

Colloidal Oatmeal from *Avena sativa*: Bioactive Constituents and Its Role in the Management of Atopic Dermatitis

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ABSTRACT

This review aims to provide a clear and concise overview of colloidal oatmeal derived from *Avena sativa*, its bioactive constituents, and its therapeutic role in managing atopic dermatitis (AD). Rich bioactive constituents in colloidal oatmeal, particularly avenanthramides (AVNs), β -glucans, lipids, and polysaccharides, provide antioxidant, anti-inflammatory, moisturizing, and skin barrier-protectant properties. AD is a chronic inflammatory condition characterized by impaired skin barrier function, pruritus, dryness, and recurrent inflammation. Colloidal oatmeal helps manage AD by reducing trans-epidermal water loss, modulating inflammatory cytokines, relieving itch, and supporting the skin microbiome. Clinical studies have demonstrated that colloidal oatmeal is a safe, natural, and effective option as either a monotherapy or adjunctive treatment for the management of mild-to-moderate AD.

Keywords: Colloidal Oatmeal, *Avena sativa*, Bioactive Constituents

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Introduction

Oats have been used for centuries in a variety of skin conditions, such as atopic dermatitis (AD), xerotic dermatoses, cercarial dermatitis, razor burns, diaper rash, and erythema(1-4). Colloidal oatmeal was first formulated and used in 1945(5). The US-FDA officially listed colloidal oatmeal as a "skin protectant" ingredient for OTC products (Category I as safe and effective) in 1989(6). The US Pharmacopeia defines colloidal oatmeal as derived from only *A. sativa* or *A. byzantina*; it does not include *A. nuda* or *A. strigosa*. It indicates that oats used to make colloidal oatmeal must meet No. 1 or 2 grade standards, i.e., 97% or 94% undamaged oats, respectively, and may contain, singly or in combination, not more than 25% wild oats and other grains for which standards have been established under the US Grain Standards Act 2[7CFR810.1001].



Figure 1: Colloidal Oatmeal Bathbombs

Constituents

Oats are rich in essential *amino acids* such as leucine (8%), phenylalanine (6%), valine (5%), and lysine (4%)(7). *Avenacosides* are inactive saponins(8, 9). Tissue damage activates these into 26-desglucoavenacosides (by the avenacosidase enzyme), which have antifungal properties(10). *Carbohydrates* include glucose, xylose, arabinose, galactose, mannose, uronic acid, fucose, and rhamnose, starches (65-85%) and β -glucan (5-7%)(11-13). *Enzymes* found in oat lipids include lipases, peroxidases, lipoxygenases, and amylases(14). *Flavonoids* such as 2',4,4',6'-tetrahydroxy-3-methoxychalcone; kaempferol-3-O-(2'',3''-di-Ep-coumaroyl)- α -L-rhamnopyranoside; linarin; tilianin; myricitrin; quercitrin; and tricrin are reported(15, 16). Calcium, phosphorus, potassium, magnesium, ferrous, manganese, copper, sodium, chloride, and cobalt have been reported(17). Avenanthramides (AVNs) are a unique group of *N*-cinnamoyl-anthranilic acids found exclusively in oats(18, 19). Other *phenolic* compounds include benzoic/cinnamic acids, quinones, flavones, chalcones, flavanones, anthocyanidins, and aminophenolics(20). Choline (755-1712ppm), niacin (5.9-44ppm), pantothenic acid (4.4-28.4ppm), and α -tocopherol (4.4-10.1ppm), thiamine, riboflavin, pyridoxine, carotene, folic acid, and biotin are reported(17).

