Catalog

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Antipruritic therapy 2022

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Corticosteroids are extremely useful drugs for the management and of many dermatologic disorders. Unfortunately, they are often misused. The purpose of the presentation is to dispel common myths about corticosteroids and understand the pituitary adrenal axis to improve the quality of life of our patients. Additionally, differences between dogs and cats will be discussed.

All corticosteroids are not created equally. The potency and duration of effect must be taken into account for each individual for maximum effect and minimal side effects.

Drug	Glucocorticoid potency	Equivalent dosage (mg)	Duration of Effect (hr)	Alternate day?
Short-Acting				
Cortisone	0.8	25	<12	–
Hydrocortisone	1	20	<12	–
Intermediate				
Prednisone	4	5	24-36	Yes
Prednisolone	4	5	24-36	Yes
Methylprednisolone	5	4	24-36	Yes
Long-Acting				
Flumethasone	15	1.3	36-48	No
Triamcinolone	40	0.5	36-48	No
Dexamethasone	40	0.5	36-54	No
Betamethasone	50	0.4	36-54	No

Only those products that are short-acting or intermediate are appropriate for alternate day dosing of anti-inflammatory or autoimmune disorders. Potency must also be considered when a topical product is selected. Long acting topical steroids will suppress the pituitary adrenal axis and are more likely to cause skin and hair follicle atrophy.

Glucocorticoids exhibit a myriad of early and late phase anti-inflammatory effects. The short term effects include mast cell stabilization, reduction in capillary permeability, and reduced leukocyte migration and function. These are the beneficial effects that are so important in treating Type I hypersensitivity reactions. The ultimate effect is to alter protein transcription. These proteins may be induced or inhibited and include lipocortin 1, cytokines, inducible nitric oxide synthetase (iNOS), and phospholipase A2, to name a few. By directly inhibiting phospholipase, the arachadonic acid cascade is blocked leading to a decrease in the pro-inflammatory mediators. Prostaglandins, thromboxanes, and leukotrienes are all decreased. Glucocorticoids may directly block cyclooxygenases further contributing to the anti-inflammatory effects. Alterations in protein synthesis may take hours to days for the necessary effect to take place.

There are several predictable effects of glucocorticoids that are not necessarily beneficial. Hyperglycemia results from gluconeogenesis and from insulin antagonism by blocking insulin from getting into cells. The physiologic reason is to protect glucose dependent brain functions. Glucocorticoids are catabolic in effect. Skeletal muscle and collagen breakdown result in muscle wasting, thin and hypotonic skin, and fragile blood vessels. These side effects may result from topical or parenteral administration. The catabolic effects on lipids result in the redistribution of fat. Glucocorticoids inhibit antidiuretic hormone and contribute to the polyuria/polydipsia seen in dogs. This may be due to both the glucocorticoid and mineralocorticoid effects of steroids.

Injectable glucocorticoids have variable actions of onset because of the carrier molecules. These esters must be hydrolyzed to release the active free form of the drug. Water soluble esters such as sodium succinate or sodium phosphate and are more rapidly hydrolyzed. These forms may be given intravenously and are indicated for acute conditions. The repositol forms are water insoluble. Carrier esters such as acetate or acetonide are more slowly hydrolyzed resulting in a prolonged effect. Not all glucocorticoids are labeled as "depo" so one must be aware of the carrier molecule to avoid unwanted effects.

Oral steroids are well absorbed and as fast in onset of the anti-inflammatory effects as injectable glucocorticoids. Giving an injection to "get things started" is not necessary

and will lead to steroid overdose if tablets are given for a controlled effect. Prednisone is converted to prednisolone and can be interchanged for most dogs. There are some individual dogs that using prednisolone may be more effective. The dosage for anti-inflammatory effects is 0.5 mg/kg b.i.d. or can be given 1.0 mg/kg/day. Once a clinical remission is achieved, the dosage may be tapered to an alternate day therapy. This will allow the pituitary-adrenal axis to rebound. Immune suppressive dosages of these drugs begin at 2mg/kg/day. Methylprednisolone (medrol®) is also an intermediate acting glucocorticoid that is good for alternate day therapy. It is slightly more potent than prednisone or prednisolone (4mg methylprednisolone = 5mg pred.) and is usually associated with less PU/PD in dogs. It is the preferred glucocorticoid for cats (author bias). The dosage for anti-inflammatory effects in cats is twice the dosage of dogs. This is due to fewer receptors and less affinity than dogs. In general, a 10# cat would receive 4mg of methylprednisolone twice daily until remission. Cats in general are more tolerant of corticosteroids than dogs and may do well with injectable forms.

There is little difference between prednisolone and prednisone. The anti-inflammatory dosage for dogs is 0.5 mg/kg given twice daily for one week, once daily for one week, and every other day for 14 days. To make this easy to calculate, take the total number of tablets for the first week and double it. Dogs can continue to receive pred. on an every other day basis if there is a history of a prolonged seasonality problem. In some cases a switch to every third day or changing to a different product (ie. Temaril-P) may maintain the effect with a lower dosage. By using a simple chart, there are no excuses for not using oral glucocorticoids

Fill in the number of tablets in each box, along with the starting day of the week

Include at the bottom some of the common side effects. Also include what should be done if side effects are problematic. Additional notes for when to recheck may also be helpful. Have the client circle the dosage once it has been given to avoid accidentally over dosing the pet.

Temaril-P is a product that contains 2 mg of prednisolone and 5 mg of trimeperazine. The dose is 1 tablet/#20 body weight with a maximum of 3 tablets. Compared to the amount of prednisolone the dog would receive with standard dosages, temaril-P is much lower. This product is not suitable for dogs >60# and is not to be used for cats. This product works very well for uncomplicated atopy cases. It is usually unsatisfactory for dogs exhibiting flea allergy, food allergy, or scabies. It may be dispensed in a decreasing dosage similar to oral pred. Some dogs are not well managed with every other day Temaril-P. Giving the tablets twice daily, every other day, may improve clinical response.

Cyclosporine (Atopica) is effective for reducing pruritus due to atopy. It is a calcineurin inhibitor that affects both TH1 and TH2 cytokines. It is an immune suppressive agent even when used for antipruritic effect. The standard dosage for dogs is 5mg/kg qd for

30 days then reduce to eod. It is recommended to treat daily beyond 30 days until a remission is reached before decreasing to eod dosing. Some dogs will require 5-6 weeks to achieve remission. We recommend CBC and UA with culture about every 4 months. Ketoconazole is a potent inhibitor of cytochrome P450 enzymes and will prolong the metabolism of cyclosporine. Cyclosporine should not be used if there was a history of demodicosis. I would recommend starting with name brand Atopica to see if it helps and then switch to a generic or compounded if requested. Compounded products have been demonstrated to be inaccurate with the amount of drug in the capsules.

