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ISSUES IN DERMATOLOGY

A Clinical Approach to Alopecia in Cats

***Cherie Pucheu-Haston**, DVM, PhD, DACVD*
Louisiana State University School of Veterinary Medicine

One of the more common dermatologic complaints for which clients seek veterinary attention for their cat is fur loss. Unfortunately, the lack of diagnostic specificity for this condition can prove challenging, especially if the client insists on a quick fix. It is beyond the scope of this article to describe in detail the clinical appearance and treatment of each of the various causes of alopecia in cats. Instead, the article lists common differentials and provides a logical algorithm for approaching these cases.

In the author's opinion (and the opinions of others), the single most important diagnostic tool for any skin disease (including alopecia) is a thorough history.[1] The following information is especially useful for pointing the practitioner toward certain categories of disease.

- Onset at a young age or exposure to other animals may suggest infectious or parasitic diseases such as dermatophytosis, *Otodectes*, and lice.[2-4]
- Dermatitis in other animals and/or humans in contact with the patient is suggestive of some form of contagious disease (e.g., dermatophytosis, ectoparasitism).[4,5]
- Onset in older patients may suggest hyperthyroidism or paraneoplastic alopecia.[6,7]
- A very recent significant illness may suggest anagen defluxion, whereas history of significant physiologic stress 1 to 3 months before alopecia onset may suggest telogen effluvium.[8-11]

It is helpful to categorize alopecia in cats as spontaneous or self-inflicted and to further subdivide self-inflicted alopecia into pruritic or nonpruritic. These categories can help focus the initial diagnostic approach and decrease the number of potential differentials (**FIGURE 1**).

In the author's experience, spontaneous alopecia is much less common than self-inflicted alopecia in cats, especially if the affected area is diffuse or widespread (**FIGURE 2**). In some cats, spontaneous alopecia can be readily demonstrated; large amounts of fur can be easily epilated by gentle tugging, especially at the edges of the alopecic area.[12,13] In the absence of such evidence, the distinction from self-inflicted alopecia can be more challenging. Clients can be

FUR-ENSIC INVESTIGATION
The workup of alopecia in cats involves a combination of thorough history, categorization, and use of targeted investigative techniques.

asked whether they have noticed the cat excessively licking, biting, or pulling at the fur or if they have seen the cat scratching. Although the client may be able to answer in the affirmative, a negative answer does not rule out self-trauma because clients may mistake these actions for normal behavior. Furthermore, some cats may prefer to groom when they are alone. You can ask clients if the cat vomits frequently (and if they have noticed fur in the vomitus), has appreciable amounts of fur in the feces, or has been constipated. Gentle brushing of the hand against the fur in the affected areas may facilitate the identification of short, broken "stubble" fur. Examination of this short fur under a microscope may demonstrate frayed or broken ends. For extreme cases, an Elizabethan collar can be placed on the cat for 2 to 3 weeks; the presence of fresh fur growth at the end of this period strongly indicates that the alopecia is indeed self-inflicted. If so, you then need to determine whether the self-inflicted condition is pruritic or nonpruritic.

SELF-INFLICTED ALOPECIA

The most common causes of self-inflicted alopecia are pruritic rather than nonpruritic, although both causes can overlap.[14] Because true cases of nonpruritic self-inflicted alopecia are relatively scarce, it is not unreasonable to preliminarily assume pruritic disease until demonstrated otherwise. This assumption may be further supported by a good clinical response to a 3- to 4-week course of medium to high anti-inflammatory doses of glucocorticoids or other antipruritic agents.[14]

