Canine atopic dermatitis: diagnosis and management

Abstract

Canine atopic dermatitis (CAD) is a common pruritic skin disease that starts in young dogs. The diagnosis is based on a set of clinical criteria as well as ruling out other pruritic skin diseases. Intradermal and serological testing are used to detect allergens for allergen-specific immunotherapy as well as allergen avoidance, but these should not be used as diagnostic tests. CAD is an incurable disease, and cases that do not respond to diet trials will require lifelong therapy. Nurses can play a valuable role in the diagnosis and long-term management of this problematic condition.

Key words: canine atopic dermatitis, symptoms, diet trials, management

Canine atopic dermatitis (CAD) is a common skin disease that small animal practitioners will see on an almost daily basis. The exact prevalence of CAD is unknown, but it is estimated that around 10–15% of dogs may be affected (Lund et al, 1999). This is a frustrating and incurable condition that has the potential to cause great distress to patients and owners. It is a common cause of 'vet-hopping', where owners move from vet to vet, seeking an elusive cure. When surveyed, 73% of owners thought that their dog's atopic dermatitis had a major impact on their pet's health-related quality of life, and 48% considered their own quality of life was affected (Linek and Favrot, 2010).

Much work has been done over the past decade in understanding the pathophysiological mechanisms underlying CAD. What is clear is that CAD is a complex, multifactorial disease (Olivry et al, 2005, 2010). Veterinary dermatologists agree that the diagnosis of CAD is based on a set of historical and clinical criteria along with exclusion of other pruritic skin disease by diagnostic testing and therapeutic trials. (*Figure 1*). This investigation is time consuming, labour intensive and relies on good owner compliance. Atopic dermatitis is an incurable condition and requires lifelong management.

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Nurses can play a vital role in the investigation and management of CAD, including:

- Taking samples for and interpreting diagnostic tests
- Advising and encouraging owners with regard to ectoparasite control programmes
- Diet trials
- Ear cleaning and shampoo therapy
- Administering allergen immunotherapy injections and performing routine skin checks.

What is atopic dermatitis?

CAD is a complex interaction between genetic, immunological, environmental and microbial factors. CAD has recently been described as:

> *'...a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features associated with IgE antibodies most commonly directed against environmental allergens'.* (Halliwell, 2006)

A smaller group of dogs (approximately 20% of cases) have symptoms of atopic dermatitis, but are negative on allergy testing; this group are described as having 'atopic-like dermatitis' (Halliwell, 2006). In addition to developing an allergy, it has also become clear that an important component of CAD is a skin barrier function defect that predisposes to dry, itchy skin, increased allergen penetration and microbial infection. There is strong evidence that CAD is an inherited condition (de Weck et al, 1997), which explains the many predisposed breeds, and breeding from affected animals should be strongly discouraged (Shaw et al, 2004).

Characteristic clinical features of CAD

CAD starts in young dogs usually between 6 months and 3 years of age. It is rare for the disease to start in dogs >6 years. The symptoms often initially present in the summer, but may become present throughout the year after the first year.

Typically, affected dogs initially present with a history of pruritus affecting the face, ears, feet, ventrum and perineum, with or without visible skin lesions. Perineal pruritus may be mistaken for anal

Figure 1. West Highland White terrier with long-standing atopic dermatitis showing generalised traumatic alopecia and lichenification.

Figure 2. Cross-bred dog with atopic dermatitis and secondary pyoderma. Note papules, pustules, collarettes and pigmented macules, which are all characteristics of superficial pyoderma.

Figure 3. Labrador retriever with atopic dermatitis and severe secondary Malassezia *dermatitis.*

sac impaction. Dorsal pruritus is more commonly associated with ectoparasitic disease, such as fleas or cheyletiellosis, although dorsal pruritus can be a feature of CAD (Favrot et al, 2010). In long-standing cases, chronic inflammatory changes can result in saliva staining, erythema, traumatic alopecia, scaling, lichenification and hyperpigmentation (*Figure 1*). Recurrent conjunctivitis is seen in many cases.

