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An allergen challenge results in a reversible reduction in stratum corneum filaggrin degradation products in sensitized atopic dogs

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A defective skin barrier occurs in dogs with atopic dermatitis, but there is controversy whether this defect preexists, or is secondary to, allergic inflammation. Our objectives were to study if an allergen challenge led to decreases in natural moisturizing factor (NMF), the main filaggrin degradation products. We challenged four house dust mite (HDM)-sensitized adult Maltese-beagle atopic dogs epicutaneously with HDMs, once, on the right lateral abdomen, while an area on the left lateral thorax served as an unchallenged control. The skin surface was swabbed before and 1, 2, 3, 7 and 28 days after a challenge on selected sites; swabs were soaked in a detergent solution and frozen until assayed. The components of NMF were measured by liquid chromatography-tandem mass spectrometry (LC/MS-MS). The allergen challenge induced the development of moderate skin lesions at the application site and to mild erythema at the control site; this prompted a 3-day administration of oclacitinib. Allergen provocation also led to significant decreases in total NMF and its components trans-urocanic acid, pyrrolidone carboxylic acid and serine at both sites. Lesion scores abated within 7 days, while NMF levels reverted to baseline by 28 days after challenge. Skin lesion scores were negatively correlated with NMF levels. In conclusion, a cutaneous allergen challenge leads, not only to the previously shown reversible reduction in epidermal lipids, but also to a transient decrease in NMF components. Altogether, these observations suggest that the skin barrier defects that are seen in atopic dogs are reversible and develop mainly following allergic inflammation.

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Efficacy of oclacitinib in allergic cats: a multicentric randomised, blinded, methylprednisolone-controlled study

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Oclacitinib is a Janus kinase inhibitor that decreases interleukin-31-induced pruritus in cats. At 0.4-0.6 mg/kg/day orally, it relieves pruritus and skin lesions in less than half of allergic cats. We aimed to evaluate the efficacy and safety of a higher dosage of oclacitinib in feline non-flea-non-food-induced hypersensitivity dermatitis (NFNFIHD). Affected cats were randomly assigned to receive oclacitinib (group A, 18 cats, 0.8-1.3mg/kg) or methylprednisolone (group B, 14, 0.5-1 mg/kg) orally, twice daily for 28 days. On Day 1 and 28, veterinarians evaluated lesions with the Scoring Feline Allergic Dermatitis (SCORFAD) scale, while owners assessed pruritus with a 10-cm visual analogue scale (VAS) and quality of life (QoL) with a validated questionnaire. Hematochemical tests were performed before and after treatment. Results were analysed with a General Linear Mixed Model (significance: $P < 0.05$). There were no significant differences between parameters at baseline. In both groups, values improved significantly over time. In cats treated with oclacitinib, the mean percentage reduction in SCORFAD and VAS was 62 and 51%, respectively. In those treated with methylprednisolone, decreases were 77% and 74%, respectively; there was no significant difference between groups at study end. No response was seen in five and one cat from groups A and B, respectively. Scores of QoL improved similarly in both groups (26 versus 23%). Hematochemical changes were not noted on any cat. In conclusion, oclacitinib at 0.8–1.3 mg/kg orally twice daily appears safe for 28 days, and it is as effective as methylprednisolone for the treatment of feline NFNFIHD.

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Influence of a *Toxocara canis* infestation in a model of *Dermatophagoides farinae*-sensitized beagles

