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# Treatment of canine atopic dermatitis: 2010 clinical practice guidelines from the International Task Force on Canine Atopic Dermatitis

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In 2009, the International Task Force on Canine Atopic Dermatitis was composed, in alphabetical order, of Emmanuel Bensignor (F), Didier Carlotti (F), Douglas J DeBoer (USA), Claude Favrot (CH), Craig Griffin (USA), Richard Halliwell (Chair; UK), Bruce Hammerberg (USA), Peter Hill (AUS), Toshiroh Iwasaki (J), Hilary Jackson (UK), Sadatoshi Maeda (J), Kenichi Masuda (J), Rosanna Marsella (USA), Ralf Mueller (D), Tim Nuttall (UK), Thierry Olivry (USA), Pascal Prélaud (F), Candace Sousa (USA) and Ton Willemse (NL).

**Conflict of Interest**

Relevant to the interventions described in this review and in the last decade, the authors report having consulted for, and/or received private funding as follows:
Thierry Olivry: Novartis Animal Health Global (Basel, Switzerland) and Virbac (Carros, France).
Douglas DeBoer: Heska Corporation (Fort Collins, CO, USA) and Greer Laboratories (Lenoir, NC, USA).
Claude Favrot: Novartis Animal Health Global (Basel, Switzerland).
Hilary Jackson: Nestlé Purina Petcare (St Louis, MO, USA) and Novartis Animal Health Global (Basel, Switzerland).
Ralf Mueller: Bayer Animal Health (Leverkusen, Germany), Boehringer Ingelheim (Copenhagen, Denmark), TEVA Animal Health-DVM Pharmaceuticals (St Joseph, MO, USA), Procter & Gamble Pet Care (Cincinnati, OH, USA), Laboratoire de Dermo-Cosmétique Animale (Castres, France), Novartis Animal Health (Greensborough, NC, USA), Pfizer Animal Health (Karlsruhe, Germany) and Virbac (Carros, France).
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None reported.

## Abstract

**Atopic dermatitis (AD) is a common chronic relapsing pruritic skin disease of dogs for which treatment has varied over time and geographical location. Recent high quality randomized controlled trials and systematic reviews have established which drugs are likely to offer consistent benefit. The International Task Force for Canine AD currently recommends a multifaceted approach to treat dogs with AD. Acute flares should be treated with a combination of nonirritating baths and topical glucocorticoids, once an attempt has been made to identify and remove the suspected causes of the flare. Oral glucocorticoids and antimicrobial therapy must be added when needed. In dogs with chronic AD, a combination of interventions should be considered. Again, factors that trigger flares of AD must be identified and, if possible, avoided. Currently recognized flare factors include food, flea and environmental allergens, *Staphylococcus* bacteria and *Malassezia* yeast. Skin and coat hygiene and care must be improved by bathing with nonirritating shampoos and dietary supplementation with essential fatty acids. The severity of pruritus and skin lesions can be reduced with a combination of anti-inflammatory drugs. Currently, medications with good evidence of high efficacy include topical and oral glucocorticoids, and calcineurin inhibitors such as oral ciclosporin and topical tacrolimus. The dose and frequency of administration of these drugs should be tailored to each patient considering each drug's efficacy, adverse effects and cost. Allergen-specific immunotherapy should be offered, whenever feasible, in an attempt to prevent recurrence of clinical signs upon further exposure to environmental allergens to which the patient is hypersensitive.**

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## Preamble

Throughout this article, recommendations for specific interventions were made using the two parameters described in Table 1.[1] Categories of evidence (COE) were assessed based on the highest evidence available at the

**Table 1.** Categories of evidence and strengths of recommendation

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| **Category of evidence** |
| **Ia** Evidence from meta-analysis or systematic reviews |
| **Ib** Evidence from at least one randomized controlled trial |
| **IIa** Evidence from at least one controlled study without randomization |
| **IIb** Evidence from at least one other type of quasi-experimental study |
| **III** Evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies |
| **IV** Evidence from expert committee reports or opinions or clinical experience of respected authorities or both |
| **LB** Evidence from laboratory-based studies |
| **Strength of recommendations** |
| **A** Directly based on category I evidence |
| **B** Directly based on category II evidence or extrapolated from category I evidence |
| **C** Directly based on category III evidence or extrapolated from category II evidence |
| **D** Directly based on category IV evidence or extrapolated from category III evidence |
| **E** Directly based on category LB evidence |
| **F** Based on consensus from Specialty Task Forces |

Modified from: Leung DYM *et al.* Ann Allergy, Asthma, Immunol 2004; 93:S1–21.

time of writing, while strength of recommendations (SOR) were derived from the highest COE for the relevant interventions. In general, COE of lower Roman numeral and SOR of lower alphabetic order should be considered of greater value than those with higher grades.

For most of the recommendations given in this article, the evidence was derived from results of clinical trials that reported statistically significant reductions in specific outcome measures. Practitioners must consider that such statistically significant reductions do not imply that the use of these interventions will always result in a response that is acceptable to the owner, or that these interventions will always be effective in every single patient. Furthermore, while reading these guidelines, veterinarians are advised to keep in mind that it is often necessary to combine several interventions in order to achieve a satisfactory outcome.

Another important point to address is that, in several sections of this document, there are statements highlighting the lack of evidence supporting the use of a particular drug or product. Insufficient evidence for recommending an intervention does not mean that the drug or product is not effective, but simply that there are no studies documenting their efficacy or lack thereof.

In this review, the recommendation for a specific drug does not imply direct support or endorsement of a particular product or company by this Task Force, but it signifies that there is a study or consensus supporting the use of this intervention. Importantly, recommendations for an intervention did not take into consideration whether a product was available in a specific country, or whether it was licensed for use in dogs – or dogs with atopic dermatitis (AD) – in that country. Before implementing these guidelines into their practice, veterinarians should first verify the legality of using the various products in their respective countries.

As far as drug designation is concerned, this text will usually provide recommendations for interventions using generic drug names. However, if the recommendation is based on specific studies that provide evidence of efficacy of a particular product, then we will refer to specific brand and company names. Finally, the reader who desires a summary of our recommendations is referred to the single-page addendum at the end of this article.

## Introduction

Atopic dermatitis is a common dermatosis of dogs defined as a genetically-predisposed inflammatory and pruritic skin disease with characteristic clinical features that is associated with IgE antibodies, most commonly directed against environmental allergens.[2] We now recognize a parallel condition termed 'atopic-like dermatitis' (ALD) that must be differentiated from AD. Patients with ALD have the same constellation of clinical signs as those with AD, but in ALD, an IgE response to environmental or other allergens cannot be documented by usual methods (Box 1).[2]

*Pathogenesis.* The pathogenesis of canine AD is not fully understood. Whereas the traditional dogma stressed the importance of IgE-mediated early and late-phase hypersensitivity reactions to airborne allergens, evidence is now mounting to suggest that epidermal barrier defects might also contribute to disease pathogenesis.[3] The current theory on the pathogenesis of canine AD can be summarized as follows.[3,4] In the acute phase of the disease, putative epidermal barrier defects are thought to facilitate contact of environmental (and possibly microbial) allergens with epidermal immune cells. Epidermal antigen-presenting cells capture allergens with allergen-specific IgE, and then migrate to the dermis and regional lymph nodes. Microbial products and immune cell-derived inflammatory mediators activate keratinocytes, which, in turn, release more chemokines and cytokines. Immunoglobulin E-coated dermal mast cells release histamine, proteases, chemokines and cytokines following contact with allergens. There is an early influx of granulocytes (neutrophils and eosinophils), allergen-specific T-lymphocytes and dermal dendritic cells. Eosinophils degranulate and release proteins that induce dermal and epidermal damage. Type-2 helper T lymphocytes release cytokines promoting IgE synthesis and eosinophil survival. Microbes, self-trauma and neuromediators might also contribute to persistent inflammation in chronic skin lesions. There is a continuous cycle of chemokine release

**Box 1.** International Task Force on Canine Atopic Dermatitis disease definition

*Canine atopic dermatitis:* A genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features associated with IgE antibodies most commonly directed against environmental allergens.

*Canine atopic-like dermatitis:* An inflammatory and pruritic skin disease with clinical features identical to those seen in canine atopic dermatitis in which an IgE response to environmental or other allergens cannot be documented.

Halliwell R. Revised nomenclature for veterinary allergy. Veterinary Immunology and Immunopathology 2006; 114: 207–8.

