Prevention of Atopic Dermatitis in Dogs: Is it Even Possible?

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1. Introduction

Canine atopic dermatitis (cAD) is a prevalent and complex skin condition in dogs, influenced by both genetic predisposition and environmental factors. Despite significant advancements in treatment options for affected dogs, research into disease prevention remains limited, particularly when compared to human atopic dermatitis.

Fernandes et al. (2023) provided a narrative review summarising existing studies and identifying potential areas for intervention.1 However, no definitive recommendations were proposed. This presentation builds upon that work, offering a broader, but definitely more speculative (though evidence-based) perspective to explore possible practical strategies for veterinarians to discuss with breeders and new dog owners. The goal is to mitigate, at least to some extent, the development of atopic dermatitis, particularly in susceptible breeds. Recommendations have been biased based on practicality and canine and / or human evidence.

2. Genetics

cAD appears to have a complex genetic basis that predisposes individuals to the development of clinical disease. This is evidenced by a strong breed predilection, with golden retrievers (GRs), West Highland White terriers, German Shepherds (GSDs), Cocker spaniels, Boxers, and French bulldogs being the most commonly reported breeds. However, the prevalence varies depending on the geographic location of the study, likely because of regional variance in the breed gene pool.[2]

Multiple single-locus genome-wide association studies and one Bayesian genome-wide association study have identified multiple candidate genes associated with canine atopic dermatitis (cAD), many linked to innate and adaptive immunity, inflammation, cell cycle, apoptosis, skin barrier formation and transcription regulation.[6] However, significant breed variability has been reported in candidate genes, strongly suggesting that the atopic genotype is breed-specific. This aligns with the distinct breed-associated phenotypic presentations of the disease.[2] Furthermore, evidence suggests that the probability of clinical expression of cAD increases with the number of expressed risk alleles.[3]

Despite this genetic complexity, and opinions that screening and breeding programmes to eliminate the condition are unlikely to succeed, [6] strong evidence indicates that selective breeding against cAD can at least reduce the prevalence of clinical disease.

Tengvall et al. (2022) demonstrated substantial genetic divergence in the risk of atopic dermatitis between gundog (black, yellow) and common (black, yellow, chocolate) Labrador Retrievers (LRs). The study also identified a divergence in risk between working (grey/black)

and show (black/tan) GSDs, attributed to distinct genetic selection signals in the two breeds (TBC1D1 in LRs and LRP1B in GSDs).[3]

A study on British LRs, GRs and their crosses estimated heritability at 0.47, indicating that nearly 50% of the risk of developing cAD in this population was genetic. Optimistically, the risk of cAD in offspring decreased to 11% when both sire and dam were classified as non-atopic.[4]

Recommendation: Breeding dogs should undergo thorough veterinary examination for early signs of atopic dermatitis (including otitis, non-infectious non-parasitic pruritus, cheilitis, pyoderma, and allergic conjunctivitis) and should not be used for breeding if signs of cAD are present. If breeding dogs develop cAD after reproduction, they should not be used for further breeding, and their offspring should not be selected to continue the breed line.

Limitations: These recommendations depend on several factors, including:

- *The accuracy of early cAD diagnosis in breeding candidates*
- *The expression of cAD at breeding age*
- *Breeder motivation to prioritize non-allergic dogs over other desirable traits*
- *If the expression of cAD risk genes in a given population was near 100% then it would be impossible to reduce the genetic risk without significant outbreeding*

3. Environmental Factors

Approximately 50% of the risk of developing both human atopic dermatitis (hAD) and canine atopic dermatitis (cAD) is attributed to environmental factors.[4] The 'hygiene hypothesis' suggests that early-life microbial exposure plays a critical role in immune system development, potentially promoting T-helper 1 (Th1) responses while downregulating T-helper 2 (Th2) responses. While extensive human research and epidemiological studies support this theory, its precise mechanisms remain incompletely understood.

Compared with hAD, research on environmental influences in cAD has been limited. It has been speculated that the increased use of antibiotics, vaccines, and anthelmintic drugs in dogs may have contributed to the rising prevalence of cAD.[5] Environmental influences, though, may vary by breed, and it has been reported that these factors do not affect the prevalence of atopic dermatitis (AD) in West Highland White terriers.[6]

While not all proposed risk factors are amenable to intervention, the following section outlines practical approaches available to both dog owners and breeders to potentially reduce cAD expression.