It is common for CAD cases to develop otitis externa and secondary bacterial and yeast infections. In some cases, secondary infections are a major cause of the pruritus associated with atopic dermatitis. Bacterial infections are usually caused by *Staphylococcus pseudintermedius* and have variable presentations, including increased pruritus, papular and pustular eruptions and patchy alopecia (*Figure 2*). Yeast infections are usually caused by *Malassezia pachydermatis* and affect skinfold areas, including the lip folds, interdigital webs, ventral neck and umbilicus (*Figure 3*). While treatable with topical and perhaps systemic therapy, infections are frequently recurrent and will continue to recur until successful management of the underlying atopic disease.

How is atopic dermatitis diagnosed?

There is no single specific test to diagnose atopic dermatitis. Essentially, it is a clinical diagnosis based on history, physical examination and the ruling out of other pruritic skin diseases, including ectoparasites and infections (see *Table 1*).

Initial diagnostic tests in any pruritic dog should include coat brushings, hair plucks and skin scrapings to look for evidence of ectoparasitic disease. However, even if negative these tests do not rule out scabies, cheyletiellosis and flea involvement, and trial therapy is usually indicated. This may involve treatment of all in-contact animals in the household, even if they are apparently unaffected. In addition, it may be necessary to treat the house environment with a combined permethrin and insect-growth regulator product, such as methoprene or pyriproxyfen. It can take time and patience to explain to owners why such trial therapy is necessary, and without such understanding they are less likely to comply. Nurses should play a key role in getting this message across by emphasising the importance of not missing a potentially curable disease at this stage before making a final diagnosis of atopic dermatitis, which would require lifelong management.

At this stage it is also necessary to identify and treat bacterial and yeast infections that may be

contributing to pruritus. *Malassezia* dermatitis and pyoderma are diagnosed on clinical signs and cytology. Stained acetate tape strips are used to look for yeast and bacterial organisms, and stained impression smears are useful to identify the presence of bacteria (*Figure 4*). Nurses should be familiar with the techniques for taking suitable samples, and with training and practice can become proficient in microscopic interpretation. Shampoo therapy with either a 2% chlorhexidine/2% miconazole (Malaseb®, Dechra) or 3% chlorhexidine (Microbex®, Virbac) product is the mainstay for treatment of yeast and superficial bacterial infections, and helps to avoid the risk of inducing resistant infections with systemic antimicrobial therapy.

If pruritus persists despite ruling out parasitic diseases and infections, it is likely that the dog is suffering from atopic dermatitis, provided the history is compatible. It is now generally accepted that in some animals exposure to particular foods can trigger atopic dermatitis symptoms (Shaw and Forsythe, 2010); the next step is to investigate the possible contribution of an adverse food reaction to the pruritus by performing an elimination diet trial.

Diet trials

An elimination diet trial consists of feeding the patient a food containing a single protein and carbohydrate to which it has not been previously exposed (so-called 'novel' ingredients); this is usually one of the proprietary dried or wet foods, although home-cooked diets are another option. In the authors' experience, compliance is improved with the convenience of a proprietary diet, which also has the benefit of being nutritionally balanced and causes fewer gastrointestinal disturbances. Another alternative is to feed a hydrolysed diet, where the parent protein source has been broken down into small peptide fragments (molecular weight <10 kDa). It is thought that these small molecules are are not be capable of invoking an allergic response, and in theory it should not matter what protein sources the patient has been previously fed. However, it has been shown that hydrolysed diets may contain larger peptide fragments (Cave, 2006) and it has been recently recommended that a hydrolysed diet originating from a novel parent protein for that patient should be selected (Olivry and Bizikova, 2010). Recently, a hydrolysed diet (Anallergenic®, Royal Canin) has been produced, where peptide fragments are no greater than 1 kDa, which may obviate this requirement although this has yet to be demonstrated in clinical trials.

