Folliculitis: Dermatophytes, *Staphylococcus* spp, and Demodicosis

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Dermatophytes

Dermatophytes are fungal organisms that invade, live in, and affect keratinized structures, i.e., the horny layer of the epidermis, hair, and claws. Under normal circumstances, they do not invade, nor can they survive in living tissue. In cats and dogs, most cases are caused by *Microsporum canis* (some cases have shown this to be a ‘natural’ part of the skin flora of healthy cats, although this finding has been disputed, and may relate to where the ‘healthy’ cats were found – in private homes or in pet shelters, for example). Less common species implicated in dermatophytosis in cats are *Microsporum gypseum* and *Trichophyton mentagrophytes* – these species are more common in dogs.

Transmission is usually animal to animal but may be soil to animal in the case of *M. gypseum*. Human to animal is quite rare, but animal to human (*M. canis* infection from cats to children) certainly may occur. A number of factors influence the susceptibility of the animal to a dermatophyte infection: age (young are more susceptible), nutritional status, concurrent disease (ie retrovirus infections), immunosuppressive drugs, etc.

The invasion of the hair shaft with fungal elements weakens it and facilitates breakage. The fungi elaborate substances that can act either as ‘toxins’ (primary irritants) or even as allergens. Clinical features may include alopecia, crusting, erythema, pruritus, papular eruption (‘miliary dermatitis’) and claw damage. Occasionally, deep infections resulting in draining tracts and/or nodules (pseudo-mycetomas) may be seen.¹ ² Dogs that like to dig in the ground and/or roam outside sometimes present with dramatic crusts and erythema on the face, the ears, with progression to the legs and even the foot pads. Often, there is intense pruritus as well. These dogs have an infection with either *T mentagrophytes* or *M. gypseum*. Do NOT be dissuaded by a lack of contagion in other animals or the owners, or even by a negative dermatophyte culture - *T mentagrophytes* especially can be difficult to culture. A biopsy, with special stains for fungi, may show the organism quicker than a culture; histology may occasionally show acantholytic cells.²b A clue is that while the face, especially the muzzle may be affected, the nasal planum (due to the lack of hair follicles) is usually spared.

Diagnosis of dermatophytosis is made by history, clinical signs, and the following: Wood’s light - must be allowed to warmup for at least 3-5 minutes. Hand-held Wood’s lamps are not effective. *M. canis* is the primary dermatophyte affecting cats that will produce fluorescence. Fluorescence is caused by tryptophan metabolites. As it is the infected hairs that fluoresce, sample these hairs for culture (fluorescence will not occur in the cultured colonies). The fluorescence is bright yellow-green; comparable to the face of watch. Not all *M. canis* infections will exhibit fluorescence, perhaps only 30-50 % will, so DO NOT rule out dermatophytes based on a negative Wood’s lamp test!
Direct microscopic examination – difficult to do. Scales and hairs are collected by plucking or scraping the suspected lesions. The material is transferred to a glass slide, a drop or two of 10 - 20% KOH or chlorphenolac added (or even mineral oil), a cover slip applied (the KOH preparation would be gently heated), and then examined microscopically. Spores should surround, and hyphae be within, hair shafts.

Fungal cultures - The most definitive method of diagnosis. The area to be cultured should be gently cleansed with water (although the author rarely does this). Material is gathered in the same method as for direct examination. Fine scales and broken hairs should be obtained: large crusts or tufts of hair should be avoided. Using a toothbrush to obtain samples from an affected carrier cat may also be used. Material may be sent to an outside lab or an in-house method may be used. Dermatophyte Test Media (DTM) is the usual media utilized. The principle behind the media is the presence of phenol red, a pH indicator. Before use, DTM is amber in color. Dermatophytes utilize the protein in the media and produce alkaline metabolites that turn the media red; most saprophytic fungi and bacteria utilize the carbohydrate in the media and produce acidic metabolites which cause no color change. REMEMBER that after using up the carbohydrates, the saprophytes will switch to protein metabolism and turn the media red.

For proper use and interpretation of DTM, several precautions need to be taken:

1) The hairs and scales should be pressed into the agar but not buried.
2) The cover should be loose to allow for adequate aeration.
3) Incubation should be done at room temperature.
4) The media should be examined daily.
5) With a dermatophyte, the red color should occur at the time the colony is first visible.
6) After prolonged growth, most saprophytes will eventually turn the media red.
7) Dermatophytes should be fluffy, light colored colonies.
8) Any colony that has a green or black coloration should be regarded as a contaminant.

A wet mount or ‘scotch’ tape preparation of the colony surface may be done for speciation purposes. This should be stained with lactophenol cotton blue (or the blue dye from a DiffQwik™ stain).

Biopsies – This is not a normal method of diagnosis, but if positive, a quicker method than culture. Usually this is the method of diagnosis when a dermatophyte infection is not being considered and the biopsy was taken for other reasons. Special stains (GMS, PAS) help to identify fungal organisms in tissue.

Treatment

Itraconazole (10 mg/kg once daily) is an anti-fungal triazole compound. It has been considered the first drug of choice in the treatment of feline dermatophytosis.³ It is lipid-soluble and best given with food to enhance intestinal absorption. Cats tolerate itraconazole better than ketoconazole, although they may rarely develop hepatic toxicosis and anorexia. Ketoconazole at 10mg/kg is the preferred drug for dogs. Neither drug should not be used in pregnant animals; adverse effects in kittens have not usually been observed. Itraconazole comes in 100 mg capsules (Sporanox®; Janssen). The capsules contain small pellets that can be put into food. This is especially useful in treating cats: a capsule’s pellets are mixed with a tablespoon of butter, frozen, and the
butter then ‘halved’. Each quarter contains 50 mg itraconazole, which is the standard dose for a 5kg cat. Alternatively, Sporonox® is available in a pediatric suspension (10mg/ml), but this is rather expensive. There are various regimens recommended for this drug, including 15 days on, 15 days off (during the last 5 days of which, a fungal culture is repeated), then 15 days on until cultures are negative; another regimen is 28 days on, then alternating weeks on and off until cultures are negative.

Fluconazole is available as a generic. The author has used it at 5mg/kg q12 hr to good effect in several cats. It is less expensive than itraconazole and well tolerated. It is mainly metabolized in the kidneys, so should be used with caution in cats with reduced renal function.

Terbinafine (Lamasil®: Novartis, also available as a generic) is an allylamine antifungal agent. It is well tolerated by cats and should be administered at a dose of 30-40 mg/kg orally once daily. It may be used in dogs and cats. It has successfully treated a dermatophyte (pseudo-) mycetoma. It has not caused problems in pregnancy in humans.

