

Mycologic Disorders of the Skin

Catherine A. Outerbridge, DVM, MVSc, DACVIM, DACVD

Cutaneous tissue can become infected when fungal organisms contaminate or colonize the epidermal surface or hair follicles. The skin can be a portal of entry for fungal infection when the epithelial barrier is breached or it can be a site for disseminated, systemic fungal disease. The two most common cutaneous fungal infections in small animals are dermatophytosis and *Malassezia* dermatitis. Dermatophytosis is a superficial cutaneous infection with one or more of the fungal species in the keratinophilic genera *Microsporum*, *Trichophyton*, or *Epidermophyton*. *Malassezia pachydermatis* is a nonlipid dependent fungal species that is a normal commensal inhabitant of the skin and external ear canal in dogs and cats. *Malassezia pachydermatis* is the most common cause of *Malassezia* dermatitis. The diagnosis and treatment of these cutaneous fungal infections will be discussed.
Clin Tech Small Anim Pract 21:128-134 © 2006 Elsevier Inc. All rights reserved.

KEYWORDS dermatophytosis, *Malassezia* dermatitis, dogs, cats, *Microsporum*, *Trichophyton*, *Malassezia pachydermatis*

Over 300 species of fungi have been reported to be animal pathogens.¹ Cutaneous tissue can become infected when fungal organisms contaminate or colonize the epidermal surface or hair follicles. The skin can be a portal of entry for fungal infection when the epithelial barrier is breached or it can be a site for disseminated, systemic fungal disease. Canine and feline skin and hair coats can be transiently contaminated with a large variety of saprophytic fungi from the environment some of which can cause opportunistic infections.^{1,2} The diagnosis of a fungal infection must be made based on isolation of the organism from affected tissues and visualization of tissue invasion by organisms with compatible morphology.³ Actual mycologic pathogens of the skin can be divided based on what layers of the skin, hair coat or claws they infect. Superficial mycoses include dermatophytosis (predominantly *Microsporum* and *Trichophyton* species), *Malassezia* spp., *Candida*, and *Trichosporon* (piedra). Subcutaneous mycotic infections include sporotrichosis, dermatophytic mycetoma, oomycosis (pythiosis and lagenidiosis), zygomycosis (mucormycosis and entomophthoromycosis), hyalohyphomycosis, and phaeohyphomycosis. The deep systemic mycoses (blastomycosis, histoplasmosis, cryptococcosis, and coccidioidomycosis) can also cause subcutaneous lesions of the skin. This paper will review the etiology, prevalence, clinical presentation, diagnosis, and treatment for the two most common cutaneous fungal infections in small animals; dermatophytosis and *Malassezia* dermatitis.

Department of Medicine and Epidemiology, School of Veterinary Medicine, University of California, Davis, CA.

Address reprint requests to Catherine A. Outerbridge, DVM, MVSc, DACVIM, Department of Medicine and Epidemiology, School of Veterinary Medicine, University of California, Davis, CA 95616. E-mail: caouterbrdge@vmth.ucdavis.edu

Dermatophytosis

Etiology

Dermatophytosis is a superficial cutaneous infection with one or more of the fungal species in the keratinophilic genera *Microsporum*, *Trichophyton*, or *Epidermophyton*. Dermatophyte genera that infect animals are divided into 3 or 4 groups based on their natural habitat. Geophilic dermatophytes are normally soil inhabitants. The most common geophilic dermatophyte to infect dogs or cats is *Microsporum gypsum*. Zoophilic dermatophytes are adapted to living on animals and are rarely found in the soil. The most common zoophilic dermatophyte to infect dogs and cats is *Microsporum canis*. Sylvatic dermatophytes are zoophilic dermatophytes adapted to living on rodents or hedgehogs. The most common sylvatic dermatophytes are *Trichophyton mentagrophytes*, *Trichophyton mentagrophytes erinacei* (hedgehogs), and in Europe *M. persicolor* (does not infect hair shafts).^{1,2} Anthropophilic dermatophytes are adapted to living on humans and do not survive in the soil and can be a cause of reverse zoonosis when dogs or cats acquire the infection from infected humans. *T. tonsurans* was the most commonly isolated dermatophyte from human patients in the United States in the mid 1990s.⁴

