10 THINGS EVERY PRACTITIONER SHOULD KNOW

ABOUT CANINE EARS

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INTRODUCTION

The 2007 data from (VPI) Veterinary Pet Insurance lists ear infections as the number **one** reason that dogs went to veterinarians in 2007 in North America and the eighth most common reason that cats went for veterinary care. These data are similar to the 2005 data, and virtually all other surveys done in veterinary medicine over the past 20 years. In addition, data from Banfield Pet Hospital in 2011 indicate that otitis was the second most common diagnosis made in their large number of affiliated hospitals in the United States.¹ Obviously, veterinary practitioners see a lot of patients with otitis externa.

There are many important concepts about otitis that can literally make the difference in practice. Knowledge can change your entire attitude about dealing

with ear disease. Instead of cringing when the appointment schedule reflects an ear case, you can be smiling. You can make a difference if you consider these 10 important points about canine ears and how they affect your ability to manage otitis externa.

KEY POINT #1 Anatomy and Physiology (Structure and Function) I'm sure that these words send needles up and down your spine, BUT you really can't understand how to manage a patient with ear disease until you understand what the ear is all about.

ear canal. These variations will affect predilection for disease, diagnosis, and treatment. For example, it can be very difficult to fully examine the external ear canal of an Irish setter, for it can be very long!

The external ear canal consists of skin overlying the auricular and annular cartilages. It has a vertical component and a horizontal component. The auricular cartilage includes the cartilage of the pinnae, which then converges to form the opening of the ear canal and the vertical component of the ear canal. The annular cartilage is a 1-2 cm long, tubular section of cartilage that extends from the auricular cartilage at the base of the vertical ear canal to the temporal bone. The auricular cartilage overlaps the annular cartilage and is connected to it with a fibrous band, which

allows for flexibility in movement.

Anatomically, the vertical canal is more open and larger in volume than the horizontal ear canal. There is a depression or pocket at the point where the auricular and annular cartilages overlap (i.e., the "opening" of the horizontal canal). The entrance to the horizontal canal is often elevated and requires manipulation of the otoscope in order to pass into the horizontal canal. There is actually a fold of skin (overlying cartilage) on the dorsal aspect of the canal that must be bypassed in order to slip the otoscope into the horizontal

ear canal. Mechanical trauma (e.g., during otoscopic examination) of this fold seems to startle the patient and may result in poor tolerance with otoscopic examination.

The skin lining the ear canal has sebaceous glands and apocrine (i.e., ceruminous) glands throughout the length. Sebaceous glands are located in the superficial part of the dermis with the apocrine glands located deeper. These apocrine glands can open directly onto the surface of the skin or in the hair follicle. Hair follicles are found throughout the length of the ear canal in most breeds, but there is breed variation as to the type of follicles and their density.²

What does a normal canine ear canal and tympanic membrane look like?

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The ear consists of the pinna, the external ear canal, the middle ear and the inner ear. There are major variations in the anatomy from breed to breed, especially with respect to the length and diameter of the external

The tympanic membrane

The tympanic membrane is at the proximal end of the external ear canal. On otoscopic examination, the tympanum appears as a vertically aligned structure, but it actually is sloped at approximately a 30-40° angle, with the top closer to the viewer. The tympanic membrane consists of two parts. The pars tensa is the tightly stretched, clear to slightly opaque whitish section of the tympanic membrane that is tightly stretched across the temporal bone. Embedded within the pars tensa is the handle (manubrium) of the malleus, the largest ossicle of the middle ear. The malleus is curved, with the concave aspect facing rostrally. The pars flaccida is located dorsal to the pars tensa. It is loosely attached and may "bulge" out in the canal. It occasionally may be seen moving with respiration, in a movement that resembles the bulging throat of a bullfrog!

The tympanic membrane is generally transparent, which allows a view of the white, bony structures of the middle ear, including the opening into the ventral cavity (i.e., bulla) of the middle ear and to a lesser extend some of the bones of the promontory, part of the temporal bone containing the inner ear (Figure 1a).

The tympanum in the cat is much more transparent, and thus is often thought to be absent by inexperienced viewers (Figure 2). The malleus is straighter than in the dog and the pars flaccida is generally not visible. Cats also have a bony septum in their middle ear that runs rostral to causal, and this septum obstructs view of the opening into the bulla or ventral cavity in the cat.

The middle ear

The middle ear may be partially visualized through a transparent tympanic membrane on ototscopic examination. The middle ear is comprised of three sections, the epitympanic recess, the tympanic cavity, and the ventral cavity or bulla. The middle ear contains ossciles (malleus, incus, stapes), some muscles (e.g., tenser tympani), vessels, and nerves, including parasympathetic nerves, sympathetic nerves and others (e.g., facial nerve).

The inner ear

The inner ear is located within the promontory, a bony protuberance of the petrous temporal bone (Figure 1b). It includes the organ of hearing (i.e., the cochlea) and the vestibular apparatus. There are two communications with the middle ear, the vestibular (oval) window to which the stapes is attached, and the cochlear (round) window that separates the middle ear from the cochlea. The promontory is located directly behind the tympanic membrane, from approximately 11:00 to 2:00 as seen on a clock.



Figure 1a. Normal canine tympanic membrane.



Figure 1b. Skull image (same perspective as 1a). Prom = promontory, vent = ventral cavity (bulla)



Figure 2. Normal feline tympanic membrane.

KEY POINT #2 Pathophysiology of canine ear disease

What and why ear disease occurs in dogs

It is very important to understand why the ear does what it does in the face of inflammation. Dr. John August first recommended dividing the pathogenic factors of otitis as follows³:

- **Predisposing factors:** these conditions "set the ear up" for inflammation. They include conformational changes, behavior and previous treatments.
- Primary factors: are those conditions that initiate inflammation in the ear. They include allergic diseases, foreign bodies, ectoparasites, autoimmune and other inflammatory skin disorders, and trauma.
- Perpetuating factors: keep the inflammatory process active and often make it significantly worse. Perpetuating factors include bacterial infections, yeast infections, hyperplastic changes and otitis media.

Simply put, there is a "WHAT" and a "WHY" when dealing with ear disease. Veterinarians must address both the "what" and "why" or ear problems will fail to resolve or recur.

What is the role of earwax?

