

- *Pediculus humanus capitis* (the head louse) is a 2-4 mm blood sucking, wingless insect.
- A louse cannot jump, but rather has 6 legs adapted for crawling along hairs at 23 cm per minute.[6]
- A louse will feed every 3-6 hours.
- Prior to feeding, the louse injects saliva into the skin.
- The life span is approximately 4 weeks and the female lays 6-8 eggs per day.
- Eggs hatch in 8 days, leaving their shell ("nit") cemented to the base of the hair.
- Head lice spread by head contact, shared fabrics, shared combs, and other fomites that are commonly in contact with the scalp and hair.[7]
- A louse can survive 2-3 days away from a human host.
- Pets are not vectors.

Diagnosis and Symptoms

Many affected individuals report no symptoms, but the most commonly reported symptom is scalp pruritus.[8] The pruritus is thought to be caused by hypersensitivity to the louse saliva that is injected into the scalp during feeding, but the itching often does not begin until 1-4 weeks after infestation. Although any part of the scalp may be colonized, there seems to be a predilection for the nape of the neck and post-auricular areas.

Skin Findings

- Often there are no significant findings on the skin.
- Pruritic, papular lesions may be found at the nape of the neck.
- There may be excoriations on the scalp.
- Secondary staphylococcal infection is possible.
- Possible enlargement of cervical / nuchal lymph nodes.

Hair Findings

- True infestation is confirmed by the presence of live adult lice or nymphs (hatched immature lice) present on the scalp with nits.
- The presence of nits alone does not confirm infestation, as an empty nit can remain cemented to a hair even after the infestation has cleared.
- The distance of the nit from the scalp can be a clue to the duration of the infestation, as it moves with the hair away from the scalp when hair grows.
- A nit within 0.6 mm of the scalp is usually a viable egg.
- To distinguish between eggs and dandruff, attempt to dislodge them from the hair shaft. Flakes of dandruff easily dislodge, whereas nits do not.

Diagnosis is best made by wet or dry combing the scalp, using a fine-toothed nit comb with teeth spaced 0.2 mm apart. One study comparing wet combing with visual inspection found that wet combing accurately diagnosed infestation 90.5% of the time, as compared to 28.6% with visual inspection.[2]

Directions for Detection by Wet Combing[9]

- Saturate hair with a conditioner.
- Remove tangles with a regular comb.
- With the nit comb against the scalp, comb to the end of the hair.
- Check the comb for lice after each pull by visual inspection and by cleaning the comb with a tissue and inspecting the contents.
- Dispose of the tissue in a plastic bag.
- Comb the entire scalp at least 5 times.
- Seal the plastic bag and dispose of it.
- If infestation is confirmed, rinse off all conditioner prior to treatment.

Treatment Options

Method	Treatment	Application	Comments
Topical Non-pediculicides	Isopropyl myristate 50% + ST-cyclomethicone 50% rinse	• 30-120 mL of solution is applied to dry hair and scalp (especially nape of the neck); leave for 10 minutes • Comb wet hair with nit comb and wash with shampoo	• Works by dissolving the outer layer of the exoskeleton of a louse, leading to dehydration and death • Resistance less likely due to mechanical mechanism • 2 applications usually necessary 7-10 days apart • Approved for use in patients ≥2 years of age • May cause erythema, burning, and dry scalp[10]
	Herbal product (HairClean 1-2-3)	• Apply product to scalp and hair; leave for 15 minutes before rinsing • Applied 3 times with 5-day intervals between applications	• Herbal product containing anise, coconut, ylang ylang oil, and isopropyl alcohol • Suggested mechanism is to invoke a "flee response" by creating an undesirable environment for the louse[9] • One manufacturer sponsored study in Israel found similar effectiveness (92%) when compared with pediculicide containing permethrin, malathion, and piperonyl butoxide[11] • Efficacy and toxicity data not available
Topical Pediculicides	Permethrin cream (1% or 5%)	• Wash hair with conditioner-free shampoo and towel dry • Apply product to scalp and hair for 10 minutes before rinsing (25 mL) • Comb wet hair with nit comb • Repeat in 7 days	• Synthetic pyrethroid, neurotoxic to lice, but low neurotoxicity in humans; avoid use in patients with a known allergy to chrysanthemum • 1% preparation is available OTC • Not ovicidal, therefore requires retreatment 7-10 days later • Approved for use in patients >2 years of age • May cause itching or burning of the scalp
	Pyrethrin 0.33% + Piperonyl butoxide 4%	• Apply product to dry hair for 10 minutes, then add water to form lather • Rinse, do not use conditioner • Repeat in 7 days	• Made from chrysanthemum extract; neurotoxic to lice, but low neurotoxicity in humans • Avoid if there is a known chrysanthemum or ragweed allergy • Approved for use in patients >2 years of age • May cause itching or burning of the scalp
	Lindane (1% gamma benzene hexachloride)	• Apply product to dry hair that is free of conditioner, gel or hairspray • Rub into hair and scalp until wet and leave in place for 4 minutes • Rinse, being careful not to spread the product to other body sites	• Organophosphate, neurotoxic to lice and humans • Second-line treatment due to the risk of toxicity, which can lead to seizures[12] • Can also cause bone marrow suppression if systemically absorbed • Contraindicated in patients <2 years of age, pregnancy, breastfeeding, and in patients with a history of seizures