Oclacitinib (Apoquel) is an antipruritic agent. It functions as a Janus kinase inhibitor (JAK) and only inhibits pruritus associated with IL-31. It is not to be used on dogs less than one year or those with bacterial or demodex infections. Studies show that it may increase the risk of these infections. Apoquel is indicated for the use of allergic pruritus. It is initially administered twice daily for 14 days then daily as needed. One of the benefits of this drug is that it reduces pruritus faster than glucococorticiod medications. The data show that this drug will not interfere with intradermal testing, so no withdrawal period is necessary. It appears to be safe when used in conjunction with commonly used medications such as cephalexin or ketoconazole. Apoquel should not be given to cats and is not licensed or approved for this use. There is anecdotal evidence that tachyphylaxis may occur with this medication.

CytoPoint is a canine monoclonal IgG antibody that binds IL-31 to inhibit binding to the receptor. It is licensed for atopy in dogs only. Human articles have shown that IL-31 is the major cause of pruritus in people with cutaneous lymphoma (CTL) but this product has not been published studies in dogs with CTL. Cytopoint is given subcutaneously every 30 days or as needed. It is not effective for all dogs. It may or may not be effective in dogs that have been unsuccessfully treated with Apoquel. Cytopoint does not interfere with intradermal allergy testing. We have used it successfully concurrently with hyposensitization induction. It is a nice off-label treatment for young dogs with sarcoptic mange, but it does not always work. It has also been used to help abate pruritus in dogs with cutaneous lymphoma.

Management of autoimmune skin disorders

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All disease is immune- mediated to some extent. Canine patients may exhibit autoimmunity leading to skin pathology. Pemphigus complex (PF), discoid lupus erythematosus (DLE), and symmetrical lupoid onychodystrophy (SLOD) are the most common. Some disorders such as vasculitis, erythema multiforme, adverse drug reactions, vasculitis, and panniculitis are immune- mediated but not necessarily autoimmune mediated.

Pemphigus foliaceus is the most common of the pemphigus complex of diseases. Middle aged dogs are most commonly affected though PF may be seen in older dogs. There does not seem to be a sex predilection. Any breed including mixed breeds can develop pemphigus. This disease is considered to be autoimmune with antibodies attacking the desmosomal proteins in the spinous layer of the stratum corneum. The resulting pathology leads to subcorneal pustules with acantholytic cells and non-degenerative neutrophils and sometimes eosinophils. Pustules rupture quickly leading to crusting and collarette lesions. The most commonly affected areas are the ears, face, footpads, and thorax but lesions can appear anywhere on the body. There is usually symmetry to the distribution of the lesions. Mucosal surfaces are usually not affected by PF. Diagnosis is made with cytology and confirming with histopathology. Severe bacterial infection can result in similar clinical findings. If bacteria are identified on cytology, it is best to resolve the infection before performing biopsy. The best biopsy is from an intact pustule. Please include the crust if a crusted lesion is selected for biopsy. Acantholytic cells may be identified within the crust itself.

Treatment for PF depends upon severity and there are many ways to manage a case. Autoimmune skin disorders are exacerbated by UV light exposure so sun avoidance is recommended. Baseline CBC, serum chemistry profile, and urinalysis should be performed. We usually recheck lab work at 2 weeks after instituting treatment then every two to three months thereafter. Obviously, it will need to be repeated if there are significant side effects. Patients that are well managed may need to be rechecked every 3-4 months. It is important that urine cultures be performed at each recheck because the immune suppressive drugs may mask the common side effects associated with a UTI. Prednisone or prednisolone should be started at 2-4 mg/kg/day. Therapy may be divided or given once daily until a

remission is achieved. The dosage can then be decreased slowly (no more than 25% reduction) until every other day therapy is achieved. Injectable and repositol steroids should be avoided. Some clinicians like to add cyclosporine at about 7mg/kg/day. This drug has a slow onset of action but may be steroid- sparing. I prefer to not add other immune suppressive agents such as azathioprine (2.2mg/kg/day) due to the severe hepatotoxicity. This drug is usually added if there are very severe side effects of steroids alone or if steroids alone do not lead to clinical remission. Chlorambucil is sometimes used but is very expensive and difficult do dose large dogs. Lufenomide is inconsistently effective. A retrospective review of canine PF revealed that concurrent antibiotic therapy with cephalexin during the induction phase of treatment led to a more favorable outcome. Most dogs will require life-long therapy. If the PF was drug induced, immune suppressive therapy may be withdrawn, but not in all cases.

Discoid lupus erythematosus (DLE) is a lupus-like reaction confined to the skin. Systemic signs of lupus such as anemia, thrombocytopenia, or proteinuria are usually absent. Lesions are commonly seen on the planum nasale but may be identified on haired skin. The diagnosis is made from biopsy and histopathology. If the planum is ulcerated, it is important to include the leading margin of the lesion in the biopsy sample. This is best accomplished by taking a surgical ellipse along the long axis of the transition from ulcer to lesser affected skin. This technique is best for ulcerated lesions of any etiology. Pemphigus foliaceus, fungal disease, bacterial infection, and vascular anomalies are important differential diagnoses. DLE is exacerbated by UV light so making the dog a vampire is an important part of therapy. This tends to be a steroid responsive disease. Prednisone or prednisolone at 1.5-2mg/kg/day is usually effective. The same monitoring protocols discussed above are appropriate. Cyclosporine at 7mg/kg/day, tapering to every other day, is also effective but slower in onset of action. Cyclosporine in general is a very good choice for treating lupus-like reactions. DLE may respond to the combination of doxycycline (5-10mg/kg bid) and niacinamide (250mg-500mg bid). This combination therapy can cause hepatotoxicity so monitoring laboratory blood work is indicated. Antibiotic resistance, frequent dosing, and inconsistent results preclude recommending this protocol for routine cases. Mildly severe patients and those with bad side effects from steroids may be reasons to consider this protocol. In many cases, therapy can be instituted until clinical remission and then withdrawn until the lesions flare again. Most cases do not need ongoing therapy, especially if light avoidance is observed.

Symmetrical lupoid onychodystrophy (SLOD) is a lupus-like reaction affecting multiple claw beds of dogs. It usually affects middle aged dogs and German shepherd breeds appear to be over represented.

Affected claws may slough, may become either soft or brittle, or may split and crack. Affected claws may become painful and secondarily infected. There are usually no clinical signs of systemic lupus erythematosus similar to DLE. This is the most common disorder affecting multiple claw beds. Neoplasia is usually confined to a single digit. Bacterial and fungal infections are important differential diagnoses. If infection is identified, it is important to resolve it before performing biopsy for histopathology. Biopsy can be collected from the tissue around the claw bed while attempting to go full thickness to the claw itself. Digital amputation and submission of the entire claw can also be performed but is usually not necessary. Treatment is the same as for DLE. Supportive pain management is usually indicated during the induction phase, obviously avoiding non-steroidal anti-inflammatory agents while steroids are being administered. It is usually beneficial to remove any loose claws and trim lesser affected claws very short at the time of anesthesia/biopsy. Periodic nail trims may need to be performed to limit secondary infections. It is my opinion that this disease can be managed (most of the time) with doxycycline/niacinamide once a clinical remission is achieved with steroids. There is no evidence that UV light avoidance must be recommended but all autoimmune skin diseases are exacerbated by UV light.