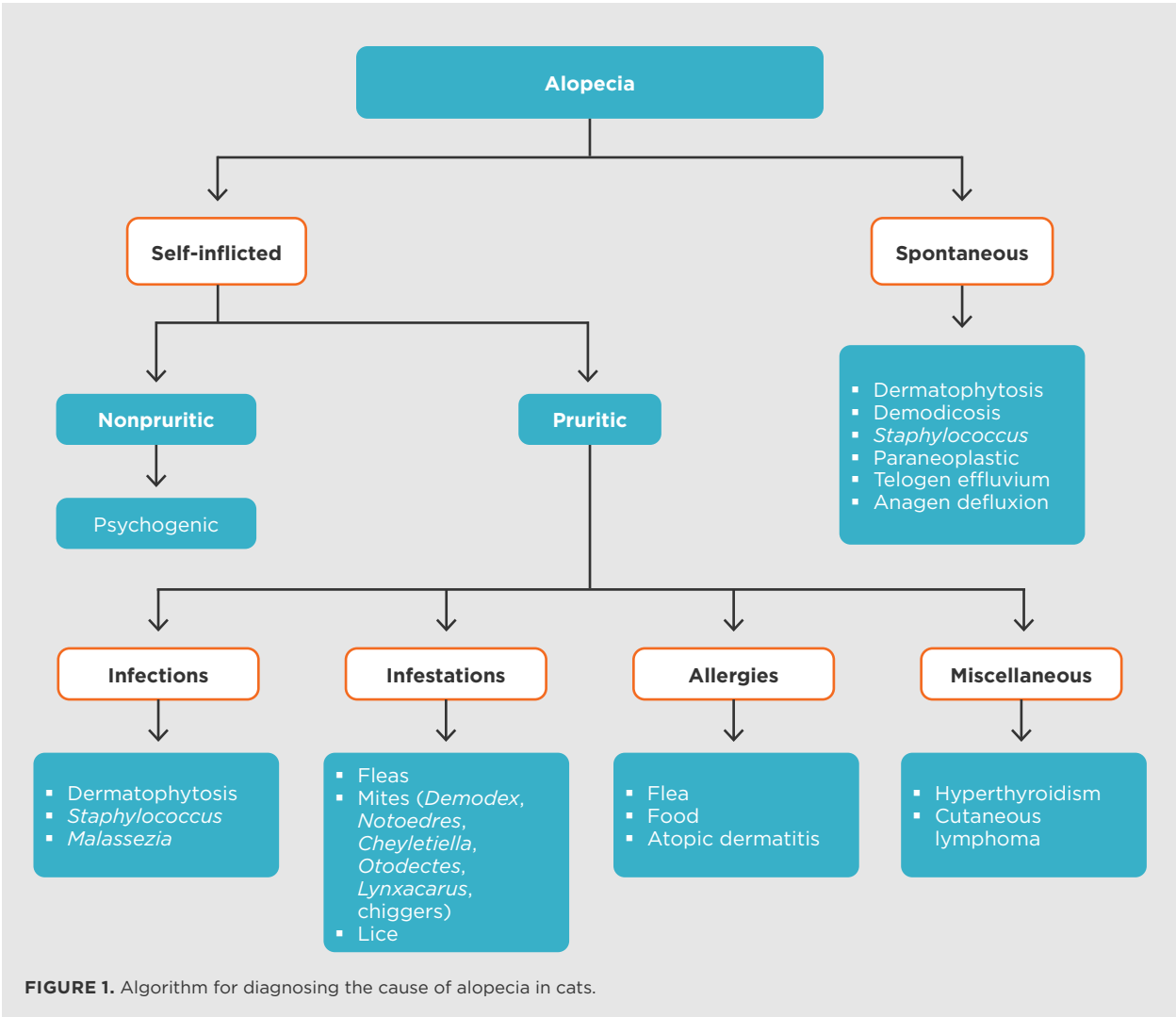

FIGURE 1. Algorithm for diagnosing the cause of alopecia in cats.

Pruritic Self-Inflicted Causes of Alopecia

After you have established self-inflicted alopecia caused by pruritus, the next question is whether the pruritus is caused by infection, infestation (e.g., ectoparasites), or hypersensitivity. Although most causes of pruritus in cats fall into 1 or more of these 3 categories, other causes are occasionally encountered (for more, see **TABLE 1** at todaysveterinarypractice.com/alopecia-diagnosis-cats).[10] At first glance, this categorization might seem to be useless because of the large number of potential differentials, especially in the first 2 categories. However, a relatively small number of inexpensive, rapid, and easily performed diagnostic tests can be used to simultaneously identify (or exclude) most of these conditions.

Identify and eliminate the easy causes first. Although many (or even most) cats with pruritic alopecia experience some form(s) of cutaneous hypersensitivity, leaping directly into a hypersensitivity disease workup is generally not the most efficient way to approach the pruritic cat for the following reasons:[10,15]

- Identification of infectious and parasitic causes of pruritus is relatively straightforward, whereas properly conducting a hypersensitivity workup can take months.
- The sensation of pruritus may be thought of as being additive. A hypersensitive cat with a secondary infection may be considerably more pruritic than a cat experiencing hypersensitivity alone. Fortunately, the reverse is also true, and the elimination of infection or parasitic disease may considerably improve the cat's condition and allow a more accurate estimation of the baseline level of pruritus.
- Infectious and parasitic causes of pruritus are generally curable, whereas most hypersensitivity disorders are manageable at best.