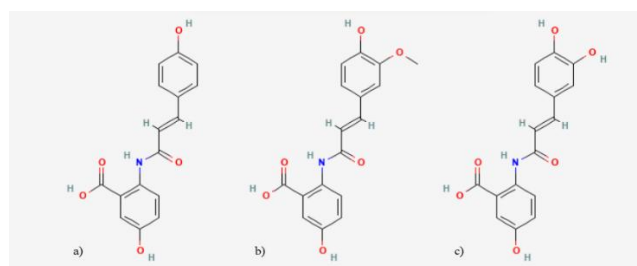


Figure 2: a) Avenanthramide A; b) Avenanthramide B; and c) Avenanthramide C

Atopic Dermatitis

Pathophysiology of AD is attributed to environmental factors and genetic predisposition, skin barrier dysregulation, immunoglobulin E (IgE) sensitization, and immune dysfunction.

Epidermis is a critical physical and functional barrier. Some of the key proteins involved in epidermal integrity include filaggrin (FLG), transglutaminases, keratins, and intercellular proteins. The filaggrin (FLG) gene, encoded on chromosome 1q2, produces filaggrin, a major structural protein in the stratum corneum (SC)(21). Pro-FLG polymers are proteolytically cleaved, and the dephosphorylated monomers bind with keratin fibers to form the SC(22). The degradation products during FLG production, urocanic acid and pyrrolidine carboxylic acid, are responsible for skin hydration and pH. Any homozygous null mutation in the FLG gene may result in AD(23-25).

Any alterations T-helper (T_H) type-2 signaling pathway, such as cytokines, TSLP, IL-25, IL-33, and NF- κ B are also associated with increased risk of AD(21, 26). The JAK/STAT pathway leads to the release of multiple pro-inflammatory cytokines, such as interleukin (IL)-4, IL-13, IL-31, and thymic stromal lymphopoietin (TSLP), thereby contributing to immune dysregulation and epidermal barrier dysfunction(27).

Upregulation of interleukin IL-4 and IL-13, or gain of functional polymorphisms in type-2 cytokine receptors (IL-4R and IL-13R), is hypothesized to lower FLG expression(28). IL-4 and IL-13 also activate T_H2 cells, inducing differentiation and activation of myeloid and atopic dendritic cells. B cells are activated, which stimulates IgE class-switching and the recruitment of eosinophils(29). Other immune-related genes implicated in the pathogenesis of AD include IL-31, IL-33, STAT 6, thymic stromal lymphopoietin (TSLP) and its receptors (IL-7R and TSLPR), interferon regulatory factor 2, Toll-like receptor 2, and high-affinity IgE receptor (Fc ϵ RI) α gene in specific populations(21, 26, 30, 31).

IL-31 also enhances the production of BDNF, resulting in a cytokine and chemokine response that causes intense itching(32). IL-25, IL-33, and TSLP also act directly on nerves to induce pruritic itch through a type-2 immune response. Antimicrobial peptides (AMPs), including cathelicidins and human β -defensins, play a key role in host defense. AMP expression is inhibited in the Th2 cytokine response. This facilitates the colonization of *Staphylococcus aureus* (*S. aureus*) on the skin and exacerbates inflammation(33, 34). Lipids are necessary to maintain the integrity of the

epidermal barrier(35). Some lipids, such as ω -O-acylceramides, long-chain FFAs, and cholesterol levels, are decreased as a result of the Th2 cytokine response. This further exposes AD patients to infections and increased transepidermal water loss (TEWL).

Literature Review

The AVNs in oatmeal are known to provide significant relief in AD by reducing pro-inflammatory cytokines, neutralizing histamine release from IgE-sensitized cells, and restoring the broken skin barrier. Some of the proposed mechanisms by which AVNs provide dermatological benefits include inhibition of NF- κ B signaling via I κ B-degradation inhibition, which subsequently decreases the production of pro-inflammatory cytokines, such as IL-1 β , IL-6, IL-7, IL-8, and TNF- α (18). The starches and β -glucans in oatmeal adhere to skin, pull in and lock the moisture into the SC, and create an occlusive film(36). It also modulates the activity of commensal skin microbes (*S. epidermidis*) by increasing the production of short-chain fatty acids (SCFAs) and lactic acid, which are major components of natural moisturizing factor(37).