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The protective influence of helminth infestation for the development of canine atopic dermatitis is suspected but unproven. We selected twelve 10-week-old beagles: six were infested orally with embryonated *Toxocara canis* (*Tc*) eggs and six served as noninfested controls. All dogs were then dewormed to avoid clinical signs associated with an active *Tc* infestation. All dogs were sensitized to *Df* with eight repeated applications of a mite extract on taped-stripped skin. *Toxocara canis*-specific IgE and IgG were measured before and after nematode infestation. All dogs were *Df*-challenged in the groin, two times for three days, and local clinical scores (LCS) were recorded (0-5 for erythema, papules and excoriations in the right and left abdominal sides). Additionally, the durations of pruritus manifestations at the site of *Df* challenge were video-recorded on Days 2 and 3 after each challenge. *Toxocara* infestation led to a marked increase in *Tc*-specific IgG and IgE, while noninfested dogs remained seronegative. Before *Df* sensitization, the LCS was zero in all dogs and 6-hour pruritus durations ranged from 0 to 55 seconds. After the second Df challenge, the LCS ranged from 6 to 18 while the pruritus durations varied from 71 to 558 seconds. The mean pruritus durations in the infested and noninfested groups were 195 and 363 seconds, respectively (one-tailed t-test, p=0.05). The mean LCS in the infested and noninfested groups were 9 and 12, respectively (t-test, p=0.1). These observations suggest a possible sensitization protection effect of the *Tc* infestation and call for further studies of this phenomenon.

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Novel insights into the pathways regulating the canine hair cycle and their deregulation in alopecia X

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Alopecia X (AX) is a hair cycle (HC) arrest disorder commonly seen in Pomeranian dogs. Histologically, kenogen and telogen hair follicles (HFs) predominate while anagen follicles are sparse. The induction of anagen relies on the activation of HF stem cells (SCs) and their subsequent proliferation and differentiation; The function of SCs depends on finely-tuned interactions of signaling molecules and transcription factors; those regulating the canine HC are not known. We performed transcriptome profiling on skin biopsies to analyze the molecular pathways that are disrupted in AX. Biopsies from six affected and five healthy Pomeranians were studied. We showed that, in mice and dogs, signaling molecules and transcription factors involved in HC control are similar. Differential gene expression analyses revealed a downregulation of key regulator genes of the WNT (*β-catenin, LEF1, TCF3, WNT10b*) and SHH (*SHH*, *GLI1, SMO, PTCH2*) pathway, whereas genes of the BMP (*BMP4*, *SMAD2, SMAD7*) pathway were dysregulated. These observations parallel the situation in mice in which BMP signaling is important for SC quiescence and WNT signaling for SC activation. Furthermore, a significant downregulation of the SC markers *SOX9* and *LGR5* and an upregulation of *NFATc1,* a quiescence marker, were also observed. Finally, in AX, genes relevant for melatonin and estrogen metabolism were dysregulated, which is in agreement with the suspected but still unproven pathomechanism for this disease. In conclusion, our results provide novel and first insights into the molecular pathways involved in the canine HC and the pathogenesis of AX.

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Efficacy of an hydrocortisone aceponate spray in dogs with flea allergy dermatitis

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The objective of this study was to evaluate the efficacy of an hydrocortisone aceponate (HCA) spray (Cortavance, Virbac, Carros, France) in dogs with flea allergy dermatitis (FAD). Sixty shelter dogs were diagnosed with FAD based on clinical signs and the presence of fleas or their feces. Dogs were treated with either a saline (30 dogs) or the HCA spray at two pumps per 100cm2 of affected skin, once daily for 7 days; dogs also received a selamectin-containing pipette (Revolution, Zoetis, Kalamazoo, USA) on Day 0. Pruritus (0-3) and lesions (0-15) were scored on Day 0, 7 and 14. On Day 7, the primary outcome measure was clinical remission (CR; a pruritus score of 0). Scores were compared between treatments by one-way ANOVA. On Day 7, a CR was obtained in 100% of dogs with HCA and in none of those treated with saline ($P < 0.05$). On Day 7, the mean percentage reduction from baseline pruritus scores were 100% (HCA) and 2 % (saline). From Day 1 onward, pruritus scores were significantly lower after HCA compared to saline ($P < 0.05$). On Day 7, mean skin lesion score reductions were 72% (HCA) and 2% (saline); lesion scores improved further with HCA on Day 14 (86%); these percentages were significantly lower with HCA than saline from Day 2 onward ($P < 0.05$). Adverse effects were not seen with either spray. In conclusion, the Cortavance spray, along with an initial parasiticide, rapidly improves the resolution of FAD in dogs.

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