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that leads to the influx and activation of leucocytes and the release of additional pro-inflammatory mediators. The failure to down-regulate pro-inflammatory mechanisms is followed by self-perpetuating cutaneous inflammation. Despite these advances in our knowledge of the pathogenesis of canine AD, the mediators that elicit the sensation of pruritus have not been elucidated. Importantly, histamine does not appear to provoke pruritus in dogs in contrast to humans and mice.[5]

*Clinical Signs and Diagnosis.* It is not within the scope of this document to provide a detailed discussion of the many facets (and controversies) in the diagnosis of AD. However, the authors wish to summarize our current understanding, and emphasize several critical elements, which we believe are most frequently misunderstood.

*The diagnosis of canine AD relies primarily on the patient's signalment, clinical signs and disease history, and not on a laboratory test*.[6,7] Atopic dermatitis is a diagnosis based on the finding of a constellation of typical history and clinical signs with the subsequent elimination of other conditions that might mimic it. Most atopic dogs usually begin manifesting signs between 6 months and 3 years of age. There is no known gender predisposition. In general, dogs have a history of pruritus with or without recurrent skin or ear infections. A history of lacrimation, ocular congestion or sneezing/rhinorrhea could be indicative of concurrent atopic conjunctivitis and rhinitis respectively. Signs might be seasonal or nonseasonal with or without seasonal exacerbation, depending principally upon the allergens involved as flare factors and the pet's environment.

Primary skin lesions usually consist of erythematous macules, patches and small papules. Most patients, however, present with lesions that occur secondary to self-trauma, for example excoriations, self-induced alopecia, lichenification and hyperpigmentation. The distribution of canine AD skin lesions is variable and likely depends upon the chronicity of the disease and allergens involved. Body areas that commonly exhibit lesions are the face, concave ear pinnae, ventral neck, axillae, groin, abdomen, perineum, ventral tail, as well as flexural and medial aspects of extremities. The dorsal and ventral paws are often involved and otitis externa is also commonly seen. Periocular and perinasal lesions might reflect co-existing pruritic atopic conjunctivitis and rhinitis respectively.

It is critically important to recognize that other dermatoses can mimic AD, or be superimposed on it. These diseases are usually of parasitic (especially scabies, and, occasionally demodicosis), infectious (e.g. *Staphylococcus* superficial pyodermas, *Malassezia* dermatitis) or of other allergic origin. Such diseases must be ruled-out or controlled before the diagnosis of AD is made. The principles of diagnosis and treatment of these clinically similar conditions are beyond the scope of these guidelines; practitioners should refer to recent reviews for best practice recommendations in their respective countries.

A set of criteria – a type of 'checklist' – has been recently recommended as an aid for diagnosing AD in dogs (Table 2).[8] One should remember, however, that these criteria are not absolute; approximately one of five cases (20%) could be misdiagnosed if these parameters were to be applied strictly! However, with the proper rule-out of ectoparasitoses and skin infections, the specificity of these criteria is expected to increase markedly. Finally, it is important to keep in mind that, in early stages of AD, lesions are unlikely to be seen at all characteristic sites, and pruritus might be present without observable lesions.

**Table 2.** Favrot's 2010 criteria for canine atopic dermatitis

1. Onset of signs under 3 years of age
2. Dog living mostly indoors
3. Glucocorticoid-responsive pruritus
4. Pruritus sine materia at onset (i.e. alesional pruritus)
5. Affected front feet
6. Affected ear pinnae
7. Nonaffected ear margins
8. Nonaffected dorso-lumbar area

A combination of five satisfied criteria has a sensitivity of 85% and a specificity of 79% to differentiate dogs with AD from dogs with chronic or recurrent pruritus without AD. Adding a sixth fulfilled parameter increases the specificity to 89% but decreases the sensitivity to 58%.

Source: Favrot C, Steffan J, Seewald W *et al.* A prospective study on the clinical features of chronic canine atopic dermatitis and its diagnosis. Veterinary Dermatology 2010; 21: 23–30.

The relationship between canine AD and cutaneous adverse food reactions (CAFR or 'food allergies') has long been the subject of controversy. Recently, this Task Force supported the concept that CAFR might manifest as AD in some canine patients, or, in other words, that food components might trigger flares of AD in dogs hypersensitive to such allergens (Box 2).[9] It should be noted that, in addition to clinical signs typical of AD, CAFR could also manifest as other syndromes, such as urticaria or pruritus without lesions or with lesions at unusual sites (e.g. flanks, dorsum, perineum, around the lips).

There is consensus that the use of allergen-specific IgE serological or intradermal tests cannot be used for the initial diagnosis of AD in dogs.[10,11] Many normal and atopic dogs exhibit positive reactions with either test, thereby markedly decreasing the tests' specificity for the diagnosis of AD. Using a serologic test or intradermal test as a primary criterion for diagnosing AD will, therefore, lead to misdiagnosis. However, such tests can be used for the following reasons: (i) to document whether or not the disease is associated with allergen-specific IgE (i.e. determining whether the dog suffers from AD or ALD), (ii) to implement allergen-avoidance interventions (e.g. house dust mite elimination measures), and/or (iii) to select allergens to be included in immunotherapy preparations. These interventions will be discussed below in greater detail.

The major objective of these practice guidelines is to improve the care of dogs with AD. The recommendations were made by a committee of the International Task Force for Canine AD for the benefit of general practitioners. This article is divided into two distinct sections: (i) the management of acute flares of AD, and (ii) the treatment of chronic skin lesions of AD. Case scenarios are provided as examples of situations that can occur in practice and that would benefit from the interventions recommended in these sections. Treatment options are

**Box 2.** Excerpts from the International Task Force on Canine Atopic Dermatitis position on the relationship between food allergy and atopic dermatitis in dogs

*Food allergy* (also known as *adverse food reaction*) is an **aetiological diagnosis**. In dogs, cutaneous clinical manifestations of food allergies have been reported as focal, multifocal or generalized pruritus, otitis, seborrhoea, superficial pyoderma and also, in some dogs, as atopic dermatitis. These cutaneous manifestations can often be accompanied with digestive signs.

*Atopic dermatitis*, in dogs and humans, is a **clinical diagnosis**. It can be exacerbated by an exposure to allergens, which can be of environmental (e.g. mites, pollens), microbial and also, in some dogs, dietary origin.

*Position statement:* the International Task Force on Canine Atopic Dermatitis supports the concept that cutaneous adverse food reactions (food allergies) might manifest as atopic dermatitis in some canine patients, or, in other words, that food components might trigger flares of atopic dermatitis in dogs hypersensitive to such allergens.

*Implications for clinical practice:* Food allergies can manifest clinically, in some dogs, as atopic dermatitis, but not every dog with food allergy will manifest it as atopic dermatitis. Atopic dermatitis can be exacerbated by food allergens, but not every dog with atopic dermatitis will have dietary-induced flares. Every dog diagnosed with nonseasonal (i.e. perennial) atopic dermatitis should undergo one or more dietary restriction-provocation challenges (i.e. 'elimination diets') to determine, and then eliminate, any dietary allergens that might cause flares of the disease.

Olivry T, DeBoer DJ, Bensignor E, Prélaud P for the International Task Force on Canine Atopic Dermatitis. Food for thought: pondering the relationship between canine atopic dermatitis and cutaneous adverse food reactions. Veterinary Dermatology 2007; 18: 390–1.

listed in a particular order, but this does not mean that all recommendations are advised or needed in that specific sequence. First and foremost, practitioners shall examine the validity of these recommendations in the context of their patient, the pet owners and the availability and cost of the products in their respective countries. Again, veterinarians must remember that it is often necessary to combine several interventions in order to achieve a satisfactory outcome.

## Treatment of acute flares of AD

*Case Scenario 1. A 3 year old male castrated English bull terrier has suffered from intermittent pruritic skin lesions diagnosed as AD for the last 2 years. It is now the peak of the season for grass pollens to which he is hypersensitive; clinical signs have begun to reappear.*

*Case scenario 1a (mild acute flares). The dog presents with erythematous and oedematous patches and excoriations of the axillae (Figure 1); he occasionally scratches his face and licks his feet.*

*Case scenario 1b (moderate to severe acute flares). The dog is affected with multifocal patches of oedema, erythema, papules and excoriations on the axillae, groin and medial thighs (Figure 2). He scratches nearly nonstop all over the body.*

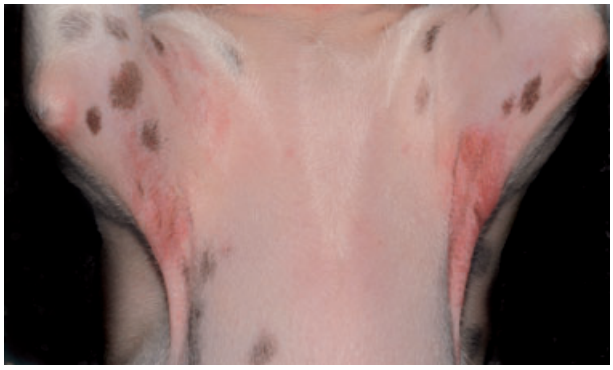

**Figure 1.** Localized acute flare of canine atopic dermatitis. This dog exhibits patches of erythema and oedema with excorations on both axillae (example of case scenario 1a).

