Corticosteroid Therapy Revisited:  
Promulgating Prudent Prednisone Policy

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INTRODUCTION:  
Judicious corticosteroid administration can be an extremely useful 
therapeutic modality for the treatment of inflammatory dermatological conditions. Unlike many other pharmacological options, the response to therapy is often quickly noticed by the client and rapidly beneficial to the patient. The dose is also easily altered. It is important to note that there is a difference between short term therapy and long term therapy. Twenty years ago, I found myself spending a great deal of time educating clients about avoiding steroid abuse. In recent years, I have found myself educating clients about the benefits of prudent steroid administration (promulgating prudent prednisone policy). Originally, I worried that I was becoming one of those “older veterinarians,” who is resistant to change. However, I realized that I have changed in many ways and instead feel compelled to offer a side of the story that is not often told in public. I would like to thank Dr. Candice Sousa for giving me the courage to repeatedly speak out regarding this topic as well as providing the permission to plagiarize the bulk of these written notes. Honestly, I have my own notes, but hers are much better. Dr. Sousa is a Diplomate of both the ACVD as well as the ABVP. She owned her own practice for years in California until she started working with Pfizer Animal Health a few years ago where she is a Senior Veterinary Specialist with their Veterinary Specialty Team. She has also recently authored an important steroid chapter in Kirk’s Current Veterinary Therapy XIV. Thanks to her, I am able to apply math and science to my clinical impressions and postulations based on my “years of experience”. Most of you already know, or were once taught, the basics of what I am about review. You may not remember all the specifics of the pharmacology, but you have witnessed the benefits of proper prednisone utilization and seen some of the side effects. I ask that you keep these in mind when comparing other therapeutic options. At the conclusion, we will review the doses of corticosteroids that I use in my practice.

WHAT ARE THEY
Steroids are compounds that are manufactured from and resemble cholesterol. Steroids are primarily produced in the cortex of the adrenal gland but other organs, such as the testicles and ovaries, also contribute to their production. Some of the metabolically active substances included in this group are the sex hormones, bile acids, and cortisone.

Corticosteroids (or corticoids) (CS) are 21 carbon steroid hormones that are primarily produced by the adrenal cortex. The major stimulus for the
adrenal cortex to synthesize and secrete steroids is adrenocorticotropic hormone (ACTH), which in turn is synthesized in and released from the pituitary gland. ACTH release is stimulated by corticotrophin-releasing factor (CRF), produced by the hypothalamus.

**Mineralocorticoids (MC)** are a CS produced primarily in the zona glomerulosa of the adrenal cortex. They exert their greatest effect on electrolyte metabolism – sodium, potassium and chloride in particular.

**Glucocorticoids (CG)** increase gluconeogenesis (i.e. cause an increase in blood sugars and liver glycogen). More than 95% of the secreted corticosteroids are considered GC. These are products of the inner zones of the adrenal cortex, the zonas fasciculata and reticularis. The primary physiologic corticoid is **cortisol**, also termed **hydrocortisone**. **Cortisone** is the inactive form of the hormone. The active form, **hydrocortisone (cortisol)**, is formed by dehydroxylation in the liver. In man, cortisol production is estimated to be produced at a rate of 10 mg/day. The daily production rate of cortisol can rise 10-fold in response to severe stress. In dogs, daily cortisol production is 0.2 to 1 mg/kg/day. No information is available for cats.

If an additional double bond is added to hydrocortisone, this results in increased glucocorticoid activity and a decreased rate of degradation. The product of this first synthetic change is **prednisone**, which is also an inactive form. Activation requires hydroxylation in the liver at the C-11 position converting **prednisone** to **prednisolone**, the biologically active form. There is good evidence that horses, and anecdotal evidence that cats, may not convert prednisone to prednisolone in the liver possibly due to a lack of 11β-hydroxydehydrogenase, making the latter a more appropriate therapeutic choice in these species.

**Methyl-prednisolone** is formed by the addition of a methyl group to **prednisolone** at C-6. When a fluorine molecule is added to **hydrocortisone** at the C-9 position, this results in increased glucocorticoid but also marked mineralocorticoid activity (**fludrocortisone, Florinef**.) Further modification of this molecule by methylation at C-16 results in **triamcinolone, dexamethasone, or betamethasone**. These molecules have high GC but low MC effects.