Earwax is a very important component of the ear. It consists of a mixture of apocrine (i.e., cerumenous) gland secretions, sebaceous secretions and epithelial cells. Sebum functions to provide lubrication in the ear canal, hydration, and as protection, both physical protection and to regulate microorganisms in the ear canal. There is a natural movement of sebum outwardly in the normal ear, facilitating natural cleaning and removal of sebum.

The lipid portion of earwax is derived from sebaceous glands and contains various waxes and fatty acids, many of which exhibit bacteriostatic and fungistatic

properties.⁴ Simply put, the lipid portion of cerumen is largely responsible for controlling microorganisms. The apocrine secretions (from "ceruminous glands") produce a water-based secretion that contains phospholipids and immunoglobin A (IgA), which also contributes to the defense of the ear. Epithelial cells contribute to the texture and consistency of the wax. Increased epithelial cell production in the ear will produce a thicker, pastier earwax.

Pathologic changes in the ear

Once the otitis has begun, certain pathologic changes occur that initiate a cascade of events that make the ear more hospitable for microorganisms and reduce the lumen size of the ear canal.⁵ Inflammatory changes are accompanied by pain, and progressive disease leads to loss of hearing. It has been determined that the pathologic changes in the ear do reduce acuity of hearing, and that some of that hearing loss is reversible, as the pathologic changes are reversed.

With inflammation comes edema and infiltration of inflammatory cells. Secretion of various growth factors will result in epidermal hyperplasia and hyperkeratosis, resulting in microfissures on the surface of the skin and increased deposition of cornified keratinocytes in the lumen of the ear. As inflammation progresses, there is fibroplasia (i.e., fibrosis) of the dermis and subcutis. Chronic inflammation of the cartilage will result in ossification of these structures.

Within the dermis, it has been shown that apocrine glands increase in size in otitis externa. The intense inflammation around apocrine glands, combined with epidermal hyperplasia (papillary proliferation) results in occlusion of ductal openings on the skin and hair follicles and may predispose the gland to rupture. When the apocrine glands rupture, there is infiltration of lymphocytes, neutrophils, mast cells, and macrophages into surrounding tissue. It would appear that the disruption of these glands significantly contributes to the inflammation, pain and fibrosis. Interestingly, sebaceous glands remain the same size, even in chronic otitis externa, though there is a qualitative change in sebum production. The net result is decreased lipid content of cerumen in ears with otitis externa. Since lipid secretions of the skin have barrier and antimicrobial functions, there is

speculation that this change further contributes to secondary infections in otitis externa.

Finally, biopsy of the ear canal in chronic otitis externa will reveal folliculitis and furunculosis. With furunculosis there is release of keratinized materials into the dermis, and the net result is a foreign body-type reaction. Furunculosis is common in ceruminous otitis externa associated with familial seborrhea of the American cocker spaniel.

KEY POINT #3 Cleaning the Ears

Basics of why and how to flush and clean canine ears

Starting with a clean ear makes a huge impact on topical management of otitis. This does not mean filling the ear with cleanser then wiping out the excess. The cleaner you get the canal, the better the chances that topical medication will work. Thorough cleaning of the ear canal will:

1. Remove debris/material that causes irritation and inflammation

Foreign material can cause irritation of the ear canal. However, accumulations of earwax (i.e., ceruminoliths) can also irritate the canal and complicate management of otitis. In addition, thorough cleansing of the ear canal will remove some of the infectious agents that act as perpetuating factors.

2. Remove debris that interferes with movement of medication deep in the ear canal

It is very common for wax, debris, and inspissated pus to completely obstruct the ear canal. In some cases, the material will become packed deep in the canal over the tympanic membrane. This material can act as a "plug" which will obstruct movement of medicine into the area. It may also serve as a nidus for reinfection, even if the therapy is effective in the outer aspects of the canal.

3. Remove debris that can interfere with the activity of some active ingredients in the canal

The efficacy of some topical medications, such as polymyxin B sulfates and aminoglycosides, is dramatically reduced in the presence of pus. So, it is to your patients' and clients' advantage to start with a thorough ear cleaning.

It is your choice, as the veterinarian, regarding the type of ear cleaning you select. For mild cases, it may suffice to use a basic technique of filling the canal with cleanser, massaging the canal, then removing excess cleanser and debris with a cotton ball — repeated until otoscopic examination confirms that most of the debris has been removed. However, severe or chronic cases warrant a more aggressive approach. In those cases, the authors recommend a deep and detailed ear cleaning (or ear flush) with the patient under general anesthesia.

Ear cleaning techniques include:

- Infusion/massage with removal using cotton balls
- 2 Waterpik[®] or AuriFlush[®]
- Cleaning with special catheters, such as gastric (duel-lumen) evacuation tubes
- 4 Deep ear cleaning/flushing

Of course, the mechanical removal of debris with alligator forceps or ear loops is often a very useful method to remove thicker debris or material packed deep in the canal. Care should be used to avoid damaging the tympanic membrane when these tools are used.

KEY POINT #4 Use Appropriate Diagnostics

What diagnostic tests are important for practitioners to use?

Collection of a thorough dermatologic history is crucial to evaluate the patient for the primary factor (i.e., underlying cause, or the "why"). When it comes to managing the perpetuating factors (e.g., current infections), it is helpful to know what medications have been used in the past. This includes amounts, frequency and duration of each treatment. The physical examination includes inspection and palpation of the entire ear canal. The mouth should be opened wide to evaluate for bullae pain, one possible indicator of otitis media.

Otoscopic examination

Otoscopy should be performed on all cases, and repeated at each re-evaluation of the patient. Both ears should be examined, even if the client believes the problem is unilateral (one ear is often worse that the other). Handheld otoscopes are very useful and there are different styles that have different levels of magnification. Several commercial video otoscopes are now affordable and they provide much better visualization down the canal.