Table 1. Common pruritic skin diseases that can affect dogs

Parasitic diseases	Infections	Allergies
Scabies	*Malassezia* dermatitis	Flea allergy dermatitis
Cheyletiellosis	Pyoderma	Food allergy and other adverse food reactions
Harvest mites		Atopic dermatitis
Lice		
Demodicosis		

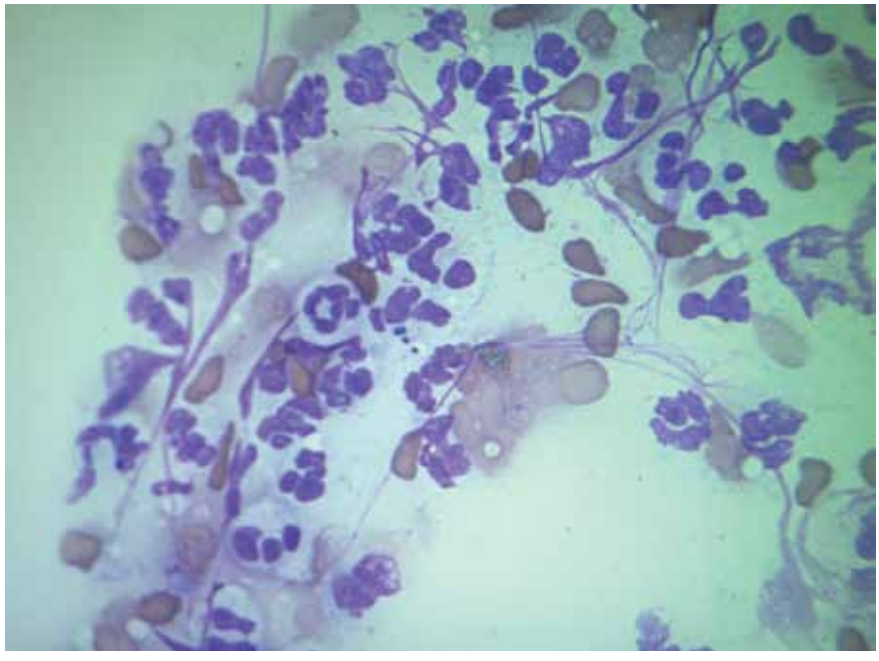

Figure 4. Cytology preparation from a case of superficial pyoderma showing neutrophils, red blood cells and coccoid bacteria (likely to be Staphylococcus pseudintermedius).

Owners should be instructed to avoid feeding other protein sources to their dog. Chews, biscuit treats, flavoured toys, flavoured toothpaste and some medications may contain protein sources to which the patient may react. All this needs to be carefully explained. In the author's practice, it has been seen that clients are much more likely to adhere to written, rather than a barrage of verbal instructions.

Owners are encouraged to continue diets for at least 6 weeks, but in some circumstances the diet trial may be prolonged as some dogs can take up to 12 weeks to respond to a change of diet (Rosser, 1993). If an adverse food reaction is involved, pruritus should resolve when the restricted diet is fed (*Figures 5a,b*); there should be a relapse on challenge with the original food, and then improvement again with re-institution of the restricted diet.

Owners can find dietary trials particularly challenging, resulting in poor compliance and missed

