How to diagnose and treat Malassezia dermatitis in dogs

Because this condition’s causative organism is a normal resident of canine skin, detection doesn’t equal diagnosis. To avoid any missteps in diagnosis as well as treatment, follow these authors’ up-to-date approach to managing Malassezia dermatitis in an itchy, smelly dog.

As the causative agent of Malassezia dermatitis, Malassezia pachydermatis contributes to or causes mild to severe pruritus in dogs. Malassezia dermatitis is common in dogs and usually occurs concurrently with other dermatoses.1 This article reviews the etiology, pathogenesis, clinical presentation, diagnosis, and treatment of Malassezia dermatitis in dogs.

Etiology and pathogenesis

Malassezia pachydermatis is lipophilic, nonmycelial, and saprophytic.1,2 This thick-walled, ovoid to ellipsoid, unpolar budding yeast can be found in or on the ear canal, anal sac, lip, chin, vagina, rectum, and skin of clinically normal dogs.1,2 It can be part of the normal cutaneous microflora of dogs, which causes confusion in accepting this yeast as a potential pathogen.

When factors allowing its overgrowth are present, M. pachydermatis is thought to act as a facultative pathogen on the skin of dogs. These factors include host predisposition and an alteration in skin microenvironment and host defenses. Skin microenvironmental factors favorable for yeast overgrowth include excessive sebum production, diminished sebum quality, moisture accumulation, a disrupted epidermal surface, and concurrent dermatoses.1,2 Diseases that cause cutaneous inflammation and altered sebum production and quality may predispose a dog to a skin microenvironment favorable to M. pachydermatis overgrowth. These diseases include allergies (atopy, food allergy, flea allergy, and contact allergy), keratinization disorders (seborrhea), bacterial skin diseases, endocrinopathies (hyperadrenocorticism, hypothyroidism, diabetes mellitus4), metabolic diseases (zinc-responsive dermatosis and superficial necrolytic dermatitis), and cutaneous or internal neoplasia.1,2,5

A recent study demonstrated that atopic dogs with cytologic evidence of yeast had significantly greater wheal-and-flare reactions to intradermal injection of M. pachydermatis extract than did atopic dogs without cytologic evidence of yeast.6 The authors of this study concluded that M. pachydermatis is capable of inducing a type-1 hypersensitivity reaction in atopic dogs. Regardless, atopic dogs, whether hypersensitive to M. pachydermatis or not, do have altered cutaneous microenvironments that could be favorable to yeast overgrowth. Interestingly, it has been demonstrated that atopic dogs have significantly greater carriage of M. pachydermatis interdigitally (especially when inflamed), directly under the base of the tail, and on clinically normal skin than do healthy dogs.7

Certain breeds seem predisposed to developing Malassezia dermatitis. These breeds include the West...
Highland white terrier, dachshund, English setter, basset hound, American cocker spaniel, Shih Tzu, springer spaniel, and German shepherd.¹,²,⁵,⁸,⁹ Many of these same breeds are commonly affected with one or more of the aforementioned diseases. In fact, one study hypothesized that predisposed breeds may have an inherent propensity for underlying conditions favorable to yeast overgrowth or infection.⁸

Antibiotic use has been suggested as a cause of *Malassezia* species overgrowth and dermatitis.²,⁹ In a group of 98 dogs with various dermatoses, the prevalence of elevated *Malassezia* species counts was significantly higher in dogs treated with antibiotics, in seborrheic dogs, and in certain dog breeds.⁹ Perhaps breed predilections and underlying predisposing dermatoses such as seborrhea were more relevant to the elevated *Malassezia* species counts than was the use of antibiotics in this study. However, in our experience, increased *Malassezia* species counts are frequently observed when seborrheic dogs are treated with antibiotics for their pyoderma, but the seborrheic condition is not addressed. Nevertheless, many dermatoses are treated effectively with antibiotics without the need for antifungal therapy.

Once colonization takes place, *M. pachydermatis* is thought to release proteases, lipases, phosphatases, and ureases that alter the sebum quality and disrupt the epidermal surface.¹⁰ It also may activate the complement cascade, as seen with the yeast *Malassezia ovalis* (formerly *Pityrosporum ovale*).¹¹ The pathogenic mechanisms of *M. pachydermatis* cause inflammation and pruritus, which further lead to a favorable microenvironment for yeast overgrowth.