Otoscopic technique

The otoscopic examination is easier when the anatomy is understood. The otoscope is rested in the intertragic notch (fissure), which can then be used as a fulcrum as the otoscope is advanced. This placement seems to calm the patient as the otoscope is moved down the ear canal. The canal should always be visualized as the scope is passed. The clinician should hold onto the pinna to facilitate restraint and to manipulate the ear canal during the examination. As the scope reaches the ventral-most aspect of the vertical canal, the pinna is gently pulled laterally (outwardly) and down to straighten out the external ear canal. There is a ridge of cartilage located at the junction of the vertical and horizontal canals, which is often traumatized during examination. Any pressure on this ridge seems to cause discomfort to the patient and will disrupt the examination, so it is very

important to pass this area as carefully. To do so, the otoscope cone should be passed along the canal as ventral as possible, and then the cone can actually be used to "lift" up the ridge of cartilage. The horizontal canal will be visible and the scope can then be slid into that area. Understanding the anatomy is also critical to facilitate proper treatment and application of medications, as will be discussed in the therapeutics session.

Cytologic evaluation is a key diagnostic procedure in patients with ear disease. Samples should be collected from both ears at the initial examination and at every recheck examination because things do change in the ear canal. Samples are usually collected by passing a cotton-tipped applicator gently into the ear canal and advancing it while performing the same traction maneuver used to facilitate passing an otoscope down the canal (pulling the pinna outwardly and down to straighten out the canal). As soon as resistance is encountered while passing the swab, it should not be advanced further. The swab is then gently rotated, then withdrawn and used to make "roll preps" on a clean glass microscope slide. The slide is then stained (with the stain of choice in your practice) and examined under the microscope (Figure 3).

A couple of tips for cytology:

- Use a clean glass slide (wipe the slide with a gauze to ensure it is clean).
- 2 Use firm pressure to roll the swab (this will increase the adhesion of material onto the slide).
- Dip or place slides into jars of stain or fixative very gently. Do not move the slide up and down after the initial placement; you may gently sway the slide in the jar to distribute stain, if needed.
- Q Rinse the slide immediately after the thiazine (blue) stain (when using Diff-Quick stain), but do not let the rinse water hit the sample directly.
- Air dry or use a blow dryer with the heat coil turned off to rapidly dry the slide. Do not overheat (or the sample may be ruined).



Figure 3. Material from both ears can be placed on one slide for easy comparison.

A normal ear may contain low numbers of bacteria (usually cocci) and yeast. However, the absolute number (e.g., number of organisms per field of view) is not important, since we all make slides differently. The cytologic findings are correlated with the clinical findings and a decision made to treat is based on all data.

Bacterial culture is indicated when:

- the cytology shows a uniform population of rodshaped bacteria (probably *Pseudomonas* spp.)
- 2 the infection has failed to respond to "standard-of-care" therapy
- When you have a known resistant organism (generally based on previous culture results)⁶

Diagnostic imaging of the ear can also be very helpful, especially in dogs with chronic disease. Radiography may help identify bony changes in the bullae that might reflect otitis media. Computerized tomography (CT) provides much better detail and is the authors' imaging of choice, due to relative low cost and high degree of detail provided. Magnetic resonance imaging is also very helpful, however, the cost is significantly higher than CT.



KEY POINT #5 Topical Therapy For Canine Ear Disease

How to use topical therapy in the ear appropriately

Topical therapy is considered sufficient to manage most cases of otitis externa, if the principles of therapy discussed earlier are followed.

Overall, the success of topical therapy depends upon:

- The efficacy of the active ingredients
- Removal or reduction of obstructions (hair, exudate) in the ear canal
- The formulation of the medication
- The technique, frequency and duration of administration
- The volume of medication instilled at each treatment
- The integrity of the tympanic membrane

Otic medications are most often in the form of an ointment (emulsions of lipid in water) or as a solution (aqueous or other carriers) **(Table 1)**. Emulsions containing lipids will enhance penetration of the active ingredient into the skin of the ear, however, most of these ointment formulations are so viscous, that they fail to penetrate **down** deep into the ear canal. They are especially ineffective in the presence of a heavy growth of hair in the canal. Less viscous medications are more likely to allow medication to distribute deeper into the canal, especially when there is significant hair in the ear canal or when the canal is hyperplastic.

In cases when topical therapy is used, the owners *must* be educated about application of medications. This should include having the owner instill medication, **in the presence** of the veterinarian or technician. Owners should be taught to massage ears for 15-30 seconds after instilling medication and to use proper amounts of medication. Once daily treatment is generally sufficient for most cases of otitis, though severe infections *may* benefit from twice daily treatment. Treatment should be continued until there is no clinical or cytologic evidence of active disease. Though in conflict with label instructions on most otic products, the authors recommend a minimum treatment time

(with topical therapy) of 30 days. This extended time is necessary to completely clear the infection. Shorter treatment times will often decrease the severity of infection and result in clinical improvement without completely eliminating the infectious agents.

Dose (volume) recommendations:

Small dogs (<15 kg)	0.4-0.5 ml
Medium dogs (15 - 20 kg)	0.7-0.8 ml
Large dogs (> 20 kg)	1.0 ml

The volume of medication applied into the ear during treatment appears to be critical. We recommend using dose syringes to accurately measure otic medications (Figure 4). Failure to apply sufficient quantities to penetrate to these areas is likely a major cause of treatment failure. Volumes used to achieve adequate penetration **down** the canal are based on pilot studies performed by one of the authors and current ongoing studies.

Keep in mind that higher volumes of otic medication may increase the likelihood of their absorption, especially glucocorticoids. It is important to understand that there may be systemic side effects as more potent glucocorticoids are used. Logically, it may be helpful to initiate therapy with medications containing potent glucocorticoids, then switching to less potent glucocorticoids as the inflammation is decreased.

The integrity of the tympanic membrane is critical in determining the best treatment options for a patient with otitis. The possibility of ototoxicosis is greatly enhanced if the medication is instilled directly into the middle ear. The best practice is to avoid topical therapy, if the tympanic membrane is torn or absent. However, there are some clinical indications, based entirely on anecdotal evidence, that vinegar: water (1:2) and enrofloxacin (parenteral formulation) are fairly safe.



Figure 4. Several types of applicator lids (that fit a range of bottle sizes) and dosing syringes are available to facilitate accurate measurement of ear medications.

Table 1.