Table 1: Topical treatment options for head lice[9-13]

Management

Traditionally, topical pediculicides have been the mainstay in the initial treatment of pediculosis. They are widely available without a prescription, which has contributed to the difficulty in gathering data on the true prevalence of infestation. Easy access and improper use has likely contributed to the significant resistance that has developed against topical pediculicides. Knockdown resistance (kdr) is a heritable insensitivity to dichlorodiphenyltrichloroethane (DDT), the pyrethrins, and the pyrethroids. A recent study examining lice collected in Quebec, Ontario, and British Columbia found the allele for resistance present in 97.1% of the 274 lice sampled.[5] These findings suggest that a significant resistance to the traditional first-line treatment options exists within Canada.

Management (continued)

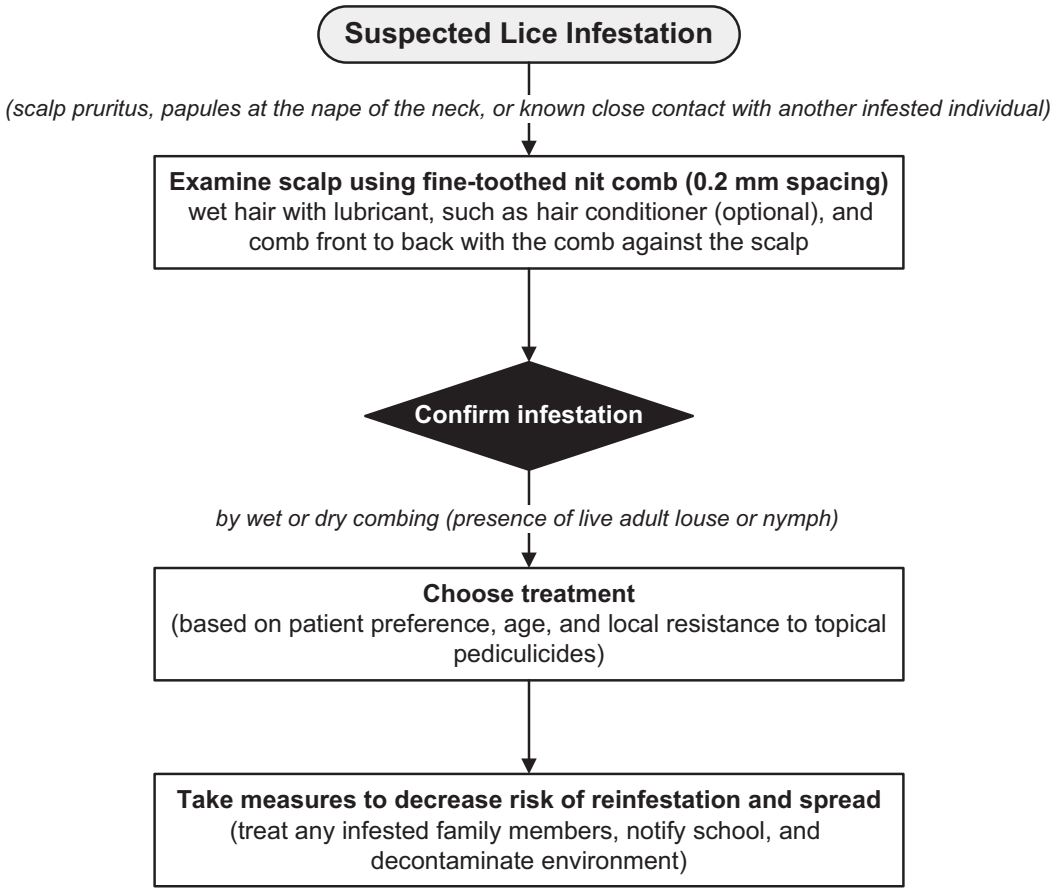
In recognition of the developing resistance, there has been an increased interest within Canada to explore effective non-pediculicidal options. A recent study found the efficacy of isopropyl myristate 50% to be significantly higher (57%) than the standard treatment with pyrethrin 0.33% + piperonyl butoxide 4%.[10]

While non-pediculicidal therapy may be efficacious against treatment resistant infestations, re-infestation from close contacts and fomite transmission is a common problem. Along with treatment, it is important to decontaminate the environment.