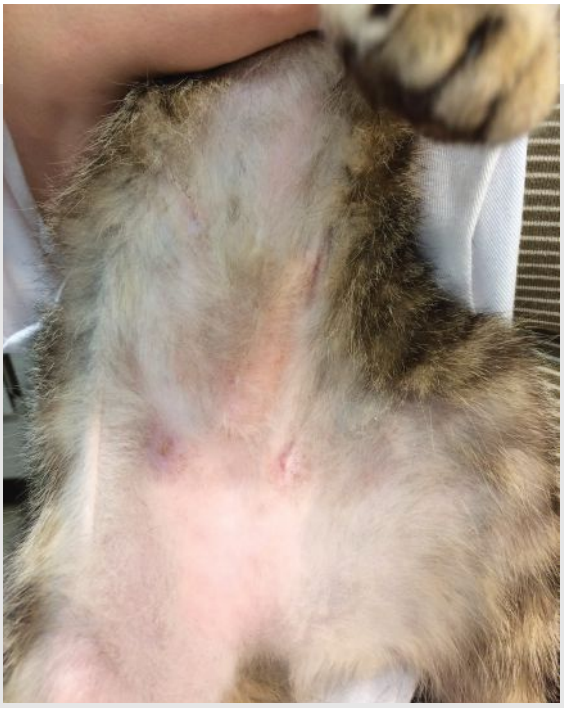

FIGURE 2. Self-inflicted alopecia on ventral abdomen of an overgrooming cat.

Infection

The 3 main infectious organisms associated with pruritus in the cat are dermatophytes (**FIGURES 3,4**), staphylococci, and yeast (particularly *Malassezia).*[10,16] With the exception of dermatophytosis, infectious causes of pruritus in cats are often secondary. Nonetheless, bacteria and yeast can contribute to cutaneous inflammation and enhance pruritus by serving as sources of pathogen-associated molecular patterns, proteolytic enzymes, and exotoxins.[17]

Parasites

Cats may be infested with a wide variety of ectoparasites, any of which can cause pruritus. Surface-dwelling parasites include fleas, lice (*Felicola subrostratus*) (**FIGURES 5–7**), *Cheyletiella* species,

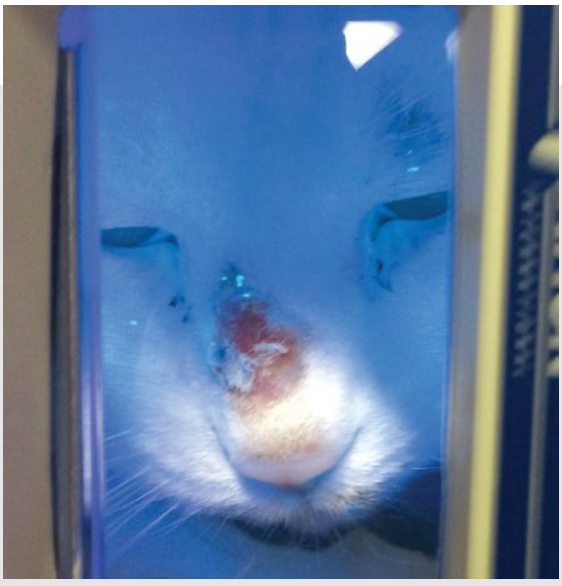

FIGURE 3. Positive Wood's lamp response: "apple green" fluorescence of fur of a cat with *Microsporum canis* infection.

fur mites (*Lynxacarus*), and chiggers (*Eutrombicula*, *Neotrombicula* species)[18-20] (**FIGURES 3,4**). *Otodectes* mites may leave the ear canals and transiently infest the skin surface.[21] Superficial-dwelling parasites include *Notoedres* (i.e., feline scabies; **FIGURES 8,9**), *Demodex gatoi*, and occasionally the unnamed feline *Demodex* mite.[21,22] The only common representative of the deep-dwelling parasite group is *Demodex cati*. Fleas may contribute to pruritus through nonspecific inflammatory responses (e.g., the unpleasant sensation associated with the fleas crawling on the skin) as well as true hypersensitivity responses.

Cutaneous Hypersensitivity

Cats, like dogs, can experience a variety of pruritic cutaneous hypersensitivities. The 3 most common hypersensitivities typically associated with significant alopecia in cats are flea hypersensitivity, food hypersensitivity, and feline atopic skin syndrome (hereafter referred to as atopic dermatitis).[15] With the exception of flea hypersensitivity (which may also be considered a parasitic cause of pruritus), diagnostic pursuit of these differentials is often deferred until after infectious or parasitic conditions are controlled or eliminated. Compared with most infectious and parasitic conditions, diagnosis of hypersensitivity-associated pruritus is typically a bit more involved. The diagnostic approaches for each of the 3 major cutaneous hypersensitivities in cats are discussed below.