### Identification and avoidance of flare factors

*Identification and removal of allergenic causes of flares. strength of recommendation SOR D (case scenarios 1a and 1b)*

*Rationale for such recommendation.* When an exacerbation of signs occurs in a dog that previously had a disease in complete or near complete remission, veterinarians must look for, and eliminate if at all feasible, the cause of such flares. Currently recognized sources of flares of canine AD include fleas, food and environmental (e.g. house dust mites, pollens) allergens. In case of acute AD exacerbation, especially in areas where flea infestation is endemic, practitioners must first verify if fleas could have contributed to the current worsening of signs. Similarly, owners should be queried about the ingestion of dietary items to which the dog is known to be hypersensitive. Finally, consultation of online pollen counts could help document whether offending pollens are currently airborne in the local geographic area. Readers are referred to sections below for further details on flea control and dietary restriction measures.

*Evaluation of use of antimicrobial therapy: SOR D (case scenarios 1a and 1b)*

*Rationale for such recommendation.* Skin and ear infections are common reasons why lesions and pruritus acutely worsen in dogs with AD. If bacterial or yeast infections are identified with some combination of clinical signs, cytology and/or culture, antimicrobial therapy is indicated, normally using a combination of topical with or without oral medications (COE IV). For skin infections, shampoos or solutions containing antibacterial (e.g. chlorhexidine, ethyl lactate, triclosan) and/or antifungal (e.g. miconazole, ketoconazole) medications are beneficial. Because of their drying and irritating effects, benzoyl peroxide-containing formulations are not recommended in dogs with AD without a subsequent topical moisturizer (COE IV). If lesions of bacterial or fungal skin infections are localized, ointments, creams, gels or wipes containing antiseptics (e.g. chlorhexidine), antibiotics (e.g. mupirocin, fusidic

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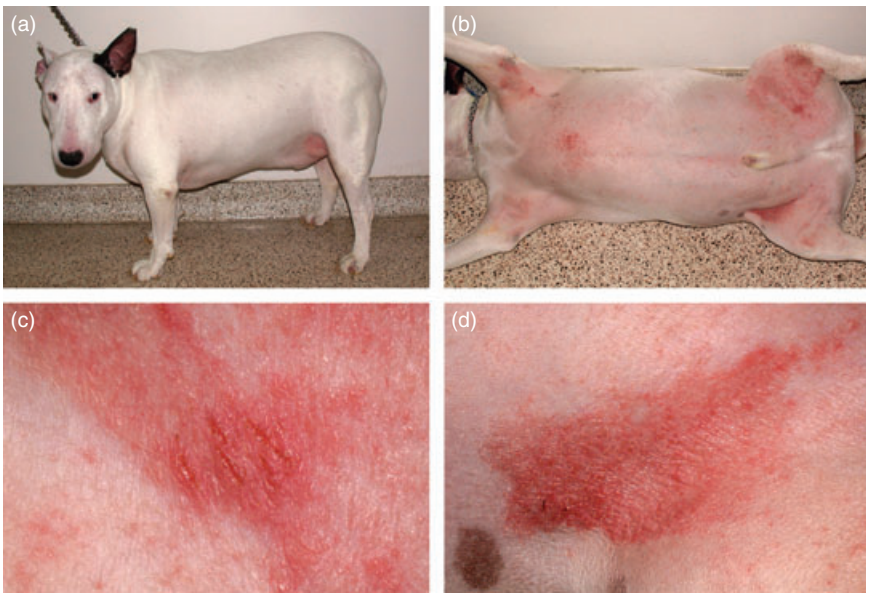


**Figure 2.** Multifocal to generalized acute flare of canine atopic dermatitis. This dog presents with an acute exacerbation of previous signs of AD. Erythema, oedema, excoriations and papules can be seen on the axillae (b,c), sternum (b), inguinal regions (a,b,d) and medial thighs (example of case scenario 1b).

acid, clindamycin or others) or antifungal drugs (e.g. miconazole, clotrimazole, ketoconazole, terbinafine) are indicated (COE IV). Pet owners should be advised to monitor for signs of worsening of pruritus and skin lesions following topical antiseptic formulations; if this were to happen, a bacterial culture and sensitivity and/or an alternative product should be considered. If lesions of infection are widespread or severe, then systemic antibiotics or antifungal drugs are normally needed (COE IV). Veterinarians should refer to best practice recommendations for oral antimicrobial drug usage in their respective countries (see 'treatment options for chronic canine AD' below for further recommendations on the responsible use of topical and systemic antibiotics).

### Improvement of skin and coat hygiene and care

*Bathing with a nonirritating shampoo: SOR B (case scenarios 1a and 1b)*

*Rationale for such recommendation.* A small double-blinded randomized controlled trial (RCT) showed that a weekly bath with a 10 min application of a shampoo containing lipids, complex sugars and antiseptics (Allermyl, Virbac, Carros, France) led to a halving of pruritus scores within 24 h in 25% of treated dogs (COE Ib).[12] When this shampoo was used in a whirlpool, the antipruritic effect was more pronounced. Interestingly, the use of the whirlpool without shampooing had a similar antipruritic benefit in one of five dogs.[12]

There is currently no evidence of any benefit from using other shampoos or conditioners containing ingredients such as oatmeal, pramoxine, antihistamine, lipids or glucocorticoids (COE IV). Taken as a whole, these findings suggest that the benefit from bathing might lie primarily in the action of washing the pet.

### Reduction of pruritus and skin lesions with pharmacological agents

*Short-term treatment with a topical glucocorticoid: SOR A (case scenarios 1a and 1b)*

*Rationale for such recommendation.* Three RCTs[13–15] and a systematic review[16] provide evidence for the high efficacy of medium potency glucocorticoid sprays [i.e. 0.015% triamcinolone solution (Genesis spray, Virbac, Ft Worth, TX, USA); or 0.0584% hydrocortisone aceponate (Cortavance spray, Virbac, Carros, France)] for reduction of skin lesions and pruritus in canine AD (COE Ia). Such intervention is especially suitable for localized skin lesions[14] and for short durations (COE Ib). Clinicians must tailor the frequency and duration of application to the severity of clinical signs (COE Ib).[15] In the absence of availability of the formulations described above, other topical glucocorticoid formulations are likely to be beneficial, but the efficacy and safety of these medications will vary with the type of glucocorticoid and vehicle used (COE IV). Clinicians should note that these treatments are intended for use only over a limited period; caution is advised with long-term use, as adverse effects are likely to occur. These usually include skin thinning with or without tearing, comedones and superficial follicular cysts (milia).[17,18]

*Short course of oral glucocorticoids: SOR A (case scenario 1b)*

*Rationale for such recommendation.* If signs are too severe or extensive to be controlled with topical formulations, then oral glucocorticoids could be needed. A systematic review of published RCTs suggests that the oral glucocorticoids prednisone, prednisolone or methylprednisolone are beneficial given at 0.5 mg/kg once to

twice daily until clinical remission occurs (COE Ia).[16] If clinical signs are very severe or do not improve rapidly, it might be necessary to maintain some dogs on longer courses at the lowest dose and frequency of administration that controls their clinical signs. The use of oral glucocorticoids is normally contra-indicated in case of widespread concurrent superficial or deep bacterial skin infections (COE IV). Side effects of oral glucocorticoids are usually proportional to drug potency, dosage and duration of administration. Treatment of acute flares of canine AD with long-acting injectable glucocorticoids is not recommended (SOR D). Because most dogs with AD have signs that respond to oral glucocorticoids, failure of rapid clinical benefit with this category of drug should prompt the clinician to reconsider alternative diagnoses or the presence of secondary complications (for example, skin infections, ectoparasitism or other nonatopic food reactions).

*Interventions likely to be of little or no benefit to treat acute flares of canine AD*

*Antihistamines.* Because of their mode of action, type-1 histamine receptor antagonists/inverse agonists (i.e. common 'anti-allergic' antihistamines such as hydroxyzine, diphenhydramine and chlorpheniramine) are unlikely to be beneficial 'after the fact' to treat acute flares of canine AD. Indeed, these drugs would not have had the time to block histamine receptors before their occupation by histamine released in early allergic reactions. When examined as a group, there is no conclusive evidence of efficacy of oral type-1 antihistamines for treatment of active AD in dogs (COE Ia).[16] Whether or not antihistamines would be beneficial in dogs with mild AD signs or to prevent the recurrence of flares has not been determined.