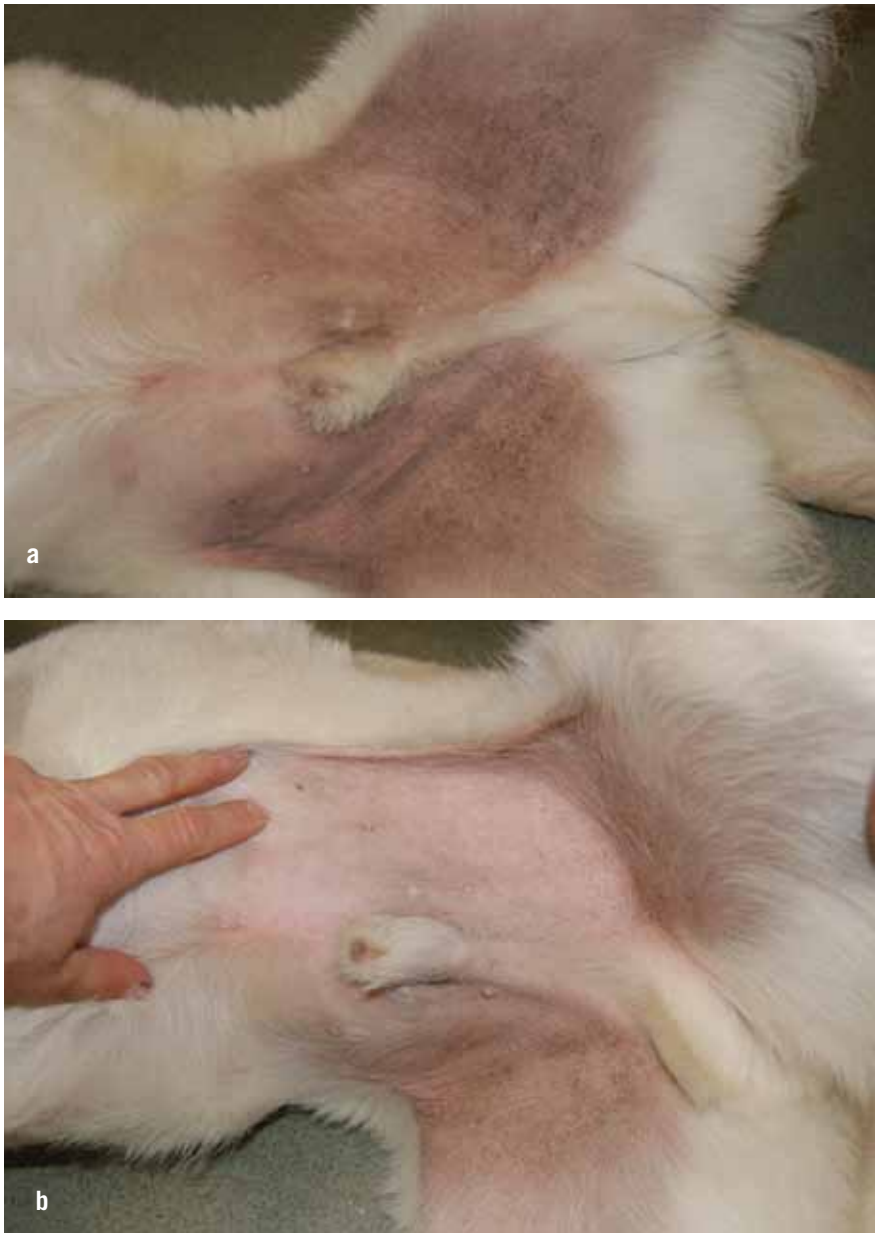

Figure 5. Groin of a pruritic Labrador retriever before (a) and after (b) a 6-week diet trial showing resolution of pigmentation (and pruritus). This dog's symptoms recurred on challenge with the original diet.

diagnoses. They may perceive that they are being unkind when they withhold treats, and that the diet trial is affecting the relationship with the pet. Nurses can play a vital support role and provide counselling to find ways around any stumbling blocks. Feeding fresh, cooked food as treats can help to improve compliance, provided that this consists of the same carbohydrate and protein as the elimination diet. Using a little imagination in the food preparation can be helpful; for example, strips of pork cooked slowly in the oven may be a tasty treat for a dog being fed a proprietary pork and potato kibble. To encourage clients to continue a diet trial, it is worth reminding them that food allergy is a 'good disease' because it is easy to control, and even if their pet responds to the diet trial, it is unlikely they will have to feed their pet such a restricted diet in the long term.

During the diet trial it is usually necessary to continue routine flea control, shampoo therapy and ear cleaning. Short-term glucocorticoid therapy may be required to control pruritus in severely affected dogs. In the author's practice prednisolone is prescribed at a dosage of 0.5–1 mg/kg orally every 24 hours, which may be administered by the owner for 3 days at a time if the dog is uncomfortably pruritic. This course may be repeated as required during the diet trial, giving the owner a means for controlling pruritus without masking any response to the diet.

Although serological tests are available for food allergies, there is currently no evidence that they are of any diagnostic value. However, there is some recent evidence that they may be of value to aid the selection of ingredients for an elimination diet (Bethlehem et al, 2012).

What happens if there is no response to the diet trial?

If there is no response to the diet trial, provided ectoparasitic and infectious causes and food allergies have been carefully excluded, the dog will require lifelong management for pruritus. There are many options for this, and there is no 'one size fits all' treatment; instead, combinations of measures are more likely to be effective. The aim is to find a method of controlling pruritus that gives the dog a good quality of life, that does not produce unacceptable side effects, that the owners feel comfortable with and that is affordable. Inevitably, some compromise of these ideals may be required.