**Clinical presentation**

*Malassezia* dermatitis can occur in dogs of any breed, age, or sex, although, as mentioned earlier, certain breeds seem predisposed.¹,²,⁵,⁸,⁹ The incidence of *Malassezia* dermatitis may increase during the summer months, but the disease can occur at any time of year, especially in warmer climates. The association between *Malassezia* dermatitis and the summer months correlates with the allergy season and higher humidity.

The most common clinical sign of *Malassezia* dermatitis is moderate to intense pruritus, which may be only partially responsive to corticosteroids and antibiotics. Affected animals typically have an offensive odor, which some clinicians refer to as yeasty or rancid. *Malassezia* dermatitis is manifested either as a generalized or localized dermatitis (lesions involving the ear, muzzle, interdigital areas, nail fold, ventral neck, medial thigh, axilla, perianal region, and intertriginous areas).¹,² Note that several of these areas are dense with sebaceous glands and are commonly associated with allergic dermatitis.

Skin lesions are not specific for *Malassezia* dermatitis and reflect the existing seborrhea and pruritus. Lesions may be erythematous, scaly (yellow to slate gray with or without plaques), greasy or dry, crusty, hyperpigmented, lichenified, and perhaps saliva-stained and alopecic (Figures 1-4). In some dogs, pruritus may be so intense that the condition may be misdiagnosed as a neurologic or behavior problem once other pruritic dermatoses, including sarcoptic mange and food allergy, have been excluded. Remember that *Malassezia* species are colonizers secondary to many different dermatologic diseases. For example, suspect increased *Malassezia* species populations in atopic dogs with erythematous feet, because these dogs have significantly higher yeast counts on cytologic examination compared with healthy dogs.⁷
**Diagnosis**

Differential diagnoses include the previously mentioned predisposing factors, as well as ectoparasitism (e.g., sarcoptic mange, demodicosis) and cutaneous drug eruption. Positive yeast recovery and identification by cytologic examination, culturing, or histopathologic examination of samples collected from affected skin suggest a diagnosis of *Malassezia* dermatitis. Cytologic examination is the diagnostic method of choice since it is quick and simple. A culture or histopathologic examination in general is not recommended in the clinical setting. When *M. pachydermatis* is recovered from a patient’s skin, always interpret this finding cautiously and in light of any clinical signs, because this yeast can be found on the skin of normal, asymptomatic dogs.

**Cytologic examination**

Cotton swab smears, skin scrapings, direct impression smears, and acetate tape impressions are all routinely used to identify *M. pachydermatis* cytologically. Ready-to-use adhesive-coated slides can also be used (e.g. Duro-Tak—Delasco). Differences in the ability to recover yeast organisms among cotton swab smears, skin scraping techniques, and direct impression smears in clinically normal dogs have not been detected. The acetate tape impression identifies yeast more frequently than the other cytologic techniques. Clinicians should be comfortable with each of these techniques and decide which might have a higher diagnostic yield, considering the affected area and skin surface. For example, direct impression and cotton swab smears are better suited to moist areas and skin folds.

In most cases, we prefer the acetate tape impression technique. With this technique, apply the adhesive side of acetate tape to affected skin. Then, stain the tape with a...
modified Wright’s stain (Diff-Quik—Dade Behring; VWR International). Since cells are already fixed to the tape, omit the fixative step, because this will dissolve the tape. Next, apply the stained tape to a glass slide with the adhesive side down. Examine the slide under oil immersion, looking for unipolar budding yeast that are described as peanut-, footprint-, or bottle-shaped organisms (Figure 5).

Confusion exists on how to differentiate between commensal and pathogenic yeast populations. To provide commensal population reference ranges, researchers have attempted to quantify the \textit{M. pachydermatis} population in clinically normal dogs. Unfortunately, the number of organisms recovered from healthy dogs varies widely. Further, the anatomical site affects the number of yeast recovered. In a study of clinically normal dogs, axillary and inguinal areas generally had the lowest number of yeast per culture. This is interesting because these areas seem to be the most commonly affected areas in dogs with \textit{Malassezia} dermatitis. This finding supports the idea that underlying predispositions allow the yeast to cause disease.