Pharmacologically, the duration of action of synthetic GC is determined by the structure of the drug molecule. This in turn determines potency and the dosage given. Generally, the larger the dose and the more potent the glucocorticoid, the longer the drug will have an effect. (Table 1). Clinically, the route of administration and the water solubility of the carrier substance are usually more important factors affecting the duration of action. Oral GC are generally formulated as a free base or an ester that is digested to the free base and subsequently absorbed. Parenteral GC (i.e. injectable) are usually esters of acetate, diacetate, sodium phosphate or sodium succinate. The sodium phosphate and succinate esters are very water-soluble and rapidly attain serum levels even when given intramuscularly. In contrast, the acetate or diacetate esters are poorly water-soluble and are slowly absorbed at a continuous low level of glucocorticoid for several days to weeks. This slow absorption may greatly prolong the adrenal suppressive effects. Concern regarding adrenal suppression
is the basis for the recommendation of alternate day dosing of short acting oral GC when long-term treatment is needed.

HOW DO THEY WORK?

Glucocorticoids influence a variety of body functions because they affect most cells in the body. They exert most of their actions by blinding to intracytoplasmic steroid receptors which are then transported to the nucleus where they bind to cellular DNA and alter gene expression.

In general, GC alter carbohydrate, fat, and protein metabolism; fibroblast proliferation (important for wound healing); the inflammatory response; electrolyte and water balance; synthesis of red blood cells; central nervous system function; gastric acid production; muscle strength and function; the immune system; and a variety of other metabolic processes. As stated earlier, they seem to have some effect on every metabolic process.

One of the most important medical uses of corticosteroids is for their anti-inflammatory effects. Inflammation comprises the changes in the tissue in response to injury. There are 4 classical signs of inflammation: pain (dolor), heat (color), redness (rubor), and swelling (tumor). When tissue injury occurs, whether it be by bacteria, trauma, chemicals, or any other phenomenon, histamine and other humoral substances are liberated by the damaged cells into the surrounding fluids. This causes an increase in local blood flow and also increases the permeability of the capillaries, allowing large quantities of fluid and cells to leak into the tissue.

Glucocorticoids: 1) Stabilize the membranes of lysosomes so that they rupture with difficulty. This helps prevent the usual tissue damage and destruction that occurs when lysosomal enzymes are released. 2) GC also decrease the production of bradykinin, which is a potent vasodilating substance. This decreases the permeability of the capillary membrane, which then prevents protein leakage into inflamed tissues. 3) GC minimize the inflammatory response through the action of lipomodulin, which inhibits phospholipase A2 which normally converts membrane phospholipids into arachidonic acid (AA), a pro-inflammatory product. The decrease in AA limits available precursor molecules for lipoxygenase and cyclo-oxygenase to produce the AA-derived mediators of inflammation. 4) Lastly GCs inhibit the expression of adhesion molecules on the endothelial cells (particularly ELAM-1 and ICAM-1) and thereby interfere with the movement of leukocytes from the vasculature into inflamed tissues. This is the cause of the commonly noted leukocytosis seen with GC administration.

Glucocorticoids block the inflammatory response to an allergic reaction exactly the same way that they block other types of inflammation. The basic allergic reaction between an antigen and antibody is not affected and even some of the secondary effects of the allergic reaction, such as the release of histamine, still occur. However, the subsequent inflammatory response is responsible for many of the serious and the sometimes fatal effects of the allergic reaction, administration of GC can be lifesaving.

A complete understanding of immunosuppression induced by CS is not known. The effect is more pronounced on the cellular arm than the humoral arm.
of the immune system. Whereas allergen specific immunotherapy (ASIT or "allergy shots" seem to affect the humoral arm). However, the two arms are interrelated. GCs have minimal effects on plasma immunoglobulin concentrations but can modulate immunoglobulin function. For example, opsonization of bacteria is inhibited.

At immunosuppressive doses (the exact dose has not been scientifically determined) GC can induce decreased production of intracellular signaling cytokines such as IL-1, IL-6, TNF-α, IFN-γ and granulocyte colony-stimulating factor (GM-CSF). These are the signals that T and B lymphocytes use to communicate and will result in an alteration of the immune system at multiple stages. In general, there is a decline in the number of leukocytes at the site of infection or inflammation and an interference with their function.