Veterinary Otic Preparations Available in North America

Product	Manufacturer	Drops/ ml*	Label dosing	Maximum treatment time (days)
Baytril [®] Otic	Bayer Animal Health	30	<35 lbs: 5-10 drops twice daily >35 lbs: 10-15 drops twice daily	14
easOtic [®]	Virbac Animal Health	NA	1 pump daily	5
Mometamax®	Intervet/Schering Plough Animal Health†	40	<30 lbs: 4 drops once daily >30 lbs: 8 drops once daily	7
Otomax [®]	Intervet/Schering Plough Animal Health†	37	<30 lbs: 4 drops twice daily >30 lbs: 8 drops twice daily	7
Posatex™	Intervet/Schering Plough Animal Health†	39	<30 lbs: 4 drops twice daily >30 lbs: 8 drops twice daily	7
Surolan [®]	Vetoquinol	45	5 drops twice daily	7
Tresaderm®	Merial		5-15 drops twice daily	7

 * Determined manually by author. Estimates \pm 2 drops/ml. \dagger Merck Animal Health USA

KEY POINT #6 Systemic Therapy for Canine Ear Disease

When to use systemic therapy for dogs with ear disease

In general systemic therapy is indicated when:

- Infections are recurrent and severe
- There are concurrent infections elsewhere, such as the skin, that would respond to the therapy
- The owners are incapable of treating topically (e.g., arthritic, elderly owner)
- The patient is entirely uncooperative
- There are severe hyperplastic changes in the canal that preclude the ability of topical medications to distribute deeper into the ear canal
- Otitis media exists

Systemic antibacterial therapy may also be indicated when inflammatory cells are seen on cytologic evaluation, when a pure infection of a gram — bacteria is present, in recurring bacterial infections, when ulcers are present in the external ear canal, or when systemic signs accompany the otitis. The antibiotic selection depends upon the organism isolated. Drugs should be dosed at the high end of the recommended range. If the calculated dose is borderline, always increase to the next table/capsule size; never skimp on systemic drug doses. Drugs should be administered for a minimum of 3 weeks, then the patient re-examined.

Systemic glucocorticoid administration may be helpful in two related situations.

 To provide anti-inflammatory effects. If prednisone is used for this purpose, the dosage is 1.1 mg/kg, given orally once daily for five days, then on alternate days in decreasing doses. Anti-inflammatory doses of glucocorticoids may exert their benefits by reducing inflammation, and thus opening the ear canals, or by managing the primary factor (e.g., atopic dermatitis) of the otitis.

2 To manage stenotic ear canals. Stenosis of the external canal is

Ear disease often occurs as a manifestation of allergic skin disease in dogs.

reversible unless fibrosis and calcification of the canal have occurred. If the canal has not become calcified, stenosis may be reversible by 1) treating the concurrent infections, and 2) using aggressive doses of glucocorticoids. If prednisone is used, the dosage recommended to reduce inflammation and edema is 2 mg/kg, given orally once daily for 5 days, then every other day for 5 doses, then 1.0 mg/kg every other day for 5 doses. If the swelling has not significantly been reduced, it is unlikely to do so. Other options that may be of value in some cases to manage stenotic canals include intralesional injections with triamcinolone or oral cyclosporine therapy.

KEY POINT #7 Control Flare Factors

Identifying food allergy, atopic dermatitis, infection and other conditions that contribute to ear disease

As mentioned in the pathophysiology section, the causes of otitis can be classified into predisposing, primary and perpetuating factors **(see Table 2)**. *Predisposing factors* are responsible for alterations in the microclimate of the ear canal, thereby increasing the likelihood of inflammation. *Primary factors* are responsible for causing inflammation directly, and *perpetuating factors* are responsible for the chronic nature of the condition. All these are potential flare

factors, and need to be identified and managed in order to control otitis successfully.

Allergic dermatitis often manifests as ear disease in dogs and underlying allergic conditions will often cause chronic otitis to flare or exacerbate. Several studies have evaluated the complex, multifaceted appearance of allergic skin disease in dogs, many of which suffer from chronic ear disease. In one study of dogs with allergic dermatitis referred to an academic dermatology service in the United States, 46% of dogs had atopic dermatitis, 23% had food allergy, 20% had atopic dermatitis and food allergy concurrently, and the remainder had various combinations of atopic dermatitis, food allergy and/or flea allergy.⁷ Of the dogs with adverse food reactions in that study, 45% had only food allergy and 40% had food allergy and atopic dermatitis concurrently. In a study from Europe, 30% of dogs with atopic dermatitis required additional treatment for otitis externa during a 9-month period of evaluation.⁸ Another study of 172 dogs with food allergic dermatitis found that over half (53%) had chronic otitis.⁹ The largest study examined the diagnostic criteria for atopic dermatitis and food allergic dermatitis in 843 dogs from 15 countries.⁹ The authors confirmed that food-induced allergic dermatitis and non-food induced atopic dermatitis cannot be clinically distinguished. Affected ear pinnae and chronic or recurrent yeast infections were two of the diagnostic criteria for allergic skin disease used in this study.

In summary, ear disease often occurs as a manifestation of allergic skin disease in dogs. Atopic dermatitis and food allergic dermatitis are clinically indistinguishable in many patients and may occur concurrently. Bacterial and yeast infections of the ear should be identified and managed appropriately but primary factors contributing to ear disease should always be investigated. Managing adverse food reactions is discussed in more detail in the next section.



Table 2.

Various Factors Influencing The Development And Perpetuation Of Canine Ear Disease.¹⁰

Predisposing Factors	 Conformation of ear Humidity of ear canal Inappropriate cleaning Irritant treatments Excessive hair growth in ear canal
Primary Factors	 Ectoparasites Atopic dermatitis Food allergy (adverse food reaction) Keratinization disorders Foreign bodies Tumors Autoimmune dermatoses
Perpetuation Factors	 Yeast infection Bacterial infection Epidermal and sebaceous hyperplasia Ulceration Otitis media

KEY POINT #8 Adverse Food Reactions In Dogs With Ear Disease

Role of food allergy/intolerance in chronic canine ear disease

An **adverse reaction to food** is an abnormal response to an ingested food or food additive. In general, pathogenic mechanisms that lead to adverse food reactions include ingestion of inciting agents followed by interaction of the agents with biological amplification systems that lead to inflammation and clinical signs. In view of the number of diverse foods that are routinely ingested by dogs, it is not surprising that there are adverse reactions to dietary substances. The fact that food-related reactions appear relatively infrequently is testimony to the effectiveness of the intestinal mucosal barrier and oral tolerance. Adverse reactions to food have been blamed for a variety of clinical syndromes in dogs, especially those involving the skin, ear and gastrointestinal tracts.