*Essential Fatty Acid Supplements.* As their mode of action requires their incorporation into cell membranes, a phenomenon that necessitates several weeks of treatment, essential fatty acids (EFA) are unlikely to be of any benefit for acute flares of AD in dogs (COE Ia).[16]

*Tacrolimus and Ciclosporin.* Similarly, even though the twice daily application of 0.1% tacrolimus ointment (Protopic, Astellas Pharma, Tokyo, Japan) has been shown to be of benefit in reducing skin lesions and pruritus in localized AD in dogs (COE Ib),[19] the slow onset of treatment effect and mild irritation observed make this intervention poorly suitable for treating acute flares of AD. As discussed in greater details in the next section, because of its delay in treatment effect, ciclosporin is unlikely to offer any benefit for treatment of acute flares of canine AD.

## Treatment Options for Chronic Canine AD

*Case Scenario 2. A 6 year old male castrated West Highland white terrier dog has been affected with atopic skin lesions and pruritus since 18 months of age. Signs are perennial and consist of erythema, lichenification, hyperpigmentation and self-induced alopecia.*
*Case scenario 2a (localized chronic AD). lesions are restricted to the front feet (Figure 3), and he chews these areas often.*
*Case scenario 2b (generalized, moderate-to-severe chronic AD). lesions are present on the face (periocular, perioral, concave pinnae), ventral neck, axillae, flanks, abdomen and feet (Figure 4). He scratches or chews these areas almost constantly.*

### Identification and avoidance of flare factors

*Performance of dietary restriction-provocation trials in dogs with perennial (nonseasonal) AD: SOR D (case scenarios 2a and 2b)*

*Rationale for such recommendation.* Food allergens can cause flares of clinical signs of AD in dogs hypersensitive to such allergens. Such patients are likely to exhibit chronic recurrent year-round clinical signs. As a result, one or more restriction-provocation dietary trials (e.g. 'elimina-

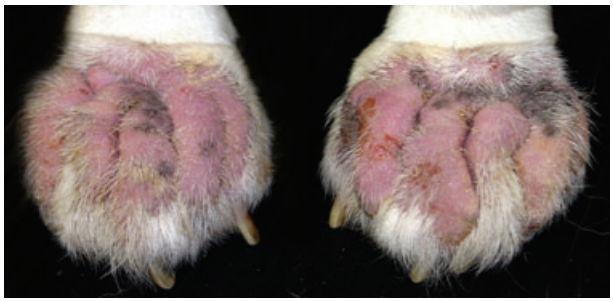

**Figure 3.** Localized chronic canine atopic dermatitis. Self-induced alopecia, oedema, erythema, excoriations and increased exudation can be seen on the dorsal metacarpi and digits (example of case scenario 2a). Courtesy of Dr Candace Sousa.

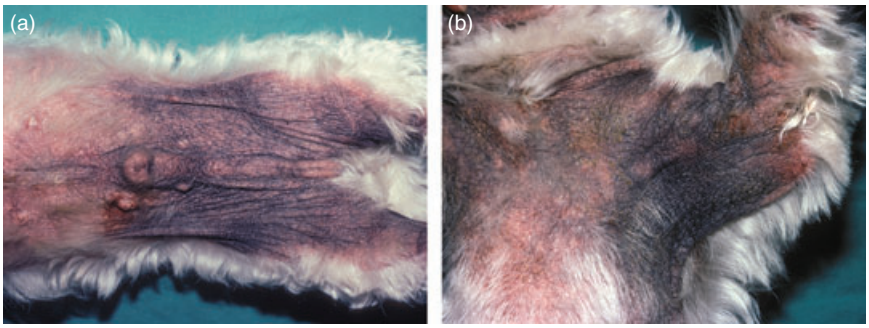


**Figure 4.** Generalized chronic canine atopic dermatitis. This dog presents with severe chronic lesions consisting of self-induced alopecia, erythema, lichenification, hyperpigmentation and scaling on the abdomen, medial thighs, groin (a), ventral neck, axillae and sternum (b) (example of case scenario 2b).

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tion diets') must be performed in all dogs with perennial (nonseasonal) AD to determine whether food allergens contribute to clinical signs in these patients (COE IV).[9] Before implementing long-term anti-inflammatory or antipruritic drug in dogs with nonseasonal AD, practitioners must remember that treatment is unlikely to be successful if a dog hypersensitive to dietary components regularly ingests offending allergens. As a result restriction-provocation dietary trials should always be considered in dogs with nonseasonal AD. Even if an attempt at controlling diets was made early in the course of the disease, this aspect might have to be reconsidered in case of disease flares, especially if anti-inflammatory therapy is not or no longer effective. Indeed, atopic dogs often acquire new hypersensitivities, and the development of a novel food allergy could be the cause of AD exacerbation.

Normally, dietary trials should be carried out for 6–10 weeks using either commercial or homemade diets employing a low number of novel or hydrolysed ingredients (COE III). At this time, however, there is no clear evidence of a superior benefit of hydrolysate-based compared to nonhydrolysed commercial diets,[20] or of homemade over commercial diets. In theory, the main value of performing trials with homemade diets is if hypersensitivity to a minor component of a commercial diet (additive, colourant, preservative, etc.) is suspected. It must be emphasized, however, that cutaneous hypersensitivity to additives has not yet been reported in dogs.

*Implementation of an effective flea control regimen: SOR D (case scenarios 2a and 2b)*

*Rationale for such recommendation.* There is evidence that the atopic status predisposes dogs to develop hypersensitivity to flea salivary antigens if exposed repeatedly to flea bites (COE IV).[21] As a result, where flea infestation is endemic, all dogs with AD should be treated with year-round flea adulticides combined with relevant environmental measures. Veterinarians should refer to standard-of-care flea control protocols in their respective countries and geographic regions. Practitioners should also bear in mind that the efficacy of topical flea control products is often limited by the frequent usage of shampoos. Careful selection, and if needed, more frequent application of the adulticide is recommended in case of repeated pet washing. The use of oral adulticides would be especially beneficial in this situation.

*Performance of allergen-specific intradermal and/or IgE serological tests to identify possible environmental allergen flare factors: SOR C (case scenarios 2a and 2b)*

*Rationale for such recommendation.* Environmental allergens, such as house dust mites, have been shown to cause flares of AD in dogs hypersensititive to these allergens (COE IIb).[22–24] The performance of allergen-specific intradermal testing (IDT) and/or IgE serological tests is helpful to identify hypersensitivity to environmental allergens in dogs with AD (COE III).[10,11] Furthermore, these tests are useful to separate AD from ALD, a disease with identical clinical signs but where hypersensitivity to aeroallergens cannot be determined.[2] Finally, the identification of IgE hypersensitivities can serve as the basis of allergen-specific immunotherapy (ASIT, see below). It must be remembered, however, that positive immediate IDT reactions and IgE serologies to environmental allergens are also common in dogs without signs of AD (COE III). As a result, these tests cannot be used to differentiate dogs with AD from normal dogs. Importantly, there is no evidence that serological and intradermal tests to determine hypersensitivity to food allergens reliably predict the results of restriction or provocation dietary trials in dogs with adverse food reactions (COE III). Consequently, such tests cannot be recommended to assess the presence of food hypersensitivity in dogs with food-induced AD (SOR C).

*Implementation of house dust mite control measures: SOR C (case scenarios 2a and 2b)*

*Rationale for such recommendation. Dermatophagoides* house dust mite glycoproteins are the most common allergens in dogs with AD, worldwide (COE III).[25] Reducing mite and their allergens in the home of a patient with mite hypersensitivity is seductive in theory, but difficult in practice.[26]

This dilemma is supported by the existing controversy surrounding the effectiveness of house dust mite control measures in the management of human patients with mite-sensitive asthma or AD (COE Ia).[27–29]

A statistically significant reduction in levels of mite allergens collected in living room carpets from households with atopic dogs was shown with the use of direct environmental flea control within the preceding year has (COE IIb).[30] One uncontrolled study reported the benefit of house dust mite control with the acaricide benzyl benzoate spray (Acarosan Spray, Bissell, Grand Rapids, MI, USA) for reduction of clinical signs of AD in mite-hypersensitive atopic dogs (COE IIb).[31] Products (carpet sprays, powders, carpet shampoos, on-animal products, etc.) containing ingredients other than benzyl benzoate are currently marketed in some countries and purport to reduce allergen levels in the household, or even on the pet. However, there is currently no evidence that these products provide any clinical benefit for dogs with AD.

To summarize: household dust mite control measures 'theoretically' should be effective for mite-allergic patients. However, even when specific products have been shown to measurably decrease dust mite allergen in the environment, this might not necessarily lead to an improvement in clinical signs in hypersensitive individuals. Nevertheless, if mite avoidance measures were to be attempted, it would seem logical to restrict this intervention to dogs sensitized to house dust mites alone, and to use a combination of measures that might include acaricides, impermeable pet mattress covers, and frequent and thorough pet mattress and environment washing and vacuuming. A benefit, if any, is likely to take some months to occur due to the long persistence of mite allergens in the environment. Additional studies are

needed to confirm the clinical benefit of mite allergen reduction strategies in atopic dogs.