The most common fungal organisms identified in the dog or cat with dermatophytosis are *M. canis*, *M. gypsum*, and *T. mentagrophytes*. These fungal organisms are adapted to colonize hair and the cornified layers of the skin where they can digest keratin protein for their nutrition. Most animals must contact a minimum infective dose of dermatophyte spores to have an infection establish. This dose will vary with each individual animal's overall health status. Some animals may have predisposing factors for developing a dermatophyte in-

fection. These predisposing factors include stress, poor nutrition, debilitating disease, compromised immune status, or young age.¹

Prevalence

Dermatophytosis is more common in cats than in dogs but the reported prevalence in a population varies considerably with geographic location, particularly between different continents. Several studies have cited the prevalence of dermatophytosis in dogs and cats, presenting to North American veterinary clinics for skin disease, to be 0.26 to 5.6%.¹ The reported prevalence of dermatophytosis is higher in areas with a warm, humid climate. When healthy pet cats in North America were microbiologically sampled the prevalence of dermatophytosis was very rare.⁵ North American studies in which shelter cats were similarly sampled (using a toothbrush technique) found a higher prevalence of dermatophytes within a shelter population of cats. Pathogenic dermatophytes were cultured in 5.5 to 19% of all shelter cats depending on the geographical region.^{6,7} *M. canis*, specifically, was found in 4 and 5% of cats but only in those geographical areas with a humid and warm climate. Of particular note is that 90 to 100% of the shelter cats that cultured positive in these studies were lesion free.^{6,7} Cats infected with FeLV or FIV did not have an increased incidence of isolation of *M. canis* compared with noninfected cats⁸ in one study but immunosuppression is a risk factor for the development of dermatophytosis. Cats positive for these retroviruses did have more diverse fungal organisms isolated from their hair coats.⁸ Persian and Himalayan cats and Yorkshire terriers appear to be predisposed to *M. canis* dermatophytosis.¹

Clinical

Dermatophytosis in dogs often results in localized lesions most commonly on the face, feet, or tail. Dogs are more likely to present with the classical circular alopecia with scale and crust and follicular papules and pustules (Fig. 1). *T. mentagrophytes* infection often results in much more dramatic clinical scaling, crusting and even scarring (Fig. 2). A kerion is a



Figure 1 Multifocal, well demarcated areas of alopecia because of *M. canis* dermatophytosis in a terrier cross dog. Scaling and follicular papules are present along the lesional borders. (Color version of figure is available online.)



Figure 2 Dermatophytosis caused by *T. mentagrophytes*. There is marked alopecia, erythema, scaling, and crusting involving the head and extremities. Scarring is also evident. The dog had been treated with systemic glucocorticoids before the diagnosis of dermatophytosis with resultant temporal muscle atrophy. (Color version of figure is available online.)

localized inflammatory lesion that results from the host's inflammatory response to a dermatophyte. It is a well-circumscribed, boggy, nodular lesion of furunculosis with draining fistulae often on the face or a distal extremity. *M. gypseum* and *T. mentagrophytes* are the most common organisms to result in kerions.¹

Dermatophytosis lesions in cats are more pleomorphic. Classic lesions include one or more areas of partial alopecia with scaling and crusting most commonly on the head or forelimbs. Lesions may be hyperpigmented.^{2,9} Dermatophytosis can also result in lesions that resemble miliarial dermatitis or focal, pruritic lesions that resemble eosinophilic plaques.^{1,9} Long haired cats may present with a complaint of a poor hair coat or excessive shedding as infected hairs are prone to breakage.⁹ Kerions are uncommon in cats but Persian and Himalayan cats can develop subcutaneous nodular lesions caused by dermatophytes called dermatophytic pseudomycetomas. These lesions are often exudative or draining and coalescing nodules (Fig. 3). Visible tissue grains may be associated with these lesions. Systemic lesions of dermatophytic pseudomycetomas have also been reported in a Persian cat.¹⁰

Diagnosis

Dermatophytosis in dogs and cats is common but is often misdiagnosed, particularly in dogs. Occasionally the diagnosis is never considered but more frequently animals are incorrectly given the presumptive diagnosis based solely on the appearance of visible, circular skin lesions. This can result in some animals receiving unnecessary therapy with antifungal drugs. This is concerning when the drugs being prescribed are expensive or capable of causing serious side effects and when the misdiagnosis results in delays in determining the actual cause of the animal's skin lesions.

