Atopic dermatitis; evidence and anecdotes. Where are we now in 2013?

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Introduction

In managing atopic dermatitis, this article is referring to dogs where food allergy (and non-allergic adverse reactions to food), flea allergy, sarcoptic mange and bacterial infection unrelated to allergy have been excluded and the clinical diagnosis of atopic dermatitis has been established.

Atopic dermatitis is a clinical diagnosis based on logically excluding the other causes of chronic pruritus and having typical signs and history.

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 without clinical lesions at onset
5. Affected front feet
6. Affected ear pinnae
7. Non-affected ear margins
8. Non-affected dorso-lumbar area

To differentiate dogs with AD from other causes of chronic pruritus, 5 satisfied criteria has a sensitivity of 85% and a specificity of 79%.
Adding a sixth fulfilled parameter increases the specificity to 89% but decreases the sensitivity to 58%.

In both dogs and humans, atopic dermatitis is associated with environmental allergens and significant defects in the epidermal barrier. A significant proportion of both dogs and humans (of the order of 30% plus) do not have significant levels of skin based or circulating immunoglobulin E; hence testing negative to both skin and blood allergen tests. Allergen tests are not used to diagnose atopic dermatitis; the only use for allergen testing is in making a desensitising vaccine. In dogs, low Ig is referred to as “atopic like-dermatitis atopic dermatitis” and in humans “intrinsic” atopic dermatitis”.

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The sensation of pruritus and the reflex of scratching are centrally processed. Nerve endings in the dermis are stimulated by chemical mediators of inflammation and self-trauma. At this stage, there is a great gap in our knowledge of what chemical mediators initiate pruritic sensation. In dogs, injections of histamine, serotonin, tryptase, substance P (a neuro-peptide) and interleukin-2 have failed to induce pruritus as distinct from local skin inflammation. Recently, interleukin-31 (produced by T helper 2 lymphocytes and skin homing T cells) has been shown to correlate with the severity of atopic dermatitis in humans. Il-31 will induce pruritus in dogs and is found in elevated levels in a significant proportion of atopic dogs.

Dogs with atopic dermatitis suffer from repeated bacterial and yeast infections further complicating the disease process. These infections are as a result of a defective skin barrier, loss of normal local defence mechanisms and the effects of immunosuppressive drugs given to treat the primary disease.

The author is of the opinion that food allergy is not necessarily a separate disease and there is a subset of dogs which will both display typical signs of atopic dermatitis that will improve but not resolve on a hypoallergenic diet. It could be argued that hypoallergenic diets may have improved micro-nutrition and higher levels of essential fatty acids. A significant proportion of cases (in the author's experience of the order of 15%) will show improvement (but not resolution) from feeding a hypoallergenic diet for 6 to 8 weeks, and then relapse within days upon diet rechallenge.

Exposure to allergens that may trigger or exacerbate atopic dermatitis can be by any route, including airborne allergens landing on the skin, being ingested or inhaled. Contact with allergens directly on the skin will also produce signs. A common event reported by clients with allergies to grasses is an immediate “grass flare” when exposed to freshly cut or wet grasses. “Contact atopy” has been demonstrated in dogs.

True contact allergy is a delayed-type hypersensitivity reaction requiring an extended period of sensitisation. Plants in the grouping of Commelineae and Tradescantieae (for example Wandering Jew) are known contact allergens. In these cases, removal of the dog from the plant source will result in resolution. With the exception of reactions to these plants, true contact allergy is an over diagnosed disease. This is because the foot and ventral distribution of atopic dermatitis lesions can lead the clinician to an incorrect diagnosis.
What approaches do we have this disease?

The pillars of treatment are:

1. Drugs to reduce inflammation and itch.
2. Immunotherapy.
4. Treatment and control of infection
5. Identification and control of any other allergies.

Unfortunately, too many dogs are only treated with immunosuppression by corticosteroids, ignoring the other options and the concept of combination therapy. These pillars of treatment are not a cherry picking choice of one. Every case is different and will need a different combination to control the signs.
Drugs to control inflammation and itch. What’s new?

There are still only two drugs that are effective for controlling the signs of atopic dermatitis in dogs. These are corticosteroids and cyclosporine. At this stage, we have no other agents proven to be effective.