*Evaluation of use of antimicrobial therapy: SOR D (case scenarios 2a and 2b)*

*Rationale for such recommendation.* The skin and ears of dogs with AD are commonly infected or colonized with *Staphylococci* and *Malassezia* species. It is suspected that these microorganisms might contribute to clinical signs in some dogs (COE IV).[32] Surface cytology of the skin and ear is useful to determine whether or not *Malassezia* or *Staphylococci* are present at lesional sites (COE III). Making antimicrobial treatment decisions based solely on microbe numbers is incorrect and inappropriate as other factors, such as microbial virulence and host response, are also likely to play an important role in the genesis of clinical signs. For example, even a 'small' number of organisms might lead to AD lesion formation if these microbes are pathogenic, superantigen or toxin-producing, and/or the dog is hypersensitive to microbial allergens. In contrast, a 'high' number of organisms might not cause any harm if these are of nonpathogenic nonvirulent species or strains, and/or the dog has mounted a protective immune response against these microbes. Consequently, the result of cytology might better be limited to the sole report of 'presence' or 'absence' of detectable bacteria or yeast.

There is evidence that a proportion of atopic dogs develop an IgE-mediated hypersensitivity to *Malassezia*[33–36] or *Staphylococcus*[37] (COE LB), but the clinical relevance of this phenomenon remains unknown. Many dermatologists perform intradermal testing with *Malassezia* extracts or assess IgE serology for the yeast in an attempt to document *Malassezia* hypersensitivity. Although positive reactions are occasionally seen, the clinical relevance of this phenomenon is not currently known. There is currently insufficient evidence to recommend the inclusion of this allergen in immunotherapy protocols (SOR D).

As surface *Staphylococcus* bacteria and *Malassezia* yeast likely contribute to the severity of AD outside of 'classical' superficial infections (e.g. bacterial folliculitis or exfoliative – spreading – pyodermas), clinicians should be prepared to use a five-step strategy to determine the importance and relevance of such surface organisms to their patient's disease. Veterinarians are encouraged to: (i) identify skin lesions suggesting microbial colonization (e.g. erythema, oedema, scaling, greasiness) at particular sites, including the ears, (ii) document the presence of bacteria/yeast at these lesional sites, (iii) implement specific antibacterial/antifungal interventions (see sections above), (iv) using cytology, observe the disappearance of organisms from previously positive sites following antimicrobial interventions, and (v) document the reduction/disappearance of skin lesions at the previous sites following antimicrobial interventions (COE IV).

The systematic prescription of antibiotics and antifungal drugs to every dog with AD is not recommended, however, as such routine use of antimicrobial drugs is likely to increase the prevalence of drug-resistant microorganisms (SOR D). Because of similar concerns, the recommendation of systemic or topical intermittent antimicrobial therapy (i.e. 'pulse' therapy) should be an exception and considered only in cases of recurrent infections that cannot be managed by any other means (SOR D).

*Investigation of the relevance of other flare factors: SOR D (case scenarios 2a and 2b)*

*Rationale for such recommendation.* In human patients with AD, environmental (e.g. low humidity, clothing, detergents) and psychological factors (e.g. stress) are known contributors to the severity of clinical signs of AD. At this time, there is insufficient evidence on the role of such factors as a cause of flares of AD in dogs (COE IV). Notwithstanding this, observant owners should still be encouraged to identify any potential disease flare factors for their animals.

**Improvement of skin and coat hygiene and care**

*Bathing with a nonirritating shampoo: SOR D (case scenarios 2a and 2b)*

*Rationale for such recommendation.* weekly bathing with a mild nonirritating shampoo and lukewarm water is likely to be beneficial for a direct soothing effect to the skin, the physical removal of surface allergens and microbes and an increase in skin hydration. At this time, there is no evidence of superiority of any particular shampoo or protocol to achieve the goals mentioned above (COE IV). If the skin is greasy and scaly, antiseborrhoeic shampoos are indicated (SOR D). If infections are deemed to contribute to clinical signs, antiseptic shampoos should be preferred (see above) (SOR D). Because frequent shampooing might further dry and irritate the skin, especially with antiseborrhoeic or antimicrobial products, owners should be reminded to report any exacerbation following bathing so that a different shampoo might be prescribed. In some cases, moisturizers might alleviate any skin dryness that would occur after the baths (COE IV).

*Dietary supplementation with EFA: SOR B (case scenarios 2a and b)*

*Rationale for such recommendation.* In normal dogs, dietary supplementation with EFA, or the feeding of EFA-rich diets (especially those rich in the omega-6 EFA linoleic acid) usually results in improvement in coat quality and gloss with an associated reduction of transepidermal water loss (COE IIb).[38] Two RCTs that tested the effect of EFA-rich diets in dogs with AD reported an improvement in coat quality with either Specific CΩD Eicosa (Leo Animal Health, Ballerup, Denmark; it is now renamed Specific Skin & Joint Support, Dechra Veterinary Products, Oslo, Norway)[39] or the Hill's Prescription Diet Canine d/d Salmon & Rice (Hill's Pet Nutrition, Hamburg, Germany) (COE Ib).[40] Not all EFA-rich diets appear to have such coat improvement effect, however.[40] At this time, there is no evidence of superiority of any particular EFA combination, dosage, ratio or formulation (including

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enriched diets) to improve skin and coat quality in dogs with AD (COE Ia).[16] In general, EFA-enriched diets provide higher amounts of EFA than their administration as oral supplements.[41,42] The benefit of EFA, if any, might not be seen before 2 months of supplementation.[16,43] Finally, the limited degree of improvement in clinical signs makes it unlikely that EFA supplements or EFA-enriched diets would be suitable for monotherapy of canine AD (COE Ia).

*Topical lipid formulations*

At this time, there is insufficient evidence supporting the use of topical formulations containing EFA, essential oils, or complex lipid mixtures for improvement of coat quality, barrier function or any other clinically relevant benefit in dogs with AD (COE IV). The authors note, however, that some lipid-based topical emollient products appear effective in human AD, and that several such products are under development and evaluation in the veterinary arena. In particular, a complex lipid mixture has been shown recently to help restore pre-existing ultrastructural lipid anomalies in a small number of dogs with AD.[44] Recommendation for usage of this or other topical lipid formulations in dogs with AD must await the performance of high quality trials that demonstrate a cost-effective and relevant clinical benefit (SOR D).

*Other dietary supplements*

Several nutritional supplements (e.g. pantothenate, choline, nicotinamide, histidine and inositol) have been shown to increase the production of ceramide skin lipids *in vitro* and to decrease transepidermal water loss *in vivo* in healthy dogs (COE LB).[45] Additional studies are needed to confirm the clinical benefit of diets containing these supplements in dogs with AD.

### Reduction of pruritus and skin lesions with pharmacological agents

*Treatment with topical glucocorticoids or tacrolimus: SOR A (case scenario 2a)*

*Rationale for such recommendation.* A recent systematic review of RCT confirmed the efficacy of topical glucocorticoids for treatment of AD in dogs (see above) (COE Ia).[20] There is RCT-grade evidence of high efficacy of a 0.015% triamcinolone spray (Genesis, Virbac, Ft Worth, TX, USA) and of a 0.0584% hydrocortisone aceponate spray (Cortavance, Virbac, Carros, France) used initially once (Cortavance) or twice daily (Genesis) then tapered (COE Ib).[13,15] Clinicians are encouraged to tailor the frequency and duration of application of topical glucocorticoids to the severity of clinical signs (COE Ib).[15] Such formulations are best suited for focal (e.g. pedal)[14] or multifocal lesions and for relatively short durations (e.g. less than 2 months). Even though largely untested in dogs with AD, other topical glucocorticoid formulations are likely to provide clinical benefit; their efficacy and side effects will depend normally on formulation type, potency and duration of application (COE IV).

The most common and important adverse events following the prolonged application of a potent topical glucocorticoid on the same area are thinning of the skin (cutaneous atrophy), comedones and superficial follicular cysts (milia).[17,18] Even though the risk of skin atrophy appears low with the new diester glucocorticoids such as hydrocortisone aceponate (Cortavance spray, Virbac, Carros, France), as shown in one RCT lasting up to 70 days,[15] experimental studies with this formulation have shown this side effect to either occur[46] or not develop.[47] Because of such atrophogenic effect, however, topical glucocorticoids might be temporarily indicated to induce a thinning of lichenified chronic skin lesions.