3.1 Bathing

Impairment of the skin barrier, as assessed via transepidermal water loss, has been positively associated with the development of hAD. Management guidelines for hAD have recommended minimising bathing frequency to reduce disruption of the skin barrier.

A study investigating bathing practices in infants revealed a nonlinear relationship between bathing frequency and the prevalence of hAD at three months. Infants bathed weekly or less

exhibited a 14.6% prevalence of AD, whereas those bathed more frequently showed a prevalence ranging from 23.2% to 26.9%. Adjusted logistic regression analysis indicated that bathing more often than weekly was significantly associated with hAD at three months.[7]

Similarly, in dogs, shampooing frequency may influence development of cAD. The previously mentioned study of LRs and GRs found that dogs bathed weekly were significantly more likely to develop cAD (odds ratio 21.47, P = 0.011) compared with dogs bathed less frequently or not at all.[5]

In humans, water hardness has also been identified as a potential risk factor in the initiation of early life skin inflammation. A positive association has been reported between living in regions with hard water (76 to > 350 mg/L CaCO3) and an increased prevalence of atopic eczema in children. Trials examining the use of water softeners in this setting have been proposed. However, there is no evidence that domestic water softeners improve objective disease severity in established AE.[8,9]

By contrast, in dogs with established cAD, shampoo treatment with ultrapure soft water (UPSW) significantly reduced pruritus and dermatitis scores, whereas shampooing with tap water did not yield similar benefits. Additionally, UPSW treatment, but not tap water treatment, led to a significant reduction in TEWL in affected dogs.[10]

Recommendation: In puppies and young dogs especially, bathing should be minimised to less than weekly, or ideally avoided completely where possible. It should only be performed where necessary for cleanliness or treatment of disease. Where bathing is performed, minimally irritant cleansers should be used (e.g. human non-soap cleansers such as Cetaphil Gentle Skin Cleanser or QV Gentle Wash), and thorough rinsing performed afterwards.

Recommendation: In hard water areas, soft water should be used for bathing dogs, particularly when young. This may be achieved by installation of a water softener, or use of distilled water.

Limitations: For those that may be concerned about the pH of infrequently used human cleansing products, note that if daily use of 50% white vinegar spray fails to make a significant impact on skin pH, [11] then an occasional bath in a pH balanced cleanser for humans (which is likely slightly more acidic than dog skin) is unlikely to make a significant impact on the dog's skin.

3.2 Antibiotic Usage

Meta-analyses of observational studies in humans suggest systemic antibiotic exposure during pregnancy or delivery, compared with no antibiotic exposure, lead to a moderate increase in the risk of the child developing hAD later in life. [9]

In children, systemic antibiotic use in the first year of life, as opposed to later, is associated with an increasing risk of hAD expression. This risk shows a dose response, and increases with increasing numbers of antibiotic courses, up to three or more. This risk is independent of the parent's history of atopic dermatitis, and one study showed that microbiome

alterations associated with both AD and systemic antibiotic usage fully mediated the increased risk.[12,13]

Recommendation: While antibiotic use has not been correlated with atopic dermatitis prevalence in dogs, the human data is compelling enough to recommend that where possible, antibiotic use should be avoided or minimised in pregnancy, and in the first year of life in dogs.

3.3 Prenatal omega-3 fatty acid supplementation

Early randomized controlled trials investigating prenatal supplementation with fish oil-derived omega-3 polyunsaturated fatty acids suggested that it may not prevent hAD in high-risk infants. However, variability in reported risk estimates did not definitively exclude the possibility of either a protective or harmful effect.[9]

A subsequent meta-analysis examined omega-3 fatty acid supplementation with reported daily intake of 900–3,071 mg (mean 1,860 mg) of combined eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) during pregnancy. This analysis also found no significant effect of fish oil supplementation during pregnancy on overall hAD incidence or IgE-associated eczema in children. In contrast, subgroup analysis indicated a significant reduction in specifically IgE-associated eczema among children younger than three years, suggesting benefits in some cases.[14]

Recommendation: 50-80mg/kg combined EPA and DHA (not α-lenolenic acid) daily from the third week of gestation to the end of lactation.