It is very important to assess response in terms of decrease of signs or decrease in the amount of corticosteroid needed to control the disease. An agent or intervention that decreases signs by 50 to 75% is still a very valuable therapeutic action. If clients expectations are managed to expect improvement but not total cure, the task of treating these dogs is made so much more rewarding for both the clinicians and the clients. Clients are encouraged to use the scale below and to evaluate the signs and not the numbers on the scale.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Extremely severe itching/almost continuous</td>
</tr>
<tr>
<td>9</td>
<td>Severe itching/prolonged episodes</td>
</tr>
<tr>
<td>8</td>
<td>Moderate itching/regular episodes</td>
</tr>
<tr>
<td>7</td>
<td>Mild itching/a bit more frequent</td>
</tr>
<tr>
<td>6</td>
<td>Very mild itching/occasional episodes</td>
</tr>
<tr>
<td>5</td>
<td>Normal dog — I don’t think itching is a problem</td>
</tr>
</tbody>
</table>

Pruritus scale as developed by Dr Peter Hill

Corticosteroids.

As every practitioner knows, corticosteroids are highly effective in suppressing the symptoms of atopic dermatitis. In fact, a failure to respond to standard doses of corticosteroids (1 mg per kilogram of prednisolone) indicates:

1. The dog may have a cause of pruritus other than atopic dermatitis. Sarcoptic mange, flea allergy and many cases of food allergy respond poorly to corticosteroids alone.

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2. Secondary infection. Malassezia and bacterial infections are intensely pruritic and respond poorly to corticosteroids.

**Systemic corticosteroids**

- Short term prednisolone for “speed-hump” crisis therapy is commonly used.
- Long-term prednisolone is poor medicine unless nothing else works and it is part of a combination.
- Depot corticosteroids in dogs have no role in long term management of atopic dermatitis

The metabolic and immunosuppressive side-effects are well known to all practitioners as are the signs of iatrogenic Cushing syndrome.

The author's explanation of corticosteroids to clients is that they are hormones that affect every tissue in the body, cause a chemical dose of AIDS and as a side effect stop the dog scratching. There is no safe dose of corticosteroids. If dogs are receiving half a milligram per kilogram of prednisolone twice a week as part of combination therapy there is a reduced risk of side-effects. These dogs should however be monitored for urinary tract infections which may be non-dose-related.

It is acceptable to use pulse corticosteroids in the induction phase of cyclosporine treatment to get over the lag period and also as short courses in the form of “speed hump therapy” to deal with flares that are not associated with infection, even when combined with immunotherapy.

**Topical corticosteroids**

There is a spectrum of potency of topical corticosteroids:

- HIGH POTENCY: Mometasone (Elocon, Mometamax ®), budesonide, hydrocortisone aceponate (Cortovance ®)
- MODERATELY HIGH: Betamethasone 17-valerate (Betnovate, Fuciderm ®)
- MODERATE POTENCY: Triamcinolone (Panolog, Aristocort ®)
- LOW POTENCY: Hydrocortisone

The potency of the topical preparation depends on the vehicle, the concentration of the active ingredient and the degree of disruption of the skin it is being applied to. For example mometasone is 700 times as potent as hydrocortisone. Elocon cream ® contains 0.1% mometasone and many of the veterinary preparations contain 5% hydrocortisone. Thus, the potency difference is still of the order of 14 times.
Lotions are less occluding and a more suitable for moist lesions. For chronic dry skin, ointments or creams are more effective.

The more potent the topical corticosteroid, the more effective it is likely to be; with higher risk of side-effects. The side-effects of topical corticosteroids are:

**Skin thinning.** Cutaneous atrophy induced by corticosteroids is a very real side-effect. The author restricts the use of potent corticosteroid topical is to 7 to 10 days of once-a-day therapy to get over flares and no more than once a week long term when using mometasone or compounded budesonide.

**Calcinosis cutis.** Local calcinosis cutis can occur at the site of corticosteroid application. This is not common but very unpleasant; resulting in nodular change and intense reaction to the calcium salts.

**Systemic absorption.** Small but significant amounts of topical steroids are absorbed through the skin and may be sufficient to depress the hypothalamic-pituitary axis. Hydrocortisone aceponate undergoes metabolism in the dermis and this serves to minimise systemic effects. Mometasone and budesonide are absorbed systemically through the skin but have short half-lives and are rapidly excreted. Systemic absorption is important when contemplating intradermal testing and more potent topical corticosteroids should be withheld for two weeks.