The clinical appearance of skin lesions is unreliable as the sole criteria to diagnose dermatophytosis. A Wood's lamp examination can be helpful in some cases but it is also not a



Figure 3 Ventral abdomen of a Himalayan cat with a *M. canis* pseudomycetoma. There are coalescing, ulcerative, erythematous nodular lesions. There are yellow granular tissue grains evident on the surface of some lesions.

reliable as the sole criteria for diagnosing dermatophytosis. If true positive, apple green, fluorescence tracking along the hair shaft is documented on a Wood's lamp examination then infection with *M. canis* is likely. However only 50% of *M. canis* infections will fluoresce and most other dermatophyte species affecting dogs and cats do not fluoresce. A negative Wood's lamp examination does not rule out dermatophytosis. False positive results can occur if sebum or topical ointments are present and the "glow" produced from them is misinterpreted as positive fluorescence from *M. canis*. Microscopic examination of hairs plucked from a suspected patient can be visualized for evidence of fungal hyphae or spores. This is facilitated by the addition of a clearing agent, either 10% potassium hydroxide KOH or chlorphenolac, to the hair sample. However, this is also not a reliable method to screen for dermatophytosis as false negative results will result if unaffected hairs are examined. Dermatophytosis can be diagnosed via a skin biopsy but fungal culture is a more sensitive diagnostic tool. Histopathology may demonstrate the presence of spores and hyphae within intrafollicular hair shafts with variable amounts of follicular inflammation. A periodic acid-Schiff stain will highlight dermatophytes a magenta color. Biopsy samples of skin or claws are very useful in diagnosing kerions, pseudomycetomas and fungal paronychia as obtaining a positive culture of the organism is more difficult in these infections because of the limited numbers of organisms.^{1,9}

The definitive diagnosis of dermatophytosis is made via culture and identification of the organism. Culture samples can be obtained from hairs plucked from suspicious lesions seen clinically or with a Wood's lamp. A sterile toothbrush can be used to sample lesions or the entire suspect animal's hair coat. Dermatophyte test medium (DTM) or Sabouraud's dextrose agar are the most commonly used culture media. To evaluate for the growth of a dermatophyte versus a saprophytic fungus the following key points should be considered:

1. Dermatophyte colonies are white or off-white in color but never brown, green or mixed colors
2. DTM will turn red when the colony is first visible. If the color change occurs several days after visible colony growth it is more likely to be a saprophytic fungus
3. All suspicious colonies should be confirmed as a particular species via microscopic evaluation. Using clear acetate tape fungal elements can be harvested by touching the top of the colony with the tape and examining the macroconidia under a microscope.

Culture samples should be incubated at 24 to 27°C as a recent study suggest that incubation at room temperature may result in false negative culture results.¹¹

Treatment

Most healthy animals will resolve their dermatophyte infection within 3 months without treatment. This occurs as the infected hairs enter the telogen phase or as an inflammatory response against the fungal organism develops in the animal. However, dermatophytosis is a highly contagious and zoonotic disease and affected animals should be treated particularly in multiple animal households or in homes with young children or with immunocompromised or geriatric individuals. One of the challenges in treating feline dermatophytosis is the fact that some cats can be chronic carriers with subclinical yet active disease that results in the production of fungal spores and environmental contamination. Many uninfected animals can transiently carry infective spores yet have no



Figure 4 The exacerbation of dermatophytosis on a cat's ventral abdomen after hair was shaved for an ovariohysterectomy. The cat had no lesions at the time of surgery but by the time of suture removal multifocal, coalescing, erythematous papules, and plaques with scaling were evident. Histopathology of skin biopsies and fungal culture confirmed infection with *M. canis*.

