Atopic dermatitis (AD) is a multifaceted disease resulting from a complex interaction between environmental and genetic factors. These factors can shape skin barrier function and the immunologic response of predisposed patients. There is increasing evidence that an impaired skin barrier plays a role in human AD. Canine AD shares many clinical and immunologic similarities with human AD, and evidence is rapidly building to support a role for skin barrier dysfunction in canine AD. It is currently proposed that genetically inherited mutations affecting barrier function in conjunction with acquired environmental stressors lead to increased allergen penetration. Impaired skin barrier function in human AD has been linked to a variety of epidermal abnormalities, although it is unclear whether they are primary, secondary, or both. Lipid deficiency in the skin of atopic patients is considered an important component of the disease.

Preliminary evidence shows that ceramides are decreased in the skin of dogs with AD and that this defect might possibly contribute to impairment of the skin barrier. Proteins important in the epidermal differentiation and cornification process have also been reported to be decreased in human patients with AD. Besides mutations in the genes encoding for proteins, there is also evidence of mutations in the genes encoding for proteases and protease inhibitors in human AD. Elevations in protease activities in humans with AD have been associated with impaired barrier function, irritation, and reduced skin capacitance. The demonstration of meaningful clinical improvement in well-conducted trials will confirm whether the approach of correcting a barrier dysfunction, if present, is valid.

Commentary: When I was in school, canine AD was called canine inhaled dermatitis because it was thought the disease was the result of inhaling allergens. Although the research on skin barrier disruptions in dogs is in its infancy, there is enough preliminary evidence to suggest that, as in people, part of the pathogenesis of AD in dogs involves abnormal skin barrier function. It’s interesting to know that human filaggrin mutations are now considered to be the most important risk factor for human AD, but how does one use this information in practice? In my practice, we emphasize to clients the importance of topical therapy. Treatment needs to be tailored to the patient and owners. Guidelines for the treatment of AD can be found in a recent issue of Clinician’s Brief (cliniciansbrief.com/column/ask-expert/canine-atopic-dermatitis). Another source, “What’s New—the Barrier Function in Canine Atopic Dermatitis,” can be found at virbacuniversity.com/Dermatology, along with modules on atopy and principles of topical therapy, as well as a video on barrier function and AD by Douglas DeBoer, DVM, Diplomate ACVD. — Karen Moriello, DVM, Diplomate ACVD

Current evidence of skin barrier dysfunction in human and canine atopic dermatitis.