Erythema multiforme (EM) is a reaction pattern, and is commonly associated with an adverse drug reaction (ADR). Not all drug reactions exhibit this type of reaction though. Clinical signs associated with EM can pretty much look like any other skin problem. Erythema, alopecia, crusting, scaling and ulceration may be seen. Target lesions are suggestive of EM but bacteria and bug reactions may also cause these lesions. The onset is quite rapidly progressive and the severity usually exceeds the typical pyoderma or dermatophyte reaction. Foot pads, planum nasale, or mucosal surfaces may be affected. A thorough drug history is important in making a preliminary diagnosis. Obviously, the offending drug should be discontinued as soon as possible. The diagnosis is made with biopsy and histopathology. Care should be taken to identify and biopsy lesions in the early, middle, and late stages of progression. The owners are usually helpful in this regard. Even with the best intention, the diagnosis of EM may not be conclusive on histopathology. Treatment is aimed at supportive care, pain management, and wound management. Any drug including glucocorticoid medications can exacerbate a drug reaction. The use of steroids is controversial and usually not recommended. Topical therapy is favored whenever possible. The prognosis can range from good to grave, depending upon the progression so it is usually best to give a guarded prognosis. These patients usually require extensive wound and pain management as well as supportive fluid and nutritional care and hospitalization may be extensive (and costly).

Vasculitis can be induced from myriad causes including: adverse drug reactions, infection due to bacteria or rickettsial causes, ectoparasites, vaccine reactions and adverse food reactions. It is reported that about 50% of vasculitis cases are idiopathic (think pinnal vasculitis). Clinical signs are usually associated with multifocal or coalescing, intensely erythematous macules that do not blanch on diascopy. There can also be erosive to crusting lesions. Angioedema may also be present. The diagnosis of vasculitis is made with biopsy and histopathology. The challenge is finding and resolving the offending cause. Though it is an "itis", steroids are usually contraindicated. Pentoxifylline at 15 mg/kg bid-tid is considered to be a treatment of choice. Other than vomiting, side effects are fairly uncommon. In the south, Ehrlichia is a common cause of vasculitis. Treatment with doxycycline is effective for both it's anti-rickettsial properties but also because of it's anti-inflammatory effects. The cycline antibiotics decrease neutrophil migration.

Panniculitis can be sterile or septic. Clinical signs include focal to multifocal soft, fluctuant, raised skin lesions that can rupture. The material can be exudative and or contain a lot of lipid. The lesions vary in pain and odor. Cytology can be helpful in identifying infectious organisms. The diagnosis is made with biopsy for histopathology and biopsy for macerated tissue culture. A non-ruptured lesion can be surgically prepared followed by aseptic biopsy collection. The sample is transferred in appropriate media to the laboratory and kept chilled but not frozen. If Pythium is a differential diagnosis, the sample should not be refrigerated because oomycete organisms will be killed. It must be requested for the sample to be macerated (chopped up). Some organisms grow better if the chunk of tissue is placed in broth or media so this would be standard procedure unless requested to be macerated. We recommend requesting aerobic, anaerobic, fungal, and opportunistic mycobacteria cultures at the time of submission. Most laboratories would like about 3, 6-8mm punch tissue samples in order to perform all of the cultures. It is best to contact the laboratory for advice prior to collection and submission. Macerated tissue culture is also the preferred method of sample collection and submission of suspected resistant staphylococcal skin infections. Sterile panniculitis is a diagnosis of exclusion of infectious organisms. Treatment is usually instituted with prednisone or prednisolone at 1.5-2mg/kg/day along with cyclosporine at 7mg/kg/day. Once remission is achieved the pred dosage should be decreased and eventually stopped. Most cases can be successfully managed with long term cyclosporine at the lowest every other day dosage. Obviously, if infectious organisms are identified they should be appropriately treated without concurrent use of immune suppressive agents.

New Perspectives on Chronic Canine Otitis

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Canine and feline otitis continues to be one of the most requested topics for continuing education seminars. The multi factorial components make diagnosis and treatment more complicated than other dermatologic diseases. Compounding the difficulty of treatment is the limited number of otic preparations that are licensed, safe, and efficacious for instillation into the ear canal. The focus of this presentation will center on treatment options for refractory otitis. A brief discussion on the predisposing and primary causes of otitis will also be covered. Because feline otitis and treatment there of can be very different, comparative aspects will be addressed. Finally, surgical options of treating otitis will be reviewed.

Otitis externa is defined as an acute or chronic inflammation of the ear canal and or pinnae. It may be unilateral or bilateral. Otitis media describes inflammation beyond or including the tympanic membrane (ear drum) and bullae. Otitis interna is reserved for cases of documented neurologic symptoms including vestibular signs.

The causes of otitis are best evaluated as predisposing, primary, and perpetuating factors. Usually there are many factors involved and otitis should not be viewed as mainly a bacteria or yeast infection. On physical examination, it is usually these perpetuating factors that get all the attention. Obviously, we need to resolve the bacteria or yeast component but we must ask ourselves “what is a nice healthy dog doing with an ear infection like this?”

Predisposing factors are those that make the conditions favorable for an otitis situation to occur. Stenotic ear canals are the classic predisposing factor. However, stenotic ear canals can also be considered a perpetuating factor in a chronic infection situation. Inappropriate topical or systemic medications can be predisposing factors. Ear canal maceration due to excessive moisture from swimming or bathing can disrupt the epithelial barrier enough to allow commensal ear organisms such as bacteria or yeast, to over- populate. Predisposing factors can best be diagnosed with a thorough history and otoscopic examination.

There are many primary causes of otitis. The partial list includes ectoparasites, foreign body, autoimmune disease, neoplasia, adverse drug reaction, viral and protozoal causes. Recurrent otitis is a common occurrence in both food allergic and atopic patients. I believe that allergy is the most common cause of otitis in adult dogs. In cases of chronic or recurrent otitis, the primary cause must be sought. Usually, the search for the primary cause is only possible once the predisposing and primary factors are resolved.

Chronic otitis associated with an adverse reaction to food is fairly common. Interestingly, these cases may present as unilateral or bilateral otitis. Concurrent pruritus of the face, feet, axillae, and or forelegs may be absent. If the distribution pattern of pruritus is suggestive of an adverse reaction to food, an elimination food trial should be instituted. There are difficult cases where the only clinical finding of adverse reaction to food is otitis. A food challenge after complete elimination test diet for at least 8 weeks may be associated with an acute relapse of the otitis.