active infection so these animals act only as fomites. Dermatophytosis can be both frustrating and challenging to manage, particularly if the infection involves a population of animals since some infected cats may have no clinical lesions and the clinical lesions in affected cats are variable. If there is a lot of movement of a population of animals within the premises (eg, an animal shelter environment) the extent of environmental contamination and the possibility of wider transmission increases. The systemic medications that are used to treat dermatophytosis can be expensive and the animal may require monitoring with laboratory tests to evaluate for adverse side effects. Animals that are housed in groups not only increase the risk of possible transmission but also increase the overall expense of therapy as all contact animals will need to be treated if dermatophytosis is diagnosed. It is difficult to have one single recommended therapeutic strategy for treating dermatophytosis as the situations may vary widely. General principles should be established. The following strategies should be employed whenever possible in treating dermatophytosis.

1. Examine and culture all contact animals.
2. Systemic therapy for all animals with positive cultures (infected) and topical therapy with lime sulfur of all infected or contact animals.
3. Environmental decontamination.

Topical therapy has long been advocated for the treatment of dermatophytosis. However numerous studies have shown that topical therapies alone are not as effective as systemic therapy.¹² The most important benefit from topical therapy is to kill infective spores on the cat or dog and limit spread of this material into the environment. There have been several studies evaluating the efficacy of various topical therapies and lime sulfur and enilconazole are the two most effective antifungal topical therapies and should be applied twice weekly.^{1,9,12} Chlorhexidene alone and povidone iodine are ineffective against infective spores and hairs and are no longer recommended for use.⁹ Topical therapy should be used, whenever permissible, in conjunction with systemic therapy.

Clipping the entire hair coat has been advocated in treating dermatophytosis. Clipping will remove infective material and make topical therapy easier. However, the clipped hair if not handled appropriately can worsen environmental contamination. Animals can have lesions spread and worsen, if clipped too closely (Fig. 4). The usefulness of clipping may depend on individual situations; long haired animals should be clipped and all cats in an affected Persian cattery should be clipped.

Systemic antifungal therapy has been shown to decrease both the duration and severity of feline dermatophytosis.^{1,9,12} Systemic therapy is of most benefit to treat the clinical signs in an individual cat or dog. It will not contain the contagious spread of dermatophytosis within a group of animals; other management practices are also needed. Griseofulvin, itraconazole, and terbinafine are all effective systemic antifungal therapies for dermatophytosis.⁹ Griseofulvin is fungistatic oral drug that interferes with nucleic acid synthesis and with spindle microtubules in the metaphase of cell mitosis.^{1,9,12} There are 2 formulations microsize (25-50 mg/kg q 24 hours) and ultramicrosize (5-10 mg/kg q 24 hours in divided treat-

ments) and the dose depends on the formulation being used. The drug needs to be administered with food to enhance absorption. There are a number of possible adverse side effects with this drug. Vomiting, diminished appetite, and diarrhea are the most common adverse effects seen with the administration of griseofulvin. It is a known teratogen and should never be given to pregnant animals and it is recommended that its use be avoided in breeding males. Myelosuppression is not common but is a known side effect and patients should have complete blood cell counts performed to monitor for leukopenia. This side effect is more common to occur in FIV positive cats, so retroviral status should be checked before initiating therapy with griseofulvin.

