

# ClinicalNotes

## Stronger at the Surface: Managing Atopic Dermatitis From the Inside Out

Joya Griffin, DVM, DACVD

Animal Dermatology Clinic

Louisville, Kentucky

Canine atopic dermatitis (CAD) and feline atopic skin syndrome (FASS) are chronic, inflammatory, pruritic skin conditions that arise from an intermix of genetic predisposition, environmental allergen exposure, immune dysregulation, and skin barrier dysfunction.<sup>1</sup>

The development of atopic disease involves increased skin inflammation, leading to exacerbation of skin barrier defects, increased transcutaneous allergen penetration, dysbiosis of the cutaneous microbiome, and perpetuated inflammation. Breaking the cycle is paramount to successfully managing atopic disease and improving clinical signs of pruritus.

### Species Overview & Differences

Atopic dogs are often presented with pruritus that is initially nonlesional, which can lead to dermatitis affecting the face, feet, ears, ventrum, and flexural surfaces. These lesions are often complicated by secondary staphylococcal or *Malassezia* spp infections.<sup>2</sup> As the disease progresses, barrier dysfunction worsens, clinical signs become more severe, and secondary infections can become more prevalent.

Cats with FASS can be presented with varying lesions. These lesions can be classified among 4 clinical reaction patterns that can mimic other dermatologic conditions: head and neck pruritus, eosinophilic granuloma complex, miliary dermatitis, or self-induced alopecia, which mirrors what typically occurs in dogs. These reaction patterns can occur alone or in combination with each other, and clinical signs can vary from year to year. Some cats may also have concurrent noncutaneous GI and respiratory signs or conjunctivitis.<sup>3</sup>

In both species, allergens penetrate a compromised skin barrier, triggering immunoglobulin E production, T-helper 2 dominant responses, and mast cell activation.<sup>4-6</sup> Barrier dysfunction is well-characterized in dogs, but much less is known in cats. Studies in dogs have shown reduced ceramides and increased transepidermal water loss in lesional skin.<sup>7,8</sup> Alterations in lipid subsets, filaggrin expression, and microbiome composition are important drivers of allergic flares.<sup>4,5</sup> Less is known about the microbiota of cats; however, fungal and bacterial dysbiosis can occur and increased adherence of staphylococcal bacteria has been reported in lesional skin of allergic cats.<sup>9,10</sup>

### The Skin Barrier: Why It Matters

In healthy skin, ceramides and sphingolipids maintain cohesion and hydration, which provide a protective mechanism. In atopic patients, ceramide depletion, abnormal lipid metabolism, and dysbiosis allow for increased allergen penetration, persistent inflammation, and subsequent worsening of clinical signs of atopic dermatitis. This altered barrier function may increase risk for secondary infections.<sup>7</sup> Supporting and repairing barrier function are key in the multimodal management of atopic dermatitis and preventing disease progression.<sup>11</sup>

### Multimodal Management Strategies

Management goals of CAD and FASS involve reducing inflammation and pruritus, preventing allergic flares, and supporting the skin barrier. Plans should be individualized and tailored to each patient's needs and severity, chronicity of the patient's clinical signs, and incidence of secondary infection. Utilizing antipruritic medications like Janus kinase inhibitors, monoclonal antibodies, cyclosporine, or glucocorticoids to reduce and control pruritus is paramount to preventing further disruption of skin barrier function while initiating a comprehensive treatment plan.

FASS management has fewer approved pharmaceutical options, with cyclosporine and glucocorticoids being the mainstays. Oclacitinib and maropitant have been used off-label to control pruritus, but limited studies on their efficacy and safety exist.<sup>12,13</sup>

Allergen-specific immunotherapy is the only treatment that alters the progression of atopic dermatitis by targeting the actual pathogenesis. Immunotherapy aims to reduce the allergic flare and prevent worsening by downregulating the immune response and promoting immune tolerance.<sup>14</sup> When immunotherapy is initiated, antipruritic medications should be utilized as anchor therapy during the induction period.

## Barrier Support

Supporting the skin barrier can be achieved topically or orally with lipids, fatty acids, and anti-inflammatory components.

## Topical Options for Skin Barrier Support

Studies suggest that phytosphingosine, glycosaminoglycan, and ceramide-containing moisturizers and lipid spot-ons can lower transepidermal water loss and clinical severity in dogs.<sup>11,15</sup> Educating owners on correct use, frequency, and duration is key to success, and some patients may be better candidates than others depending on patient and owner factors.