Some general principles to consider in the management of CAD are:

- Control of flare factors
- Allergen-specific immunotherapy and avoidance of causative allergens
- Improved skin and coat care
- Pharmacotherapies.

Control of flare factors

So-called flare factors include secondary bacterial and yeast infections and parasitic diseases such as fleas or harvest mite infestation. Control of flare factors can be key to successful management in some cases of CAD. It is common for CAD patients to be apparently well-controlled and then experience a sudden flare of pruritus; it is therefore important to

identify why this has occurred. The owner may call in to report this and a recheck should be arranged unless the reason can be readily identified on the telephone. Most commonly it is because the dog has developed a secondary bacterial or yeast infection that should be identified on clinical signs and cytology and treated appropriately.

Allergen-specific immunotherapy and allergen avoidance

The identification of causative allergens can be helpful to tailor treatment. 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 (Bousquet, 1998; Loewenstein and Mueller, 2009). Uncontrolled studies have reported that ASIT is efficacious in the treatment of CAD in 50–100% of cases (Loewenstein and Mueller, 2009). However, there is only one placebo controlled study on the use of ASIT in CAD and this study reported that 59% of cases showed a 51% or greater improvement (Willemse et al, 1984). Allergen avoidance measures may also be helpful in relieving symptoms in some cases, and are listed in *Table 2*. These measures require identification of causative allergens, and this is the role of allergy testing in CAD. Allergy tests do not diagnose CAD because healthy dogs with no evidence of skin disease are frequently positive on these tests (Lian and Haliwell, 1998).

Intradermal testing involves injecting a panel of allergens directly into the dermis and detecting the presence of allergen-specific IgE antibody in the skin (*Figure 6*). Interpreting the results of intradermal tests requires experience, and the cost of maintaining a suitable panel of allergens makes this a technique used mainly by veterinary dermatologists. Serology detects the presence of IgE in the circulation. Various laboratories offer such tests and will also give advice on which allergens to include in a course of immunotherapy as well as supply the treatment. Serological testing is appropriate in most general practice situations.

ASIT can take many months to be effective, and additional treatment to control pruritus is often required during this interim period. If effective, ASIT should be continued for life. Nurses can play a role in the administration of immunotherapy injections and performing routine skin and ear checks.

Improved skin and coat care

Dry skin lowers the pruritic threshold, and defective barrier function encourages the penetration of irritants, allergens and microbes; thus measures to improve skin health and hydration may be of benefit in managing CAD.

Shampoos can be soothing, anti-pruritic, moisturising and facilitate the removal of allergen and

Table 2. Allergen avoidance measures*

House dust mites

- Use impermeable covers on soft furnishings and bedding
- Keep the pet's bedding areas clutter free and remove soft toys to prevent dust build up
- Keep the pet away from dusty areas
- Vacuum the house weekly with a high-efficiency particulate absorption (HEPA) filter. Keep the pet outdoors during vacuuming and for 1 hour afterwards
- Substitute laminate flooring for carpets wherever possible
- Wash bedding weekly in a hot wash (>130˚ F; 54.4˚ C)
- Reduce relative humidity in the home to 30–45% where practicable (e.g. dehumidifier, air conditioning)

Molds

- Avoid freshly cut grass, compost heaps, grass clippings, leaf piles etc
- Reduce relative humidity in the home to 30–45%
- Avoid damp rooms such as basements, bathrooms and utility areas
- Keep bedding clean and dry
- Store food in a dry environment
- Wipe damp areas with a fungicide or dilute sodium hypochlorite solution (one part bleach to nine parts water)

Pollens

- Pollen avoidance is nearly impossible to achieve. Advice to people allergic to pollens includes keeping windows closed and using air conditioning, minimising outdoor activities between 05.00 and 10.00, staying indoors on high-pollen days and windy days, and avoiding freshly cut grass. This may or may not be practicable in pollen allergic pets

*From Hillier (2002)

Figure 6. Positive intradermal test. Note positive reactions to dust and storage mite allergens along the top row.