Environmental Decontamination[14]

- Family members and close contacts should be examined and be treated for any infestation.
- Any clothing, linens, combs, toys, and fabrics used by the individuals in the 3 days preceding treatment should be decontaminated.
- Fabrics can be washed in high heat and put in a hot dryer for 20 minutes.
- Items that cannot be washed can be sealed in a plastic bag for 14 days or placed in the freezer for 24 hours.
- Brushes can be soaked in rubbing alcohol for 1 hour.
- Floors and furniture can be cleaned by vacuuming.
- Spraying the home with a pediculicide is not recommended.
- No nit policies at schools are unnecessary.

Management Tree



Homeophathic Treatment

The clinical efficacy of homeopathic remedies (e.g., petroleum jelly, peanut butter, mayonnaise, tea tree oil, and vinegar) remains unproven.

Manual Removal

Some patients may prefer to attempt mechanical treatments prior to topical therapy. Wet combing, as described earlier, can be both diagnostic and therapeutic. To attempt this method the patient should wet comb the entire scalp until no more lice are found every 3-4 days for 3 weeks, or at least 2 weeks after the last adult louse was found.[14]

Treatment Failure

Treatment failure is commonly the result of inadequate (insufficient amount of the product used) or improper treatment (repeating the treatment before 7 days), resistance, or reinfestation. If environmental decontamination was performed and the treatment was properly administered, then immediate retreatment with a different agent is advised.

Patients should be advised that scalp itchiness can occur after the application of insecticides and does not necessarily indicate a resistance to treatment or reinfestation.

Conclusion

Head lice infestation is a common problem for children in Canada. The first-line treatment of using topical pediculicides is unfortunately not as effective as it once was because of a heritable resistance that seems to be rising in prevalence. Topical non-pediculicides may be an effective option in the case of failed treatment due to louse resistance to standard treatment.

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Skin Barrier Repair in the Management of Atopic Dermatitis

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Background

The term "atopy" was first coined by Cooke and Coca in 1923, derived from the Greek word *atopos*, which means out of place and denotes an immune reaction that is "strange or eccentric". Atopic dermatitis (AD) is a chronic, waxing and waning, often symmetric inflammatory eruption that is characterized by pruritus and xerosis (dry skin). AD frequently emerges in the first few months of life, but its prevalence decreases with increasing age. It has been reported to affect up to 15% of children worldwide[1] and can persist into adulthood. This pathology is probably caused by the interplay of genetic and environmental factors.

Genetic Factors in Atopic Dermatitis

- A strong genetic involvement in AD has been clearly established.
- Linkage analysis studies and the examination of polymorphisms in a number of candidate genes have identified several chromosomal loci and potential genes as possible susceptibility factors.
- The genetic variants in these loci and genes are postulated to be involved in
  - immunoglobulin E (IgE) antibody production
  - regulation of the immune response in the skin and mucosa
  - epidermal barrier dysfunction, via modulation of epidermal maturation.

Environmental Factors in Atopic Dermatitis

- Genetic factors alone, however, cannot explain the results of epidemiological studies showing the recent significant increase in prevalence of atopic dermatitis, especially in industrialized countries.
- Therefore, a key role for environmental factors in mediating disease expression has been suggested.
- Allergic sensitization to house dust mites and some foods may play an important etiologic role in some patients.
- However, non-allergic factors may also contribute to the pathophysiology of AD by influencing dysregulation, resulting in disruption of the skin barrier. These include:
  - low environmental humidity level
  - *Staphylococcus aureus* (*S. aureus*) colonization
  - exposure (or the absence of it) to certain microbial factors in early infancy
  - exposure to pollutants, detergents, and other irritants
  - excessive heat.