Clinical Features

Adverse reactions to food were reported in dogs as early as 1920. Adverse food reactions are usually suspected when a client or veterinarian establishes a historical association between ingestion of certain foods and the appearance of certain clinical signs. No gender predisposition is noted in reported canine cases. Ages of reported cases have ranged from 4 months to 14 years for dogs. Up to one-third of canine food allergy cases may occur in dogs less than 1-yearold. Most of the reported adverse reactions have been termed food allergy, although no specific tests were performed to confirm an immunologic basis for the clinical signs. Two series of cases could not relate the onset of clinical signs with recent changes in the diet. This suggests that dogs may develop food allergy after prolonged exposure to one brand, type or form of food.

In dogs, food allergy typically is manifested as nonseasonal pruritic dermatitis that is occasionally accompanied by gastrointestinal signs. Pruritus is of varying severity. The location of pruritus is often indistinguishable from that seen with inhalant allergies; feet, face, axilla, perineal region, inguinal region, rump and ears. **One-quarter of food allergic dogs only develop lesions in the ear region. Dogs with chronic or recurrent otitis externa should always be evaluated for food allergy.**

A variety of primary and secondary skin lesions occur in food allergic dogs. These lesions include papules, erythroderma, excoriations, hyperpigmentation, epidermal collarettes, pododermatitis, seborrhea sicca, and otitis externa. Food allergy often mimics other common canine skin disorders including pyoderma, pruritic seborrheic dermatoses, folliculitis, and ectoparasites. Twenty to 30 percent of dogs with suspected food allergy have concurrent allergic disease such as flea allergy dermatitis and/or atopic dermatitis.

Gastrointestinal signs of canine food allergy include vomiting and diarrhea. Clinical response to dietary modification suggests that hypersensitivity to food antigens plays a role in dogs with chronic idiopathic or plasmacytic-lymphocytic colitis. It is not known if the chronic colitis or other forms of inflammatory small bowel disease are a direct manifestation of an adverse food reaction or if dietary modification is merely palliative in some dogs.

Food Allergens

In general, the major food allergens that have been identified in people are water-soluble glycoproteins that have molecular weights ranging from 10,000 to 70,000 daltons and are stable to treatment with heat, acid and proteases. [Molecular weights are expressed in units called daltons. The dalton is an arbitrary unit of mass established by Sir John Dalton (1766-1844), an English chemist and physicist who was the founder of atomic theory. One dalton is $1/12^{\text{th}}$ the mass of the nuclide of carbon-12, equivalent to 1.657 x 10^{-24} grams. It also called an atomic mass unit.]

Fifteen different studies, representing a total of 278 dogs, have described cutaneous lesions associated with adverse reactions to specific foods or ingredients.^{11,12} In these studies, adverse reactions to beef, dairy products, and wheat have accounted for over two-thirds of all the reported cases in dogs. Adverse reactions to chicken, chicken egg, lamb, or soy accounted for approximately 25% of the reported canine cases. Adverse reactions to corn, pork, rice or fish ingredients are rarely reported in dogs from these studies. Specific allergens identified in food allergic dogs include chicken serum albumin (chicken), bovine serum albumin (beef), bovine IgG (cow's milk, beef), ovine IgG (lamb), muscle phosphoglucomutase (beef, lamb) and Gly proteins 50 & 75 kD (soy).

Human allergy reference books often contain phylogenetic tables of animal and vegetable foods, and food-allergic persons are often advised to avoid other closely related foods. In clinical practice, human patients often report cross-reactivity among various fish and among various crustaceans, but less cross-reactivity is reported within vegetable food groups. Results of oral food challenges in children demonstrate that clinically important cross-reactivity to legumes (peanut, soybean, green bean, lima bean, peas, lentils) is very rare. Wheat, rye and barley show cross reactivity in allergic human beings but oats allergens appear to cross react only weakly with these other three grains. Cross-reactivity between milk proteins from cows, goats and sheep has been noted and chicken egg-allergic children have also been shown to cross-react with egg proteins of other birds. Certain allergens are apparently common to both foods and pollens. Common allergens have been reported in melon, banana and ragweed pollen; celery and mugwort pollen, as well as apple and birch pollen. Cross-reactivity among food allergens has not been well investigated in pet animals.

Review of Literature on Canine Food Allergy (1967-present)

278 canine cases (problem ingredient was clearly identified)



Elimination Food Trials

The Ideal Elimination Food

Dietary elimination trials are the main diagnostic method used in dogs and cats with suspected adverse food reactions. At the present time, intradermal skin testing, radioallergosorbent (RAST) tests, and ELISA testing for food hypersensitivity are considered unreliable in dogs with dermatologic disease.

The ideal elimination food should include:

- protein hydrolysates or reduced numbers of novel, highly digestible protein sources
- 2 avoid protein excesses
- 3 avoid additives and vasoactive amines
- e nutritionally adequate for the pet's life stage and condition

Ingredients in an ideal elimination food should provide protein hydrolysates or a limited number of novel protein sources to which the patient has not been previously exposed. This may include a commercial product containing protein hydrolysates, or a commercial or homemade food with one animal protein source and one vegetable protein source. In the dermatologic patient, excess dietary protein levels should be avoided to reduce the amount of potential allergens to which the animal is exposed. A dietary protein level of 16-20% (dry matter basis) for adult dogs is recommended. In patients with hypoproteinemia, hypoalbuminemia, and/or weight loss associated with severe gastrointestinal disease, a higher dietary protein level may be necessary to counteract gastrointestinal losses.

Commercial Elimination Foods

A variety of limited and different protein foods are now being manufactured by several companies. These commercial products are attractive because they are convenient, often contain novel protein sources and are nutritionally complete and balanced for either dogs or cats. Protein digestibility has been compared for some of these commercial products and varies considerably. It is important to understand that few of these commercial foods have been adequately tested in dogs with known adverse food reactions. To the authors' knowledge, only a few commercial foods have undergone the scrutiny of a clinical trial in patients with dermatologic or gastrointestinal disease. In the thirds to three-fourths of the patients with suspected adverse food reactions showed significant improvement in clinical signs.¹²

published clinical trials using commercial foods, two-

Note: A list of currently available commercial elimination foods can be found at the Mark Morris Institute website (markmorrisinstitute.org) under the section for updated tables from the *Small Animal Clinical Nutrition* textbook (*SACN* 5th Edition Table Updates; Section 10 - Tables 31-5 and 31-6).