How many yeast are clinically relevant? It’s probably best to use your clinical judgment. Some clinicians diagnose \textit{Malassezia} dermatitis by finding 10 or more yeast per 0.5-sq-in area of microscopic glass slide. But many dermatologists consider finding any yeast from clinically affected areas diagnostic. Regardless, try to grade yeast numbers in the most objective manner possible. A scale of 1+ to 4+ or quantitative terminology such as mild, moderate, or severe can be used. This aids in assessing response to therapy.
Histopathologic examination

In general, histopathologic examination has a low sensitivity in detecting Malassezia species, because the yeast are removed from the skin surface during processing. Consequently, the pathologist may not find any yeast in the processed biopsy sample. But if a few yeast are seen in the stratum corneum, reevaluate the patient for Malassezia dermatitis, because there may be many more yeast elsewhere on the skin. Occasionally, yeast may be found within the hair follicle, which is always considered to be clinically relevant.

In addition to possibly finding yeast, histopathologic examination can increase suspicion of Malassezia dermatitis if certain histopathologic characteristics are found. These include lymphocytic superficial perivascular to interstitial dermatitis with parakeratotic hyperkeratosis, irregular epidermal hyperplasia, diffuse intercellular edema, lymphocytic exocytosis, and, perhaps, eosinophilic microabscesses. Keep in mind that these findings, while suggestive of Malassezia dermatitis, are not specific. Histopathologic evaluation by a dermatopathologist may also help identify an underlying cause of Malassezia dermatitis.

Treatment

When you treat Malassezia dermatitis, it is important to identify any predisposing factors that can lead to yeast overgrowth, since M. pachydermatis is usually a contributing factor, not the sole cause, of clinical signs. Failure to treat concurrent problems may result in partial treatment success, treatment failure, or a relapse of Malassezia dermatitis.

Topical therapy

To treat generalized Malassezia dermatitis, use topical agents alone or in combination with systemic antifungals. We have found topical agents, initially used as sole therapy, to be beneficial in most cases. Topical therapy kills yeast (antifungal agents) or disrupts yeast colonization by altering the cutaneous microenvironment (degreasing or antiseborrhic agents). Prophylactic use of topical agents to prevent recurrence is beneficial in relapsing cases once the active infection is eliminated. Many different topical products and formulations (e.g. shampoos, dips, creams, lotions) are available. Familiarize yourself with the properties of different topical ingredients and their functions when treating Malassezia dermatitis (Table 1; also see “Noninsecticidal shampoo therapy” in the October 1998 issue).12

When seborrhea oleosa (greasy dander) is present, use degreasing antiseborrhic agents. Benzoyl peroxide, benzoyl peroxide with sulfur, 1%
selenium sulfide, and tar shampoos have good degreasing activity and remove crusts and oily scale. When mild to moderate seborrhea sicca (dry, flaky dander) is present, use antiseborrheic agents, such as sulfur, salicylic acid, and 1% selenium sulfide. By removing surface debris, these agents eliminate cutaneous conditions favorable to yeast overgrowth; if needed, antifungal agents should then be able to penetrate yeast more effectively. Use these shampoos at least twice a week. Some cases require the addition of a topical antifungal agent. Use a degreasing or antiseborrheic agent before applying the topical antifungal to enhance the effectiveness of the antifungal agent. Some of the newer antifungal shampoos that have vehicles with antiseborrheic properties (e.g., Malaseb—DVM Pharmaceuticals; KetoChlor—Virbac; Seba-Hex—EVSCO Pharmaceuticals) have proved in our hands to be very beneficial in treating *Malassezia* dermatitis.

Ketoconazole, miconazole, clotrimazole, and chlorhexidine have broad-spectrum antifungal activity and are found in many topical formulations. When treating generalized *Malassezia* dermatitis, clinicians usually use the shampoo formulation. Antifungal shampoos (ketoconazole, miconazole, 3% or 4% chlorhexidine, miconazole-chlorhexidine combination, or ketoconazole-chlorhexidine combination) are liberally applied to the coat and skin and left on for 10 to 15 minutes before rinsing. When using shampoos without antiseborrheic properties, apply a degreasing or antiseborrheic product before using the antifungal shampoo.