The perpetuating factors include bacterial and yeast infections, and rarely dermatophytosis. Otitis media may be a perpetuating or a primary cause of recurrent (or chronic) otitis. As the perpetuating factors continue to progress, fibrosis and ear canal calcification may develop. These progressive pathologic changes usually result in the necessity of surgical intervention.

After an adequate history if collected, an otoscopic examination is the single most useful diagnostic procedure. A thorough examination of the pinnae, vertical and horizontal ear canal, and status of the tympanum must be documented. A diagnosis of ear mites is best made with an otoscope. Ulcerations and primary lesions can be identified. The status of the tympanic membrane (intact, torn, inflamed, absent) will determine subsequent diagnostic and therapeutic considerations.

After otoscopic examination and before ear cleaning, ear exudate cytology should be performed. This is done by gently inserting a cotton-tipped swab into the ear canal and rolling the contents onto a clean glass slide. The slide should be heat fixed (until warm) and stained with a modified Wright's stain such as Dif-Quik. A second slide should be prepared for Gram Stain if indicated. The presence of many rod- shaped bacteria would be a reason to perform this extra procedure. Once dried, the slide should be viewed under high power (100X) oil immersion objective for the morphology of bacteria or the presence of yeast. This stain is helpful in determining the morphology of the bacteria (rod or cocci) but does not reveal the Gram staining properties. Cocci bacteria are commonly Staphylococcus pseudintermedius or Streptococcus sp. Common rod bacteria include E.coli, Pseudomonas, Proteus, Klebsiella, and Corynebacteria, although others are occasionally identified. Yeast within the ear canal are almost always Malassezia organisms and as such, therapy should be directed at this pathogen. Occasionally Candida are identified based on culture. Morphologically speaking, it is impossible to tell these two apart on cytology alone. Ectoparasites such as demodex or sarcoptiform mites are occasionally seen on exudate cytology. It is important to identify the cells of the inflammatory response. Neutrophils are commonly identified in exudative, ulcerative otitis. Proteinaceous debris may be present without identification of inflammatory cells. Some cases of chronic otitis may have little inflammatory exudation.

Once cytology samples are collected, it is best to collect a sample from each ear canal for culture They can always be thrown out if culture and susceptibility are not needed, but it is hard to get a good culture after cleaning the ears. Wipe out the excess debris within the external canal. Use a sterile otoscopic cone and insert a culturette swab through it into the middle ear. A sterile cone should be used for each ear to prevent cross contamination.

In many cases of chronic otitis the ear canal may be so full of waxy debris or exudation that visualization of the tympanic membrane cannot be determined. When this happens, a thorough ear cleaning must be performed. In mild or acute cases products designed for cleaning and maintenance (Oti Rinse, Oticalm, etc.) can be used. In chronic cases, anesthesia and thorough ear flushing may be indicated. Sterile warmed saline is probably the best and safest ear cleaning agent. However, in chronic cases it is difficult to know the status of the tympanum until cleaning is completed. An ear bulb syringe is all that is needed for lavaging the ear canal. Rubber catheters and water-pik devices should be used with caution as either can rupture an intact tympanic membrane or damage the oval window. A feeding tube attached to a large syringe can be inserted into the ear canal to remove excess water once cleaning is

complete. Suction apparatus may also be useful. If oti-liths are present, the gentle use of an ear curette can be beneficial. Chlorhexidine as a cleaning and flushing agent should be avoided due to its ototoxicity potential. Iodine agents have been shown to be ototoxic to cats. In general, iodine agents break down quickly in exudation and are rarely useful for routine cleaning.

Treatment of otitis includes the complete resolution of all predisposing, primary and perpetuating factors. In many cases the resolution of the perpetuating factors (bacteria and or yeast) can be formidable. For the treatment of bacterial otitis there are many commercially available products. If bacterial are identified on cytology a topical agent selected to treat the bacterial component is indicated. Selection of a topical agent is based on the morphology of the bacteria and the historical information of what products have been used previously. Neomycin is a good broad spectrum antibiotic effective against some rod and cocci bacteria. It is a good first line antibiotic but resistance may be seen with continued usage. Neomycin has been reported to be a cause of contact dermatitis in human beings. Contact reactions to any topical medication may occur in dogs and may be another perpetuating factor of otitis. Florfenicol containing products such as Osurnia (terbinafine, betamethasone acetate) are effective against Gram positive cocci and yeast. This product is to be instilled into clean dry ears once, then repeated in 7 days. Claro also contains florfenicol and terbinafine but has mometasone furoate as its steroid. It is applied as a single application and left alone for 30 days. Care should be taken to not get these products of the patient (or clinician). Gentamicin becomes inactivated in the presence of exudation and should not be placed into an ear canal full of pus. In cases where resistance is suspected to these topical agents, polymixin B containing products may be used. Polymixin B is highly effective against Gram negative rods but has limited efficacy against cocci bacteria. Neomycin is added to this product to increase the spectrum of activity to include cocci bacteria. Corticosporin Otic (and generic formulations) contain hydrocortisone and Burrow's solution to help decrease inflammation. Surolan is a product that contains miconazole, polymixin B and prednisolone acetate. In highly resistant cases silver sulfadiazine by itself may be used. The cream may be diluted with warm water to make it into a workable otic preparation. This is the only product mentioned so far that does not contain corticosteroids and may be a good choice of a topical agent if avoidance of corticosteroids is indicated. Silver sulfadiazine is not licensed and approved for use in the ear canal of dogs. It is a very potent agent against Gram negative rod bacteria and will help with re-epithelialization of ulcerated ear canals. The author is unaware of any reports of ototoxicity induced by this agent. Because of the potential for adverse reactions in human beings sensitive to sulfur drugs, it is best to recommend that the owner wear gloves when handling this product. Baytril otic contains both enrofloxacin and silver sulfadiazine and no corticosteroid. It is beneficial in treating Gram negative rod bacteria, Gram positive cocci and highly exudative otitis. Posatex is a product that contains orbafloxacin, mometasone and posaconazole. The glucocorticoid is very potent so use is limited to once daily application. I have used ticarcillin prepared as an otic preparation for highly refractory bacterial pathogens with good success. Since ticarcillin is expensive and hard to obtain, we have substituted piperacillin with equally good success. The compounding is the same. There is a recipe at the end of the notes.

Chlorhexidine has been shown to be highly ototoxic and products containing this ingredient are not labeled for otic usage. The fact that one product was taken off of the market should be sufficient warning of the potential risk. Xenodyne is a buffered iodine containing

product that is effective against bacteria and yeast and does not contain a corticosteroid. This product is contraindicated in cats because of ototoxicity but this has not been reported in dogs. This is a good time to make the point that dog and cat otitis is very different.