Synthetic AVNs, such as dihydroavananthramide D (dhAvD), have been shown to restore barrier function and normalize the expression of tight junction proteins and natural moisturizing factors in skin, supported by various studies conducted on Human adult low-Calcium high-Temperature (HaCaT) cell lines. AVNs and dhAvD also block the release of histamine and substance P from sensory nerve fibers and mast cells, effectively interrupting the itch-scratch cycle(38).

Listane and colleagues conducted two single-center, single-arm clinical trials in patients with mild-to-moderate AD to evaluate the effects of 1% OTC cream administered for 14 days. Study 1 assessed the Eczema Area and Severity Index (EASI) and Investigator's Global Atopic Dermatitis Assessment (IGADA) on day 3, and itch severity using a Visual Analogue Scale (VAS) immediately after application as primary efficacy endpoints. Compared to baseline (20%), improvements in EASI (53.6%) and IGADA scores (25.0%) were observed at day 3. On day 14, these percentages were 82.8%, 62.1%, and 85.7%, respectively. In study 2, the primary efficacy endpoint was the change from baseline in patients' assessment of itch. Itch severity and EASI score improved significantly after application and sustained until day 14. TEWL was significantly reduced, and skin hydration was significantly increased(3).

In another double-blind study by Lisante, 90 eligible patients (6 months to 18 years) were randomized to OTC or prescription cream. EASI scores, IGADA scores, and patients/caregivers' itch severity, using VAS, were measured on day 0 & weeks 1, 2, and 3. At week 3, EASI scores showed that the OTC cream was non-inferior to the prescription cream. IGADA (0.106) and VAS itch (0.103) scores improved in both groups. No safety issues were identified. OTC 1% oatmeal cream was equally effective and safe as the prescription barrier cream for the symptomatic treatment of mild-to-moderate AD in children(39).

A *post hoc* analysis in 49 Black or African American children (age 2-15 years) was conducted by Lisante and Co. At week 3, mean (SD) change from baseline in EASI scores was -2.4 with 1% oatmeal cream and -2.1 with barrier cream; improvements were observed in week 1. At week 3, mean (SD) changes from baseline in IGADA scores were -0.6 and -0.7, respectively. Subjective ratings of signs and symptoms of eczema showed significant improvements(40).

A 2015 single-center, randomized trial studied an OTC Body Cream (containing 1% colloidal oatmeal, licochalcone A from *Glycyrrhiza inflata* roots, ceramide 3) for 38 weeks. After a 2-week washout period, subjects were randomized to cleanser plus daily moisturizing with Body Cream (test) or cleanser only (control) for 6 months or until flare. The incidence of flare was significantly lower in the test group compared to the control. The median time to flare was shorter in the control group (28 vs. >180 days). Flare risk was reduced by 44.1% after 6 months of Body Cream application(41).

In another randomized-controlled trial by Capone, a 1% cream was compared with a standard non-fragranced moisturizer in 61 patients with mild-to-moderate AD. Patients applied the assigned product twice daily for 14 days. The 1% cream reduced EASI scores by 51% and ADSI scores by 54% at day 14. It was also associated with favorable microbiome changes (reduced *Staphylococcus* prevalence and increased diversity) and significantly improved skin pH, barrier function, and hydration, whereas the standard moisturizer only improved hydration(42).

A study by Whiting suggests a moisturizer containing colloidal oatmeal, patented filaggrin technology, niacinamide, and tocopheryl acetate selectively inhibited the growth and adhesion of pathogenic *S. aureus* while promoting commensal *S. epidermidis* on reconstructed human epidermis (RHE) ($P < 0.001$)(43).

Conclusion

Colloidal oatmeal is a time-tested, safe, and effective ingredient for AD management. Since it is effective on its own, it offers a minimal skincare approach, reducing reliance on chemical-based formulations. In addition, it is a highly sustainable, biodegradable product with a very low environmental footprint.

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