Several new shampoos containing boric and acetic acid acidify the cutaneous microenvironment, making the

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**Use a degreasing or antiseborrheic agent before applying the topical antifungal to enhance the effectiveness of the antifungal agent.**
skin less favorable to yeast growth. These products may be used in combination with antiseborrheic agents to control infection or may be used prophylactically to prevent yeast overgrowth. A solution of equal parts of white vinegar and water is an inexpensive acidifying topical agent with residual activity. It is applied to the skin and left to dry.

In addition to shampoo therapy, leave-on products such as conditioners, creams, lotions, sprays, wipes, and dips can be applied after bathing in cases of generalized *Malassezia* dermatitis. These products may also be used to spot-treat localized areas of *Malassezia* dermatitis. We tend to use these leave-on products on non-bathing days in mild cases, or every day (once or twice a day) in more severe cases.

Topical treatments must be effective, well-tolerated by the patient and client, and not extraordinarily labor-intensive or expensive. With so many new topical preparations, you should be able to devise a therapeutic plan amenable to both the patient and the client.

### Systemic therapy
Consider systemic antifungal therapy in severe cases of *Malassezia* dermatitis or when topical therapy is unsuccessful. Azoles such as ketoconazole, itraconazole, and fluconazole have shown efficacy against *Malassezia* species. Their use is considered extralabel, since none are licensed for veterinary use in the United States. Azoles (imidazoles and triazoles) inhibit the cytochrome P-450 enzyme lanosterol 14-alpha-demethylase, preventing the demethylation of lanosterol and thus the synthesis of ergosterol, the main sterol in fungal cell membranes. By preventing the synthesis of ergosterol, the agent alters the cell’s permeability, resulting in its death. Azoles may also disrupt intracellular oxidative and peroxidative enzymes. These drugs are considered fungistatic, but they may be fungicidal at higher concentrations. Azole toxicity is directly related to its binding of mammalian cytochrome P-450 enzymes. The net effect of this binding is decreased cholesterol and steroid hormone production. Itraconazole and fluconazole have less affinity for mammalian cell enzymes than does ketoconazole.

Of the azoles, ketoconazole is the most common one used to treat *Malassezia* dermatitis. The dosage is 5 to 10 mg/kg orally once a day (or divided twice daily) for 30 days.

Dosages of 5 to 10 mg/kg daily for 10 days followed by alternate-day therapy at the same dose for another 10 days have been reported to be successful. In our experience, concurrent use of topical antiseborrheic therapy may reduce the duration of ketoconazole administration.

Ketoconazole is a good choice for *Malassezia* dermatitis because it is excreted through sebum and eccrine glands. Some of its success in treating *Malassezia* dermatitis may be related to its immunomodulatory and anti-inflammatory effects. Oral ketoconazole is better absorbed in an acidic environment and when taken with food. Ketoconazole can alter the metabolism of some drugs, because it inhibits cytochrome P-450 enzymes. So be aware of any concomitantly used medications and possible drug interactions. Ketoconazole is contraindicated in pregnant bitches and in patients with liver dysfunction, since it is a known teratogen and hepatotoxin. Document serum liver enzyme activities before you administer ketoconazole to determine whether the drug is contraindicated. Many veterinary dermatologists do not routinely monitor liver parameters unless a patient is receiving long-term or high-dose therapy, experiencing adverse effects, or has another medical condition that warrants testing. Adverse effects include anorexia, nausea, vomiting, diarrhea, elevated serum liver enzyme activities, icterus, pruritus, and a reversible lightening of the haircoat. These effects are usually related to dose and length of treatment. Doses greater than 10 mg/kg/day may suppress adrenal cortisol production.

Oral itraconazole and fluconazole are also effective against *Malassezia* species. Both are given orally once a day (itraconazole at 5 to 10 mg/kg and fluconazole at 2.5 to 5 mg/kg) for two to four weeks. We have used itraconazole successfully at 5 mg/kg/day. Because of itraconazole’s keratinophilic and lipophilic properties, it remains in the skin and adnexa in high concentrations for prolonged times in people. Consequently, the dosing regimens of itraconazole in human dermatology reflect these properties. Some veterinary dermatologists treat *Malassezia* dermatitis with a lower dose of itraconazole (2.5 mg/kg/day), because of these cuta-
neous properties. Recently, itraconazole was shown to be effective when given at a dose of 5 mg/kg/day for two consecutive days a week for three weeks. Fluconazole also remains at high concentrations in the skin and nails in people. Absorption of itraconazole capsules (but not itraconazole solution or fluconazole) is increased with food. Itraconazole is metabolized in the liver and excreted in the urine and feces, while fluconazole is excreted in the urine relatively unchanged. Because of itraconazole’s metabolism, document serum liver enzyme activities before its use. Both of these drugs have fewer adverse effects and lower risks for drug interactions than ketoconazole, but they are considerably more expensive. Anorexia is the most common adverse effect with both drugs.