The value of clipping dermatophyte-infested cats has been questioned: clipping may spread the organism and provide it access through an abraded epidermis. Lime-sulfur (Sulfurated Lime, Dechra Overland Park, KS 66211 USA at 1 cup to a gallon of water is also an effective topical treatment modality. Topical miconazole shampoos and leave-on conditioners have become more popular than older topical antifungal products. Patient compliance is, needless to say, a concern.

Treatment of the environment – ‘The most important part of decontamination is the “hard clean”: mechanical removal of debris followed by washing of the surface with a surface safe detergent until visibly clean. Disinfectants are only needed to kill spores remaining after the hard clean (often the hard clean alone will remove all of the infective spores). A 10 minute contact time is recommended. If there are only one or two infected pets AND OWNERS ARE USING A TOPICAL ANTIFUNGAL ON THE PET(S), owners can do a hard clean/disinfectant application once or twice a week provided they do adequate mechanical removal of debris followed by a one-step cleaner with antifungal efficacy against Trichophyton mentagrophytes on the label. There are many alternatives to bleach, accelerated hydrogen products are a good option.

Minimally, all bedding should be disposed of, as should all air filters. Vacuum exposed areas thoroughly on a daily basis. Use 10% bleach on nonporous surfaces daily, and soak grooming tools in 10% bleach or discard. Clipping long-haired cats or cats with generalized disease (#10 blade) is controversial.

In cases of multicat households, catteries, or shelters, there are two excellent articles. Briefly put, in the latter study a three-area system was used: healthy animals (no lesions and negative cultures), subclinical carrier animals (no lesions but with positive cultures) and clinically affected animals (lesions and positive cultures). The cats were examined and inspected under a Wood’s lamp and had samples taken for fungal culture every 2 weeks. Thirty-three per cent of the cats had a positive fungal culture at the start of the study. Clinically affected animals and carriers were treated with a 0.2% enilconazole lotion (Imaverol® – not available in the USA) twice a week and given itraconazole 5 mg/kg once daily orally every other week. The environment was treated once a day with a 1% bleach solution and once a week with a 0.6% enilconazole (Clinafarm) solution. Treated animals were considered cured after two consecutive
negative fungal cultures. All cats were cured within 56 days. No relapses were observed based on the fungal cultures taken from the cats and the environment over the first 10 months.  

References

Specifics on Feline Dermatophytes
The author is indebted to Dr. Karen Moriello, College of Veterinary Medicine, University of Wisconsin, and the World Association for Veterinary Dermatology’s Guidelines for the Treatment of Dermatophytosis (available at www.WAVD.org).

INTRODUCTION
*M. canis*, the most common cause of dermatophytosis, is NOT part of the normal skin microbiome of cats. Dermatophytes over all are not that common: a study from the United States detailing the causes of skin disease in cats revealed that dermatophytosis was 136 found in only 45 of 1407 (2.4%) cats.

Dermatophyte spores don’t live in the environment, and do not multiply on, they can’t grow in any of the home surface – they are like dust, not mildew, easily removed by mechanical cleaning and washing with a detergent and water. Spores do not represent a respiratory risk.

RISK FACTORS
-Seropositive FIV and/or FeLV status in cats alone does NOT increase the risk of dermatophytosis.
- There are NO reports of dermatophytosis in pemphigus foliaceus-afflicted cats treated with immunosuppressive drugs.
- Persian cats maybe at greater risk to develop dermatophytosis, almost certainly to develop the sever subcutaneous form.
- Cats from large scale hoarding environments were found to be at a higher risk of dermatophytosis
- Microtrauma from bites/scratches, ectoparasites, matted hair, maceration of the skin from high humidity associated with dampness or post-cleaning – all are risk factors
- Cat's inability to groom

**DIAGNOSIS**
Confirmation of clinical suspicion is needed, not only for treatment, but also to limit contagion to other susceptible animals and people.

Questions to determine a diagnostic test’s application:
1. What test(s) confirm the presence of an active infection in order to make an informed decision, i.e. treat or not treat, euthanize, quarantine?
2. What test(s) confirm the absence of an active infection, i.e. the animal poses no infection risk, when is the animal cured?

**Conclusions: No one test is the ultimate “gold standard”**.
Dermatophytosis is diagnosed by utilizing a number of complementary diagnostic tests, including Wood’s lamp and direct examination to document active hair infection, dermatophyte culture by toothbrush technique to diagnose fungal species involved and monitor response to therapy, and biopsy with special fungal stains for nodular or atypical infections.

Wood’s lamp: The anecdotal comment that “not all ‘strains of M. canis will fluoresce on all cats” is NOT supported by the findings from the experimental studies in which experimental infection resulted in 100% fluorescence in all cats.

Another anecdotal statement is that bathing or topical therapy will ‘change or destroy fluorescence’. Reviews of experimental or field studies using Wood’s lamp examinations to monitor response to therapy did NOT report loss of fluorescence due to topical shampoo therapy or with the use of lime sulfur or enilconazole rinses. Wood’s lamps must be the kind that plug into the wall, as battery-powered give inconsistent results. They do NOT have to ‘warm up, but human eyes need time to adapt to the dark. The metabolite that grows is only seen on the hairs, it will not appear in culture. One characteristic finding in cats under treatment or after cure is the persistent presence of “glowing tip fluorescence will remain long after the hair shafts are culture negative. In Dr. Moriello’s laboratory, hairs were observed to retain fluorescence even after 18 years!

Wood’s lamp examination is likely positive in most cases of *M.canis* dermatophytosis. Fluorescing hairs are most likely to be found in untreated infections; fluorescence may be difficult to find in treated cats. False positive and false negative results are most
commonly due to inadequate equipment, lack of magnification, patient compliance, poor technique or lack of training.

**PCR (polymerase chain reaction):**
PCR detection of dermatophyte DNA can be helpful, however a positive PCR does not necessarily indicate active infection, as dead fungal organisms from a successfully treated infection will still be picked up on PCR, as will non-infected fomite carriers.

Negative PCR in a treated cat is compatible with cure. Negative fungal culture from cats with no lesions and a negative Wood’s lamp (except for glowing tips) is compatible with cure. From a medical-legal perspective the most established method to confirm species identification of a dermatophyte from a positive direct examination preparation or a lesional, suspect animal is via fungal culture with a brush technique usually used to collect hairs and/or scale.