Glucocorticoids cause marked changes in leukocyte numbers and distribution. A mature neutrophilia is a characteristic component of a physiologic stress response and to exogenous GC treatment. This occurs from a combination of an increased release of mature neutrophils from the bone marrow, decreased margination and decreased migration of neutrophils out of the blood vessels, resulting in a prolonged circulatory half-life.

In contrast, the administration of GC leads to a decreased number of circulating lymphocytes, eosinophils, monocytes and basophils. Lymphopenia results from a redistribution of circulating lymphocytes to nonvascular lymphatic compartments such as the lymph nodes. As lymphopenia is not a marked or consistent component of the feline stress leukogram, this species is considered relatively steroid resistant.

Systemic glucocorticoids are probably the most commonly used drugs in veterinary medicine and are undoubtedly the most commonly used and abused drugs in veterinary dermatology. Their intended and appropriate use in veterinary dermatology is for their anti-pruritic, anti-inflammatory, and immunomodulatory properties. A beneficial response is seen in animals with allergic disorders, inflammatory skin diseases, and autoimmune or immune-mediated dermatoses. The specific GC affects that will occur with their therapeutic use are summarized below and need be considered particularly if ongoing therapy will be needed.

1. suppress the release of ACTH by the pituitary gland and therefore suppress the release of corticosteroids by the adrenal cortex
2. reduce the number of circulating lymphocytes through redistribution and suppress T lymphocyte function
3. reduce the number of circulating eosinophils
4. help maintain cell membrane activity
5. inhibit macrophage function
6. suppress antibody production
7. inhibit the release of endogenous pyrogen (IL-1)
8. depress prostaglandin and leukotriene synthesis
9. alter the complement and kinin cascades
10. interfere with leukocyte migration and adhesion
11. suppress lysosomal release from neutrophils by stabilizing lysosomal membranes
12. suppress phagocytosis
13. reduce fibroblast activity resulting in delayed healing and thinning of the skin
14. effect enzyme actions and other cellular functions

SIDE EFFECTS

As with any other class of drugs, corticosteroids have clear value when used to treat a disorder for which they have proven therapeutic benefit and when administered at the appropriate dose, frequency, and duration of administration. Their recognized anti-inflammatory and immunosuppressive effects make them a valuable addition to veterinary medicine. There is no question that side effects do occur with GC therapy. However, excessive concern for these may prevent the appropriate use of this class of drugs when they are indeed indicated. Yet CS tend to be shunned by some practitioners who worry about iatrogenic suppression of the hypothalamic-pituitary-adrenal (HPA) axis, immune suppression and other side effects.

The benefits of any therapy must always be weighed against the possible and / or probable side effects. It is well-recognized that the excessive use of GC can be associated with many adverse effects. The anti-inflammatory and immunosuppressive actions of GC, though desired for their therapeutic effects, may facilitate the establishment or spread of other infectious or parasitic diseases. As a result, dogs treated with GC have a tendency to develop secondary bacterial infections of the skin, urinary tract or respiratory tract. Urinary tract infections have been documented in 18 to 39% of dogs who are treated with 0.28 to 0.8 mg/kg of GC for more than 6 months.

The most serious side effects of CS are related to prolonged use of large doses which may suppress the HPA axis. The effects of chronic elevations in glucocorticoid levels are readily seen with naturally occurring hyperadrenocorticism (Cushing’s disease). Unfortunately those same problems can be created by overuse of GC by the veterinarian and / or client, even when administered on an alternate-day basis. These high levels of exogenous steroids can result in hyperglycemia, fat redistribution, decreased skin elasticity, atrophy of the skin, poor wound healing, a pendulous abdomen secondary to a redistribution of body fat, poor quality coarse hair, alopecia (e.g. hair loss from breakage and failure to regrow), comedones (e.g. follicular plugs or blackheads), a variety of bacterial infections (especially of the bladder and skin) and even calcinosis cutis (e.g. mineral deposits in the skin). Localized dermal and adnexal atrophy following subcutaneous and occasional intramuscular repositol GC have also been reported. If the glucocorticoid used also has mineralocorticoid effects then polyuria (e.g. production of an increased amount of urine) and polydipsia (e.g. drinking an excessive amount) may also be present.