Griseofulvin has no effect against Malassezia species.

Follow-up examinations
Every three or four weeks, perform follow-up examinations on patients receiving topical or systemic therapy. Note the clinical response, and collect samples from new and old skin lesions for cytologic examination. Before deciding to continue therapy, compare the most recent cytologic results with those of the last and the initial visit. Monitor serum liver enzyme activities if the patient will be maintained on systemic therapy. Frequent reexamination may reduce the duration of therapy and any unforeseen adverse effects. Continue systemic antifungals until clinical signs resolve or until there is no cytologic evidence of yeast. Topical therapy should be continued beyond resolution and may be used in a prophylactic manner thereafter.

Zoonotic potential
Malassezia pachydermatis was identified recently in a human intensive care nursery. The yeast was thought to have been transmitted to 15 infants from the contaminated hands of dog-owning healthcare workers. So consider M. pachydermatis to be a potential zoonotic agent, especially in immunoincompetent or immunocompromised individuals.

Summary
Malassezia pachydermatis is a commensal of canine skin. It becomes an opportunistic pathogen when normal cutaneous conditions are altered, favoring its growth. A patient’s history,
clinical signs, response to previous therapy, and cytologic examination findings are all used in diagnosing Malassezia dermatitis. It is paramount to diagnose and treat any underlying predisposing factors. Tailor treatment options, whether topical or systemic therapy or both, to the individual.

REFERENCES

CE Questions

You can earn two hours of Continuing Education credit from Kansas State University by answering the following questions on Malassezia dermatitis in dogs. Circle only the best answer for each question, and transfer your answers to the form on page 624.

Article #2

1. Malassezia pachydermatis can be found in or on the ________ of clinically normal dogs.
   a. Rectum
   b. Lip
   c. Ear canal
   d. Chin
   e. All of the above

2. Which factor is not favorable for yeast overgrowth?
   a. Cutaneous inflammation
   b. Low humidity
   c. Cutaneous erosions
   d. Seborrhea
   e. Allergies

3. Which clinical sign is associated with Malassezia dermatitis?
   a. Interdigital skin lesions
   b. Pruritus
   c. Lichenified skin lesions
   d. Greasy skin lesions
   e. All of the above

4. Which of the following would not be a differential diagnosis for Malassezia dermatitis based on skin lesions and clinical signs?
   a. Sarcoptic mange
   b. Food allergy
   c. Epitheliotropic lymphoma
   d. Pemphigus vulgaris
   e. Seborrheic dermatitis

5. Which of the following is the diagnostic method of choice for clinicians trying to rule out Malassezia dermatitis?
   a. Histopathologic examination
   b. Culture
   c. Cytologic examination
   d. Fluorescence with a Wood's lamp
   e. Trichography

6. Malassezia pachydermatis has been described as ________-shaped organisms when examined under light microscopy.
   a. Hot dog
   b. Peanut
   c. Block
   d. Footprint
   e. Both B and D

7. Which topical agent does not have good degreasing activity?
   a. Miconazole
   b. Benzoyl peroxide
   c. Selenium sulfide
   d. Tar shampoo
   e. All of the above

8. Which of the following is primarily a top-
ical antiseborrheic agent?
- Chlorhexidine
- Salicylic acid
- Miconazole
- Oatmeal
- White vinegar and water

9. Which of the following systemic antifungal agents is ineffective in treating *Malassezia* dermatitis?
- Fluconazole
- Ketoconazole
- Itraconazole
- Griseofulvin
- Both C and D

10. Ketoconazole may cause __________.
- Anorexia
- Vomiting
- Elevated serum liver enzyme activity
- Suppressed adrenal cortisol production
- All of the above

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