Commercial Foods Containing Novel Protein Sources

Commercial foods containing unique or novel protein ingredients have been available for more than

45 years. Novel protein sources are usually defined as animal or vegetable ingredients containing protein, which are not commonly used in pet foods and/or are not commonly associated with adverse food reactions. Examples of such protein sources include lamb, venison, rabbit, various fish, rice, potato, and green pea. Beef, dairy products and wheat are the most commonly reported ingredients causing adverse reactions in dogs and should be avoided. Commercial foods containing novel protein ingredients are frequently enhanced with fatty acids and antioxidants to help

control inflammation and improve skin barrier function. Several published clinical studies support the use of commercial foods containing novel protein sources in management of adverse food reactions in dogs.^{12,13}

Commercial Foods Containing Protein Hydrolysates

The newest concept for managing veterinary patients with suspected adverse food reactions is use of commercial foods containing hydrolyzed protein ingredients. Veterinary therapeutic foods containing protein hydrolysates offer several hypothetical advantages over traditional commercial or homemade elimination foods. Protein hydrolysates of appropriate molecular weight (less than 10,000 daltons) will not elicit an immunologically mediated response and may be regarded as truly "hypoallergenic" ingredients. Novel or unique protein sources are less important with protein hydrolysates. Protein hydrolysates have been used for many years in human infant formulas and for human patients with various gastrointestinal diseases.

Protein hydrolysates used in some commercial veterinary therapeutic foods have been found to have substantially lower immunogenicity than the parent proteins. These protein hydrolysates are excellent ingredients for elimination foods. Palatability of veterinary therapeutic foods containing protein hydrolysates vary; however, taste is often better than or at parity with traditional products containing novel protein sources such as duck, venison, fish and potato.

In general,

homemade foods

lack a source of

calcium, essential

fatty acids, certain

vitamins and other

micronutrients.

Several published clinical studies document use of foods containing protein hydrolysates in veterinary patients.¹² These studies showed that over 90% of dogs with confirmed adverse food reactions remained free of clinical signs when consuming a commercial protein hydrolysate food. Clinical trials with protein hydrolysate foods have been conducted in patients seen in private and specialty practices with dermatological and gastrointestinal disease. These studies showed that two-thirds of dogs have complete or partial improvement in clinical signs when fed a commercial protein hydrolysate-based food.14,15

Homemade Elimination Foods

Homemade elimination foods usually include a single protein source or a combination of a single protein source and a single carbohydrate source. Ingredients recommended often for homemade canine foods include lamb, rice, potato, fish, rabbit, venison and tofu.

In a previously published survey, most of the homemade foods recommended for initial management of dogs with suspected food allergy were nutritionally inadequate for growth or adult maintenance.¹⁶ This failure to meet nutritional requirements occurs in most homemade foods because rations are devised to include a minimum of ingredients. In general, homemade foods lack a source of calcium, essential fatty acids, certain vitamins and other micronutrients. Complete and balanced homemade food recipes are available in other references.¹⁷

The use of nutritionally inadequate homemade foods in young dogs for over 3 weeks may result in nutritional disease.¹⁸ Clinical signs of anorexia and poor growth occur in puppies within 10 to 20 days of starting a thiamin deficient diet. Many previously recommended homemade elimination foods have a severe inverse calcium-phosphorus ratio of 1:10. Foods with this severe mineral imbalance can cause skeletal disease in young dogs within 4 weeks and should not be fed for longer than 3 weeks. In young growing dogs, a source of calcium such as additive-free oyster shell calcium (calcium carbonate) should always be used in homemade recipes.

Non-flavored, additive-free vitamin and mineral supplements that do not contain animal or vegetable protein are unlikely to be a source of ingested allergens and should be used in all homemade diets. Intolerance to calcium supplements in atopic children has been reported but is rare. Calcium supplementation should be routinely used in homemade recipes for dogs less than 10 months old.

A source of essential fatty acids, such a vegetable oil, should also be included in homemade rations. Studies show that human beings allergic to peanuts and soybeans can safely ingest peanut oil or soybean oil. This supports the concept that vegetable oils are not a routine source of ingested allergens and can be used in homemade rations. Fatty acid supplements, which contain fish oils are more likely to be contaminated with trace amounts of protein. Recipes recommended for homemade foods should also provide the optimal amount of protein.

Performing an Elimination Food Trial

Before an elimination food trial is initiated, it is useful for the client to keep the dog or cat on its usual food for 7 to 14 days. During this time the client should record the type and amount of food ingested, any other ingested food items such as table scraps, treats or snacks, and the occurrence and character of adverse reactions. Secondary causes of pruritus such as *Malassezia* infection or pyoderma should be eliminated before initiating the dietary trial. The patient is then placed on a controlled elimination food for 4 to 12 weeks. In addition to the dietary change, no other ingested substances such as treats, flavored vitamin supplements, chewable heartworm prevention tablets, fatty acid supplements or chew toys should be offered. Dogs that roam should be confined during the trial.

In dermatologic patients, a tentative diagnosis of an adverse food reaction is made if the level of pruritus markedly decreases. This improvement may be gradual and may take 4 to 12 weeks to become evident. Patients with primarily gastrointestinal signs will usually improve more rapidly.

Provocation involves introducing single dietary ingredients until as many positive reactions as possible can be documented. Clients and veterinarians are often reluctant to pursue challenge and provocation once clinical signs have improved or been eliminated. Provocation may also be difficult in many dogs and cats because commercial pet foods contain such a large number of ingredients and because these ingredients can not often be duplicated in challenge studies. As an example, use of chicken meat in a provocative food challenge may not duplicate the types of antigens found in poultry by-product meal.

Elimination trials are often difficult to interpret in some dogs because of concurrent allergic skin disease. In several studies of food allergic dogs and cats, 20 to 30% or more had concurrent allergies. The response to an elimination trial may be only partial in these patients. Flea allergic dermatitis and atopic dermatitis are common and should be eliminated through other diagnostic testing.

