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The ACVD task force on canine atopic dermatitis (VIII): is the epidermal lipid barrier defective?

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Abstract

In humans with atopic dermatitis (AD), it is suspected that the epidermal lipid barrier is abnormal because of combined insufficient extrusion of lipid-containing organelles into the superficial epidermal intercellular spaces as well as skin lipid metabolic defects. To date, studies investigating skin hydration and lipids in atopic dogs are scarce and unfortunately have yielded conflicting data. Whether or not dogs with AD exhibit dry skin and an inadequate stratum corneum barrier, therefore, remains the subject of speculation.

Keywords: Allergic disease; Atopy; Dog; Fatty acids; Lipid; Skin

1. Introduction

The outermost layer of the epidermis is composed of desquamating corneocytes surrounded by intercellular lipids. These lipids are thought to be critical for normal skin barrier function and provide a major protective function for the host. To fully understand the nature of the epidermal lipid barrier, the reader is referred to recent review papers on epidermal lipids, although almost all of the information in these articles was derived from studies using rodent and human specimens (Kwochka, 1993; Bouwstra et al., 2000; Wertz, 2000).

To date, very few studies have focused on the characteristics of epidermal lipids in dogs. Canine skin samples analyzed by means of low-temperature scanning electron microscopy were found, in fact, to contain very little intercellular lipids, in contrast to those of other

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mammalian species (Mason and Lloyd, 1993). In this paper, we will highlight results of studies investigating skin hydration and epidermal lipids in dogs with atopic dermatitis (AD).

2. Skin hydration and epidermal lipids in humans with atopic dermatitis

Human patients with AD commonly exhibit xerosis, a term describing abnormal skin dryness (Linde, 1992). It is hypothesized that a defective extrusion of lipid-containing organelles, the lamellar bodies, results in changes in the chemical composition of the epidermal lipid barrier and increased transepidermal water loss (Fartasch and Diepgen, 1992; Fartasch, 1994). Additionally, a novel enzyme with sphingomyelin deacylase activity has been found recently in the skin of humans with AD (Hara et al., 2000). This enzyme competes with sphingomyelinase and β-glucocerebrosidase for sphingomyelin or glycosylceramide substrates and thus, causes a deficiency in epidermal ceramides (Hara et al., 2000).

3. Skin hydration and epidermal lipids in dogs with atopic dermatitis

To date, only rare studies have focused on the hydration status and lipid content of the epidermis in dogs with AD. Recently, Chesney studied skin moisture, hygroscopicity and water-holding capacity of canine atopic skin by measuring the skin electrical capacitance (Chesney, 1995). Because water absorption–desorption values of dogs with AD did not differ from that of normal dogs, it was hypothesized that a defect in the epidermal lipid barrier leading to ''dry'' skin would be unlikely in this disease (Chesney, 1995).

In dogs with AD, studies on cutaneous and plasma lipid compositions have yielded conflicting results. In 1990, Van den Broek showed that the peak serum triglyceride concentration recorded after feeding corn oil was significantly lower in atopic than normal dogs (Van den Broek and Simpson, 1990). Thus, it was postulated that an impaired fat absorption or increased plasma triglyceride clearance might be present in atopic dogs (Van den Broek and Simpson, 1990).

At the same time, an unpublished abstract reported that normal humans and dogs exhibited slightly lower levels of plasma linoleic acid (LA, 18:2n6) and higher levels of dihomogammalinolenic (DGLA, 20:3n6) and arachidonic (AA, 20:4n6) acids than subjects with AD. These findings were deemed consistent with a δ-6 desaturase deficiency resulting in abnormal fatty acid metabolism[1].

In contrast, two recent studies did not support the concept of a universal δ-6 desaturase deficiency in dogs with AD (Scott et al., 1997; Taugbol et al., 1998). In fact, these two reports revealed the existence of abnormalities in DGLA metabolism possibly due to a decreased function of the δ-5 desaturase enzyme (Scott et al., 1997; Taugbol et al., 1998).

[1] Horrobin, D., Dobbin, S., Reynolds, B., 1990. Plasma fatty acids in dogs and their response to essential fatty acid supplementation. In: von Tscharner, C., Halliwell, R.E.W. (Eds.), Advances in Veterinary Dermatology. Vol. 1, Baillière-Tindall, London, pp. 473–474 (Abstract).

Finally, and to add to the confusion, a last unpublished report established that there were no significant differences in serum or skin abrasion fluid fatty acid profiles between normal, atopic, or seborrheic dogs[2]. Hence, the issue that a defect in fatty acid metabolism could underlie the pathogenesis of canine AD is far from being resolved.

Nevertheless, a recent pilot study established, by means of ruthenium tetroxide electron microscopy, that the epidermal lipid barrier was disrupted in nonlesional atopic canine skin. Indeed, the length and thickness of stratum corneum lipid deposits were lower in nonlesional atopic than normal canine skin. Moreover, intercellular lipid lamellae exhibited many structural defects in the stratum corneum of dogs with AD. An abnormal extrusion of lamellar bodies was not found in canine atopic skin, however. These morphologic studies suggested that an epidermal barrier defect could be present in atopic canine skin[3] (Inman et al., 2001).

4. Conclusions

Additional studies should be performed to corroborate whether or not an epidermal lipid barrier defect exists in canine atopic skin. Projects aimed at characterizing the biochemical composition of normal and atopic canine skin lipids should be undertaken. Furthermore, the influence of a defective epidermal barrier defect on epidermal allergen contact could be studied. Correction of this putative abnormality with pharmacological or nutritional agents then might be attempted. Indeed, such a therapeutic approach was validated with the recent demonstration that various dietary fat sources can influence both serum and cutaneous fatty acid concentrations (Campbell et al., 1995). Moreover, supplementation with linoleic acid-enriched diets can result in a significant decrease in transepidermal water loss, thus, suggesting that orally administered fatty acids were incorporated in the epidermal intercellular lipids (Marsh et al., 2000).

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