Tris EDTA is a product that improves the efficacy of an antibiotic against both Gram positive and negative bacteria. There are also commercially available products (Triz-EDTA) that are convenient and excellent. Ten minute contact time is essential. This product has detergent like properties to help break down exudation within the ear canal. It can disrupt the lipopolysaccharide membrane of Gram- negative bacteria and kill the organism. It can also allow better penetration of additional antibacterial agents. Studies support the benefical effect against Gram positive bacteria as well. These products are buffered to a pH of 8 which is compatible with our aminoglycoside and fluorinated quinolone topical medications. The author does not recommend mixing this product with antibiotics as safety and efficacy have not been scientifically demonstrated. There are commercially available product which contains both Tris EDTA and ketoconazole. These are beneficial in mixed bacteria and yeast infections. The author has not identified any adverse reactions associated with these products.

pH compatibility is important when more than one topical agent is selected. In general, aminoglycoside and fluorinated quinolone medications are buffered to a basic pH. There are many cleaning agents available that are acidic. It is recommended that compatible products are used. In general, Tris EDTA is an excellent cleaning agent. Any residual product remaining in the ear canal will not alter the efficacy of the selected antibacterial agent.

There are limited products available for the topical treatment of yeast infections within the ear canal. Complicating things even further is the fact that the literature is not consistent with which active ingredients are actually efficacious. Recommendations are based on personal experience. Miconazole (Conofite lotion) containing products are the single best anti-yeast agent available. Efficacy may be regionally dependent. Clotrimazole has very good *in vitro* efficacy against Malassezia organisms, however its clinical effectiveness has not been as consistently good. Posaconazole (Posatex) is a product on the market that contains orbafloxacin and mometasone. Nystatin has good efficacy against Candida but poor effectiveness against Malassezia yeast. Thiabendazole is variably effective against Malassezia organisms. Iodine products such as Xenodine has good efficacy against yeast but may stain the haircoat, can break down in exudation, and can cause ototoxicity in cats. Because yeast prefer a waxy and greasy environment with a high pH, products that modify the local environment can be fungi static. Most cleaning and drying types of products contain an acidifying agent such as boric acid, salicylic acid, acetic acid, etc. A 2.5% acetic acid solution can be effective against yeast but if it is applied to ulcerated ear canals it may be painful to the patient. I have used Bur-otic with and without hydrocortisone with good success both as a treatment and also as a maintenance product for dogs that swim frequently. This product contains Burrow's solution which is an astringent (drying agent) and has an acidic pH to limit Malassezia proliferation. HB 101 is a similar product. In refractory cases of Malassezia otitis media, oral ketoconazole at 10mg/kg once daily is an effective adjunct treatment. Products containing Tris-EDTA and ketoconazole are now available. The author has been pleased with the clinical efficacy of these products. There are products for human beings that contain clotrimazole and other antifungal agents. The literature is replete with information supporting efficacy of these products for the treatment of canine yeast otitis.

Corticosteroids can be very beneficial in treating otitis. These products are anti-inflammatory, anti-pruritic, and reduce the amount of sebum and scale. Because of the beneficial effects of corticosteroids, they are added to virtually all of the licensed otic preparations. There are times, however, when corticosteroids are either not necessary or contraindicated. If a corticosteroid is indicated, but not available in the product selected, consider using oral prednisolone rather than adding a steroid to an otic preparation. There are many home concoctions that I have heard about over the years and the only recommendation is to be cautious. Some active ingredients are very unstable, and mixing may precipitate or inactivate them.

For most cases, 14 days of therapy is indicated. If there is inadequate resolution then a recheck cytology should be performed. One of the most common reasons for treatment failure is poor owner compliance. Other possible reasons are resistance of the organism or a different pathogen may be present because of treatment. It is necessary to keep the ears free of debris prior to treatment. I prefer not to send home a cleaning product simultaneously with a medicating product. The residue from the cleaning product may dilute the drug or pH changes may inactivate the active ingredient. It is usually advised to clean the ears before the patient goes home and have the owner return for a recheck if an excessive amount of debris builds up in the canal.

An oral antibiotic or antifungal agent is always indicated if otitis media is present. This should be selected on the basis of a culture and susceptibility results. It is not necessary to culture a sample that contains only yeast organisms. Oral ketoconazole at 10mg/kg qd with food is usually beneficial for dogs with otitis media associated with Malassezia yeast. Oral antibiotics are not indicated as a supplemental therapy for otitis externa under most cases. Severe proliferative otitis externa may benefit from oral antibiotics, however by the time these changes are present, otitis media is usually present.

Otitis media with mild or absent otitis externa does occur. On otoscopic exam the tympanum may appear hyperpigmented, erythematous, or billowing outward. Myringotomy is the term referring to surgical excision of the tympanum for a sample collection for culture and sensitivity. The dog should be heavily sedated or anesthetized for this procedure. Clean and disinfect the ear canal as well as possible with an iodine cleaning agent. Insert a sterile otoscope cone into the canal to visualize the tympanum. Gently insert a sterile spinal needle down the cone and through the tympanic membrane, attempting to stay to the ventrolateral aspect of the tympanum. A syringe should be attached to the needle for aspiration of material from the middle ear canal. This sample should be submitted for culture and susceptibility testing for aerobe and anaerobic bacteria. If the tympanum is highly inflamed, a microtip culturette may be pushed through the tympanum. In normal canine ears, the tympanum may heal in about 3 weeks.

Studies on healing times for inflamed tympanic membranes have not been performed.

Chronic otitis implies an underlying cause. Many chronic ears have been treated with about every steroid and antibiotic imaginable. At first visit careful palpation of the ear canals should be performed to identify calcification. Calcified ears are surgical ears in almost all cases. It is usually recommended to perform a CBC, serum profile, and thyroid profile to identify underlying causes and the effects of chronic therapy. If the patient is currently receiving topical medications containing corticosteroids, T4 levels will likely be lower than normal.

If there is an opportunity to treat the patient medically, then cytology, culture and aggressive ear cleaning under anesthesia is indicated. The selection of a topical medication is based on something stronger than what the patient has already received. A systemic antibiotic should be selected based upon culture and susceptibility testing. Most chronic ears have highly resistant bacteria and antibacterial therapy can be very expensive. It is always best to pick the right antibiotic the first time. Initial therapy should be for 3 weeks with a mandatory recheck. Under most cases an ear cleaning and culture would be indicated. If all is well, therapy should be continued for an additional 3 weeks. Treatment for chronic otitis is neither cheap nor easy! Success is totally dependent on owner compliance.

