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Experimental model for peanut allergy by epicutaneous sensitization in atopic beagle dogs

Companion Animal Research Review Issue 2 with Nick Cave

Authors: Marsella, R.

Summary: This study in five atopic and five normal Beagles assessed whether they could be sensitised by 8 weeks of peanut paste applied epicutaneously and whether subsequent dermatitis flare-ups could be elicited by epicutaneous and oral challenges. Pruritic erythematous macules and papules developed in the area of paste application in all atopic dogs and two control dogs. Total clinical scores (Canine Atopic Dermatitis Extent and Severity Index Score) differed between groups, and an ANOVA indicated group ($p = 0.0139$), time ($p < 0.0001$) and group x time interactions ($p = 0.0067$). After oral

challenge, pruritic dermatitis developed in all atopic dogs within 15 min and peaked one hour after ingestion, while after epicutaneous challenge, macules and papules developed at the application site in all atopic dogs and in one control dog. Allergen-specific IgE was detected in the serum of all of the atopic dogs and two of the control dogs, and the number of IgE-positive cells in skin biopsies was significantly higher in atopic than control dogs ($p = 0.033$).

Comment: Images of Beagles smeared with peanut butter may be something you'd find during a dubious and rather niche internet search, but this study provides evidence to support one of the most interesting hypotheses of the aetiology of food allergic dermatitis in recent years. It has previously been assumed that sensitisation to food allergens occurs through the intestinal tract, and that the aberrant immunological event occurs within the intestinal mucosa. What is lacking in that assumption, is the ability to explain how a loss of mucosal tolerance to an ingested antigen can result in cutaneous food hypersensitivity, rather than clinical signs of gastrointestinal disease. What explains those cases where only the skin is affected? More recently, the now widely accepted hypothesis is that sensitisation to the food proteins actually occurs through the dermis itself. Sensitisation through the dermis has been demonstrated in mice, and can lead to local, and even systemic IgE production resulting in allergen-primed mast cells in the dermis and other tissues. Oral feeding of the allergen can then produce cutaneous signs, or even systemic anaphylaxis. A pre-requisite for this to occur is the lack of oral exposure prior to epicutaneous sensitisation, whereby the induction of oral tolerance lessens, or may even prevent dermal sensitisation. Likewise, small doses of food allergen applied to a disrupted or abnormally permeable dermis can prevent the development of normal oral tolerance when that food is subsequently ingested in mice. The study by Marsella et al. is the first to show the same response in dogs, and gives further support to the hypothesis that cutaneous food hypersensitivity is a cutaneous allergy, and not an oral allergy, even though clinical signs are elicited following ingestion. It may also explain the close clinical association between atopic dermatitis and cutaneous food hypersensitivity. And if cutaneous food hypersensitivity is due to cutaneous sensitisation, it opens the intriguing possibility that strategies employing the induction of oral tolerance, such as gradual oral reintroduction of the offending allergen, might be successful therapeutic approaches.

Reference: Exp Dermatol. 2015;24(9):711-2

http://www.animalhealthreview.co.nz/an/animal-health-area/Companion-Animal/Companion-Animal/Companion-Animal-Research-Review-Issue-2.aspx