**Topical corticosteroid options**

**Registered products.**

Fuciderm ® or Mometamax ® are useful for treating focal pruritic inflammatory disease where there is surface infection with bacteria (and yeast in the case of Mometamax ). These products are not effective for dealing with follicular-based infections (superficial pyoderma).

**Hydrocortisone aceponate Cortovance ®** has been demonstrated to be effective in placebo-controlled studies. It is only registered for short-term use at ONE pump per 10x10cm area.


- 28 Dogs, 15 treated, 13 placebo
- TWO pumps per 10x10cm area once daily for 28 days
- Duration of trial 70 days
- No changes in blood parameters or ACTH stimulation
- At day 28, 11/15 and 7/15 HCA dogs had 50% + reductions in lesional score and pruritus respectively.
- Coat length did not influence the results.
Off label topical corticosteroids

Mometasone (Elocon ®) is commonly used as a cream by veterinary dermatologists. The author has found this a useful product in hairless skin that does not have secondary infection. Long-term it should not be used more than once a week and the dog monitored for skin thinning the owners should wear gloves when applying.

Budesonide. A single placebo-controlled trial using 0.025% budesonide as a leave-on conditioner has shown benefits in a crossover study. The vehicle may significantly influence the efficiency of the product and the compounded budesonide formulations may not be as efficient as the preparation tested in the clinical trial. Cases need to be monitored carefully for skin thinning and off label consent must be obtained.

Hydrozole ® is a human product containing clotrimazole and 1% hydrocortisone and is used in people for treating fungal infections of the foot. The author finds this a useful product where a low-powered corticosteroid is desirable and there is a risk of surface infection with bacteria or yeast such as the feet and the medial pinna. Clotrimazole is effective against gram positive bacteria and Malassezia. In case of flares, some additional Elocon ® can boost its anti-inflammatory action.

Cyclosporine

Cyclosporine is the only drug, other than corticosteroids, that is effective, in a significant proportion of cases, in controlling the symptoms of atopic dermatitis.

We now have had this as a registered drug for almost 10 years and experience has borne out the initial clinical studies.

Effectiveness. Cyclosporine given at 5 mg per kilogram daily for six weeks will result in approximately 75% to 80% improvement in 75 to 85% of dogs. It is the only current drug alternative to topical and systemic corticosteroids.

Reasons for cyclosporine failure:

Misdiagnosis. Cyclosporine is not effective against parasite allergy and food allergy in dogs. 
Infection. Cyclosporine will fail in the presence of an uncontrolled bacterial or yeast infection.
Compounded or generic products. The micro-emulsion used by Novartis is the only product that has been studied. Compounded or generic products have not been studied in dogs with respect to their efficiency and most veterinary dermatologists will see cases that fail to respond to these products. If they are to be used, they should only be used after a baseline has been established using the Novartis product and relapse can be evaluated.
Food. Cyclosporine absorption is reduced by food. It should be given on an empty stomach. One study, however, showed no difference in outcome when cyclosporine was given with food.
**Owner expectations.** Cyclosporine is a slow drug; it takes at least three weeks to begin to see a significant clinical effect and it should not be judged a success or failure until six weeks of use. The owner also must expect that success is a 75% or better reduction in clinical sign.

**Inherent failure rate.** Cyclosporine will fail in 20 to 25% of atopic dogs.

One other major limitations of high cyclosporine use is the cost. Being a biological agent derived from a fungus, the cost of production is high. Veterinarians need to consider their retail margins on this drug, given that it is very effective in many cases and often will require lifelong therapy.

**Vomiting with cyclosporine**

Vomiting in the initial stages of therapy is a relatively common side effect of cyclosporine. The author always pre-warns clients of this potential. In case of vomiting, the following protocol will assist the majority of dogs:

1. Initially if vomiting, freeze the capsule and give with a little food.
2. If this fails to stop the vomiting, premedicate 30mins before with metoclopramide. Also, the cyclosporine dose may need to be lowered temporarily. After 2-3 weeks, the metoclopramide should be able to be discontinued.
3. There will be some dogs who are not able to tolerate cyclosporine

**Cyclosporine and ketoconazole combination.**

It has been known for a significant amount of time that ketoconazole and cyclosporine compete both for cytochrome P-450 3A (an enzyme involved in the excretion of these agents) and P-glycoprotein (actively pumping these drugs out of the intestines and CNS). The combination of ketoconazole and cyclosporine results in decreased elimination and higher bioavailability; resulting in increased blood levels.