The Impairment of the Barrier Function

- The epidermis of the skin functions not only as a physical and anatomical barrier, but also as a vast immunological organ.
- This barrier constantly protects against the entry of different microbes, allergens, and irritants.
- In AD, a dysfunctional skin barrier has been shown to provoke increased transepidermal water loss (TEWL), resulting in pronounced cutaneous dehydration.
- Such a damaged barrier can allow allergens, microbes, and irritants to penetrate the skin and cause a pro-inflammatory reaction that typically characterizes AD.
- The extent of barrier dysfunction strongly correlates with the degree of inflammation within AD lesions.[2]
- The stratum corneum (SC) of the skin has been compared to a brick wall, consisting of terminally differentiated keratinocytes (bricks) that are surrounded by a matrix of specialized lipids.
  - The major lipids in the SC are:
    - ceramides (50% by mass)
    - cholesterol (25% by mass)
    - fatty acids (10-20% by mass).
  - These elements create a barrier that helps to
    - keep water within the body
    - prevent the entrance of pathogens and allergens.
- It has been shown that AD patients have reduced levels of the SC lipids (e.g., ceramides).[3,4]

The Impairment of the Barrier Function (continued)

- This barrier defect affects not only the involved, but also the uninvolved skin, which correlates with a decrease in the ceramide fraction of the SC.
- Genetic abnormalities in protease inhibitor expression and reduced levels of cornified envelope proteins, such as filaggrin,[5] fuel the skin damage seen in eczematoid conditions.
- Stress may also aggravate this barrier dysfunction by the production of endogenous glucocorticoids, which suppress epidermal lipid synthesis.
- Lastly, the intense itching and extensive scratching that is associated with AD can also be an important factor leading to the disruption of the cutaneous barrier.

Options for Management

A range of treatments exist for atopic dermatitis, depending on the severity of the disease. Successful management aimed at reducing flares requires a combination of medical treatment, non-pharmacologic therapy, and lifestyle modification.

Topical Corticosteroids

- For several decades, topical corticosteroids have been the mainstay of treatment for AD flare-ups.
- A number of agents are available in various vehicles, potencies, and concentrations.
- Low-potency agents should be used in infants and on sensitive skin areas (e.g., face, neck, groin, and axillae) in order to minimize side-effects, such as skin atrophy, acne, and adverse systemic effects.
- Topical corticosteroids should be used for the shortest duration and at the lowest potency possible, while still allowing good control of flare-ups, to limit adverse effects.
- Conversely, misinformed patients and/or parents demonstrating steroid phobia should be advised that withholding appropriate treatment affects their/their children's wellbeing and unnecessarily prolongs the course of sometimes debilitating disease.

Topical Calcineurin Inhibitors (TCIs)

- TCIs are indicated in the management of
  - mild to moderate AD (pimecrolimus)
  - moderate to severe AD (tacrolimus).
- TCIs inhibit T cell activation and release of cytokines involved in the pro-inflammatory cascade of AD.
- Their side-effects include skin irritation and burning at the start of therapy, but usually subside with time.
- Their long-term safety is unknown and rare reports of malignancies have surfaced;[6,7] however, there is no evidence of an increased rate of lymphoma when compared to the general population.[8]
- TCIs are generally used in patients who are unresponsive or show unacceptable side-effects with classic therapy.[6,7]

Oral Antihistamines

- There has been a lack of evidence supporting the use of sedative or non-sedative antihistamines for the treatment of atopic dermatitis.
- The first generation antihistamines (diphenhydramine and hydroxyzine) are sometimes recommended at night for their sedative effects.

Antimicrobial Agents

- Secondary superinfection with *S. aureus* is common and is treated with short courses of antibiotics with anti-staphylococcus coverage.
- Antiseptic baths have been advocated by some experts in those chronically colonized.

Emollients

- Emollient therapy maintains hydration levels and epidermal barrier defenses, thereby reducing the frequency and severity of AD flares.
- Emollients soften and soothe the skin.
- Common emollients include petroleum jelly, animal oils, butyl stearate, cocoa butter, lanolin, lipids, mineral oil, and shea butter.
- There is no strong evidence that emollients improve AD directly.
- However, emollients are widely recommended because they improve the appearance and symptoms of dry skin that is commonly present in AD.[7]
- Studies have shown that emollients may reduce the need for topical steroids and enhance the therapeutic response to them.[8,9]
- In the absence of good studies showing the superiority of one emollient over another, patient preference should guide their usage.

Barrier Repair Creams

- Given the importance of the dysregulated barrier function in AD, the use of topical agents aimed at accelerating its improvement represents a new therapeutic approach.
- As important as emollients are for the alleviation of AD symptoms, these agents may be ineffective at correcting TEWL and the ceramide deficiency resulting from the defective skin barrier of AD patients.
- The efficacy and tolerability of new ceramide-dominant skin repair creams with a more physiologic 3:1:1 ratio of ceramides, free fatty acids, and cholesterol have been studied in two company sponsored trials.[12,13]

Options for Management (continued)

- In one multicenter, investigator-blinded, randomized pediatric study using a ceramide-dominant skin barrier cream (EpiCeram®), similar efficacy was demonstrated when compared with the mid-potency steroid fluticasone propionate 0.05%.[12] No significant adverse events were observed in either treatment arms. However, 4 of 59 patients in the barrier group experienced an initial flare-up that required short-term fluticasone cream.
- EpiCeram®, a steroid-free, lipid-based barrier repair cream, was approved by Health Canada in September 2009 as a Class 2 medical device and is only available by prescription.
- A thin layer is applied to affected skin areas 2 times daily or as needed, massaging gently into the skin. Following application, a transient tingling sensation may persist for 10-15 minutes.