As an alternative to topical glucocorticoids, 0.1% tacrolimus ointment (Protopic, Astellas Pharma, Tokyo, Japan) has been shown to be effective, especially in dogs with localized AD (COE Ib).[19,48] The efficacy of tacrolimus ointment appears highest when used twice daily for 1 week with ensuing reduced frequency of application as needed to control signs. As in humans with AD, the application of tacrolimus might be followed by signs suggesting mild irritation.[19] As mentioned in a preceding section, the relatively slow onset of clinical benefit of tacrolimus ointment suggests that this formulation is not suitable to treat acute flares of canine AD.

*Treatment with oral glucocorticoids or ciclosporin: SOR A (case scenarios 2a and 2b)*

*Rationale for such recommendation.* Systematic reviews of clinical trials have established the efficacy of oral glucocorticoids[16] and ciclosporin[16,49] for treatment of AD in dogs (COE Ia). Such oral medications are especially suited for dogs with nonlocalized AD, and when other flare factors have been identified and eliminated (COE IV). The onset of clinical benefit arises earlier with glucocorticoids than with ciclosporin (COE Ia).

As discussed above, oral glucocorticoids (e.g. prednisone, prednisolone, methylprednisolone) should be commenced at approximately 0.5 mg/kg once to twice daily, and then decreased, as signs abate, to the lowest dose and frequency (e.g. twice daily to once daily to every other day) needed to maintain good quality of life, control of signs and minimal side effects (COE Ia).[16] Side effects of oral glucocorticoids (e.g. polyuria, polydipsia, polyphagia, predisposition to urinary tract infections) are common and normally proportional to dosage and duration of administration (COE Ia).[16] Clinicians should be aware that the long-term use of glucocorticoids can also result in calcinosis cutis and, sometimes, predispose to the development of demodicosis. The inflammation associated with these conditions can cause owners to erroneously believe that the allergic signs are flaring, thereby prompting them to inappropriately increase the frequency of use of topical or oral glucocorticoids. At this time, because of the risk for adverse effects, the use of long-acting injectable glucocorticoids is not recommended unless there is an inability to treat the patient orally (SOR D).

In an attempt to reduce the dose of oral glucocorticoids needed to control clinical signs of AD, veterinarians are encouraged to investigate the simultaneous administration of additional medications or supplements that might have a steroid-sparing effect.

For example, an early crossover trial reported that a combination of the antihistamine trimeprazine and the glucocorticoid prednisolone [Vanectyl-P (Temaril-P), Pfizer Animal Health, Kirkland, Canada] had a higher antipruritic efficacy than trimeprazine or prednisolone given alone (COE IIa).[50] Whether or not such steroid-sparing effect would be seen with other antihistamines has not been established.

Similarly, a RCT established that the daily administration of a specific EFA liquid supplement (Viacutan Plus, Boehringer Ingelheim, Ingelheim, Germany) permitted the reduction of the dose of prednisolone needed to control pruritus in dogs with AD (COE Ib).[51] The statistically significant reduction in prednisolone dosage occurred after approximately 2 months. Whether or not similar glucocorticoid-sparing effects would be seen with other EFA supplements or enriched diets is unknown.

Finally, a RCT demonstrated that a Chinese herbal supplement (Phytopica, Intervet-Schering Plough Animal Health, Milton Keynes, UK) allowed a statistically significant reduction of the dosage of methylprednisolone needed to treat dogs with moderate to severe AD (COE 1b).[52]

Modified ciclosporin (Atopica, Novartis Animal Health, Basel, Switzerland) should be started at a dosage of 5 mg/kg once daily and continued at this dosage until a halving or a satisfactory decrease of severity of signs is achieved (COE Ia). After this improvement is reached, the dose should be reduced by either increasing dosage intervals (e.g. going from every day to every other day) or by decreasing the daily dose by half. After a further reduction of signs exceeding approximately 75%, the administration could be reduced to twice weekly or a 75% reduction of the original daily dose (COE Ia).[16,49] Other dose-reducing regimens also might be helpful, but these have not been fully tested. After beginning ciclosporin administration, the onset of satisfactory clinical benefit normally cannot be expected before four to 6 weeks. Consequently, the response to this drug should not be assessed, nor dose adjustments be made, for at least 1 month after commencing therapy. To increase the speed of clinical sign improvement, the administration of a short course of oral glucocorticoids – as described above – during the first 2 weeks of ciclosporin administration might be beneficial (SOR D). Minor adverse events (e.g. vomiting, diarrhoea) are common after initiating ciclosporin therapy; most improve spontaneously upon further administration of this drug (COE Ia).[16,49] The concurrent long-term administration of oral ciclosporin and glucocorticoids – especially at higher dosages of either or both drugs – should be monitored carefully, as potent combined immune suppression is likely to result in a higher risk for development of potentially severe opportunistic infections of the skin or other organs.

*Treatment with subcutaneous interferons: SOR A (case scenarios 2a and 2b)*

*Rationale for such recommendation.* Two RCTs provide evidence of the efficacy of recombinant canine gamma-interferon (Interdog, Toray Industries, Tokyo, Japan) to treat dogs with AD in Japan (COE Ib).[53,54] Suggested effective dosages are 5000–10 000 units/kg, subcutaneously, three times weekly for 4 weeks then once weekly. Side effects appear to be minimal.[53,54] Two studies, including one RCT, suggest that subcutaneous injections of recombinant feline omega interferon (Virbagen Omega, Virbac, Carros, France) might have some clinical efficacy to treat dogs with AD (COE IIb).[55,56] Suggested doses of one to four million units per injection over 6 months and then every month appear to be well tolerated. Whether or not repeated injections of recombinant feline omega interferon in dogs lead to a host immune response against this heterologous protein, later followed by a progressive reduction in this biological drug's efficacy, is unknown. Further recommendations for this intervention must await the performance of larger and longer clinical trials.

*Interventions likely to be of little or no benefit to treat chronic canine AD*

As a group, first (i.e. sedating) and second (i.e. lower sedation) generation oral type 1 histamine receptor inverse agonists ('type 1 antihistamines') are unlikely to be of clinical benefit in dogs with chronic skin lesions (COE Ia).[16] This low efficacy of type 1 antihistamines might be due to the lack of relevance of histamine and/or type 1 histamine receptors in the persistence of canine AD chronic skin lesions. Alternatively, a lack of clinical benefit could be due to inappropriate dosages, frequency of administration or type of antihistamine used. For example, clemastine is a type 1 antihistamine that has been used for almost two decades, yet it has been shown recently not to be bioavailable and to lack effect after oral use in dogs (COE IIb).[57] In the absence of convincing clinical trials, if veterinarians wish to use type 1 antihistamines, they should limit their prescription to those drugs with demonstrable inhibitory effect of intradermal histamine injections in dogs. At this time, antihistamines with such proven effect are hydroxyzine (2 mg/kg twice daily, COE IIb) [58] and cetirizine 0.5–1.0 mg/kg once daily (COE IIb).[59] Antihistamines should be given as preventatives, that is every single day at the recommended dosage, to maintain H1 receptors in an inactive state before histamine is released during immediate allergic reactions (SOR D). Type 1 antihistamines might be better suited for dogs with mild skin lesions or pruritus manifestations (SOR D). Even though antihistamines, when given as a single drug, do not appear effective as a group, a combination of the type 1 antihistamines hydroxyzine and chlorpheniramine maleate (Histacalmine, Virbac, Carros, France) has been reported to be clinically beneficial in dogs with AD (COE Ib).[60] Whether or not other antihistamine combinations would show a similar efficacy is not known.

As discussed above, there is evidence of potential glucocorticoid sparing effect of the type 1 antihistamine trimeprazine (COE IIa).[50] Whether or not such phenomenon would be seen with other type 1 antihistamines has not been determined. Clinical trial results suggest that some type 1 antihistamines can induce sedation as a side effect of their administration in dogs with AD (COE Ia).[16] This adverse event might be responsible for the small

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benefit seen in this class of drugs in some dogs with AD, and it might be especially useful in dogs with pruritus-associated disturbed sleep patterns.[61,62] In one study, however, diphenhydramine was reported to have limited sedation potential in dogs (COE IIb).[63]

The limited improvement in clinical signs following treatment, as shown in the systematic review of RCT, means that EFA supplements, EFA enriched diets and nutritional or herbal supplements are unlikely to provide meaningful benefit if given alone for relief of inflammation and/or pruritus (COE Ia).[16] As discussed above, EFA might be useful to improve coat quality and ameliorate dry skin, but, at this time, there is no evidence of superiority of any particular EFA combination, dosage, ratio or formulation (including enriched diets) to achieve skin barrier, coat quality or anti-allergic effect (COE Ia). As reported in a preceding section, one EFA combination (Viacutan Plus) and one Chinese herbal supplement (Phytopica; Intervet-Schering Plough Animal Health) have been shown to have steroid-sparing effects in well-designed RCTs (COE Ib). Whether or not similar observations would be made with other nutritional supplements has not been established, and care must be taken to not extrapolate these findings to other untested products.