**Culture:**  
There is **NO** advantage to keeping the culture in the dark, or in 25 vs 30 vs 37 degrees C.  

A culture plate with one cfu (colony forming unit) per plate and another with confluent growth would both be reported as “positive”, but need to be interpreted differently with the latter being typical of an animal with true infection and the former a recovery or fomite carriage. Two negative cultures are often used to confirm cure.

**Direct examination of hairs:**  
The following is recommended to simplify and maximize the success of this diagnostic test:  
- Examine only Wood’s lamp positive hairs.  
- Hairs need to be plucked in the direction of growth using forceps and mounted directly into a small drop of mineral oil.  
- Clearing agents are not needed. The advantage of mineral oil is that it is readily accessible and will not damage the microscope lens.  
- A drop of new methylene blue can be added to the mineral oil. The fragile and damaged hairs will absorb the stain, making them easier to visualize.  
- One potentially frustrating problem with direct examinations can be the difficulty finding suspect hairs microscopically. This is readily solved by holding a Wood’s lamp over the microscope slide to locate the fluorescing hair(s) and then repositioning the slide to bring them into the field of vision. Once the hairs are located, normal illumination can be used.  
- Infected hairs can be readily identified at x4 or x10 magnification, appearing pale, wide and filamentous compared with normal hairs  
- On high magnification (x40) cuffs of arthrospores are visible. *M canis* is an ectothrix infection and careful examination will reveal large cuffs of spores on the surface of hairs before the hair shaft is invaded by the organism.

It is cost effective to be proficient in performing a Wood’s lamp and direct examination of hair and scale.

**Biopsy:**
Review of the literature reveals three clinical presentations where diagnosis via skin biopsy has been reported. The first is the work up of a non-healing wound or nodule caused by dermatophytosis (kerion, pseudomycetoma, mycetoma). Histological similarities between dermatophytosis and pemphigus included acantholytic intra-epidermal pustules and interface dermatitis, although this has not been reported in cats. The third is the work up of animal with unusual skin lesions not easily attributed to other causes. In any of these situations, routine haematoxylin and eosin staining (H&E) may or may not identify dermatophytes and, as such, Periodic acid-Schiff (PAS) and Grocott methenamine silver (GMS) are used. Histological staining cannot identify the dermatophyte species

TREATMENT

Topical Therapy:

Conclusions

Twice weekly application of lime sulfur, enilconazole (not available in the USA) or a miconazole/chlorhexidine shampoo are currently recommended effective topical therapies in the treatment of generalized dermatophytosis in cats.

Accelerated hydrogen peroxide products as well as climbazole and terbinafine shampoos show promise, but cannot be definitively recommended until more in vivo studies documenting efficacy are available.

Miconazole shampoos are effective in vitro but in vivo are most effective when combined with chlorhexidine. Chlorhexidene alone is poorly effective and NOT recommended.

For localized treatment, clotrimazole and miconazole have some data to document effectiveness. These are recommended as concurrent treatments, but not as sole therapy.

More on Lime sulfur (calcium polysulfide) leave-on:

Lime sulfur can be used at a concentration of 4 or 8 oz/gallon (15 to 30 mL/L); however in shelters the clinical impression is that the higher concentration was more efficacious as determined by shorter treatment times.

Documented cutaneous side effects of lime sulfur were drying of the footpads, loss of hair on the ears, drying of the hair coat, and with repeated application yellow discoloration of the hair coat of white cats. Oral ulceration associated with an irritant reaction from contact (i.e. licking) with lime sulfur on the hair coat has not been documented in any of the shelter studies where it was used at a dilution of 8 oz/gal or 30 ml/L. Reports of oral ulcerations in cats under treatment in shelters occurred concurrently with fever and development of upper respiratory infections and did not have an irritant pattern. There are two likely explanations for these reports. The first is confusion with ulcers associated with upper respiratory infections. The second explanation is dilution error of resulting in a solution that is 3x-4x as concentrated.

SYSTEMIC THERAPY

Conclusions

Itraconazole (non-compounded) and terbinafine are the most effective and safe treatments for dermatophytosis.

Ketoconazole, fluconazole are less effective treatment options and ketoconazole
has more potential adverse side effects.

- Griseofulvin is effective but also has more potential side effects compared to itraconazole and terbinafine.
- Lufenuron has NO *in vitro* efficacy against dermatophytes, does not prevent or alter the course of dermatophyte infections, does not enhance the efficacy of systemic antifungal or topical antifungal treatments and has no place in the treatment of dermatophytosis.

Antifungal vaccines in cats do NOT protect against challenge exposure but may be a useful adjunct therapy.

**More on Itraconazole:**

Due to its highly lipophilic character, itraconazole accumulates in adipose tissue and sebaceous glands. Distribution to these tissues is extensive and tissue concentrations are many times higher than plasma concentrations. Levels in the stratum corneum of skin areas with a high density of sebaceous glands were up to 10 times higher than plasma levels. In people, the drug has been shown to persist in the epidermis for up to 4 weeks post discontinuation of treatment. Concentrations of itraconazole in cat hairs were measured after domestic short haired cats received 5 mg/kg or 10 mg/kg once daily for 14 days. The drug was rapidly detected in all hairs, however lower concentrations were found in areas with fewer sebaceous glands or slower hair growth. Hyporexia, vomiting and/or diarrhea were the most common adverse effect, Review of the literature did not reveal any documented cases of fatal liver toxicity in cats receiving therapeutic doses for dermatophytosis.

A commonly used protocol is 5mg/kg per day, one week on, one week off for a total of 6 weeks (so a total of 21 days of itraconazole).

**More on Terbinafine:**

- Terbinafine is a synthetic allylamine, and exerts is antifungal effects by inhibiting fungal sterol biosynthesis.

- Compared to itraconazole, fluconazole, ketoconazole and griseofulvin, terbinafine has the lowest mean inhibitory concentration (MIC) for *Microsporum* sp. and *Trichophyton* spp. However, authors concluded that *M. canis* was not significantly less susceptible to terbinafine compared to other dermatophytes. It is important to remember that the drug is stored in body fat and differences between studies may be due to age of the cats, body condition score, and number of hairs in anagen (i.e. kittens vs adult cats). In one study, eight weeks after the last dose of terbinafine, eight of 10 cats had hair concentrations of terbinafine above the MIC used for the common dermatophytes.