Limitations: While the evidence supporting use of prenatal omega-3 fatty acids as a preventative of cAD is absent, human evidence is supportive, supplementation is cheap, easy and low risk.

3.4 Avoidance of passive tobacco smoking

Studies in humans have shown that children exposed to tobacco smoke when very young have a higher risk of developing allergic diseases (including atopic eczema) than unexposed children. One study in adult dogs showed a significant increase in risk in cAD associated with high levels of exposure to tobacco smoke for at least 12 months, compared with non-exposed dogs. There appeared to be an exponential dose-response relationship between smoke exposure and prevalence of cAD.[15]

Recommendation: Smoking should be avoided around the dog if possible.

Limitations: This recommendation is based on very limited veterinary data from adult dogs, not pups.

3.5 Outdoor activity

A single study found that outdoor exposure on multiple occasions through the day, and at least an hour of sunlight exposure in pups 4-8 weeks of age significantly reduced the risk for later life otitis (and likely cAD) development.

Recommendation: Pups should get outdoor and sunlight exposure daily where possible from 4-8 weeks of age.

Limitations: This recommendation is derived from a single observational study and causality is not demonstrated, but is cheap, low risk and simple to implement.

3.6 Dietary manipulation: Non-processed meat based diets

Several observational studies, mainly from Finland and Sweden, support the role of home cooked (HC) diets (including human leftovers) and more specifically non-processed meat-based (NPMB) diets for both dam and pups in reducing the risk of clinical signs consistent with cAD. All the studies suggested that NPMB diets did not feed to be fed exclusively, and that most of the benefits were seen when the NPMB diet was 20-30% of the total food intake.[16,17,18] Evidence suggests the role of HC / NPMB diets seems at least as important in the dam as the pups, with one study reporting feeding puppies home-made diets including minced beef from 2 to 6 months was associated with a higher risk of cAD incidence, but only if the dam was not fed similar noncommercial products during lactation.[17,19] On the contrary, feeding more than 80% of the diet to pups 2-6 months of age as commercial dry food (CDF) was associated with an increased risk of clinical signs consistent with cAD.

The mechanism of how NPMB diets may impact development of cAD is unclear, though NPMB diets are reported to have more diverse and abundant microbial composition in the gut microbiota than dogs fed commercial feed,[20] and a small study of normal dogs and dogs with AD showed eight upregulated genes innate immune function, inflammation and antioxidants in the skin of dogs eating RMBD compared with CDF demonstrable effects on the skin.[21]

It is worth noting that in the studies, not all the NPMB diets were home cooked – some frozen commercial NPMB diets were used.[21]

Recommendation: The dam should be fed at least 30% of a nutritionally appropriate non-processed meat-based diet from 3 weeks of gestation to the end of lactation. Pups should be fed at least 30% of a nutritionally appropriate non-processed meat-based diet from starting solid food to 26 weeks of age. Nutritionally appropriate human leftovers should not be excluded.

Limitations:

- *most of the studies are observational, demonstrating associations but not necessarily causality*
- *the bulk of the studies arise from a single institution in Finland, by many of the same authors*
- *several studies are based owner-reported data via questionnaires and the inherent biases associated with this (recall bias, selection bias)*

- *the details of non-processed meat based diets were not detailed; nutritional quality may be an issue*
- *the risk of opportunistic infections may be higher in dogs fed NPMB diets*[20]
- *the ingredients of a home cooked diet (including a meat exclusion diet) can have a marked impact on the intestinal microbiome and this may impact outcomes*[22]
- *randomised controlled clinical trials from other centres are needed to confirm whether feeding a raw or partly raw diet during early life can minimise the risk for cAD, and whether some recipes are better than others*

3.7 Dietary Manipulation: Other

In an observational study of puppy feeding habits from two to six months of age, Hemida *et al* (2021) found several factors associated with a significantly increased risk of clinical signs consistent with cAD:

- Feeding of fruit
- Frequent dosing of omega 6 / omega 3 fatty acid supplements
- Feeding of dried animal parts
- Drinking from muddy puddles

Recommendation: Feeding of fruits, omega fatty acid supplements, and dried animal parts, as well as drinking from muddy puddles should be avoided in pups under 6 months of age.