Iatrogenic secondary adrenocortical insufficiency is a side effect that can be seen after withdrawal of the glucocorticoid therapy. When an animal is treated
with a glucocorticoid, the adrenal gland, in natural response to the effect of the exogenous GC on the HPA, will stop producing steroid hormones for some period of time. The duration of this suppressive effect is known to be dependent on the type of steroid and duration of treatment. However, the precise degree and length of suppression in any individual dog can not be predicted. Generally speaking the longer the therapy and the higher the dosage, the longer the time before natural production of steroid hormones resumes by the adrenal gland. This resultant lack of endogenous (physiological) GC is the cause of weakness and possible circulatory collapse that can occur with cessation of exogenous glucocorticoid therapy.

One intravenous injection of dexamethasone at 0.1 mg/kg, which equals approximately 3 mg of hydrocortisone (cortisol) or 3X the highest daily natural production can suppress the HPA for 32 hours in a healthy dog.

**USE OF CORTICOSTEROIDS IN VETERINARY DERMATOLOGY**

Cortisone and ACTH were first used to treat a variety of inflammatory dermatoses in humans in the 1950s. The major indications for their use in veterinary dermatology include treatment of allergic or pruritic dermatoses, autoimmune dermatoses and feline eosinophilic granulomas.

The use of glucocorticoids is an art that requires the clinician to skillfully integrate the many details about the patient, the owner, and the disease so that an appropriate type and dose of glucocorticoid can be used. Changes and adjustment in dosages and even the type of corticosteroid used must be made depending on the response of the disease and the side effects that develop. Physiologic doses of GC are those that approximate the daily cortisol production by normal individuals. In dogs, daily cortisol production has been reported to be 0.2 to 1 mg/kg/day. A pharmacologic dose of GC exceeds physiologic requirements. There is no optimal dosage established in the veterinary literature and each case should be treated individually. There are guidelines, however, which serve as a good starting point. Using oral prednisone or prednisolone (or methylprednisolone) in dogs as the drug of choice the recommendations are:

- **Antipruritic doses**: 0.5 mg/kg/day for 7 to 10 days then decreased to lowest effective dose
- **Anti-inflammatory doses**: 1.0 - 1.5 mg/kg/day for 7 to 10 days then decreased to the lowest effective dose
- **Immunosuppressive doses**: 2.0 – 6.0 mg/kg/day for induction then decreased as possible to maintain control of the disease

Compared with dogs, cats seem to require about twice the dose of oral GC to achieve the same effects.

**LONG TERM USAGE OF GLUCOCORTICOIDS**

This section now presents Dr. Sousa’s personal beliefs regarding a safe dose of GC used long term as there is no true evidence-based formula. Every animal and every disease condition differs.
The following is her formula for dogs. Starting with the fact that dogs manufacture 0.2 to 1 mg/kg/day of cortisol and need this to survive, and using a 40 kg (88#) dog as an example:

\[ 40 \text{ kg} \times 0.4 \text{ mg} \times 365 \text{ days} = 5840 \text{ mg of cortisol produced / year} \]

Since prednisone is considered to be about 4 times as potent as hydrocortisone (cortisol), dividing 5840 by 4 is 1460 mg of prednisone. Thus, this 40 kg dog would “see” in a normal physiologic state approximately 1460 mg of prednisone (or prednisolone) / year

From these calculations I have I have developed what I called my “safe annual steroid dose” formula:

\[
\text{BW (kg)} \times 30 = \text{mg prednisone / year}
\]

or

\[
\text{BW (lb)} \times 15 = \text{mg prednisone / year}
\]

This number (30 is based on a combination of several publications reporting the side effects of GC as related to dose and on over 10 years of using this in my own practice and seeing the safe use.

Again considering the 40 kg dog I would calculate

\[ 40 \times 30 = 1200 \text{ mg of prednisone to be the “safe annual steroid dose”}. \]

This value is less than the range of what is considered physiologic for that dog.

If this dog required more than what I believed to be the “safe annual dose” of prednisone or prednisolone to control its dermatologic disease (i.e. pruritus from allergies or atopic dermatitis) then I would either add a second medication in an effort to decrease the amount of GC needed or change medications (e.g. to cyclosporine.) Steroid treatment protocols generally begin with higher doses and then are tapered but again looking at these calculations in this way can be helpful guides.