Key points

- Canine atopic dermatitis (CAD) is a common skin disease involving a complex interaction between genetic, immunological, environmental and microbial factors.
- CAD starts in young dogs aged 6 months to 3 years; dogs initially present with a history of pruritus affecting the face, ears, feet, ventrum or perineum.
- It is common for CAD cases to develop otitis externa and secondary bacterial and yeast infections.
- Clinical diagnosis is based on history, physical examination and the ruling out of other pruritic skin diseases, such as ectoparasites and infections.
- As exposure to particular foods can trigger CAD symptoms, an elimination diet trial should be instigated to identify any adverse food reaction contributing to the pruritus.
- If there is no response to the diet trial and ectoparasitic and infectious causes have been excluded, the dog will require lifelong management for pruritus.
- Management of CAD includes control of flares, allergen-specific immunotherapy and allergen avoidance, improved skin and coat care, and pharmacotherapies.

microbes from the skin surface. Once- or twice-weekly shampoo treatments can be a helpful part of the management of atopic dermatitis.

Spot-on products containing lipids and fatty acids are available that are claimed to improve skin barrier function, and a clinical benefit to this has also been demonstrated (Piekutowska et al, 2008).

Diet affects the quality of skin and hair coat, and there is evidence that diets high in essential fatty acids are beneficial in the management of CAD (Taugbøl et al, 2004; Bensignor et al, 2008).

Pharmacotherapies

Although the above measures can be successful, some cases require additional therapy to control pruritus (Olivry et al, 2010). These treatments include essential fatty acid supplements, antihistamines, systemic glucocorticoids and ciclosporin (Atopica®, Novartis).

Despite their widespread use, there is little conclusive evidence to support the efficacy of antihistamines for the treatment of CAD (Olivry et al, 2010). This is borne out by clinical experience although there are occasional cases that may benefit from antihistamine therapy; although they have the advantage of minimal side effects, they can be sedating in some dogs (Paterson, 1994). Similarly, essential fatty acid supplementation has only a mild benefit, but was shown to reduce the amount of prednisolone required to control pruritus in some CAD patients (Saevik et al, 2004).

Topical and systemic glucocorticoids are effective in controlling pruritus in many cases. Topical therapy using hydrocortisone aceponate has been shown to be safe and effective in the control of pruritus in CAD, but is mainly useful for treating focal areas. Systemic glucocorticoids are effective but carry the risk of longer-term adverse effects.

Wherever possible, short-acting, orally administered preparations should be used, such as prednisolone or methylprednisolone, and preferably administered on alternate days at the lowest possible dosage that controls pruritus. Ciclosporin is as effective as prednisolone in controlling pruritus, without producing the unacceptable adverse effects. However, it can take at least 2–4 weeks of treatment before any reduction in pruritus can be expected. The main adverse effect of ciclosporin is vomiting and loose stools, and it is an expensive therapy. Recently, the janus kinase inhibitor, oclacitinib, has been shown to be effective for the management of pruritus in CAD and has recently been licensed in Europe for this indication (Cosgrove et al, 2013). This treatment may have a significant impact on the way atopic dermatitis is managed.

Conclusion

In summary, CAD is a common pruritic skin disease that starts in young dogs and is a complex interplay between genetic predisposition and environmental factors. The diagnosis is based on careful history taking, clinical diagnostic criteria and a systematic approach to rule out other pruritic diseases. Many cases are predisposed to secondary bacterial and yeast infections that contribute to pruritus. The majority, but not all, cases are associated with hypersensitivity to dietary ingredients or environmental allergens.Treatment should be tailored to the individual client and patient and should take into account severity of the disease, owner expectations, cost and the potential for adverse effects. **VN**

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References

Bensignor E, Morgan DM, Nuttall T (2008) Efficacy of an essential fatty acid-enriched diet in managing canine atopic dermatitis: a randomised, single-blinded, cross-over study. *Vet Dermatol* **19**: 156–62

Bethlehem S, Bexley J, Mueller RS (2012) Patch testing and allergen-specific serum IgE and IgG antibodies in the diagnosis of canine adverse food reactions. *Vet Immunol Immunopathol* **145**: 582–9

Bousquet J, Lockey R, Malling HJ et al (1998) Allergen immuno-therapy: therapeutic vaccines for allergic diseases. World Health Organization. American Academy of Allergy, Asthma and Immunology. *Ann Allergy Asthma Immunol* **81**: 401–5

Cave NJ (2006) Hydrolyzed protein diets for dogs and cats. *Vet Clin North Am Small Anim Pract* **36**: 1251–68, vi