Flea hypersensitivity: Generally, the first step in a hypersensitivity workup is elimination of fleas, assuming that aggressive flea control has not already been instituted as part of the workup for parasitic causes of pruritic alopecia. Although practitioners in areas where fleas are not commonly found may be tempted to skip this step, a good faith effort must be made to eliminate the possibility of flea infestation because few geographic areas can truly be considered flea free.[23] Fortunately, modern flea control agents have made elimination of fleas possible in all but the most challenging circumstances. A detailed discussion of the available agents and their selection is beyond the scope

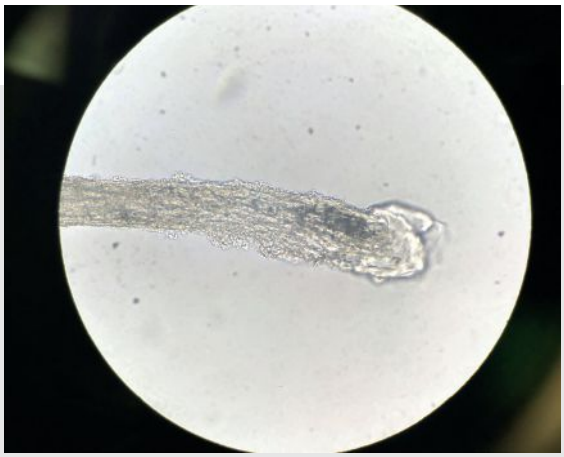

FIGURE 4. Distorted, broken shaft of hair affected by dermatophytes, showing indistinct edges lined with fungal elements.

FIGURE 5. Flea comb, demonstrating large amounts of "flea dirt," a flea **(orange arrow)**, and several lice **(blue arrows)**.

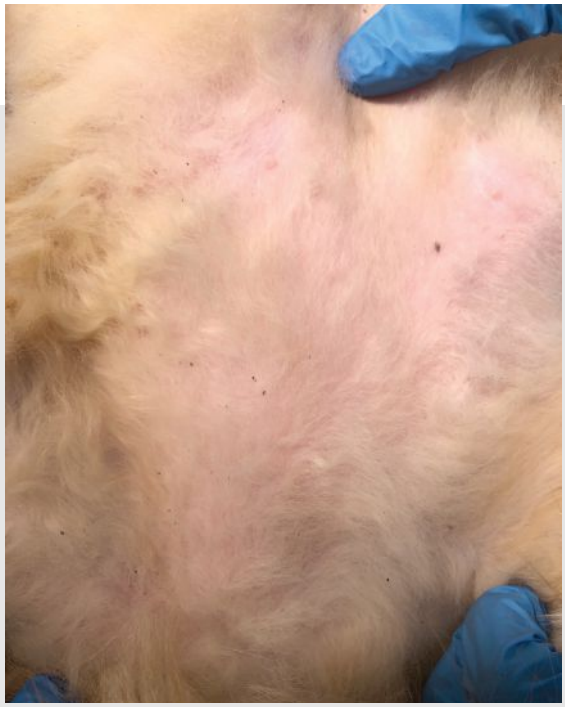

FIGURE 6. Self-inflicted alopecia on a flea-infested cat, showing scattered "flea dirt" and 1 flea.

Top left: Courtesy of Dr. Chi-Yen Wu (Louisiana State University School of Veterinary Medicine, Department of Veterinary Medical Sciences).

of this article, but other resources are available.[24] Regardless of the agent(s) chosen, treatment must be maintained for a minimum of 4 to 6 weeks to be certain that the local flea population has been eliminated. All in-contact animals should also be treated, regardless of whether they are demonstrating clinical signs. Free-roaming cats should be confined for the duration of the treatment trial. Environmental flea control measures may include frequent vacuuming of the area to which the cat has access and spraying the household with insect growth regulators (such as pyriproxyfen). If dogs share the household, measures should be taken to ensure that they do not come into contact with open sheds, crawl spaces, etc., which may be "reseeded" by wildlife that may also access these areas.[25]

Food hypersensitivity: Despite the plethora of alternate diagnostics (e.g., IgE serology, quantification of fecal or salivary IgE), the only valid way to diagnose food hypersensitivity and identify the culprit foods in cats is controlled dietary elimination trials.[26] Elimination diets may contain novel proteins (whether home cooked or commercial) or hydrolyzed proteins (low molecular weight peptides that can be absorbed by the digestive tract with a reduced risk of triggering an immune reaction).

- If a **novel protein** diet is selected, the ingredients should be carefully selected from sources phylogenetically distant from the patient's normal diet ingredients. For example, a rabbit-based diet may be an acceptable empirical choice for a cat normally fed a fish-based diet.
- If a **hydrolyzed protein** diet is selected, ultrahydrolyzed diets (fragments of 1 to 2 kilodaltons) are considered superior to semihydrolyzed diets (fragments of ~10 kilodaltons) for diagnostic purposes.[27,28]

There is no consensus on the required length of a diet trial. A recent review article recommends a minimum of 8 weeks, with 10 to 12 weeks required to identify 90% of cases.[29] For at least the first few weeks of the trial, it may be prudent to provide the patient with anti-inflammatory/antipruritic support (such as prednisolone). Doing so may be advisable not only for