Ketoconazole is an imidazole that inhibits ergosterol synthesis in the fungal cell wall affecting the permeability of the fungal cell wall. It is not used typically to treat feline *M. canis* infections as up to 25% of systemically treated cats developed adverse side effects; diminished appetite, vomiting, diarrhea, and hepatotoxicity.¹² Ketoconazole can be used to treat dogs with dermatophytosis at a dose of 10 mg/kg orally once per day.¹ Itraconazole is a triazole antifungal that inhibits ergosterol synthesis. It can be used in both dogs and cats to treat dermatophytosis at a dose of 10 mg/kg once orally per day. It is now considered the drug of choice for treating feline dermatophytosis as adverse reactions are uncommon and in experimental models it is equal or superior to griseofulvin.^{13,14} However, the expense of itraconazole can be prohibitive. There are now a number of protocols that utilize the medication in a pulse or cycle fashion.⁹ Fluconazole is an oral, water soluble triazole that has been used to treat a variety of fungal infections including dermatophytosis. A generic of this drug is now available and it is likely that there will be future clinical trials treating dermatophytosis with this drug. Terbinafine is allylamine antifungal that interferes with ergosterol synthesis by inhibiting the fungal enzyme squalene epoxidase and has specific activity against dermatophytes. It has been used in both cats and dogs at dosages of 30 to 40 mg/kg per day. Terbinafine is well tolerated by both dogs and cats. It may be possible to administer this medication in a pulse or cycle therapy fashion.^{9,14} Lufenuron is a benzoylphe-nylurea drug that is used as a flea control product as it disrupts chitin synthesis in the insect. As chitin is a component of the fungal cell wall it has been proposed as a systemic therapy for dermatophytosis. A retrospective study proposed that lufenuron was protective against dermatophytosis and the same investigators reported successful therapeutic response with this drug.¹⁵ There is conflicting evidence in the literature as to how efficacious this therapy actually is. Recent scientific studies suggest that pretreatment with lufenuron before either experimental or natural exposure to *M. canis* was not protective.^{16,17} Lufenuron is an extremely safe, well-tolerated drug. The current recommended dose is 100 to 120 mg/kg to be administered with food. In the original report affected animals received a single dose. However, as lufenuron has failed to resolve all cases of dermatophytosis, more frequent dosing intervals have been proposed. The most effective dosing regime and assessment of the true efficacy of this medication for treating dermatophytosis is still in question and it is not recommended as a therapy for dermatophytosis.^{9,14}

Topical and systemic therapy should be continued until

serial negative cultures have been obtained. The first culture should be obtained 4 weeks after initiating therapy and 2 negative cultures: 2 weeks apart for individual cat households or 3 negative cultures 2 weeks apart are desirable in multiple cat households.¹⁴ If cultures can not be performed then treatment should be continued for 2 to 4 weeks after resolution of all clinical signs. Typically treatment is often for 6 to 10 weeks.

Environmental decontamination is very important in preventing spread of infection to other animals or re-infection. However, environmental decontamination is extremely difficult, if not impossible, to accomplish in some environments. It should be assumed that environmental contamination with *M. canis* spores is widespread and that uninfected cats may transport spores on their hair coats. The best agents for disinfection continue to be debated. Studies have evaluated the efficacy of many antifungal disinfectants. When contaminated surfaces were cultured after a single application of a disinfectant only undiluted bleach (100% elimination), a 1:10 dilution of bleach (22% elimination) and enilconazole (33% elimination) had efficacy with a one time surface application.¹⁸ An environment with infected cats within it will need to be treated repetitively as the cats will continue to shed infective hair and spores. Furnace filters, fabric pet beds, and blankets should be replaced or discarded. The environment can also be problematic to disinfect if there is lots of fabric or carpet used to cover surfaces. Carpets and furniture can be steam cleaned and this will decrease but not eliminate all spores.

There are commercial dermatophyte vaccines for cats, cattle, and foxes. In cats they have not shown the same efficacy as in cattle. There is no evidence that the feline vaccine offers any protection against dermatophytosis. In clinical trials, cats given the vaccine had a decrease in the size of their lesions but did not resolve their infection any sooner than unvaccinated cats.^{9,13}

Malassezia Dermatitis

Etiology

The genus *Malassezia* is known to include six lipid dependent species (*M. furfur*, *M. symbodialis*, *M. sloofiae*, *M. globosa*, *M. obtusa*, *M. restricta*) and one lipid independent species *M. pachydermatitis*.^{19,20} There have also been recent reports of three new lipid dependent species which include *M. equi*, *M. dermatis*, and *M. nana*.²¹ *Malassezia* species are identified based on differences in their morphologic, ultrastructural and molecular characteristics along with colony appearance and biochemical properties.²⁰ *M. pachydermatitis* is a normal commensal inhabitant of the skin and external ear canal in dogs and cats. In dogs the lipid dependent species *M. furfur* and *M. sympodialis* have also been isolated.²⁰ In cats the lipid dependent species *M. symbodialis*, *M. globosa*, *M. furfur*, and *M. nana* have also been identified.^{19,21} *Malassezia* organisms may cause dermatitis as a result of inflammatory or hypersensitivity reactions by the host animal to yeast antigens or products. Most dogs with *Malassezia* dermatitis have concurrent dermatoses. Dogs with allergic dermatitis often have increased numbers of *Malassezia* colonizing their skin and when treated appropriately with antifungal therapies both

