

## **Integrative Medicine Approach to Eczema (atopic dermatitis)**

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“Eczema is a term that refers to a group of inflammatory skin conditions. Atopic dermatitis, seborrheic dermatitis, contact dermatitis, nummular eczema, dyshidrotic eczema and lichen simplex are all in the eczema family.<sup>i</sup> The most common type of eczema is atopic dermatitis, which is defined as eczema occurring in a patient with a personal or family history of allergic conditions such as asthma or hay fever.”<sup>ii</sup> It is also the most common chronic illness in children. “Atopic dermatitis is a chronic skin disease characterized by itching, an impaired epithelial barrier, inflammation, significant bacterial colonization with frequent infections, and a clinical course punctuated by flares and remissions.”<sup>iii</sup> “There is no universal consensus for the meaning of the term atopy, although it is often used to refer to an inherited tendency to develop allergic conditions, such as asthma, hay fever, and AD (the atopic triad).”<sup>iv</sup> Atopic dermatitis (eczema) has a prevalence of 2-4 % to 7% percent of the population.<sup>v</sup> The current etiopathogenesis of atopic dermatitis involves the interplay of the following:

- Cutaneous barrier dysfunction and mutations in genes coding for skin proteins (filaggrin)
- Dysregulation of the immune system and the role of thymic stromal lymphopoietin in allergic inflammation
- Environmental factors

There is a myriad of endogenous and exogenous factors that may contribute to the pathogenesis of eczema, which include:

- Exogenous factors: phthalates, topical drugs, plants, cosmetics, metal compounds, dyes, detergents, chemicals, latex, wool, synthetic fibers, mineral oils, and bacterial and fungal pathogens. Pervasive exposure to phthalates can play a role in the pathogenesis of AD by stimulating the production of a thymic stromal lymphopoietin (IL-7 like cytokine) secreted by barrier-defective skin cells.<sup>vi</sup>
- Endogenous factors: immunological abnormalities (i.e. family history), environmental elements (e.g. food allergies), and emotional influences (e.g. stress).
- “Individuals with endogenous eczema may also demonstrate reduced ceramide content of the stratum corneum, reduced skin itch threshold, decreased antimicrobial peptide production of

keratinocytes, intestinal *Candida* overgrowth, increased pro-inflammatory cytokine production and intestinal dysbiosis.”<sup>vii</sup> An overgrowth of *Candida albicans* in the gastrointestinal tract may contribute to AD. Elevated levels of anticandidal antibodies are common in atopic individuals.<sup>viii</sup>

- “Gastric *H. pylori* directly stimulates epidermal cells to secrete thymic stromal lymphopietin, and *H. pylori* antibody is positive in up to 70% of AD patients.”<sup>ix</sup>

Food allergens can be a major contributing factor in atopic dermatitis (eczema). “One hundred thirteen patients with severe atopic dermatitis were evaluated for food hypersensitivity with double-blind placebo-controlled oral food challenges. Fifty-six percent of children experienced 101 positive food challenges; skin symptoms developed in 84% of the challenges. Eggs peanut, and milk accounted for 72% of the hypersensitivity reactions induced. When patients were given appropriate restrictive diets based on oral food challenge results, most showed significant improvement in their clinical course compared with patients in whom no food allergy was documented.”<sup>x</sup> “Since AD and food allergy are highly associated, patients with AD exhibit a much higher rate of food allergy than the general population. Elevated titers of IgE to specific allergens can be found in up to 85% of patients with AD.”<sup>xi</sup>

“Diagnosis of food allergy is usually best achieved via the elimination diet and challenge method. This approach is especially useful in childhood eczema. Elimination of milk products, eggs, peanuts, tomatoes and artificial colors and preservatives result in significant improvement in at least 75% of cases. If laboratory methods are used to identify food allergies in eczema, the most useful method appears to be the enzyme-linked immunosorbent assay (ELISA) IgE and IgG<sub>4</sub>.”<sup>xii</sup> Intestinal hyperpermeability may be a contributing factor for individuals with AD. “A defective skin barrier and increased intestinal permeability appear to facilitate allergen sensitization.”<sup>xiii</sup>

Most Common Allergic Foods by Age<sup>xiv</sup>

<b>Infants</b>	<b>Children (2-10 years)</b>	<b>Adolescents and Young Adults</b>
Cow’s milk	Cow’s milk	Peanut
Egg	Egg	Tree nuts
Wheat	Peanut	Fish
Soy	Tree nuts	Shellfish
	Fish	Sesame
	Shellfish	
	Sesame	
	Kiwi fruit	