Because this drug is not licensed for use in small animals there are no published target animal safety studies for review. A safety and tolerability study in cats receiving either 10-20 mg/kg or 30-40 mg/kg terbinafine orally revealed no changes outside of normal laboratory ranges for serum biochemistries or complete blood counts. In a another study, two cats were reported to develop systemic clinical signs including lethargy, anorexia and weight loss one week after the 14 day drug trial. In addition, these two cats developed intense facial pruritus and a macular to papular skin reaction seven to 14 days after discontinuation of the drug. Histological findings were suggestive of an allergic reaction. (The cats in this study were privately owned, lived in a semitropical region, and it is unknown if the cats were from the same household.).
A protocol of 30mg/kg once daily would seem safe to use.

ENVIRONMENT

Conclusions

Environmental decontamination’s primary purpose is to prevent fomite contamination and false positive fungal culture results.

- Infection from the environment alone is rare.
- Minimizing contamination can be accomplished via clipping of affected lesions, topical therapy, and routine cleaning.
- Confinement needs to be used with care and for the shortest time possible.
- Dermatophytosis is a curable disease, but behavior problems and socialization problems can be life-long if the young or newly adopted animals are not socialized properly. Veterinarians need to consider animal welfare and quality of life when making this recommendation.

Infective material is easily removed from the environment; if it can be washed, it can be decontaminated.

- Spores don’t live in the environment, and do not multiply on or grow on any of the home’s surface areas – they are like dust, not mildew, easily removed by mechanical cleaning and washing with a detergent and water. Spores do not represent a respiratory risk.
- If it can be washed, it can be decontaminated. Laundry should be washed 2x or until all visible hair has been removed. Bleach and hot water are not better than cold water. The most important was long agitation times and not over stuffing the washing machine.
- Carpets can be disinfected with hot water extraction, repeated washing with carpet shampooer or if needed after pre-treatment with a disinfectant.
- Hard surfaces (floors, countertops if cats have access, etc.) are disinfected by mechanical removal of debris, washing with a detergent until visibly clean, then rinsing and subsequent removal of all water. Disinfectants are only need to kill spores remaining after the hard clean. If there are only one or two infected pets and a topical antifungal treatment is being used, owners can do a hard clean/disinfectant 1-2 times per week. Accelerated hydrogen peroxide is as effective as bleach.
- There are no safe surface disinfectants for wood floors, however Dr. Karen Moriello has successfully decontaminated wood floors via daily removal of hair and dust using commercial disposable cleaning clothes designed for dry mopping floors (Moriello 2016, unpublished data). Floors were then washed twice weekly with a wood oil soap for floors.

Finally: Monitoring of response to therapy includes clinical response, use of Wood’s lamp if possible, and fungal culture. The number of colony forming units is helpful in monitoring response to therapy (see second paragraph under Culture, above).

FURTHER READING:


Moriello KA, Hondzo H. Efficacy of disinfectants containing accelerated hydrogen peroxide against conidial arthrospores and isolated infective spores of *Microsporum canis* and *Trichophyton* sp. *Vet Dermatol*. 2014;25:191-4


**Staphylococcal Folliculitis**

**Cause:** *Staphylococcus pseudintermedius*, *S. aureus*, and *S. schleiferi*. Occasionally coagulase-negative staphylococcal species.

**Affected hosts:** Dogs, less commonly cats.

**Geographic Distribution:** Worldwide

**Major Clinical Signs:** epidermal collarettes, pruritus, erythema, papules, pustules, alopecia.

**Human Health Significance:** *S. pseudintermedius* prefers not to colonize humans, but zoonotic transmission has been reported. *S. aureus* infections in dogs likely represents reverse zoonotic transmission from humans to dogs. *S. schleiferi* has also been reported to infect humans.

**Etiology and Epidemiology**

The bacteria most commonly associated with pyoderma in the dog are *Staphylococcus pseudintermedius* (previously identified as *S. intermedius*)¹, *S. aureus*, and *S. schleiferi* (including the coagulase-negative variant) roughly in that order of frequency.²,³ The role of other coagulase-negative staphylococcal (CoNS) species is probably more important than previously thought.⁴
Gram-negative bacteria such as *Pseudomonas aeruginosa*, *Proteus* spp., and *Escherichia coli* may be involved in dogs with deep pyoderma. These organisms are thought to invade secondary to alteration of local environmental conditions by *S. pseudintermedius*.

*S. pseudintermedius* is a normal resident of mucosal sites such as the anus and nares and is thought to colonize the skin transiently following grooming and excessive licking in dogs with pruritus. In contrast, the resident flora of the canine skin includes coagulase-negative staphylococci, *Micrococcus* spp., α-hemolytic streptococci, aerobic coryneforms, *Acinetobacter* spp., and anaerobes. The exact number of species that make up the canine cutaneous biome is continually expanding as various higher throughput methods are brought to bear on this research. Similar research is ongoing in the cat.

In the cat, pyoderma is less common, and is more likely to be caused by either *S. aureus* or *S. pseudintermedius*. Feline acne is often a form of deep pyoderma, complicated by keratinization abnormalities.

Pyoderma is usually secondary to some disruption in the animal’s cutaneous barrier and/or immune system. The usual primary diseases are allergic (flea allergy, atopic dermatitis, cutaneous adverse food reaction [CAFR]), endocrine (hypothyroidism, hyperadrenocorticism, diabetes mellitus [less commonly]) or demodicosis, but other diseases that cause severe pruritus or suppress the immune system have occasionally been implicated.

Over the past decade, clonal spread of methicillin-resistant *S. pseudintermedius* (MRSP) has occurred across Europe and North America. Methicillin resistance has also been described among *S. aureus* (MRSA) and *S. schleiferi* (MRSS) isolates from dogs and cats with pyoderma. These methicillin-resistant staphylococci (MRS) encode an altered penicillin binding protein that confers resistance to all β-lactam antimicrobials, and many also demonstrate resistance to other antimicrobials becoming multiple drug resistant (MDR) isolates.

**Clinical Features**

*Staphylococcus* spp may infect the superficial epidermal layers that lie immediately under the stratum corneum and/or the portion of the hair follicle above the sebaceous duct (the infundibulum). This is the most common type of pyoderma. It includes superficial bacterial folliculitis, superficial spreading pyoderma, and "puppy pyoderma" (also known as impetigo or juvenile pustular dermatitis). Early lesions of superficial pyoderma are erythematous follicular papules (papules from which a hair shaft protrudes). As pus accumulates within the epidermis and hair follicles, these lesions become pustules. Papules and pustules are primary skin lesions. Epidermal collarettes are circles of epidermal scale with a free edge facing toward the center of the circle and may be remnants of ruptured pustules. In thick-coated dogs, the scale may become trapped throughout the haircoat as epidermal collarettes exfoliate. A recent article showed that microscopically epidermal collarettes consisted of interfollicular, epidermal spongiotic pustules. This may mean that epidermal collarettes do not always denote a folliculitis, although they almost always denote a pyoderma. Hair fragments are shed from damaged follicles, which results in alopecia. Crusted papules, epidermal collarettes, and alopecia are secondary lesions.