the overall appearance of the skin and the level of pruritus will improve. The diagnosis of generalized *Malassezia* infection in a cat is strongly linked to concurrent, serious, systemic disease: diabetes mellitus, positive retroviral status or internal neoplasia (thymoma and pancreatic adenocarcinoma).^{8,22}

Prevalence

Malassezia is a commensal organism that has been isolated from the skin, ear canals, anal sacs and mucosal surfaces of healthy dogs and cats.¹⁹ It becomes a pathogen when there are sufficient predisposing factors existing for a particular animal. Most predisposing factors alter either the cutaneous microenvironment or the host animal's immune system. Increased humidity in the cutaneous microenvironment promotes yeast growth and offers an explanation for the prevalence of *Malassezia* dermatitis in ear canals and intertriginous areas.¹⁹ *Malassezia* dermatitis is also more common in warm, humid climates and times of the year.²¹ Disturbances in the amount or composition of surface lipids in the skin can also promote yeast growth. These alterations in lipid can occur in response to hormonal changes, cornification disturbances, nutritional disturbances, and the presence of bacterial lipases.^{19,21} The epidermal barrier is an important component of an animal's innate immune system. The cellular and lipid structure of the stratum corneum protects the skin from infection. If this barrier is disrupted then secondary bacterial and yeast infections can occur. There are a large number of cutaneous and systemic diseases that can compromise the integrity of this barrier function. Allergic dermatitis and ectoparasitic diseases are very common predisposing causes for *Malassezia* dermatitis and otitis externa as they result in inflammation and pruritus with self trauma disturbing the normal epithelial barrier. Endocrinopathies and metabolic diseases alter surface lipids and/or immune responses predisposing to secondary *Malassezia* infections. *Malassezia* dermatitis is much more common in dogs than in cats. Certain breeds appear predisposed. Normal basset hounds have increased cutaneous carriage of *M. pachydermatitis* than other breeds of dogs.²¹ Other breeds with apparent predisposition include West Highland white terriers, cocker spaniels, dachshunds, Shih Tzus, and English setters.^{19,21}

The administration of antibiotics has been documented to promote *Candida* fungal overgrowth in humans but the evidence for secondary *Malassezia* overgrowth in dogs receiving antibiotics is lacking.¹⁹

Clinical

Skin lesions resulting from *Malassezia* dermatitis may be localized or generalized. Localized lesions most commonly involve the external ear canal, interdigital skin, ventral neck, axillae, inguinal region, or intertriginous areas. Affected areas are erythematous, alopecic, and often lichenified with or without hyperpigmentation. The lesional surface is often greasy or exudative and affected dogs may have a malodorous, musty smell. Chronic lesions are often thickened, lichenified, and hyperpigmented with a halo of erythema (Fig. 5). Lesions are variably pruritic. It is likely that those individuals with intense pruritus associated with their *Malassezia* dermatitis may have a hypersensitivity reaction to *Malassezia* antigens or products.^{19,21}

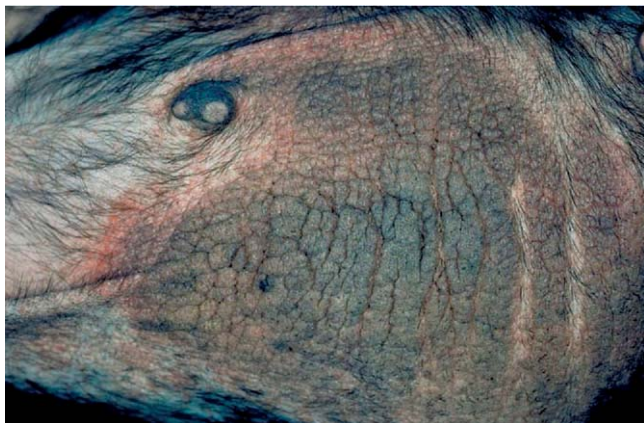


Figure 5 The ventral abdomen of a dog with malassezia dermatitis secondary to atopic dermatitis. Marked hyperpigmentation, thickening, and lichenification of the inguinal skin. There is a halo of erythema surrounding the affected skin at the advancing edges of the lesion.