Limitations: These recommendations are derived from a single observational study, and as always, causality is not established. They are however cheap, low risk and simple to implement.

4. Interventions that were considered but excluded

Several interventions were considered but subsequently excluded from definite recommendation.

Prospective use of Lokivetmab or oclacitinib as a preventative for AD. Rationale for exclusion: IL-31 knockout mice can still develop contact hypersensitivity[23], and in dogs, IL-31 inhibition is insufficient to prevent the expression of other proinflammatory mediators in acute AD.[24] Oclacitinib failed to prevent epicutaneous sensitisation in a colony of Beagle dogs genetically predisposed to cAD.[25]

Prospective use of administration dust mite extract or other arthropod or crustacean proteins to develop oral tolerance. Rationale for exclusion: In humans, while there is some cross reactivity between house dust mites, cricket, black soldier fly and shrimp allergens (particularly tropomyosins),[26-28] and foods based on all of the above except dust mites are commercially available in Australia, twice daily prospective use of house dust mite allergen oral immunotherapy for 12 months failed to prevent dust mite sensitisation or allergy symptoms in human infants <12 months of age.[29]

Location of puppies. Rationale for exclusion: Meury *et al* (2011) found with LRs, GRs and their crosses that puppies kept in a shed outside the breeder's house during puppyhood

developed cAD more often (odds ratio 19.6; P=0.006) than dogs kept indoors during the first months of life.[5] In contrast with this, Hemida *et al* (2023) reported that dogs that had been raised on a dirt floor (earth) or lawn had a 0.7-fold lower incidence of otitis later versus dogs using other floor types.[18] More prospective data is required before a recommendation can be made.

Reduction in frequency of gastrointestinal worming. Rational for exclusion: While one study in dogs with established cAD administered *Trichuris vulpis* eggs showed improvement in pruritus and Canine Atopic Dermatitis Extent and Severity Index scores, this failed to be reproduced in second placebo controlled study.[2] Zwicki et al (2018) reported total and Toxacara canis-specific IgE levels were higher in non-atopic compared to atopic dogs, and it was speculated that *T. canis* infection may have a protective effect against the development of canine atopic dermatitis.[30] However, Nødtvedt et al (2007) found there was no correlation between the number of times a puppy was treated with anthelminthics prior to leaving to breeder and subsequent development of cAD.[17] In contrast to this, Hemida *et al* (2023) reported puppies treated with gastrointestinal anthelminthics 2-10 times in the first twelve months of life were reported as being significantly more at risk of later development of otitis compared with puppies treated less than twice in the same time period.[18] Because of the pathogenic potential of worms in young dogs, more definite data is required before a recommendation can be made regarding the appropriate dosing of anthelminthics with respect to cAD risk.

Use of emollients prospectively in pups. Rationale for exclusion: Early small-scale studies supported the use of emollients for hAD prevention; however, subsequent reviews and larger trials, primarily conducted among high-risk infants, did not confirm this benefit. Notably, the PreventADALL trial, which combined emollient application with frequent bathing (4–7 days per week), suggested a potential increased risk of hAD. Conversely, more recent smaller trials investigating ceramide-containing emollients, when initiated within the first days of life in high-risk infants, indicated a tendency toward reduced hAD risk. [9] While adopting this protocol using human ceramide-based emollients in dogs may hold theoretical promise, practical limitations, including the high compliance demands on owners and the challenges posed by increasingly dense hair, make this an impractical option.

A limited number of studies have reported mild to moderate benefits of emollients, both human and veterinary formulations, including some containing ceramides, in the treatment of established cAD.[31-36] Their role in preventing cAD has not been documented.