A recent study has shown that giving cyclosporine and ketoconazole at 2.5 mg per kilogram each, results in equivalent blood and skin levels to cyclosporine at 5 mg per kilogram alone.

The author's preference is to use the Novartis micro-emulsion product (Atopica ®) at the registered dose of 5 mg per kilogram daily for six weeks to establish a baseline. Then, in the case of large dogs, ketoconazole can be trialled to see if the dose can be halved. In refractory cases, ketoconazole at 2.5 to 5 mg per kilogram can be added to standard doses of cyclosporine. Practitioners need to be aware that this increases the risk of side-effects due to immunosuppression and off label consent is required from the owners. Clinicians need to be aware that there are cases of non-dose-related hepatopathy recorded with ketoconazole use in dogs.

**Antihistamines**

Antihistamines remain a relatively ineffective means of controlling atopic dermatitis in the majority of cases. This is to be expected as histamine has not been shown to itself induce pruritus in dogs. The registered product, chlorpheniramine, has been the
least effective in the author's hands. Some dogs will occasionally respond to cetirizine at 1 mg per kilogram or fexofenadine at 15-18 milligrams per kilogram daily.

A recent workshop of a large number of veterinary dermatologists (WVDC 2012) concluded that 45% of respondents regarded the success rate of antihistamines less than 10% and that a further 51% regarded the success rate as 10 to 20%.

It is highly unlikely that antihistamines will be able to control a case of atopic dermatitis as monotherapy. There is some role for antihistamine trials as a means of reducing the dependence of dogs on immunosuppressive agents, particularly corticosteroids.

Australian practitioners should be aware that hydroxyzine is not available in Australia and that cetirizine is its active metabolite. Combinations of antihistamines remain anecdotal; in the EU there is a registered combination product of chlorpheniramine and hydroxyzine.

**Other agents**

**Essential fatty acids.** The majority of veterinary dermatologists use essential fatty acids omega six and Omega three. There is still no consensus on the optimal dose and the optimal 3 to 6 ratio. It is highly unlikely that fatty acid therapy alone will control the signs of atopic dermatitis. Its role is as a safe measure capable in a significant proportion of cases to reduce the amount of drug that is needed to control the signs. The author favours the use of a high-quality fish oil product at one mil per 3 kg daily in divided doses.

**Interferons.** There have been some studies using high-dose and low-dose interferons, particularly recombinant feline interferon Ω, recombinant canine interferon γ or human interferon α2A. Trials have show responses but evidence is still lacking on a defined protocol.

**Methotrexate.** The antimetabolite methotrexate, in humans, is relatively common therapy for atopic dermatitis where there is a failure to control with topical treatment and the patient is reliant on corticosteroids. One protocol has been developed in a small number of dogs (Pinn et al 2012) using weekly dosing at 2.5 to 10mg of methotrexate weekly with monitoring. Significant improvement was noted after five months of therapy. The author is currently using this protocol in a single patient who was unable to tolerate corticosteroids and cyclosporine.

**Pentoxifylline** is relatively weaker inhibitor of tumour necrosis factor-α and other mediators of inflammation. It has a good safety profile and side effects are generally limited to gastrointestinal disturbances. At the WVDC 2012 workshop, 75% of respondents regarded pentoxifylline as effective in less than 10% of cases. A further 17% regarded it as ineffective in 80 to 90% of cases. When used as part of a combination, it is given at 15-20mg/kg twice a day.
**Tacrolimus topically** has been reported to be effective in local treatment of atopic dermatitis. In Australia, there is no registered formulation and the author has not enjoyed any success in the few cases he has used the compounded product. **Cyclosporine topical micro-emulsion** has shown benefits in a single published trial.