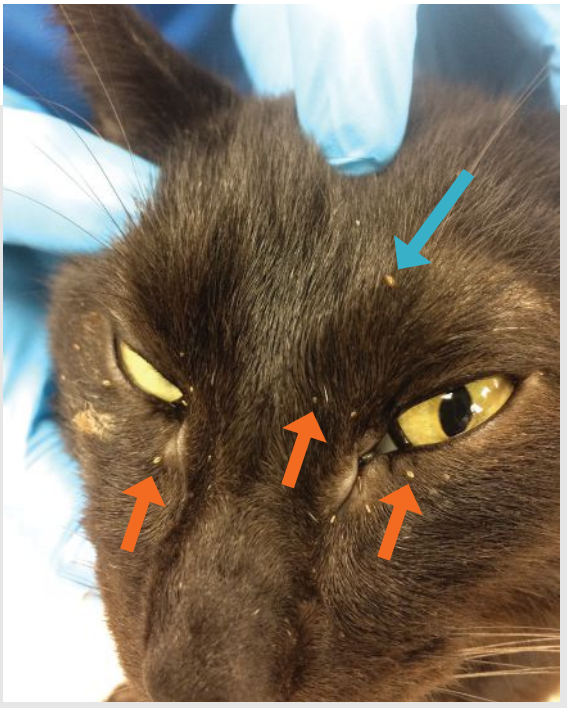

FIGURE 7. Cat with lice **(orange arrows)** and fleas **(blue arrow)**.

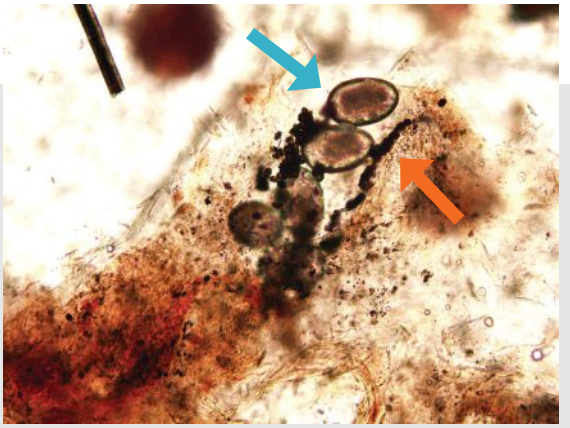

FIGURE 8. *Notoedres cati* eggs **(blue arrow)** and granular brown feces **(orange arrow)** from a superficial skin scraping of a cat infested with *Notoedres* mites.

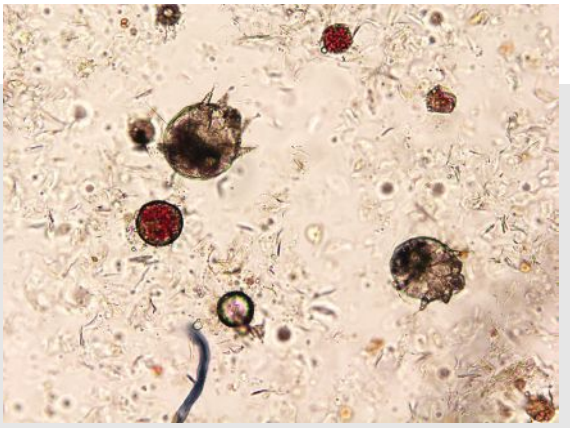

FIGURE 9. *Notoedres cati* mites obtained by superficial skin scraping.

patient welfare but also because some evidence suggests that early suppression of cutaneous inflammation may shorten the necessary duration of the diet trial.[30]

Even if the cat does not become asymptomatic, all elimination diets should ideally be followed by rechallenge with the cat's previous diet. Exacerbation of clinical disease after challenge both confirms the diagnosis of food hypersensitivity in asymptomatic cats and demonstrates a food hypersensitivity component in cats that showed limited improvement during the trial.

Atopic dermatitis: Hypersensitivity testing is often mistakenly used to diagnose atopic dermatitis. However, there is currently no in vivo or in vitro test that can clinically distinguish between atopic and nonatopic cats.[31,32] As is already known in dogs with atopic dermatitis, cats may also have clinically irrelevant positive reactions or may have false-negative reactions for a variety of reasons (e.g., stress during testing, drug interference). Atopic dermatitis is thus considered to be a clinical diagnosis of exclusion and should be made only after all other factors have