Universal Criteria for the Diagnosis of Atopic Dermatitis<sup>xv</sup>

- A. Essential features; must be present and, if complete, are sufficient for diagnosis
  - 1. Pruritus
  - 2. Eczematous changes that are acute, subacute, or chronic
    - a. Typical and age-specific patterns
      - (i) Facial, neck, and extensor involvement in infants or children
      - (ii) Current or prior flexural lesions in adult/any age
      - (iii) Sparing of groin and axillary regions
    - b. Chronic or relapsing course
- B. Important features that are seen in most cases, adding support to the diagnosis
  - 1. Early age onset
  - 2. Atopy (IgE reactivity)
  - 3. Xerosis
- C. Associated features: Clinical associations; help in suggesting the diagnosis of AD but are too nonspecific to be used for defining or detecting AD for research and epidemiologic studies
  - 1. Keratosis pilaris/Ichthyosis/Palmar hyperlinearity
  - 2. Atypical vascular responses
  - 3. Perifollicular accentuation/Lichenification/Prurigo
  - 4. Ocular/periorbital changes
  - 5. Perioral/periauricular lesions
- D. Exclusions: Firm diagnosis of AD depends on excluding conditions such as scabies, allergic contact dermatitis, seborrheic dermatitis, cutaneous lymphoma, ichthyoses, psoriasis, and other primary disease entities

Treatment Strategies for Atopic Dermatitis (Eczema)

Therapeutic objectives<sup>xvi</sup>

- 1. Identify and eliminate foods that may contribute to or exacerbate eczema
- 2. Identify environmental toxin exposure and eliminate (e.g. phthalates)
- 3. Assess gastrointestinal system for dysbiosis (e.g. intestinal hypermeability, *H. pylori* infection, candidiasis, hypochlorhydria, digestive enzyme insufficiency, and inadequate probiotics)
- 4. Botanical medicine
- 5. Stress reduction (emotional stress can provoke itching)

6. Increase consumption of seafood (fish oil) to increase omega-3 fatty acids (used wild caught). Most clinical improvement correlates with increase in concentration of DHA in serum phospholipids
7. Increase consumption of fruits and vegetables to augment ascorbic acid, bioflavonoid, iron, vitamin A and vitamin E intake (use organic if possible)
8. Increase consumption of low-allergenic nuts and seeds to improve iron, niacin, selenium, vitamin E, omega-3 fatty acids and zinc intake
9. Increase lean meat consumption to improve iron, niacin, selenium, vitamin E, and zinc intake.

#### Basic Daily Adult Supplementation Considerations

- High potency multiple vitamin and mineral formula
- Vitamin E (400 IU of mixed tocopherols)
- Fish oil (1000 to 3000 mg EPA and DHA)
- Flaxseed oil (1 tablespoon)
- Ascorbic acid (250 mg b.i.d.)
- Probiotics (Lactobacilli rhamnosus  $1 \times 10^9$  cfu/ gram b.i.d.) Other probiotics to consider (Lactobacillus acidophilus and Bifidobacterium bifidum 5 to 10 cfu)

#### Herbal Medicine Strategies<sup>xvii</sup>

- The immune response can be balanced with immune modifiers, particularly Echinacea root. Experience that the herb does not aggravate atopic dermatitis (and may even shift responses away from Th-2 to Th-1). Boosting the immune response with Echinacea root, Astragalus, and Andrographis may help control *S. aureus* infection.
- The allergic and inflammatory responses can be controlled with anti-allergic herbs (e.g. Albizia, Baical skull cap, nettle leaf) and anti-inflammatory herbs (e.g. licorice, gotu kola, Bupleurum)
- Bitter herbs and aromatic digestives will improve digestion (if indicated)
- Long-term treatment with depuratives such as burdock, nettle leaf, cleavers, yellow dock, and sarsaparilla is aimed at correcting the metabolic imbalances underlying the disorder.
- Bacterial colonization of the skin can be corrected and inflammation allayed by topical treatment with anti-inflammatory and antiseptic herbs. The antiseptic herbs will help control skin microflora imbalance and infection with *S. aureus*. Calendula has both antiseptic and anti-inflammatory properties, and topical treatment with myrrh and Echinacea root may improve the cutaneous response. Golden seal contains antimicrobial alkaloids (it should not be used with tannins).

Sample Herbal Liquid Formulas to Consider

Formula 1 (3 ml of herbal mixture with warm water b.i.d.)

- |                        |             |       |
|------------------------|-------------|-------|
| – Echinacea root blend | 1:2 extract | 50 ml |
| – Baical skull cap     | 1:2 extract | 25 ml |
| – Nettle leaf          | 1:2 extract | 25 ml |

Formula 2 (5 ml of herbal mixture with warm water b.i.d.)