There are some basic guidelines when surgical intervention is indicated. Calcified ear canals are surgical. Calcification is a chronic progressive and irreversible process that causes tremendous pain for the patient. Ear canals that are fibrotic may be surgical. If it is possible to get medication down the ear canal, all hope is not lost. Stenotic ear canals may be a predisposing factor for otitis or may be a result of chronic inflammation. Historical information may be helpful in defining these circumstances. In some cases a vertical ear canal ablation may be a beneficial adjunct to treatment. However, if an underlying primary cause is not found then relapse is likely. Progressive proliferative changes may lead to occlusion of the ear canal. Surgery is usually indicated to remove this tissue. Pinnal ablation is not the best way to resolve this condition. Ear canal ablation with plastic surgery will yield a cosmetically pleasing result. Because of the difficulty and expense of obtaining diagnostic bulla radiographs, it is usually recommended to allow a specialty hospital to perform this procedure. A C.T is usually more cost effective and a better diagnostic tool than radiology. M.R.I. is a superior diagnostic tool but current procedures are very expensive and time consuming compared to routine C.T.

Ear canal ablation with bulla osteotomy is reserved for cases of medically intractable infection, calcified ear canals, neoplasia of the ear canal or bulla, or chronic changes of the bullae including calcification or lytic changes. Ear polyps are also an indication for surgical intervention. This procedure is difficult and labor intensive after the surgery. It is usually advisable to refer these cases to a reputable surgeon that likes ear surgery. Possible complications include Horner's syndrome due to facial nerve damage and head tilt. Residual sepsis is also a possibility. All dogs with ear canal ablation with bulla osteotomy will have significant hearing loss. Usually they experience substantial hearing deficits prior to surgery and because of the slow progression, most dogs adapt to the handicap easily.

APPENDIX

Ticarcillin Ear Solution:

Concentrate

6g vial of ticarcillin in 12ml of sterile water
Divide into 2ml portions in syringes and freeze

Solution:

mix 2ml concentrate with 40ml sterile saline
divide into four 10ml portions and freeze

once thawed for use it should be refrigerated
discard any solution after 7 days

***Courtesy of Aiden Foster** BVSc, PhD, MRCVS
Foster AP, DeBoer DJ. The role of Pseudomonas in canine ear disease. *The Compendium*, 20(8)1998. 909-919.

Compounded Baytril Otic
4oz bottle of Tris EDTA, remove 16ml
Add Dexamethasone SP 4mg/ml (4ml vol.)
Add Lg Animal Baytril 100mg/ml (12ml vol.)

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Non-inflammatory alopecia in the dog

Non-inflammatory alopecia in the dog can be one of the most challenging of cases. There are more similarities than differences making the diagnosis difficult. We will be evaluating the various underlying problems, diagnostic methods, and treatment options.

Endocrine disorders are notorious for causing symmetrical alopecia in the dog. Hypothyroidism, Hyperadrenocorticism, Hyperestrogenism, and Hypotestosteronism can all look alike. Judicious use of history taking skills along with appropriate laboratory testing may potentially lead to an accurate diagnosis.

Hypothyroidism is usually associated with symmetrical alopecia to varying extent. Additional clinical findings may include lethargy, heat seeking, and weight gain. A "tragic look" is sometimes used to describe affected patients. The alopecia may present as diffuse to complete with variable hyperpigmentation and scale. Sometimes only, the haired skin of the nose is affected. Thyroid testing is straight forward, however many cases are in the grey zone, making a definitive diagnosis challenging without repeated testing. Response to hormonal therapy does not confirm the diagnosis as euthyroid dogs may exhibit hair regrowth due to the stimulation of the hair follicles. Biopsy by itself is not diagnostic; however, enlarged erector pili muscles may be identified. Response to therapy is very good and return to normal hair coat is expected. Incomplete resolution of the alopecia would indicate the need for investigation of a second problem.

Hyperadrenocortism is usually associated with alopecia. Similar to hypothyroidism, the alopecia may be diffuse or complete. Both differential diagnoses should be considered for a dog that presents with an alopecic tail when there is no evidence of self-trauma. Concurrent clinical findings are usually present including: PU/PD, potbelly appearance, polyphagia, and recurrent infection. Most dogs are senior. In the early stages, alopecia with scale are common. If secondary infection or calcinosis cutis are present, the alopecia will be exacerbated and inflammatory. Laboratory testing makes the diagnosis straightforward, usually. Other than calcinosis cutis, which is pathognomonic for iatrogenic or hyperadrenocorticism, there are no key identifying features on biopsy. The hairs may present as miniaturized and the dermis may show thinning.

Hyperestrogenism as a spontaneous disease process is rare. Young intact female dogs may be affected. Clinical signs include ventral alopecia and pruritus. Spaying the dog will be curative. Most dogs have very low circulating levels of estrogen so the diagnosis is made on history, clinical signs, and spaying. Hyperestrogenism is seen with iatrogenic exposure to topical estrogen replacement therapy in people. The alopecia tends to be complete and affects mostly the ventrum. A CBC should be monitored to check for anemia. Clinical signs should resolve once exposure is reduced or eliminated. Dogs should be discouraged from sleeping in the bed of people using estrogen replacement products. Biopsy findings are non- specific for an accurate diagnosis.

Flank alopecia (seasonal flank alopecia) is a clinical finding of dogs with short, bristly hair coats. Commonly affected breeds include the Bulldog, Boxer, Doberman, and Pit bulls. The lesions are usually fairly symmetrical along the flank regions but may extend up the thorax. The alopecia is usually fairly complete with well-demarcated margins. The skin in the affected areas is usually hyper pigmented. Biopsy along with a history of lesions in the area is diagnostic. The histopathologic findings are classic for a definitive diagnosis. Similar lesions may be seen with hypothyroidism and concurrent illness may occur. Flank alopecia is ultimately cosmetic in nature. Treatment with melatonin may be instituted but is likely ineffective in most cases. Although there is breed predilection, the genetics of this problem have not been elucidated.

Pattern baldness is a diagnosis by exclusion. Clinical findings overlap with other diseases and biopsy is not definitive. Pattern baldness usually occurs in young adult dogs and may continue to progress over time. Ears and ventrum are usually affected and the alopecia can be complete or diffuse. Hyperpigmentation is variable. The Dachshund and Chihuahua are over-represented breeds.

Black hair follicular dysplasia is an uncommon problem. It tends to occur in young adult dogs. Only the black hairs are lost in the affected dog. The skin may be hyper pigmented in the alopecic regions. Biopsy in the alopecic areas is diagnostic as dysplastic hair follicles will be present. The diagnosis may be made on clinical findings alone. There is no treatment for these dogs.