Routine application of **Allerderm Spot-On** (Virbac), which contains ceramides, cholesterol, and fatty acids, could theoretically align with the more promising human studies. However, its availability in the Australian market is limited. **Dermoscent Essential 6** (Blackmores), a topical supplement which is available in Australia, contains essential oils and unsaturated fatty acids but lacks ceramides. While it has demonstrated some benefits in established cAD,[35,36] its potential as a preventive measure remains untested. If **Dermoscent Essential 6** were to be considered for preventive use though, weekly application from one week of age in high-risk breeds would be the theoretical recommendation. However, even within a speculative framework, the absence of supporting evidence for its preventive efficacy, as

well as the critical role of formulation of emollients for the treatment of hAD, precludes a formal recommendation.

Adoption age less than eight weeks. Rationale for exclusion: Meury et al (2011) found in LR, GR and their crosses that dogs adopted between the ages of 8 and 12 weeks developed cAD more often (odds ratio 4.89; P = 0.011) than dogs adopted at less than eight weeks of age.[5] However, puppies adopted at less than four to eight weeks of age have been reported to subsequently show more frequent signs of fear, anxiety, and attachment and attention-seeking tendencies compared to those adopted at a later age.[37] As such early adoption cannot be recommended as a preventative strategy for cAD.

Prenatal, postnatal and puppy probiotics. Rationale for exclusion: There is no doubt that there is a link between intestinal microbiota and atopic dermatitis in dogs – several studies have identified intestinal dysbiosis and reduction in microbial diversity in atopic dogs compared with normal control dogs. Furthermore, probiotics have shown some positive clinical outcomes (though inconsistent) in established cAD, with better outcomes potentially associated with improving diversity of the intestinal flora. Interestingly, there are conflicting results on the impact of controlling cAD with oclacitinib on improvements in the intestinal microbiota diversity with one study each showing either no, or significant improvement in diversity of intestinal microbiota.[38-40] Lastly, use of daily probiotics in Beagles genetically predisposed to cAD showed reduced IgE at 6 months of age, in addition to measurable immunological effects 3 years after cessation of the probiotics at 6 months of age. There was no clinical difference on cAD outcomes though between the control and treatment groups.[41,42]

In 2015, the World Allergy Organization, in a review of the literature to that date, concluded that probiotics, when dosed with single strains (typically *Lacticaseibacillus* (prev *Lactobacillus) paracasei* or *L. rhamnosus,* or *Bifidobacteria longum*, *animalis*, or *lactis*) and administered to mothers prenatally (usually in the final trimester), reduced the risk of infant hAD. It made a conditional recommendation, based on overall very low certainty, for using probiotics directly for infants at risk of developing AD in addition to supplementing probiotics to both pregnant women and breastfeeding mothers of high-risk infants. Subsequent to that, a further nine systematic reviews and meta-analyses have been performed, and while most of those supported the use of probiotics to reduce the risk of hAD in generally high risk infants, there was no consensus to achieve best outcome on dose, timing of commencement of treatment, duration of treatment, ideal strain or strains to dose with, and whether to dose the mother, infant or both.[43]

Although these findings highlight the likely association between the gut microbiota and AD, the specific relationship among probiotics, gut microbiota, and AD remains unclear. A better understanding of this relationship is likely going to be needed to more precisely manipulate gut microbiota to attenuate clinical manifestation of AD in humans and dogs.[9] This is compounded by the significant impact of diet on the intestinal microbiota, even in the absence of probiotics.[20, 22]

As such, a formal recommendation for dosing of probiotics to prevent cAD cannot be made. If probiotics are desired to be used regardless, then a logical dosing strategy would be to

dose the dam from week three of gestation to the end of lactation, and the pups from the point of starting solid food to 6 months of age. An appropriate dose would be >1x10^9 colony forming units (CFU) daily for the pups and >2x10^9 (CFU) for the dam, using a mixture of *Bifidobacterium bifidum, Lactobacillus acidophilus, Enterococcus faecium*, *Latilactobacillus* (*Lactobacillus) sakei probio-65, Lacticaseibacillus (Lactobacillus) paracasei K71* and/or *Lacticaseibacillus (Lactobacillus) rhamnosus strain GG*.

5. Conclusion

A good understanding of successful prevention of cAD is in its infancy, and there are multiple potentially useful strategies that need to be prospectively explored to confirm their usefulness. Until then, these practical strategies remain speculative.

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