Because the lesions of bacterial pyoderma are directly visible, in many cases, diagnosis can be made based on a thorough physical examination. Use of a hand lens may be helpful. In some cases, clipping of small areas of the haircoat may be required. This can help to identify epidermal collarettes in dogs with dry scale. Careful attention should be paid to the presence of follicular...
papules, pustules, crusted papules, epidermal collarettes, alopecia, nodules, fistulous tracts, and the presence or absence of pruritus. The distribution of lesions should be noted because it can be a clue to underlying disease, such as demodicosis or flea allergy. In dogs with deep pyoderma, the skin may feel warm to the touch and peripheral lymphadenomegaly may be detected.

**Diagnosis**

The diagnostic approach to pyoderma should involve confirmation of the presence of pyoderma and a thorough search for possible underlying causes. In addition to a thorough physical examination, diagnostic procedures that may be necessary include skin scrapings, cytologic examination, aerobic bacterial culture and susceptibility, and skin biopsy. Identification and subsequent treatment of underlying allergic skin disease may require additional testing such as intradermal skin testing, serologic tests for environmental allergy, and/or a limited allergen diet trial.

**Skin Scrapings**

Skin scrapings should be performed in any dog suspected to have pyoderma to assess for underlying demodicosis. Pustules or papules that are associated with hair follicles should be targeted, because *Demodex* mites have a predilection for this site.

**Cytologic Examination**

Cytology can be performed on tape preparations of the skin or smears made after swabbing pustules or draining tracts. Cytologic examination is essential in order to identify concurrent infection with *Malassezia* spp. Slides should be air-dried and stained with a modified Wright’s stain such as Diff-Quik®. The presence of cocci suggests a staphylococcal infection. Infection is supported by the presence of degenerate neutrophils and intracellular cocci. Inflammatory cells may be absent in dogs with underlying immunosuppressive disorders or those receiving glucocorticoid treatment. It is the opinion of the authors that the failure to see cocci on cytologic examination does NOT rule out a staphylococcal pyoderma, especially if clinical signs such as epidermal collarettes are present.

**Culture and Susceptibility Testing**

Due to the rapidly increasing prevalence of MRS worldwide, aerobic bacterial culture and susceptibility testing is playing a more important role in diagnosis of canine pyoderma. While probably not necessary for dogs with a first-time diagnosis of pyoderma culture, susceptibility testing is never contraindicated and should always be offered in dogs with: recurrent or refractory pyoderma, a history of recent treatment with systemic antimicrobial drugs, or who have had surgery or a hospital stay within the previous 6-12 months. Refractory pyoderma may be defined as pyoderma that fails to respond to treatment within a 3- to 4-week treatment period. Culture also can definitively identify the staphylococcal species involved, which may have public health implications. The results of culture and susceptibility testing must always be interpreted in light of the clinical signs present and any history of antimicrobial drug treatment.

The best lesions for culture in dogs with superficial pyoderma are pustules. A thorough search for pustules is recommended. If pustules cannot be detected, culture can be performed on swab
specimens collected from the skin that lies beneath crusts or from epidermal collarettes. No surface antisepsis should be performed. Any hair should be clipped from the lesion using sterile scissors, and crusts should be lifted with sterile forceps. Pustules can be ruptured using a sterile needle, and a swab used to obtain purulent material. Papules can be biopsied using local anesthesia and submitted for culture. In this case, surface antisepsis with a single 70% alcohol wipe may be performed before collection of the biopsy. Culture of biopsy specimens from nodules and furuncles is best for dogs with deep pyoderma. After collection of the biopsy, the epidermis can be removed using a sterile blade and the deeper tissues submitted for macerated tissue culture.

Minimum reporting by microbiology laboratories should include complete speciation of staphylococci – regardless of tube coagulase status– and complete antibiotic susceptibility results (antibiogram). The pathogenic potential of any CoNS isolate obtained from a skin lesion should be interpreted in light of the clinical presentation and with respect to any other pathogenic species of bacteria that may be co-isolated with it. Other suggestions about interpretation of antibiograms are:

- Beta-lactam antibiotics should not be used for MRS infections, irrespective of susceptibility results. This includes third generation cephalosporins (cefovecin and cefpodoxime) as they do not have efficacy against MRS.

- When possible, fluoroquinolones should be avoided for empirical therapy when an MRS is suspected.

- If the staphylococcal species is reported as susceptible to clindamycin but resistant to erythromycin, another antibiotic should be chosen, as this may be an organism in which clindamycin may induce its own resistance.

- Choice of a tetracycline antibiotic is complicated by the fact that resistance to these antibiotics is mediated by four different genes. The genes most commonly expressed by S. pseudintermedius are tet(M) and tet(K). Strains which possess only tet(K) maintain susceptibility to minocycline but not to other tetracyclines. The newly approved canine breakpoints for doxycycline are a reasonable surrogate for minocycline susceptibility.

Histopathology
Findings on histopathology in dogs with uncomplicated pyoderma include a neutrophilic infiltrate and abundant bacterial organisms. Folliculitis, furunculosis, or the features noted in epidermal collarettes (noted above) are all potential findings. Histopathologic evaluation of skin biopsy specimens allows classification of the extent and type of pyoderma and identification of other conditions such as allergic dermatitis, demodicosis, dermatophytosis, pemphigus foliaceus, neoplasia, deep mycoses, and vasculitides. Biopsy should be considered in recurrent or refractory cases, in dogs with deep pyoderma, or when the underlying cause is not apparent. Biopsies can be performed under local anesthesia using a 6-mm punch biopsy. When possible, the biopsy sample can be halved with half submitted for culture and half for histopathology. The biopsy site should be sutured after the specimen has been collected.
Polymerase chain reaction/ Molecular typing
Molecular strain typing methods are research tools used to investigate the epidemiology and ecology of MRS. However, the clinical value of strain typing largely depends on the organism’s population structure, the typing method(s) used, and the goals of the investigation. Strain typing often has no impact on patient- or clinic-level management, even in the context of most outbreaks.20

Treatment and Prognosis
Factors to consider when treating dogs for pyoderma include the underlying disease, the severity and extent of lesions, and the local geographic prevalence of staphylococcal resistance. Veterinarians’ reliance on systemic antimicrobial drug treatment has been challenged by the increasing prevalence of multidrug-resistant staphylococci. Topical treatments that help to restore skin structure and function, as well as topical antimicrobial therapy, should be considered as alternatives. In dogs with mild disease, treatment aimed at the underlying disorder may be sufficient to resolve infection (see Prevention).