## Oral Options for Skin Barrier Support

Certain ingredients in oral supplements provide essential building blocks for barrier repair and hydration:

- **Omega-3 fatty acids:** Omega-3 fatty acids reduce proinflammatory eicosanoids and support barrier lipids.<sup>16</sup>
- **Beta-1,3/1,6-glucans:** These polysaccharides modulate immune pathways. Clinical and experimental studies suggest they may have benefits in reducing pruritus.<sup>17</sup>
- **Micronized palmitoylethanolamide (PEA):** An endocannabinoid-related lipid first isolated from soy lecithin in 1957, micronized PEA has been found to stabilize mast cells, decrease mast cell degranulation and histamine-related pruritus, and modulate inflammatory mediators. In a multicenter trial, dogs with moderate CAD that received PEA supplementation demonstrated significant improvement in pruritus and lesions,<sup>18</sup> and 45% of study dogs achieved quality of life scores like that of normal dogs.<sup>18</sup>
- **Ceramides:** Ceramides play a role in forming the lipid lamellae that reduces transepidermal water loss. They maintain skin hydration and flexibility and help lessen penetration of environmental allergens and microorganisms.<sup>4,5</sup>
- **Hardy kiwi extract (*Actinidia arguta*):** Traditionally, hardy kiwi has been used in Eastern medicine to promote healthy response to allergens. Oral administration in animal models has been shown to help reduce immunoglobulin E production,

## Key Takeaways

- Skin barrier dysfunction is a key driver of CAD and FASS, making skin barrier repair an important focus of case management.
- Dogs with atopic disease are generally presented with pruritus and often secondary infections, whereas cats tend to have a more variable clinical presentation.
- Allergen-specific immunotherapy is the only intervention that can modify the allergic disease process and should be considered when appropriate.
- An individualized, multimodal management approach that includes antipruritics, infection control, and barrier support is required for long-term success in CAD and FASS.
- Oral and topical barrier support products can be utilized to help reduce patient flares and improve long-term outcomes.

mast cell degranulation, and dermatitis severity.<sup>19</sup> It has also been found to reduce pruritus scores associated with atopic dermatitis when combined with glucocorticoids, and these scores were maintained after cessation of the glucocorticoids.<sup>19</sup>

One oral option for skin barrier support is Dermaquin<sup>®</sup> Skin Support Supplement, which has been reformulated with the addition of micronized PEA. It also contains beta-1,3/1,6-glucan, hardy kiwi extract, and omega-3 fatty acids. This combination serves to help fortify the skin barrier, provide hydration to the skin, and support a healthy response to allergens through immune modulation, neuroinflammatory control, and barrier lipid support.<sup>20</sup> Another oral option is Welactin<sup>®</sup>, an omega-3 supplement that provides high levels of EPA and DHA. For dogs requiring higher omega-3 supplementation when targeting inflammation-modulating levels of administration, Welactin<sup>®</sup> is a quality adjunct. Welactin<sup>®</sup> Canine Liquid delivers ≈1,610 mg of omega-3s (≈1,330 mg EPA + DHA) per each 6-mL scoop.<sup>21</sup>

## Conclusion

CAD and FASS are lifelong, multifactorial diseases. Barrier dysfunction is not merely a feature but a central driver, making barrier support integral to successful management. A layered multimodal approach that includes antipruritic medications, immunotherapy, and oral and topical barrier support offers the best outcomes. ●

For references, please visit  
[cliniciansbrief.com/article/stronger-surface-managing-atopic-dermatitis-from-inside-out](https://cliniciansbrief.com/article/stronger-surface-managing-atopic-dermatitis-from-inside-out)