Compliance with Dietary Elimination Trials

Techniques should be used to assure a high degree of compliance with dietary trials. One strategy is to use diet diaries. During the dietary elimination trial, the client should continue daily documentation of the type and amount of food ingested, and the occurrence and character of adverse reactions. This daily diary is important for documenting both the progression of clinical signs during the elimination trial, as well as, whether a strict elimination trial has been performed in the home environment. The diary will often document different findings than those offered to the veterinarian by the client during the recheck examination.

Monitoring Patients

For most adverse food reactions, avoiding the offending foods or food additives is the most effective treatment. How selective or meticulous an avoidance diet must be depends upon the individual patient's sensitivity. Some dogs may suffer adverse reactions to even trace quantities of an offending food or food additive while others may have a higher tolerance level. Concurrent allergies will influence the threshold level of clinical signs in some dogs. Symptomatic therapy in pruritic dogs can also include corticosteroids and antihistamines. Systemic and topical therapy of the ear will often be necessary during elimination food trials in patients with chronic otitis.

Both homemade and commercial foods can be used for long-term maintenance of patients with suspected food allergy. An attempt should always be made to find an acceptable commercial food that raises owner compliance with the dietary change and ensures a nutritionally adequate ration. It is highly recommended to consult with a clinical veterinary nutritionist when formulating a homemade food that will be fed long-term.

KEY POINT #9 Dietary Management Of Dogs With Ear Disease

How to improve skin barrier function and use fatty acid supplementation for patients with inflammatory skin disease

Skin Barrier Function and Allergic Skin Disease

The skin is the largest organ of the body and the anatomic and physiologic barrier between the dog and its environment. The skin protects against water loss and physical, chemical and microbiologic injuries. Recent evidence suggests that skin barrier function is altered in dogs with allergic skin disease.^{19,20}

One measure of epithelial barrier function, transepidermal water loss (TEWL), is increased in atopic compared with normal dogs. This occurs after allergen challenge in sensitized dogs and is also increased in "atopic" versus "nonatopic" predilection sites such as the axilla, chin, periocular region, pinna and thorax. It is not known if patients with canine atopic dermatitis have an intrinsic epithelial cell defect, which allows increased penetration of environmental allergens, sensitization and inflammation, or whether epithelial barrier dysfunction is secondary to immunologic disturbances and associated inflammation. Regardless of whether it is a primary or secondary condition, altered skin barrier function appears to be a consistent finding in patients with atopic dermatitis and is one target for therapeutic intervention.

Therapeutic nutrition can be used to help manage skin inflammation and improve skin barrier function. Key nutritional factors for patients with allergic dermatitis include omega-6 fatty acids, omega-3 fatty acids, antioxidant nutrients such as vitamin E and selenium, zinc, biotin, pantothenic acid, inositol and other B-vitamins. Supplementation of complete and balanced pet foods with these nutrients has been shown to reduce TEWL and improve coat softness and appearance.²¹⁻²⁶ Similar supplementation may improve skin barrier function in patients with allergic dermatitis and decrease epicutaneous absorption of environmental allergens.

Fatty Acid Supplementation

See the separate proceedings "Fatty Acid Supplementation: Does It Work For Dogs with Ear and Skin Disease?" for information on use of fatty acid supplementation.

KEY POINT #10 Client Education

How to get compliance for successful long-term management of canine ear disease

First, remember that all of dermatology is a bit like

archeology. That is, clinicians must keep on digging until they find the underlying civilization (i.e., primary factor). If the clinician does not address the underlying problem, then the perpetuating (i.e., secondary) factors will fail to respond to treatment or will recur. So, it makes sense to explain this to clients *at the beginning*, understanding that it is unlikely that a client will give consent to spend a lot of time and money searching for a cause of first-time otitis. At the very first visit of a patient with otitis to your clinic, you should "plant the seed" by educating the client about the pathogenesis of otitis. You don't need to go into great detail, but simply explain that there is a "what" and a "why" when it comes to ear disease. That way, when the problem recurs (notice I didn't say "if"), they just may remember that you tried to explain this to them. As the pet requires repeated treatments for recurring otitis, the owner may finally understand your recommendation to find the underlying (primary) cause!

Second, ear models are great for explaining otitis.

Several companies have provided these to veterinarians in the past, so ask your reps about one! It is especially helpful to explain the "L" shaped ear canal and why we have to medicate the way we do.

A third suggestion to improve client communication is to consider the value of a video otoscope to get

clients more involved. Though some units may be a bit expensive, they are well worth the investment. Allowing clients to see the condition of their pet's ears will definitely help to convince them that cleaning and medications are warranted. In addition, clients LOVE seeing their pet's ears before *and after* cleaning or before and after treatment. Many clients even learn to ask for photos of the ears at each visit so that they can keep a running log of the problem.

Fourth, it is also very important to educate clients on the best techniques for applying medicine in the ears

of their pet. It is strongly recommended that you (or your technician) demonstrate application of the product you are dispensing, then have the owner apply the medication in the second ear while you watch and provide constructive feedback.

Long-term therapy maintenance

Once a pet develops otitis externa, "flare-ups" tend to occur for the remainder of the pet's life even if the primary factor is identified and appropriately managed. Some type of maintenance therapy is frequently necessary to prevent frequent full-scale recurrence of disease and to keep the pet comfortable and the owner happy. Maintenance may be accomplished with once weekly cleaning of ears, especially if the ear cleanser has some inherent antibacterial and anti-yeast activity, or through the use of intermittent topical medications (effective against the perpetuating factor found in the patient).

The authors strongly recommend the use of an antiseptic, such as acetic acid/boric acid or aluminum acetate (Burow's solution) in lieu of intermittent use of an antibiotic or antifungal drug. We do not want to facilitate development of any microbial resistance through the use of sub-optimal concentrations of these agents.

The value of good client education:

- 1 Better client compliance
- 2 More cooperative clients
- Better success with treatment plans
- 4 Increased business
- 5 Everybody wins

SUMMARY

These 10 key points can change the way you address a patient with otitis and make veterinary practice more enjoyable. The most important key is good client communication. Owners really do need to understand the basics of the pathogenesis of otitis, so that they can understand why you are managing their pet the way you do. Increased communication will improve owner compliance and overall success.