Diagnosis

Malassezia dermatitis or otitis externa is most commonly diagnosed based on cytologic documentation of increased numbers of the yeast organisms. Cytologic samples can be obtained from performing superficial skin scrapings, directly placing a glass slide onto lesional skin, rubbing a cotton swab on the skin surface of interest or repetitively pressing clear acetate tape to lesional skin (tape stripping). Each method has advantages and disadvantages based on the ease of sampling a given area with a given technique. Clear acetate tape impressions are often the most versatile as they can access difficult areas (between toes or around eyes) and are equally effective for moist and dry lesions. Cotton swabs are best used for obtaining cytologic samples from ear canals. Cytologic samples are placed on a glass slide, heat fixed and stained (Diff-Quick or methylene blue). Acetate tape preparations are not heat fixed and are stained only with the purple stain from the Diff Quick or methylene blue and applied to a glass slide sticky side down. Slides are examined under oil immersion (1000 \times magnification) looking for the presence of round to oval, monopolar budding yeasts often adhering to keratinocytes.^{1,21} A small number of organisms may be identified in cytological samples from normal animals. The number of organisms that must be present to be pathogenic is not well defined and the finding of one yeast organism per oil immersion field in the animal with compatible clinical signs should be considered significant.²¹ *Malassezia* organisms can be cultured but as they are normal commensal organisms interpreting a positive culture must be made in context with clinical signs and history. Culturing is rarely indicated as organisms if present should be visualized on cytology. *Malassezia* organisms can be seen on histopathology of skin biopsies. The organisms are typically visualized within the stratum corneum and/or the follicular infundibulum.¹⁹ The stratum corneum is often disturbed or lost during processing of a skin biopsy and consequently histopathology is not a sensitive test for diagnosing *Malassezia* dermatitis.

Treatment

Treatment for *Malassezia* dermatitis involves the use of topical therapy with or without concurrent systemic therapy.

Topical therapies that can be used include miconazole, ketoconazole, chlorhexidine, clotrimazole, enilconazole, nystatin, and selenium sulfide).^{1,21} *M. pachydermatitis* is most susceptible to topical azoles. There are numerous commercial shampoos, leave-on lotions, sprays, and wipes that utilize the above listed topical agents. In chronic or generalized cases systemic therapy with one of the azole antifungals may be necessary. Ketoconazole is administered at 5 to 10 mg/kg orally once every 24 hours for 3 to 4 weeks.¹⁹ Itraconazole has been reported to be equally effective at 5 mg/kg once orally every 24 hours for 21 days compared with 5 mg/kg orally per day for 2 consecutive days per week for 3 weeks.²³ Fluconazole has activity against *Malassezia* in humans. With the availability of generic fluconazole, treatment for *Malassezia* with this drug in dogs and cats is likely to become common. Griseofulvin has no activity against *Malassezia*. Identification of underlying predisposing factors should also be pursued whenever appropriate to diminish recurrence of infection. If relapses are common or predisposing factors cannot be controlled, routine topical therapy or pulse oral medications may be utilized.^{1,21}

Conclusion

Dermatophytosis and *Malassezia* dermatitis are the most common cutaneous fungal infections in dogs and cats. Both infections can occur in association with other skin diseases such as allergic dermatitis or cornification disturbances. They can also occur in association with underlying systemic disease such as endocrine disorders or neoplasia. Diagnosis is based on compatible clinical signs and demonstration of the organism on fungal culture, histopathology, or cytology. Both topical and systemic therapy are utilized in the management of these cutaneous fungal infections typically with good success provided predisposing factors are identified and for dermatophytosis that the environment is not a source of reinfection.

Acknowledgement

Thank you to Dr. Autumn P. Davidson for guest editing this article.

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