Copyright 2025, Educational Concepts, LLC

## References

1. Hensel P, Saridomichelakis M, Eisenschenk M, et al. Update on the role of genetic factors, environmental factors and allergens in canine atopic dermatitis. *Vet Dermatol*. 2024;35(1):15-24. doi:10.1111/vde.13210
2. Griffin CE, DeBoer DJ. The ACVD task force on canine atopic dermatitis (XIV): clinical manifestations of canine atopic dermatitis. *Vet Immunol Immunopathol*. 2001;81(3-4):255-269. doi:10.1016/s0165-2427(01)00346-4
3. Santoro D, Pucheu-Haston CM, Prost C, Mueller RS, Jackson H. Clinical signs and diagnosis of feline atopic syndrome: detailed guidelines for a correct diagnosis. *Vet Dermatol*. 2021;32(1):26-e6. doi:10.1111/vde.12935
4. Popa I, Remoue N, Osta B, et al. The lipid alterations in the stratum corneum of dogs with atopic dermatitis are alleviated by topical application of a sphingolipid-containing emulsion. *Clin Exp Dermatol*. 2012;37(6):665-671. doi:10.1111/j.1365-2230.2011.04313.x
5. Martins GC, de Oliveira Melo Júnior OA, Botoni LS, et al. Clinical-pathological and immunological biomarkers in dogs with atopic dermatitis. *Vet Immunol Immunopathol*. 2018;205:58-64. doi:10.1016/j.vetimm.2018.10.009
6. Halliwell R, Banovic F, Mueller RS, Olivry T. Immunopathogenesis of the feline atopic syndrome. *Vet Dermatol*. 2021;32(1):13-e4. doi:10.1111/vde.12928
7. Santoro D, Marsella R, Pucheu-Haston CM, Eisenschenk MN, Nuttall T, Bizikova P. Review: pathogenesis of canine atopic dermatitis: skin barrier and host-micro-organism interaction. *Vet Dermatol*. 2015;26(2):84-e25. doi:10.1111/vde.12197
8. Shimada K, Yoon JS, Yoshihara T, Iwasaki T, Nishifuji K. Increased transepidermal water loss and decreased ceramide content in lesional and non-lesional skin of dogs with atopic dermatitis. *Vet Dermatol*. 2009;20(5-6):541-546. doi:10.1111/j.1365-3164.2009.00847.x
9. Meason-Smith C, Diesel A, Patterson AP, et al. Characterization of the cutaneous mycobiota in healthy and allergic cats using next generation sequencing. *Vet Dermatol*. 2017;28(1):71-e17. doi:10.1111/vde.12373
10. Older CE, Diesel A, Patterson AP, et al. The feline skin microbiota: the bacteria inhabiting the skin of healthy and allergic cats. *PLoS One*. 2017;12(6):e0178555. doi:10.1371/journal.pone.0178555
11. Drechsler Y, Dong C, Clark DE, Kaur G. Canine atopic dermatitis: prevalence, impact, and management strategies. *Vet Med (Auckl)*. 2024;15:15-29. doi:10.2147/VMRR.S412570
12. Mueller RS, Nuttall T, Prost C, Schulz B, Bizikova P. Treatment of the feline atopic syndrome - a systematic review. *Vet Dermatol*. 2021;32(1):43-e8. doi:10.1111/vde.12933
13. Bajwa J. Feline atopic syndrome - an update. *Can Vet J*. 2021;62(11):1237-1240.
14. Mueller RS. A systematic review of allergen immunotherapy, a successful therapy for canine atopic dermatitis and feline atopic skin syndrome. *J Am Vet Med Assoc*. 2023;261(S1):S30-S35. doi:10.2460/javma.22.12.0576
15. Tretter S, Mueller RS. The influence of topical unsaturated fatty acids and essential oils on normal and atopic dogs. *J Am Anim Hosp Assoc*. 2011;47(4):236-240. doi:10.5326/JAAHA-MS-5607
16. Kar P, Rajesh JB, Behera SK, et al. Omega fatty acids and its role in amelioration of canine dermatologic disorders. *J Sci Res Reports*. 2024;30(11):910-921. doi:10.9734/jsrr/2024/v30i112618
17. Amaral AR, Risolia LW, Rentas MF, et al. Translating human and animal model studies to dogs' and cats' veterinary care: Beta-glucans application for skin disease, osteoarthritis, and inflammatory bowel disease management. *Microorganisms*. 2024;12(6):1071.
18. Noli C, Della Valle MF, Miolo A, Medori C, Schievano C; Skinalia Clinical Research Group. Efficacy of ultra-micronized palmitoylethanolamide in canine atopic dermatitis: an open-label multi-centre study. *Vet Dermatol*. 2015;26(6):432-440, e101. doi:10.1111/vde.12250
19. Marsella R, Messinger L, Zabel S, et al. A randomized, double-blind, placebo-controlled study to evaluate the effect of EFF1001, an *Actinidia arguta* (hardy kiwi) preparation, on CADESI score and pruritus in dogs with mild to moderate atopic dermatitis. *Vet Dermatol*. 2010;21(1):50-57. doi:10.1111/j.1365-3164.2009.00773.x
20. Nutramax Laboratories. Dermaquin®. Nutramax website. Accessed August 2025. <https://www.dermaquin.com/product-information>
21. Nutramax Laboratories. Welactin®. Nutramax website. Accessed August 2025. <https://www.welactin.com/omega-3-supplements/dogs/liquid>