TABLE 2 Diagnostics Useful in the Workup of Cats With Spontaneous Fur Loss

DIAGNOSTIC TOOLS	EXPECTED FINDINGS	COMMENTS	LIMITATIONS
FOLLICULITIS			
Wood's lamp examination, dermatophyte culture, examination of plucked hairs, fungus PCR	Dermatophyte infection	See **TABLE 1** online at todaysveterinarypractice.com/alopecia-diagnosis-cats.	
Skin cytology	Presence of bacteria	See **TABLE 1** online.	
Deep skin scraping	*Demodex cati* or the unnamed *Demodex* mite	See **TABLE 1** online.	
PHYSIOLOGIC STRESS			
Targeted clinical history[1]	Anagen defluxion	Abrupt hair loss appears soon (days to weeks) after a physiologic stress or cytotoxic drug administration.	Close chronologic association between stressor and hair loss facilitates diagnosis.
	Telogen effluvium	Diffuse, noninflammatory hair loss 1–3 months after severe physiologic stress is suggestive of telogen effluvium.	Delayed appearance of alopecia can complicate association with the inciting event.
Examination of plucked hairs[1]	Anagen defluxion	Hairs may be deformed, narrowed, or broken.	
	Telogen effluvium	Most or all of the hairs may be in telogen.	Normal hairs may be seen if regrowth has already begun.
Skin biopsy[1]	Anagen defluxion	Hairs and hair follicles may appear dysplastic or deformed.	Not always seen
	Telogen effluvium	Biopsy soon after alopecia begins may demonstrate synchronization of the hairs in telogen.	Biopsy samples taken later may demonstrate only empty follicles or actively regrowing hairs.
TUMOR-ASSOCIATED HAIR LOSS			
Thoracic imaging[1,2]	Thymoma-associated alopecia	Imaging may demonstrate a cranial mediastinal tumor.	
Abdominal radiographs or ultrasound[3,4]	Paraneoplastic alopecia	Abdominal radiographs or ultrasound may demonstrate a pancreatic or hepatic mass.	The underlying tumor is not always immediately apparent.
Skin biopsy[1,3,4]	Thymoma-associated alopecia	May see scaling, individual keratinocyte death, sebaceous gland atrophy, inflammation at dermal–epidermal junction and around hair follicles.	These findings are not completely specific; interpretation should be made in the context of the clinical appearance.
	Paraneoplastic alopecia	May see follicular miniaturization and telogenization, epidermal thickening, occasional yeast overgrowth.	

been eliminated or identified. The true purpose of hypersensitivity testing is to identify potential allergenic triggers so that they can be avoided or desensitization can be attempted. Both intradermal testing and IgE serology are valid tools for these purposes. The methods used in serologic assays vary greatly between laboratories and can significantly affect study results.[33] The practitioner is advised to seek the advice of a veterinary dermatologist before selecting a laboratory.

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Other Causes of Pruritus

Hyperthyroidism: Although hyperthyroidism may not be the first differential that comes to mind for an alopecic cat, it can nonetheless be associated with pruritus and alopecia secondary to overgrooming.[6] Affected cats are typically older and also demonstrate other clinical signs of hyperthyroidism. However, the author has seen one cat in which the first sign noted was fur pulling.

Cutaneous lymphoma: Epitheliotrophic cutaneous lymphoma may occasionally be associated with pruritic (or occasionally nonpruritic) alopecia, often accompanied by scaling and erythema.[34] Affected cats are typically mature to older. Depending upon the extent of the disease, cats may or may not be clinically ill at the time of presentation.

Nonpruritic Self-Inflicted Causes of Alopecia

The most well-known cause of nonpruritic self-inflicted alopecia is psychogenic disease. Affected cats methodically or episodically lick or bite at their fur in reaction to a psychologic stressor. Evidence suggests that psychogenic alopecia is widely overdiagnosed, and it may be the least common of the potential differentials for alopecia. In a recent study, only 2 of 21 cats presented for evaluation of "psychogenic alopecia" were found to have a behavioral disorder as their only problem. In contrast, 16 of those cats were found to have medical abnormalities (e.g., food hypersensitivity or atopy) and the other 3 cats were found to have both psychogenic and medical causes for their overgrooming.[14] Indeed, cats may first have a medical abnormality and then a later, secondary obsessive or behavioral component to their disease (possibly secondary to endorphin release), similar to what is believed to occur in some dogs with acral lick dermatitis.

For this reason, in the absence of chronologic association with the appearance of an obvious stressor (e.g., new house, addition or loss of a family member, stressors associated with poor social status), psychogenic alopecia is usually considered only when pruritic self-inflicted alopecia is ruled out. However, failure to respond to an antipruritic agent trial may lend support to the possibility of psychogenic disease and may lead practitioners to consider investigating this differential sooner than they otherwise might. Recently, Titeux et al. developed a welfare score to aid assessment of cats with idiopathic ulcerative dermatitis.[35] In their study, identification and elimination of stressors and environmental enrichment resulted in the near-immediate cessation of this previously chronic, treatment-refractory disorder. Indeed, adaptation of this score for the evaluation of cats with suspected psychogenic alopecia may be of considerable diagnostic and therapeutic value. For an in-depth workup, the practitioner is encouraged to seek the advice of a qualified veterinary behaviorist.