- Studies on concomitant use of both topical steroids and skin barrier repair creams are not yet available.

Non-pharmacological Factors and Lifestyle Modification

- Flare-ups of AD can be reduced by:
  - wearing soft cotton clothing
  - washing clothes with mild detergents
  - avoiding the use of fabric softeners
  - controlling the ambient temperature and humidity of the home
  - implementing avoidance measures to decrease exposure to dust mites in sensitized individuals
  - more rarely, avoiding specific foods in sensitized patients. If a food trigger is suspected, it may be useful to consult an allergist.
- It is important to emphasize that no good evidence supports highly restrictive diets, which might have a significant psychological impact and can lead to malnutrition.
- Educational programs have demonstrated significant improvement in AD severity and treatment satisfaction in intervention groups compared with control groups.[14]

Conclusion

Because of the better appreciated role of the skin barrier in AD disease pathogenesis, use of agents that can stabilize epidermal defenses may reduce the current exclusive dependence on topical steroids and immunomodulators. These barrier repair creams do not target inflammation directly, but rather act at an earlier stage in the disease process to normalize the barrier function and reduce pro-inflammatory signaling. This approach could potentially lead to better treatment outcomes with lesser side-effects.

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Seal of Approval Initiative to Assess Skin Care Product Claims

Dermatology Review Panel

For the average consumer, choosing nonprescription skin care products can be confusing, time consuming, and stressful. Personal care in Canada is a $5 billion industry that offers thousands of competing products, many of which make skin care-related claims. Advertisers inundate us with messages about what their products can do for us, but how can we be sure that the products we buy will actually live up to their claims?

Phrases such as "Dermatologist Tested" or "Dermatologist Approved" offer reassurance that a nonprescription skin care product has been reviewed by a professional and is likely to provide the desired results. But, in reality, there is no standard for what these phrases mean; they can, in fact, simply indicate that several dermatologists have tried a product at the manufacturer's request.

For these reasons, several organizations have created seals of approval for products; for example, the Canadian Dermatology Association (CDA) created a seal of approval for sunscreens, much like the American and Canadian Dental Associations have done for dental products like toothpaste. Products that carry a "Seal of Approval" can take

In response to this consumer confusion, a professional review process for over-the-counter skin care products has been formed. In July 2007, the Dermatology Review Panel (DRP) was established to provide professional reviews of skin care product claims. The overall purpose of the DRP is two-fold. First, it will assist consumers and medical professionals to easily identify nonprescription skin care products that meet independent approval standards with regard to product claims. Second, it will encourage manufacturers to engage in more clinical research.

The Review and Approval Process

The DRP is comprised of leading dermatologists from across Canada. The Panel provides independent dermatological assessments of the available scientific data supporting the skin care-related product claims in order to ensure that they meet the criteria set out by the DRP.

Manufacturers are invited to submit skin care products to the DRP for review. The DRP accepts applications for Canadian over-the-counter skin care products and other consumer products that make skin care-related claims. Each product's scientific data is reviewed by a minimum of three reviewers, who are chosen from the panel depending on their expertise in a relevant product area. The reviewers independently assess each submission to verify that there is enough scientific data to support the product's claims. Evaluations are tabulated and a final decision is rendered by the Board of Governors, which is comprised of three additional nationally recognized dermatologists.

The Dermatology Review Panel Seal of Approval is only granted to products that meet the criteria set out by the DRP. The DRP seal is easy to recognize and can be prominently displayed on approved products; its visual impact is meant to encourage Canadians who want to make educated choices about their skin care products.

Using the Seal

Manufacturers can display the Seal of Approval on a product's packaging, advertisements, and any other promotional vehicles within Canada. Prominently placing the seal on products and promotional materials can help consumers and medical professionals easily identify products that have been reviewed by professionals and meet the criteria set out by the DRP.

The DRP Seal of Approval has received a number of submissions to date and several products have already received approval.

For more information about The Dermatology Review Panel and to learn what products have been approved, go to www.dermatologyreviewpanel.ca.

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