**Miscellaneous agents** with low quality evidence. Gabapentin. 61% of workshop responding dermatologists have not used this drug and a further 33% have found it to be ineffective in atopic dermatitis. Only 3% of respondents have found this drug to be effective in over 50% of cases. There is some low quality evidence supporting misoprostol, Chinese herbs, maropitant (Cerenia, ©Pfizer). The author has no experience with any of these agents.

**Future developments**

There are a wide variety of receptors that respond to cytokines and other chemical messengers produced from lymphocytes, histiocytic cells and keratinocytes. Many of these receptors respond by triggering a phosphorylation cascade utilising tyrosine kinase enzymes. Tyrosine kinase-based receptors have actions that include regulation of inflammation, haematopoiesis, cell differentiation and growth.

In human and animal medicine, tyrosine kinase inhibitors have been developed as antineoplastic agents. The canine antineoplastic agent masitinib (not available in Australia) has shown benefit in a significant proportion of refractory atopic dermatitis cases. Dogs receiving masitinib needed to be monitored for protein-losing nephropathy.

**The Janus Kinase (JAK) family of receptors (JAK1, JAK2, JAK3 and TyK2).**

JAK1 and JAK3 are major targets for inflammatory cytokines such as IL-2, IL-4, IL-7, IL-9 and IL-31. Tofacitinib, a tyrosine kinase inhibitor targeting JAK1 and JAK3, has begun use in humans for treating refractory rheumatoid arthritis.

Oclacitinib, a new veterinary JAK inhibitor preferentially targeting JAK1, has shown extremely effective antipruritic properties in dogs and has the potential to be a major breakthrough in the clinical management of canine atopic dermatitis.

While, not yet registered in Australia, Oclactinib (Apoquel ® Zoetis) was just received U.S. FDA approval and an application for approval has been lodged with the APVMA. Readers are directed to [www.apoquel.com](http://www.apoquel.com) and [www.itchcycle.com](http://www.itchcycle.com) for further information and developments.

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The JAK-STAT pathway

Activated T cells, histiocytes, and keratinocytes release inflammatory cytokines such as interleukins, growth factors, and interferons.

Cytokine bound to JAK receptor on cell membrane surface

Intracellular portion of receptor with tyrosine kinase components. TK portion auto-phosphorylates.

STAT (Signal Transducer and Activator of Transcription) monomers are phosphorylated and form dimers that bind to nuclear DNA.
Developments with allergen specific immunotherapy.

Allergen specific immunotherapy (desensitising) still remains the principal means, shown in multiple placebo-controlled studies, to be effective in reducing dog’s dependence on drugs. Multiple studies have shown that allergen specific immunotherapy will reduce the severity of clinical signs by at least 50% in approximately 2/3 dogs. It is not a substitute for drug therapy; it is part of the therapeutic combination.

Sublingual immunotherapy. At present, immunotherapy is given by injection usually administered by the client. A recent study combined with anecdotal experience of some veterinary dermatologists indicates that sublingual immunotherapy in a glycerine vehicle (given twice daily) has a similar efficiency rate to that reported for injectable immunotherapy. It is interesting that a similar efficiency rate also occurred in dogs that had failed to respond to injectable immunotherapy; the reason for this remains unclear. In Australia, we are yet to have a registered sublingual vehicle and allergen combination.

Allergen testing. There are two principal means of testing for allergen candidates to formulate immunotherapy vaccines: serum allergen testing and intradermal allergen testing. It is important to understand that they measured two different things. serum allergen testing measures circulating IgE while intradermal allergy testing measures IgE bound to cutaneous mast cells. Unfortunately, correlation between the two tests is often weak.

The Heska Allercept® IgE ELISA offers potential increased specificity through selecting for IgE capable of binding with the FCε mast cell specific receptor. The Greer IgE ELISA is also accepted to be a reliable repeatable test. Clinician should be wary of overseas laboratories offering poorly validated tests especially if they include dubious food allergen results.

Which test to use and when.