There is some evidence of anti-allergic efficacy of oral pentoxifylline and misoprostol (COE Ia).[16] Because of their modest benefit, relatively high costs and adverse effects, these medications should probably not be used as first line medications to treat dogs with AD (SOR A). A recent clinical trial tested the efficacy of the dual cyclooxygenase and 5-lipoxygenase inhibitor tepoxalin (Zubrin; Intervet-Schering-Plough Animal Health, Boxmeer, The Netherlands) in dogs with AD (COE Ib).[64] The very limited improvements in pruritus and skin lesions seen in most dogs during this trial suggest that this drug might not offer much advantage for treatment of dogs with AD. The combination of a nonsteroidal anti-inflammatory agent and glucocorticoids, furthermore, should be avoided because of the risk of inducing gastric or duodenal ulceration (SOR D).

A recent systematic review confirmed that there is some evidence of very low, or complete lack of efficacy of leukotriene inhibitors, dextromethorphan or capsaicin to treat dogs with AD (COE Ia).[16] Consequently, these drugs should not be used to treat dogs with this disease (SOR A). Other medications have not been tested sufficiently to ensure an appropriate recommendation for or against their use for treatment of canine AD.

### Implement strategies to prevent recurrence of signs

*Avoidance of flare factors. SOR D (case scenarios 2a and 2b)*

*Rationale for such recommendation.* Avoidance of known flare factors is the strategy most optimal to prevent recurrence of signs in patients with AD. As discussed in the sections above, the maintenance of the dog on a diet not containing ingredients to which it is hypersensitive, the implementation of an effective flea control and a reduction of contact with provocative environmental or microbial allergens would be ideal, wherever and whenever possible.

*Implementation of preventive pharmacotherapy: SOR F (case scenarios 2a and b)*

*Rationale for such recommendation.* In humans with AD, there is evidence of high benefit, cost effectiveness and low risk of proactive intermittent applications of topical glucocorticoids and tacrolimus to skin areas repeatedly affected during flares of AD (COE Ib).[65–68] Such intermittent application of potent anti-inflammatory drugs onto healed skin appears to delay or prevent flares of AD skin lesions. Whether or not a similar strategy would be equally effective in dogs with AD has not been established at this time, but because of the possible benefit, low risk and low cost, such interventions are worth considering in dogs with recurrent moderate or severe AD.

The proactive administration of other drugs (e.g. type 1 antihistamines, other immunomodulators) or supplements (e.g. Chinese herb mixtures, EFA, etc.) theoretically might help prevent the recurrence of flares of AD in a dog whose signs had previously reached remission. The benefit and cost effectiveness of such a concept have not yet been tested in clinical practice.

*Implementation of allergen-specific immunotherapy: SOR A (case scenarios 2a and b)*

*Rationale for such recommendation*. Allergen-specific immunotherapy (ASIT) is the practice of administering gradually increasing quantities of an allergen extract to an allergic subject to ameliorate the symptoms associated with subsequent exposure to the causative allergen.[69,70] A systematic review established that subcutaneous ASIT appeared effective and safe to reduce signs of AD in dogs (COE Ia).[16] As suggested in a previous review[71] (SOR D), ASIT should be considered in any dog where a diagnosis of AD – but not ALD – has been made, in whom IDT or IgE serology have permitted the identification of allergens that are likely to be contributing to the disease and in whom allergen contact is unavoidable. Moreover, the dog's owners should be able to afford the time, expense and technical aspects of this regimen. In addition, when symptomatic anti-inflammatory therapy is ineffective, or associated with unacceptable or potentially unacceptable side effects (e.g. glucocorticoids), or is impractical to maintain for an extended period of time, then ASIT is indicated, even in dogs with seasonal disease of short duration. Finally, due to its unique mode of action, ASIT is the only intervention that has the potential to prevent the development of signs and alter the long-term course of the disease.

As written above, veterinarians can use either allergen-specific intradermal or IgE serological tests to identify hypersensitivity to common environmental allergens as there is no clear evidence that the response to ASIT is superior using allergens selected by IDT or serology (COE III).[72,73] To be included in the ASIT preparations, allergens identified must fit exacerbation patterns of clinical signs

and there must be likely exposure based on the clinical history and geographical location (SOR D).

It is expected that between approximately 50% and 80% of dogs with AD that have been treated with ASIT for six to twelve months will exhibit an improvement in signs and/or a decrease in anti-inflammatory or antipruritic medication use.[74] At this time, there appears to exist no clear advantage of a particular ASIT protocol over other ones (traditional, rush or low-dose) (COE Ia).[16] Most importantly, injection frequencies and amounts injected must be tailored to each patient depending upon the clinical improvement observed and the presence of adverse events (e.g. increases in pruritus after each injection). Because of the delay in ASIT effect, anti-inflammatory drugs should be given temporarily, as needed to maintain good quality of life until such time as the ASIT is judged to be effective (see sections above). There is currently no evidence suggesting that the concurrent administration of topical or systemic anti-inflammatory drugs alters the clinical benefit of ASIT in dogs (COE IV). Because the onset of clinical benefit might not appear for months, ASIT must be continued for at least 1 year to properly evaluate efficacy (COE III). Whether or not ASIT must be continued for the reminder of the life of the patients has not been determined in dogs with AD.

As more details on ASIT methodology are beyond the scope of this review, readers are referred to a recent review [74] for additional information on the various protocols of ASIT and the various factors that might affect treatment outcome.

## Conclusion

In summary, the treatment of canine AD must be individualized for each patient. Treatment regimens should depend principally upon whether veterinarians are treating acute flares or chronic skin lesions of AD, and whether signs are localized or generalized. Treatment of chronic canine AD is most challenging and should incorporate a combination of detective work to identify flare factors, elimination of these factors (if feasible), optimization of skin care, reduction of skin lesions and pruritus and prevention of recurrence of signs after remission.

*Not all interventions will be suitable for every patient; drugs will not be equally effective for, or tolerated by, every dog. Veterinarians are encouraged to abide by the evidence-based medicine principles highlighted in this review. They must also, at the same time, follow pet owners' preferences – which includes the cost and ease of the various interventions – and, ultimately, consider the quality of life of each patient in the context of the recommendations described herein.*

### Future concepts and strategies

The authors note that, as of the time of this writing, several therapeutic interventions for canine AD are under active study and might be promising candidates for future recommendations. For example, drugs that inhibit the tyrosine kinase family of enzymes are under study for use in both neoplastic and inflammatory conditions, including canine AD. In addition, there is active discussion over the possible benefit of improving epidermal barrier function (via a dietary supplement or topical means) in dogs with AD. Methods to study barrier function, and the influence of such therapies on canine skin, are under active development. Results from clinical trials in dogs with AD have yet to be reported for these different interventions, but the theoretical concepts provide reason for hope that additional tools in our arsenal against canine AD might be available in the future.

## Supporting Information

Additional Supporting Information may be found in the online version of this article:

**Folder S1.** Translations of the paper 'Treatment of canine atopic dermatitis: 2010 clinical practice guidelines from the International Task Force on Canine Atopic Dermatitis'.

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**Résumé** La dermatite atopique (AD) est une dermatose canine prurigineuse chronique récidivante fréquente pour laquelle le traitement varie selon les périodes et les régions. De récentes études contrôlées randomisées de haute qualité et des revues systématiques ont établies que les traitements offrent un bénéfice conséquent. L' « International Task Force » de la dermatite atopique canine recommande actuellement une approche multiple du traitement des chiens atopiques. Les crises aigues devraient être traitées avec des bains non-irritants et des glucocorticoïdes topiques en même temps qu'identifier et traiter la cause sous-jacente. Les glucocorticoïdes oraux et une thérapeutique antimicrobienne doivent être ajoutés si nécessaire. Chez les chiens atopiques chroniques, un ensemble d'interventions doit être envisagé. Là encore, les facteurs déclenchant les crises d'atopie doivent être identifiés et, si possible, éliminés. Ces facteurs actuellement reconnus regroupent alimentation, puces et allergènes environnementaux, staphylocoques et levures Malassezia. L'hygiène et l'entretien de la peau et du pelage doivent comprendre des shampooings non-irritants et une supplémentation alimentaire en acides gras essentiels. L'intensité du prurit et des lésions cutanées peut être diminuée par une association d'anti-inflammatoires. Actuellement, les traitements ayant une haute preuve d'efficacité comprennent les glucocorticoïdes topiques et oraux ainsi que les inhibiteurs de la calcineurine tels que la ciclosporine et le tacrolimus topique. La dose et la fréquence d'administration de ces traitements doivent être adaptées à chaque animal en fonction de l'efficacité des molécules utilisées, de leurs effets secondaires et de leur coût. L'immunothérapie spécifique d'allergène devrait être proposée, lorsque possible, dans le but de prévenir la récidive des signes cliniques en cas de future exposition aux allergènes environnementaux auxquels le patient est sensibilisé.