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Hepatocutaneous syndrome has in the past been called: superficial necrolytic dermatitis, necrolytic migratory erythema, and diabetic dermatopathy. The age of onset is usually seen in older dogs and there is no breed or sex predilection. Clinical signs are usually associated with scaly to exfoliative dermatitis affecting the foot pads, bony prominences, perianal region and commissures of the mouth. As the lesions on the feet progress, cracking and fissuring of the foot pads with crusting and secondary infection are common. The claw beds are usually affected with increased severity. Perianal lesions may become erosive to ulcerative with secondary infection. Pruritus is variable with increased severity with secondary infection. Serum chemistry may reveal elevations in ALP, and ALT but some dogs may be within normal limits. Serum glucose may be elevated and bile acids are variable. There are usually no overt clinical signs of liver disease in these patients. Ultrasonography of the liver reveals a "honeycomb" pattern which is almost pathognomonic for this disease. Skin biopsy is the gold standard of diagnosis. The pattern of inflammation is classic and is referred to as "red, white, and blue disease". The red represents abundant parakeratosis of the stratum corneum. The white is the marked edema of the epidermis and the blue represents the hyperplastic stratum basale and dermal inflammation. Because of the marked parakeratosis, zinc responsive dermatosis is an important differential. The edema and inflammation are not usually seen with zinc responsive. The prognosis is poor as a cure is rarely achieved. The goal of therapy is quality of life. Most of these dogs have low levels of amino acids so therapy consists of amino acid replacement. The gold standard is IV administration of amino acids such as Aminosyn. The product is very hypertonic and it must be administered very slowly over about 8 hours. Clinical improvement can be seen within a week of administration. Usually it is administered weekly until an adequate remission is achieved, then as needed. The aminosyn is not very expensive but the hospitalization and insertion of a central line (to prevent phlebitis) can be costly. Nutritional support includes: high protein diet, Zn supplementation, the addition of cooked egg whites (yolks are mostly cholesterol) and oral amino acid supplementation. The GNC company makes an amino acid capsule and can be given once or twice daily. Fatty acid supplementation is recommended by some clinicians but there is no evidence of efficacy. Protein powders for people can be offered but are frequently unpalatable for dogs. If secondary infection is present it must be treated topically and parenterally based upon culture and susceptibility. The overall response to therapy is quite variable. Unfortunately, many patients are humanely euthanized.

True fatty acid deficiencies are rare in dogs. We use omega 3 and omega 6 fatty acids to help our atopic dogs. Generally speaking, omega 3 fatty acids are considered to be anti-inflammatory and anti-pruritic. The omega 6 fatty acids help with coat conditioning and epithelial barrier. Arachidonic acid is an omega 6 fatty acid. These can be broken down via the arachidonic acid cascade and can help create pro-inflammatory mediators. The perfect ratio of omega 6 to omega 3 has not been elucidated in vivo. Many over the counter fish oil capsules do not contain enough omega 3 fatty acids to affect pruritus. We recommend about 100mg/kg of combined DHA and EPA once daily for dogs. There are formulations specifically for dogs with skin conditions that are about this concentration. This allows for package dosing accuracy without a lot of calculations. Some diets such as Purina JM are very high in omega 3

fatty acids so additional supplementation may not be needed. Fatty acids and antihistamines are not used as commonly as before because of the availability of alternative antipruritic agents to steroids. Studies have shown that it may take six weeks or longer to see visible changes to the coat condition after adding these supplements.

Vitamin A may be used at higher dosages to treat scaling disorders of dogs. Dosages vary but can be used at about 10,000 IU/day. Side effects may include dry eyes and hepatotoxicity. It had not been shown to be effective for dogs with sebaceous adentitis. It would be considered a treatment of choice for idiopathic seborrhea in dogs. Vitamin A responsive dermatosis (with scale) may be identified with histopathology but is not associated with a pathognomic change. The pathologist will indicate that it is suggestive of but not diagnostic for vitamin A responsive scaling. We generally recommend baseline blood work and Schimer tear test before instituting therapy. We would likely monitor both every 3-4 months, but sooner if there are problems. Synthetic retinoids are rarely used in veterinary medicine due to the likelihood of causing birth defects. This is a concern if people accidentally ingest this medication.

Vitamin E is a free radical scavenger and has antioxidant properties as well as anti-inflammatory properties. It is rarely used as a sole supplement but may have tremendous value as adjunct therapy for many autoimmune skin disorders. The drawback of vitamin E is that it is best given on an empty stomach, 2 hours before or after a meal. This is very challenging for our clients and may be a reason why we may not be achieving maximum benefits from this supplement. Vitamin E supplementation may be considered for lupus-like reactions, and dermatomyositis. At one time it was considered to be a treatment for demodicosis but it turned out that the dogs in the study were severely vitamin E deficient already. Most dog food already has adequate levels of vitamin E so supplementation for clinically normal patients is not indicated. Sources vary but 200-800 IU/day have been suggested, based on the size of the patient. Current formulations make exact dosing impossible. Additionally, many omega 3 fatty acid products formulated for dogs frequently have vitamin E added.

Zinc responsive dermatosis is most commonly seen in Arctic breeds but others can be affected. Age of onset is usually young to middle aged dogs. There is no sex predilection. Clinical signs are associated with increased scale mostly affecting the bony prominences, foot pads, and periorbital regions. Alopecia is variable. There are many differential diagnoses. Biopsy reveals marked parakeratosis suggestive of zinc responsive dermatosis. Parakeratosis can also be seen in healing wounds, hepatocutaneous syndrome, and Malassezia dermatitis. Treatment is zinc supplementation. Zinc sulfate can be given at 10mg/kg/day (or can be divided). Zinc methionine seems to be better tolerated and is given at 2mg/kg/day. The ZinPro product is available as a chewable making administration easy. Cases that do not improve clinically within a few months may benefit from concurrent prednisone/prednisolone at anti-inflammatory doses (1mg/kg/day tapered). Lethal acrodermatitis is an inherited autosomal recessive trait characterized by low zinc levels and immune deficiency. The prognosis is grave with median survival of 7 months. This disease does not improve with zinc treatment.

Probiotics are exogenous bacteria that are healthy for the gut. They help to stimulate the immune system and compete with pathogenic bacteria. Prebiotics are essentially food for our GI bacteria flora. Sources of prebiotics are plant-based fiber including inulin, psyllium, and various brans and flax seeds. One of the side effects of feeding high levels of prebiotics is that they can lead to the production of excess gas and soft stool. Several studies have shown the importance of the gut-brain axis. New products like Nestle Purina Calming Care have shown clinical evidence with this strain of probiotic.

Probiotics are used for acute and chronic diarrhea. They are also important for treatment of antibiotic induced diarrhea. There are many products available on the internet with little scientific or clinical evidence of efficacy. It is recommended that those with a clinical track record should be selected such as those from Nutromaxx or Nestle Purina. In general, human probiotic formulations are not recommended for dogs. The exception is the human product VSL#3 which is sometimes used by veterinary gastroenterologists.