Serum allergy testing has the advantage of being able to be done in general practice. Corticosteroid withdrawal times before testing may be shorter. Serum allergen tests have recently been shown in 2 studies (West Highland White terriers and Golden Retrievers) to give significantly more positives in non-atopic individuals as compared to atopic patients of the same breed. Despite this evidence and the lack of correlation between serum and intradermal tests, two large retrospective studies have failed to show a difference in outcome of immunotherapy based on test method. These findings, however, need to be tempered with the fact that in both studies the vaccine allergens were selected by dermatologists and would have been based on clinical signs and patient history and not just numbers read off a set of results.
Intradermal testing is more specific (less false positives) but requires access to testing allergens and skill and experience to interpret the results. There is evidence that the combination of intradermal and serum allergen testing produces improved results. The author initially performs intradermal testing. If the results correlate with the clinical history and our knowledge of cross reacting allergens, a vaccine is made based on this. Should the intradermal test be negative or produce dubious results, serum allergen test is performed and, if the combined results are meaningful, a vaccine is made based on this.

It is most important to understand that a proportion of dogs with atopic dermatitis do not produce IgE and hence will be negative to both tests. As positive tests occur in non-atopic dogs, neither test should be used to diagnose atopic dermatitis but rather only used to formulate a vaccine for the management of atopic dermatitis when other causes of pruritic skin disease have been excluded and the diagnosis made on clinical grounds.

Correlates with success. Several studies have been done to determine what factors influence the success of allergen specific immunotherapy. Success appears to be independent of age, breed, number of allergens and type of allergens. Success is correlated with the skill and experience of the clinician and the amount of contact between the client and the veterinarian during the immunotherapy process.

Corticosteroid withdrawal times. A recent evidence based review has shown that there is no evidence for prednisolone withdrawal longer than four weeks before intradermal testing. The author would still lean towards a longer withdrawal time if the course of prednisolone has been prolonged. Potent topical corticosteroids, including ear drops, should be withdrawn for two weeks. Hydrocortisone cream and antihistamines should be withdrawn 7 to 10 days before intradermal testing. The length of time that depot steroid injections need to be withdrawn is unclear but it is certainly extremely prolonged. This is one of the reasons the author is of the opinion that these agents have no role in the long-term management of atopic dermatitis in dogs. Withdrawal periods for serum allergen testing are unclear and probably depend on the test method. It is wise to maximise steroid withdrawal before serum IgE testing.

Infection control.

It is absolutely vital to control yeast and bacterial infections. Dogs with atopic dermatitis suffer from re-occurring infections because of a loss of barrier function, reduced skin immunological and non-immunological defence mechanisms, increased bacterial adherence and the effects of systemic immunosuppressive drugs.

Total failure of management is guaranteed if infections are not addressed.
If low doses of corticosteroid failed to control a confirmed case of atopic dermatitis or an existing case rapidly becomes out of control, the clinician should suspect an infection.

Much is written on this topic and the reader is directed to Web-based references listed below. It is most important to understand that superficial bacterial infections such as folliculitis require at least three weeks of antibiotics at effective dose rates (cephalexin 25 mg per kilogram twice a day). Yeast infections require at least four weeks of systemic therapy.

**Antibiotic resistance.** In Australia, fortunately, we are not YET commonly finding the multidrug resistant *Staphylococcus pseudintermedius* (formerly S. intermedius) strains that are seen on a regular basis in North America and Europe. We need to be extremely vigilant to the emergence of these strains and also to follow accepted guidelines on antibiotic use so as not to promote resistance.

**Managing defects in the epidermal barrier.**

In recent years, it has become apparent that both in dogs and humans there is a defect in the epidermal barrier in atopic individuals. This can be demonstrated by electron microscopy showing defects in the lipid lamellae within the epidermis, trans-epidermal water loss studies and biochemical studies on the composition of skin lipids.

In humans, there is a significant body of thought that at least a proportion of atopic individuals may have a primary epidermal barrier defect, which initiates the whole atopic cascade. In dogs, it is unclear whether the demonstrated epidermal defects are primary or secondary.

In humans, moisturising is a cornerstone of therapy. There is limited evidence that moisturisers containing sphingolipids may be more effective than standard moisturisers. In dogs, there is no doubt there is a barrier defect in the majority of dogs and dry skin is often seen in clinical cases. What is unclear in dogs is which moisturisers are more effective than others. There are no placebo-controlled studies to demonstrate the effectiveness of moisturising in atopic dogs. However, on a logical and do no harm basis it has a huge role to play.

**Moisturisers have two properties:**

**Emollient.** These oils provide a barrier between the epidermis and environment reducing trans-epidermal water loss and providing a physical barrier to allergens. The principal examples are paraffin oil and lanolin.