Cosgrove SB, Wren JA, Cleaver DM et al (2013) Efficacy and safety of oclacitinib for the control of pruritus and associated skin lesions in dogs with canine allergic dermatitis. *Vet Dermatol* **24**: 479-e114

de Weck AL, Mayer P, Stumper B, Schiessl B, Pickart L (1997) Dog allergy, a model for allergy genetics. *Int Arch Allergy Immunol* **113**: 55–7

Favrot C, Steffan J, Seewald W, Picco F (2010) A prospective study on the clinical features of chronic canine atopic dermatitis and its diagnosis. *Vet Dermatol* **21**: 23–31

Halliwell R (2006) Revised nomenclature for veterinary allergy. *Vet Immunol Immunopathol* **114**: 207–8

Hillier A (2002) Allergy testing and treatment for canine atopic dermatitis. *Vet Med* **97**: 210–24

Lian TM, Haliwell RE (1998) Allergen-specific IgE and IgGd antibodies in atopic and normal dogs. *Vet Immunol Immunopathol* **66**: 203–23

Linek M, Favrot C (2010) Impact of canine atopic dermatitis on the health-related quality of life of affected dogs and quality of life of their owners. *Vet Dermatol* **21**: 456–62

Loewenstein C, Mueller RS (2009) A review of allergen-specific immunotherapy in human and veterinary medicine. *Vet Dermatol* **20**: 84–98

Lund EM, Armstrong PJ, Kirk CA, Kolar LM, Klausner JS (1999) Health status and population characteristics of dogs and cats examined at private veterinary practices in the United States. *J Am Vet Med Assoc* **214**: 1336–41

Olivry T, Bizikova P (2010) A systematic review of the evidence of reduced allergenicity and clinical benefit of food hydrolysates in dogs with cutaneous adverse food reactions. *Vet Dermatol* **21**: 32–41

Olivry T, Deboer DJ, Favrot C et al (2010) Treatment of canine atopic dermatitis: 2010 clinical practice guidelines from the International Task Force on Canine Atopic Dermatitis. *Vet Dermatol* **21**: 233–48

Olivry T, Marsella R, Maeda S, Pucheu-Haston CM, Hammerberg B (2005) Mechanism of lesion formation in canine atopic dermatitis. In: Hillier A, Foster AP, Kwochka KW, eds. *Advances in Veterinary Dermatology*. Vol 5. Blackwell Science, Oxford: 10–16

Paterson S (1994) Use of antihistamines to control pruritus in atopic dogs. *J Small Anim Pract* **35**: 415–19

Piekutowska A, Pin D, Reme CA, Gatto H, Haftek M (2008) Effects of a topically applied preparation of epidermal lipids on the stratum corneum barrier of atopic dogs. *J Comp Pathol* **138**: 197–203

Rosser EJ Jr (1993) Diagnosis of food allergy in dogs. *J Am Vet Med Assoc* **203**: 259–62

Saevik BK, Bergvall K, Holm BR et al (2004) A randomized, controlled study to evaluate the steroid sparing effect of essential fatty acid supplementation in the treatment of canine atopic dermatitis. *Vet Dermatol* **15**: 137–45

Shaw SC, Forsythe PJ (2010) Atopic dermatitis and food allergy: are they the same? In: De Boer DJ, Affolter VK, Hill PB, eds. *Advances in Veterinary Dermatology*. Vol 6. Wiley-Blackwell, Oxford: 363–9

Shaw SC, Wood JL, Freeman J, Littlewood JD, Hannant D (2004) Estimation of heritability of atopic dermatitis in Labrador and Golden Retrievers. *Am J Vet Res* **65**: 1014–20

Taugbøl B, Vroom MW, Nordberg L et al (2004) A randomised, double-blinded, placebo-controlled multicenter study on the efficacy of a diet with high levels of eicosapentaenoic acid and gamma-linolenic acid in the control of canine atopic dermatitis. *Vet Dermatol* **15**(Suppl 1): 11–12

Willemse A, van den Brom W E, Rijnberk A (1984) Effect of hyposensitization on atopic dermatitis in dogs. *J Am Vet Med Assoc* **184**: 1277–80