If the dog needed more than the “safe annual steroid dose” and the owner declined further diagnostic work up or other therapy then I recommend monitoring for weight gain and urinary tract infection as these are the most prevalent side effects with ongoing GC therapy. First, I would discuss in detail my recommendations for feeding and have the dog weighed to make sure that it wasn’t gaining weight. I would also perform a cystocentesis for urinalysis and urine culture and sensitivity test. It is critical that the urine be cultured as in dogs receiving steroid therapy due to the dilution of the urine and the anti-inflammatory effects of the steroids, actual bactiuria or the suggestive urinalysis findings of infection my not be detected. Although it would be expected that the urine specific gravity to be low (about 1.012) if there was protein or glucose in the dilute urine a serum chemistry to assess for any early renal disease or diabetes would be indicated. I would expect the alkaline phosphatase and alanine
aminotransferase (ALT) to be elevated as well as for the CBC to reveal a stress leukogram.

In summary, the rational use of glucocorticoids in veterinary dermatology requires the clinician to be familiar with the pharmacological and physiological effects and side effects of steroids as a class and the individual formulations used in their practice. Glucocorticoid use should be limited and kept to a minimum by using adjunctive therapies (such as antibiotics, antihistamines, topical therapies, etc.) whenever possible. A diagnosis should be made prior to determining the therapeutic regimen. And long-term therapy should be monitored with frequent examinations and laboratory testing as indicated.

Table 1
Comparison of Water-Soluble Glucocorticoids Used in Dogs and Cats

<table>
<thead>
<tr>
<th>Generic Drug</th>
<th>Relative Mineralocorticoid Potency</th>
<th>Relative Glucocorticoid Potency (Anti-inflammatory Potency)</th>
<th>Equivalent Dose (mg)</th>
<th>Plasma Half-life (Hours)</th>
<th>Biologic Half-life in Humans (Hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrocortisone</td>
<td>1</td>
<td>1</td>
<td>20</td>
<td>1</td>
<td>8-12</td>
</tr>
<tr>
<td>Prednisone / prednisolone</td>
<td>0.8</td>
<td>4</td>
<td>5</td>
<td>(?) 1</td>
<td>12-36</td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>0.5</td>
<td>5</td>
<td>4</td>
<td>1.5</td>
<td>12-36</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>0</td>
<td>3 - 5</td>
<td>4</td>
<td>(?) 4</td>
<td>24-48</td>
</tr>
<tr>
<td>Triamcinolone acetonide</td>
<td>a</td>
<td>15</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flumethasone</td>
<td>0</td>
<td>30</td>
<td>0.75</td>
<td>2</td>
<td>35-54</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>0</td>
<td>30</td>
<td>0.6</td>
<td>(?) 5</td>
<td>&gt;48</td>
</tr>
<tr>
<td>Betamethasone</td>
<td>0</td>
<td>30</td>
<td>0.6</td>
<td>(?) 5</td>
<td>&gt;48</td>
</tr>
</tbody>
</table>

a Triamcinolone acetonide has a greater potency (approaching that of dexamethasone)

“A Dunny’s Guide to Dermatitis” has been derived from Dunbar Gram’s many years of practice and thought, but only a relatively few days of hard science and math. When faced with a pruritic patient, a few important questions arise. If I am running behind in my busy practice, the staff is trained, to give me a “one liner” history (duration, historical seasonality and steroid responsiveness). Naturally the physical exam, a more in-depth history and ancillary tests are also important, but the “one liner” is like a “pick up” line when I walk in the exam room.

If a pruritic and inflamed patient is not steroid responsive, I will evaluate the dose of steroids that had been used and compare to anti-inflammatory doses discussed in Dr. Sousa’s notes. Like Dr. Sousa, I find many newer veterinarians do not seem to have the training or the desire to use steroids appropriately. If the dose has simply not been high enough, I will consider a higher dose for short term therapy for the benefit of the patient. The long term management is a different lecture. If the dose has been adequate, but the pruritus has not
subsided, I worry that the patient may have a pruritic dermatitis that is not steroid responsive or that perhaps the symptoms have become refractory to the once beneficial particular steroid.