**Resumen** La dermatitis atópica (AD) es una enfermedad crónica pruriginosa recidivante de la piel de perros para la cual ha variado el tratamiento a lo largo del tiempo y según la localización geográfica. Estudios

de calidad recientes al azar y controlados, así como revisiones sistemáticas ha establecido qué fármacos tienen mas posibilidades de ser beneficiosos de forma consistente. La fuerza internacional para el estudio de la dermatitis atópica canina actualmente recomienda una estrategia con múltiples facetas para tratar a los perros con AD. Los ataques agudos deben tratarse con una combinación de baños no irritantes y glucocorticoides tópicos, una vez que se ha llevado a cabo un intento de identificar y retirar la causa sospechosa del ataque. Los glucocorticoides por vía oral y la terapia antimicrobiana deben añadirse cuando sea necesario. En perros con AD crónica, se debe considerar una intervención combinada. De nuevo los factores que inician los ataques de AD deben ser identificados y si es posible, evitarlos. Actualmente factores iniciadores reconocidos incluyen comida, pulgas y alergenos ambientales, bacterias estafilococicas y levaduras del genero Malassezia. La higiene y cuidado de la piel y el pelo deben mejorarse con baños con champús no irritantes y suplementos dietético de ácidos grasos esenciales. La severidad del prurito y las lesiones de la piel pueden reducirse con una combinación de fármacos antiinflamatorios. Actualmente, los medicamentos con evidencia de una eficacia elevada incluyen glucocorticoides tópicos y orales, inhibidores de calcineurina, tales como ciclosporina y tacrolimus por vía tópica. La dosis y frecuencia de administración de estos fármacos debe adaptarse para cada paciente considerando la eficacia de cada fármaco, los efectos adversos y el coste. La inmunoterapia especifica de alergeno debe ofrecerse, cuando sea factible, en un intento de prevenir recidiva de los signos clínicos tras nuevas exposiciones a alergenos ambientales a los que el paciente es sensible.

**Zusammenfassung** Die atopische Dermatitis (AD) ist eine chronische wiederkehrende juckende Hauterkrankung von Hunden, für die sich die Behandlung mit der Zeit und mit der geographischen Region geändert hat. Jüngste randomisierte Studien von hoher Qualität und systematische Reviews haben festgestellt, welche Medikamente am wahrscheinlichsten von konstantem Nutzen sind. Die Internationale Task Force für canine AD schlägt momentan eine vielseitige Herangehensweise bei der Behandlung von Hunden mit AD vor. Akute Schübe sollten mit einer Kombination aus nicht-reizenden Bädern und topischen Glukokortikoiden behandelt werden, nachdem der Versuch gemacht wurde, die vermeintlichen Ursachen der Schübe zu identifizieren und zu beseitigen. Glukokortikoide per os und eine antimikrobielle Therapie müssen nach Bedarf hinzugefügt werden. Bei Hunden mit chronischer AD sollte eine Kombination dieser Interventionen überlegt werden. Wiederum müssen Ursachen, welche die Schübe der AD auslösen, identifiziert werden und sollten, wenn möglich, verhindert werden. Zu den zurzeit bekannten Schübe-auslösenden Faktoren gehören Futter, Flöhe und Umgebungsallergene, Staphylokokkenbakterien und Malassezia-Hefepilze. Die Hygiene und Pflege von Haut und Fell müssen durch Baden mit nicht-reizenden Shampoos und mit diätetischer Unterstützung durch essentielle Fettsäuren verbessert werden. Der Schweregrad von Juckreiz und Hautläsionen kann mit einer Kombination von entzündungshemmenden Medikamenten vermindert werden. Momentan gehören zu den Medikamenten mit nachweislich hoher Wirksamkeit topische und orale Glukokortikoide, Calcineurininhibitoren wie Ciclosporin und topischer Takrolimus. Die Dosis und Frequenz dieser Medikamente sollte auf jeden einzelnen Patienten abgestimmt sein, wobei auf die Wirksamkeit des Inhaltsstoffes, Nebenwirkungen und Kosten geachtet werden sollte. Eine Allergen-spezifische Immuntherapie sollte, wann immer möglich, angeboten werden, als Versuch ein Wiederauftreten von klinischen Symptomen bei weiterer Exponierung zu Umweltallergenen, auf die der Patient hypersensibel reagiert, zu verhindern.

**要約**　アトピー性皮膚炎（AD）は犬によくみられる慢性再発性そう痒性皮膚疾患でその治療法は時期や地域によって異なっている。近年、質の高い無作為化比較対照試験と系統的レビューにより、どの薬物に一貫した有用性があるかを評価することが可能となった。国際犬アトピー性皮膚炎調査委員会（the international Task Force for Canine AD）ではアトピー性皮膚炎の犬の治療に多面的なアプローチをすることを推奨した。急性の悪化時期では疑われる悪化原因を調査し、それが除去されたら、刺激性のない温浴とコルチステロイドの外用薬の組み合わせで治療するのがよい。経口グルココルチコイド剤と抗菌剤を用いた治療は必要なときに追加する。犬の慢性 AD では、治療法の組み合わせを検討する。また、AD の悪化の原因となる因子を探索し、可能であれば回避、除外する。現在までに確認された悪化因子はフード、ノミ、環境中アレルゲン、ブドウ球菌などの細菌、マラセチアがある。非刺激性のシャンプーを用いた洗浄と必須脂肪酸のサプリメントによって皮膚と被毛の衛生とケアを向上させる。そう痒の重症度と皮疹は抗炎症薬の組み合わせで低減らすることができる。近年、外用と経口のグルココルチコイド、経口シクロスポリンや外用のタクロリムスなどのカルシニューリン阻害薬による薬物治療は高い効果を示すといエビデンスがある。それらの薬剤の用量と投与間隔は各薬物に対する患者の反応、副作用、費用に合わせて調整する。患者が過敏反応を示す環境中のアレルゲンへの暴露にたいする再発性の臨床症状を回避するために、可能であればアレルゲン特異的免疫療法を実施する。

## Addendum

### Treatment of acute flares of canine atopic dermatitis

*a. Identification and avoidance of flare factors:*
   i. Identification and elimination, whenever possible, of allergenic flare factors (fleas, food and environmental allergens)
   ii. Evaluation of use of antimicrobial therapy if clinical signs of infection or colonization with bacteria or yeast are present on the skin or in the ears

*b. Improvement in skin and coat hygiene and care:*
   i. Bathing with a nonirritating shampoo

*c. Reduction of pruritus and skin lesions with pharmacological agents:*
   i. Treatment with topical glucocorticoids, especially for localized lesions, as needed to control signs
   ii. Treatment with oral glucocorticoids, especially for widespread or severe lesions, as needed to control signs

### Treatment of chronic canine atopic dermatitis

*a. Identification and avoidance of flare factors:*
   i. Dietary restriction-provocation trials in dogs with nonseasonal signs
   ii. Implementation of an effective flea control regimen in areas where fleas are present
   iii. Performance of allergen-specific intradermal and/or IgE serological tests to identify possible environmental allergen flare factors
   iv. Possible implementation of house dust mite control measures, if relevant and feasible
   v. Evaluation of use of antimicrobial therapy if signs of infection or colonization with bacteria or yeast are present on the skin or in the ears

*b. Improvement in skin and coat hygiene and care:*
   i. Bathing with a nonirritating shampoo or an antiseborrhoeic/antimicrobial shampoo, depending on the skin lesions seen
   ii. Dietary supplementation with essential fatty acids

*c. Reduction of pruritus and skin lesions with pharmacological agents:*
   i. Treatment with topical glucocorticoids or tacrolimus, especially for localized lesions, as needed to control signs
   ii. Treatment with oral glucocorticoids, ciclosporin or subcutaneous interferon, especially for widespread or severe lesions, as needed to control signs. These agents would not normally be combined together.
   iii. Use of steroid-sparing agents, such as essential fatty acids, Chinese herbs and antihistamines, if glucocorticoids are being used as a long term treatment option.

*d. Implementation of strategies to prevent recurrence of signs*
   i. Avoidance of known flare factors, as identified above
   ii. Consideration of preventive pharmacotherapy, if feasible and relevant
   iii. Implementation of allergen-specific immunotherapy, if feasible. This can be used alongside all the above treatment options in an attempt to provide long term amelioration of the aberrant immune response

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