**Humectant.** These agents such as propylene glycol or glycerine actively draw water into the skin by osmosis. Oatmeal has a weak humectant action.
Atopic dogs have abnormal epidermal sphingolipid levels. There are new veterinary moisturisers containing sphingolipids such as phytosphingosine (a plant-based sphingolipid). Nutriderm Conditioner® Paws Blackmores is an Australian-registered sphingolipid veterinary moisturiser.

The author's preferred moisturising regimes are:

- Paws Nutriderm Conditioner® diluted 1:2 with water and used as a spray.
- Chlorhexidine-containing leave-on conditioners (e.g., Pyohex Conditioner® Dermcare, Resichlor® Virbac) where infection and barrier issues coexist.
- Sorbolene (both humectants and emollient) applied in a perfume and alcohol-free base to non-haired skin.
- Propylene glycol 33% in water as a spray
- Alpha Keri Oil® suspended in water 1:50 as a spray.
- Some clients have informed the author that they are having some success with a pawpaw-based moisturiser available over-the-counter. While not specifically recommending this form of treatment, the author has no reason to steer clients away from something they are using that they feel is giving benefit.

**Shampooing.** There is little doubt that shampooing with antiseptic shampoo assists in clearing superficial bacterial infections. Chlorhexidine-based shampoos and leave on conditioners have been shown to persist in hair after shampooing. The other side of the coin is that, in humans, excess use of shampoos and detergents has been shown to have a detrimental effect on the epidermal barrier function. The author does not encourage shampoos beyond what is necessary to deal with or to prevent bacterial or yeast overgrowth of the skin.

**Summary.**

There have been new developments in the management of atopic dermatitis but fundamentally the pillars of treatment remain:

1. Immunosuppressive and anti-inflammatory agents to control pruritus.
2. Allergen specific immunotherapy.
3. Control of infections.
5. Detection of other flair factors such as diet, fleas and avoidable allergens.

Clinicians need to be aware that corticosteroids only comprise point 1(a) of this list and that each case is individual and will need combination therapy.
Recommended web based readings


Rosychuk RAW. Small Animal Veterinary Dermatology – A Therapeutic Update
http://www.delawarevalleyacademyvm.org/pdfs/PruriticDogCat.pdf

Zoetis www.itchcycle.com Detailed information on the latest concepts of the pathogenesis, diagnosis and treatment of atopic dermatitis.

Specific other references available by request.

The author would like to thank Dr Anne Woolley for assistance with proof reading this article.
Figure 1. Chronic atopic dermatitis showing skin thickening (lichenification) and hyperpigmentation. Complicated by bacterial and yeast overgrowth, barrier defects and chronic systemic corticosteroid use.
Figure 2. Atrophic changes on a dog’s abdomen cause by excessive use of high potency topical steroids. Note thin skin, prominent veins, light scale and “wavy” changes.

Figure 3. Localised calcinosis cutis induced by topical steroid agents.
Figure 4. Multiple epidermal collarettes typical of superficial pyoderma, secondary to atopic dermatitis
Dr Robert Hilton BVSc(Hons) MACVSc(Canine Medicine) CertVD MRCVS graduated with first class honours from the University of Melbourne in 1975. Between 1976 and 2000 he was in mixed then small animal practice, gaining a MACVSc in canine medicine along the way. Since 2001 he has been focusing is professional activities on Veterinary Dermatology, his long time professional interest.

In 2006, he successfully completed all the requirements and examinations of the Royal College of Veterinary Surgeons Certificate in Veterinary Dermatology. Rob is a member of the Dermatology Chapter of the Australasian College of Veterinary Scientists, a full member of the European Society for Veterinary Dermatology and an active member of Vetdermlist.

Rob’s particular interests include allergic skin disease (including immunotherapy) and any other skin disease relating to dysfunction of the immune system. He is the author of several articles and clinical reviews and has given a number of seminars locally and further afield. Rob’s private interests include climbing mountains on foot or by skis and trying not to fall off on the way down.

Rob’s practice is restricted to referrals and consultations in Veterinary Dermatology at the Lort Smith (North Melbourne) and Yarrambat Veterinary Hospitals. Rob can be contacted on 0433-853560 and by email on rob_hilton@yahoo.com