Topical Treatments
Topical therapy should be used as the sole on-animal antibacterial treatment for surface and superficial infections whenever the pet and owner can be expected to be compliant.

Antibacterial shampoos may be effective alone in some dogs with surface or mild superficial pyoderma and should be used as adjunctive therapy in dogs with deep pyoderma. They aid in debridement, reduce surface bacterial numbers, and decrease pain and pruritus. Options include shampoos that contain ethyl lactate, chlorhexidine, miconazole, benzoyl peroxide, or triclosan. Initially, they should be used at least twice weekly with a 10-minute contact time. A combination of chlorhexidine and miconazole may be particularly effective.24 Benzoyl peroxide shampoos can be drying and so are best reserved for dogs with greasy dermatitides. Daily shampooing may be required for dogs with deep pyoderma. If available, treatment with daily whirlpool baths containing chlorhexidine may also be helpful for these dogs. A few important points about shampoos:

- Water temperature should be lukewarm (not hot); if the dog is bathed outside using the hose, the owner should be told to run the water first to make sure it is not hot - water in hoses in the summer can easily get up to temperatures that can burn dogs.

- Most medical shampoos do not lather well, and owners may get frustrated with the amount of shampoo that they deem adequate for obtaining lather relative to the cost. Thus owners should be instructed to use approximately a hand-palm size amount, often placing this in 0.5 to 1 gallon of water first, then applying to the dog.

- The shampoo should always be applied to the dog in the direction of the hair, not against it. Aggressive scrubbing should be avoided, as this may lead to follicular damage and even post-bathing folliculitis-furunculosis.25

Sprays and conditioners with the same or similar medications, used following or in between shampoo sessions may also be useful.26,27
Diluted sodium hypochlorite (bleach) has been used as a leave on, then rinse off after, application. Starting as a commercial 6.15 % solution, a dilution of 1:32 with water was effective in vitro against *S pseudintermedius*, including methicillin-resistant strains.\textsuperscript{28,29}

Topical antimicrobial drugs and antiseptics in ointment or cream form result in high concentrations of drug at the skin surface that can overwhelm bacterial drug resistance mechanisms, and can be useful when pyoderma is limited to small areas of skin. Aside from having the potential to be messy, these drugs have minimal side effects and result in minimal exposure of bystander organisms (such as gut flora) to antimicrobial drugs. Examples of topical drugs with excellent activity against staphylococci include neomycin, gentamicin, polymyxin B, bacitracin, hydroxylic acids (such as acetic acid), novobiocin, and silver sulfadiazine. Mupirocin and fusidic acid are additional topical antimicrobial drug preparations that may also be useful for treatment of pyoderma that is restricted to small areas. While resistance to mupirocin and fusidic acid has been documented in methicillin-resistant *S. aureus* isolates from humans\textsuperscript{30,31}, in vitro evidence of resistance of *S pseudintermedius* to these two medications is rare.\textsuperscript{24,32} However, there is a report of asymptomatic mupirocin-resistant MRSA nasal carriage in a pet dog, so some caution is warranted.\textsuperscript{33}

**Systemic Antimicrobial Drug Therapy**

When pyoderma is widespread, deep, or topical treatment is ineffective or impractical, treatment with systemic antimicrobial drugs may be required. If an antibiotic is selected for empiric treatment, it should be based on the local prevalence of resistance, and narrow-spectrum drugs that target staphylococci are preferable. Clindamycin, first-generation cephalosporins (such as cephalaxin), and amoxicillin-clavulanic acid are reasonable first choices. Other acceptable alternatives when the local regional susceptibility of *S. pseudintermedius* is known include doxycycline, trimethoprim- or ormetoprim-potentiated sulfonamides, lincomycin, and erythromycin. The use of third-generation cephalosporins for empiric therapy is controversial because their use in particular has been associated with selection for methicillin-resistant staphylococci in humans and they have the potential to select for resistant populations of gram-negative bacteria in the gastrointestinal tract.\textsuperscript{34} Whether this holds true for dogs and cats is unknown at present. What is known is that the percentage of MRS and MDR isolates among all species of *Staphylococcus* is increasing.\textsuperscript{11,20,35} However there is little evidence for a difference in outcome between MRS and methicillin-susceptible *Staphylococcus* infections in animals, and the prognosis for MRS skin infections in pets is good, depending on the underlying cause and co-morbidities.\textsuperscript{20} There is controversy about using the fluoroquinolones for canine pyoderma, however the author has used them successfully when susceptibility testing so indicates. Chloramphenicol should be reserved for those cases with no other viable alternative. A small minority of dogs treated with this antibiotic may show a (usually) reversible neuropathy which resembles a thoraco-lumbar disc neuropathy.

Treatment should be for a minimum of 4 weeks for superficial pyoderma and 8 weeks for deep pyoderma. Dogs should be re-evaluated at the end of the suggested periods whilst still receiving the antibiotic. Continued clinical signs such as pruritus in the face of resolution of the pyoderma (reduction or absence of epidermal collarettes or follicular papules) should prompt investigation of underlying diseases. For superficial pyodermas, treatment should be continued for at least 7 days beyond clinical resolution of lesions, because inflammation subsides before infection is completely
resolved. For deep or recurrent superficial pyoderma, treatment should be continued for 14 days beyond clinical resolution.

Although intermittent administration of antimicrobials on a regular basis (“pulse therapy”) has been used to treat some dogs with recurrent pyodermas, there is concern for induction of resistance using these protocols. This mode of therapy was initiated in the early 1980s, when the current high incidence of resistance was not present, and it may not be relevant or advisable at this time. If necessary, referral to a veterinary dermatologist is recommended.

**Prevention**

Prevention of recurring pyoderma relies on addressing the underlying cause or causes whenever possible. This may involve investigating the possibility of food allergy, flea allergy, atopic dermatitis, *Demodex* spp infestation, hypothyroidism or hyperadrenocorticism. This could include skin scraping, initiating or increasing flea control, frequent bathing to restore skin condition, dietary management for dogs with food allergies, management of atopic dogs, and/or breeding practices that aim to select against atopic and dogs with abundant skin folds. Specific prevention of methicillin-resistant staphylococcal infections relies on hand-washing, disinfection practices as well as judicious use of antimicrobial drugs, as outlined earlier. Previous treatment with antibiotics, previous hospitalization and cohabitant dogs have been identified as risk factors for the development of infection with methicillin resistant *Staphylococci*.11,36,37 There is currently not enough evidence to recommend routine decolonization of MRS carrier animals.20

When treatment of the underlying cause or use of topical therapy is unsuccessful in preventing recurrence of pyoderma, subcutaneous administration of autogenous bacterins or commercial bacterial antigens (such as Staphage Lysate®, Delmont Laboratories, USA) may be beneficial in some dogs.38,39 These are generally given once or twice weekly.