SPONTANEOUS ALOPECIA

Compared with self-inflicted alopecia, significant spontaneous fur loss in the cat is fairly uncommon and the potential causes are relatively few (**TABLE 2**). Fur loss may result from fungal, bacterial, or parasitic folliculitis, in which irritation of the hair follicle causes damage to and/or premature shedding of the hairs. Affected cats may experience significant fur loss without demonstrating evidence of pruritus (e.g., scratching, overgrooming, broken hair). Significant physiologic stress may result in acute (anagen defluxion) or delayed (telogen effluvium) fur loss.[8]

Less commonly, spontaneous alopecia may represent a cutaneous manifestation of systemic neoplastic disease (**FIGURES 10,11**). Some cats with thymomas will

develop marked exfoliative scaling associated with progressive fur loss and easily epilated fur.[36] Another variant of alopecic dermatitis has been associated with other forms of neoplasia (most frequently pancreatic, hepatocellular, or bile duct carcinomas).[37,38] Cats affected by any of those 3 tumors may rapidly shed clumps of fur, particularly on the ventrum. Histologically, severe follicular atrophy and hair bulb miniaturization can be seen.[38,39] Secondary infection with *Malassezia* is not uncommon and may be associated with significant pruritus.[40] Infected cats may obsessively groom their bellies, resulting in the development of shiny skin. These cats are frequently systemically ill at the time of presentation.

Similar to workups for cats with pruritic skin disease, it is prudent to begin the workup of the cat with spontaneous alopecia by first focusing on more common causes and leaving the more esoteric entities until after the common conditions have been identified or eliminated. Of note, some cats may have more than one problem (e.g., secondary overgrowth with *D cati* may develop in a cat with paraneoplastic alopecia).

CONCLUSIONS

The workup of alopecia in cats involves a combination of thorough history, categorization, and use of targeted investigative techniques. Although there are a large number of differential diagnoses for feline alopecia, a very large proportion of these differentials can be identified or excluded by using a relatively small number of inexpensive, rapid, and easy diagnostic techniques. TVP

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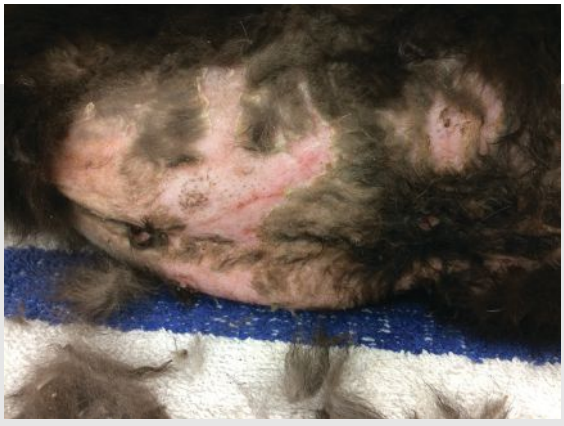

FIGURE 10. Cat with paraneoplastic alopecia associated with internal malignancy. Note the smooth, furless areas. Tufts of fur on the towel were epilated by gentle tugging.

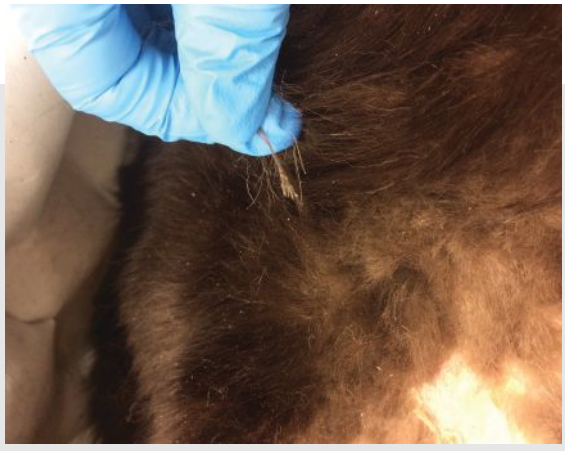

FIGURE 11. Easily epilated fur in a cat with paraneoplastic alopecia.

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Cherie Pucheu-Haston

Dr. Pucheu-Haston is an associate professor of veterinary dermatology and immunology at Louisiana State University. She received her veterinary degree from Louisiana State University and completed residency training at North Carolina State University. After a brief hiatus in private practice dermatology, she returned to NCSU to pursue a PhD in immunology, followed by 3 years of postdoctoral study at the U.S. Environmental Protection Agency's Immunotoxicology Branch. Her clinical and research interests are cutaneous and respiratory allergic diseases in cats, dogs, and horses and the immunologic response to cutaneous fungal infections.