References

¹Banfield[®] Pet Hospital State of Pet health: 2011 Report. Available at: http://www.banfield.com/Banfield/files/ bd/bd826667-067d-41e4-994d-5ea0bd7db86d.pdf. Accessed May 23, 2012.

²Angus JC, Lichtenstieger C, Campbell KL, et al. Breed variations in histopathologic features of chronic otitis externa in dogs: 80 cases (1995-2001). *J Am Vet Med Assoc.* 2002;221:1000-1006.

³August JR. Otitis externa: A disease of multifactorial etiology. *Vet Clin N Am Sm Anim Pract*. 1988;18:731-742.

⁴Huang HP, Fixter LM, Little CJ. Lipid content of cerumen from normal dogs and otitic canine ears. *Vet Rec.* 1994;134:380-381.

⁵Zur G, Lifshitz B, Bdolah-Abram T. The association between the signalment, common causes of canine otitis externa and pathogens. *J Small Anim Pract*. 2011;52:254-258.

⁶Cole LK, Kwochka KW, Kowalski JJ, et al. Microbial flora and antimicrobial susceptibility patterns of isolated pathogens from the horizontal ear canal and middle ear in dogs with otitis media. *J Am Vet Med Assoc*. 1998;212:534-538.

⁷Jackson HA, Murphy KM, Tater KC, et al. The pattern of allergen hypersensitivity (dietary or environmental) of dogs with non-seasonal atopic dermatitis cannot be differentiated on the basis of historical or clinical information: a prospective evaluation 2003-2005 (abstract). In: *Proceedings*. North American Veterinary Dermatology Forum 2005;96.

⁸Colombo S, Hill PB, Shaw DJ, et al. Requirement for additional treatment for dogs with atopic dermatitis undergoing allergen-specific immunotherapy. *Vet Record*. 2007;160(25):861-864.

⁹Favrot C, Steffan J, Seewald W, et al. A prospective study on the clinical features of chronic canine atopic dermatitis and its diagnosis. *Vet Dermatol*. 2010;21(1):23-31.

¹⁰Griffin CE. Otitis externa and media. In: Griffin CE, Kwochka KW, MacDonald JM, eds. *Current Veterinary Dermatology*. St. Louis: Mosby-Year Book;34:244-262.

¹¹Roudebush P. Ingredients associated with adverse food reactions in dogs and cats. *Adv Am Anim Med Surg.* 2002;15(9):1-3.

¹²Roudebush P, Guilford WG, Jackson HA. Adverse reactions to food. In: Hand MS, Thatcher CD, Remillard RL, Roudebush P, Novotny BJ, eds. *Small Animal Clinical Nutrition, 5th ed.* Topeka, KS: Mark Morris Institute; 2010:609-635.

¹³Fritsch DA, Roudebush P, Allen TM, et al. Effect of two therapeutic diets in dogs with chronic nonseasonal pruritic dermatitis. *Intern J Appl Res Vet Med*. 2010;8:146-154.

- ¹⁴Loeffler A, Lloyd DH, Bond R, et al. Dietary trials with a commercial chicken hydrolysate diet in 63 pruritic dogs. *Vet Rec.* 2004;154:519-522.
- ¹⁵Loeffler A, Soares-Magalhaes R, Bond R, et al. A retrospective analysis of case series using home-prepared and chicken hydrolysate diets in the diagnosis of adverse food reactions in 181 pruritic dogs. *Vet Dermatol.* 2006;17:273-279.

¹⁶Roudebush P, Crowell CS. Results of a hypoallergenic diet survey of veterinarians in North America with a nutritional evaluation of homemade diet prescriptions. *Vet Dermatol.* 1992;3:23-28.

¹⁷Remillard RL, Crane SW. Making pet foods at home. In: Hand MS, Thatcher CD, Remillard RL, Roudebush P, Novotny BJ, eds. *Small Animal Clinical Nutrition, 5th ed.* Topeka, KS: Mark Morris Institute; 2010:207-223.

¹⁸Debraekeleer J, Gross KL, Zicker SC. Feeding growing puppies: postweaning to adulthood. In: Hand MS, Thatcher CD, Remillard RL, Roudebush P, Novotny BJ, eds. *Small Animal Clinical Nutrition, 5th ed.* Topeka, KS: Mark Morris Institute; 2010:311-319.

¹⁹Cornegliani L, Vercelli A, Marsella R. Transepidermal water loss in healthy and atopic dogs, treated and untreated: a comparative preliminary study. *Vet Dermatol.* 2012;23:41-44.

²⁰Hightower K, Marsella R, Flynn-Lurie A. Effect of age and allergen exposure on transepidermal water loss in a house dust mite sensitized beagle model of atopic dermatitis. *Vet Dermatol*. 2010;21:88-95.

²¹Campbell KL, Roudebush P. Effects of four diets on serum and cutaneous fatty acids, transepidermal water losses, skin surface lipids, hydration and condition of the skin and haircoat of dogs (abstract). In: *Proceedings*. Annual Meeting of AAVD/ACVD 1995.

²²Jewell DE, Yu S, Joshi DK. Effects of serum vitamin E levels on skin vitamin E levels in dogs and cats. *Vet Ther*. 2002;3(3):235-243.

²³Markwell P, Watson A, Fray T. Skin barrier function: can nutrition influence the final frontier? In: *Proceedings*. 22nd Annual ACVIM Forum 2004;550-551.

²⁴Marsh KA, Ruedisueli FL, Coe SL, et al. Effects of zinc and linoleic acid supplementation on the skin and coat quality of dogs receiving complete and balanced diet. *Vet Dermatol.* 2000;11:277-284.

²⁵Roudebush P, Schoenherr WD. Skin and hair disorders. In: Hand MS, Thatcher CD, Remillard RL, Roudebush P, Novotny BJ, eds. *Small Animal Clinical Nutrition, 5th ed.* Topeka, KS: Mark Morris Institute; 2010:237-265.

²⁶Yu S, Wedekind KJ, Kirk CA, et al. Primary hair growth in dogs depends on dietary selenium concentrations. *J Anim Physiol Anim Nutr (Berl)*. 2006;90:146-151.