**References**


Demodicosis

Canine demodicosis is a noncontagious parasitic skin disease caused by an overpopulation of the host-specific follicular mites of the genus *Demodex*. Most cases of canine demodicosis are caused by *Demodex canis*, although two other species of demodicid mites are reported, *D. injai* and *D. cornei*, however the latter has been shown based on partial sequences of mitochondrial 16S rDNA to be a sub-species of *D canis*.\(^1\text{-}^4a\)

Localized demodicosis is a common mild and benign self-limiting disease. Generalized demodicosis, in contrast, is a serious and potentially life-threatening disease. Most cases of generalized demodicosis are juvenile in onset and develop in dogs less than 1 year of age. Interestingly, yet there is no universally agreed to definition of ‘generalized’, or for the term ‘adult onset ’ (as opposed to juvenile). For the purposes of these notes, ‘adult onset’ will be defined as any case diagnosed in a dog at over 2 years of age, and ‘generalized’ will follow the suggestion of Mueller: ‘…involvement of an entire body region, more than 5 focal areas, and/or paw involvement’.\(^1\)

A genetically preprogrammed immunologic defect probably is responsible for the juvenile onset, generalized demodicosis. Adult onset demodicosis has been reported to be caused by immunosuppressive treatment for neoplasias or auto-immune disorders, or be associated with diseases altering the immune response such as hypothyroidism, hyperadrenocorticism, leishmaniasis, and neoplasias\(^5\text{-}^9\). In the author’s practice the most common underlying cause is the long-term use of systemic corticosteroids – these may be at relatively low doses. Perhaps 25% of the dogs have no demonstrable underlying disease.

Diagnostic tests

Diagnosis is by demonstration of the mite on deep skin scraping in a dog fulfilling the lesional and age requirements noted above. The author prefers to use a medical grade spatula (Fisherbrand* Microspatula with Flat-Ended Blade, catalogue number 21-401-20, Fisher Scientific; \url{http://www.fisherscientific.com}), as this blade is just sharp enough to scrape deep enough to the first level of capillaries (and hence deep enough to be at the follicular depth of the mites). Dogs which have very thick skin (especially on the feet) due to chronic inflammatory skin disease associated with furunculosis may need to be biopsied in order to demonstrate the mites.

For adult onset generalized demodicosis, the owner should be informed of the potential of an underlying disease, and the clinician should perform diagnostic tests searching for such an etiology. Minimum data base should include a complete blood count and biochemical profile, but depending upon the presentation of the dog (as well as the willingness of the owner to spend money) other tests such as abdominal ultrasound, thoracic radiographs, thyroid hormone panel (T4, free T4, TSH level) and ACTH stimulation test could be performed. If an underlying disease is found, the disease should be treated, as well as proceeding with appropriate miticidal treatment.

Treatment
It is important to realize that most if not all dogs with demodicosis will have a secondary pyoderma. This is usually caused by *Staphylococcus pseudintermedius*, but if there is concurrent immunosuppression (for example, from exogenous corticosteroids) other bacteria may be contributing to the pyoderma.

There are several different miticidal treatments that are available to treat generalized demodicosis. In all cases the dog’s mite population should be monitored by means of a deep skin scraping once monthly. The owners should be instructed to continue treatment until the dog has 2 consecutive negative scraping sessions. ‘Negative’ in this instance means NO live or dead adult mites, nymphs, larva, or eggs. Thus, minimum miticidal treatment time will be 2 months. In actuality, most dogs will need to be treated for at least 4 months, although most will show improvement within the first 2 months. If the dog still has positive scrapings 6 months after continuous treatment, but is clinically normal (or dramatically improved) the owner should be informed that the disease can be controlled but that treatment is probably necessary for the rest of the dog’s life.

It should be emphasized that with the newer class of isoxazoline oral anti-parasiticides, their demonstrated efficacy against *Demodex canis* have now made them the author’s treatment of choice. These medications are: fluralaner (Bravecto™; Merck), afoxolaner (Nexgard™; Merial), and Sarolanor (Simparica™; Zoetis). Fluralaner is given q 60 days, the others once monthly. Because these are approved to be given indefinitely for flea and tick control, we may see a decrease in those dogs with demodicosis whose disease relapse (within the first year, after treatment is discontinued) or recur (after the first year), although based on a retrospective study now being done at UC Davis, these numbers were quite low (7% or less).

Thus the following list of treatments may be more for historical purposes!

*Ivermectin*- this should NOT for be used in Collies, or related breeds:
Border Collies, Bearded Collies, Old English Sheep Dogs, Australian herding breeds, Shetland sheepdogs, or anything that could be considered a cross of one of these breeds. The author uses the bovine injectable 1% solution (Ivomec®; Merial); obviously, this is off-label usage. The usual dose is 0.3 mg/kg given ORALLY q24 h for the first week, then 0.6 mg/kg. Some dogs find this medication bitter tasting, so putting it in a small amount of food (vanilla ice cream works well!) is helpful. Adverse effects, although rare, include lethargy, edematous wheals, ataxia and mydriasis. These effects may be seen early or late in the treatment. If the dog shows adverse effects, the drug should be stopped. Most dogs recover within 48 hours of stopping the drug.
Negative outcomes (with ivermectin or moxidectin) have been associated with the use of either diazepam (or similar drugs) or barbituates to control seizures: the preferred drug may be propranolol.

Collies are particularly sensitive to adverse reactions of ivermectin with over 75% of Collies being either carriers or homozygous for the mutant MDR1 allele leading to neurotoxicity. Information on testing for this mutation can be found at [http://www.vetmed.wsu.edu/depts-vcpl/](http://www.vetmed.wsu.edu/depts-vcpl/). Other herding breeds such as the Shetland Sheepdog, Australian Shepherd, and Border Collie also carry or be homozygous for this allele. However, idiosyncratic toxicity may be seen in any breed.

*Imidacloprid & Moxidectin (Advantage Multi®, Advocate®, Bayer)* -
Label claim for demodicosis in Europe, weekly application works best for mild to moderate cases; author’s choice at present for non-collie dogs or dogs that cannot tolerate ivermectin.