Drug	Subclass	Spectrum	Dosage	Formulations	Toxicity/side effects	Misc
1st generation cephalosporins		G+ Not so great for G- Bad for anaerobes **Staph pseud**	22-30mg/kg BID → 30mg for skin infections	250/500mg capsules 150/300/600mg chewable tablets → $$$	Vomiting (>10%), other GI issues uncommon	**Not in cats!!** → always causes BAD vomiting Tx for 30 days → need to educate the clients on abx resistance and that we only have one shot with this
	Cephalexin	#1 choice for Staph pseud				
	Cefazolin			Injectable – used in emergency – IM + IV, but can use SQ		
	Cefadroxil			50/100/200mg tabs (Cefatab) → have candy coating (enteric coating → less GI upset + allows it to get to the SI)		
2nd generation cephalosporins	Ceftiofur	Bad for G+ Good for G-				Exclusively in food animals
3rd generation cephalosporins		G- rods Anaerobes → broad spectrum so it affects the rest of the body Not as good as 1st gen for staph so we don't consider them to be first line			Vomiting → give Cerenia	
	Cefovecin (brand name Convenia)		8mg/kg SQ Q14d (Convenia: 80mg/ml)	Convenia: short half-life → once opened, only have 56 days until it goes bad, and it		Tolerated by cats! Much more expensive, tends to be used w/ small dogs or if dogs aren't tolerating oral cephalosporins

Drug	Subclass	Spectrum	Dosage	Formulations	Toxicity/side effects	Misc
				needs to be refrigerated		Highly protein bound so stays in body for a long time
	Cefpodoxime (brand name: Simplicef)		5-10mg/kg QD → >7mg for skin, mainly aim for 10mg	100/200mg tablets	vomiting	Broad spectrum and expensive
Amoxicillin + ampicillin		Anaerobes Not good for G+ or G- b/c the bacteria make Beta-lactamase	22-30mg/kg BID (TID better – use higher dosage)	inexpensive and available in many forms Ampicillin IV	Diarrhea, treats anaerobes) → Tx w/ probiotics	Amoxicillin better absorbed in guts in dogs, ampicillin can be IV so good for emergency. Excreted in urine and concentrated in epithelial lining of bladder wall.
	Clavulanate (Clavamox)	Good for G+ cocci	13-22mg/kg TID (higher dose b/c increasing resistance)	62.5/125/250/375mg	Lots of GI upset	The 500mg augmentin has the same ratio as clavamox Binds to B lactam ring and allows it to be interacted w/ Conc, best in epithelial lining of the bladder Covers #1 and #2 most common UTIs
Macrolides	(azithromycin, clarithromycin)	Bacteriostatic, so don't want other drugs suppressing the immune system G+ Great for anaerobes Not for G-			Diarrhea If resistant to one then become resistant to all of them	Conc most in bone → best tx for canine osteomyelitis (mainly caused by Staph pseud) Azithromycin – stays in body for a long time b/c highly protein bound, so induces resistance
	Clindamycin		5.5-11mg/kg BID, 11mg used	25/75/150/300mg capsules and tablets		Most important If staph infxn in skin, HAVE to use 11mg/kg BID 150 + 300mg are cheaper b/c people use it

Drug	Subclass	Spectrum	Dosage	Formulations	Toxicity/side effects	Misc
						Tx of choice for dogs + cats w/ dental dz Resistance occurs v quickly Staph schleiferi – resistant to cephalexin + susceptible to clinda
	Erythromycin					Oldest and worst, except for use against Rhodococcus equi w/ Rifampin (horses)
	Lincomycin					All pigs are resistant b/c they were treated for greasy pig dz aka Staph hyicus
Fluoroquinolones	(marboflox, orbiflox) Marbo: Zenequin	G+ Used to be good for G- rods but many have resistance now Not for anaerobes Good for mycoplasma b/c they don't affect the cell wall + myco doesn't have walls	Marbo: 5mg/kg QD	$$$	Cartilage defects in young dogs	Help to induce multiple drug resistance → may be why we so much resistant Staph now Only use w/ C + S Inhibit DNA gyrase Dose dependent, not time-dependent (like aminoglycosides) Orbi: crappy when used orally, but great for ophthalmic Conc v highly in WBC + bone so good for osteomyelitis
	Enrofloxacin (Baytril)		2.5-20mg/kg QD	Oral, 36/68/135mg tablets – also injectable	Cats – retinal degeneration **IV bolus may induce seizures**	Metabolised to active metabolite ciprofloxacin as bonus
	Ciprofloxacin		30mg/kg qd		Induces multiple drug resistance	Poorly available to dog → 40-60% bioavailability in canine gut
TMS					Resistance develops very quickly so used potentiated w/ sulphonamides → the	

Drug	Subclass	Spectrum	Dosage	Formulations	Toxicity/side effects	Misc
					whole is better than the sum of its parts	
TMPS		Tx of choice for Nocardia			Folic acid inhibitor so causes blood dyscrasias (→ anemia) so need to monitor CBC → only use based on C + S Can also affect eyes (KCS) → do Schirmer tear test before to check that the eye is normal beforehand	Side effects are not dose dependent Endocrine: decr total T4 → incr TSH → will look like hypoT → takes a minimum of 6 weeks for pituitary thyroid axis to go back to normal (8wks is better)
	Ormetroprim sulfa			Primor	Doesn't affect TT4 → better + safer Still has the other side effects though	
Aminoglycosides	(amikacin, gentamycin)	Bactericidal G+ Sometimes G- Not for anaerobes Often good for highly resistant Staph (do C + S)	15mg/kg QD Can't give oral		Nephrotoxic Ototoxic	Generally, don't do well in the skin Ototoxic – toxic to CN8; kills the hair cells in the cochlea Don't give oral b/c they break down v quickly in acidic environments → neutros make it acidic when they burst so they will break down v quickly in exudative ears Not absorbed well orally either
Cyclines	(tetracycline)					Highly lipophilic so great for Txing intracellular parasites Sometimes highly resistant Staph is resistant → use mino (C + S first)
	Doxycycline		5-15mg/kg BID		Diarrhea	

Drug	Subclass	Spectrum	Dosage	Formulations	Toxicity/side effects	Misc
					Cats: esophageal strictures	
	Minocycline		5-10mg/kg BID for Staph		Makes dogs generally feel lousy	Safest + fewest side effects
Chloramphenicol		G+ cocci Great for anaerobes Bad for G-	35mg/kg TID (must be TID!)		Aplastic anaemia in people Diarrhea guaranteed Hind limb weakness – worse in larger breeds – reverses once stopped	Good for getting into almost all tissue compartments eg BBB, uterus, prostate quickly develops resistance
Rifampin		Used for highly resistant Staph	6mg/kg QD + need careful monitoring		Highly nephrotoxic so do CBC + liver panel weekly	Dye w/ multi-bacti properties → pee is fluorescent Almost always used w/ other drugs b/c resistance WILL occur