Osurnia®
(florfenicol-terbinafine-betamethasone acetate)

Otic gel
For Otic Use in Dogs Only

CAUTION: Federal (USA) law restricts this drug to use by or on the order of a licensed veterinarian.

BRIEF SUMMARY (for full prescribing information, see package insert)

DESCRIPTION: OSURNIA contains 10 mg florfenicol, 10 mg terbinafine and 1 mg betamethasone acetate per mL and the inactive ingredients propylene carbonate, glycerol formal, hypromellose, phospholipid, oleic acid and BHT in an off-white to slightly yellow translucent gel.

INDICATION: OSURNIA is indicated for the treatment of otitis externa in dogs associated with susceptible strains of bacteria (*Staphylococcus pseudintermedius*) and yeast (*Malassezia pachydermatis*).

DOSAGE AND ADMINISTRATION: OSURNIA should be administered in the clinic. Clean and dry the external ear canal before administering the initial dose of the product. Administer one dose (1 tube) per affected ear(s) and repeat administration in 7 days. Do not clean the ear canal for 45 days after the initial administration to allow contact of the gel with the ear canal. Cleaning the ear may affect product effectiveness (see **Effectiveness** in the product insert). If alternative otic therapies are required it is recommended to clean the ear(s) before application. Open tube by twisting the soft tip. Insert the flexible tip into the affected external ear canal(s) and squeeze entire tube contents into the external ear canal(s). After application, gently massage the base of the ear to allow the gel to penetrate to the lower part of the ear canal.

CONTRAINDICATIONS: Do not use in dogs with known tympanic perforation (see **Precautions** in the product insert). Do not use in dogs with a hypersensitivity to florfenicol, terbinafine, or corticosteroids.

WARNINGS: Not for use in humans. Keep this and all medications out of reach of children. Consult a physician in case of accidental ingestion by humans. In case of accidental skin contact, wash area thoroughly with water. Avoid contact to the eyes.

PRECAUTIONS: Do not administer orally. The use of OSURNIA in dogs with perforated tympanic membranes has not been evaluated. The integrity of the tympanic membrane should be confirmed before administering this product. Reevaluate the dog if hearing loss or signs of vestibular dysfunction are observed during treatment. Use of topical otic corticosteroids has been associated with adrenocortical suppression and iatrogenic hyperadrenocorticism in dogs (see **Animal Safety** in the product insert). Use with caution in dogs with impaired hepatic function (see **Animal Safety** and **Adverse Reactions** in the product insert). The safe use of OSURNIA in dogs used for breeding purposes, during pregnancy, or in lactating bitches, has not been evaluated.

ADVERSE REACTIONS: The following adverse reactions were reported during the course of a US field study for treatment of otitis externa in dogs treated with OSURNIA with 1 tube per affected ear(s) and repeated after 7 days. The following adverse events are listed in decreasing order: elevated alkaline phosphatase, vomiting, elevated AST, ALT, ALP, weight loss (>10% body weight), and hearing decrease/loss. To report suspected adverse events, for technical assistance or to obtain a copy of the SDS, contact Dechra Veterinary Products at (866) 933-2472. For additional information about adverse drug experience reporting for animal drugs, contact FDA at 1-888-FDA-VETS or online at http://www.fda.gov/AnimalVeterinary/SafetyHealth.

EFFECTIVENESS: Effectiveness was evaluated in 235 dogs with otitis externa. The study was a double-masked field study with a placebo control (vehicle without the active ingredients). One hundred and fifty-nine dogs were treated with OSURNIA and seventy-six dogs were treated with the placebo control. All dogs were evaluated for safety. Treatment (1 mL) was administered to the affected ear(s) and repeated 7 days later. Prior to the first administration, the ear(s) were cleaned with saline but not prior to the Day 7 administration. Six clinical signs associated with otitis externa were evaluated: pain, erythema, exudate, swelling, odor and ulceration. Total clinical scores were assigned for a dog based on the severity of each clinical sign on Days 0, 7, 14, 30 and 45. Success was determined by clinical improvement at Day 45. The success rates of the two groups were significantly different (p=0.0094); 64.78% of dogs administered OSURNIA were successfully treated, compared to 43.42% of the dogs in the placebo control group.

STORAGE: OSURNIA should be stored under refrigerated conditions between 36° - 46° F (2° - 8° C). To facilitate comfort during administration, OSURNIA may be brought to room temperature and stored for up to three months.

MANUFACTURED FOR:
Dechra Veterinary Products
7015 College Boulevard, Suite 525
Overland Park, KS 66211 USA

Product of Great Britain

NADA # 141-437, Approved by FDA

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