Moxidectin which has been evaluated either injected subcutaneously 0.200 mg/kg q7-14 days for 1 to 4 treatments, or 200-400 mcg/kg/day orally\textsuperscript{20-22}. Success rates and adverse effects were similar to milbemycin.

**Milbemycin oxime**- formerly the author’s drug of choice for the ivermectin-sensitive breeds. It would probably be the drug of choice for all breeds except it is more expensive that the bovine ivermectin solution. It was available as an oral heartworm preventative (Interceptor\textsuperscript{®}: Novartis). The author uses a dose of 2 mg/kg per day. A paper from the USA supports the use of this dose\textsuperscript{11}. A more recent article from Sweden suggests that lower doses (mean 0.75 mg/kg) may be effective\textsuperscript{12}. The author has had patients which eventually could be controlled (although not cured) with the lower dose, even if given q 48 h. Adverse effects are stupor, ataxia, trembling, transient vomiting, and lethargy\textsuperscript{13,14} and are generally reversible upon discontinuing the drug. No longer available as a stand-alone drug in the USA.

**Doramectin (Dectomax\textsuperscript{®}, Zoetis), not approved in the U.S for demodicosis.**

300 micrograms/kg/day orally used successfully in Australia, New Zealand & Japan; side effects similar to those seen with ivermectin. Has been used sub-cutaneously with anecdotal success; in one report; the dosage used was 0.4 – 0.6 mg/kg SQ weekly, for 5-23 weeks. In another, Remission was achieved in 94.8% of dogs treated with weekly subcutaneous injections of doramectin at a dose rate of 0.6 mg/kg body weight. Adverse events were rare with two suspected instances (0.5%) being recorded. The mean duration of treatment was 7.1 weeks.\textsuperscript{23a} Mydriasis, anorexia, weight loss, tremors have been noted at 10x this dose.\textsuperscript{23}

**Amitraz**- The author has used this product as a topical 0.025% solution applied once weekly, as has been reported by others\textsuperscript{16}. The hair coat should be clipped in thick-coated and long-haired dogs, and an antibacterial shampoo used to remove crusts and bacteria, then the amitraz solution applied, and NOT rinsed off. While most owners were able to perform the application at home, they were always advised to do this in a well-ventilated area wearing gloves and long-sleeved shirts, as respiratory problems have been observed in humans.\textsuperscript{9,15} In general, this is an effective treatment, but as in other treatment modalities, adult onset generalized demodicosis cases responded less favorably to therapy\textsuperscript{1} than juvenile cases, or at least required longer duration of treatment\textsuperscript{16}. Most cases have to be treated once weekly for 4-6 months. Higher concentrations may have greater cure rates, but also had a greater chance of eliciting adverse effects: depression, sleepiness, ataxia, polyphagia/polydipsia and vomiting and diarrhea\textsuperscript{1,17}. It is the impression of the author, and others\textsuperscript{6} that smaller dogs may be at greater risk of showing clinical signs. The author has noted hypoglycemia in conjunction with sleepiness or depression in some dogs following amitraz treatment: feeding the dogs a small amount of honey or similar sugar-containing food before the next treatment ameliorated or prevented signs of lethargy. This is contrary to one early experimental study which documented amitraz-induced hyperglycemia in dogs\textsuperscript{18}.

Three less common uses of amitraz should be noted. First, in cases of severe pododermatitis, the author and others\textsuperscript{9} have used 1ml of the 12.5% amitraz concentrate mixed in 30 ml of mineral oil for daily application. Obviously, owners should wear gloves. The second use involves a report utilizing amitraz (9%)-containing collars which were replaced every 3 weeks
in 2 dogs with adult onset demodicosis\textsuperscript{19}. There are anecdotal reports of a amitraz/fipronil anti-flea and tick topical medication (Certifect\textsuperscript{TM}, Merial) as a successful treatment for demodicosis when applied q1-2 weeks. As no follow-up period was specified for these uses, it is difficult to judge long-term efficacy.

**Feline Follicular Demodicosis**

Feline follicular demodicosis due to *D. cati* is rare and may present as either localized or generalized follicular demodicosis. Feline localized demodicosis, similar to canine localized demodicosis, is a mild and self-limiting disease. Feline generalized follicular demodicosis due to *D. cati*, similar to canine generalized demodicosis, seems to require diminished immune response. Immunosuppressive diseases such as feline immunodeficiency virus infection (FIV), feline leukemia virus (FeLV), diabetes mellitus, hyperglucocorticoidism, and neoplasia may initiate feline generalized follicular demodicosis.

Feline generalized follicular demodicosis is a less severe disease than canine generalized demodicosis. Predominantly asymptomatic and subtle erythema with variable alopecia, scaling, and crusting may be seen. Lesions most commonly affect the face, neck, trunk, or extremities. Secondary pyoderma is rare contributing to less morbidity. Systemic signs referable to underlying immunosuppressive systemic diseases may be noted. **Treatment** with various medications have been somewhat effective. These include ivermectin orally and moxidectin topically, as noted for dogs. Feline topical Bravecto’s efficacy has not been tested to date. The underlying disease, if present must be treated.

**Feline Superficial Demodicosis**

Feline superficial demodicosis does not have a true canine counterpart. It is believed to be rare in most of North America, but is found more commonly in localized enzootic regions of the southern and southeastern U.S.A. Cases seem to be ‘spreading’ up the Mississippi and Missouri river valleys, especially in animal shelters (Katrina refugees?) Feline superficial demodicosis may be increasing in frequency where modern insect-specific parasiticides that do not kill acarids are used for flea control. The organism is *D. gatoi*.

Clinical features vary from asymptomatic alopecia to alopecia with variable pruritus and self-trauma. If pruritus is absent, cats can present with diffuse, bilaterally symmetric alopecia, plus or minus scaling, affecting the ventral and lateral trunk and caudal legs. Pruritus, if present, usually is intense leading to erythema, crusting and excoriation. Skin scrapings may not yield mites or eggs in pruritic cats since excessive grooming can remove surface-living mites. Skin scrapings of non-pruritic cats may yield large numbers of mites. There is good evidence that this form of demodicosis is contagious among cats.

Treatment with imidacloprid combined with moxidectin (Advantage-Multi\textsuperscript{®} ([Advocate\textsuperscript{®} in Europe]; Bayer) weekly has been successful, as has treatment with lime-sulfur 2x/week. Again, feline topical Bravecto may be effective but studies have not been reported.

**References**