- |                        |             |       |
|------------------------|-------------|-------|
| – Astragalus           | 1:2 extract | 25 ml |
| – Echinacea root blend | 1:2 extract | 25 ml |
| – Gotu kola            | 1:2 extract | 20 ml |
| – Feverfew             | 1:5 extract | 10 ml |
| – Bupleurum            | 1:2 extract | 20 ml |

Topical Treatments<sup>xviii,xix</sup>

- Matricaria recutita (Chamomile) 1:2 liquid extract 20 ml  
Glycyrrhiza glabra (Licorice) 1:1 liquid extract 15 ml  
Reduce both of these extracts by half and pour, while hot, into 100 grams of base cream. Apply to lesions twice daily.
- Ceramide-containing emollients can be used to reduce trans epidermal water loss (e.g. CervVe, Nature Pure’s Hippophae-Ceramide Cream, Hippophae Oil [sea buckthorn], ceramides, squalane, olive oil, beeswax, extracts of St. John’s wort, ginko biloba, calendula, and chamomile)
- Glycyrrhetic acid-containing commercial preparations may be helpful. Chamomile, feverfew, oatmeal, and cardiospermum preparations are also popular and their use is supported in the scientific literature.
- Atopiclair: Prescription medication containing glycyrrhetic acid, shea butter, bisabolol (chamomile), vitamin E, aloe vera, and grape seed.
- 1% feverfew (parthenolide-free extract: Aveeno Daily Moisturizer, Ultra-Calming)
- Colloidal oatmeal contains starches and beta glucans that have protective and water holding functions, polyphenols (avenanthramides) are antioxidant, anti-inflammatory, and UV absorbers.
- Saponins have a cleaning activity (Aveeno Overnight Itch Relief Cream)

## Resources

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- <sup>i</sup> Saavedra JM. Atopic Dermatitis and Diet. In: Kohlstadt I. *Advancing Medicine with Food and Nutrients*. 2<sup>nd</sup> Ed. Boca Raton: CRC Press; 2013. p. 435.
- <sup>ii</sup> Gaby AR. *Nutritional Medicine*. Concord; Fritz Perlberg Publishing; 2011. p. 687.
- <sup>iii</sup> Lee KC, Lio PA. Integrative Management of Atopic Dermatitis. In: Norman RA, Shenefelt PD, Rupani RN. *Integrative Dermatology*. Oxford: Oxford University Press; 2014. p. 262.
- <sup>iv</sup> Saavedra JM. Atopic Dermatitis and Diet. In: Kohlstadt I. *Advancing Medicine with Food and Nutrients*. 2<sup>nd</sup> Ed. Boca Raton: CRC Press; 2013. p. 435.
- <sup>v</sup> Murray MT, Traub M. Atopic Dermatitis (Eczema). In: Pizzorno JE, Murray MT. *Textbook of Natural Medicine*. 4<sup>th</sup> Ed. St. Louis: Elsevier; 2013. p. 1246.
- <sup>vi</sup> *Ibid.* p. 1247.
- <sup>vii</sup> Leach MJ. The Dermatological System. In: Hechtman L. *Clinical Naturopathic Medicine*. Australia: Churchill Livingstone; 2012. p. 645-646.
- <sup>viii</sup> Murray MT, Traub M. Atopic Dermatitis (Eczema). In: Pizzorno JE, Murray MT. *Textbook of Natural Medicine*. 4<sup>th</sup> Ed. St. Louis: Elsevier; 2013. p. 1248.
- <sup>ix</sup> *Ibid.* p. 1247.
- <sup>x</sup> Lee KC, Lio PA. Integrative Management of Atopic Dermatitis. In: Norman RA, Shenefelt PD, Rupani RN. *Integrative Dermatology*. Oxford: Oxford University Press; 2014. p. 44.
- <sup>xi</sup> Saavedra JM. Atopic Dermatitis and Diet. In: Kohlstadt I. *Advancing Medicine with Food and Nutrients*. 2<sup>nd</sup> Ed. Boca Raton: CRC Press; 2013. p. 438.
- <sup>xii</sup> Murray MT, Traub M. Atopic Dermatitis (Eczema). In: Pizzorno JE, Murray MT. *Textbook of Natural Medicine*. 4<sup>th</sup> Ed. St. Louis: Elsevier; 2013. p. 1248.
- <sup>xiii</sup> *Ibid.* p. 1247.
- <sup>xiv</sup> *Ibid.* p. 439.
- <sup>xv</sup> *Ibid.* p. 436.
- <sup>xvi</sup> Leach MJ. The Dermatological System. In: Hechtman L. *Clinical Naturopathic Medicine*. Australia: Churchill Livingstone; 2012. p. 648.
- <sup>xvii</sup> Bone K. *A Clinical Guide to Blending Liquid Herbs*. St. Louis: Churchill Livingstone; 2003. p. 36.
- <sup>xviii</sup> *Ibid.* p. 46.
- <sup>xix</sup> Murray MT, Traub M. Atopic Dermatitis (Eczema). In: Pizzorno JE, Murray MT. *Textbook of Natural Medicine*. 4<sup>th</sup> Ed. St. Louis: Elsevier; 2013. p. 1250.