In the latter situation as well as “routine allergic patients”, I often use Dexamethasone Sodium Phosphate (DexSP): 4mg/mL injection for horses which is equivalent to 3mg Dexamethasone/ml. Based on the above chart, 0.75 mg of Dexamethasone (Dex) is equivalent to 5mg of pred. My standard **DexSP dose is 1 mg/10 pounds** SQ or IV or 0.75mg Dex/10 pounds, (0.75mg Dex/4.54 kg X 5mg pred/0.75mg dex = 1.1mg Pred/kg). As stated earlier, the anti-inflammatory dose of pred is 1.0-1.5mg/kg/day, for 7-10 days. The anti-inflammatory dose is approximately 2-3 times the antipruritic dose. However, most of my pruritic patients are also inflamed. In these cases, I **simply** give one injection of Dex SQ to “break the inflammatory cycle” and often do not need any more steroids while utilizing other medications. If more steroids are needed, I will then start oral prednisone 1-2 days latter, at a standard antipruritic dose of 0.5mg/kg every 2-3 days. If the inflammation is severe, I will use a higher dose. For **convenience** sake, I often simply have the clients start the pred pills the day after the injection. For practical reasons, I typically administer the dex subcutaneously, but will consider intravenously (IV) if necessary. I use the IV route if the client and patient need the quickest response possible (a dinner party that evening or their therapist has cancelled) or if the client is uncertain regarding the response to steroids in the past. In the latter case, we call the client 24 hours later to document the response. Clinically, this technique works well in patients who have been well controlled with immunotherapy and/or other forms of medical therapy, but have suffered an exacerbation.

I emphasize the words **simply** and **convenience** because the patient’s primary care giver (the client) is often burdened with many other tasks. I feel this approach is both medically appropriate and helps prevent care giver burnout as well as many financial concerns. I use the term emotionally and financially exhausted when I write my referral letters to describe clients that are overburdened with caring for an allergic pet. The doses were arrived at by comparing it to what would be necessary if prednisone was utilized, but trying to avoid the mineralocorticoid side effects. I justify its use by comparing the dose used in a high dose dexamethasone suppression test. My naïve thought is that if I had been taught to use the same drug at a similar dose for a diagnostic test in a diabetic ketoacidotic cushings suspect (high dose dexamethasone suppression test), without worrying about the side effects, then why not consider it for therapeutic reasons.

For example, a 40 pound (18kg) dog receiving a high dose dex test, based on the 0.1mg/kg or 1mg/kg IV or IM, would received 1.8-18mg dexamethasone or 0.6-6.0ml of DexSP. The same size dog that was itching would receive 1.0ml of DexSP. One of my associates actually commonly utilizes ½ of my dose and has good results. A “Dunny’s Guide” to mathematical estimation implies that this dose of dexamethasone should not be repeated more than every 22 days (based
on dogs normally manufacturing cortisol at the low end of 0.2mg/kg/day. In my practice, before I had “done the math” I typically had been telling my clients to do this as seldom as possible, but if one injection a month works for them, then it is likely to be safe. Using Dr. Sousa calculations for a safe dose of glucocorticoids used long term (based on cortisol production of 0.4mg/kg/day), it would be every 33 days. I do realize that the patient is also producing cortisol. Adding the different numbers together is complex, considering the patient’s own production varies from day to day. However, if we utilize conservative endogenous cortisol production numbers, the figures proposed by Dr. Sousa seem quite reasonable. If we use the “high end of endogenous cortisol production” then this dex dose is equivalent to 4-5 days of endogenous cortisol.

Dr. Sousa and I studied dermatology at two totally different universities, practiced on two different coasts for many years and seem to have reached a similar conclusion. I call it “convergent evolution.” Corticosteroids can be used safely for the benefit of our patients and clients. You intuitively know this. As with any drug, the potential for side effects exist and should be considered when comparing therapeutic options. Oral and short acting injectable corticosteroids quickly exert their beneficial effects thus enable rapid dose alteration or reinitiation. They should not be overlooked as a therapeutic option for both acute and chronic therapy. In my opinion, they are the treatment of choice to gain control of an itchy and inflamed allergic patient.

References from Dr. Sousa


Sousa, C. Kirk’s Current Veterinary Therapy XIV, Elsevier: 2009: PAGE 400